



**The Washington
Manual of Surgery**
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The Washington
Manual of Surgery

Full Text, NO Tables, NO Figures by Mandrake-GN

Version: Full Images (1.5MB)

Platform: PalmOS

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Recommended: 33MHz PalmOS v4.0 or later PDA w/ expansion and hi-res, 16-bit display.

- Greetings -

Respect goes to MedScut (the iSilo king), the PDA
People, xylarr, taihenchen, ZD, the "group" moderators and those that have
made and still make the Skyscape stuff possible.

p.s. thanks to **How, Guiga, Super Falcão**, the "Brazilian PalmBoys"

- Comments -

Send your constructive comments/criticisms to: hifixz@yahoo.com.br

Dedication

To the patients who have taught us
how to best care for them, with the
hope that we will remember the
lessons of the past and recognize
the lessons of the future

Foreword

This is the third edition of *The Washington Manual of Surgery*. This effort reflects the ongoing commitment of the Department of Surgery at Washington University to medical education both within and outside the institution. This commitment to resident and medical student education began with our first full-time Chairman of Surgery, Everts A. Graham (1919–1951). Not only did Dr. Graham help to found the American Board of Surgery, but he was also instrumental in several other areas. He developed new surgical techniques (including the one-stage pneumonectomy), collaborated to develop oral cholecystography with Mallinckrodt Institute of Radiology investigators, and utilized an epidemiologic approach to make the first link of cigarette smoking to lung cancer development.

Dr. Carl Moyer (1951–1965), who was regarded as a superb educator, continued Dr. Graham's legacy. Dr. Walter Ballinger (1967–1978) brought with him the Hopkins tradition of resident education and a focus on the importance of the surgeon/scientist. Dr. Samuel A. Wells, Jr. (1978–1997) was responsible for assembling a world-class faculty and significantly increasing the number of surgeon/scientists within the department. Dr. Wells emphasized basic and translational research and placed a great emphasis on educating academic leaders of surgery. Dr. Wells is currently the Group Chair and Principal Investigator of the American College of Surgeons Oncology Group (ACOSOG).

In keeping with this rich tradition, residents of the Department of Surgery authored this third edition of *The Washington Manual of Surgery*, with each resident assisted by a senior faculty co-author. This edition of the manual provides a complete reference that can be utilized by medical students, house officers, and practicing surgeons, presenting brief and logical approaches to the management of patients with various surgical problems. The manual does not attempt to extensively cover pathophysiology or history, but does provide the most up-to-date and important diagnostic and management information for a given topic. There are selected references included in each chapter that the reader may use to further their education about the topic. The manual is also standardized with respect to structure so that the reader will be able to most easily obtain information.

Dr. Gerard M. Doherty continues to serve as the principal editor of the manual. Four associate editors, each of whom is currently a chief resident in the Department of Surgery, assist Dr. Doherty. The manual begins with general care of the surgical patient that includes areas of general knowledge such as nutrition, life support, perioperative medical care, and critical care management. The second section is devoted to the evaluation of abdominal diseases as well as gastrointestinal surgical disease. The third section includes discussions on vascular disease, endocrine surgery, trauma, and transplantation surgery. The final section of the manual is devoted to the surgical subspecialties and unique problems associated with them as well as common surgical procedures. This edition also has an appendix of commonly used laboratory values and formulas.

I am extremely proud of the residents and faculty who have done such an outstanding job in this third edition. They have made this edition easier to read, which I hope will result in it becoming a more convenient reference for the management of surgical diseases. It is a fine example of the unique collegiality that exists in our department between faculty and residents and of our commitment to surgical education. I hope that you will find this edition an often-used reference.

Timothy J. Eberlein, M.D.

Preface

This third edition of *The Washington Manual of Surgery* has been designed to complement *The Washington Manual of Medical Therapeutics*. This book was written by members of the Department of Surgery and presents a brief, rational approach to the management of patients with surgical problems. The text was directed to the reader at the level of the second- or third-year surgical house officer, although surgical and nonsurgical attendings, medical students, physician assistants, and others who provide care for patients with surgical problems will find it of interest and assistance. The book provides a succinct discussion of surgical diseases, with algorithms for addressing problems based on the opinions of the physician authors. Although multiple approaches may be reasonable for some clinical situations, this manual attempts to present a single, effective approach for each. We have limited coverage to diagnosis and therapy; this is not an exhaustive surgical reference. Coverage of pathophysiology, the history of surgery, and extensive reference lists have been specifically excluded from most areas. This third edition of the manual, which was published initially in 1997, followed by a second edition in 1999, includes updated coverage of each topic, as well as substantial new material.

This is a resident-prepared manual. Each chapter was revised by a resident with assistance from a faculty co-author. The project was separated into four subsections; editorial oversight was performed for each section by one of the four chief resident co-editors (John E. Mason, M.D., Chapters 1–10; Jennifer K. Lowney, M.D., Chapters 11–21; Michael A. Smith, M.D., Chapters 22–32; and Scott I. Reznik, M.D., Chapters 33–43 and the Appendixes). The tremendous effort of all involved residents and faculty members, and particularly the chief resident co-editors, is reflected in the quality and consistency of the chapters.

I am grateful for the invaluable assistance of Lisa Williams, who has served unfailingly as the editorial coordinator; she has kept each of us in line. Lisa McAllister and Lisa Consoli from Lippincott Williams & Wilkins have been understanding and encouraging as they carried the project through to production. We are very fortunate to have such outstanding support from the publisher, which allows the attention to detail necessary for such a project.

Finally, we have all benefited from the guidance of an outstanding department chair during the production of this book. Timothy J. Eberlein, M.D., has actively participated in the book by serving as senior author for two chapters and has served as an inspiration for each of us by his leadership and example of hard work and honesty.

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1 General Care of the Patient

Nahush A. Mokadam and Jeffrey F. Moley

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Introduction

Patient care is not only dependent on a fund of medical knowledge but also on appropriate and accurate documentation of that care. Proper documentation ensures communication with other physicians as well as administrators and provides the basis for all reimbursement. It is imperative for all documentation to include a date, time, and signature.

Patient Care

I. Hospital orders

- A. **Admission orders** detail every aspect of a patient's care for the administration, nursing staff, pharmacy, and ancillary care services. The mnemonic **ABC DAAVVIDD** is a simple device to signify routine admission, postoperative, and transfer orders.
1. **Admit.** Include nursing division, surgical service, attending physician, and admission status (in-patient vs. 23-hour observation).
 2. **Because (diagnosis).** The principal diagnosis and, if relevant, care path. Any operations performed should also be included.
 3. **Condition.** Distinguish between good, satisfactory, serious, and critical.
 4. **Diet.** Include diet type (regular, American Diabetes Association, renal, etc.) and consistency (clear liquids, full liquids, pureed, etc.) as well as supervision instructions, if applicable. Also include "ins and outs."
 5. **Allergies.** Include specific reactions if known.
 6. **Activity.** Include necessary supervision and weightbearing status, if applicable.
 7. **Vitals and other nursing orders.** Include frequency and special instructions, such as pulse oximetry, neurologic checks, and vascular checks. Also include dressing care, drain care, urine output monitoring, and antiembolic stockings. Include specific parameters for physician notification for abnormal results (such as low urine output or low blood pressure).
 8. **Ventilator settings** (if applicable). Include mode, tidal volume, rate, pressure support, positive end-expiratory pressure, and oxygen percent.
 9. **Intravenous fluids.** Include fluid type, rate, and time interval.
 10. **Drugs (medicines).** Include home medicines if appropriate. Reference to patient-controlled anesthesia forms should be made here.
 11. **Diagnostics.** All necessary laboratory and radiographic investigations should be listed here, as well as electrocardiograms, cardiac diagnostic laboratory testing, pulmonary function tests, and other special procedures.
- B. **Review orders with nursing staff.** All orders should be reviewed with the nursing staff, particularly any unusual orders or orders that must be expedited.
- C. **STAT orders** should be designated as such on the order form and brought to the attention of the nursing staff. This is especially true for orders for new medicines because the pharmacy must be notified and the medicine brought to the floor.
- D. **Discharge orders**
1. **Discharge** should include location and condition. If a transfer to another institution is planned, copies of all medical records and a copy of current orders should be included.
 2. **Activity limitations**, if applicable, should be included. Workplace or school documentation may also be necessary.
 3. **Medicines.** Prescriptions for new medicines as well as detailed instructions are required.
 4. **Follow-up.** Follow-up plans with the appropriate physicians should be clearly indicated. Contact information for their offices should also be included.
 5. **Special.** Wound care, catheter care, physical therapy, or special studies should be described before discharge.

II. Hospital notes

- A. **History and physical examination.** The admission history and physical examination should be a complete record of the patient's history. Include past medical and surgical history, social history and family history, allergies, and home medicines with dosage and schedule. Outpatient records are often helpful and should be obtained if possible.
- B. **Preoperative notes** summarize the pertinent laboratory and other investigations before one proceeds to the operating room ([Table 1-1](#)).

Table 1-1. Preoperative note

- C. **Operative notes.** A brief operative note should be placed in the written medical record immediately following the operation, including the operative findings. The surgeon should also complete a dictated operative note immediately after the operation ([Table 1-2](#)). In addition, a dictated note should include specific operative indications, preparation and drape position, sponge and instrument count, and copy distribution.

Table 1-2. Brief operative note

- D. **Postoperative check.** Several hours after an operation, a patient should be examined and vital signs and urine output reviewed. Documentation in the medical record in the form of a SOA/P (subjective-objective-assessment/plan) note should be included. **Discharge summary.** A detailed account of a patient's hospitalization should be dictated at the time of discharge ([Table 1-3](#)). If a dictation confirmation number is provided, it should be recorded in the written medical record as the final note of the hospitalization. A dictated discharge summary must accompany any patients who are being transferred to other institutions.

Table 1-3. Discharge summary (dictated)

III. Informed consent

- A. **Obtaining informed consent.** The patient must choose whether or not to undergo a medical procedure or operation. The physician provides information to the patient so that an informed decision can be made. To be informed, the patient must understand the disease process, the natural course of the disease, the risks and benefits of the procedure under consideration, and potential alternative therapies. The most common and serious risks of the procedure and the patient's condition that might affect the outcome of a planned procedure or might place the patient at increased risk should be discussed. Recovery time, including amount and expected duration of postoperative pain, should also be reviewed. The use of invasive monitoring devices, including arterial and pulmonary artery catheters, should be explained. These discussions should use terms that are readily understood by the patient.
- B. **Documentation of informed consent.** An informed consent form is completed and signed by the patient before any elective operative procedure. In addition to the generic consent form, informed consent discussions should be documented in the progress notes section of the medical record. These notes should document the salient features of the informed consent discussion and specifically document that the potential complications and outcomes were explained to the patient. The patient's refusal to undergo a procedure that has been advised by the physician should be documented clearly in the chart. In certain situations, such as a medical emergency, it is impossible to obtain informed consent. Inability to obtain consent should be documented carefully in the medical record. Local medical bylaws generally have provisions for these types of situations and should be consulted on a case-by-case basis.

IV. Advanced directives. These are legal documents that allow patients to provide specific instructions for health care treatment in the event that the patient is unable to make or communicate these decisions personally. Advanced directives commonly include standard living wills and durable powers of attorney for health care. With the growing realization that medical technology can prolong life considerably and sometimes even indefinitely beyond the point of significant or meaningful recovery, the importance of these issues is clear. Patients should be offered the opportunity to execute an advanced directive on admission to the hospital.

- A. **Living wills** provide specific instructions for the withdrawal of medical treatment in the event that a patient is unable to make treatment decisions and is terminally ill. Living wills do not include withdrawal or withholding of any procedure to provide nutrition or hydration.
- B. **Durable powers of attorney for health care.** These directives allow a patient to legally designate a surrogate or proxy to make health care decisions if the patient is unable to do so.
- C. **Implementation.** Advanced directives are personal documents and therefore differ from patient to patient. These documents should be reviewed carefully before implementation. Advanced directives are legal documents, and they should be displayed prominently in the medical record. To be legally binding, the documents must be executed properly. If there is any question of validity, the risk management or legal staff of the hospital should be consulted. The most effective advance directives include specific instructions for health care decisions. Important issues to be addressed include the following:
1. **Intravenous fluids**
 2. **Enteral and parenteral nutrition**
 3. **Medicines**
 4. **Inotropic support**
 5. **Renal dialysis**
 6. **Mechanical ventilation**
 7. **Cardiopulmonary resuscitation**
- D. **Conflicts.** Although advance directives can be helpful in the management of critically ill patients, their implementation often is difficult. Advance directives, by their nature, cannot provide for every medical situation. For this reason, it is important to communicate with the patient and family before the execution of an advance directive and with the family in the event that a patient becomes incapacitated. If no advance directive is available, the physician and family must consider carefully when life-prolonging medical treatments are no longer beneficial to the patient. In such a case, the state's interest in preserving life might conflict with the desires of the family and physician. Advance directives avoid these legal ambiguities and protect the desires of a patient once he or she is incapacitated. If the family and physician do not agree, the hospital ethics committee or risk management staff should be consulted.

V. Occupational transmission of disease. As a rule, all occupational exposures should be reported to appropriate staff (student health, employee health), and a frank discussion regarding chemoprophylaxis should ensue.

- A. **Bloodborne pathogens.** Health care workers are at risk of becoming infected with bloodborne pathogens, such as hepatitis B virus (HBV), hepatitis C virus (HCV), and human immunodeficiency virus (HIV), through occupational exposure. Surgeons are at increased risk because of the frequency of percutaneous injuries during operative procedures. Although occupational exposure to HIV is a frightening aspect of the surgical profession, a surgeon is, in fact, much more likely to become infected with HIV through other means.
1. **Risk of disease transmission.** The risk of disease transmission depends on several factors, including the prevalence of disease in the patient population, frequency of exposure, and efficiency of disease transmission after exposure. Recognizing the risk of occupational exposure to bloodborne pathogens, the Centers for Disease Control and Prevention have issued guidelines for universal infection control practices (*MMWR Morb Mortal Wkly Rep* 37:377, 1988). Adherence to these guidelines now is mandated and regulated federally by the U.S. Occupational Safety and Health Administration.
 2. **Universal precautions.** Occupational Safety and Health Administration regulations emphasize the concept of universal precautions, stressing that every patient should be treated as if infected with bloodborne pathogens. Barrier protection should be used at all times to prevent skin and mucous membrane contact with blood or bloody body fluids. Other body fluids that might be infectious for HIV include amniotic, pericardial, peritoneal, pleural, synovial, and cerebrospinal fluids as well as semen and vaginal secretions. Fluids that are not considered infectious for HIV include saliva, sputum, tears, urine, sweat, vomitus, and stool. Specific recommendations include the following:
 - a. **Wear two pairs of gloves** when direct contact with blood or bloody body fluids is expected, when touching mucous membranes or nonintact skin, or when performing venipuncture or other invasive procedures.
 - b. **Wear a mask or protective eyewear**, or both, when there is a risk of splashes of blood or body fluids.
 - c. **Wear a gown** when clothing is likely to become soiled.
 3. **Behavior modification.** Although universal precautions are an important element of risk prevention, they do not prevent exposure that is associated with sharp objects, such as needles. Sharps injuries are a well-documented route of transmission of HBV, and sharps injury with hollow-bore needles is the most common event leading to HIV transmission in health care workers. To avoid sharps injuries, the following behavior modifications are recommended:
 - a. **Needles should not be bent or recapped.**
 - b. **Routine double gloving in the operating room.** Double gloving decreases the risk of perforation and, in the event of perforation, is associated with a decreased inoculum size.
 - c. **Palpation of sharps.** One's hand should not be used to palpate sharps during suturing in the operating room.
 - d. **Suture needles.** Instruments should be used to hold suture needles at all times.
 - e. **Wound retraction.** Wound retraction should be performed with instruments only. Gloved hands should not be used for wound retraction.
 - f. **Instrument tray.** An instrument tray should be positioned between the scrub nurse and surgeon, so that sharps can be placed on a tray instead of passed directly.
 4. **HIV**
 - a. **Risk.** The risk of occupational exposure to HIV has been evaluated prospectively in multicenter trials (*Curr Probl Surg* 29:197, 1992). These trials reveal that blood or bloody body fluids were the source of infection in all well-documented cases. Needle-stick injury (hollow-bore needle) is the primary cause of occupational HIV infection. The risk of HIV infection after percutaneous exposure is estimated at 0.3%. The risk of HIV infection after mucocutaneous exposure is too low to quantify, although transmission by this route has been documented. In needle-stick injuries, the risk of transmission depends on the volume of the inoculum, the quantity of virus in the source, the depth of penetration, and the type of needle (hollow-bore needle injury is associated with a higher risk than that of suture needle). No transmission by aerosols, casual contact, or suture needles in the operating room has been documented.
 - b. **Response to occupational exposure**
 1. **Cleanse wound immediately** with soap and water or with an effective antiviral agent.
 2. **Report exposure** to the employee health clinic.
 3. **Evaluate the patient for HIV, HBV, and HCV** after informed consent.
 4. **Baseline serology.** Evaluate the affected health care worker for evidence of HIV, HBV, and HCV immunity.
 5. **Counseling**
 6. **Chemoprophylaxis**
 - c. **Chemoprophylaxis**
 1. **Decision to institute chemoprophylaxis.** Risk factors for infection should be balanced against the toxicities of the antiretroviral regimen. Chemoprophylaxis is recommended after high-risk percutaneous exposures to patients with known HIV infection. Chemoprophylaxis should be offered after lower-risk exposures to patients with known HIV infection. After exposures to patients with unknown HIV status, the decision to institute

chemoprophylaxis should be made on a case-by-case basis.

2. **Prophylaxis regimen.** The latest recommendations of the Public Health Service (*MMWR Morb Mortal Wkly Rep* 45:468, 1996) are for a combination of zidovudine with lamivudine, with the addition of the protease inhibitor indinavir for highest-risk exposures. These medications should be initiated as soon as possible after exposure and continued for 4 weeks. The recommended doses are as follows:
 - a. **Zidovudine**, 200 mg p.o. t.i.d.
 - b. **Lamivudine**, 150 mg p.o. b.i.d.
 - c. **Indinavir**, 800 mg p.o. t.i.d.
- d. **Follow-up care.** Periodic testing for HIV should be performed for 6 months after exposure, after which time seroconversion is extremely rare.
- e. **Risk of transmission to patient.** Only one documented cluster of HIV infections has been associated with an infected health care worker, but this case has been highly publicized. Based on statistical factors, the estimated rate of transmission of HIV to patients from an HIV-positive surgeon would be extremely low, at 1 per 83,000 hours of surgery (*J Am Coll Surg* 184:403, 1997).

5. HBV

- a. **Risk of transmission to health care worker.** Although HIV and HBV are transmitted in the same manner, the efficiency of HBV transmission is much greater. Transmission after needle-stick exposure from a source who is hepatitis B early antigen positive is estimated to be 30%. Unlike HIV, HBV is a preventable disease, and the following prophylactic measures are strongly recommended:
 - b. **Prophylaxis**
 1. **Hepatitis B vaccine.** All surgeons should receive the hepatitis B vaccine unless antibody status documents active immunity. The vaccine is an inactivated or recombinant subunit vaccine and is administered intramuscularly at 0, 1, and 6 months.
 2. **Antibody titers.** Although routine monitoring of HBV antibody titers is not recommended, antibody titers should be checked after a known exposure to HBV to determine whether further prophylactic therapy is required. Antibody titer levels of greater than 10 mIU/mL within the 24 months preceding exposure are considered protective.
 3. **Hepatitis B immune globulin** is recommended after exposure to HBV unless antibody status reveals active immunity in the health care worker.
 - c. **Risk of transmission to patient.** Transmission of HBV from surgeon to patient has been well documented (particularly for surgeons who are positive for hepatitis B early antigen), which underscores the importance of disease prevention. Surgeons who are infected actively with, or who are carriers of, HBV should consult with the local hospital infection control committee before operating.
6. **HCV.** HCV is a recently characterized bloodborne pathogen. Chronic hepatitis develops in more than 50% of infected adults.
- a. **Risk of transmission to health care worker.** The risk of disease transmission from an infected patient to a health care worker through a percutaneous exposure is estimated at 3–10% (*N Engl J Med* 332:444, 1995). Transmission through mucocutaneous contact has been documented, but the risk has not been quantified.
 - b. **Postexposure treatment.** No vaccine is available for HCV, and no benefit is associated with treatment with immune globulin or any other prophylactic regimen. After a possible exposure to HCV, health care workers should undergo baseline testing for HCV antibodies, followed by repeat testing 6–9 months later. The risk of secondary transmission of HCV by exposed health care workers through patient contact or other means appears to be low.

B. Tuberculosis (TB)

1. **Risk of transmission to health care workers.** TB is a well-documented occupational hazard for health care workers. The resurgence of TB in the general population and the emergence of drug-resistant strains of TB have increased this risk. One study of physicians at Barnes Hospital demonstrated that 8.6% of the study population had undergone skin test conversion (*Infect Control Hosp Epidemiol* 15:95, 1994). The predominant route of disease transmission is respiratory.
2. **Respiratory isolation.** All patients with active TB should be maintained in respiratory isolation. Respiratory isolation includes caring for the patient in a room with negative-pressure ventilation and the use of suitable respiratory protection devices. Surgical masks are not considered adequate respiratory protection. Although respiratory isolation is an important element in limiting the risk of occupational exposure to TB, health care workers remain at risk from exposure to unidentified cases of TB.
3. **TB surveillance and prophylaxis**
 - a. **Annual TB skin tests are recommended.** Physicians who undergo skin test conversion should be evaluated with a chest X-ray and considered for prophylactic therapy.
 - b. **Prophylaxis.** Isoniazid therapy is considered effective in preventing the development of active TB and is recommended after skin test conversion. Therapy also is recommended for health care workers who are exposed to TB and have medical conditions, such as renal insufficiency, diabetes, and HIV infection, all of which increase the risk of developing active TB. The dose of isoniazid is 300 mg per day for 1 year. Hepatic toxicity is one of the adverse effects of isoniazid therapy.

2 Nutrition

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Introduction

Metabolism

I. Metabolism of proteins, carbohydrates, and fats

II. Stress metabolism

Nutritional Assessment and Administration

I. Nutritional assessment

II. Administration of nutrition

Introduction

Nutritional support in the surgical patient remains an essential component of perioperative care. Approximately one-half of all hospitalized patients experience or are at risk for **malnutrition**, with severe protein-calorie malnutrition (PCM) evident in 1 in 10 patients. Malnourished surgical patients have increased mortality and are two to three times more likely to incur complications. In addition, these patients have longer lengths of hospital stay and higher hospital charges [*Nutr Clin Pract* 16(2):69, 2001]. Many studies have found that appropriate nutritional intervention can improve postoperative outcomes and decrease costs. Most patients who undergo surgical procedures have adequate fuel reserves to tolerate a short period of starvation and catabolism; however, some individuals require nutritional support—in particular, those in whom complications of major surgery, trauma, or sepsis have developed, as well as those with cancer-related cachexia. Adequate nutrition sustains basal metabolism, wound healing, and the immune response, all of which are essential for timely recovery.

Metabolism

I. Metabolism of proteins, carbohydrates, and fats

- A. **Proteins** are important for the biosynthesis of enzymes, structural molecules, and immunoglobulins. Accordingly, the balance between protein synthesis and degradation is critical.
1. **Digestion of proteins** yields dipeptides and single amino acids, which are actively absorbed. The duodenum is the site of the majority of protein digestion and absorption, although exposure to pepsin in the stomach initiates the process. Pancreatic proteases, activated on exposure to enterokinase found throughout the duodenal mucosa, are the principal effectors of protein degradation. This accounts for the fact that almost 50% of protein absorption occurs in the duodenum, and complete protein digestion is achieved by the midjejunal level. Protein absorption can effectively occur at every level of the small intestine; therefore, clinically significant protein malabsorption is relatively infrequent, even after extensive intestinal resection. The quality of a protein is related to its amino acid composition. The 20 amino acids are divided into essential amino acids and nonessential amino acids, depending on whether they can be synthesized *de novo* in the body.
 2. **Major roles of amino acids** include the following:
 - a. **Synthesis and recycling** of proteins
 - b. **Catabolic reactions**, resulting in energy generation and the production of carbon dioxide
 - c. **Incorporation of nitrogen** into the production of nonessential amino acid and nucleotides
 3. **Metabolism of absorbed amino acids**, primarily by the liver, regulates accumulation of plasma amino acids. Administration of parenteral nutrition initially bypasses the liver by delivering amino acids directly into the systemic circulation.
 4. **Total body protein** in a 70-kg person is approximately 10–11 kg, concentrated mostly in skeletal muscle. Daily protein turnover is 250–300 g, or approximately 3% of total body protein. The primary site of protein turnover is the gastrointestinal (GI) tract, where shed enterocytes and secreted digestive enzymes are regularly lost. Excessive GI tract losses from a fistula, ileostomy, or draining gastrostomy, as well as partial- or full-thickness skin burns or seeping wounds, provide other potential sources of significant protein loss in surgical patients. Protein turnover decreases with age, from 25 g/kg per day in the neonate to 3 g/kg per day in the adult.
 5. **Protein requirements** in the average healthy adult without excessive losses are approximately 0.8 g/kg body weight. In the United States, the typical daily intake is, on average, twice this amount. Requirements for patients with acute illness increase to 1.2 g/kg per day, and up to 2 g/kg per day is necessary for severely physiologically stressed patients in the intensive care unit. Amino acids contribute only 15% of the normal energy expenditure, with the remainder supplied by carbohydrates and fat. Each gram of protein can be converted into 4 kcal energy.
- B. **Carbohydrates** are the body's primary energy source, providing 30–40% of calories in a typical diet.
1. **Carbohydrate digestion** is initiated by the action of salivary amylase, and absorption is generally completed within the first 1.0–1.5 m of the small intestine. Salivary and pancreatic amylases cleave starches into oligosaccharides on contact. Surface oligosaccharidases then hydrolyze and transport these molecules across the GI tract mucosa. Deficiencies in carbohydrate digestion and absorption are rare in surgical patients. Because pancreatic amylase is abundant, even in patients with limited pancreatic function, maldigestion of starch does not usually occur. Diseases that result in generalized mucosal flattening (e.g., celiac sprue, Whipple's disease, and hypogammaglobulinemia) may cause diminished uptake of carbohydrate byproducts because of resultant deficiencies in oligosaccharidases.
 2. **Glucose stores.** More than 75% of ingested carbohydrate is broken down and absorbed as glucose. Carbohydrate stores are exhausted after a 24-hour fast. Liver glycogen is used first, followed soon thereafter by muscle glycogen. Glucose drives insulin secretion, which in turn influences protein synthesis. An intake of as little as 400 calories of carbohydrate per 24 hours minimizes protein breakdown, particularly after adaptation to starvation. Uptake of glucose, under normal conditions, also inhibits lipolysis. Glucose is essential for wound repair, but excessive amounts can have adverse effects, including hepatic steatosis and neutrophil dysfunction. Each gram of enteral carbohydrate provides 4 kcal energy, similar to protein. Parenteral carbohydrate (e.g., dextrose) provides 3.4 kcal/g.
- C. **Lipids**, the third fuel component, provide the remaining 25–45% of calories in the typical diet. During starvation, the majority of calories are provided by lipids in the form of ketone bodies converted by the liver from long-chain fatty acids (see [section II.A](#)).
1. **Digestion and absorption of lipids** is complex and requires coordination between **biliary and pancreatic secretions**, as well as functional jejunum and ileum. The introduction of fat to the duodenum leads to secretion of cholecystokinin and secretin, which in turn produces gallbladder contraction and pancreatic enzyme release. **Pancreatic secretions** contain a combination of lipase, cholesterol esterase, and phospholipase A₂. In the alkaline environment of the duodenum, lipase hydrolyzes triglycerides to a monoglyceride and two fatty acids. Bile salts lead to emulsification. **Micelle formation** is the most important step in lipid absorption, facilitating absorption of fats across the mucosal barrier. Reabsorption of bile salts is necessary to maintain the bile salt pool. The liver is able to compensate for the moderate intestinal bile salt losses by increased synthesis from cholesterol. Major ileal resection may lead to depletion of the bile salt pool and subsequent fat malabsorption. Lipolysis is stimulated by steroids, catecholamines, and glucagon but is inhibited by insulin. **Stress results in dramatic lipolysis.** Each gram of lipid provides 9 kcal energy.
 2. The **essential fatty acids**, linoleic and linolenic acid, are required for cell membrane integrity. Dietary fats are the sole precursors to eicosanoid production and as such are potent immunomodulators. Arachidonic acid, a vital component in prostaglandin synthesis, can be manufactured from linoleic acid. Clinical deficiency results in a generalized scaling rash, poor wound healing, hepatic steatosis, and bone changes. This condition is usually a consequence of long-term, fat-free parenteral nutrition, in which high glucose levels inhibit lipolysis, preventing peripheral essential fatty acid liberation. It is remedied by providing as little as 3% of calories as parenteral lipid.

II. Stress metabolism

- A. **Starvation.** After an overnight fast, liver glycogen is rapidly depleted because of a fall in insulin and a rise in glucagon levels in plasma. In the first few days of starvation, caloric needs are supplied by fat and protein degradation. Generally, a baseline nitrogen loss of 10–15 g per day occurs through urinary losses. Most of the available protein is from the breakdown of skeletal and visceral muscle and is converted to glucose by gluconeogenesis in the liver. The brain preferentially uses this endogenously produced glucose, with the remainder consumed by red blood cells and leukocytes. If starvation continues for more than a few days, the brain then uses fat as its fuel source. Because the brain cannot use free fatty acids in the same manner as other tissues do, it relies on keto acids produced by the liver. This adaptation to ketone usage has a protein-sparing effect. In summary, the adaptive changes in uncomplicated starvation are a decrease in energy expenditure, a change in type of fuel consumed (which maximizes the caloric potential), and preservation of protein.
- B. **Physiologic stress.** The interaction of metabolic and endocrine responses that result from major operation, trauma, or sepsis can be divided into three phases:

1. **Catabolic phase.** After major injury, the metabolic demand is dramatically increased, as reflected in a significant rise in the urinary excretion of nitrogen (beyond that seen in simple starvation). After a major surgical procedure, protein depletion inevitably occurs because patients are commonly prevented from eating in addition to having an elevated basal metabolic rate. The hormonal response of physiologic stress includes elevation in the serum levels of glucagon, glucocorticoids, and catecholamines and decreased levels of insulin.
2. The **early anabolic phase** is also called the *corticoid-withdrawal phase*. Depending on the severity of stress, the body shifts from catabolism to anabolism. The timing of this event is variable, ranging from several days to several weeks. The period of anabolism can last from a few weeks to a few months, depending on many factors, which include the ability of the patient to obtain and use nutrients and the extent to which protein stores have been depleted. This phase is marked by a positive nitrogen balance, and there is a rapid and progressive gain in weight and muscular strength. The total amount of nitrogen gained is equivalent to the amount lost in the catabolic phase; however, the rate of repletion is much slower than the rapid rate of protein depletion after the original insult.
3. The **late anabolic phase** is the final period of recovery and may last from several weeks to months. Adipose stores are replenished gradually, and nitrogen balance equilibrates. Weight gain is much slower during this period than in the early anabolic phase due to the high caloric content of fat—the primary energy stores deposited during the early anabolic phase—as compared to protein.

Nutritional Assessment and Administration

I. Nutritional assessment is essential in identifying patients who are at risk for development of complications related to significant PCM. Preoperative nutritional support can significantly reduce perioperative morbidity and mortality in patients with severe PCM. In addition, the incidence of postoperative morbidities, such as intraabdominal abscess, anastomotic leakage, and ileus, may be decreased by the use of preoperative enteral or parenteral hyperalimentation in patients with severe PCM. Preoperative nutritional support for patients with only mild or moderate PCM is not routinely indicated (*ASPEN nutrition support practice manual*, 2nd ed. Gaithersburg, MD: ASPEN Publishers, 1998). [Table 2-1](#) presents several nutritional and biologic indices that were developed to predict the risk of perioperative complications and mortality (*Nutr Clin Pract* 8:171, 1998).

Table 2-1. Nutritional indices

A. **Types of malnutrition.** Two forms of malnutrition have been identified ([Table 2-2](#)).

Table 2-2. Types of malnutrition

1. **Marasmus** is malnutrition that is typically caused by illness-induced anorexia, without catabolism. It is a chronic nutritional deficiency marked by losses in weight, body fat, and skeletal muscle mass (as identified by anthropometric measurements). Visceral protein stores remain normal. Patients with marasmus may lose substantial body weight but are able to resist infection and respond appropriately to minor or moderate stress.
 2. **Kwashiorkor** may be, in some patients, an extension of marasmus. This form of malnutrition develops when the period of starvation is prolonged or if the stress is severe. Increased visceral protein loss and metabolic stress lead to hypoalbuminemia, edema, and anergy. Even in a well-nourished patient, a severe stress (e.g., major burn or prolonged sepsis) may rapidly lead to the depletion of visceral protein stores and impairment in immune function. Conventional anthropometric measurements may not identify these patients as being significantly malnourished.
- B. **Evaluation of preexisting deficits.** A dietary history, physical examination (including anthropometric measurements), and relevant laboratory data are the appropriate tools needed to make an accurate evaluation of a patient's preoperative nutritional status (*The science and practice of nutrition support: a case based core curriculum*. Dubuque, IA: Kendall/Hunt Publishing, 2001).
1. A **history** of weight fluctuation or a change in dietary habits is particularly relevant. In most cases, the possibility of malnutrition is suggested by the underlying disease or by a history of recent weight loss. Anorexia, nausea, vomiting, dysphagia, odynophagia, gastroesophageal reflux, or a history of generalized muscle weakness should prompt further evaluation. Recent weight loss (5% in the last month or 10% over 6 months) or a current body weight of 80–85% (or less) of ideal body weight suggests significant malnutrition. A complete history of current medications is essential to alert caretakers to potential underlying deficiencies as well as drug-nutrient interactions.
 2. **Physical examination** may identify muscle wasting (especially thenar and temporal muscles), loose or flabby skin, and peripheral edema (as a result of hypoproteinemia). More subtle findings of nutritional deficiency include skin rash, pallor, glossitis, gingival lesions, hair changes, hepatomegaly, neuropathy, and dementia (*ASPEN nutrition support practice manual*, 2nd ed. Gaithersburg, MD: ASPEN Publishers, 1998).
 3. **Anthropometric measurements**, such as triceps skinfold thickness and midarm muscle circumference, are a reflection of body-fat stores and skeletal muscle mass, respectively. These values are standardized for gender and height, and they should be reported as a percentage of the predicted value. Typically, anthropometric measurements include assessment of body weight, height, and body mass index, and these values allow the clinician to assess the patient's visceral and somatic protein mass and fat reserve.
 4. **Laboratory tests** that suggest malnutrition have a direct correlation with perioperative morbidity.
 - a. **Serum albumin** of less than 3.0 g/dL in a stable, hydrated patient; half-life of 14–20 days.
 - b. **Serum prealbumin** with a half-life of 2–3 days may be a more useful indicator of nutritional status. 10–15 mg/dL = mild depletion, 5–10 mg/dL = moderate depletion, and less than 5 mg/dL = severe depletion.
 - c. **Serum transferrin** of less than 200 mg/dL; half-life of 8–10 days.
 5. **Immune function** is frequently altered by malnutrition and may be determined by assessing.
 - a. **Delayed-type hypersensitivity** (anergy to common skin antigens)
 - b. **Total lymphocyte count (TLC)** is calculated by the following formula:

[Equation](#)

where 1,500–1,800 mm³ = mild depletion, 900–1,500 mm³ = moderate depletion, and less than 900 mm³ = severe depletion.

C. **Estimation of caloric and protein requirements** is necessary to provide adequate substrates for healing and tissue repair. Failure to provide adequate amounts of both calories and protein leads to further depletion of lean body mass.

1. **Basal energy expenditure (BEE)** can be predicted using the Harris-Benedict equation:
 - BEE in kcal per day for men = 66.4 + (13.7 × weight in kg) + (5.0 × height in cm) – (6.7 × age in years)
 - BEE in kcal per day for women = 655 + (9.6 × weight in kg) + (1.8 × height in cm) – (4.7 × age in years)
2. These equations provide a reliable estimate of the energy requirements in approximately 80% of hospitalized patients. The actual caloric needs may be substantially greater than the BEE during periods of metabolic stress ([Table 2-3](#)). Most stressed patients require 25–35 kcal/kg per day.

Table 2-3. Disease stress factors used in calculation of total energy expenditure

3. **Estimates of protein requirements.** The appropriate calorie-nitrogen ratio is approximately 150:1. In the absence of severe renal or hepatic dysfunction, approximately 1.5 g protein per kg body weight should be provided daily ([Table 2-4](#)).

Table 2-4. Estimated protein requirements in various disease states

- a. **Twenty-four-hour nitrogen balance** is calculated by subtracting nitrogen excretion from nitrogen intake. Nitrogen intake is the sum of nitrogen delivered from enteral and parenteral feedings. Nitrogen output is the sum of nitrogen excreted in urine, fistula drainage, diarrhea, and so forth. The usual approach is to measure the urea nitrogen concentration of an aliquot of a 24-hour urine collection and then to calculate nitrogen content from the urine volume. A correction factor is added to account for nitrogen losses in the stool and from skin exfoliation. The difference in nitrogen intake minus output estimates the 24-hour nitrogen balance.
- b. **Creatinine-height index (CHI)** can be used to determine the degree of malnutrition. A 24-hour urinary creatinine excretion is measured and compared to normal standards. Creatinine height index is calculated by the following equation:

Equation

where greater than 80% = zero to mild depletion, 60–80% = moderate depletion, and less than 60% = severe depletion.

II. Administration of nutrition

- A. **Indications.** The need for nutritional support should be assessed continually in all patients preoperatively and postoperatively. The majority of surgical patients do not require nutritional supplementation. Most patients have adequate fuel reserves to withstand common catabolic stresses and partial starvation for at least 1 week. For these patients, intravenous fluids with appropriate electrolytes and a minimum of 100 g glucose daily (to minimize protein catabolism) is adequate. However, even **patients who were well nourished** before a major operation or trauma who subsequently enter a prolonged hypermetabolic or severely catabolic period may require nutritional support (*Curr Probl Surg* 32:833, 1995). Without nutritional intervention, these patients may have complications that are attributable to impaired immune function and poor wound healing as a result of depletion of visceral protein stores (see [Chapter 9](#)). **Patients with a significant degree of preoperative malnutrition** have less reserve, tolerate catabolic stress and starvation poorly, and are therefore at higher risk for postoperative complications.
- B. **Enteral nutrition.** In general, the enteral route is preferred over the parenteral route and should be the initial step in alimentation. Enteral feeding is simple, physiologic, relatively inexpensive, and well tolerated by most patients. Enteral feeding maintains the GI tract cytoarchitecture and mucosal integrity (through trophic effects), absorptive function, and normal microbial flora. This results in less bacterial translocation and exotoxin release from the intestinal lumen to the bloodstream (*Semin Respir Infect* 9:248, 1994). **Enteral feedings are indicated** for patients who have a functional GI tract but are unable to sustain an adequate oral diet. **Enteral feedings may be contraindicated** in the patient with an intestinal obstruction, ileus, GI bleeding, severe diarrhea, vomiting, enterocolitis, or high-output enterocutaneous fistula. Contraindications to enteral feeding are in most cases relative or temporary rather than absolute. Choice of an appropriate feeding site, administration technique, formula, and equipment may circumvent many of these problems.
 1. **Feeding tubes.** Nasogastric, nasojejunal, gastrostomy, and jejunal tubes are available for the administration of enteral feeding products. Gastrostomy tubes can be placed using minimally invasive techniques, such as endoscopic or laparoscopic insertion. Jejunal tubes are preferred for long-term access and require a continuous infusion rather than bolus administration. The techniques for placement of these tubes and the common complications associated with their use are summarized in [Chapter 8](#).
 2. **Enteral feeding products.** A variety of commercially available enteral feeding formulas are available ([Table 2-5](#)). Standard solutions provide 1 kcal/mL; calorically concentrated solutions (>1 kcal/mL) are available for patients who require volume restriction. Currently available dietary formulations for enteral feedings can be divided into polymeric (blenderized and nutritionally complete commercial formulas), chemically defined formulas (elemental diets), and modular formulas ([Table 2-5](#)).

Table 2-5. Enteral formulas

- a. **Blenderized tube feedings** can be composed of any food that can be blenderized. Caloric distribution of these formulas should parallel that of a normal diet.
 - b. **Nutritionally complete commercial formulas** (standard enteral diets) vary in protein, carbohydrate, and fat composition. Several formulas use sucrose or glucose as carbohydrate sources and are suitable for lactose-deficient patients. Commercial formulas are convenient, sterile, and low in cost. They are recommended for patients experiencing minimal metabolic stress who have normal gut function.
 - c. **Chemically defined formulas** are commonly called *elemental diets*. The nutrients are provided in predigested and readily absorbed form. They contain protein in the form of low-molecular-weight free amino acids or polypeptides. Amino acid (elemental) and polypeptide diets are efficiently absorbed in the presence of compromised gut function. However, they are more expensive than nutritionally complete commercial formulas and are hyperosmolar, which may cause cramping and diarrhea.
 - d. **Modular formulations** include special formulas that are used for specific clinical situations (e.g., pulmonary, renal, or hepatic failure or immune dysfunction).
3. **Enteral feeding protocols.** In the past, elaborate protocols for initiating tube feedings were used. Currently, it is recommended that feedings be started with full-strength formula begun at a slow rate and steadily advanced. This reduces the risk of microbial contamination and achieves full nutrient intake earlier. This approach can also be used with high-osmolality or elemental products. Conservative initiation and advancement rates are recommended for patients who are critically ill, those who have not been fed for some time, and those who are receiving high-osmolality or calorie-dense formula.
- a. **Bolus feeding.** In general, bolus feedings are used in patients with nasogastric or gastrostomy feeding tubes. Feedings are administered by gravity and begin at 50–100 mL every 4 hours and are increased in 50-mL increments until the intake goal is reached (usually 240–360 mL every 4 hours). Tracheobronchial aspiration is a potentially serious complication. To prevent this, the patient's head should be elevated to 30–45 degrees during feeding and for 1–2 hours after each feeding. The residual gastric volume should be measured every 4 hours and before administration of the feeding bolus. If the gastric residual volume is greater than 50% of the previous bolus, the next feeding should be withheld. The feeding tube should be flushed with approximately 30 mL water after each use.
 - b. **Continuous infusion** administered by a pump is generally required for nasojejunal, gastrojejunal, or jejunal feeding tubes. Feedings are initiated at 20–50 mL per hour and increased in 10- to 20-mL-per-hour increments, every 4–6 hours, until the desired goal is reached. The feeding tube should be flushed with approximately 30 mL water every 4 hours. It may be difficult to aspirate these small-bore tubes to check for residuals; however, if volumes are high, the feedings should be slowed or held. For some patients, the entire day's feeding volume can be infused over an 8- to 12-hour period at night to allow the patient to be disconnected from the infusion pump during the day.
 - c. **Conversion to oral feeding.** When indicated, an oral diet is resumed gradually. In an effort to stimulate appetite, enteral feeding can be modified by the following measures:
 1. **Providing fewer feedings**
 2. **Holding daytime feedings**
 3. **Decreasing the volume of feedings.** When oral intake provides approximately 75% of the required calories, tube feedings can be discontinued.
 - d. **Administration of medications.** Many oral medications can be administered through feeding tubes. The elixir form is preferred but is not always available. Medications that are not suitable for administration through a feeding tube include the following:
 1. **Enteric-coated medications**
 2. **Drugs in gelatinous capsules**
 3. **Medications that are designed for sublingual use**
 4. **Most sustained-release medications**

- e. The following medications have been associated with tube clogging:
1. **Sucralfate**
 2. **Hydrochlorothiazide-triamterene (Dyazide)**
 3. **Ibuprofen**
 4. **Psyllium**
 5. **Extended-release theophylline (Theo-Dur sprinkles)**
 6. **Chlorpromazine (Thorazine)**
4. **Complications**
- a. **Metabolic complications.** Abnormalities in serum electrolytes, calcium, magnesium, and phosphorus can be minimized through vigilant monitoring. **Hyperosmolarity** may lead to the development of mental lethargy or obtundation. The treatment for this is the administration of free water by giving either 5% dextrose in water intravenously or additional water in the tube feedings. Volume overload and subsequent congestive heart failure may occur as a result of **excess sodium administration**, observed especially in patients with impaired ventricular function or valvular heart disease. **Hyperglycemia** may occur in any patient but is particularly common in individuals with preexisting diabetes or sepsis. The serum glucose level should be determined frequently, and regular insulin should be administered accordingly.
 - b. **Clogging** can usually be prevented by careful attention to routine flushing of the feeding tube. Wire stylets should not be used to unclog a feeding tube because of the risk of tube perforation and injury to the esophagus or stomach. Instillation of carbonated soda, cranberry juice, or meat tenderizer (1 teaspoon papain in 30 mL water) is sometimes useful for unclogging feeding tubes. Tubes that are refractory to these remedies, as well as those with cracks, leaks, or defective connectors, should be replaced.
 - c. **Tracheobronchial aspiration** of tube-feeding solutions may occur with patients who are fed into the stomach or proximal small intestine and may lead to the development of pneumonia. Patients at particular risk are those with central nervous system abnormalities and those who are sedated. Methylene blue (1 mL/L) added to the tube-feeding solutions, or glucose test strips, can be used to detect tube-feeding formula in tracheal aspirates. Historically, jejunal feeding has been the preferred route for patients who are at risk for aspiration; however, one comparison of tube feeding via jejunal and gastric routes found no difference in rates of aspiration pneumonia or other complications (*J Parenter Enteral Nutr* 24:103, 2000).
 - d. **High gastric residuals** of tube feedings as a result of outlet obstruction, dysmotility, intestinal ileus, or bowel obstruction may limit the usefulness of nasogastric or gastrostomy feeding tubes. Treatment of this problem should be directed at correcting the underlying cause. If gastric retention prevents the administration of sufficient calories and intestinal ileus or obstruction can be excluded, a nasojejunal or jejunostomy feeding tube may be necessary.
 - e. **Diarrhea** is a potential consequence of enteral feeding, occurring in 10–20% of patients; however, other causes of diarrhea (e.g., *Clostridium difficile* or other infectious colitis) should be considered. Diarrhea may result from numerous causes: too rapid an increase in the volume of hyperosmolar tube feedings, some medications (e.g., metoclopramide), a diet that is high in fat content, or the presence of components not tolerated by the patient (e.g., lactose). If other causes of diarrhea can be excluded, the volume or strength of tube feedings should be diminished. If no improvement occurs, a different formula should be used. Antidiarrheal agents (e.g., loperamide) should be reserved for patients with severe diarrhea. In the case of the surgical patient, *C. difficile* is a frequent cause of diarrhea due to the common use of perioperative antibiotics. Laboratory confirmation can be made with a *C. difficile* toxin assay. Treatment options include either metronidazole (Flagyl; oral or intravenous) or oral vancomycin.
- C. **Parenteral nutrition** is indicated for patients who require nutritional support but cannot meet their nutritional needs through oral intake and for whom enteral feeding is contraindicated or not tolerated.
1. **Peripheral parenteral nutrition (PPN)** is administered through a peripheral intravenous catheter. The osmolarity of PPN solutions generally is limited to 1,000 mOsm (approximately 12% dextrose solution) to avoid phlebitis. Consequently, unacceptably large volumes of solution (>2,500 mL) are necessary to fulfill the typical patient's total nutritional requirements. Temporary partial nutritional supplementation with PPN may be useful in selected patients but typically is not indicated.
 2. **Total parenteral nutrition (TPN)** provides complete nutritional support (*Surgery* 64:134, 1968). The selection of a particular solution, volume of administration, and solution additives is individualized for the patient and based on an assessment of the nutritional requirements (*N Engl J Med* 325:527, 1991).
 - a. **Access.** TPN solutions must be administered through a central venous catheter. A dedicated single-lumen catheter or a multilumen catheter can be used. Catheters should be replaced for unexplained fever or bacteremia.
 - b. **TPN solutions.** TPN solutions generally are administered as a 3-in-1 admixture of protein, as amino acids (10%; 4.0 kcal/g); carbohydrate, as dextrose (70%; 3.4 kcal/g); and fat, as a lipid emulsion of soybean or safflower oil (20%; 9.0 kcal/g, respectively). Alternatively, the lipid emulsion can be administered as a separate intravenous "piggyback" infusion. Standard preparations of TPN are used for most patients and provide total calories that are broken down as 50–60% carbohydrate, 24–34% fat, and 16% protein. Special solutions that contain low, intermediate, or high protein and nitrogen concentrations as well as varying amounts of fat and carbohydrate are available for some patients with diabetes, renal failure, or hepatic dysfunction.
 - c. **Additives.** Other elements can be administered in conjunction with the basic caloric and protein solutions.
 1. **Electrolytes** (e.g., sodium, potassium, chloride, acetate, calcium, magnesium, and phosphate) that are added to the TPN solution should be adjusted daily. A suggested formulation often is listed on a prewritten order sheet, and these concentrations are designed for the patient whose current serum electrolytes and renal function are normal. Suggested ranges for these additives include Na, 60–80 mEq per day; K, 30–60 mEq per day; Cl, 80–100 mEq per day; Ca, 4.6–9.2 mEq per day; Mg, 8.1–20.0 mEq per day; and PO₄, 12–24 mmol per day. The number of cations and anions must balance; this is achieved by altering the concentrations of chloride and acetate. If the serum bicarbonate is low, the solution should contain more acetate. The calcium and phosphate ratio must be monitored to prevent salt precipitation.
 2. **Medications**, such as albumin, H₂-receptor antagonists, heparin, iron, dextran, insulin, and metoclopramide, can be administered in TPN solutions; however, not all medications are compatible with 3-in-1 admixtures. Regular insulin should initially be administered subcutaneously according to a sliding scale, based on determination of the blood glucose level. After a stable insulin requirement has been established, insulin can be administered in the TPN solution, generally at two-thirds of the daily subcutaneous insulin dosage.
 3. **Other additives.** Trace elements are added to the TPN solution daily using a commercially prepared mixture (e.g., 1 mL Trace Element-5: 1 mg copper, 12 µg chromium, 0.3 µg manganese, 60 µg selenium, and 5 mg zinc). Multivitamins generally are added daily to the TPN solution using a commercially prepared mixture (e.g., 10 mL MVI-12). Vitamin K is not included in most multivitamin mixtures and must be added separately (10 mg once a week). Vitamins A and C and zinc are particularly important for proper wound healing.
 - d. **Routine physiologic and laboratory monitoring** should occur on a scheduled basis. This can be performed less frequently for patients whose postoperative course has stabilized and who are receiving a consistent TPN regimen. The initial frequency of monitoring includes vital signs and serum glucose every 6 hours; weight, serum electrolytes, and blood urea nitrogen daily; and triglycerides, complete blood cell count, prothrombin time, liver enzymes, and bilirubin weekly. A weekly 24-hour urine collection is valuable in estimating nitrogen losses because it provides a value for the urine urea nitrogen (UUN). Nitrogen balance is calculated as follows:

Equation

where total nitrogen loss (g per day) = 1.2 × [24-hour UUN (g per day)] + 2 g per day.

- e. **Administration of TPN.** Orders, written daily, should reflect the patient's dynamic nutritional status and biochemical profile ([Table 2-6](#)).

Table 2-6. Barnes-Jewish Hospital parenteral nutrition order form

1. **Introduction of TPN** should be gradual. For example, approximately 1,000 kcal is provided the first day. If there is metabolic stability (e.g., normoglycemia), this is increased to 1,500 kcal the second day. The amount is increased by 500 kcal per day until the caloric goal is reached.
2. **TPN solutions** are delivered most commonly as a continuous infusion. A new 3-in-1 admixture bag of TPN is administered daily with a constant infusion rate over 24 hours.
3. **Cyclic administration of TPN** solutions may be useful for selected patients, including (1) those who will be discharged from the hospital and subsequently receive home TPN; (2) those with limited intravenous access who require administration of other medications, such as chemotherapeutic agents; and (3) those who are metabolically stable and desire a period during the day when they can be free of an infusion pump. Cyclic TPN is administered for 8–16 hours, most commonly at night. This should not be done until metabolic stability has been demonstrated for patients on standard, continuous TPN infusions.

4. **Discontinuation of TPN** should take place when the patient can satisfy 75% of his or her caloric and protein needs with oral intake or enteral feeding. The calories provided by TPN can be decreased in proportion to calories from the patient's increasing enteral intake. To discontinue TPN, the infusion rate should be halved for 1 hour, halved again the next hour, and then discontinued. It is not necessary to taper the rate of TPN infusion if the patient is receiving less than 1,000 kcal per day. This is done to prevent complications that are caused by hyperinsulinemia.

f. **Complications associated with TPN**

1. **Catheter-related complications** can be avoided by strict aseptic technique and routine catheter care (*N Engl J Med* 290:757, 1974).
2. **Metabolic complications.** Hyperglycemia and hyperosmolarity may lead to coma or death. In addition, hyperglycemia may be the first indication of occult infection. As noted in [section II.C.2.d](#), the serum glucose level should be monitored frequently. Patients with a serum glucose level of 200–400 mg/dL should have subcutaneous regular insulin. Patients with a serum glucose level that exceeds 400 mg/dL should have intravenous infusions of regular insulin. Hyperglycemia can result in the generation of excess carbon dioxide, which may cause respiratory difficulties in patients with marginal pulmonary reserve. If such patients are ventilator dependent, they may be particularly difficult to wean (*JAMA* 243:1444, 1980). Conversely, hypoglycemia may develop on discontinuation of TPN. The refeeding syndrome may occur when TPN is administered to a severely malnourished patient, resulting in a dramatic shift of extracellular ions into the intracellular space. Additional supplementation of K, Mg, and PO₄ may be needed. A large parenteral sodium load in a severely malnourished patient may precipitate congestive heart failure. The consequences of the various electrolyte abnormalities are discussed in [Chapter 4](#).
3. **Refeeding syndrome** occurs when excessive carbohydrate calories are administered to malnourished patients, causing a precipitous drop in serum phosphate. Phosphate levels need to be monitored frequently in these patients.
4. **Hepatic dysfunction** is a common manifestation of long-term TPN support. Steatosis is associated with mild elevations of the transaminases, alkaline phosphate, and bilirubin.
5. **Cholecystitis**, particularly the acalculous type, is common in patients who receive TPN for extended periods. Cholecystostomy or cholecystectomy is indicated for symptomatic patients. To avoid cholestasis and prevent this complication, gallbladder contraction can be stimulated with the C-terminal octapeptide of cholecystokinin, 0.02 µg/kg i.v. per day.

- D. **Specialized substrates.** Promise has been shown for “nutritional pharmacology,” in which interventions with cytokines, hormones, and substrates can augment basic nutritional repletion. Cytokines, such as interleukin-1, interleukin-6, and tumor necrosis factor-alpha, contribute to the acute-phase response of inflammation, loss of skeletal muscle protein, and use of exogenous nutrients. Pharmacologic manipulation of these agents may reverse their deleterious effects (*Cancer* 79:1828, 1997). Exogenous growth hormone has been shown to accelerate lean body mass and protein gain in stable patients, thus offsetting the deposition of fat and extracellular water that are found with standard methods of nutritional support (*J Parenter Enteral Nutr* 25:519, 2001). However, complications that are associated with growth hormone administration must be fully understood and may limit its use. In addition, substrates such as arginine, glutamine, omega-3 fatty acids, and nucleic acids (RNA) may be beneficial in select patient populations. (*Nutr Clin Pract* 16:6, 2001).

E. **Disease-specific nutrition**

1. **Thermal injury** has a tremendous impact on metabolism because of prolonged, intense neuroendocrine stimulation. The increase in metabolic demands following thermal injury is proportionate to the extent of ungrafted body surface. Decreasing the intensity of neuroendocrine stimulation by providing analgesia and thermoneutral environments lowers the accelerated metabolic rate in many of these patients and helps to decrease catabolic protein loss until the burned surface can be grafted (*Compr Ther* 17:47, 1991).
2. **Diabetes** often complicates nutritional management. Complications that are associated with TPN administration (e.g., catheter-related sepsis) are more common with prolonged hyperglycemia. Unopposed glycosuria may cause osmotic diuresis, loss of electrolytes in urine, and nonketotic coma. The goal in glucose-intolerant patients is to maintain the serum glucose level at 100–200 mg/dL. Hypoglycemia can result in shock, seizures, or vascular instability. This can be prevented by adjusting the insulin dosing, with the understanding that insulin requirements will decrease as the patient recovers from the initial stress that is associated with the illness.
3. **Renal failure** may be associated with glucose intolerance, negative nitrogen balance (resulting from increased losses through dialysis), loss of protein with decreased protein synthesis, and diminished excretion of phosphorus. Dialysis should be adjusted accordingly, and these patients should be nutritionally replenished according to their calculated needs. Patients who receive peritoneal dialysis absorb approximately 80% of the dextrose in the dialysate fluid (assuming a normal serum glucose level). These factors must be considered when designing a nutritional support strategy.
4. **Hepatic failure** may result in wasting of lean body mass, fluid retention, vitamin and trace metal deficiencies, anemia, and encephalopathy. More than 70–80 g per day of amino acids is required to maintain nitrogen balance in these patients. It may be difficult or impossible to limit the amount of nitrogen that a patient receives each day yet still provide adequate nutritional support. Branched-chain amino acids are metabolized by skeletal muscle and serve as an energy source during periods of stress. These amino acids are available enterally or parenterally to decrease the levels of aromatic amino acids and, therefore, the severity of encephalopathy; however, their efficacy has not been proved (*J Parenter Enteral Nutr* 14:225, 1990).
5. **Cachexia and cancer** are associated with lean muscle wasting. More than two-thirds of patients with cancer experience significant weight loss during their illness, and malnutrition is a contributing cause of mortality in 20–40% of these individuals. Reasons for this development include decreased nutrient intake and impaired nutrient use. Antineoplastic therapies, such as chemotherapy, radiation therapy, or operative extirpation, can worsen preexisting malnutrition. Although the addition of TPN to these modalities in clinical studies has shown improvement in weight, nitrogen balance, and biochemical markers, there is little evidence to suggest better response rates or survival. Use of specialized formulas supplemented with various substrates (arginine, glutamine, nucleic acids, and omega-3 fatty acids) may reduce morbidity and length of hospital stay, but ongoing studies need to be done before these formulas are routinely recommended.
6. **Short-bowel syndrome** commonly occurs in patients with less than 200 cm of functional jejunum. It may result from mesenteric ischemia, Crohn's disease, or necrotizing enterocolitis. It is characterized by nutrient malabsorption, electrolyte imbalance, diarrhea, and dehydration. Most of these patients require intravenous nutrition for life, at costs of more than \$100,000 per year, with frequent hospitalizations for conditions such as catheter sepsis, progressive organ dysfunction, and osteoporosis. The estimated length of small bowel that is required for adult patients to become independent of TPN is greater than 120 cm without colon or greater than 60 cm with some colonic continuity. Salvage of the ileocecal valve improves outcome. Intestinal adaptation may occur in some patients, thereby allowing for the transition from intravenous to enteral feeding. Uniquely formulated diets (supplemented with glutamine and growth hormone) show promise for accelerating this process (*Curr Probl Surg* 34:393, 1997).
7. **Patients with AIDS** develop PCM and lose weight. Malnourished AIDS patients require 35–40 kcal and 2.0–2.5 g protein/kg per day. In addition to the required electrolytes, vitamins, and minerals, they should receive glutamine, arginine, nucleotides, omega-3 polyunsaturated fats, branched-chain amino acids, and trace metal supplements. Those with normal gut function should be given a high-protein, high-calorie, low-fat, lactose-free oral diet. Patients with compromised gut function require an enteral (amino acid, polypeptide, or immuno enriched) diet or TPN.

3 Life Support

Ernest W. Franklin IV and Bradley D. Freeman

Introduction

Life Support and Arrest Algorithms

I. BLS

II. ACLS

IV. Specific arrest algorithms

Introduction

The **time** from cardiopulmonary arrest to the initiation of **basic life support (BLS)** and **advanced cardiac life support (ACLS)** is critical to the ultimate outcome. The following guidelines were developed by the American Heart Association to standardize treatment for most adult patients [*Circulation* 102(Suppl I), 2000]. These guidelines do not preclude specific interventions based on an individual patient's characteristics.

Life Support and Arrest Algorithms

I. BLS. The ABCs of BLS—airway, breathing, and circulation—are critical to successful resuscitative efforts for both respiratory and cardiac arrest. This algorithm has been recently updated to include BLS and ACLS in one format and can be represented in either simplified (not shown) or detailed forms ([Fig. 3-1](#) and [Table 3-1](#)). After a person collapses, the following procedures are recommended:

Fig. 3-1. Comprehensive emergency cardiovascular care algorithm for the evaluation and management of persons after collapse. BLS, basic life support; CPR, cardiopulmonary resuscitation; PEA, pulseless electrical activity; VF, ventricular fibrillation; VT, ventricular tachycardia. [Adapted from A guide to the international ACLS algorithms. *Circulation* 2000;102(Suppl I):I-142-I-157.]

Table 3-1 Primary ABCD Survey

- A. **Determine unresponsiveness** by gently tapping or shaking the victim and asking, "Are you okay?" **Do not shake the victim's head or neck** unless trauma to these areas has been excluded.
- B. **Call for help if there is no response.** In the field, activate the emergency medical service system by calling the local emergency telephone number (e.g., 911). Call for a defibrillator.
- C. **Airway**
 1. **Position the patient** supine on a firm, flat surface. While the patient is being moved, his or her head and neck should remain in the same plane as the torso, and his or her body should be moved as a unit.
 2. **Open the airway.** If there is no evidence of head or neck trauma, the head-tilt, chin-lift maneuver is used: The rescuer should place the palm of one hand on the patient's forehead and apply firm pressure to tilt the head backward. At the same time, the rescuer places the index and middle fingers of the other hand under the patient's chin and displaces the mandible anteriorly, raising the tongue away from the posterior pharynx. For the patient with a suspected neck injury, the neck tilt should be avoided, and the modified jaw thrust should be used. This is performed by grasping the angles of the patient's mandible with the fingers of both hands and moving the mandible anteriorly.
- D. **Breathing**
 1. **Assess for the presence of spontaneous respiration** with the patient's airway open. The rescuer should listen and feel for airflow while observing for movement of the patient's chest. Maintenance of an open airway may suffice for spontaneous respiration to resume.
 2. **Perform rescue breathing** if spontaneous respiration is not present. Gently occlude the patient's nose, making a tight seal over the patient's mouth, and ventilate twice with slow, full breaths (1–2 seconds each, with a 2-second pause between breaths). Adequate ventilation is indicated by observing the chest rise and fall and hearing and feeling the air escape during exhalation. Rapid and high-pressure breaths might result in gastric distention. Health care professionals should be proficient in the use of the pocket mask to help prevent the transmission of infection during BLS. Proper technique involves holding the mask in place with the middle and ring fingers of the hands at the angle of the patient's mandible while executing the head-tilt maneuver. Because it is difficult to maintain a leak-proof seal, only well-trained and experienced personnel should use a bag-valve device with a mask. If the patient cannot be ventilated, his or her head should be repositioned, and ventilation should be attempted again. If these attempts are still unsuccessful, the foreign body–airway obstruction maneuver should be performed (see [section I.F](#)).
- E. **Circulation**
 1. **Assess for the presence of a pulse** by palpating the carotid artery. If a carotid pulse is present, rescue breathing should be continued at a rate of 10–12 breaths per minute until spontaneous respiration resumes.
 2. In the absence of a carotid pulse, **deliver chest compressions** at a rate of 80–100 per minute. Chest compression is performed by placing the heel of one hand on the back of the other with the fingers extended or interlocked. With elbows locked and shoulders located directly above the hands, the rescuer's hands are placed 1 inch cephalad to the xiphoid process. The patient's sternum is then compressed 1.5–2.0 inches by thrusting with the heel of the hand directly toward the spine. Compressions should be smooth and regular, with an equal amount of time allowed for compression and release. Without moving the hands, pressure must be completely released from the chest after each compression. During **one-rescuer BLS**, 15 compressions are delivered before ventilating twice. For **two-rescuer BLS**, the compression-ventilation ratio is 5:1, and the rescuer responsible for airway management should assess the adequacy of compressions by palpating periodically for the carotid pulse. Once the patient is intubated, ventilation can be given at a rate of 12–15 breaths per minute without pausing for compressions.
 3. **BLS should be stopped** for 5 seconds after four cycles of compressions and ventilation and every 2–3 minutes thereafter to assess whether the patient has resumed spontaneous breathing or circulation. If a pulse is present, ventilation should be continued as needed. When the defibrillator is available, it should be attached. Otherwise, BLS should not be stopped for more than 5 seconds except to intubate or defibrillate.
- F. **Foreign body–airway obstruction management.** If an unconscious patient cannot be ventilated after two attempts at repositioning the airway, abdominal thrusts (Heimlich maneuver) should be performed. The rescuer straddles the victim's thighs and places the heel of one hand against the victim's abdomen in the midline slightly cephalad to the umbilicus. The second hand is placed directly on top of the first, and thrusts are delivered by pressing posteriorly and cephalad. After 6–10 quick thrusts, the victim's mouth is opened, and a finger sweep is performed to remove any debris. Ventilation is then attempted, and if unsuccessful, the sequence of abdominal thrusts and the finger sweep are repeated. Cricothyrotomy and transtracheal ventilation should be performed if effective ventilation cannot be established.

II. ACLS. Properly performed BLS is critical to the successful performance of ACLS, which is a team effort that depends on effective supervision by a team leader. The leader should ensure that the following measures are taken:

- Effective BLS is performed.
- Early defibrillation is used when appropriate.
- Adequate intravenous access is obtained expeditiously.
- Endotracheal intubation occurs as soon as possible without excessive interruption of BLS (>30 seconds).
- Specific pharmacologic treatment is initiated in the appropriate sequence and dosage (see [section III](#) for specific arrest algorithms).

III. The team leader also is responsible for determining when resuscitative efforts should be terminated.

A. Primary therapies

1. **Airway management and oxygen therapy** are essential to the resuscitative effort. Oxygen (100%) should be delivered by mask, preferably with an adjunctive oropharyngeal or nasopharyngeal airway. Endotracheal intubation should be performed as soon as practicable in the resuscitative effort. Proper tube placement must be documented by auscultating breath sounds in the midaxillary line bilaterally and over the epigastrium and by end-tidal carbon dioxide determination. Ventilation with a well-fitting mask is preferable to multiple unsuccessful attempts at intubation. If airway obstruction is present and cannot be relieved by abdominal thrusts, transtracheal catheter ventilation or cricothyrotomy is indicated (see [Chapter 27](#)).
2. **Electrical defibrillation** is the major determinant of survival in cardiac arrest due to ventricular fibrillation (VF). Electrocardiographic monitoring should be established immediately. Defibrillation can be performed with manual or automatic external units. Quick-look paddles on the manual defibrillator can be used initially to avoid delay. If VF or pulseless ventricular tachycardia (VT) is present, defibrillation should be performed immediately ([Fig. 3-2](#)). Asystole should be confirmed in at least two leads or monitor paddle positions, because fine VF can be mistaken for asystole in a single-lead or monitor paddle position. Proper electrode position is essential for successful defibrillation. Standard placement is with one paddle along the patient's upper right sternal border just below the clavicle and the other paddle lateral to the left nipple centered in the midaxillary line. An alternative position is with one paddle anteriorly over the patient's left precordium and the other posterior to the heart, in the right infrascapular location. Conductive gel or pads should be used. Firm paddle pressure should be used to reduce transthoracic resistance. Self-adhesive monitor and defibrillator electrode pads also are effective and can be used in either location. Automatic external defibrillators are a recent device that have proved to be of significant value. Automatic external defibrillators require 5–15 seconds to analyze the electrocardiographic rhythm. There are three contraindications: Automatic external defibrillators are contraindicated in patients younger than 8 years of age or less than 25 kg and should not be used in any patient who is wet, lying in water, or atop a metal surface. Patients with a pacemaker, an automatic implantable cardioverter-defibrillator, or transdermal medication patches can be defibrillated externally without injury if a defibrillation paddle or electrode is not placed directly over the device. These devices may be reset by defibrillation and should be interrogated after resuscitation. The person operating the defibrillator must ensure that all personnel are away from the bed and the patient during defibrillation.

[Fig. 3-2](#). Ventricular fibrillation (VF)/pulseless ventricular tachycardia (VT) algorithm. CPR, cardiopulmonary resuscitation. [Adapted from A guide to the international ACLS algorithms. *Circulation* 2000;102(Suppl I):I-142-I-157.]

B. Drug therapies

1. **Route of administration.** Drugs may be administered via peripheral or central venous catheter. If peripheral venous access is used during resuscitative efforts, intravenous medications should be administered rapidly by bolus injection and be followed by a 30- to 50-mL bolus of intravenous fluid and elevation of the extremity. If there is delay in gaining intravenous access, epinephrine, lidocaine, and atropine can be administered through the endotracheal tube. Medications given endotracheally should be administered at 2.0–2.5 times the intravenous dose by dilution in 10 mL of normal saline or sterile water.
2. **Epinephrine** increases myocardial and cerebral blood flow during cardiopulmonary resuscitation, principally because of its alpha-adrenergic receptor–stimulating properties. The recommended dose is 1.0 mg i.v. every 3–5 minutes (2.0–2.5 mg per endotracheal tube). If this dosing of epinephrine fails, higher doses may be tried (0.2 mg/kg i.v. every 3–5 minutes).
3. **Arginine vasopressin (vasopressin)** has vasoconstrictive properties at pharmacologic doses and may have a more favorable side effect profile than epinephrine. In the setting of cardiac arrest, a single bolus of 40 units i.v. can be administered. Vasopressin was not found to be superior to epinephrine for resuscitation from cardiac arrest in a recent randomized controlled trial (*Lancet* 358:105, 2001).
4. **Antiarrhythmics**
 - a. **Amiodarone** affects the sodium, potassium, and calcium channels and is an alpha- and beta-adrenergic antagonist. Amiodarone is administered as 300 mg in 20–30 mL of D5W as an i.v. bolus for shock-resistant VF or pulseless VT. If these arrhythmias recur, a second dose of 150 mg i.v. may be indicated. With return of a spontaneous perfusing rhythm, an amiodarone drip of 150 mg i.v. over the first 10 minutes, then 360 mg i.v. over the next 6 hours, and finally a maintenance infusion of 540 mg i.v. over the next 18 hours are administered. The maximum cumulative dose is 2.2 g over 24 hours.
 - b. **Lidocaine** is now a second-line choice for VT or VF. The initial dose for VF is 0.5–0.75 mg/kg i.v. every 5–10 minutes with electrocardioversion attempts after the medication. The maximal dosage is 3 mg/kg. With return of a spontaneous perfusing rhythm, a lidocaine drip can begin with a loading dose of 1.0–1.5 mg/kg followed by a continuous infusion of 1–4 mg per minute. The maintenance dose of lidocaine should be reduced by 50% in the presence of impaired hepatic blood flow (e.g., patients with acute myocardial infarction, congestive heart failure, or circulatory shock) and in patients older than 70 years. Myocardial depression, circulatory depression, and neurologic symptoms, including seizure, are manifestations of lidocaine toxicity.
 - c. **Magnesium sulfate** is the treatment of choice in patients with torsades de pointes (polymorphic VT). For torsades de pointes, higher doses (up to 5–10 g) may be used. Rapid administration of magnesium can cause flushing, sweating, mild bradycardia, and hypotension. Depressed reflexes, flaccid paralysis, circulatory collapse, respiratory paralysis, and diarrhea may occur with hypermagnesemia.
 - d. **Procainamide's** indications include VF or VT refractory to other agents. Procainamide load is 30 mg per minute up to a total of 17 mg/kg (1.2 g for a 70-kg patient) followed by a maintenance infusion of 1–4 mg per minute. Procainamide may induce hypotension, heart block, or even cardiac arrest. Arterial blood pressure and electrocardiographic monitoring are required during intravenous administration.
 - e. **Atropine** is useful in treating symptomatic bradycardia (0.5–1.0 mg every 3–5 minutes i.v. to a total dose of 0.03 mg/kg) and might be beneficial in asystole (1.0 mg every 3–5 minutes i.v.). A total dose of 3 mg may result in full vagal blockade and should not be exceeded. Denervated transplanted heart does not respond to atropine and would require pacing, catecholamine infusion, or both. Atropine can induce tachycardia, which may be deleterious in the setting of myocardial ischemia. Anticholinergic syndrome of delirium, tachycardia, coma, flushed and hot skin, ataxia, and blurred vision can occur with excessive doses of atropine.
 - f. **Dopamine infusion** at a rate of 5 µg/kg per minute may be used for symptomatic bradycardia not responding to atropine if transcutaneous pacing is not immediately available. At these doses of dopamine, the alpha₁- and beta-adrenergic effects cause increased myocardial contractility, cardiac output, heart rate, and blood pressure. The infusion may be increased up to 20 µg/kg per minute for a predominately alpha-adrenergic effect if hypotension is associated with the bradycardia.
 - g. **Adenosine** depresses atrioventricular node conduction and is used to terminate atrioventricular node reentrant tachycardias, such as paroxysmal supraventricular tachycardia. It is not recommended for wide-complex tachycardias. A 6-mg intravenous bolus over 1–3 seconds is used and, if ineffective, is repeated with a 12-mg dose. Adenosine acts transiently (5–10 seconds) and produces few hemodynamic effects. Therapeutic levels of theophylline or related methylxanthines, such as caffeine and theobromine, can block adenosine's effects. Dipyridamole may potentiate adenosine's effects. Adenosine should be avoided or its dosage adjusted in patients receiving these drugs.
 - h. **Diltiazem** is useful in the management of atrial fibrillation or flutter. It can be used also in paroxysmal supraventricular tachycardia if adenosine has failed in patients who are not hypotensive and have a narrow QRS complex. The initial dose is 15–20 mg (0.25 mg/kg) i.v., followed by a second dose of 20–25 mg (0.35 mg/kg) i.v. in 15 minutes, if necessary. The maintenance infusion rate is 5–15 mg per hour to control the ventricular rate. Diltiazem should not be used concomitantly with intravenous beta-adrenergic blocking agents. Diltiazem should also be avoided or used with caution in patients with sick sinus syndrome, atrioventricular block in the absence of a functioning pacemaker, or Wolff-Parkinson-White syndrome and atrial fibrillation or flutter.
 - i. Although **calcium** has not been shown to be an effective primary therapy in patients with cardiac arrest, it is indicated for the treatment of myocardial instability secondary to hypocalcemia or hyperkalemia. A 10% calcium chloride may be given as an intravenous bolus of 2–4 mg/kg.
 - j. **Sodium bicarbonate** is not recommended for routine use early in the resuscitative effort. Bicarbonate may be beneficial in patients with preexisting metabolic acidosis, hyperkalemia, or tricyclic or phenobarbital overdose. When bicarbonate is used, 1 mEq/kg should be given as the initial dose, with one-half this dose administered every 10 minutes thereafter. One ampule of sodium bicarbonate contains 8.4% of bicarbonate (50 mEq/50 mL).

C. Adjunctive therapies

1. **A solitary precordial thump** can convert VT and, less often, VF to sinus rhythm. This should be used only for witnessed arrests when a defibrillator is not available and the patient is pulseless. A precordial thump should not be used in patients with VT who have a pulse because it may convert a pulse-forming into a pulseless rhythm (asystole, VF, or electromechanical dissociation).

2. When **internal cardiac defibrillation** is performed, the recommended starting defibrillation energy is 5 joules (J) with no more than 50 J used per discharge.
 3. **Emergency pacemaker therapy** is indicated in patients with hemodynamically unstable bradycardia unresponsive to pharmacologic treatments (i.e., atropine, dopamine). Transcutaneous cardiac pacing is the initial method of choice because of the speed with which it can be initiated and because it is widely available. Transvenous pacing is best instituted in the postresuscitation period.
- D. **Postresuscitation management** depends on the underlying disease process and the continued maintenance of hemodynamic and electrical stability. All patients should be transferred to an intensive care unit for continued care.

IV. Specific arrest algorithms. The following algorithms are applicable to a broad range of arrest scenarios but should be modified as the clinical situation demands (*JAMA* 268:2171, 1992).

- A. **Universal algorithm of adult emergency cardiac care** (Fig. 3-1 and Table 3-1)
1. **Assess responsiveness, breathing, and circulation.**
 2. **Institute BLS.**
 3. **Defibrillate early** for pulseless VT or VF.
 4. **Institute ACLS** and arrhythmia-specific algorithm.
- B. **VF or pulseless VT** (Fig. 3-2)
1. There are **two phases in the algorithm** for this dysrhythmia: The first involves electrocardioversion, and the second involves pharmacologic and electrocardioversion. In the first phase, sequentially defibrillate three times at 200 J, 200–300 J, and 360 J in rapid succession for refractory VF or VT. Do *not* pause for a pulse check if the monitor clearly displays persistent VF or VT.
 2. There are **four guidelines for the second phase**: Follow a **drug-shock-drug-shock** sequence, shock within 30–60 seconds of drug administration, assess for a pulse after each shock, and maximize one antiarrhythmic before administering another to limit proarrhythmic drug-drug interactions. First-line antiarrhythmic medications are either epinephrine, 1 mg i.v. every 3–5 minutes, or vasopressin, 40 units i.v. once. Ten to 20 minutes should elapse after the administration of vasopressin until epinephrine can be given.
 3. **Second-line antiarrhythmic medications** are amiodarone, lidocaine, magnesium sulfate, procainamide, and bicarbonate.
- C. **Pulseless electrical activity**, previously referred to as **electrical mechanical dissociation** (Fig. 3-3) is almost uniformly fatal unless the underlying cause can be identified and treated. Potentially reversible causes follow the acronym *5H 5T*:

Fig. 3-3. Pulseless electrical activity (PEA) algorithm. ACS, acute coronary syndrome; CPR, cardiopulmonary resuscitation; EMT, emergency medical treatment; OD, overdose; VF, ventricular fibrillation; VT, ventricular tachycardia. [Adapted from A guide to the international ACLS algorithms. *Circulation* 2000;102(Suppl I):I-142–I-157.]

1. **Hypovolemia**, especially resulting from hemorrhage, is the most common cause of pulseless electrical activity.
 2. **Hypoxia**
 3. **Hypothermia**
 4. **Hydrogen ions**
 5. **Hyperkalemia**
 6. **Tablets/toxins**
 7. **Tension pneumothorax**, evidenced by tracheal deviation and decreased ipsilateral breath sounds, should be treated by insertion of a large-bore angiocatheter (14-gauge) into the pleural space in the second intercostal space in the ipsilateral midclavicular line, followed by a thoracostomy tube.
 8. **Pericardial tamponade** is treated by pericardiocentesis during cardiac arrest.
 9. **Thrombosis of coronary vessels**
 10. **Thrombosis of pulmonary vessels**
- D. **Asystole** (Fig. 3-4) is associated with a poor prognosis in resuscitation. Asystole should be confirmed in two leads because fine VF might be difficult to distinguish from asystole. If the rhythm is unclear, the presence of fine VF should be assumed. Transcutaneous pacing is useful if the asystolic period is brief. Epinephrine and atropine are indicated irrespective of the down time.

Fig. 3-4. Asystole, the silent heart algorithm. CPR, cardiopulmonary resuscitation; VF, ventricular fibrillation; VT, ventricular tachycardia. [Adapted from A guide to the international ACLS algorithms. *Circulation* 2000;102(Suppl I):I-142–I-157.]

- E. **Bradycardia** (Fig. 3-5). The indications for treatment of bradycardia are absolute bradycardia (pulse rate of <60) or relative bradycardia with signs and symptoms (e.g., chest pain, shortness of breath, decreased level of consciousness, hypotension, or congestive heart failure). It should be remembered that some athletes and patients on beta-blockers may have a resting heart rate of less than 60. The sequential intervention sequence is the following:

Fig. 3-5. Bradycardia algorithm. AV, atrioventricular; BP, blood pressure; ECG, electrocardiogram. [Adapted from A guide to the international ACLS algorithms. *Circulation* 2000;102(Suppl I):I-142–I-157.]

1. **Atropine**, 0.5–1.0 mg i.v. every 3–5 minutes, up to 3 mg total dose
 2. **Transcutaneous pacing**
 3. **Dopamine**, 5–20 µg/kg i.v. per minute
 4. **Epinephrine**, 2–10 mg i.v. per minute
 5. **Isoproterenol**, 2–10 µg i.v. per minute. Isoproterenol is a class IIB/III medication (may be harmful) because it causes increased myocardial oxygen demand and peripheral hypotension. It is solely a temporizing measure until transcutaneous or transvenous pacing is available.
- F. **Tachycardia** (Fig. 3-6). Stable patients are treated according to the specific tachycardia. Unstable patients with serious signs or symptoms (e.g., chest pain, shortness of breath, decreased level of consciousness, hypotension, pulmonary edema, or acute myocardial infarction) attributable to the tachycardia are treated initially with cardioversion if the ventricular rate is more than 150 beats per minute. If the patient's condition becomes critical, then unsynchronized cardioversion is permitted.

Fig. 3-6. The tachycardia overview algorithm. CHF, congestive heart failure; DC, direct current; ECG, electrocardiogram; SVT; supraventricular tachycardia; VF, ventricular fibrillation; VT, ventricular tachycardia; WPW, Wolff-Parkinson-White syndrome. [Adapted from The tachycardia algorithms. *Circulation* 2000;102(Suppl I):I-158–I-165.]

1. **Necessary equipment** includes an oxygen saturation monitor, oral suction equipment, intravenous access, sedation, and possibly analgesics.

2. **Synchronized cardioversion for VT** should be performed starting at 100 J and increasing to 200, 300, and 360 J, if necessary. Synchronized cardioversion starting with 50 J is appropriate for atrial dysrhythmias.

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The Washington Manual of Surgery

4 Fluid, Electrolyte, and Acid-Base Disorders

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Diagnosis and Treatment of Fluid, Electrolyte, and Acid-Base Disorders

I. Definition of body fluid compartments

II. Osmolality and tonicity

III. Common electrolyte disorders

IV. Parenteral fluid therapy

V. Acid-base disorders

Diagnosis and Treatment of Fluid, Electrolyte, and Acid-Base Disorders

I. Definition of body fluid compartments. Water constitutes 50–70% of lean body weight. Total body water content is slightly higher in men, is most concentrated in skeletal muscle, and declines steadily with age. Total body water is divided into an intracellular fluid compartment, comprising 40% of total body weight, and an extracellular fluid compartment, comprising 20%. The extracellular fluid compartment consists of a plasma or intravascular compartment, comprising 5% of total body weight, and an interstitial compartment, comprising 15%. The extracellular and intracellular compartments have distinct electrolyte compositions. The principal extracellular cation is Na^+ , and the principal extracellular anions are Cl^- and HCO_3^- . In contrast, the principal intracellular cations are K^+ and Mg^{2+} , whereas the principal intracellular anions are phosphates and negatively charged proteins.

II. Osmolality and tonicity. *Osmolality* means the number of osmoles of solute particles per kilogram of water. Total osmolality is composed of both effective and ineffective components. Effective osmoles cannot freely permeate cell membranes and are restricted to either the intracellular or extracellular fluid compartments. The asymmetric accumulation of effective osmoles in either extracellular fluid (e.g., Na^+ , glucose, mannitol, glycine) or intracellular fluid (e.g., K^+ , amino acids, organic acids) causes transcompartmental movement of water. Because the cell membrane is freely permeable to water, the osmolalities of the extracellular and intracellular compartments are equal. The effective osmolality of a solution is synonymous with its tonicity. Ineffective osmoles, in contrast, freely cross cell membranes and therefore are unable to effect shifts in water between fluid compartments. Such ineffective solutes (e.g., urea, ethanol, and methanol) contribute to total osmolality but not to tonicity. For example, the plasma of uremic patients may be hyperosmolar but not hypertonic because the concentration of urea is equally distributed between the intracellular and extracellular fluid compartments. *Tonicity*, not osmolality, is the physiologic parameter that the body attempts to regulate.

III. Common electrolyte disorders

A. Sodium

1. **Physiology.** The normal individual consumes 3–5 g NaCl (50–90 mmol Na^+) per day. Balance is maintained primarily by the kidneys. Normal Na^+ concentration is 135–145 mmol/L. Potential sources of significant Na^+ loss include sweat, urine, and GI secretions (Table 4-1). Na^+ concentration largely determines the plasma osmolality (P_{osm}), which can be approximated by the following equation:

Equation

Table 4-1. Composition of gastrointestinal secretions

Normal P_{osm} is 290–310 mOsm/L. In general, hypotonicity or hypertonicity coincides with the presence of hyponatremia or hypernatremia. However, Na^+ concentration and total body water are controlled by independent mechanisms. As a consequence, hyponatremia or hypernatremia may occur in the setting of hypovolemia, hypervolemia, or euvolemia.

2. Hyponatremia

- a. **Causes and diagnosis.** The diagnostic approach to hyponatremia is illustrated in Fig. 4-1. Hyponatremia may occur in the setting of hypertonicity, isotonicity, or hypotonicity. Consequently, it is necessary to measure the serum osmolality to evaluate patients with hyponatremia.

Fig. 4-1. Diagnostic approach to hyponatremia. CHF, congestive heart failure; ECF, extracellular fluid; GI, gastrointestinal; SIADH, syndrome of inappropriate antidiuretic hormone secretion; TURP, transurethral resection of the prostate. (Adapted from Narins RG, Jones ER, Stom MC, et al. Diagnostic strategies in disorders of fluid, electrolyte, and acid-base homeostasis. *Am J Med* 1982;72:496.)

1. **Isotonic hyponatremia.** Hyperlipidemic and hyperproteinemic states result in an isotonic expansion of the circulating plasma volume and cause a decrease in serum Na^+ concentration. The reduction in serum sodium (mmol/L) can be estimated by multiplying the measured plasma lipid concentration (mg/dL) by 0.002 or the increment in serum protein concentration above 8 g/dL by 0.25. Isotonic, sodium-free solutions of glucose, mannitol, and glycine are restricted initially to the extracellular fluid and may similarly result in transient hyponatremia [see section III.A.2.c.(5)].
2. **Hypertonic hyponatremia.** Hyperglycemia may result in transient fluid shift from the intracellular to the extracellular compartment, thus diluting the serum Na^+ concentration. The expected decrease in serum Na^+ is approximately 1.3–1.6 mmol/L for each 100-mg/dL increase in blood glucose above 200 mg/dL. Rapid infusion of hypertonic solutions of glucose, mannitol, or glycine may have a similar effect on Na^+ concentration [see section III.A.2.c.(5)].
3. **Hypotonic hyponatremia** is classified on the basis of extracellular fluid volume. Hypotonic hyponatremia generally develops as a consequence of the administration and retention of hypotonic fluids (e.g., D5W, 0.45% NaCl) and rarely from the loss of salt-containing fluids alone.
 - a. **Hypovolemic hypotonic hyponatremia** in the surgical patient most commonly results from replacement of sodium-rich fluid losses (i.e., from the GI tract, skin, or lungs) with an insufficient volume of hypotonic fluid (e.g., D5W, 0.45% NaCl).
 - b. **Hypervolemic hypotonic hyponatremia.** The edematous states of congestive heart failure, liver disease, and nephrosis occur in the setting of inadequate circulating blood volume. This serves as a stimulus for the renal retention of sodium and of water. Disproportionate accumulation of water results in hyponatremia.
 - c. **Isovolemic hypotonic hyponatremia**
 - i. **Water intoxication** typically occurs in the patient who consumes large quantities of water in the setting of mildly impaired renal function (primary polydipsia). Alternatively, it may be the result of the administration of large quantities of hypotonic fluid in the patient with generalized renal failure.
 - ii. **K^+ loss**, either from GI fluid loss or secondary to diuretics, may result in isovolemic hyponatremia.
 - iii. **Reset osmostat.** Normally, the serum “osmostat” is set at 285 mOsm/L. In some individuals, the osmostat is “reset” downward, thus maintaining a lower serum osmolality. Several chronic diseases (e.g., tuberculosis and cirrhosis) predispose to this condition. Patients thus affected respond normally to water loads with suppression of antidiuretic hormone (ADH) secretion and excretion of free water. In contrast to

the syndrome of inappropriate ADH secretion (SIADH), the administration of exogenous water does not worsen the hyponatremia.

- iv. **SIADH** is characterized by plasma hypo-osmolality; urine osmolality that exceeds 100–150 mOsm/kg; urine Na^+ concentration of greater than 20 mmol/L; normal adrenal, renal, and thyroid function; normal serum K^+ concentration; and normal acid-base balance. The major causes of SIADH include pulmonary disorders (e.g., atelectasis, emphysema, pneumothorax, respiratory failure), central nervous system disorders (e.g., trauma, meningitis, tumors, subarachnoid hemorrhage), drugs (e.g., cyclophosphamide, cisplatin, nonsteroidal antiinflammatory drugs), and ectopic ADH production (e.g., small cell lung carcinoma).
- b. **Clinical manifestations.** Symptoms associated with hyponatremia are predominantly neurologic and result from hypo-osmolality. A decrease in P_{osm} causes intracellular water influx, increased intracellular volume, and cerebral edema. Symptoms include lethargy, confusion, nausea, vomiting, seizures, and coma. The likelihood that symptoms will occur is related to the degree of hyponatremia and to the rapidity with which it develops.
- c. **Treatment**
 1. **Isotonic and hypotonic hyponatremia** correct with resolution of the underlying disorder.
 2. **Hypovolemic hyponatremia** can be managed with administration of 0.9% NaCl to correct volume deficits and replace ongoing losses.
 3. **Water intoxication** responds to fluid restriction (1,000 mL per day).
 4. **Hypervolemic hyponatremia** may respond to water restriction (1,000 mL per day) to return Na^+ to greater than 130 mmol/L. In the setting of severe congestive heart failure, optimizing cardiac performance (e.g., with nitrates or angiotensin-converting enzyme inhibitors) may assist in Na^+ correction. If the edematous hyponatremic patient becomes symptomatic, plasma Na^+ can be increased to a safe level by the use of a loop diuretic (furosemide, 20–200 mg i.v. every 6 hours) while replacing urinary Na^+ losses with 3% NaCl. A reasonable approach is to replace approximately 25% of the hourly urine output with 3% NaCl. Hypertonic saline should not be administered to these patients without concomitant diuretic therapy.
 5. **Transurethral resection syndrome** refers to hyponatremia in conjunction with cardiovascular and neurologic manifestations, which infrequently follow transurethral resection of the prostate. This syndrome results from intraoperative absorption of significant amounts of irrigation fluid (e.g., glycine, sorbitol, or mannitol). Isotonic, hypotonic, or hypertonic hyponatremia may occur. Management of these patients may be complicated.
 6. For **SIADH**, water restriction (1,000 mL per day) should be attempted initially. Hypertonic saline (3% NaCl) is indicated in the presence of symptoms or extreme hyponatremia ($\text{Na}^+ < 110$ mmol/L). Serum Na^+ should be corrected to approximately 120 mmol/L. The quantity of 3% NaCl that is required to increase serum Na^+ to 120 mmol/L can be estimated by calculating the Na^+ deficit:

Equation

(Each liter of 3% NaCl provides 1,283 mmol Na^+ .) The use of a loop diuretic (furosemide, 20–200 mg i.v. every 6 hours) may increase the effectiveness of 3% NaCl administration. Central pontine demyelination occurs in the setting of SIADH. The risk factors for demyelination are controversial but appear to be related to the chronicity of hyponatremia and the rate of correction. The plasma Na^+ should be increased by no more than 12 mmol/L over the first 24 hours of treatment (i.e., $\text{Na}^+ < 0.5$ mmol per hour). The patient's volume status should be carefully monitored over this time, and the serum Na^+ should be determined frequently (every 1–2 hours). Once the serum Na^+ concentration reaches 120 mmol/L, administration of hypertonic saline can be discontinued. Water restriction (1,000 mL per day) should be continued until serum Na^+ concentration normalizes.

3. Hypernatremia

- a. **Diagnosis.** Hypernatremia is uniformly hypertonic and typically the result of water loss in excess of solute. Patients are categorized on the basis of their extracellular fluid volume status. The diagnostic approach to hypernatremia is illustrated in [Fig. 4-2](#).

Fig. 4-2. Diagnostic approach to hypernatremia. ECF, extracellular fluid; GI, gastrointestinal. (Adapted from Narins RG, Jones ER, Stom MC, et al. Diagnostic strategies in disorders of fluid, electrolyte, and acid-base homeostasis. *Am J Med* 1982;72:496–520.)

1. **Hypovolemic hypernatremia.** Any net loss of hypotonic body fluid results in extracellular volume depletion and hypernatremia. Common causes in the surgical patient include diuresis as well as GI, respiratory (especially patients with tracheostomies who are breathing unhumidified air), and cutaneous (particularly burn) fluid loss. Chronic renal failure and partial urinary tract obstruction also may cause hypovolemic hypernatremia.
2. **Hypervolemic hypernatremia** in the surgical patient is most commonly iatrogenic and results from the parenteral administration of hypertonic solutions (e.g., NaHCO_3 , saline, medications, nutrition).
3. **Isovolemic hypernatremia**
 - a. **Hypotonic losses.** Constant evaporative losses from the skin and respiratory tract, in addition to ongoing urinary free water losses, require the administration of 300–500 mL electrolyte-free water (e.g., D5W) daily to parenterally maintained afebrile patients. Inappropriate replacement of these hypotonic losses with isotonic fluids is the most common cause of isovolemic hypernatremia in the hospitalized surgical patient.
 - b. **Diabetes insipidus** is characterized by isovolemic hypernatremia in association with the production of large volumes of hypotonic urine (urine osmolality < 200 mOsm/kg). *Central diabetes insipidus* (CDI) describes a defect in hypothalamic secretion of ADH and may occur after head trauma or hypophysectomy. CDI may occur as a result of intracranial tumors, infections, vascular disorders (aneurysms), hypoxia, or medications (e.g., clonidine, phencyclidine). *Nephrogenic diabetes insipidus* (NDI) describes renal insensitivity to normally secreted ADH. NDI may be familial or drug induced (e.g., lithium, demeclocycline, methoxyflurane, glyburide) or may occur as a result of hypokalemia, hypercalcemia, or intrinsic renal disease. If CDI and NDI are not distinguishable clinically, they can be differentiated by dehydration testing.
- b. **Clinical manifestations.** Symptoms of hypernatremia are primarily neurologic and include lethargy, weakness, and irritability initially. These may progress to fasciculations, seizures, coma, and irreversible neurologic damage.
- c. **Treatment**
 1. **Water deficit** associated with hypernatremia can be estimated:

Equation

Rapid correction of hypernatremia can result in cerebral overhydration and permanent neurologic damage. Consequently, only one-half of the water deficit should be corrected over the first 24 hours, with the remainder corrected over the following 2–3 days. Serial Na^+ determinations are necessary to ensure that the rate of correction is adequate but not excessive. Oral fluid intake is acceptable for replacing water deficits. If oral intake is not possible, D5W or D5/0.45% NaCl can be substituted. In addition to the actual water deficit, insensible losses and urinary output must be replaced.

2. Diabetes insipidus

- a. **CDI** can be treated with desmopressin acetate administered intranasally [0.1–0.4 mL (10–40 μg) daily] or s.c. or i.v. [0.5–1 mL (2.0–4.0 μg) daily].
- b. **NDI treatment** requires removal of any potentially offending drug and correction of electrolyte abnormalities. If these measures are ineffective, dietary sodium restriction in conjunction with a thiazide diuretic may be useful (hydrochlorothiazide, 50–100 mg per day p.o.).

B. Potassium

1. **Physiology.** K^+ is the major intracellular cation, with only 2% of total body K^+ located in the extracellular space. Normal serum concentration is 3.3–4.9 mmol/L. Approximately 50–100 mmol K^+ is ingested and absorbed daily. Ninety percent of K^+ is renally excreted, with the remainder eliminated in stools.
2. **Hypokalemia**
 - a. **Causes.** K^+ depletion from inadequate K^+ intake alone is rare. The most common causes of K^+ depletion in the surgical patient include GI (e.g., diarrhea, persistent vomiting, nasogastric suctioning), renal (e.g., diuretics, fluid mobilization, amphotericin B), and cutaneous (e.g., burn) losses. Other causes of hypokalemia include acute intracellular K^+ uptake (associated with insulin excess, metabolic alkalosis, myocardial infarction, delirium tremens, hypothermia, or theophylline toxicity). Hypokalemia may also occur in the malnourished patient after the initiation of total parenteral nutrition (the refeeding syndrome) caused by the incorporation of K^+ into rapidly dividing cells. Hypomagnesemia frequently accompanies hypokalemia and generally must be corrected to replenish K^+ .
 - b. **Clinical manifestations.** Mild hypokalemia ($\text{K}^+ > 3.0$ mmol/L) is generally asymptomatic. The symptoms that are present with severe K^+ deficiency ($\text{K}^+ < 3.0$ mmol/L) are primarily cardiovascular. Early electrocardiogram (ECG) manifestations include ectopy, T-wave depression, and prominent U waves. Severe depletion increases susceptibility to reentrant arrhythmias.

- c. **Treatment.** In the absence of factors that cause redistribution, the relationship between the degree of hypokalemia and the extent of total body K^+ depletion is relatively predictable. For each 1-mmol/L decrement in the serum K^+ concentration, total body K^+ stores decrease by 200–400 mmol. When serum K^+ concentration falls below 2.0 mmol/L, the total body deficit may exceed 1,000 mmol. In mild hypokalemia, oral replacement is suitable. Typical daily therapy for the treatment of mild hypokalemia in the patient with intact renal function is 40–100 mmol KCl p.o. in single or divided doses. Parenteral therapy is indicated in the presence of severe depletion, significant symptoms, or oral intolerance. K^+ concentrations (administered as chloride, acetate, or phosphate) in peripherally administered intravenous fluids should not exceed 40 mmol/L, and the rate of administration should not exceed 20 mmol per hour. However, higher K^+ concentrations (60–80 mmol/L) administered more rapidly (with cardiac monitoring) are indicated in cases of severe hypokalemia, for cardiac arrhythmias, and in the management of diabetic ketoacidosis. Administration of high K^+ concentrations via subclavian, jugular, or right atrial catheters should be avoided because local K^+ concentrations may be cardiotoxic.

3. Hyperkalemia

- a. **Causes and diagnosis.** Hyperkalemia may occur with normal or elevated stores of total body K^+ . *Pseudohyperkalemia* is a laboratory abnormality that reflects K^+ release from leukocytes and platelets during coagulation. Spurious elevation in K^+ may result from hemolysis or phlebotomy from a strangulated arm. Abnormal redistribution of K^+ from the intracellular to the extracellular compartment may occur as the result of insulin deficiency, beta-adrenergic receptor blockade, acute acidemia, rhabdomyolysis, cell lysis (after chemotherapy), digitalis intoxication, reperfusion of ischemic limbs, and succinylcholine administration.
- b. **Clinical manifestations.** Mild hyperkalemia ($K^+ = 5.0$ – 6.0 mmol/L) is generally asymptomatic. Signs of significant hyperkalemia ($K^+ > 6.5$ mmol/L) are, most notably, ECG abnormalities: symmetric peaking of T waves, reduced P-wave voltage, and widening of the QRS complex. If untreated, severe hyperkalemia ultimately may cause a sinusoidal ECG pattern. ECG changes are more likely to develop with rapid increases in K^+ .
- c. **Treatment**
- Mild hyperkalemia** ($K^+ = 5.0$ – 6.0 mmol/L) can be treated conservatively by the reduction of daily K^+ intake and the possible addition of a loop diuretic (e.g., furosemide) to promote renal elimination. Any medication that is capable of impairing K^+ homeostasis (e.g., nonselective beta-adrenergic antagonists, angiotensin-converting enzyme inhibitors, K^+ -sparing diuretics, nonsteroidal antiinflammatory drugs) should be discontinued, if possible.
 - Severe hyperkalemia** ($K^+ > 6.5$ mmol/L)
 - Temporizing measures** produce shifts of potassium from the extracellular to the intracellular space.
 - NaHCO₃** [1 mmol/kg, or 1–2 ampules (50 mL each) of 8.4% NaHCO₃] can be infused intravenously over a 3- to 5-minute period. This dose can be repeated after 10–15 minutes if ECG abnormalities persist.
 - Dextrose** (0.5 g/kg body weight) infused with insulin (0.3 units regular insulin/g dextrose) transiently lowers serum K^+ (usual dose is 25 g dextrose with 6–10 units regular insulin given simultaneously as an intravenous bolus).
 - Inhaled beta-agonists** [e.g., albuterol sulfate, 2.0–4.0 mL of 0.5% solution (10–20 mg) delivered via nebulizer] have been shown to lower plasma K^+ , with a duration of action of up to 2 hours. Although only modest increases in heart rate and blood pressure have been reported when the nebulized form of this drug is used, caution is warranted in patients with known or suspected cardiovascular disease.
 - Calcium gluconate 10%** (5–10 mL i.v. over 2 minutes) should be administered to patients with profound ECG changes who are not receiving digitalis preparations. Calcium functions to stabilize the myocardium.
 - Therapeutic measures** decrease total body potassium by increasing potassium excretion.
 - Sodium polystyrene sulfonate** (Kayexalate), a Na^+ - K^+ exchange resin, can be administered orally (20–50 g of the resin in 100–200 mL 20% sorbitol every 4 hours) or rectally (as a retention enema, 50 g of the resin in 50 mL 70% sorbitol added to 100–200 mL water every 1–2 hours initially, followed by every-6-hour administration) to promote K^+ elimination. A decrease in serum K^+ level typically occurs 2–4 hours after administration.
 - Hydration** with 0.9% NaCl in combination with a loop diuretic (e.g., furosemide, 20–100 mg i.v.) should be administered to patients with adequate renal function to promote renal K^+ excretion.
 - Dialysis** is definitive therapy in severe, refractory, or life-threatening hyperkalemia.

C. Calcium

- Physiology.** Serum calcium (8.9–10.3 mg/dL or 2.23–2.57 mmol/L) exists in three forms: ionized (45%), protein bound (40%), and complexed to freely diffusible compounds (15%). Only free ionized Ca^{2+} (4.6–5.1 mg/dL or 1.15–1.27 mmol/L) is physiologically active. Daily calcium intake ranges from 500 to 1,000 mg, with absorption varying considerably. Normal calcium metabolism is under the influence of parathyroid hormone (PTH) and vitamin D. PTH promotes calcium resorption from bone and reclamation of calcium from the glomerular filtrate. Vitamin D increases calcium absorption from the intestinal tract.
- Hypocalcemia**
 - Causes and diagnosis.** Hypocalcemia most commonly occurs as a consequence of calcium sequestration or vitamin D deficiency. Calcium sequestration may occur in the setting of acute pancreatitis, rhabdomyolysis, or rapid administration of blood (citrate acting as a calcium chelator). Transient hypocalcemia may occur after total thyroidectomy, secondary to vascular compromise of the parathyroid glands, and after parathyroidectomy. In the latter case, serum Ca^{2+} reaches its lowest level within 48–72 hours after operation, returning to normal in 2–3 days. Hypocalcemia may occur in the setting of Mg^{2+} depletion, which simultaneously impairs PTH secretion and function. Acute alkalemia (e.g., from rapid administration of parenteral bicarbonate or hyperventilation) may produce clinical hypocalcemia with a normal serum calcium concentration due to an abrupt decrease in the ionized fraction. Because 40% of serum calcium is bound to albumin, hypoalbuminemia may decrease total serum calcium significantly. A fall in serum albumin of 1 g/dL decreases serum calcium by approximately 0.8 mg/dL (0.2 mmol/L). Ionized Ca^{2+} is unaffected by albumin. As a consequence, the diagnosis of hypocalcemia should be based on ionized, not total serum, calcium.
 - Clinical manifestations.** Tetany is the major clinical finding and may be demonstrated by Chvostek's sign (facial muscle spasm elicited by tapping over the branches of the facial nerve). Additionally, hypocalcemia can be associated with QT-interval prolongation and ventricular arrhythmias.
 - Treatment**
 - Parenteral therapy.** Asymptomatic patients, even those with moderate hypocalcemia (calcium, 6.0–7.0 mg/dL or 1.5–1.75 mmol/L), do not require parenteral therapy. Symptoms such as overt tetany, laryngeal spasm, or seizures are indications for parenteral calcium. Approximately 200 mg elemental calcium is needed to abort an attack of tetany. Initial therapy consists of the administration of a calcium bolus (10–20 mL of 10% calcium gluconate over 10 minutes) followed by a maintenance infusion of 1–2 mg/kg per hour elemental calcium. Serum calcium level typically normalizes in 6–12 hours with this regimen, at which time the maintenance rate can be decreased to 0.3–0.5 mg/kg per hour. In addition to monitoring calcium levels frequently during therapy, one should check Mg^{2+} , phosphorus, and K^+ levels and supplement as necessary. Calcium should be administered cautiously to patients who are receiving digitalis preparations because they may potentiate digitalis toxicity. Once the serum calcium level is normal, patients can be switched to oral therapy.
 - Oral therapy.** Calcium salts are available for oral administration (Table 4-2). In chronic hypocalcemia, with serum calcium levels of 7.6 mg/dL (1.9 mmol/L) or higher, the daily administration of 1,000–2,000 mg elemental calcium alone may suffice. When hypocalcemia is more severe, calcium salts should be supplemented with a vitamin D preparation. Daily therapy can be initiated with 50,000 IU calciferol, 0.4 mg dihydrotachysterol, or 0.25–0.50 μ g of 1,25-dihydroxyvitamin D₃ p.o. Subsequent therapy should be adjusted as necessary.

Table 4-2. Composition of common parenteral fluids^a

3. Hypercalcemia

- Causes and diagnosis.** Causes of hypercalcemia include malignancy, hyperparathyroidism, hyperthyroidism, vitamin D intoxication, immobilization, long-term total parenteral nutrition, thiazide diuretics, and granulomatous disease. The finding of an elevated PTH level in the face of hypercalcemia supports the diagnosis of hyperparathyroidism. If the PTH level is normal or low, further evaluation is necessary to identify one of the previously cited diagnoses.
- Clinical manifestations.** Mild hypercalcemia (calcium <12.0 mg/dL or <3.0 mmol/L) is generally asymptomatic. The hypercalcemia of hyperparathyroidism is associated infrequently with classic parathyroid bone disease and nephrolithiasis. Manifestations of severe hypercalcemia include altered mental status, diffuse weakness, dehydration, adynamic ileus, nausea, vomiting, and severe constipation. The cardiac effects of hypercalcemia include QT-interval shortening and arrhythmias.
- Treatment of hypercalcemia** depends on the severity of symptoms. Mild hypercalcemia (calcium <12.0 mg/dL or <3.0 mmol/L) can be managed conservatively by restricting calcium intake and treating the underlying disorder. Volume depletion should be corrected if present, and vitamin D, calcium

supplements, and thiazide diuretics should be discontinued. The treatment of more severe hypercalcemia may require one or more of the following measures.

1. **NaCl 0.9% and loop diuretics** may rapidly correct hypercalcemia. In the patient with normal cardiovascular and renal function, 0.9% NaCl (250–500 mL per hour) with furosemide (20 mg i.v. every 4–6 hours) can be administered initially. The rate of 0.9% NaCl infusion and dose of furosemide should subsequently be adjusted to maintain a urine output of 200–300 mL per hour. Serum Mg^{2+} , phosphorus, and K^+ levels should be monitored and replaced as necessary. The inclusion of KCl (20 mmol) and $MgSO_4$ (8–16 mEq or 1–2 g) in each liter of fluid may prevent hypokalemia and hypomagnesemia. This treatment may promote the loss of as much as 2 g calcium over 24 hours.
2. **Salmon calcitonin**, in conjunction with adequate hydration, is useful for treatment of hypercalcemia that is associated with malignancy and with primary hyperparathyroidism. Salmon calcitonin can be administered either s.c. or i.m. Skin testing by s.c. injection of 1.0 IU is recommended before progressing to the initial dose of 4 IU/kg i.v. or s.c. every 12 hours. A hypocalcemic effect may be seen as early as 6–10 hours after administration. If this dose is unsuccessful after 48 hours of treatment, it can be doubled. The maximum recommended dose is 8 IU/kg every 6 hours.
3. **Pamidronate disodium**, in conjunction with adequate hydration, is useful for the treatment of hypercalcemia that is associated with malignancy. For moderate hypercalcemia (calcium = 12.0–13.5 mg/dL or 3.0–3.38 mmol/L), 60 mg pamidronate diluted in 1 L of 0.45% NaCl, 0.9% NaCl, or D5W should be infused over 24 hours. For severe hypercalcemia, the dose of pamidronate is 90 mg. If hypercalcemia recurs, a repeat dose of pamidronate can be given after 7 days. The safety of pamidronate for use in patients with significant renal impairment is not established.
4. **Plicamycin** (25 µg/kg, diluted in 1 L of 0.9% NaCl or D5W, infused over 4–6 hours each day for 3–4 consecutive days) is useful for treatment of hypercalcemia that is associated with malignancy. The onset of action is between 1 and 2 days, with a duration of action of up to 1 week.

D. Phosphorus

1. **Physiology.** Extracellular fluid contains less than 1% of total body stores at a concentration of 2.5–4.5 mg/dL (0.81–1.45 mmol/L). Phosphorus balance is regulated by a number of hormones that control calcium metabolism. As a consequence, derangements in concentrations of phosphorus and calcium frequently coexist. The average adult consumes 800–1,000 mg per day phosphorus, which is predominantly renally excreted.
2. **Hypophosphatemia**
 - a. **Causes**
 1. **Decreased intestinal phosphate absorption** results from vitamin D deficiency, malabsorption, and the use of phosphate binders (e.g., aluminum, magnesium, calcium, or iron-containing compounds).
 2. **Renal phosphate loss** may occur with acidosis, alkalosis, diuretic therapy (particularly acetazolamide), during recovery from acute tubular necrosis, and during hyperglycemia as a result of osmotic diuresis.
 3. **Phosphorus redistribution**, from the extracellular to the intracellular compartment, occurs principally with respiratory alkalosis and administration of nutrients such as glucose (particularly in the malnourished patient). This transient decrease in serum phosphorus is of no clinical significance unless there is a significant total body deficit. Significant hypophosphatemia may also occur in malnourished patients after the initiation of total parenteral nutrition (refeeding syndrome) as a result of the incorporation of phosphorus into rapidly dividing cells.
 4. Hypophosphatemia may develop in **burn patients** as a result of excessive phosphaturia during fluid mobilization and incorporation of phosphorus into new tissues during wound healing.
 - b. **Clinical manifestations.** Moderate hypophosphatemia (phosphorus, 1.0–2.5 mg/dL or 0.32–0.81 mmol/L) is usually asymptomatic. Severe hypophosphatemia (phosphorus <1.0 mg/dL or <0.32 mmol/L) may result in respiratory muscle dysfunction, diffuse weakness, and flaccid paralysis.
 - c. **Treatment.** Hypophosphatemia can be corrected by the administration of oral or intravenous phosphate salts. For mild hypophosphatemia (phosphorus = 1.0–2.5 mg/dL or 0.32–0.81 mmol/L), potassium phosphate or sodium phosphate should be administered i.v. (0.08–0.16 mmol/kg over 6–8 hours). For more severe hypophosphatemia (phosphorus <1.0 mg/dL or <0.32 mmol/L), correction should be 0.16–0.24 mmol/kg potassium phosphate or sodium phosphate over 6–8 hours. Serum phosphorus levels should be checked frequently until the patient's condition is stable. Risks of intravenous therapy include hyperphosphatemia, hypocalcemia, hypotension, hyperkalemia (with potassium phosphate), hypomagnesemia, hyperosmolality, metastatic calcification, and renal failure. The hypophosphatemic patient may require intravenous replenishment for 5–7 days before intracellular stores are corrected. Once the serum phosphorus level exceeds 2.0 mg/dL (0.65 mmol/L), the patient can be switched to oral therapy. Oral therapy can be initiated with a sodium-potassium phosphate salt [e.g., Neutra-Phos, 250–500 mg (8–16 mmol phosphorus) p.o. four times a day; each 250-mg tablet of Neutra-Phos contains 7 mmol each of K^+ and Na^+].
3. **Hyperphosphatemia**
 - a. **Causes** include impaired renal excretion and transcellular shifts of phosphorus from the intracellular to the extracellular compartment (e.g., tissue trauma, tumor lysis, insulin deficiency, or acidosis). Hyperphosphatemia is also a common feature of postoperative hypoparathyroidism.
 - b. **Clinical manifestations**, in the short term, include hypocalcemia and tetany. In contrast, soft tissue calcification and secondary hyperparathyroidism occur with chronicity.
 - c. **Treatment** of hyperphosphatemia, in general, should eliminate the phosphorus source, remove phosphorus from the circulation, and correct any coexisting hypocalcemia. Dietary phosphorus should be restricted. Urinary phosphorus excretion can be increased by hydration (0.9% NaCl at 250–500 mL per hour) and diuresis (acetazolamide, 500 mg every 6 hours p.o. or i.v.). Phosphate binders (aluminum hydroxide, 30–120 mL p.o. every 6 hours) minimize intestinal phosphate absorption and can induce a negative balance of more than 250 mg phosphorus daily, even in the absence of dietary phosphorus. Hyperphosphatemia secondary to conditions that cause phosphorus redistribution (e.g., diabetic ketoacidosis) resolves with the treatment of the underlying condition and requires no specific therapy. Dialysis can be used to correct hyperphosphatemia in extreme conditions.

E. Magnesium

1. **Physiology.** Mg^{2+} (1.3–2.2 mEq/L or 0.65–1.10 mmol/L) is predominantly an intracellular cation. Renal excretion and retention play the major physiologic role in regulating body stores. Mg^{2+} is not under direct hormonal regulation.
2. **Hypomagnesemia**
 - a. **Causes.** Hypomagnesemia on the basis of dietary insufficiency is rare. Common etiologies include excessive GI or renal Mg^{2+} loss. GI loss may result from diarrhea, malabsorption, vomiting, or biliary fistulas. Urinary loss occurs with marked diuresis, primary hyperaldosteronism, renal tubular dysfunction (e.g., renal tubular acidosis), or chronic alcoholism, or as a drug side effect (e.g., loop diuretics, cyclosporine, amphotericin B, aminoglycosides, cisplatin). Hypomagnesemia may also result from shifts of Mg^{2+} from the extracellular to intracellular space, particularly in the setting of acute myocardial infarction or alcohol withdrawal or after receiving glucose-containing solutions. After parathyroidectomy for hyperparathyroidism, the redeposition of calcium and Mg^{2+} in bone may cause dramatic hypocalcemia and hypomagnesemia. Hypomagnesemia is usually accompanied by hypokalemia and hypophosphatemia and is frequently encountered in the trauma patient.
 - b. **Clinical manifestations.** Symptoms of hypomagnesemia are predominantly neuromuscular and cardiovascular. With severe depletion, altered mental status, tremors, hyperreflexia, and tetany may be present. The cardiovascular effects of hypomagnesemia are similar to those of hypokalemia and include T-wave and QRS-complex broadening, as well as prolongation of the PR and QT intervals. Ventricular arrhythmias most commonly occur in patients who receive digitalis preparations.
 - c. **Treatment**
 1. **Parenteral therapy** is preferred for the treatment of severe hypomagnesemia (Mg^{2+} <1.0 mEq/L or <0.5 mmol/L) or in symptomatic patients. In the setting of life-threatening arrhythmias, 1–2 g (8–16 mEq) $MgSO_4$ can be administered over 5 minutes, followed by a continuous infusion of 1–2 g per hour for the next several hours. The infusion subsequently can be reduced to 0.5–1.0 g per hour for maintenance. The normal range of Mg^{2+} (1.3–2.2 mEq/L or 0.65–1.10 mmol/L) probably is below its physiologic optimum. Thus, except in the setting of renal failure, vigorous correction of either severe or symptomatic hypomagnesemia is warranted. In less urgent situations, $MgSO_4$ infusion may begin at 1–2 g per hour for 3–6 hours, with the rate subsequently adjusted to 0.5–1.0 g per hour for maintenance. Mild hypomagnesemia (1.1–1.4 mEq/L or 0.5–0.7 mmol/L) in an asymptomatic patient can be treated initially with the parenteral administration of 50–100 mEq (6–12 g) $MgSO_4$ daily until body stores are replenished. Treatment should be continued for 3–5 days, at which time the patient can be switched to an oral maintenance dose. Intravenous $MgSO_4$ remains the initial therapy of choice for torsades de pointes (polymorphologic ventricular tachycardia). Furthermore, its use to achieve hypermagnesemia is therapeutic for eclampsia and for preeclampsia.
 2. **Oral therapy.** Magnesium oxide is the preferred oral agent. Each 400-mg tablet provides 241 mg (20 mEq) Mg^{2+} . Other formulations include magnesium gluconate [500-mg tablet provides 27 mg (2.3 mEq) Mg^{2+}] and magnesium chloride [535-mg tablet provides 64 mg (5.5 mEq) Mg^{2+}]. Depending on the level of depletion, oral therapy should provide 20–80 mEq per day Mg^{2+} in divided doses.
 3. **Prevention of hypomagnesemia** in the hospitalized patient who is receiving prolonged parenteral nutritional therapy can be accomplished by providing 0.35–0.45 mEq/kg per day Mg^{2+} [i.e., the addition of 8–16 mEq (1–2 g) $MgSO_4$ to each liter of intravenous fluids].
 4. **Serum Mg^{2+} levels should be monitored during therapy.** In the setting of renal insufficiency, the dose of Mg^{2+} should be reduced.
3. **Hypermagnesemia**
 - a. **Causes.** Hypermagnesemia occurs infrequently, is usually iatrogenic, and is seen most commonly in the setting of renal failure.
 - b. **Clinical manifestations.** Mild hypermagnesemia (Mg^{2+} , 5.0–6.0 mEq/L or 2.5–3.0 mmol/L) is generally asymptomatic. Severe hypermagnesemia (Mg^{2+} >8.0 mEq/L or >4.0 mmol/L) is associated with depression of deep tendon reflexes, paralysis of voluntary muscles, hypotension, sinus bradycardia, and

prolongation of PR, QRS, and QT intervals.

- c. **Treatment.** Cessation of exogenous Mg^{2+} is necessary. Calcium gluconate 10% (10–20 mL over 5–10 minutes i.v.) is indicated in the presence of life-threatening symptoms (e.g., hyperreflexia, respiratory depression, or cardiac conduction disturbances) to antagonize the effects of Mg^{2+} . NaCl 0.9% (250–500 mL per hour) infusion with loop diuretic (furosemide, 20 mg i.v. every 4–6 hours) in the patient with intact renal function promotes renal elimination. Dialysis is definitive therapy in the presence of intractable symptomatic hypermagnesemia.

IV. Parenteral fluid therapy.

The composition of commonly used parenteral fluids is presented in [Table 4-2](#).

- A. **Crystalloids**, in general, are solutions that contain sodium as the major osmotically active particle. Crystalloids are relatively inexpensive and are useful for volume expansion, maintenance infusion, and correction of electrolyte disturbances.
 1. **Isotonic crystalloids** (e.g., lactated Ringer's solution, 0.9% NaCl) distribute uniformly throughout the extracellular fluid compartment so that after 1 hour, only 25% of the total volume infused remains in the intravascular space. Lactated Ringer's solution is designed to mimic extracellular fluid and is considered a balanced salt solution. This solution provides a HCO_3^- precursor and is useful for replacing GI losses and extracellular fluid volume deficits. In general, lactated Ringer's solution and 0.9% NaCl can be used interchangeably. However, 0.9% NaCl is preferred in the presence of hyperkalemia, hypercalcemia, hyponatremia, hypochloremia, or metabolic alkalosis.
 2. **Hypertonic saline solutions** alone and in combination with colloids, such as dextran, have generated interest as a resuscitation fluid for patients with shock or burns. These fluids are appealing because, relative to isotonic crystalloids, smaller quantities are required initially for resuscitation. However, the intravascular hypertonic benefit rapidly dissipates as the fluid redistributes between the intravascular and extravascular spaces. Side effects of hypertonic solutions include hypernatremia, hyperosmolality, hyperchloremia, hypokalemia, and central pontine demyelination with rapid infusion. These solutions should be used with caution in patients with impaired renal function.
- B. **Hypotonic solutions** (D5W, 0.45% NaCl) distribute throughout the total body water compartment, expanding the intravascular compartment by as little as 10% of the volume infused. For this reason, hypotonic solutions should not be used for volume expansion. They are used to replace free water deficits.
- C. **Colloid solutions** contain high-molecular-weight substances that remain in the intravascular space. Early use of colloids in the resuscitation regimen may result in more prompt restoration of tissue perfusion and may lessen the total volume of fluid required for resuscitation. However, there are no situations in which colloids have unequivocally been shown to be superior to crystalloids for volume expansion. Because colloid solutions are substantially more expensive than crystalloids, their routine use in hypovolemic shock remains controversial. The use of colloids is indicated when crystalloids fail to sustain plasma volume because of low colloid osmotic pressure (e.g., increased protein loss from the vascular space, as in burns and peritonitis). Synthetic and human-derived colloids carry minimal risk of transmitting infection.
 1. **Albumin preparations** ultimately distribute throughout the extracellular space, although the initial volume of distribution is the vascular compartment. Preparations of 25% albumin (100 mL) and 5% albumin (500 mL) expand the intravascular volume by an equivalent amount (450–500 mL). However, 25% albumin is indicated in the edematous patient to mobilize interstitial fluid into the intravascular space. The cost per liter of albumin is more than that of other colloid solutions and 30 times the cost of the intravascular volume-equivalent amount of crystalloid solutions. Consequently, albumin preparations should be used judiciously. They are not indicated in the patient with adequate colloid oncotic pressure (serum albumin >2.5 mg/dL, total protein >5 mg/dL), for augmenting serum albumin in chronic illness (cirrhosis or nephrotic syndrome), or as a nutritional source.
 2. **Dextran** is a synthetic glucose polymer that undergoes predominantly renal elimination. In addition to its indications for volume expansion, dextran also is used for thromboembolism prophylaxis and promotion of peripheral perfusion. Dextran solutions expand the intravascular volume by an amount equal to the volume infused. Side effects include renal failure, osmotic diuresis, coagulopathy, and laboratory abnormalities (i.e., elevations in blood glucose and protein, interference with blood cross-matching).
 - a. **Dextran 40** is the fraction of branched dextrose polysaccharide with an average molecular weight of 40 kd, available as a 10% solution in either 0.9% NaCl or D5W. Recommended dosing for acute volume expansion is 2 g/kg (20 mL/kg of 10% solution) per 24 hours. If use continues beyond 24 hours, the dosing should be reduced to 1 g/kg (10 mL/kg) per 24 hours. Use should not extend beyond 5 days.
 - b. **Dextran 70** is a polysaccharide with an average molecular weight of 70 kd that is available as a 6% solution in normal saline or a 5% solution in inert sugar. Recommended dosing for acute volume expansion should not exceed 1.2 g/kg (20 mL/kg) in the first 24 hours. If therapy is extended beyond 24 hours, the dose should not exceed 0.6 g/kg (10 mL/kg) daily. The usual adult dose for acute volume expansion is 30 g (500 mL). In emergent situations, dextrose 70 can be given at a rate of 1.2–2.4 g per minute (20–40 mL per minute). In normovolemic or nearly normovolemic patients, the rate of infusion should not exceed 0.24 g per minute (4 mL per minute).
 3. **Hydroxyethyl starch (hetastarch)** is a synthetic molecule resembling glycogen that is available as a 6% solution in 0.9% NaCl. Hetastarch infusion, like 5% albumin, increases the intravascular volume by an amount equal to or greater than the volume infused. Hetastarch is less expensive than albumin and has a more favorable side effect profile than dextran formulations, making it an appealing colloid preparation.
 - a. **Indications** include use as a plasma volume-expanding agent in shock from hemorrhage, trauma, sepsis, and burns. Urine output typically increases acutely secondary to osmotic diuresis and must not be misinterpreted as a sign of adequate peripheral perfusion in this setting.
 - b. **Elimination** is hepatic and renal. Patients with renal impairment are particularly subject to initial volume overload and tissue accumulation of hetastarch with repeated administration. In these patients, initial volume resuscitation accomplished with hetastarch should be maintained with another plasma volume expander, such as albumin or crystalloid.
 - c. **Laboratory abnormalities** include elevations in serum amylase to approximately twice normal without associated alteration in pancreatic function.
 - d. **Dosing** of hetastarch 6% solution is 30–60 g (500–1,000 mL), with the total daily dosage not exceeding 1.2 g/kg (20 mL/kg) or 90 g (1,500 mL). In hemorrhagic shock, hetastarch solution can be administered at a rate of 1.2 g/kg per hour (20 mL/kg per hour). Slower rates of administration generally are used in patients with burns or septic shock. In individuals with severe renal impairment (creatinine clearance <10 mL per minute), the usual dose of hetastarch can be administered, but subsequent dosage should be reduced 50–75%.
- D. **Principles of fluid management.** A normal individual consumes an average of 2,000–2,500 mL water daily. Daily water losses include approximately 1,000–1,500 mL in urine and 250 mL in stool. The minimum amount of urinary output that is required to excrete the catabolic end products of metabolism is approximately 800 mL. An additional 750-mL insensible water loss occurs daily via the skin and respiratory tract. Insensible losses increase with hypermetabolism, fever, and hyperventilation.
 1. **Maintenance.** Maintenance fluids should be administered at a rate that is sufficient to maintain a urine output of 0.5–1.0 mL/kg per hour. Maintenance fluid requirements can be approximated on the basis of body weight as follows: 100 mL/kg per day for the first 10 kg, 50 mL/kg per day for the second 10 kg, and 20 mL/kg per day for each subsequent 10 kg. Maintenance fluids in general should contain Na^+ (1–2 mmol/kg per day) and K^+ [0.5–1.0 mmol/kg per day (e.g., D5/0.45% NaCl + 20–30 mmol K^+/L)].
 2. **Preoperative management.** Preexisting volume and electrolyte abnormalities should be corrected before operation whenever possible. Consideration of duration and route of loss provides important information regarding the extent of fluid and electrolyte abnormalities.
 3. **Intraoperative fluid management** requires replacement of preoperative deficit as well as ongoing losses ([Table 4-3](#)). Intraoperative losses include maintenance fluids for the duration of the case, hemorrhage, and “third-space losses.” Maintenance fluid requirement is calculated as detailed above (see [section IV.D.1](#)). Acute blood loss can be replaced with a volume of crystalloid that is three to four times the blood loss or with an equal volume of colloid or blood. Intraoperative insensible and third-space fluid losses are dependent on the size of the incision and the extent of tissue trauma and dissection and can be replaced with an appropriate volume of lactated Ringer's solution. Small incisions with minor tissue trauma (e.g., inguinal hernia repair) result in third-space losses of approximately 1–3 mL/kg per hour. Medium-sized incisions with moderate tissue trauma (e.g., uncomplicated sigmoidectomy) result in third-space losses of approximately 3–7 mL/kg per hour. Larger incisions and operations with extensive tissue trauma and dissection (e.g., pancreaticoduodenectomy) can result in third-space losses of approximately 9–11 mL/kg per hour or greater.

Table 4-3. Estimation of intraoperative fluid loss and guide for replacement

4. **Postoperative fluid management** requires careful evaluation of the patient. Continued sequestration of extracellular fluid into the sites of injury or operative trauma can continue for 12 hours or more after operation. Urine output should be monitored closely and intravascular volume replenished to maintain a urine output of 0.5–1.0 mL/kg per hour. Gastrointestinal losses that exceed 250 mL per day from nasogastric or gastrostomy tube suction should be replaced with an equal volume of crystalloid. Mobilization of perioperative third-space fluid losses typically begins 2–3 days after operation. Anticipation of postoperative fluid shifts should prompt careful evaluation of the patient's volume status and, if needed, consideration of diuresis before the development of symptomatic hypervolemia.

V. Acid-base disorders

A. Diagnostic approach

1. General concepts

- Acid-base homeostasis** represents equilibrium between the concentration of H^+ , PCO_2 , and HCO_3^- . In clinical practice, H^+ concentration is expressed as pH.
 - Normal pH** is 7.35–7.45. **Acidemia** refers to pH of less than 7.35, and **alkalemia** refers to pH of more than 7.45.
 - Acidosis and alkalosis** describe processes that cause the accumulation of acid or alkali, respectively. The terms *acidosis* and *acidemia*, and *alkalosis* and *alkalemia*, are often used interchangeably, but such usage is inaccurate. A patient, for example, may be acidemic while alkalosis is occurring.
 - Laboratory studies** that are necessary for the initial evaluation of acid-base disturbances include arterial pH, $PaCO_2$ (normal is 35–45 mm Hg), and serum electrolytes [HCO_3^- (normal is 22–31 mmol/L)]. Although base excess or base deficit calculations can be made, this information does not add substantially to the evaluation.
2. **Compensatory response to primary disorders.** Disorders that initially alter $PaCO_2$ are termed *respiratory acidosis* or *alkalosis*. Alternatively, disorders that initially affect plasma HCO_3^- concentration are termed *metabolic acidosis* or *alkalosis*. Primary metabolic disorders stimulate respiratory responses that act to return the ratio of PCO_2 to HCO_3^- (and therefore the pH) toward normal. Similarly, primary respiratory disturbances elicit countervailing metabolic responses that also act to normalize pH. As a general rule, these compensatory responses do not normalize pH because to do so would remove the stimulus for compensation. By convention, these compensating changes are termed *secondary* or *respiratory* or *metabolic* compensation for the primary disturbance. The amount of compensation to be expected from either a primary respiratory or metabolic disorder is presented in [Table 4-4](#). Significant deviations from these expected values suggest the presence of a mixed acid-base disturbance.

Table 4-4. Expected compensation for simple acid-base disorders

B. Primary metabolic disorders

- Metabolic acidosis** results from the accumulation of nonvolatile acids, reduction of renal acid excretion, or loss of alkali. The most common causes of metabolic acidosis are listed in [Table 4-5](#). Metabolic acidosis has few specific signs. The appropriate diagnosis depends on the clinical setting and laboratory tests.

Table 4-5. Causes of metabolic acidosis

- Anion gap** (AG; normal is 3–11 mmol/L) represents the anions, other than Cl^- and HCO_3^- , that are necessary to counterbalance Na^+ electrically:

Equation

It is useful diagnostically to classify metabolic acidosis into increased or normal AG metabolic acidosis.

- Increased AG metabolic acidosis** occurs in the setting of toxic ingestions (e.g., salicylates, methanol, ethylene glycol), rhabdomyolysis, or overproduction of endogenous acids (e.g., lactic acidosis or ketoacidosis) or as a consequence of renal failure. Lactic acidosis (normal serum lactate, 0.3–1.3 mmol/L) represents one of the most common causes of severe metabolic acidosis that is encountered in the critically ill surgical patient.
 - Normal AG (hyperchloremic) metabolic acidosis** results from the loss of bicarbonate-rich fluids from the GI tract (e.g., diarrhea, pancreatic or biliary fistula) or kidney (e.g., renal tubular acidosis, K^+ -sparing diuretics, carbonic anhydrase inhibitors). It may also occur after the administration of HCl or its precursor (e.g., in parenteral nutrition).
- b. **Treatment of metabolic acidosis** must be directed primarily at the underlying cause of the acid-base disturbance. Bicarbonate therapy should be considered in patients with moderate to severe metabolic acidosis, only after the primary cause has been addressed. The HCO_3^- deficit (mmol/L) can be estimated by the following equation:

Equation

This equation serves only as a rough estimate because the volume of HCO_3^- distribution and the rate of ongoing H^+ production are variable.

- Rate of HCO_3^- replacement.** In nonurgent situations, the estimated HCO_3^- deficit can be administered as a continuous intravenous infusion over 4–8 hours [50-mL ampule of 8.4% $NaHCO_3$ solution (provides 50 mmol HCO_3^-) can be added to 1 L D5W or 0.45% NaCl]. In urgent situations, the entire deficit can be administered as a bolus over several minutes.
 - The **goal of HCO_3^- therapy** should be to raise the arterial blood pH to 7.20 or the HCO_3^- concentration to 10 mmol/L. One should not attempt to normalize pH with bicarbonate administration because the risks of bicarbonate therapy (e.g., hypernatremia, hypercapnia, cerebrospinal fluid acidosis, or overshoot alkalosis) are likely to be increased. Serial arterial blood gases and serum electrolytes should be obtained to assess the response to HCO_3^- therapy.
 - Lactic acidosis.** Correction of the underlying disorder is the primary therapy for lactic acidosis. Reversal of circulatory failure, hypoxemia, or sepsis reduces the rate of lactate production and enhances its removal. Because the use of $NaHCO_3$ in lactic acidosis is controversial, no definite recommendations can be made.
2. **Metabolic alkalosis** ([Table 4-6](#))

Table 4-6. Causes of metabolic alkalosis

a. Causes

- Chloride-responsive metabolic alkalosis** in the surgical patient is typically associated with extracellular fluid volume deficits. The most common causes of metabolic alkalosis in the surgical patient include inadequate fluid resuscitation or diuretic therapy (e.g., contraction alkalosis), acid loss through GI secretions (e.g., nasogastric suctioning, vomiting), and the exogenous administration of HCO_3^- or HCO_3^- precursors (e.g., citrate in blood). Posthypercapnic metabolic alkalosis occurs after the rapid correction of chronic respiratory acidosis. Under normal circumstances, the excess in bicarbonate that is generated by any of these processes is excreted rapidly in the urine. Consequently, maintenance of metabolic alkalosis requires impairment of renal HCO_3^- excretion, most commonly due to volume and chloride depletion. Because replenishment of Cl^- corrects the metabolic alkalosis in these conditions, each is classified as a Cl-responsive metabolic alkalosis.
- Chloride-unresponsive metabolic alkalosis** is encountered less frequently in surgical patients and usually results from mineralocorticoid excess. Hyperaldosteronism, marked hypokalemia, renal failure, renal tubular Cl^- wasting (Barter's syndrome), and chronic edematous states are associated

with chloride-unresponsive metabolic alkalosis.

- b. **Diagnosis.** Although the cause of metabolic alkalosis is usually apparent in the surgical patient, measurement of urinary chloride concentration may be useful for differentiating these disorders. Urine Cl^- concentration of less than 15 mmol/L suggests inadequate fluid resuscitation, ongoing GI loss from emesis or nasogastric suctioning, diuretic administration, or posthypercapnia as the cause of the metabolic alkalosis. Urine Cl^- concentration greater than 20 mmol/L suggests mineralocorticoid excess, alkali loading, concurrent diuretic administration, or the presence of severe hypokalemia.
- c. **Treatment principles** in metabolic alkalosis include identifying and removing underlying causes, discontinuing exogenous alkali, and repairing Cl^- , K^+ , and volume deficits. Because metabolic alkalosis generally is well tolerated, rapid correction of this disorder usually is not necessary.
 1. **Initial therapy** should include the correction of volume deficits (with 0.9% NaCl) and hypokalemia. Patients with vomiting or nasogastric suctioning also may benefit from H_2 -receptor antagonists or other acid-suppressing medications.
 2. **Edematous patients.** Chloride administration does not enhance HCO_3^- excretion because the reduced effective arterial blood volume is not corrected by this therapy. Acetazolamide (5 mg/kg per day i.v. or p.o.) facilitates fluid mobilization while decreasing renal HCO_3^- reabsorption. However, tolerance to this diuretic may develop after 2–3 days.
 3. **Severe alkalemia** ($\text{HCO}_3^- > 40$ mmol/L), especially in the presence of symptoms, may require more aggressive correction. The infusion of acidic solutions is occasionally indicated in the patient with severe refractory metabolic alkalosis and chloride loss, typically due to massive nasogastric drainage or complete prepyloric obstruction. Ammonium chloride (NH_4Cl) is hepatically converted to urea and HCl. The amount of NH_4Cl that is required can be estimated by the following equation:

[Equation](#)

NH_4Cl is prepared by adding 100 or 200 mmol (20–40 mL of the 26.75% NH_4Cl concentrate) to 500–1000 mL of 0.9% NaCl. This solution should be administered at a rate that does not exceed 5 mL per minute. Approximately one-half of the calculated volume of NH_4Cl should be administered, at which time the acid-base status and Cl^- concentration should be repeated to determine the necessity for further therapy. NH_4Cl is contraindicated in hepatic failure.

4. **HCl** (0.1 N, administered i.v.) corrects metabolic alkalosis more rapidly. The amount of H^+ to administer can be estimated by the following equation:

[Equation](#)

To prepare 0.1 N HCl, mix 100 mmol HCl in 1 L sterile water. The calculated amount of 0.1 N HCl must be administered via a central venous catheter over 24 hours. HCO_3^- concentration can be safely reduced by 8–12 mmol/L over 12–24 hours.

5. **Dialysis** can be considered in the volume-overloaded patient with renal failure and intractable metabolic alkalosis.

C. Primary respiratory disorders

1. **Respiratory acidosis** occurs when alveolar ventilation is insufficient to excrete metabolically produced CO_2 . Common causes in the surgical patient include respiratory center depression (e.g., drugs, organic disease), neuromuscular disorders, and cardiopulmonary arrest. Chronic respiratory acidosis may occur in pulmonary diseases, such as chronic emphysema and bronchitis. Chronic hypercapnia may also result from primary alveolar hypoventilation or alveolar hypoventilation related to extreme obesity (e.g., pickwickian syndrome) or from thoracic skeletal abnormalities. The diagnosis of acute respiratory acidosis usually is evident from the clinical situation, especially if respiration is obviously depressed. Appropriate therapy is correction of the underlying disorder. In this setting, there is no indication for NaHCO_3 administration.
 2. **Respiratory alkalosis** is the result of acute or chronic hyperventilation. The causes of respiratory alkalosis include acute hypoxia (e.g., pneumonia, pneumothorax, pulmonary edema, bronchospasm), chronic hypoxia (e.g., cyanotic heart disease, anemia), and respiratory center stimulation (e.g., anxiety, fever, gram-negative sepsis, salicylate intoxication, central nervous system disease, cirrhosis, pregnancy). Excessive ventilation may also cause respiratory alkalosis in the mechanically ventilated patient. Depending on its severity and acuteness, hyperventilation may or may not be clinically apparent. Clinical findings are nonspecific. As in respiratory acidosis, the only effective treatment is correction of the underlying disorder.
- D. Mixed acid-base disorders.** When two or three primary acid-base disturbances occur simultaneously, a patient is said to have a mixed acid-base disorder. As summarized in [Table 4-4](#), the respiratory or metabolic compensation for a simple primary disorder follows a predictable pattern. Significant deviation from these patterns suggests the presence of a mixed disorder. [Table 4-7](#) lists some common causes of mixed acid-base disturbances. The diagnosis of mixed acid-base disorders depends principally on evaluation of the clinical setting and on interpretation of acid-base patterns. However, even normal acid-base patterns may conceal mixed disorders.

Table 4-7. Common causes of mixed acid-base disorders

5 Hemostasis and Transfusion Therapy

Joseph J. Wizorek, Brian G. Rubin, and Morey A. Blinder

Hemostasis

I. Mechanisms of hemostasis

II. Evaluation of hemostasis

Bleeding Disorders

I. Platelet disorders

II. Disorders of coagulation

Therapy

I. Anticoagulation therapy

II. Antiplatelet therapy

III. Fibrinolytic therapy

Anemia, Transfusions, and Hemostatic Agents

I. Anemia

II. Transfusion therapy

III. Local hemostatic agents

Hemostasis

I. Mechanisms of hemostasis. Coagulation is a complex equilibrium between thrombotic, anticoagulant, and fibrinolytic processes. The disruption of these processes can lead to severe hemorrhagic or thrombotic complications.

- A. **Platelets** serve a primary role in hemostasis physically, by filling the defect, as well as biochemically, through the release of thrombotic mediators. Exposure of platelets to subendothelial collagen causes adhesion, a process that requires von Willebrand factor (vWF) and activation, which results in platelet aggregation through exposure of glycoproteins IIb and IIIa. Activation of platelets also precipitates release of vasoconstrictor agents, including adenosine diphosphate, thromboxane A₂, and serotonin.
- B. **Endogenous antiplatelet agents**
 1. **Prostacyclin** is produced by endothelium and acts to inhibit resting and activated platelet aggregation. Disruption of the endothelial surface at the site of vessel injury results in augmented platelet plug formation owing to the loss of prostacyclin.
 2. **Nitric oxide** is produced by endothelium and platelets and acts to downregulate P-selectin expression, thereby decreasing platelet aggregation. Chronic cigarette smoking decreases platelet nitric oxide production and may lead to a propensity for thrombosis.
- C. **Coagulation cascade** is a series of reactions involving activation of serine proteases (factors) that eventually leads to the production of a proteinaceous plug of cross-linked fibrin. Fibrin is produced by the action of thrombin on fibrinogen, and this insoluble fibrin then is cross-linked by activated factor XIII (factor XIIIa). The extrinsic and intrinsic pathways merge in the production of factor Xa, forming the common pathway (Fig. 5-1). The extrinsic pathway involves exposure of plasma and, in particular, factor VIIa to tissue factor, a lipoprotein that is found only in extravascular tissues. The physiologic events that lead to intrinsic pathway initiation have not been elucidated thus far. In addition to the enzymatic clotting factors, cofactors (e.g., factors V and VIII), phospholipids from platelet and endothelial cell surfaces, and calcium are required for normal coagulation. Deficiencies of any of the coagulation factors (except factor XII, high-molecular-weight kininogen, and prekallikrein) can lead to abnormal bleeding.

Fig. 5-1. Blood coagulation cascade. Plasma zymogens are sequentially converted to active proteases (arrows). Nonenzymatic protein cofactors (ovals) are required at several stages of the cascade. Factors IX and X and prothrombin are activated on phospholipid surfaces. Thrombin cleaves fibrinogen, yielding fibrin monomers that polymerize to form a clot. HMW, high molecular weight. (From Ewald GS, McKenzie CR, eds. *Manual of medical therapeutics*, 28th ed. Boston: Little, Brown, 1995, with permission.)

- D. **Endogenous anticoagulants** are important to restrict the procoagulant response to the specific area of vascular injury. Deficiencies in these anticoagulants lead to thrombosis.
 1. **Antithrombin III (AT III)** is an important inhibitor of the coagulation cascade. AT III complexes irreversibly with thrombin and factors IXa, Xa, and XIa. This inhibition is accelerated by endothelium-produced heparan sulfates and exogenously administered heparin.
 2. **The thrombomodulin–protein C–protein S system** also is important in the regulation of hemostasis *in vivo*. Protein C, a vitamin K–dependent proenzyme, is cleaved by thrombin to form activated protein C, a serine protease that inactivates factors Va and VIIIa in the presence of protein S. Activation of protein C is enhanced by thrombomodulin on intact endothelial cell surfaces.
- E. **Fibrinolytic system.** Once formed, fibrin can be degraded by plasmin. Plasmin is formed from the specific activation of fibrin-bound plasminogen by tissue plasminogen activator or by the direct activation of plasminogen by urokinase or streptokinase-plasminogen complex. Naturally occurring inhibitors of plasmin include plasminogen activator inhibitor, alpha₂-antiplasmin, and alpha₂-macroglobulin, along with the pharmacologic agents aprotinin and epsilon-aminocaproic acid (EACA).

II. Evaluation of hemostasis

- A. **History and physical examination.** A history of prolonged bleeding after injury, childbirth, or surgical or dental procedures should be sought from each patient before operation and provides important clues as to the risk for surgical bleeding. Patients with a platelet disorder typically have signs of skin or mucosal bleeding, such as petechiae, frequent bruising, epistaxis, menorrhagia, and prolonged bleeding after minor injuries. On the other hand, patients with deficiencies in one or more coagulation factors typically describe delayed development of soft-tissue hematomas or hemarthroses. The use of medications that affect platelet function and coagulation (e.g., aspirin, nonsteroidal antiinflammatory drugs, ethanol, contraceptives, or anticoagulants) should be determined. A family history of bleeding should be elicited. Physical examination should include an inspection of the patient's skin, oral mucosa, and joints for evidence of occult bleeding and liver or kidney disease, malignancy, or malnutrition. A personal or family history of venous thrombosis, particularly occurring in those younger than age 50 years, may also point to an increased perioperative risk of venous thromboembolism.
- B. **Laboratory evaluation**
 1. **Platelet function**
 - a. **Platelet count** (140,000–400,000/μL) usually is determined as part of an automated blood count. Abnormal values should be confirmed by a review of the blood smear. Ethylenediaminetetraacetic acid in the blood transport tube can cause platelet clumping and lead to artifactually low platelet counts.
 - b. **Bleeding time** (2.5–9.0 minutes) is a functional assay of platelet function. Thrombocytopenia as well as qualitative platelet disorders, von Willebrand's disease (vWD), vasculitides, and connective tissue disorders can prolong the bleeding time; however, the sensitivity and specificity of this test to predict surgical bleeding are poor, and it is not a reliable predictor of surgical bleeding.
 - c. **Platelet aggregometry** can be used to classify congenital platelet disorders but is less useful in the evaluation of acquired platelet dysfunction.
 - d. **vWF assays** include ristocetin cofactor activity (based on the ability of ristocetin to enable vWF to interact with platelet glycoprotein IIb *in vitro*), vWF antigen (factor VIII–related antigen), and vWF multimer analysis (to detect the quantity and function of vWF and allow for the classification of vWD).
 2. **Coagulation factor activity** is measured as the time that citrate-anticoagulated plasma takes to clot after the addition of calcium, phospholipid, and the appropriate activating agent. Proper collection and prompt performance of testing must be ensured for accuracy. Polycythemia creates an elevated anticoagulant-plasma ratio, thereby artificially lengthening the clotting times. Clotting factor deficiencies, heparin, fibrin degradation products (FDPs), or coagulation factor inhibitors can prolong clotting times as well.
 - a. **Prothrombin time (PT)** is the clotting time measured after the addition of thromboplastin, phospholipids, and calcium. This test assesses the factors of the extrinsic and common pathways and is most sensitive to factor VII deficiency. In most laboratories, the normal PT is 10–13 seconds, but variability occurs between facilities. The thromboplastins used vary in their responsiveness to warfarin-induced anticoagulation; therefore, the International

Normalized Ratio (INR) is used to standardize PT reporting between laboratories. The INR is the ratio of the measured PT to a mean laboratory control PT corrected for the sensitivity of the thromboplastin.

- b. **Partial thromboplastin time (PTT)** is the clotting time (26–36 seconds) for plasma that is preincubated in particulate material (i.e., contact activation) followed by the addition of phospholipid and calcium. Inhibitors or deficiencies of factors in the intrinsic or common pathways cause prolongations of the PTT. A prolonged PTT should be evaluated by a 50:50 mixture with normal plasma. Factor deficiencies are corrected by the addition of normal plasma, whereas prolongation due to inhibitors is not.
- c. **Thrombin time (TT)** is the clotting time for plasma after the addition of thrombin. Hypo- and dysfibrinogenemia, the presence of FDPs from disseminated intravascular coagulation (DIC), and heparin cause elevations of the normal TT (11–18 seconds). Prolongation of the TT by heparin can be corrected by the addition of protamine sulfate to the assay.
- d. **Factor assays**
 1. **Fibrinogen level** (150–360 mg/dL) can be underestimated by the TT (e.g., in DIC) by the presence of FDPs. Therefore, clotting assays are generally used to determine fibrinogen concentration.
 2. **FDP elevations** (>8 µg/mL) occur in DIC, in association with thromboembolic events and during administration of fibrinolytic therapy. D-dimer levels reflect fibrinolysis and have been used in early identification of patients with venous thromboembolism but offer no advantage in the diagnosis of DIC compared to the FDP.
 3. **Specific factor assays** measure the ability of a patient's plasma to correct specific factor-deficient plasma clotting times so that each factor can be determined individually. The normal range for most factors is 60–160% of activity of normal pooled plasma.

Bleeding Disorders

I. Platelet disorders

- A. **Thrombocytopenia** is defined as a platelet count of less than 140,000/µL. Significant bleeding usually does not occur with platelet counts greater than 50,000/µL, and severe spontaneous bleeding rarely occurs with platelet counts greater than 10,000/µL, provided that platelet function is normal. Platelet counts greater than 50,000/µL should be maintained during surgical procedures, using platelet transfusions or other appropriate therapy administered at the start of surgery. In cases of serious hemorrhage, however, platelet transfusions should be administered to achieve platelet counts greater than 100,000/µL wherever possible. Each unit of platelets increases the platelet count by 5,000–10,000/µL and is usually administered as a pool of 6 units. Venipuncture and intramuscular injections and rectal examinations, suppositories, and enemas should be limited in patients with severe thrombocytopenia. Drugs that are known to inhibit platelet function (e.g., nonsteroidal antiinflammatory drugs and aspirin) should be discontinued. Nearly all thrombocytopenia is acquired. It can be caused by decreased platelet production, increased platelet destruction, or platelet sequestration. Isolated thrombocytopenia with normal red blood cell (RBC) and white blood cell (WBC) counts typically occurs with platelet destruction.
 1. **Drug-induced thrombocytopenia.** Many drugs can affect platelet production or cause increased platelet destruction ([Table 5-1](#)). Increased destruction of platelets is most commonly the result of an immune mechanism in which platelets are destroyed by complement activation by drug-antibody complexes. **All nonessential drugs should be discontinued until the cause of the thrombocytopenia is identified.** Drug-induced thrombocytopenia typically resolves within 7–10 days after cessation of the offending agent. Occasionally, the thrombocytopenia may be long-lived if the responsible medication is not cleared rapidly from the body (e.g., gold salts). Prednisone (1 mg/kg per day p.o.) may facilitate recovery of platelet counts.

Table 5-1. Commonly used drugs affecting platelet number and function

Heparin-induced thrombocytopenia is a potentially life-threatening reaction that may cause venous and arterial thromboembolic events. A mild thrombocytopenia (>100,000/µL) that occurs 2–10 days after the initiation of heparin therapy is not immune mediated and does not require cessation of heparin. A severe immune-mediated syndrome caused by heparin-dependent antiplatelet antibodies occurs 4–15 days after initial exposure to heparin but within hours after reexposure. In 10–50% of cases, thromboembolic events ensue, including extensive arterial thrombosis. Bleeding complications are rare, despite the thrombocytopenia. Diagnosis can be confirmed by ¹⁴C-serotonin release studies or via an enzyme-linked immunosorbent assay. This phenomenon is not type, dose, or route dependent. All heparin, including heparin flushes and heparin-coated lines, may cause or contribute to the reaction and must be discontinued or removed. Initiation of warfarin in the setting of heparin-induced thrombocytopenia is contraindicated and is associated with the development of venous limb gangrene. Anticoagulation with a thrombin inhibitor (hirudin or argatroban) or a heparinoid (danaparoid) is recommended.

2. **Autoimmune or idiopathic thrombocytopenic purpura** (see [Chapter 19](#))
3. **Thrombotic thrombocytopenic purpura** is a syndrome of microangiopathic hemolytic anemia and thrombocytopenia that occurs spontaneously or in association with pregnancy, carcinoma, infection, chemotherapy, or transplantation. It is suggested by recognizing schistocytes on the peripheral blood smear. Treatment consists of daily plasmapheresis using fresh frozen plasma (FFP). Prednisone (1 mg/kg per day p.o.) usually is used as initial therapy as well. Aspirin (325 mg per day p.o.) might reduce the frequency of thrombotic complications. Splenectomy appears to be beneficial in the treatment of relapsed disease.
4. **Dilutional thrombocytopenia** can occur with rapid blood product replacement for massive hemorrhage. No formula predicts accurate platelet requirements with the transfusion of blood cells. Therefore, frequent platelet counts should be used to guide platelet transfusion therapy. In the setting of ongoing hemorrhage, empiric platelet transfusion is appropriate.
5. **Other causes of thrombocytopenia** include pregnancy, HIV infection, DIC, sepsis, and hematopoietic disorders.
- B. **Thrombocytosis** is defined as a platelet count of greater than 600,000/µL. It can occur primarily, as with myeloproliferative disorders, or secondarily (e.g., after splenectomy) with iron deficiency, with carcinomas, or with chronic inflammatory disorders. Essential thrombocytosis, a myeloproliferative disorder with platelet counts greater than 600,000/µL may cause a thrombotic or hemorrhagic syndrome and can be treated with hydroxyurea (15 mg/kg per day p.o.) or anagrelide (1–4 mg per day p.o. in 2–4 divided doses) to lower platelet counts and decrease the associated risk of thrombosis and bleeding. Secondary thrombocytosis requires no specific therapy and is generally not associated with an increased thrombotic risk.
- C. **Qualitative platelet dysfunction**
 1. **Acquired defects** (e.g., those that result from uremia, liver disease, or cardiopulmonary bypass or from the use of such drugs as aspirin, nonsteroidal antiinflammatory drugs, and beta-lactam antibiotics) account for most qualitative platelet disorders. Treatment is aimed at the underlying cause. Because the effect of aspirin on platelets is irreversible, this medication should be discontinued at least 1 week before elective operations to allow new, functional platelets to form. For severe bleeding, platelet transfusions may be necessary. Desmopressin acetate (DDAVP, 0.3 µg/kg i.v. before operation) may limit bleeding from platelet dysfunction, particularly in uremic patients. Conjugated estrogens (0.6 mg/kg per day i.v. for 5 days) also can improve hemostatic function.
 2. **vWD** is an autosomal disorder characterized by a prolonged bleeding time secondary to a qualitative or quantitative deficiency of vWF. Classification of vWD subtype is required before therapy is started. Type 1 (mild quantitative deficiency) accounts for 80–90% of vWD. DDAVP (0.3 µg/kg every 12–24 hours i.v.) stimulates endothelial release of vWF and usually is effective for 2–3 days in this form of vWD. Because type 2B vWD results from accelerated clearance of platelets due to a vWF-platelet interaction, DDAVP should not be used in patients with type 2B vWD. If replacement therapy is necessary, factor VIII concentrates are the safest blood products and contain high concentrations of vWF. Cryoprecipitate (1–2 bags/10 kg i.v.) also corrects bleeding times in patients with vWD but carries the risk of viral transmission.
 3. **Congenital platelet disorders**, such as Bernard-Soulier syndrome, Glanzmann's thrombasthenia, and storage pool defects, are rare and are generally treated with platelet transfusions to control bleeding.

II. Disorders of coagulation

A. Inherited factor deficiencies

1. **Hemophilia** is an X-linked deficiency of either factor VIII (hemophilia A) or factor IX (hemophilia B, Christmas disease) and can present with spontaneous hemarthroses, hemorrhage, or prolonged bleeding after injury or operation. Patients with more than 5–20% of normal factor activity rarely experience spontaneous bleeding but may bleed extensively during surgery or after trauma. Patients with 1–5% activity have prolonged bleeding with minor injuries but rarely develop spontaneous hemarthroses. In patients with less than 1% activity, frequent hemarthroses and severe bleeding episodes develop. The diagnosis is suggested by an elevated PTT, normal PT, and normal bleeding time. Factor activity assays confirm the diagnosis.
 - a. **Treatment of hemophilia A** is based on the degree of bleeding and the severity of the disease.

1. **Minor bleeding** often is controlled locally without factor replacement therapy. DDAVP (0.3 µg/kg every 12–24 hours i.v.) stimulates the release of vWF, which complexes with factor VIII, thereby sheltering it from the circulation. It can increase factor VIII levels two- to threefold and therefore helps to control minor bleeding in patients with milder disease. For minor dental procedures, EACA, an inhibitor of fibrinolysis, can be used in conjunction with 50% factor replacement for control of bleeding. EACA (50–100 mg/kg every 6 hours p.o. or i.v.) is administered the day before operation and for 3–5 days afterward. EACA should not be used for the treatment of hematuria.
 2. **Major bleeding** (e.g., during surgical procedures) requires factor VIII replacement. Factor VIII activity should be restored to 100% immediately before operation and be maintained at least to 50% for 3–5 days afterward. Neurosurgical patients and those with head trauma should be restored immediately to 100% activity and be maintained for 5 days at greater than 80%. The usual dosing scheme to maintain factor levels at 50–100% is 50 units/kg loading dose followed by 25 units/kg every 8–12 hours. Plasma-purified and recombinant factor VIII are effective in preventing and controlling bleeding episodes, but product availability and cost may affect the choice of treatment. All available data suggest that these products are free of viral contamination. Cryoprecipitate contains factor VIII, vWF, and fibrinogen and can be used to treat patients with suspected hemophilia A for control of bleeding. However, each unit of cryoprecipitate carries the same risk of viral transmission as a unit of packed RBCs.
- b. **Treatment of hemophilia B.** The treatment of choice for factor IX replacement therapy is purified factor IX. Because factor IX has more extensive extravascular distribution than does factor VIII, the dose required to correct factor IX deficiency is roughly twice that required to replace factor VIII activity [see [section II.A.1.\(a\)\(2\)](#)]. FFP can be used for minor bleeding. Cryoprecipitate and DDAVP are not useful in the treatment of factor IX deficiency.
 - c. **Factor inhibitors for the treatment of hemophilia.** Antibodies to factor VIII and factor IX occur in 5–10% of patients with hemophilia A and 1% of those with hemophilia B, and they occasionally occur spontaneously. Patients with low titers of antibodies can respond to factor replacement therapy in higher than normal doses. Patients with high antifactor antibodies should not receive factor replacement therapy for minor episodes of bleeding. Hemorrhage in high-titer patients should always be considered life threatening, and any surgery should be undertaken with extreme caution. Recombinant factor VIIa has been approved for treatment in this setting. Prothrombin complex concentrate and activated prothrombin complex concentrate can be used but are associated with thromboembolic disease. Porcine factor VIII can be used to treat factor VIII inhibitors but may lead to the formation of specific antibodies. Inhibitory antibody titers can be lowered by plasmapheresis, immunoadsorption, immunoglobulin therapy, or immunosuppressive therapy.
2. **Other inherited factor deficiencies** account for fewer than 10% of severe inherited factor deficiencies. Deficiencies of factors XII, high-molecular-weight kininogen, or prekallikrein do not cause bleeding and require no treatment. Factor XI deficiency may lead to bleeding with operation or injury. Factor deficiencies can be corrected with FFP, if required for bleeding diatheses.
- B. Acquired factor deficiencies**
1. **Vitamin K deficiency** leads to the production of inactive, noncarboxylated forms of prothrombin; factors VII, IX, and X; and proteins C and S. The diagnosis should be considered in a patient with a prolonged PT, which corrects with a 50:50 mixture of normal plasma. Vitamin K deficiency can occur in patients without oral intake for 1 week, with biliary obstruction, with malabsorption, and in those receiving antibiotics or warfarin.
 - a. **Prophylaxis.** Patients on nothing-by-mouth status should receive vitamin K₁ s.c. (10 mg 3 times a week) prophylactically or as a supplement in total parenteral nutrition.
 - b. **Treatment.** For mild deficiencies, vitamin K₁ (10–15 mg every 1–3 days s.c. or i.v.) corrects the deficit. Severe deficiencies (e.g., with warfarin overdose) might require much higher doses and longer duration of therapy. If patients have a PT that is greater than 1.5 times the control caused by vitamin K deficiency or warfarin therapy and have ongoing hemorrhage or require emergent surgery, FFP (2–4 units i.v.) should be used for correction along with vitamin K. Further FFP therapy is guided by the degree of bleeding and the PT.
 2. **Liver dysfunction** causes decreased production of all coagulation factors except vWF. Additionally, the coagulopathy associated with decreased circulating factors is worsened by thrombocytopenia from hypersplenism. Parenteral vitamin K₁ administration (10–15 mg every 1–3 days s.c. or i.v.) infrequently corrects the coagulopathy. FFP administration often improves the hemostatic defect transiently, and platelet transfusion can be used to augment the platelet count.
 3. **DIC** has many inciting causes, including sepsis (particularly associated with gram-negative bacteria, meningococcemia, Rocky Mountain spotted fever, and viral infections), extensive trauma or burns, necrotic tissue, intravascular antibody-antigen immune reactions, hematologic and other malignancies, liver failure, obstetric complications, intravascular prosthetic devices, and toxins or venoms. In DIC, coagulation and fibrinolytic pathways are overactive beyond normal constraints. Usually, DIC presents initially with microvascular thrombotic complications that involve the vascular beds of the kidney, brain, lung, and skin. As the process develops, however, the consumption of coagulation factors, particularly fibrinogen, may lead to bleeding. Activation of the fibrinolytic system lyses fibrinogen and fibrin clots, worsening the disorder and leading to elevations of FDP. Laboratory findings in DIC include thrombocytopenia, hypofibrinogenemia, increased FDPs (or D-dimer), and prolonged TT and PTT. The treatment of DIC initially involves the treatment of the underlying cause. Conscientious management of hemodynamic and oxygenation support is critical. Correction of coagulopathy with platelet transfusions, FFP, and cryoprecipitate should be undertaken for bleeding complications. Early in DIC, when microvascular thrombosis is occurring, the thrombotic process may be inhibited when heparin is used in low doses (initially, 500 units per hour i.v. without a bolus) and titrated upward as tolerated, but the benefit remains unproven.
- C. Hypercoagulable states**
1. **Inherited**
 - a. **AT III deficiency** is an autosomal disorder that presents with recurrent thrombosis, usually in the second decade of life. The thromboses are typically venous, but even aortic thrombosis has been reported. A family history of recurrent thromboses should be determined. Assays for AT III levels typically are falsely decreased in the setting of acute thrombosis and while the patient is receiving heparin. Treatment of acute thromboembolism is with heparin, which can usually be administered successfully if the AT III level is greater than 50% of normal. Patients with AT III deficiency and an episode of thrombosis commonly are treated with lifelong oral anticoagulation. Women with AT III deficiency should receive full-dose heparin or low-molecular-weight heparin (LMWH) anticoagulation during pregnancy to prevent the risk of deep venous thrombosis (DVT). AT III-deficient patients should have the AT III level restored to more than 80% of normal activity with AT III concentrate before operation or peripartum.
 - b. **Protein C deficiency** can lead to a propensity for venous and arterial thrombosis by lowering factor Va and VIIIa inactivation, thereby allowing coagulation to continue. Besides the inherited type, protein C deficiency is encountered in patients with liver failure and in those who are receiving warfarin therapy. Initial treatment for symptomatic patients includes heparin anticoagulation followed by warfarin therapy. In individuals with diminished protein C activity, full heparin anticoagulation must be confirmed before the initiation of warfarin because warfarin transiently lowers protein C levels further and potentially worsens the hypercoagulable state. Patients with protein C deficiency but with no history of thrombosis do not require prophylactic anticoagulation.
 - c. **Protein S deficiency** can lead to thrombotic complications similar to those in protein C deficiency. Therapy is similar to that for protein C deficiency.
 - d. **Factor V Leiden** is a genetic mutation in factor V that renders it resistant to breakdown by activated protein C (*N Engl J Med* 330:517, 1994). This mutation occurs in approximately 5% of those of European heritage but is much less common in others. The relative risk of DVT in heterozygotes is five- to ten-fold and, in homozygotes, up to 80-fold (*Blood* 85:1504, 1995). Research to date suggests that routine preoperative screening in asymptomatic patients is unnecessary. Therapy for venous thromboembolism consists of standard warfarin therapy, but optimal duration has not yet been established.
 - e. **Hyperhomocystinemia** in adults is defined as a plasma homocysteine level above the ninety-fifth percentile (18.5 µmol/L). It may be an independent risk factor for venous thromboembolic disease and atherosclerotic disease. Clinically important elevations may be present in 5–10% of the population; these are the result of a genetic alteration in the enzymes involved in homocysteine metabolism. Treatment with folate and vitamin B₁₂ is effective in lowering homocysteine levels, but these patients may also require oral anticoagulation with warfarin, particularly if they also have factor V Leiden.
 - f. **Prothrombin gene mutation** occurs in approximately 2% of patients of European heritage and is associated with a two- to threefold increased risk of venous thromboembolic disease.
 2. **Acquired**
 - a. **Lupuslike inhibitors** are IgG, IgA, or IgM immunoglobulins that are targeted against antigens composed in part of platelet and endothelial cell phospholipids. The antibodies may be detected in patients with systemic lupus erythematosus or other immunologic disorders, in those with infections (e.g., HIV), or in those who receive predisposing medications (e.g., chlorpromazine, procainamide, or hydralazine). Approximately half of patients with lupuslike inhibitors have no identifiable predisposing conditions and are said to have antiphospholipid antibody syndrome. The diagnosis is suggested by a prolonged PTT and can be confirmed using specific tests, such as Russell's viper venom clotting time (for lupus anticoagulant) and anticardiolipin antibody immunoassay. Patients with these antibodies are at risk for arterial and venous thrombosis. In many cases, therefore, long-term warfarin anticoagulation is indicated. In some instances, the immunoglobulins resolve spontaneously or with immunosuppressive therapy.
 - b. **Other acquired hypercoagulable states** include sepsis, malignancies, pregnancy or the use of estrogen therapy, intravascular hemolysis (e.g., hemolytic anemia or after cardiopulmonary bypass), and the localized propensity to thrombosis in arteries that have recently undergone endarterectomy or angioplasty or in new prosthetic vascular grafts.

Therapy

I. Anticoagulation therapy

- A. **Principles and indications.** Anticoagulation is used to prevent and treat thrombosis and thromboembolic events. Before therapy is instituted, careful consideration must be given to the risk of thromboembolism and to anticoagulation-induced bleeding complications. Current indications for antithrombotic therapy are discussed in detail with specific diseases; they include atrial fibrillation, mechanical prosthetic heart valves, DVT, pulmonary embolism (PE), stroke prevention, and acute arterial or graft occlusion. Relative contraindications to anticoagulation therapy are listed in [Table 5-2](#).

Table 5-2. Relative contraindications to anticoagulation therapy

B. Heparin and related therapy

1. **Standard unfractionated heparins** act principally by potentiating the action of AT III and by accelerating the inhibition of factor Xa and thrombin. AT III also inhibits platelet function.
 - a. **Administration.** Heparin is administered parenterally, either s.c. or i.v. Therapy for prophylaxis of venous thrombosis (typically 5,000 units s.c.) should be administered before surgery and every 8–12 hours thereafter. Treatment of venous thrombosis requires a higher daily dose, typically given as 100–150 units/kg i.v. bolus, followed by 1,000–2,000 units per hour (or 18 units/kg per hour continuous i.v.). The amount of heparin is typically prepared as 25,000 units/250-mL bag of intravenous fluid to prevent inadvertent massive overdose. PTT should be measured before initiation of heparin and 6 hours after the initial bolus or at each change in dosing. A therapeutic PTT of 1.5–2.0 times the control (approximately 50–80 seconds) should be maintained. Typical dosages of intravenous heparin range from 800–1,500 units per hour. High-dose heparin therapy for arterial embolism and PE can be used in some patients who are not operative candidates. For these individuals, 20,000 units of heparin are given as an intravenous bolus, followed by 2,000–4,000 units per hour i.v. Platelet counts should be measured daily when initiating heparin therapy and periodically thereafter. The development of unexplained thrombocytopenia (<100,000/ μ L) should prompt discontinuation of heparin therapy.
 - b. The **toxicity** that is associated with heparin therapy is most frequently bleeding. Immediate reassessment of the PT, PTT, and complete blood cell count should be undertaken. Gastrointestinal (GI) bleeding that occurs while a patient is therapeutically anticoagulated suggests an occult source and warrants further evaluation. Heparin-induced thrombocytopenia is an uncommon but potentially devastating complication of heparin therapy and must be recognized early to prevent irreversible morbidity (see Bleeding Disorders, [section I.A](#)).
 - c. **Heparin clearance** is accomplished within hours after cessation of heparin administration. Reversal can be achieved more quickly with protamine sulfate. Each milligram of protamine sulfate reverses 100 units of heparin. Therefore, for patients who are receiving continuous intravenous heparin infusions, enough protamine sulfate should be administered to neutralize approximately one-half of the preceding hour's dose by slow intravenous infusion (i.e., <50 mg/10 min). The PTT or activated clotting time can be used to assess the adequacy of the reversal. Anaphylactic reactions can occur to protamine, especially in diabetic patients who have received protamine-insulin [neutral protamine Hagedorn (NPH)] preparations.
 2. **LMWHs** (enoxaparin, dalteparin and tinzaparin) are fractionated forms of heparin that are playing an ever-increasing role in anticoagulant therapy. The anticoagulant effect of LMWHs depends less on thrombin inhibition than does that of unfractionated heparin and is due to a more potent and consistent factor Xa inhibition. The advantages of LMWHs include a more predictable anticoagulant effect, less platelet interaction, and a longer half-life. Therefore, dosing is based on weight, and laboratory monitoring is not typically needed. In prevention of postoperative DVT and PE, LMWH is as effective as low-dose unfractionated heparin in patients who are undergoing laparotomy and appears more effective in patients undergoing orthopedic procedures or after multiple trauma or spinal injury. Fixed-dose subcutaneous regimens of LMWH are as effective as adjusted-dose intravenous unfractionated heparin in the initial treatment of established DVT or PE that does not require fibrinolysis or surgery. These drugs also appear to be of particular value for longer-term therapy in patients with a contraindication to oral anticoagulant treatment (e.g., pregnant patients who cannot take warfarin). Because LMWHs have a longer half-life and no effective antidote, they must be used with caution in surgical patients or in those in whom a bleeding risk has been substantiated.
 3. **Danaparoid** is an anti-factor Xa heparinoid that is an effective anticoagulant in patients in whom heparin is contraindicated.
- C. **Direct thrombin inhibitors**
1. **Recombinant hirudin and hirudin analogues** (lepirudin) are a class of compounds derived from leeches, which are direct inhibitors of thrombin. Early studies indicate excellent prophylaxis of DVT without bleeding complications.
 2. **Argatroban** is a synthetic direct thrombin inhibitor with actions that are similar to those of the recombinant and naturally occurring hirudins.
- D. **Warfarin** is an oral anticoagulant that prevents vitamin K–mediated carboxylation of factors II, VII, IX, and X as well as proteins C and S. The vitamin K–dependent factors decay with varying half-lives: factor II (prothrombin), 3 days; factor VII, 6 hours; factor IX, 24 hours; and factor X, approximately 2 days, so that the full anticoagulant effect of warfarin is not apparent for 5–7 days. Therefore, when immediate anticoagulation is necessary, heparin or related agents must be used initially.
1. **Administration.** Warfarin usually is initiated at 5.0–10.0 mg per day for 2 days followed by dosage adjustment based on daily INR. Use of the INR standardizes the assessment of anticoagulation by warfarin and should be used in place of the PT or PT ratio. Elderly patients, those with hepatic insufficiency, and those who are receiving parenteral nutrition or broad-spectrum antibiotics should be given lower initial doses of warfarin. Daily doses of warfarin usually range from 2 to 15 mg per day p.o. for adequate anticoagulation. An INR of 2–3 is therapeutic for most indications, but patients with prosthetic heart valves should be maintained with an INR of 2.5–3.5. Once a stable INR is obtained, it can be monitored biweekly or monthly.
 2. **Toxicity.** Bleeding in patients who are treated with warfarin occurs with a frequency of approximately 10% per year, with fatal hemorrhagic events developing in 0.5–1.0% of patients annually. The risk of bleeding correlates directly with the INR. Warfarin-induced skin necrosis, caused by dermal venous thrombosis, occurs rarely when warfarin therapy is initiated in patients who are not already anticoagulated and is usually associated with hypercoagulability caused by protein C deficiency. **Because warfarin can produce significant birth defects and fetal death, it should not be used during pregnancy.** Changes in medications and diet that affect warfarin or vitamin K levels ([Table 5-3](#)) require more vigilant INR monitoring and warfarin adjustment.

Table 5-3. Commonly used drugs that affect the prothrombin time in patients receiving oral anticoagulation

3. **Reversal of warfarin-induced anticoagulation** requires up to a week after discontinuation of therapy. Vitamin K administration can be used to correct warfarin anticoagulation within 1–2 days. The appropriate vitamin K dosage depends on the INR and the urgency with which correction must be accomplished. For patients with an INR of 6–10 and no serious bleeding, 0.5–1.0 mg i.v. vitamin K can be used. In patients with an INR of 10–20, 3–5 mg vitamin K is generally recommended to improve the coagulopathy. For patients with bleeding or an INR of more than 20, 10 mg of vitamin K should be administered. Serial INR should be followed every 6 hours. In addition, FFP (2–4 units i.v.) is administered to patients with a coagulopathy and ongoing hemorrhage.

II. Antiplatelet therapy

- A. **Aspirin** irreversibly acetylates cyclooxygenase, inhibiting platelet synthesis of thromboxane A₂. It is useful in the prevention of transient ischemic attacks, stroke, myocardial infarction, and coronary and vascular graft occlusion. The optimal dose of aspirin for platelet inhibition is unknown, but 80–325 mg per day p.o. is recommended. Chronic aspirin use can lead to the development of peptic ulcers and elevation of the blood urea and uric acid. Use of aspirin in patients who are receiving anticoagulant therapy predisposes to increased bleeding complications.
- B. **Thienopyridine derivatives.** Ticlopidine (125 mg b.i.d. p.o.) alters the platelet membrane, thereby inhibiting platelet aggregation. This agent has been shown to lower the stroke, myocardial infarction, and vascular death rate of patients who have had a previous stroke (*Lancet* 1:1215, 1989). Ticlopidine can cause reversible neutropenia, pancytopenia, and agranulocytosis. Clopidogrel, an agent with similar mechanism of action, appears to have fewer side effects than ticlopidine.
- C. **Dextran** decreases platelet aggregation and adhesion, but the mechanism of this action is not completely understood. It also improves blood flow by volume expansion and reduces factor VIII activity. Dextran (500 mL i.v. given just before surgery followed by 500–1,000 mL per day i.v. during the postoperative period) can be used for prevention of DVT and PE. In addition, femoral-to-tibial bypass graft patency improves with perioperative dextran use (500 mL at 100 mL per hour i.v. on induction of anesthesia, followed by 500 mL at 75 mL per hour per day i.v. starting on the day of surgery for 4 days) (*J Vasc Surg* 1:765, 1984). Complications of dextran infusion include bleeding, volume overload, and allergic or anaphylactoid reactions. Allergic reactions are prevented or decreased by

preinfusion of short-chain dextrans. Of the two types of dextran that are available, dextran 40 leads to less volume overload and bleeding complications than does dextran 70.

- D. **Glycoprotein IIb/IIIa inhibitors** are a new class of drugs that block platelet aggregation and platelet adhesion to fibrin. The first of this class is a chimeric monoclonal antibody, abciximab. Other peptidomimetic compounds have also been put into clinical trials. These agents have been used primarily to prevent restenosis after coronary angioplasty. Hemorrhagic complications have been relatively infrequent; however, there are occasional reports of acute profound thrombocytopenia with this therapy.

III. Fibrinolytic therapy

- A. **Indications and contraindications.** Thrombolytic therapy is used for iliofemoral DVT; axillary vein and superior vena caval thrombosis; PE in hemodynamically unstable patients; acute thrombosis of peripheral, mesenteric, and coronary arteries; acute vascular graft occlusion; thrombosis of hemodialysis access grafts; and occlusion of venous catheters. Contraindications to fibrinolytic therapy are listed in [Table 5-4](#).

Table 5-4. Contraindications to fibrinolytic therapy

- B. **Dosage** depends on the agent and the indication. Tissue plasminogen activator or recombinant analogues (alteplase, reteplase), as well as streptokinase, are used for lysis of catheter, venous, and peripheral arterial thrombi. The appropriate doses for systemic and catheter-directed fibrinolytic therapy are described in [Chapter 7](#) and [Chapter 23](#).

Anemia, Transfusions, and Hemostatic Agents

I. Anemia

- A. **Evaluation.** *Anemia* is a decreased circulating RBC mass and is defined as a hemoglobin level of less than 12 g/dL in women and less than 14 g/dL in men. In addition to hemoglobin and hematocrit, peripheral blood smear, measurement of the reticulocyte count, and mean cellular volume help to evaluate and identify the cause of the anemia. The blood smear is used to identify abnormalities in RBC size and shape, WBCs, and platelets. The reticulocyte count assesses the bone marrow response to anemia. A normal or low reticulocyte count in the presence of anemia suggests bone marrow suppression. The mean cellular volume helps to classify the anemia and distinguish its cause.
- B. **Causes of anemia** ([Table 5-5](#))

Table 5-5. Classification of anemia based on red blood cell (RBC) kinetics

1. Anemias associated with increased RBC destruction

- a. **Bleeding** is the most frequently encountered cause of RBC destruction. Most surgical patients have an obvious etiology for blood loss; however, sources of occult bleeding include the GI tract, uterus, urinary tract, and retroperitoneum.
- b. **Hemolytic anemias**
1. **Hereditary hemolytic anemias** include the hemoglobinopathies (e.g., sickle cell disease), RBC membrane abnormalities (e.g., hereditary spherocytosis), and the RBC enzymopathies (e.g., glucose 6-phosphate dehydrogenase deficiency). Sickle cell disease is caused by abnormal hemoglobin that polymerizes under decreased oxygen tension. Dehydration and hypoxia must be avoided to prevent sickling. This is particularly true in patients with sickle cell disease who undergo general anesthesia. Transfusion to increase hemoglobin levels to 10 g/dL may be effective in reducing complications after major operations, with fewer complications than more aggressive exchange transfusion regimens (*N Engl J Med* 333:106, 1995).
 2. **Acquired hemolytic anemias** include autoimmune hemolytic anemia, drug-induced hemolytic anemia, microangiopathic hemolytic anemia, and traumatic hemolytic anemias, such as those induced by malfunctioning prosthetic aortic valves or vascular bypass grafts.

2. Anemias associated with decreased RBC production

- a. **Aplastic anemia** is an acquired defect of bone marrow stem cells associated with leukopenia and thrombocytopenia. Aplastic anemia may result from drug-related causes [approximately 20% (e.g., phenylbutazone, gold, D-penicillamine, anticonvulsants, sulfonamides, chloramphenicol, benzene)], viral infection [e.g., hepatitis, Epstein-Barr virus, cytomegalovirus (CMV)], and, most commonly, idiopathic causes. The diagnosis is confirmed with a bone marrow biopsy. Initial treatment is supportive, with cessation of any possibly offending agents. Bone marrow transplantation or immunosuppressive therapy often is required.
- b. **Iron-deficiency anemia** is most commonly caused by blood loss from either menstrual bleeding or occult GI blood loss. Sources of GI blood loss include peptic ulcer disease, gastritis, hemorrhoids, angiodysplasia, and colonic adenocarcinoma. In postmenopausal women and in men with iron-deficiency anemia, a complete GI evaluation for a potential source of blood loss must be performed. Iron requirements for women are increased during pregnancy owing to the transfer of iron to the fetus. Iron uptake by the intestine might be diminished in patients who have had a partial or total gastrectomy, patients with achlorhydria, and patients with chronic diarrhea or intestinal malabsorption. The diagnosis is suggested by hypochromic microcytic anemia, low serum iron levels (<60 µg/dL), increased total iron-binding capacity (>360 µg/dL), and low serum ferritin levels (<12 µg/L). A bone marrow biopsy that demonstrates absence of iron staining or a response to a trial of iron therapy establishes the diagnosis definitively. Oral iron replacement (ferrous sulfate, 325 mg 3 times a day p.o. between meals) is usually sufficient; it is generally administered with a stool softener, such as docusate sodium, to prevent constipation and obstipation. Iron polysaccharide (150 mg b.i.d. p.o.) appears to be a better-tolerated alternative. Iron dextran also can be administered intramuscularly (100 mg per day) or as a single-dose intravenous preparation (1–2 g i.v. over 3–6 hours) in patients with malabsorption or intolerance of oral preparations.
- c. **Megaloblastic anemias** are associated with a deficiency of cobalamin (vitamin B₁₂) or folic acid. These deficiencies cause decreased hematopoietic cell DNA synthesis. Cobalamin, derived in the diet from meat and dairy products, is dependent for absorption on intrinsic factor production by the gastric parietal cells and by specific ileal receptors. Therefore, pernicious anemia, gastrectomy, ileal resection or ileitis, intestinal parasites, and bacterial overgrowth can lead to vitamin B₁₂ deficiency. However, because only a small portion of the body's stores is used each day, vitamin B₁₂ deficiency takes several years to manifest. In addition to anemia, vitamin B₁₂ deficiency can create numbness and paresthesias of the extremities, weakness, ataxia, and poor finger coordination. In contrast to cobalamin deficiency, folic acid deficiency can develop within weeks of decreased intake. It can arise from decreased intake (e.g., alcohol abuse), malabsorption, or increased use (e.g., pregnancy or hemolysis). Serum vitamin B₁₂ levels (<100 pg/mL) and serum folate levels (<4 ng/mL) are used to establish the diagnosis of deficiency. RBC folate levels are a more sensitive measurement than are serum levels. Therapy for vitamin B₁₂ deficiency involves replacement with cyanocobalamin (1 mg per day i.m. for 7 days, then weekly for 1–2 months, then every month). Folic acid is replenished, 1 mg per day p.o., until the deficiency is corrected. An incomplete response to therapy might indicate a coexisting iron deficiency and occurs in one-third of patients.
- d. **Other anemias** associated with decreased RBC production include anemia due to renal insufficiency, chronic disease, chemotherapy, and the thalassemias. Anemia from renal failure is treated effectively with erythropoietin (50–100 units/kg s.c. 3 times a week).

II. Transfusion therapy. Indications for transfusion should be noted in the medical record. The **risks and benefits** of transfusion therapy must be considered carefully in each situation. **Informed consent** also should be obtained before blood products are administered, if possible. Before elective operations that are likely to require blood transfusion, the available options (e.g., autologous or directed donation of blood) should be discussed with the patient in time to allow for the collection process.

A. RBC transfusion

1. **Indications.** RBC transfusions are used to treat anemia or anticipated anemia to improve the oxygen-carrying capacity of the blood. Routinely, a hemoglobin level of 7–8 g/dL is ample for tissue oxygenation in a normovolemic patient. However, therapy must be individualized based on the patient's age and cardiovascular and pulmonary status, the type of transfusion necessary (i.e., homologous vs. autologous), and the expectations of further blood loss. Correctable causes of anemia, such as folate, vitamin B₁₂, and iron deficiency, must be identified because these patients typically do not require blood transfusion. Chronic anemia, particularly the anemia due to renal disease, is treated with erythropoietin (50–100 units/kg s.c. 3 times a week).
2. **Preparation.** RBCs currently are administered most commonly as packed RBCs. When available, whole blood can be used for blood volume replacement that is associated with recent hemorrhage (i.e., in GI bleeding, major surgery, or trauma). Packed RBCs can be stored for approximately 1 month after collection, depending on the preservative. Before administration, blood must be tested to decrease transfusion reactions. Blood typing and screening test the recipient's RBCs for antigens A, B, and Rh and screen the recipient's serum for the presence of antibodies to a panel of RBC antigens. Each unit must be cross-matched against the recipient's serum to check for preformed antibodies in the recipient's serum against RBC antigens on the donor's RBCs. In emergency situations, blood transfusions might be required before the 30 minutes that are necessary for cross-matching. In this situation, type O/Rh-negative blood that has been prescreened for reactive antibodies is administered. After blood typing, type-specific blood can be given. The patients involved must be monitored closely for evidence of a transfusion reaction, and the transfusion should be discontinued immediately if such a reaction occurs.
3. **Administration.** Proper identification of the blood unit and patient is necessary to prevent transfusion errors, particularly in the operating room. Packed RBCs should be administered through a standard filter (170–260 μm) and an 18-gauge or larger intravenous catheter. One unit of packed RBCs raises the hemoglobin level approximately 1 g/dL and the hematocrit approximately 3%. The rate of transfusion is determined by the clinical situation; typically, however, blood must be administered within 4 hours. Patients are monitored for adverse reactions during the first 5–10 minutes of the transfusion and frequently thereafter. Those who need chronic transfusion therapy and organ transplant patients should be administered blood through a leukocyte-depleting filter. Immunocompromised patients and those who receive blood from first-degree relatives should be given irradiated blood to prevent graft-versus-host disease (GVHD).
4. **Complications**
 - a. **Infections.** Despite the institution of aggressive testing of the blood supply, the spread of transmissible agents is still a big concern with homologous blood transfusion because up to 1 per 34,000 units donated harbors residual infection (*N Engl J Med* 334:1685, 1996). Hepatitis C and B account for over 88% of the overall risk, where the risk of an infected donation passing all tests is approximately 1 per 100,000 and 1 per 60,000, respectively. The risk of HIV-1 transmission is approximately 1 per 500,000 units and that for human T-cell lymphoma virus is less than 1 per 600,000. Furthermore, new screening procedures have been calculated to reduce the risks by a further 27–72%. CMV transmission is a risk in CMV-negative immunocompromised patients and can be lowered by using either leukocyte-depleting filters or CMV-negative blood products. Bacteria and endotoxins can be infused with blood products, particularly those that are stored for prolonged periods. Parasitic infections also can be transmitted, although rarely, with blood products.
 - b. **Immune reactions**
 1. **Acute hemolytic reactions** are caused by preformed recipient antibodies to transfused RBC antigens. These reactions result in intravascular hemolysis and usually are produced by ABO or Rh incompatibility. Initially, symptoms in an awake patient might include restlessness, anxiety, flushing, chest or back pain, tachypnea, tachycardia, and nausea. These symptoms might progress to shock and renal failure with hemoglobinuria. In anesthetized or comatose patients, the first signs of a transfusion reaction can be excessive incisional bleeding or oozing from the mucous membranes. When a transfusion reaction is suspected, the infusion should be stopped immediately, and all intravenous tubing should be changed. A check of the identity of the donor unit and recipient is required because most ABO reactions today are the result of clerical error. A repeat of the cross-match should be performed with samples of the recipient's serum and the remaining donor unit. Plasma hemoglobin should be determined as well as serum bilirubin and blood coagulation parameters. Treatment includes maintenance of intravascular volume, hemodynamic support as needed, and preservation of renal function. Urine output should be maintained at greater than 100 mL per hour using volume resuscitation, diuretics, and mannitol, if needed. Alkalinization of the urine to a pH of more than 7.5 by adding sodium bicarbonate to the intravenous fluids (2–3 ampules of 7.5% sodium bicarbonate in 1,000 mL D5W) helps prevent precipitation of hemoglobin in the renal tubules.
 2. **Delayed hemolytic transfusion reactions** result from an anamnestic antibody response to antigens to which the recipient has been exposed. Transfused RBC survival is normal initially, but 1–25 days later, the RBCs are lysed rapidly. A decline of hemoglobin and an elevated bilirubin suggest this diagnosis. Specific treatment rarely is necessary, but severe cases should be treated like acute hemolytic reactions, with volume support and maintenance of urine output.
 3. **Nonhemolytic immune transfusion reactions** usually are caused by reactions to transfused WBCs, platelets, or plasma antigens. Fever, chills, urticaria, pruritus, and respiratory distress are consequences of these reactions. To avoid these reactions, most patients should be treated before transfusion with acetaminophen and diphenhydramine unless they are specifically contraindicated. Patients with antibodies against WBCs and platelets can receive RBCs from which WBCs have been removed by filters. Patients who lack IgA can acquire antibodies to IgA and develop severe anaphylactic reactions to blood transfusions that contain IgA. These patients should receive IgA-deficient blood or washed RBCs. Symptomatic treatment for nonhemolytic reactions includes acetaminophen and diphenhydramine (25–50 mg p.o. or i.v.), epinephrine, and glucocorticoid therapy. Meperidine (25–100 mg) is particularly helpful for shaking chills associated with transfusion reactions. Prophylaxis with acetaminophen, diphenhydramine, and hydrocortisone (100–150 mg i.v.) should be used before future transfusions in patients who have experienced severe reactions.
 4. **Noncardiogenic pulmonary edema** can be caused by preformed antileukocyte antibodies in the donor unit. This condition is transient and is managed with supportive care.
 5. **GVHD** can occur after transfusion of immunocompetent T cells into immunocompromised recipients or human leukocyte antigen-identical family members. GVHD presents with a rash, elevated liver function tests, and pancytopenia and has an associated mortality of more than 80%. Irradiation of donor blood from first-degree relatives of immunocompetent patients and all blood for immunocompromised patients prevent this complication.
 - c. **Volume overload after blood transfusion** can occur in patients with poor cardiac or renal function. Careful monitoring of the volume status and judicious administration of diuretic therapy can reduce the risk of this complication.
 - d. **Massive transfusion**, defined as the transfusion of blood products that are greater in volume than a patient's normal blood volume in less than 24 hours, creates several risks not encountered with a lesser volume or rate of transfusion.
 1. **Coagulopathy** might arise as a result of platelet and coagulation factor depletion. Transfusion of platelets, FFP, or cryoprecipitate should not be performed empirically but instead should be based on the clinical situation and laboratory values.
 2. **Hypothermia** can result from massive volume resuscitation with chilled blood products but can be prevented by using blood warmers. Hypothermia can precipitate cardiac dysrhythmias and coagulopathy.
 3. **Citrate toxicity** can develop after massive transfusion in patients with hepatic dysfunction. Hypocalcemia can be treated with 10 mL 10% calcium gluconate i.v.
 4. **Electrolyte abnormalities**, including acidosis and hyperkalemia, occur rarely after massive transfusions, especially in patients with preexisting hyperkalemia.
5. **Alternatives to homologous transfusion** exist and may provide advantages in safety and cost when used in elective procedures with a high likelihood for significant blood loss. However, it must be stressed that meticulous surgical technique can in itself provide for significant reductions in transfusion requirements.
 - a. **Autologous predonation** is the preferred alternative for elective transfusions and has become standard practice for some high-risk procedures. Up to 20% of patients still require allogeneic transfusion, however, and transfusion reactions may result from clerical errors in storage. Despite its intrinsic advantages, predonation is not cost effective when the risk of transfusion is moderate or low.
 - b. **Isovolemic hemodilution** is a technique in which whole fresh blood is removed with simultaneous infusion of crystalloid in the immediate preoperative period. The blood is stored at room temperature and reinfused after acute blood loss has ceased. Moderate hemodilution (hematocrit 32–33%) is as effective as autologous predonation in reducing the need for allogeneic transfusion but is much less costly.
 - c. **Intraoperative autotransfusion**, wherein blood from the operative field is returned to the patient, can decrease allogeneic transfusion requirements. Equipment to separate and wash recovered RBCs is required in most cases, and contraindications include neoplasm and enteric or purulent contamination.
 - d. **Erythropoietin** may be effective in decreasing allogeneic transfusion requirements when given preoperatively. Appropriate dosages (1,000–3,500 units/kg) can be calculated based on anticipated transfusion requirement and are administered over 2–4 weeks. Adjunctive use with autologous predonation has not consistently been shown to be effective.

B. Platelet transfusion

1. **Indications.** Platelet transfusions are used to control bleeding that is caused by thrombocytopenia and to prevent spontaneous bleeding in situations of severe thrombocytopenia. For ongoing hemorrhage and before major operations, platelet counts greater than 100,000/μL should be obtained; however, platelet counts greater than 50,000/μL usually are sufficient for minor surgical procedures if the platelet function is normal. In patients with severe thrombocytopenia, platelet counts should be maintained above 10,000/μL to prevent spontaneous bleeding.
2. **Administration.** Random donor platelets are used routinely, but in patients who require long-term platelet replacement and those who are refractory to platelet transfusions, single-donor transfusions may be more effective in increasing the platelet count. Generally, a “six pack” of random donor platelets or 1 unit of single-donor platelets increases the platelet count by 30,000–60,000/μL.
3. **Complications specific to platelet transfusions**

- a. **Alloimmunization** occurs in 50–75% of patients receiving long-term platelet transfusions and presents as a failure of the platelet count to increase significantly after transfusion. Single-donor platelets might result in a better response than would random-donor platelet products. In patients who likely will need long-term platelet therapy, human leukocyte antigen–matched single-donor platelets slow the onset of alloimmunization.
- b. **Posttransfusion purpura** is a rare complication of platelet transfusions seen in previously transfused individuals and multiparous women. It is usually caused by recipient antibodies to platelet antigen PIA1. This condition presents with severe thrombocytopenia, purpura, and bleeding occurring 7–10 days after platelet transfusion. Fatal bleeding can occur. Plasmapheresis or immunoglobulin therapy raises platelet levels. Platelet transfusions are ineffective.
- C. **FFP** contains the clotting activity of all the coagulation factors. Therefore, it can be used to correct coagulopathies that are due to deficiencies of all coagulation factors and is particularly useful when multiple factor deficiencies exist (e.g., liver disease or massive transfusion). Dosage is 10–15 mL/kg, where each unit of FFP is 225 mL. However, deficiencies of factors VIII and IX are best treated with their respective factor concentrates. The activity of factors V and VIII diminishes with time if plasma is stored unfrozen.
- D. **Cryoprecipitate** is the cold precipitate of fresh plasma and contains approximately 80% of factor VIII and vWF as well as approximately 80% of plasma fibrinogen. This is particularly useful in replacing vWF in vWD and in correcting the fibrinogen deficiency in DIC or during massive transfusion. Typical replacement therapy consists of 1 unit/10 kg body weight.

III. Local hemostatic agents can aid in the intraoperative control of bleeding from needle holes, vascular suture lines, or areas of extensive tissue dissection. Anastomotic bleeding usually is best controlled with local pressure or a simple suture. These agents speed hemostasis by providing a matrix for thrombus formation.

- A. **Gelatin sponge** (e.g., Gelfoam) can absorb many times its weight of whole blood by capillary action and provides a platform for coagulation. Gelfoam itself is not intrinsically hemostatic. It resorbs in 4–6 weeks without significant inflammatory reaction.
- B. **Oxidized cellulose** (e.g., Surgicel) is a knitted fabric of cellulose that allows clotting by absorbing blood and swelling into a scaffold. Its slow resorption can create a foreign body reaction.
- C. **Collagen sponge** (e.g., Helistat) is produced from bovine tendon collagen and promotes platelet adhesion. It is only slowly resorbed and creates a foreign body reaction similar to that of cellulose.
- D. **Microfibrillar collagen** (e.g., Avitene, Hemotene) can be sprayed onto wounds and anastomoses for hemostasis, particularly in areas that are difficult to reach. It, too, stimulates platelet adhesion and promotes thrombus formation. Because microfibrillar collagen can pass through autotransfusion device filters, these adjuncts should be avoided during procedures that involve cell-saver devices.
- E. **Topical thrombin** can be applied to the various hemostatic agents or to dressings and placed onto bleeding sites to achieve a fibrin-rich hemostatic plug. Topical thrombin, usually of bovine origin, is supplied as a lyophilized powder and can be applied directly to dressings or dissolved in saline and sprayed onto the wound. Repeated use of bovine thrombin may result in formation of inhibitors to thrombin or factor V, which rarely is associated with a clinical bleeding disorder. Because all that is necessary for hemostasis is fibrinogen, topical thrombin can be used effectively in anticoagulated patients.
- F. **Gelatin matrices** (e.g., Floseal) are often used in combination with topical thrombin intraoperatively. Typically, bovine thrombin, 5,000 units, is sprayed onto the matrix, which is then applied to the site of bleeding.
- G. **Other agents** include topical cryoprecipitate, which can be sprayed onto the wound with topical thrombin for a fibrin-rich coagulum, as well as topical EACA and topical aprotinin. Topical EACA and aprotinin inhibit fibrinolysis.

6 Anesthesia

Douglas W. Green and Richard S. Hotchkiss

Preparing the Patient

I. Patient preparation for operation

Types of Anesthesia

I. Local anesthetics

II. Regional anesthesia

III. General anesthesia

Intubation and Sedation

I. Emergent intubation by rapid-sequence induction

II. Sedation for procedures

Postoperative Medication and Complications

I. Postoperative analgesia

II. Sleeping medications

Preparing the Patient

I. Patient preparation for operation

A. Preoperative evaluation

1. A **comprehensive preoperative evaluation** is critical to the safe administration of anesthetic care.
 - a. A **thorough history**, including medication usage and prior anesthetic usage, should be obtained.
 - b. An **examination of airway**, vascular access, and other pertinent anatomy tailored to the anticipated operation should be undertaken.
2. For **patients without preexisting disease**, preoperative screening and testing are determined primarily by age.
 - a. **Hemoglobin or hematocrit** may be the only test required in healthy patients younger than 40 years.
 - b. A **serum pregnancy test** should be obtained for female patients of childbearing age.
 - c. A **screening chest X-ray and electrocardiogram (ECG)** are obtained for patients who are 40 years or older, unless an indication is found from the history or physical examination, or both.
3. **Additional testing** may be required when clinically indicated.
 - a. **Serum electrolytes** must be evaluated in patients with diabetes or renal insufficiency and in patients who are taking diuretics.
 - b. **Coagulation studies** (prothrombin time, partial thromboplastin time, bleeding time) must be evaluated in patients who are receiving anticoagulation therapy or have a personal or family history that is suggestive of abnormal bleeding.
 - c. **Additional testing or consultation** may be required in patients with evidence of severe coexisting disease, especially those with cardiac, pulmonary, or renal compromise.

B. Nothing by mouth (n.p.o.) status

1. It is customary for patients to abstain from any oral intake except for medications with sips of water for 8 hours before elective surgery. However, the following are **aspiration prophylaxis regimens** for adult patients who are not considered to be at increased risk for aspiration of gastric contents.
 - a. **Solid food** is permitted until 6 hours before surgery.
 - b. **Clear liquids** (which do not include milk or juices containing pulp) are permitted until 2 hours before surgery (*Anesthesiology* 90:896–905, 1999).
2. **Patients with slowed or incomplete gastric emptying** (e.g., those who are morbidly obese, diabetic, or on narcotic therapy) may require longer fasting periods and additional pretreatment with metoclopramide, histamine H₂-receptor antagonists, or sodium citrate. Rapid-sequence induction can be considered in these patients. Maintenance intravenous fluids should be started in n.p.o. in-patients.

C. Medications

1. In-patients can receive **benzodiazepines or narcotics** to alleviate preoperative anxiety.
2. **Cardiovascular or other pertinent medications** usually are administered on the morning of surgery with small sips of water. Patients who normally receive scheduled insulin doses should instead be placed on sliding-scale insulin, with blood sugars checked every 6 hours while n.p.o.

Types of Anesthesia

I. Local anesthetics are categorized into two groups. **Esters** include tetracaine, procaine, cocaine, and chlorprocaine. **Amides** include lidocaine, mepivacaine, bupivacaine, and etidocaine. Characteristics of commonly used local anesthetic agents are summarized in [Table 6-1](#).

Table 6-1. Local anesthetics for infiltration

A. Mechanism of action

1. **Local anesthetics** work by diffusing through the nerve plasma membrane and causing blockade of sodium channels. The nerve cell is unable to depolarize, and axonic conduction is inhibited.
2. **Local tissue acidosis** (e.g., from infection) causes local anesthetic molecules to become positively charged and less able to diffuse into the neuron. This slows the onset and decreases the intensity of analgesia.

B. Toxicity (dose dependent, except for allergic reactions)

1. Central nervous system (CNS)

- a. **Signs and symptoms** include mental status changes, dizziness, perioral numbness, a metallic taste, tinnitus, visual disturbances, and seizures. Seizures resulting from inadvertent intravascular injection usually last only minutes. Continuous infusion of local anesthetics may result in high plasma levels and prolonged seizures.
- b. **Treatment** involves airway support and ventilation with 100% oxygen, which should always be available. Prolonged seizures may require administration of benzodiazepines (midazolam, 1–5 mg i.v.; diazepam, 5–15 mg i.v.; or lorazepam, 1–4 mg i.v.). Intubation may be required to ensure adequate ventilation.

2. Cardiovascular

- a. **Signs and symptoms** range from decreased cardiac output to hypotension and cardiovascular collapse. Most local anesthetics cause CNS toxicity before cardiovascular toxicity. Bupivacaine is an exception, and its intravascular injection can result in severe cardiac compromise.
- b. **Treatment** includes fluid resuscitation, administration of vasopressors, and cardiopulmonary resuscitation, if necessary (see [Chapter 3](#)).

3. Hypersensitivity reactions, although rare, have been described with ester-based local anesthetics and are attributed to the metabolite *p*-aminobenzoic acid. True amide-based local anesthetic reactions are questionable.

- a. **Signs and symptoms** can range from mild to life threatening. These include urticaria, bronchospasm, hypotension, and anaphylactic shock.
- b. **Treatment** is similar to that for hypersensitivity reactions from other etiologies. Urticaria responds to diphenhydramine, 25–50 mg i.v. Bronchospasm is treated with inhaled bronchodilators (e.g., albuterol) and oxygen. Hypotension is treated with fluid resuscitation and vasopressors [e.g., phenylephrine hydrochloride (Neo-Synephrine)] as required. Anaphylactic cardiovascular collapse can be treated with epinephrine, 0.5–1.0 mg i.v. bolus.

C. Epinephrine (1:200,000, 5 µg/mL) is mixed with local anesthetic solutions to prolong the duration of neural blockade and reduce systemic drug absorption. Its

use is contraindicated in areas where arterial spasm would lead to tissue necrosis (e.g., nose, ears, fingers, toes, or penis).

II. Regional anesthesia

A. In the operating room

1. General considerations

- The **importance of preoperative communication between anesthesiologist and surgeon** cannot be overemphasized. The extent and duration of the procedure must be appreciated by the anesthesiologist so that the appropriate area and duration of analgesia can be achieved. If the possibility of a prolonged or very involved operative procedure is likely, a general anesthetic may be more appropriate. Patient positioning for various surgical procedures is poorly tolerated by awake patients (e.g., steep Trendelenburg may cause respiratory compromise); in these instances, a general anesthetic is appropriate.
- Supplements to regional anesthesia.** No regional anesthetic technique is foolproof, and various degrees of failure may occur because of inexperience or adverse anatomy. Local infiltration by the surgeon may be required if there is an incomplete block or one that is slow to set up fully. Intravenous sedation using short-acting benzodiazepines, narcotics, barbiturates, or propofol can also be helpful. General anesthesia may be required when a regional technique provides inadequate analgesia.
- n.p.o. status.** Because any regional anesthetic may progress to a general anesthetic, n.p.o. requirements for regional and general anesthetics are identical (see Preparing the Patient, [section I.B.1](#) and [section I.B.2](#)).
- Monitoring requirements** are no different from those for general anesthesia. Heart rhythm, blood pressure (BP), and arterial oxygen saturation should be monitored regularly during regional or general anesthesia. Other monitoring may be indicated, depending on coexisting disease states.

2. Types of regional anesthesia

- Spinal anesthesia** involves the injection of small volumes of local anesthetic solution into the subarachnoid space at the level of the lumbar spine.
 - Anatomy and placement** ([Fig. 6-1](#))

Fig. 6-1. Anatomy for epidural and spinal anesthesia.

- Using sterile technique, and after local anesthetic infiltration of the skin and subcutaneous tissues, a small (22- to 27-gauge) **spinal needle is passed between two adjacent lumbar spinous processes**. The needle is passed through the following structures: supraspinous ligament, interspinous ligament, ligamentum flavum, dura mater, and arachnoid mater. Cerebrospinal fluid (CSF) is aspirated, and the appropriate local anesthetic solution is injected.
 - The needle can be removed (single-shot method), or a catheter can be placed to allow repeated dosing for potentially longer procedures (continuous spinal).
- Level of analgesia**
 - Multiple variables** affect the spread of analgesia. The **baricity** of the agent (solution density compared to that of CSF) and the position of the patient immediately after injection are major determinants of level. The **total dose injected** (increased dose results in higher spread) and the **total volume injected** (increased volume results in higher spread) are also important determinants of anesthetic level.
 - Older patients** tend to have greater spread of anesthesia by a few dermatomes. Such a difference may not be clinically significant.
 - Onset and duration of analgesia**
 - The **specific characteristics of the local anesthetic** used and the **total dose injected** are the primary determinants of onset and duration of action. Epinephrine added to the solution increases the duration of analgesia.
 - Variability in length of analgesia** is significant, ranging from as little as 30 minutes (lidocaine) to up to 6 hours (tetracaine with epinephrine).
 - Complications**
 - Hypotension** may occur as a result of sympatholytic-induced vasodilation and bradycardia. It may be more severe in hypovolemic patients or in those with preexisting cardiac dysfunction. Treatment includes volume resuscitation (crystalloid, 500–1,000 mL), vasopressors (epinephrine, 5–10 µg i.v. for adults; phenylephrine hydrochloride, 50–100 µg i.v.), and positive chronotropic drugs.
 - High spinal blockade.** Inadvertently high levels of spinal blockade may result in hypotension, dyspnea (loss of chest proprioception or intercostal muscle function), or apnea (decreased medullary perfusion secondary to hypotension). Respiratory dysfunction may necessitate intubation and ventilatory support.
 - Headache** after spinal anesthesia or diagnostic lumbar puncture is encountered with higher frequency in young or female patients. A postural component is always present (i.e., symptoms worsened by sitting up or standing). The recent use of smaller-gauge spinal needles has reduced the frequency of this complication. Treatment includes oral or intravenous fluids, oral analgesics, and caffeinated beverages. Severe refractory headache may require placement of an epidural blood patch.
 - CNS infection** after spinal anesthesia, although extremely rare, may result in meningitis, epidural abscess, or arachnoiditis.
 - Permanent nerve injury** is exceedingly rare and is seen with the same frequency as in general anesthesia.
 - Urinary retention with bladder overdistention** occurs in patients with spinal anesthesia whose bladders are not drained by urethral catheters. Catheters should remain in place until after the spinal anesthesia has been stopped and full sensation has returned.
 - Contraindications**
 - Absolute contraindications** to spinal anesthesia are localized infection at the planned puncture site, increased intracranial pressure, generalized sepsis, coagulopathy, and lack of consent.
 - Relative contraindications** include hypovolemia, preexisting CNS disease, chronic low back pain, platelet dysfunction, and aortic stenosis.
- Epidural anesthesia**
 - Anatomy and placement** ([Fig. 6-1](#))
 - Inserting an epidural needle is similar to placing a spinal needle except that the **epidural needle is not advanced through the dura**. No CSF is obtained. The tip of the epidural needle lies in the epidural space between the ligamentum flavum posteriorly and the dura mater anteriorly. Local anesthetic solution can then be injected.
 - The **needle is either removed** (single-shot method), or, more commonly, **a flexible catheter is passed through the needle** into the space and the needle is withdrawn over the catheter (continuous catheter technique). Local anesthetics or opiates can be dosed intermittently or infused as needed.
 - Level of analgesia**
 - Once injected into the epidural space, the local anesthetic solution diffuses through the dura and into the spinal nerve roots, resulting in a **bilateral dermatomal distribution of analgesia**.
 - The spread of nerve root blockade is primarily determined by the **volume** of injection and, to a lesser degree, by patient **position, age, and area of placement**.
 - Onset and duration of analgesia**
 - Epidural anesthesia develops more slowly than does spinal anesthesia**, because the local anesthetic solution must diffuse farther. The rate of onset of sympathetic blockade and hypotension also is slowed, enabling more precise titration of hemodynamic therapy compared to spinal anesthesia.
 - The **dosing interval** depends on the agent used.
 - Complications** are similar to those that are encountered with spinal anesthesia.
 - Spinal headache** may result from inadvertent perforation of the dura.
 - Epidural hematoma** is rare and usually occurs with coexisting coagulopathy. Emergent laminectomy may be required to decompress the spinal cord and avoid permanent neurologic injury.
 - Combined spinal and epidural anesthesia**
 - Anatomy and placement**
 - small-gauge spinal needle** is placed through an epidural needle once the epidural space has been located. The dura is punctured only by the spinal needle, and placement is verified by CSF withdrawal. Subarachnoid local anesthetics or narcotics can then be administered via the spinal needle.
 - The **spinal needle** is withdrawn after the initial dosing, and an epidural catheter is threaded into the epidural space through the existing epidural needle.

2. **Onset and duration.** This procedure combines the quick onset of spinal analgesia with the continuous dosing advantages of epidural analgesia.
 3. **Complications** are similar to those seen in spinal and epidural anesthesia.
 - d. **Comparison of spinal or epidural anesthesia with general anesthesia.** Although the incidence of thromboembolic complications and total blood loss is reduced in certain surgical procedures with spinal or epidural anesthesia, there is no evidence that long-term mortality is reduced compared to general anesthesia (*Br J Anaesth* 58:284, 1986).
 - e. **Brachial plexus blockade.** Injection of local anesthetic solution into the sheath surrounding the brachial plexus results in varying degrees of upper-extremity blockade. This technique is indicated for any procedure involving patients' shoulders, arms, or hands. The approach taken depends on the distribution of blockade desired.
 1. **Axillary block.** The needle is placed into the brachial plexus sheath from the axilla. Blockade above the patient's elbow is unreliable.
 2. **Supraclavicular blockade.** The needle is directed caudally from behind the posterior border of the inner one-third of the clavicle. This technique reliably blocks the entire upper extremity, sparing the patient's shoulder. The risk of pneumothorax is low.
 3. **Interscalene blockade** involves the cervical as well as brachial plexus and reliably blocks the patient's shoulder. Ulnar nerve block is unreliable. The high incidence of phrenic nerve block increases the risk of pulmonary complications in patients with chronic obstructive pulmonary disease and also serves as a contraindication to bilateral blockade.
 - f. **Cervical plexus blockade** is indicated primarily for carotid endarterectomy and is the anesthetic method of choice for this procedure at many institutions. Inadvertent blockade of neighboring structures does occur.
 1. **Phrenic nerve blockade** may result in transient diaphragmatic paralysis. Simultaneous bilateral cervical plexus blockade is therefore contraindicated.
 2. **Ipsilateral cervical sympathetic plexus blockade** may result in Horner's syndrome, producing transient ptosis, miosis, and facial anhidrosis.
- B. Outside the operating room**
1. **Intercostal nerve block** is indicated after thoracotomy or before chest tube placement.
 - a. **Anatomy and placement** ([Fig. 6-2](#))

Fig. 6-2. Anatomy and placement for intercostal nerve block. **A:** The anesthesiologist's hand closest to the patient's head (cephalic) first locates the target interspace and then **(B)** retracts the skin over the rib above. **C:** The hand closest to the patient's feet (caudad) places the needle and attached syringe containing local anesthetic through the skin onto the rib at approximately a 30-degree angle, with the needle bevel directed cephalad. **D:** The cephalic hand then grasps the needle while maintaining contact with the patient, and allows the tension of the retracted skin to walk the needle off the inferior edge of the rib and advance 2–3 mm.

1. The **posterior axillary line is identified** and, using sterile technique, a 23-gauge needle is placed perpendicular to the patient's skin until contact is made with his or her rib. The needle is then walked caudad off the patient's rib and advanced several millimeters. After negative aspiration, 5 mL of bupivacaine 0.25–0.50% with epinephrine (1:200,000) is injected.
2. Usually, **five interspaces** (including 2 above and 2 below the interspace of interest) are injected.
- b. **Complications** include pneumothorax and intravascular injection causing arrhythmias. Injection into the nerve sheath with retrograde spread back to the spinal cord can produce a high spinal or epidural block (see [section II.A.2.a.\(4\)\(b\)](#)).
2. **Digital block** is indicated for minor procedures of the fingers.
 - a. **Anatomy and placement**
 1. From the **dorsal surface of the hand**, a 23-gauge needle is placed on either side of the metatarsal head and inserted until the increased resistance of the palmar connective tissue is felt. An injection of 1–2 mL of lidocaine 1–2% is made as the needle is withdrawn.
 2. **Supplemental injection** of 0.5–1.0 mL of lidocaine 1–2% in the interdigital web on either side may be required.
 - b. **Epinephrine is contraindicated.**
 - c. **Local infiltration**
 1. **In the operating room**, the area of incision can be infiltrated before incision or at the conclusion of the operation. Some evidence suggests that infiltration before incision is associated with less postoperative discomfort and reduced analgesic use (*Anesth Analg* 74:495, 1992). Bupivacaine is frequently used.
 2. **Outside the operating room**, local anesthetic infiltration may also be useful during wound débridement, central venous catheter placement, or repair of minor lacerations. The agent of choice is lidocaine 1–2% due to its quick onset and low toxicity. The area of interest should be injected liberally. Frequent aspiration helps to avoid intravascular injection. Injection should be repeated as necessary.
 3. **Epinephrine** should not be used in areas at risk of vascular compromise from arterial spasm (e.g., nose, ears, fingers, toes, or penis).

III. General anesthesia provides hypnosis (unconsciousness), analgesia, amnesia, and skeletal muscle relaxation.

- A. All patients who are undergoing general anesthesia require an appropriate **preoperative evaluation** and optimization of any coexisting medical problems (see *Preparing the Patient*, [section I.A.1](#) and [section I.A.2](#)).
- B. **Monitoring.** Basic monitoring requirements for general anesthesia are similar to those for regional anesthesia (see [section II.A.1.d](#)).
- C. **Induction of general anesthesia.** Intravenous agents are most widely used owing to rapid onset and ease of administration.
 1. **Thiopental**, a barbiturate (3–5 mg/kg i.v.), has a rapid onset and redistribution. Often, there is an associated decrease in cardiac output and BP.
 2. **Propofol**, a phenol derivative (1–3 mg/kg i.v.), is used for induction and maintenance of anesthesia. This agent has hemodynamic properties that are similar to those of thiopental but is associated with a low incidence of postoperative nausea and vomiting.
 3. **Etomidate**, an imidazole derivative (0.3 mg/kg i.v.), has only mild direct hemodynamic depressant effects.
 4. **Ketamine**, a phencyclidine derivative (1–4 mg/kg i.v.), increases cardiac output and BP in patients who are not catecholamine depleted. Ketamine raises intracranial pressure and is not used in patients with head trauma. The **use of ketamine is limited** owing to the emergence of delirium and nightmares.
- D. **Airway management.** Ventilation during general anesthesia may be spontaneous, assisted, or controlled.
 1. **Mask ventilation** with spontaneous respiratory effort can be used during limited (usually peripheral) procedures that do not require neuromuscular relaxation. Because the airway is unprotected, this technique is contraindicated in patients at risk for aspiration.
 2. **Endotracheal intubation** secures the airway, allows control of ventilation, and protects against aspiration. Although frequently performed orally with the laryngoscope, intubation can also be accomplished nasally and, in anatomically challenging patients, can be performed with the aid of a fiberoptic bronchoscope via oral or nasal routes.
- E. **Neuromuscular blockade** facilitates tracheal intubation and is required for many surgical procedures. It provides the surgeon with improved working conditions and optimizes ventilatory support. Agents that produce neuromuscular blockade act on postsynaptic receptors in the neuromuscular junction to antagonize the effects of acetylcholine competitively. Agents are categorized as either depolarizing or nondepolarizing ([Table 6-2](#)).

Table 6-2. Agents producing neuromuscular blockade

1. **Succinylcholine** is a rapid-acting (60 seconds), rapidly metabolized depolarizing agent that allows return of neuromuscular function in 5–10 minutes. This agent causes a transient mild hyperkalemia that may be exaggerated in patients with severe burns, trauma, or paralysis; patients on prolonged bed rest; or patients with other neuromuscular disorders. In addition, it can cause increases in intraocular, intracranial, and gastric pressures.
2. **Nondepolarizing muscle relaxants** can be divided into short-, intermediate-, and long-acting agents. Associated hemodynamic effects and elimination pathways vary.
 - a. **These agents are often used in an intensive care setting** when paralysis is necessary for adequate ventilation of an intubated patient. Such patients must have adequate sedation and analgesia before and during paralysis. Dosage should be monitored by train-of-four stimulus every 4 hours, with one-quarter twitches the goal. Corticosteroids and aminoglycosides should be avoided to reduce the risk of myopathy.
 - b. **Reversal of neuromuscular blockade** for patients who are receiving nondepolarizing muscle relaxants usually is performed before extubation to ensure full return of respiratory muscle function and protective airway reflexes. The diaphragm is less sensitive to muscle relaxants than are the muscles of the

head and neck; a spontaneously ventilating patient may be unable to protect the airway. The definitive test to assess the degree of remaining paralysis is to have the patient raise the head from the bed for 5 seconds or more. **Anticholinesterases** (neostigmine, 0.06–0.07 mg/kg, and edrophonium, 0.1 mg/kg) act to increase the availability of acetylcholine at the neuromuscular junction. The binding frequency of the nondepolarizing muscle relaxant (a competitive acetylcholine antagonist) is reduced, and the blockade is reversed.

F. Maintenance of anesthesia

1. The **goal of anesthesia** is to provide unconsciousness, amnesia, analgesia, and, usually, muscle relaxation. Balanced anesthesia involves the combined use of inhalational agents, narcotics, and muscle relaxants to attain this goal.
2. **Inhalational agents**
 - a. All inhalational agents provide varying degrees of unconsciousness, amnesia, analgesia, and muscle relaxation.
 - b. **Isoflurane** is the most commonly used inhalational agent due to its low rate of metabolism. Enflurane, halothane, sevoflurane, and desflurane are also used.
 - c. **Halothane and nitrous oxide** are used extensively as induction agents in pediatric patients owing to the decreased irritating effects of halothane on the airway. Halothane sensitizes the myocardium to catecholamines; thus, another inhalational agent should be used if epinephrine solutions have been injected into the surgical field. Sevoflurane is also used in children because, like halothane, it causes minimal irritation of the airways.
 - d. **Nitrous oxide** by itself cannot provide surgical anesthesia. When combined with other inhalational agents, it reduces the required dose and subsequent side effects of the other agents. Nitrous oxide is extremely soluble and readily diffuses into any closed gas space, increasing its pressure. As a result, this agent should not be administered to patients with intestinal obstruction or suspected pneumothorax.
3. **Intravenous agents**
 - a. **Narcotics** can be administered continuously or intermittently. These agents provide superior analgesia but unreliable amnesia. Commonly used narcotics include fentanyl, sufentanil, alfentanil, remifentanyl, morphine, and meperidine.
 - b. **Hypnotics, benzodiazepines, and propofol**. Propofol infusion provides excellent hypnosis (unconsciousness) but insignificant analgesia and unreliable amnesia. Its rapid dissipation of effects and low incidence of postoperative nausea have contributed to its widespread use in outpatient surgery. The maintenance dose is 0.1–0.2 mg/kg per minute.
 - c. **Ketamine** by itself can provide total anesthesia. The associated emergence of delirium and nightmares limits its use.

G. Recovery from general anesthesia. The goal at the conclusion of surgery is to provide a smooth, rapid return to consciousness with stable hemodynamics and pulmonary function, protective airway reflexes, and continued analgesia.

1. **Preparation for emergence from anesthesia** usually begins before surgical closure, and communication between the surgeon and anesthesiologist facilitates prompt emergence of the patient at the procedure's termination.
2. **Laryngospasm** is a potentially life-threatening complication that may occur after extubation (see [section III.H.2](#)).
3. Patients recover from the effects of sedation or general or regional anesthesia in the **postanesthesia care unit**. Once they are oriented, comfortable, hemodynamically stable, ventilating adequately, and without signs of anesthetic or surgical complications, patients are discharged to the appropriate ward or to home.

H. Complications of general anesthesia

1. **Malignant hyperthermia** is a hypermetabolic disorder of skeletal muscle characterized by intracellular hypercalcemia and rapid adenosine triphosphate consumption. This condition is initiated by exposure to one or more anesthetic triggering agents, including desflurane, enflurane, halothane, isoflurane, sevoflurane, and succinylcholine.
 - a. **Signs and symptoms may occur in the operating room or more than 24 hours postoperatively** and include tachycardia, tachypnea, hypertension, hypercapnia, hyperthermia, acidosis, and skeletal muscle rigidity.
 - b. **Treatment** involves immediate administration of dantrolene (1 mg/kg i.v. up to a cumulative dose of 10 mg/kg), which attenuates the rise in intracellular calcium. Repeat doses are given as needed if symptoms persist. Each vial contains 20 mg dantrolene and 3 g mannitol and must be mixed with 50 mL sterile water. Intensive care monitoring for 48–72 hours is indicated after an acute episode of malignant hyperthermia to evaluate for recurrence, acute tubular necrosis, and disseminated intravascular coagulation.
2. **Laryngospasm**
 - a. **During emergence from anesthesia**, noxious stimulation of the vocal cords can occur at light planes of anesthesia. Additionally, blood or other oral secretions can irritate the larynx. As a result, the vocal cords may be brought into forceful apposition, and the flow of gas through the larynx is restricted or prevented completely.
 - b. **Treatment** involves the use of positive-pressure ventilation by mask to break the spasm. Such therapy usually is sufficient. Succinylcholine may be required in refractory cases to allow successful ventilation.
3. **Nausea and vomiting**
 - a. **Cortical** (pain, hypotension, hypoxia), **visceral** (gastric distention, visceral traction), **vestibular**, and **chemoreceptor trigger zone** (narcotics) afferent stimuli all can play a role in postoperative nausea and vomiting. Overall incidence is approximately 30%. It is more common in preadolescents 11–14 years old, women, and obese patients. Narcotics, etomidate, and isoflurane have been implicated.
 - b. **Treatment** includes avoiding gastric distention during ventilation as well as administering prochlorperazine (Compazine), an antidopaminergic agent, 10 mg i.v. or p.o. every 4–6 hours as needed. For severe cases of postoperative nausea and vomiting, ondansetron (Zofran), 4 mg i.v., can be given. The dosing can be repeated every 6–8 hours if symptoms persist.
4. **Urinary retention**
 - a. Although very common with spinal anesthesia [see [section II.A.2.a.\(4\)\(f\)](#)], **urinary retention occurs in only 1–3% of cases involving general anesthesia**. It most commonly occurs after pelvic operations and in the setting of benign prostatic hypertrophy.
 - b. **Treatment** ranges from conservative (early ambulation, having patient sit or stand while attempting to micturate) to aggressive intervention (bladder catheterization).
5. **Hypothermia**
 - a. General anesthesia induction causes **peripheral vasodilation**, which leads to internal redistribution of heat, resulting in an increase in peripheral temperature at the expense of the core temperature. Core temperature then decreases in a linear manner until a plateau is reached. Such hypothermia is more pronounced in the elderly.
 - b. **Treatment** includes passive warming during an operation by insulation of all exposed surfaces. Additionally, active warming with forced-air convective warmers is effective, but care should be taken in patients with vascular insufficiency (warmers should not be used on unperfused extremities) and to prevent thermal injury.
6. **Nerve injury**
 - a. **Nerve palsies** can occur secondary to improper positioning of the patient on the operating table or insufficient padding of dependent regions. Such palsies can be long-lasting and debilitating.
 - b. **Prophylactic padding** of sensitive regions and attention to **proper positioning** remain the most effective therapies.

Intubation and Sedation

I. Emergent intubation by rapid-sequence induction

- A. **Patients in respiratory distress** outside the operating room may require intubation to ensure adequate oxygenation and ventilatory support. Whenever possible, an anesthesiologist should be alerted and present at the time of intubation to assist if necessary; however, intubation should not be unduly delayed while waiting for an anesthesiologist to arrive.
- B. **Airway support with 100% oxygen mask ventilation should be initiated before intubation**. In the emergent setting or with the hemodynamically unstable patient in whom paralysis is necessary to intubate, rapid-sequence induction of anesthesia with etomidate followed by succinylcholine is preferred. Intubation can then be performed via laryngoscopy using a size 8 tube for men and size 7 tube for women. After inflation of the cuff, bilateral and equal breath sounds should be auscultated, end-tidal CO₂ and pulse oximetry measured, and a portable chest X-ray ordered to ensure proper placement. The patient should be continued on 100% oxygen until being transferred to an intensive care setting.

II. Sedation for procedures

A. Monitored anesthesia care

1. In monitored anesthesia care or local standby cases, **an anesthesiologist is present** to monitor and sedate the patient during the procedure. The surgeon is responsible for analgesia, which is accomplished with local infiltration or peripheral nerve blockade. Sedating or hypnotic medications (e.g., propofol) provide sedation only and, when given in the presence of inadequate analgesia, may result in a disinhibited, uncooperative patient.
2. **Monitoring is identical to that required for general or regional anesthesia**. Supplemental oxygen is provided by face mask or nasal cannula.

3. **n.p.o. criteria** are identical to those for general or regional anesthesia.
 4. **Considerable variation exists** regarding the response of patients to sedating medications, and protective airway reflexes may be diminished with even small doses.
- B. Local procedures in the operating room**
1. *Local* implies that **an anesthesiologist is not required** to monitor the patient or provide sedation. It still is advisable for the physician performing the procedure to monitor the ECG, arterial oxygen saturation, and BP even if sedation is not given.
 2. **Painful stimuli** can increase vagal tone, resulting in bradycardia, hypotension, and hypoventilation.
- C. Sedation outside the operating room**
1. **Indications** are to relieve patient anxiety and avoid potentially detrimental hemodynamic sequelae during invasive procedures or diagnostic tests.
 2. **Oxygen** should be supplied by nasal cannula or face mask when sedation is given. When benzodiazepines and narcotics are combined, even healthy patients breathing room air may become hypoxic.
 3. **Monitoring** should include pulse oximetry, continuous ECG, and BP.
 4. **Agents.** All medications should be titrated, with adequate time between doses to judge clinical effects. The end result should be a calm, easily arousable, cooperative patient. Oversedation may result in hypoventilation, airway obstruction, or disinhibition. Dosages of commonly used sedatives are summarized in [Table 6-3](#).

Table 6-3. Medications for short-term sedation and analgesia during procedures

5. **Side effects** that result from benzodiazepine administration include oversedation, respiratory depression, and depressed airway reflexes. Flumazenil (Romazicon), a benzodiazepine antagonist, can be used to reverse such effects. A dose of 0.2 mg i.v. should be administered and repeated every 60 seconds as required to a total dose of 1 mg. It can produce seizures and cardiac arrhythmias, and re sedation can occur after 30–60 minutes.

Postoperative Medication and Complications

I. Postoperative analgesia is provided to minimize patient discomfort and anxiety, attenuate the physiologic stress response to pain, enable optimal pulmonary toilet, and enable early ambulation. Analgesics can be administered by the oral, intravenous, or epidural route ([Table 6-4](#)).

Table 6-4. Adult dosages of commonly used oral analgesics

- A. Intravenous route.** Many patients are unable to tolerate oral medications in the immediate postoperative period. For these patients, narcotics can be administered i.v. by several mechanisms.
1. **As needed (p.r.n.)**
 - a. **Narcotics**
 1. The **intermittent administration of intravenous or intramuscular narcotics by nursing staff** has the potential disadvantages of being given too infrequent, too late, and in insufficient amounts to provide adequate pain control. This may be the only choice in patients who are functionally unable to operate a patient-controlled analgesia (PCA) device.
 2. **Morphine**, 2–4 mg i.v. every 30–60 minutes, or **meperidine**, 50–100 mg i.v. every 30–60 minutes, should provide adequate analgesia for most patients. Orders should be written to withhold further injections for a respiratory rate of less than 12 breaths per minute or in cases of oversedation.
 - b. **Nonsteroidal antiinflammatory drugs (NSAIDs)**
 1. **Ketorolac is an NSAID available in oral and in injectable forms** that is an effective adjunct to opioid therapy. The usual adult dose is 30 mg i.m. followed by 15–30 mg every 6 hours.
 2. **Ketorolac shares the potential side effects of other NSAIDs** and should be used cautiously in the elderly and in patients with a history of peptic ulcer disease, renal insufficiency, steroid use, or volume depletion.
 2. **PCA**
 - a. With PCA, the patient has the ability to self-deliver analgesics within **preset safety parameters**.
 - b. Patients initially receive either **morphine** (100 mg in 100 mL, with each dose delivering 1 mg), **hydromorphone** (50 mg in 100 mL, with each dose delivering 0.25 mg), or **meperidine** (1,000 mg in 100 mL, with each dose delivering 20 mg), with a maximum of one dose every 10 minutes. If this treatment provides inadequate pain control, the concentration of the drug meperidine can be increased and the lockout time period can be changed.
 3. **Continuous “basal” narcotic infusions** are used rarely to treat patients who require sustained high serum narcotic concentrations. Continuous infusions should be used with great caution and only in patients with adequate monitoring and supervision to prevent respiratory depression and oversedation.
- B. Epidural infusions** are useful to treat postoperative pain caused by thoracotomy, extensive abdominal incisions, or orthopedic lower-extremity procedures. Narcotics, local anesthetics, or a mixture of the two can be infused continuously through catheters placed in the patient's lumbar or thoracic epidural space.
- C. Oral agents.** Dosages of commonly used oral analgesics are summarized in [Table 6-4](#).
- D. Side effects and complications**
1. **Oversedation and respiratory depression**
 - a. **Arousable, spontaneously breathing patients** should be given supplemental oxygen and monitored closely for signs of respiratory depression until mental status improves. Medications for pain or sedation should be decreased accordingly.
 - b. **Unarousable but spontaneously breathing patients** should be treated with oxygen and naloxone (Narcan). One vial of naloxone (0.4 mg) should be diluted in a 10-mL syringe, and 1 mL (0.04 mg) should be administered every 30–60 seconds until the patient is arousable. Adequate ventilation should be confirmed by arterial blood gas measurement. Current opioid administration should be stopped and the regimen decreased. In addition to continuous pulse oximetry, the patient should be monitored closely for potential re sedation as the effects of naloxone dissipate. Naloxone must be used carefully in patients with a history of coronary artery disease.
 2. **Apnea**
 - a. **Treatment** involves immediate intubation and ventilation.
 - b. **Naloxone**, 0.2–0.4 mg i.v., should be given immediately.
 3. **Hypotension and bradycardia**
 - a. **Local anesthetics administered via lumbar epidurals** decrease sympathetic tone to the abdominal viscera and lower extremities and greatly increase venous capacitance. Thoracic epidurals can additionally block the cardioaccelerator fibers, resulting in bradycardia.
 - b. The **treatment** of choice for any of these situations (excluding bradycardia) is volume resuscitation. Epinephrine can be used to raise BP acutely; 10 mg is diluted in 100 mL and given 1 mL i.v. at a time. If needed, this mixture can be infused i.v. starting at 15 mL per hour (25 µg/min). Bradycardia can be treated with atropine, 0.4–1.0 mg i.v., or glycopyrrolate given i.v. in 0.2-mg increments every 3–5 minutes as needed.
 4. **Nausea and vomiting**
 - a. **Naloxone** in small doses (0.04–0.1 mg i.v. p.r.n.) is helpful.
 - b. **Metoclopramide** (10 mg i.v. every 6 hours) is also useful.
 5. **Pruritus**
 - a. **Naloxone**, 0.04–0.1 mg i.v., is effective.
 - b. **Diphenhydramine**, 25–50 mg i.v. p.r.n., may provide symptomatic relief.
 6. **Monoamine oxidase inhibitors** (e.g., isocarboxazid, phenelzine) may, through unknown mechanisms, **interact adversely with narcotics**, resulting in severe hemodynamic swings, respiratory depression, seizures, diaphoresis, hyperthermia, and coma. Meperidine has been most frequently implicated and

should be avoided. Although morphine and fentanyl are believed to be safe, narcotics should be avoided whenever possible.

II. Sleeping medications

- A. **Insomnia** is common during hospitalization, and treatment should be readily available. Medications for insomnia taken before admission should be continued.
- B. **Diphenhydramine (Benadryl)** provides an alternative to benzodiazepines. Patients with severe chronic obstructive pulmonary disease or pulmonary hypertension from another etiology should be treated initially with this drug, 25–50 mg p.o., to avoid potential benzodiazepine-induced respiratory depression and hypercapnia. Elderly or debilitated patients also should receive diphenhydramine initially.
- C. **Zolpidem tartrate (Ambien)** is an hypnotic that is used for short-term treatment of insomnia. It is structurally dissimilar to benzodiazepines yet has much or all of its actions explained by its effects on benzodiazepine receptors. It impairs cognitive and motor performance to a greater degree in the elderly and should therefore be closely monitored in this age group.
- D. **Benzodiazepines** (rarely used) include flurazepam, temazepam, and triazolam. Temazepam and triazolam have less “hangover” effects because of a shorter half-life. Starting doses are as follows: flurazepam, 15–30 mg p.o.; temazepam, 15–30 mg p.o.; and triazolam, 0.125–0.25 mg p.o.

7 Perioperative Medical Care

Steven Hunt and Bradley D. Freeman

Care of the Surgical Patient

I. Preoperative evaluation and management

II. Postoperative care of the patient.

III. Complications

Care of the Surgical Patient

I. Preoperative evaluation and management

- A. **General evaluation of the surgical patient.** Preexisting medical conditions are common in the surgical population. The goal of preoperative evaluation is to identify and, if possible, modify risk factors that might influence surgical treatment adversely.
1. **History.** Key elements of the history should include preexisting medical conditions and family history known to increase operative risk, such as myocardial infarction (MI) and diabetes mellitus. Prior operations and any operative complications should be noted. Patients should be questioned carefully on their use of tobacco, alcohol, drugs, or other personal habits that increase operative risk. Review of systems should screen for cardiac, pulmonary, neurologic, endocrine, peripheral vascular, infectious, renal disease, deep venous thrombosis (DVT), pulmonary embolism (PE), and occult bleeding disorders.
 2. **Physical examination.** Physical examination includes measurement of vital signs; assessment of the patient's head and neck; and lung, cardiac, abdominal, neurologic, and peripheral vascular examination.
 3. **Routine diagnostic testing.** Minor surgical procedures and procedures on young, healthy patients often require minimal or no diagnostic testing. [Table 7-1](#) lists routine preoperative diagnostic tests and justifications for their use. Inclusion or exclusion of these tests should be considered on a case-by-case basis.

Table 7-1. Routine preoperative testing

4. **Preoperative medications.** In general, patients should continue their medications in the immediate preoperative period. Exceptions to this rule include diabetic medications (see [section I.B.6](#)), anticoagulants (see [section I.B.9.a](#)), and antiplatelet agents.
- B. **Specific considerations in preoperative management**
1. **Cerebrovascular disease.** Perioperative stroke is an uncommon surgical complication, occurring in fewer than 1% of general patients and in 2–5% of cardiac surgical patients. The majority (>80%) of these events are postoperative, and they most often are caused by hypotension or cardiogenic emboli during atrial fibrillation. Acute surgical stress might cause focal signs from a previous stroke to recur, mimicking acute ischemia.
 - a. **Risk factors for perioperative stroke** include age, hypertension, coronary artery disease (CAD), diabetes, and tobacco use. Known or suspected cerebrovascular disease requires special consideration.
 1. **The asymptomatic carotid bruit** is relatively common, occurring in approximately 14% of surgical patients who are older than age 55 years. However, fewer than 50% of bruits reflect hemodynamically significant disease. Although no increase in risk of stroke has been demonstrated during noncardiac surgery in the presence of an asymptomatic bruit, it is reasonable to evaluate such patients with carotid duplex before major general surgical procedures.
 2. **Patients with recent transient ischemic attacks** are at increased risk for perioperative stroke and should have preoperative neurologic evaluation (e.g., head CT, echocardiography, carotid Doppler). Patients with symptomatic carotid artery stenosis should have an endarterectomy before elective general surgery. The timing of carotid endarterectomy in the setting of cardiac surgery remains controversial.
 3. **Elective surgery for patients with recent cerebrovascular accident** should be delayed for a minimum of 2 weeks, ideally for 6 weeks, depending on the severity of the event.
 2. **Cardiovascular disease.** Death from cardiac causes is one of the leading causes of death after noncardiac surgery; therefore, risk stratification by the operating surgeon, anesthesiologist, and consulting internist is important for identifying patients at low, medium, and high risk for adverse cardiac outcomes.
 - a. **Risk factors.** The following risk factors have been associated with perioperative cardiac morbidity:
 1. The **patient's age** (>70 years) has been identified as an independent multivariate risk factor for cardiac morbidity in many studies.
 2. **Stable angina** has not been identified consistently as a multivariate risk factor for adverse cardiac outcome. Nevertheless, because angina is associated strongly with hemodynamically significant (>90%) coronary stenoses, patients with stable angina usually should undergo further diagnostic evaluation.
 3. **Unstable angina** is defined as chest pain that does not correlate with the level of physical activity and, therefore, occurs at rest or with minimal physical exertion. It typically implies a more serious degree of CAD. Elective operation in patients with unstable angina is contraindicated and should be postponed pending further evaluation.
 4. A **recent MI** is a well-defined risk factor for cardiac morbidity. The risk of reinfarction is significant if an operation is performed within 6 months of an MI (11–16% at 3–6 months). This risk still is increased substantially after 6 months, in contrast to patients without a history of MI (4–5% vs. 0.13%).
 5. **Untreated CHF** is a predictor of perioperative cardiac morbidity. Consequently, these patients should be optimized before any operative procedures are performed.
 6. **Diabetes mellitus** is now thought to confer additional independent risk for an adverse cardiac outcome. In particular, patients with diabetes as well as peripheral vascular disease might warrant dipyridamole thallium scanning regardless of other risk factors.
 7. **Valvular heart disease.** Aortic stenosis is a significant risk factor and may confer a 14-fold increase in relative risk independent of the manifestations of CHF. Consequently, any uncharacterized systolic ejection murmur deserves further evaluation with echocardiography. Other valvular heart diseases have not been evaluated systematically as risk factors for adverse cardiac outcome. However, patients with unexplained symptoms of dyspnea on exertion, shortness of breath, chest pain, or syncope should undergo further diagnostic evaluation before elective operation. All patients with valvular heart disease, even hemodynamically insignificant disease (excluding patients with mitral valve prolapse alone without a murmur), should receive prophylactic antibiotics before any operation that can introduce bacteria into the bloodstream or any dental procedure to reduce the risk of infective endocarditis ([Table 7-2](#)).

Table 7-2. Antibiotic prophylaxis of bacterial endocarditis for adult patients

8. **Peripheral vascular disease.** The perioperative cardiac risk for patients with peripheral vascular disease undergoing nonvascular surgery is not known. Because of the high coexistence of CAD with peripheral vascular disease, a lower threshold for obtaining diagnostic testing is warranted.
 9. **Type of procedure.** Patients who are undergoing thoracic surgery, vascular surgery, or upper abdominal surgery are at a higher risk of adverse cardiac outcome.
 10. **Functional impairment.** Patients with a poor functional capacity have a significantly higher risk of experiencing a postoperative cardiac event.
- b. **Revised Cardiac Risk Index.** This index stratifies patients into risk groups and helps to identify those in whom additional cardiac evaluation might be indicated ([Table 7-3](#)).

Table 7-3. Revised cardiac risk index*

- c. **Preoperative testing.** Patients at risk for a perioperative myocardial event require additional studies to minimize the occurrence of adverse outcomes.
 1. A **preoperative electrocardiogram (ECG)** is warranted in any patient with a history of angina or MI and in all men older than age 40 and all women older than age 50. Any ECG abnormalities that indicate a previous MI should be further evaluated by noninvasive testing.
 2. **Noninvasive testing.** Patients who are identified to be at risk of a perioperative cardiovascular event by the revised cardiac risk index or who have other risk factors (e.g., peripheral vascular disease, unexplained chest pain, diabetes, ECG abnormalities, etc.) should undergo further evaluation.
 - a. **Exercise stress testing** provides useful information for risk stratification. An inability to achieve even modest levels of exercise or the presence of ECG changes with exercise identifies patients at significant risk of adverse outcome.
 - b. **Dipyridamole thallium imaging** is the best-studied and most widely accepted imaging test for predicting adverse cardiac outcomes. This test is highly sensitive (>90%) and reasonably specific (>60%). It is most efficacious when used in conjunction with clinical risk stratification.
 - c. **Dobutamine stress echocardiography** is becoming accepted as an excellent predictor of cardiac outcome after noncardiac surgery. The test is easier to perform than thallium imaging and might be more appropriate for patients who are taking theophylline.
 3. **Invasive testing.** High-risk subgroups of patients based on clinical risk factors and individuals with positive noninvasive tests should undergo cardiac catheterization. Patients with significant cardiac lesions should have definitive treatment (angioplasty or coronary artery bypass grafting) before any elective general surgical procedures are performed.
 - d. **Preoperative management**
 1. **Patients with pacemakers** should have their pacemakers turned to the uninhibited mode (e.g., DDO) before surgery. In addition, a bipolar cautery should be used when possible in these patients. If it is necessary to use a unipolar cautery, the grounding pad should be placed away from the heart.
 2. **Patients with internal defibrillators** should have these devices turned off during surgery.
3. **Pulmonary disease.** Preexisting lung disease confers a dramatically increased risk of perioperative pulmonary complications.
 - a. **Preoperative evaluation and screening**
 1. **Risk factors**
 - a. **Chronic obstructive pulmonary disease (COPD)** is by far the most important risk factor, increasing pulmonary complications four- to fivefold. Patients with restrictive lung disease probably are at increased risk as well.
 - b. **Smoking**
 - c. **Increased age**
 - d. **Obesity**
 - e. **Type of surgery.** Pulmonary complications occur at a much higher rate for thoracic and upper abdominal procedures than for other procedures.
 - f. **Acute respiratory infections.** Postoperative pulmonary complications occur at a much higher rate for patients with acute respiratory infections; therefore, elective operations should be postponed in these individuals.
 - g. **Functional status.** In the patient with pulmonary disease or a history of smoking, a detailed evaluation of the patient's ability to climb stairs, walk a certain distance, or perform daily duties is vital to stratify risk.
 2. **Physical examination** should be performed carefully, with attention paid to signs of lung disease (e.g., wheezing, prolonged expiratory-inspiratory ratio, clubbing, or use of accessory muscles of respiration).
 3. **Laboratory evaluation**
 - a. **Chest X-ray (CXR)** is useful for identifying parenchymal or pleural abnormalities preoperatively and for providing a baseline for comparison with postoperative studies. It has no role as a routine screening examination for stratifying risk.
 - b. An **arterial blood gas (ABG)** is useful in patients with a history of lung disease or smoking to provide a baseline for comparison with postoperative studies. This test is not useful for routine screening.
 - c. **Preoperative pulmonary function testing** is of unproved benefit for patients who are undergoing nonthoracic operations. Nevertheless, certain high-risk patient groups can benefit from testing ([Table 7-4](#)). Based on the results of pulmonary function testing, patients can be stratified into risk groups ([Table 7-5](#)).

Table 7-4. Potential indications for preoperative pulmonary function tests

Table 7-5. Risk prediction in abdominal or nonresective thoracic surgery

- b. **Preoperative prophylaxis and management**
 1. **Pulmonary toilet.** Increasing functional residual capacity by the use of preoperative incentive spirometry is potentially effective in reducing pulmonary complications.
 2. **Antibiotics** do not reduce pulmonary infectious complications in the absence of preoperative infection. Elective operations should be postponed in patients with respiratory infections. If emergent surgery is required, patients with acute pulmonary infections can be treated with cefuroxime (1 g i.v. every 8 hours).
 3. **Cessation of smoking.** The return of mucociliary function usually requires cessation of smoking 2–4 weeks before surgery and should be urged in all patients undergoing elective surgery.
 4. **Bronchodilators.** In the patient with obstructive airway disease and evidence of a significant reactive component, bronchodilators might be required in the perioperative period. When possible, elective operation should be postponed in the patient who is wheezing actively.
4. **Renal disease**
 - a. **Preoperative evaluation of patients with existing renal insufficiency**
 1. **Risk factors**
 - a. **Underlying medical disease.** A substantial percentage of patients who require chronic hemodialysis for chronic renal insufficiency (CRI) have diabetes or hypertension. Furthermore, the incidence of CAD is substantially higher in these patients. Thus, much of the perioperative morbidity and mortality arises from these coexisting illnesses.
 - b. **Metabolic and physiologic derangements of CRI.** A variety of abnormalities in normal physiology that occur as a result of CRI can affect perioperative outcome adversely; they include alterations in electrolytes, acid-base balance, platelet function, the cardiovascular system, and the immune system. Specifically, the most common abnormalities in the perioperative period include hyperkalemia, intravascular volume overload, and infectious complications.
 - c. **Type of operative procedure.** Minor procedures under local or regional anesthesia are usually well tolerated in patients with CRI; however, major procedures are associated with an increased morbidity and mortality and include electrolyte abnormalities, bleeding, and infection.
 2. **Evaluation**
 - a. **History.** It is important to ascertain the specific etiology of CRI because patients with hypertension or diabetes and CRI are at a substantially increased risk of perioperative morbidity and mortality. The timing of last dialysis, the amount of fluid removed, and the preoperative weight provide important information about the patient's expected volume status.
 - b. **Physical examination** should be performed carefully to assess the volume status. Elevated jugular venous pulsations or crackles on lung examination can indicate intravascular volume overload.
 - c. **Diagnostic testing**

- i. **Laboratory data.** Serum sodium, potassium, calcium, phosphorus, magnesium, and bicarbonate levels should be measured, as well as blood urea nitrogen (BUN) and creatinine levels. A complete blood cell (CBC) count should be obtained to evaluate for significant anemia or a low platelet level. Determination of the bleeding time can be indicated in patients who are undergoing major operative procedures.
 - ii. **Supplemental tests,** including diagnostic cardiac evaluation (a dipyridamole thallium imaging test or dobutamine stress echocardiography), can be indicated regardless of the presence of other cardiac risk factors in patients with CRI.
3. **Management**
- a. **Timing of dialysis.** Dialysis should be performed within 24 hours of the planned operative procedure.
 - b. **Intravascular volume status.** CAD is the most common cause of death in patients with CRI. Consequently, because of the high incidence of coexisting CAD, patients with CRI undergoing major operations might require invasive monitoring in the intraoperative and postoperative period. Hypovolemia and volume overload are both tolerated poorly.
- b. **Patients at risk for perioperative renal dysfunction.** The reported incidence of acute renal failure (ARF) after operations in patients without preexisting CRI ranges from 1.5% to 2.5% for cardiac surgical procedures to more than 10% for patients who are undergoing repair of suprarenal abdominal aortic aneurysms.
- 1. **Risk factors** for the development of ARF include elevated preoperative BUN or creatinine, CHF, advancing age, intraoperative hypotension, sepsis, aortic cross-clamping, and intravascular volume contraction. Additional risk factors include administration of nephrotoxic drugs, such as aminoglycosides, and the administration of radiocontrast agents.
 - 2. **Preventive management**
 - a. **Intravascular volume expansion.** Adequate hydration is the single most important preventive measure for reducing the incidence of ARF because all mechanisms of renal failure are exacerbated by renal hypoperfusion caused by intravascular volume contraction.
 - b. **Radiocontrast dye administration.** Patients undergoing radiocontrast dye studies have an increased incidence of postoperative renal failure. Fluid administration (1–2 L isotonic saline) alone appears to confer protection against ARF. Additional measures to reduce the incidence of contrast dye–mediated ARF include the use of low-osmolality contrast agents.
 - c. **Other nephrotoxins,** including aminoglycoside antibiotics, nonsteroidal antiinflammatory drugs, and various anesthetic drugs, can predispose to renal failure and should be used judiciously in patients with other risk factors for the development of ARF.
5. **Prevention of infectious complications.** Infectious complications may arise in the surgical wound itself or in other organ systems. They may be initiated by changes in the physiologic state of the respiratory, genitourinary, or immune systems arising from the stress of surgery. It is impossible to overemphasize the importance of frequent hand washing by all health care workers to prevent the spread of infection.
- a. **Assessment of risk.** Risk factors for infectious complications after surgery can be grouped into risk factors arising from the type of procedure or patient-specific risk factors.
 - 1. **Surgical risk factors** include the type of procedure and degree of wound contamination ([Table 7-6](#)), the duration of operation, and the urgency of operation.

Table 7-6. Classification of surgical wounds

- 2. **Patient-specific risk factors** include age, diabetes, obesity, immunosuppression, malnutrition, preexisting infection, and chronic illness.
- b. **Prophylaxis**
- 1. **Surgical wound infection.** Antibiotic prophylaxis has contributed to a reduction in superficial wound infection rates (see [Table 7-7](#) for specific recommendations). Coverage should be initiated before the skin incision is made and, in the absence of gross contamination or overt infection, should not be administered beyond 24–48 hours after surgery. Repeat doses should be administered every 6–8 hours during the operation.

Table 7-7. Recommendations for antibiotic prophylaxis

- 2. **Respiratory infections.** Risk factors and measures to prevent pulmonary complications are discussed in [section I.B.3](#).
 - 3. **Genitourinary infections** may be caused by instrumentation of the urinary tract or an indwelling urinary catheter. Preventive measures include sterile insertion of the catheter and removal of the catheter as soon as possible postoperatively. A prophylactic dose of antibiotics (ciprofloxacin, 400 mg i.v., or cefazolin, 1 g i.v.) should be given after a difficult catheter insertion or excessive manipulation of the urinary tract.
6. **Diabetes mellitus.** Diabetic patients experience significant stress during the perioperative period and are at an estimated 50% increased risk of morbidity and mortality over nondiabetic patients. Diabetic patients experience more infectious complications and have impaired wound healing. Most importantly, vascular disease is common in diabetics, and silent CAD must always be considered. MI, often with an atypical presentation, is the leading cause of perioperative death among diabetic patients.
- a. **Preoperative evaluation** of diabetic patients must include an assessment of the chronic complications of diabetes and an estimation of control of blood sugars. Diabetic patients should be evaluated to exclude the existence of common comorbid conditions such as cardiovascular and renal disease. Preoperative assessment of glucose control is essential in all diabetic patients. In general, diabetic patients should be well controlled before elective surgery, with procedures scheduled early in the day. All diabetic patients should have their blood glucose checked on call to the operating room and during general anesthesia to prevent unrecognized hyper- or hypoglycemia.
 - 1. Patients with **diet-controlled diabetes mellitus** can be maintained safely without food or glucose infusion before surgery.
 - 2. **Patients who are taking oral hypoglycemic agents** should discontinue these medications the evening before scheduled surgery. Patients who take such long-acting agents as chlorpropamide or glyburide should discontinue these medications 2–3 days before surgery. These patients may require intravenous glucose when fasting or purified human insulin to manage glucose levels of greater than 250 mg/dL perioperatively.
 - 3. **Patients who normally take insulin** require insulin and glucose preoperatively to prevent ketosis and catabolism. Patients undergoing major surgery should receive one-half of their morning insulin dose and 5% dextrose i.v. at 100–125 mL per hour. Subsequent insulin administration by either subcutaneous sliding-scale or insulin infusion is guided by frequent (every 4–6 hours) blood glucose determinations. Subcutaneous insulin pumps should be inactivated the morning of surgery.
 - 4. For **poorly controlled diabetic patients with diabetic ketoacidosis (DKA)** who require emergency surgery, every attempt should be made to correct acidosis, electrolyte imbalance, hypokalemia, and volume depletion before operation. Control of blood glucose in these critically ill patients is best managed with insulin infusion (see [section III.G.1.e](#)).
7. **Thyroid disorders**
- a. **Preoperative evaluation.** Patients with known thyroid disorders should be evaluated for symptoms of hyper- and hypothyroidism. Thyroid function should be measured in those who are experiencing symptoms. In the majority of cases, measurement of thyroxine (T_4) and thyroid-stimulating hormone suffices for diagnosis. Conditions such as pregnancy and stress may change the level of circulating thyroid-binding globulin and, as a result, total T_4 concentration. In these cases, the free T_4 index is used.
 - b. **Hyperthyroidism.** Nonthyroid surgery in the treated hyperthyroid patient generally is well tolerated. The principal risk in confronting unrecognized or untreated hyperthyroid surgical patients is development of thyroid storm. Hyperthyroid patients also are at increased risk for perioperative cardiac arrhythmias (i.e., atrial fibrillation) and ischemia. Preoperative screening for cardiopulmonary disease is therefore essential.
 - 1. **Clinically euthyroid patients** who are treated medically with either propylthiouracil (PTU) or methimazole should take their medication the day of surgery, and these drugs should be resumed either orally or per nasogastric tube within 72 hours of surgery.
 - 2. **Untreated hyperthyroid patients** should have elective procedures postponed up to several months while medical therapy is undertaken. Thyrotoxic patients who require emergency surgery should be given 1 g PTU p.o., followed in 1 hour by sodium iodide, 500 mg/L i.v. fluid, given over 12 hours. Propranolol can be given (2–10 mg i.v. every 4 hours), except in asthmatics or in CHF, to control tachycardia, hypertension, and arrhythmias. Stress-dose steroids also should be administered. These agents should be continued postoperatively because the risk of thyroid storm remains high in the first 18 hours after surgery.
 - c. **Hypothyroidism:** surgery in hypothyroid patients. In general, the presence of medically controlled thyroid disease or even mild to moderate untreated

disease does not adversely affect the outcome of nonthyroid surgery. These patients may have an impaired febrile response to infection and require close observation for infectious complications. Fluid and electrolyte disorders, prolonged ileus, and neuropsychiatric problems also may be encountered with increased frequency.

1. **Clinically euthyroid** patients may have their thyroid hormone withheld the day of surgery. Postoperatively, patients may resume thyroid replacement when tolerating oral intake. Intravenous thyroid replacement seldom is necessary because the half-life of T_4 exceeds 7 days.
 2. **Mild to moderate hypothyroid patients** with inadequate replacement should have their thyroid medication continued perioperatively. If oral intake is inadvisable, equivalent i.v. T_4 can be given. Newly diagnosed or noncompliant hypothyroid patients should have surgery postponed until they are medically treated.
 3. **Profoundly hypothyroid patients** tolerate surgical stress poorly. These individuals should receive 300–500 mg of i.v. T_4 and stress steroids if they are subjected to trauma or emergent surgery. Intraoperative hypotension should be anticipated.
8. **Adrenal insufficiency/steroid dependence**
- a. **Exogenous steroids** commonly are used to treat a variety of diseases that are encountered in surgical patients. Perioperative management of these individuals requires knowledge of the dose and type of steroid (long- vs. short-acting), schedule, and length of treatment with exogenous steroids.
 - b. **Perioperative stress-dose steroids** are indicated for patients who are receiving chronic steroid replacement or immunosuppressive steroid therapy.
 - c. **Dosage recommendations** for perioperative steroids reflect estimates of normal adrenal responses to major surgical stress. The normal adrenal gland produces 250–300 mg cortisol per day under maximal stress, peaking at 6 hours after surgery and returning to baseline after 24 hours unless stress continues. A regimen of hydrocortisone sodium succinate, 100 mg i.v., on the evening before major surgery, at the beginning of surgery, and every 8 hours on the day of surgery, approximates the normal adrenal stress response. Tapering is not necessary in uncomplicated cases. Patients who are undergoing minor surgery or diagnostic procedures usually do not require stress-dose steroids.
9. **Anticoagulation.** The most common indications for warfarin therapy are atrial fibrillation, venous thromboembolism, and mechanical heart valves.
- a. **Elective procedures.** Mitigation of warfarin's anticoagulant effect occurs only after several days of cessation from the drug, and it requires several days to reestablish the effect after warfarin is resumed. Recommendations for the management of anticoagulation (summarized in [Table 7-8](#)) in the perioperative period require weighing the risk of subtherapeutic anticoagulation (thromboembolic events) against the benefits (reduced incidence of perioperative bleeding).

Table 7-8. Recommendations for preoperative and postoperative anticoagulation in patients taking oral anticoagulants ^a

1. **Preoperative anticoagulation.** It is generally considered safe to perform surgery when the International Normalized Ratio (INR) value is below 1.5. Patients whose INR is maintained between 2.0 and 3.0 normally require withholding of the medication for 4 days preoperatively. For patients whose INR is maintained at a value greater than 3.0, withholding for a longer period of time is necessary. The INR should be measured the day before surgery, if possible, to confirm that the anticoagulation is reversed and, if progress is inadequate, to allow the option of administering a small dose of vitamin K (1.0 mg s.c.). Alternate prophylaxis should be considered for the preoperative period when the INR is less than 2.0 ([Table 7-8](#)).
2. **Postoperative anticoagulation.** The anticoagulant effects of warfarin require several doses before therapeutic levels are reached. For this reason, in patients who can tolerate p.o. or nasogastric medications, warfarin therapy can be resumed on postoperative days 1 or 2. If indicated ([Table 7-8](#)), i.v. heparin should not be restarted until 12 hours after surgery and should be delayed even longer if there is any evidence of bleeding.
- b. **Emergent procedures.** In urgent or emergent situations in which there is no time to reverse anticoagulation before surgery, plasma products must be administered. To provide sufficient functional coagulation factors, several units of fresh frozen plasma are necessary. Vitamin K requires hepatic synthesis of new coagulation factors and is inadequate for reversal of anticoagulation in the emergent setting.

II. postoperative care of the patient. The specifics of postoperative care must be individualized. This section summarizes general considerations in all postoperative patients, then addresses specific concerns in patients with significant comorbid disease.

A. Routine postoperative care

1. **i.v. fluids.** Significant amounts of insensible fluid losses occur during long procedures as a result of evaporative losses from the wound. Vast amounts of fluid are also redistributed into the third space in procedures in which extensive dissections and traumatic handling of the tissues occur. As a general rule, patients should be maintained on maintenance i.v. fluids until they are tolerating p.o. intake. For patients who have undergone extensive abdominal procedures, i.v. fluids in the early postoperative period should be administered according to the extent of the procedure (see [Chapter 4](#)).
2. **DVT prophylaxis.** Many postoperative patients are not immediately ambulatory. In these individuals, it is important to provide prophylactic therapy to reduce the risk of DVT and PE (see [Table 7-9](#) for the recommended prophylactic regimen). Prophylaxis should be started preoperatively because venous stasis and relative hypercoagulability occur during the operation. Management of patients with a history of DVT or PE is discussed in [section I.B.9.a](#).

Table 7-9. Prophylaxis for deep venous thrombosis and pulmonary embolus

- a. **Mechanical prophylaxis** includes graded compression stockings and intermittent pneumatic compression devices, either of which are nearly as effective as unfractionated heparin in reducing DVT in most low- to moderate-risk patients who are undergoing general surgical procedures. These devices alone are inadequate prophylaxis for high-risk (especially cancer) patients and should be avoided in individuals with peripheral vascular disease.
 - b. **Unfractionated heparin**, 5,000 units s.c., starting 2 hours before surgery and continuing every 8–12 hours postoperatively, markedly decreases the incidence of DVT after general surgery. No increase in major hemorrhagic complications is observed with this regimen, although the rate of wound hematoma is higher. This increase may be detrimental in operations that involve prosthetic materials (i.e., hernia repair with mesh), in which case intermittent pneumatic compression devices can be substituted.
 - c. **Low-molecular-weight heparins**, such as enoxaparin (1 mg/kg s.c. every 12–24 hours), are derivatives of unfractionated heparin and have become an important class of agents in the prevention and treatment of thromboembolic disease. These agents have a clinical efficacy that is nearly equivalent to that of i.v. heparin, with fewer hemorrhagic complications and a lower incidence of heparin-induced thrombocytopenia, and they do not require frequent measurement of coagulation times. Prophylactic efficacy has been demonstrated in patients who have undergone total hip replacement and in those who are undergoing elective abdominal or pelvic operation for malignancy, as well as after major trauma.
 - d. **Warfarin** is the agent of choice only for prophylaxis in total hip arthroplasty but can also be used in other orthopedic procedures and in certain high-risk general surgery patients (i.e., antithrombin III deficiency). Warfarin prophylaxis can be instituted at 5 mg p.o. on the night before surgery, with 5 mg p.o. on the evening of surgery. Dosing thereafter is targeted to an INR of 2.0–3.0. This method of prophylaxis carries a significantly higher rate of major postoperative bleeding (5–10%) than with low-dose heparin and should be avoided, if possible, in general surgery patients.
3. **Tubes and drains.** When postoperative orders are written, each tube and drain (including Foley catheters, nasogastric tubes, chest tubes) should be specifically addressed in the orders. If multiple drains are present, they should be numbered and referred to by these numbers in the orders. [Chapter 8](#) addresses the specific management of different types of tubes and drains.
 4. **Wound care.** [Chapter 10](#) addresses the specific considerations in wound care; however, even the care of simple wounds should be addressed in the postoperative orders.
 5. **Pulmonary toilet.** Pain and immobilization in the postoperative patient conspire to decrease the clearance of pulmonary secretions and decrease alveolar recruitment. Fevers, hypoxemia, and even frank pneumonias can develop in patients who have inadequate pulmonary toilet. These complications can be avoided by incentive spirometry, cough and deep breathing exercises, and early mobilization. Adequate pain control and patient education are necessities in providing effective pulmonary toilet.
 6. **Medications**
 - a. **Antiemetics.** Postoperative nausea is common in patients after general anesthesia and in patients receiving narcotics. Although individual preferences dictate the choice of antiemetics, these drugs should be considered in all patients after general anesthesia.
 - b. **Ulcer prophylaxis.** Patients with a history of peptic ulcer disease should have some form of ulcer prophylaxis in the perioperative period, either with

acid-reducing agents or cytoprotective agents, such as sucralfate. Routine ulcer prophylaxis in patients without a history of peptic ulcer disease has only been of proven benefit in those with a coagulopathy or prolonged ventilator dependence; however, it is a common practice to use antiulcer agents in all patients who are n.p.o. for a prolonged period of time.

- c. **Pain control.** Inadequate pain control can significantly slow the recovery or contribute to complications in postoperative patients. Individuals whose pain is poorly controlled are less likely to ambulate and may tend to take shallow breaths, contributing to atelectasis and interfering with the clearance of pulmonary secretions. Pain can also contribute to tachycardia and relative hypertension, increasing myocardial work and possibly increasing the risk of cardiac complications.
- d. **Antibiotics.** Surgeon preferences often dictate the use of postoperative antibiotics in particular cases. Recommendations for specific procedures can be found in [Table 7-7](#). Antibiotic therapy for specific infectious etiologies is discussed in [section III.E.2](#).

7. **Laboratory tests.** Postoperative laboratory tests should be individualized; however, the following considerations are important when planning laboratory evaluations:

- a. A **CBC** should be obtained in the immediate postoperative period and on subsequent postoperative days in any procedure in which considerable blood loss occurred. This test is also important on subsequent postoperative days when there is concern for postoperative or ongoing infection. Serial hematocrits should be ordered in any patient in whom there is concern for ongoing blood loss.
- b. **Serum electrolytes, BUN, and creatinine** are important postoperatively in patients who are n.p.o. or are receiving large volumes of i.v. fluids, total parenteral nutrition, or transfusions. In patients with large transfusion requirements, it is important to obtain frequent calcium and magnesium measurements.
- c. **Coagulation studies** are important in patients who have had insults to the liver or large transfusion requirements.
- d. **CXRs** are a necessity after any procedure in which the thoracic cavity is entered or when central venous access is attempted. CXRs on subsequent postoperative days should be considered on an individual basis if significant pulmonary or cardiovascular disease is present.

B. Specific considerations in postoperative care

1. **Seizure disorders.** Perioperative management of patients with known seizure disorders should be directed toward an understanding of the type (i.e., partial vs. generalized, simple partial vs. complex partial), frequency, and degree of control of the disorder. Well-controlled seizure disorders pose little additional risk if proper attention is paid to perioperative management of anticonvulsants. Standard seizure precautions should be included in the postoperative nursing orders.
 - a. **Phenytoin and phenobarbital** are available in parenteral form. If patients are expected to resume oral intake within 24 hours of operation, these oral anticonvulsants can be withheld. If oral intake is withheld for more than 24 hours, the equivalent parenteral dose can be given, using a divided schedule.
 - b. **Carbamazepine, ethosuximide, and valproic acid** are not available parenterally; decisions to withhold these medications or to substitute parenteral phenytoin must take into account type, frequency, and severity of seizures. Patients with frequent generalized tonic-clonic seizures might require parenteral anticonvulsant substitution. Phenytoin can be given intravenously as a loading dose of 18–20 mg/kg over 30 minutes (<50 mg per minute) followed by 5 mg/kg per day as maintenance, divided into three daily doses.
2. **Cardiovascular disease**
 - a. **CAD**
 1. **Control of precipitants.** A basic tenet in the postoperative management of patients with known or suspected CAD is the avoidance of stressors that may exacerbate ischemia through increased myocardial oxygen consumption.
 - a. **Acute hypertension** increases oxygen demand by increasing ventricular wall stress. The management of acute hypertension is described in [section III.H.2](#).
 - b. **Pain control** is critical in CAD patients, as pain can precipitate tachycardia and hypertension.
 - c. **Oxygen** should be administered in the early postoperative period to maximize the oxygen content of the blood.
 - d. **Anemia** should be avoided in patients with known CAD, as it decreases the oxygen-carrying capacity. Transfusions should be considered when the hemoglobin falls below 9.0.
 2. **Monitoring.** Patients at high risk for perioperative myocardial morbidity should have telemetry monitoring in the early postoperative period. Invasive monitoring should be considered in patients undergoing urgent or emergent surgery with a history of CAD or poorly compensated CHF, those with unstable or severe angina, and those with a recent MI for whom operation cannot be delayed.
 3. **Rule out MI protocol.** No consensus has been reached regarding the appropriate protocol to rule out MI or the indications for this protocol. Daily ECGs and a series of three troponin-I levels 12 hours apart are appropriate in patients with significant risk factors who have had a major surgical procedure.
 4. **Medications**
 - a. Patients who are receiving **beta-adrenergic receptor antagonists** (beta-blockers) should have this medication continued through the perioperative period. Additionally, beta-blockers should be considered in all patients undergoing noncardiac major surgery who have or are at risk for CAD. Perioperative beta-blockers can be administered in either the oral or parenteral form (metoprolol, 25–50 mg p.o. b.i.d. or 2.5–5.0 mg i.v. every 6 hours).
 - b. Patients who are receiving **nitrate therapy** should be given these medications during the perioperative period. Those who are receiving oral nitrate therapy can be switched to topical therapy. Additionally, nitrates can benefit patients who are deemed at substantially increased risk of perioperative cardiac morbidity, such as those who are undergoing emergent operation and those with severe or unstable angina.
 - c. **Calcium channel blockers** should also be continued in the perioperative period if they were taken preoperatively.
 - b. **Congestive heart failure.** Ischemic heart disease is frequently the cause of CHF, and patients with known CHF should be considered to have CAD and managed as described previously. Patients with poor preoperative cardiac function should be monitored in an intensive care setting with invasive monitoring to allow for careful titration of volume replacement. Daily CXRs also assist in detecting pulmonary edema that results from volume overload. Patients who are given digoxin therapy for CHF should continue receiving this drug in the postoperative period, either in the p.o. or parenteral form. The management of fluid failure in the postoperative period is discussed in [section III.B.2](#).
 3. **Pulmonary disease.** Patients with COPD are at high risk for pulmonary complications in the postoperative period. Bronchodilators should be continued in this period, either in the nebulized form or as metered-dose inhalers. These patients require supplemental oxygen and frequent assessment of oxygen saturations. Individuals with severe COPD may have a respiratory drive that is solely dependent on maintaining adequate oxygenation of the blood, having lost their respiratory drive in response to hypercarbia. In these patients, excessive supplemental oxygen decreases their respiratory drive and minute ventilation, leading to hypercarbia and possibly obtundation. Frequently, such patients must be maintained with oxygen saturations in a narrow range between 88% and 92% to avoid hypercarbia.
 4. **Renal disease**
 - a. **Fluids and electrolytes.** Patients with chronic renal insufficiency require replacement of operative fluid losses in the same manner as normal patients; however, care should be taken to avoid excessive fluid replacement. Fluids given i.v. to these patients should not contain potassium. Frequent measurement of serum electrolytes is needed in these individuals while they are receiving i.v. hydration. Early dialysis may be necessary because of hyperkalemia or intravascular volume overload.
 - b. **Drug therapy in renal insufficiency.** Many drugs and their metabolites are cleared by the kidneys. In patients with reduced creatinine clearance, it is important to adjust the dose and the dosing interval of many drugs, including antibiotics. Meperidine is contraindicated in patients with renal insufficiency, as one of its metabolites can accumulate and precipitate seizures.
 5. **Diabetes.** Postoperative management of diabetic surgical patients centers on maintenance of euglycemia and management of chronic diabetic complications.
 - a. **Metabolic control** of diabetic patients requires blood glucose in the range of 100–250 mg/dL. All diabetic patients should have blood glucose checked on arrival to the postanesthesia recovery room.
 1. **Diet-controlled diabetics** infrequently need glucose infusion or insulin therapy after minor surgery. After major surgery, these patients require dextrose infusion while fasting, blood glucose monitoring every 6 hours, and sliding-scale insulin coverage as needed.
 2. **Diabetic patients who are receiving oral hypoglycemic agents** frequently need insulin postoperatively. These patients require dextrose infusion while fasting, q.i.d. blood glucose monitoring (performed before meals and at bedtime), and subcutaneous regular insulin coverage as needed. When oral intake is tolerated, the preoperative oral hypoglycemic regimen should be resumed.
 3. **Patients who are taking insulin preoperatively** usually require insulin postoperatively to achieve adequate control of the serum glucose level. Brittle diabetics must receive their basal insulin requirements or they are at risk for development of DKA. DKA may develop in the fasting or metabolically stressed patient with a serum glucose level of only 100–200 mg/dL. Patients generally require at least their total daily dose given in divided doses postoperatively. Stress related to anesthesia, the operative procedure, and concurrent illness increases this requirement, especially during the first 24–48 hours after surgery. Regularly scheduled insulin dosing based on basal requirements plus anticipated patient needs is much more physiologic and thus preferable to the more common sliding-scale method. Parenteral glucose (100–150 g per day), given as 5% dextrose in intravenous fluids, should be given to all fasting diabetic patients to promote anabolism and to prevent ketosis and inadvertent hypoglycemia.
 - a. **Intermittent dosing of subcutaneous insulin** can be given as intermediate-acting [neutral protamine Hagedorn (NPH)] insulin twice a day, with hyperglycemia managed by supplemental dosing of regular insulin based on blood glucose determinations performed every 4–6 hours.

Alternatively, regular insulin can be given in four divided doses before meals and at midnight if the patient is eating or simply every 6 hours if the patient is receiving intravenous glucose.

- b. **Continuous intravenous insulin infusion** is indicated in patients with hyperglycemia that is not controlled by intermittent subcutaneous dosing. The infusion should be started at 1–2 units per hour and titrated to effect. Initial blood glucose determinations should be done hourly. Conversion to a scheduled dose of regular or intermediate-acting subcutaneous insulin is based on clinical stability and should begin before discontinuation of the intravenous insulin infusion.

III. Complications

A. Neurologic complications

1. Perioperative stroke

- a. **Presentation.** Transient ischemic attacks (neurologic deficits that resolve in 24 hours) and stroke cannot be differentiated at the onset of symptoms. The patient usually describes rapid onset of focal loss of neurologic function (unilateral weakness or clumsiness, sensory loss, speech disorder, diplopia, or vertigo). Massive strokes can also present with altered mental status.
- b. **Physical examination.** A thorough neurologic examination, in addition to vital signs, finger-stick glucose, and pulse oxymetry, should be assessed.
- c. **Evaluation**
 1. **Laboratory evaluation** should include a CBC, electrolytes, BUN, creatinine, and coagulation studies. An ECG should be done to rule out cardiac arrhythmia.
 2. A **CT scan of the head** should be obtained urgently to rule out a hemorrhagic stroke.
 3. **Further studies**, including echocardiography, carotid and transcranial ultrasound, and MR scan should only be ordered in consultation with a neurologist.
- d. **Treatment**
 1. **General supportive measures** include supplemental oxygen and i.v. fluid.
 2. **Aspirin** (325 mg p.o.) should be given immediately in ischemic stroke, as it has been shown to reduce death and dependency modestly.
 3. **Thrombolysis** has been proven effective in improving outcomes from ischemic strokes; however, it is usually contraindicated in postoperative patients and should only be initiated in close consultation with a neurologist.

2. **Seizures.** Evaluation and treatment of postoperative seizures involve the same principles as those encountered in other settings. Most seizures in surgical patients without a history of seizure are generalized and can be attributed to metabolic derangements, including electrolyte abnormalities (e.g., hyponatremia, hypocalcemia), hypoglycemia, sepsis, fever, and drugs (e.g., imipenem).

- a. **Determine from patient history whether a true seizure was witnessed;** if so, note its type, characteristics (i.e., general vs. focal onset), and similarity to any previous seizures. New-onset seizures are worrisome, and iatrogenic causes (e.g., medications) and cerebrovascular accident must be considered. A history of preoperative alcohol use should prompt an evaluation for substance withdrawal.
- b. **Complete physical and neurologic examination** should focus first on oxygenation and hemodynamics, then on any sequelae of seizure, including trauma, aspiration, or rhabdomyolysis. A focally abnormal neurologic examination, especially in the setting of a new-onset focal seizure, suggests a possible cerebrovascular event.
- c. **Laboratory and diagnostic studies.** Begin with rapid blood glucose determination, CBC, and serum chemistries, including calcium and magnesium as well as an oxygen saturation. Serum levels of anticonvulsants should be measured in patients receiving these medications. Patients with new-onset seizures who do not have identifiable metabolic or systemic causes warrant further evaluation with a head CT scan and a lumbar puncture.
- d. **Treatment** of new-onset, single, nonrecurrent seizures or recurrent generalized seizures with identifiable metabolic or systemic causes usually requires only correction of the underlying abnormality.
 1. **Recurrent generalized tonic-clonic seizures** require anticonvulsant therapy. A 15- to 20-mg/kg load of phenytoin, given parenterally in three divided doses followed by maintenance dosing of 5 mg/kg per day in three divided doses, controls most seizures. Therapeutic serum levels are 10–20 mg/mL.
 2. **Status epilepticus** is a medical emergency.
 - a. **Monitor cardiopulmonary parameters and stabilize the patient's airway with a soft oral or nasal airway.** Endotracheal intubation might be required to protect the airway, especially in the setting of status epilepticus when significant doses of antiepileptic agents may be required to terminate the seizure. Intravenous access should be established peripherally.
 - b. **Administer parenteral anticonvulsants promptly.**
 - i. **Diazepam** (5–10 mg i.v. every 5–10 minutes) or **lorazepam** (1–2 mg i.v. every 5–10 minutes) should be administered to patients with generalized convulsions of greater than 5 minutes' duration. Results usually are obtained within 10 minutes. These agents are relatively short acting, however, and a second parenteral anticonvulsant should be started concurrently.
 - ii. **Phenytoin** administered parenterally is the first choice to supplement the benzodiazepines in this setting, with dosing as noted previously.
 - iii. **Phenobarbital** is a second-line agent and should be used when phenytoin is contraindicated (e.g., heart block) or ineffective. A loading dose of 10 mg/kg can be given at 100 mg/min. Maintenance doses of 1–5 mg/kg per day i.v. or orally are required to achieve therapeutic plasma levels. Institution of a phenobarbital coma should be considered if status epilepticus continues.

3. **Delirium.** Delirium is common in patients (especially the elderly) who undergo the stress of operation. An underlying cause usually can be identified and in most cases involves medications or infection. Other causes include hypoxemia, electrolyte abnormalities, cardiac arrhythmias, MI, and stroke. Alcohol withdrawal, discussed in [section III.A.4](#), is another common cause of postoperative delirium.

- a. **Symptoms** often include impaired memory, altered perception, and paranoia. Altered sleep patterns result in drowsiness during the day and wakefulness and agitation at night (sundowning). Combativeness is common.
- b. **Treatment** begins with determination of the underlying cause. Pulse oximetry to assess oxygenation is included with determination of pulse, BP, and temperature. Electrolyte abnormalities and infection can be assessed with routine blood work and urinalysis. Other testing, including ECG, ABG, and CXR, is dictated by clinical suspicion. Medications should be reviewed carefully, with attention directed toward anticholinergic agents, opiate analgesics, and antihistamines. If no underlying organic cause is identified, alteration in sleep patterns or sensory deprivation can be invoked, and haloperidol (1–5 mg p.o. or i.m.) can be prescribed. Often, family reassurance or transfer to a naturally lighted room is curative. Physical restraints might be necessary to prevent self-harm.

4. **Alcohol withdrawal.** Alcohol withdrawal can occur in the surgical patient who abruptly decreases or stops the regular consumption of alcohol. This syndrome carries significant risk of morbidity and mortality, and prevention based on a high index of suspicion is necessary. Clues to withdrawal potential include a prior history of withdrawal symptoms and a regular pattern of alcohol intake preoperatively. Some patients have ill-defined histories of intermittent treatment for seizures.

- a. **Symptoms.** Minor withdrawal can begin 6–8 hours after cessation of alcohol intake and is characterized by anxiety, tremulousness, anorexia, and nausea. Signs include tachycardia, hypertension, and hyperreflexia. These signs and symptoms generally resolve within 24–48 hours. Delirium tremens can occur 24–48 hours or longer after cessation of alcohol intake and is characterized by disorientation, hallucinations, and autonomic lability that includes tachycardia, hypertension, fever, and profuse diaphoresis.

b. Treatment

1. **Benzodiazepines**, such as chlordiazepoxide, 25–100 mg p.o. every 6 hours; oxazepam, 5–15 mg p.o. every 6 hours; or diazepam, 5–20 mg p.o. or i.v. every 6 hours can be used as prophylaxis in alcoholics who have a history of withdrawal or to alleviate symptoms of minor withdrawal. Patients with delirium tremens should be given diazepam, 5–10 mg i.v. every 10–15 minutes, to control symptoms. Oversedation must be avoided through close monitoring. The dosage of benzodiazepines should be reduced in patients with liver impairment. Moderate alcohol intake with meals can be a simple way to prevent and treat alcohol withdrawal.
2. **Clonidine**, 0.1 mg p.o. q.i.d., or atenolol, 50–100 mg p.o., every day, can be used to treat tachycardia or hypertension resulting from autonomic hyperactivity. These patients require close hemodynamic monitoring during therapy.
3. **General medical care.** Fluid and electrolyte abnormalities should be corrected, and fever should be treated with acetaminophen or cooling blankets as needed. Thiamine, 100 mg i.m. for 3 days, followed by 100 mg p.o. every day, should be given to all suspected alcoholic patients to prevent development of Wernicke's encephalopathy. Many chronic alcoholics have hypomagnesemia, and, if it is present, magnesium sulfate should be administered to patients with normal renal function as either 2 g i.m. every 6 hours for three doses or p.o. in the form of magnesium-containing antacids, 30 mL every 4 hours. Folate should be given 1 mg i.m. or p.o. every day.
4. **Restraints** should be used only when necessary to protect the patient from self-harm.
- c. **Alcohol withdrawal seizures** occur within 24 hours of cessation of alcohol and are most often generalized tonic-clonic. They are usually brief and self-limited, although status epilepticus occurs in approximately 3% of cases. Benzodiazepines are most helpful in preventing recurrent seizures.

B. Cardiovascular complications

1. Myocardial ischemia and infarction

- a. The **presentation** of myocardial ischemia in the postoperative patient is often subtle, as incisional pain may be difficult to differentiate from chest pain. Frequently, perioperative MI is silent or presents with dyspnea, hypotension, or atypical pain.

- b. In postoperative patients who present with chest pain, the **differential diagnosis** includes myocardial ischemia or infarction, PE, pneumonia, and, less commonly, pericarditis, aortic dissection, and pneumothorax.
- c. **Evaluation**
1. **Physical examination** should be performed carefully to assess BP, heart rate, and general organ and tissue perfusion. The lungs should be auscultated for signs of pulmonary edema (with myocardial ischemia or infarction and CHF) and diminished or absent breath sounds unilaterally (pneumothorax). Auscultation of the heart can reveal a new murmur suggestive of ischemic mitral regurgitation or a pericardial friction rub suggestive of pericarditis. All peripheral pulses should be palpated, and BP should be measured in both arms.
 2. **Diagnostic testing**
 - a. An **ECG** is warranted in virtually all cases of postoperative chest pain, and comparison should be made to prior tracings.
 - b. **Laboratory data**
 - i. **Cardiac enzymes.** An elevated troponin-I level (>0.4 ng/mL) is diagnostic of MI. This laboratory test has replaced other biochemical markers of myocardial injury (lactate dehydrogenase, creatine kinase). A series of three samplings of troponin-I 12 hours apart has a sensitivity and specificity of more than 90% for detecting myocardial injury.
 - ii. **Routine chemistries and hemoglobin** should be determined.
 - iii. **Oxygen saturation** should be determined via pulse oximetry, and supplemental oxygen should be administered. Patients with chest pain should have arterial oxygen saturation determined with a noninvasive oxygen saturation monitor. Patients with oxygen saturations of less than 93% on room air or with symptoms of dyspnea or other signs of respiratory compromise should have ABG determined. Significant hypoxia can be seen with MI, CHF, pneumonia, and PE.
 - c. **CXR** should be obtained and evaluated for pneumothorax, infiltrate, or evidence of pulmonary edema.
 - d. **Further diagnostic evaluation** [e.g., echocardiography, coronary catheterization, or ventilation-perfusion (V/Q) scintigraphy] should be pursued as indicated by the diagnostic workup.
 3. **Treatment**
 - a. **Telemetry** should be used in all patients with suspected myocardial ischemia.
 - b. **Oxygen therapy.** The arterial oxygen saturation should be kept at greater than 90% with supplemental oxygen. Endotracheal intubation and mechanical ventilation are indicated for patients with hypoxia that is refractory to supplemental oxygen therapy, progressive hypercapnia, or respiratory fatigue.
 - c. **Pharmacologic therapy**
 - i. **Nitrates.** In the absence of hypotension (systemic BP <90 mm Hg), initial management of patients with chest pain of presumed cardiac origin includes the use of sublingual nitroglycerin (0.4 mg), which can be repeated every 5 minutes. Additionally, topical nitrate therapy (0.5–2.0 in. every 6 hours) can be instituted. Ongoing myocardial ischemia or infarction should be treated with intravenous nitroglycerin, starting with an infusion rate of 5 μ g/min and increased at 5- μ g-per-minute increments until the chest pain is relieved or significant hypotension develops (systemic BP <90 mm Hg).
 - ii. **Beta-adrenergic receptor antagonists.** In the absence of significant contraindications (e.g., heart failure, bradycardia, heart block, or significant COPD), patients should be treated with intravenous beta-adrenergic receptor antagonists (e.g., metoprolol, 15 mg i.v., in 5-mg doses every 5 minutes, followed by a 50- to 100-mg oral dose every 12 hours).
 - iii. **Intravenous morphine sulfate** (1–4 mg i.v. every hour) is also useful in the acute management of chest pain to decrease the sympathetic drive of an anxious patient.
 - iv. **Antiplatelet therapy** in the form of a chewed non-enteric-coated aspirin (325 mg) can also be given, assuming the patient is at low risk of perioperative bleeding.
 - d. **Other therapeutic measures.** Thrombolytic therapy, anticoagulation, or coronary catheterization should be considered on an individual basis in consultation with a cardiologist.
2. **Congestive heart failure**
- a. **Differential diagnosis** of shortness of breath or hypoxia in the perioperative period includes CHF, pneumonia, atelectasis, PE, reactive airway disease (asthma, COPD exacerbation), and pneumothorax. These conditions are discussed in [section III.C](#).
 - b. **Evaluation**
 1. **History.** CHF typically occurs immediately postoperatively as a result of excessive intraoperative administration of fluids, or 24–48 hours postoperatively, related to mobilization of fluids that are sequestered in the extracellular space. Myocardial ischemia or infarct can also lead to CHF.
 2. **Physical examination** should be directed toward signs and symptoms of fluid overload and myocardial ischemia. Net fluid balance and weight for the preceding days should be assessed.
 3. **Diagnostic testing**
 - a. **Laboratory data**
 - i. **Pulse oximetry**
 - ii. **ECG**
 - iii. **CXR**
 - iv. An **echocardiogram** is frequently indicated in patients with new CHF to evaluate the valves, assess the dimensions of each cardiac chamber, and rule out tamponade.
 - c. **Management of CHF**
 1. **Supplemental oxygen** should be administered. Mechanical ventilation (either noninvasive or via endotracheal intubation) is indicated in patients with refractory hypoxemia.
 2. **Diuretics.** Treatment should be initiated with furosemide (20–40 mg i.v. push), with doses up to 200 mg every 6 hours as necessary to achieve adequate diuresis. Fluid intake should be limited to 1,000 mL per day. Serum potassium should be monitored closely.
 3. **Morphine** (1–4 mg i.v. push every 1 hour)
 4. **Arterial vasodilators.** To reduce afterload and help the failing heart in the acute setting, sodium nitroprusside should be administered to lower the systolic BP to 90–100 mm Hg. Alternatively, enalaprilat (0.625–2.5 mg i.v. every 6 hours) or oral angiotensin-converting enzyme inhibitors can be used.
 5. **Inotropic agents.** Digoxin increases myocardial contractility and can be used to treat patients with mild failure. Patients with fluid failure may need invasive monitoring and titration of drips if they do not respond to these measures. If there is a low cardiac index (<2.5 L/min/m²) with elevated filling pressures, inotropic agents are indicated. Therapy can be initiated with dobutamine (3–20 μ g/kg per minute) to increase the cardiac index to a value near 3 L/min/m². If the hypotension is accompanied by low systemic vascular resistance, vasopressors may be useful ([Table 7-10](#)).

Table 7-10. Doses of commonly used vasopressors (alpha-adrenergic agonists)

C. Pulmonary complications

1. The **differential diagnosis** of dyspnea includes atelectasis, pneumonia, CHF, COPD or asthma exacerbation, pneumothorax, PE, and aspiration.
2. **Evaluation**
 - a. **History.** Additional factors that help to differentiate disease entities include the presence of a fever, chest pain, and the time since surgery.
 - b. **Physical examination** with attention to jugular venous distention, breath sounds, symmetry, and respiratory effort
 - c. **Diagnostic testing**
 1. **Laboratory.** CBC, chemistry profile, and pulse oximetry or ABG
 2. **ECG** should be obtained for any patient older than age 30 years with significant dyspnea or tachypnea to exclude myocardial ischemia and in any patient who is dyspneic in the setting of tachycardia.
 3. **CXR** is mandatory in all dyspneic patients.
 4. **V/Q scan**, pulmonary angiogram, or chest CT scan might be required in special circumstances.
3. **Management** of specific diagnoses
 - a. **Atelectasis** commonly occurs in the first 36 hours after operation and typically presents with dyspnea and hypoxia. Therapy is aimed at reexpanding the collapsed alveoli. For most patients, deep breathing and coughing along with the use of incentive spirometry is adequate. Postoperative pain should be sufficiently controlled so that pulmonary mechanics are not significantly impaired. In patients with significant atelectasis or lobar collapse, chest physical therapy and nasotracheal suctioning might be required. In rare cases, bronchoscopy can aid in clearing mucus plugs that cannot be cleared using less

invasive measures.

- b. **Pneumonia** is discussed in [section III.E.2.b](#).
- c. **PE** is discussed in [section III.F.2](#).
- d. **Gastric aspiration** usually presents with acute dyspnea and fever. CXR might be normal initially but subsequently can demonstrate a pattern of diffuse interstitial infiltrates. Therapy is supportive, and antibiotics are typically not given empirically.
- e. **Pneumothorax** is treated with tube thoracostomy. If tension pneumothorax is suspected, immediate needle decompression through the second intercostal space in the midclavicular line using a 14-gauge needle should precede controlled placement of a thoracostomy tube.
- f. **COPD and asthma** exacerbation present with dyspnea or tachypnea, wheezing, hypoxemia, and possibly hypercapnia. Acute therapy includes administration of supplemental oxygen and inhaled beta-adrenergic agonists [albuterol, 3.0 mL (2.5 mg) in 2 mL normal saline every 4–6 hours of nebulization]. Beta-adrenergic agonists are indicated primarily for acute exacerbations rather than for long-term use. Anticholinergics such as ipratropium bromide metered-dose inhaler (Atrovent, 2 puffs every 4–6 hours) can also be used in the perioperative period, especially if the patient has significant pulmonary secretions. Patients with severe asthma or COPD may benefit from parenteral steroid therapy (methylprednisolone, 50–250 mg i.v. every 4–6 hours) as well as inhaled steroids (beclomethasone metered-dose inhaler, 2 puffs q.i.d.), but steroids require 6–12 hours to take effect.

D. Renal complications

1. ARF

- a. **Causes.** The etiologies of postoperative renal insufficiency can be divided into prerenal, intrinsic renal, and postrenal classes ([Table 7-11](#)).

Table 7-11. Etiologies of postoperative renal failure

1. **Prerenal azotemia** results from decreased renal perfusion that might be secondary to hypotension, intravascular volume contraction, or decreased effective renal perfusion (CHF, hepatorenal syndrome).
 2. **Renal.** Intrinsic renal causes of ARF include drug-induced acute tubular necrosis, pigment-induced renal injury, radiocontrast dye administration, acute interstitial nephritis, and prolonged ischemia from suprarenal aortic cross-clamping.
 3. **Postrenal causes** of ARF can result from obstruction of the ureters or bladder. Operations that involve dissection near the ureters, such as colectomy, colostomy closure, or total abdominal hysterectomy, have a higher incidence of ureteral injuries. In addition to ureteral injuries or obstruction, causes of postrenal ARF can be functional obstruction of the bladder from an enlarged prostate or postoperative pain and medication administration.
- b. **General evaluation**
 1. **History**
 2. **Physical examination**
 3. **Laboratory evaluation**
 - a. **Urinalysis**
 - b. **Serum chemistries**
 - c. **Urinary index** helps to classify ARF into prerenal, postrenal, or intrinsic renal categories ([Table 7-12](#)). Fractional excretion of sodium (Fe_{Na}) can be calculated from this formula:

Table 7-12. Laboratory evaluation of oliguria and acute renal failure

Equation

4. **Other diagnostic testing**
 - a. **Renal ultrasonography** can be used to exclude the diagnosis of obstructive uropathy.
 - b. **Radiologic studies** using intravenous contrast are contraindicated in patients with suspected ARF due to the potential to exacerbate renal injury.
- c. **Management of specific problems**
 1. **Oliguria** (<500 mL per day) in the postoperative period
 - a. **Evaluation.** Laboratory evaluation should include serum chemistries, BUN, and creatinine and the urinary indices, such as Fe_{Na} , U_{Na} , and U_{Osm} . Because management of the causes of oliguria differs substantially, the goal of this evaluation is to determine the patient's intravascular volume status and to differentiate the causes of oliguria.
 - b. **Differential diagnosis** ([Table 7-11](#))
 - c. **Management**
 - i. **Prerenal.** In most surgical patients, oliguria is caused by hypovolemia. Initial management includes fluid challenges (normal saline, 500 mL). Patients with adequate fluid resuscitation and CHF may benefit from invasive monitoring and optimization of cardiac function.
 - ii. **Intrinsic renal.** Management includes maintaining the patient in a euvolemic state and the use of diuretics (furosemide, 40–200 mg i.v.).
 - iii. **Postrenal.** Ureteral injuries or obstruction can be treated with percutaneous nephrostomy tubes and generally are managed in consultation with a urologist. Urinary retention and urethral obstruction can be managed by placement of a Foley catheter, or, if necessary, a suprapubic catheter.
 2. **Elevated creatinine and ARF**
 - a. **Evaluation.** The laboratory and diagnostic evaluation for patients with a rising creatinine is similar to the evaluation for patients with oliguria and is discussed above.
 - b. **Management**
 - i. **Medical management** includes careful attention to the intravascular volume status. The patient should be weighed daily, and intakes and outputs should be recorded carefully. Serum electrolytes should be monitored closely. The patient should be maintained in a euvolemic state. Protein intake should be reduced to 0.5 g/kg per day to decrease nitrogenous wastes, although this is controversial. Sodium and potassium intake should be reduced. Hyperkalemia, metabolic acidosis, and hyperphosphatemia are common problems in patients with ARF and should be managed as discussed in [Chapter 4](#). Medication doses should be adjusted appropriately.
 - ii. **Dialysis.** Indications for dialysis include intravascular volume overload, hyperkalemia, severe metabolic acidosis, and complications of uremia (encephalopathy, pericarditis).

E. Infectious complications

1. Management of postoperative infection and fever

- a. **Evaluation of fever** should take into context the time after operation in which the fever occurs.
 1. **Intraoperative fever** may be secondary to malignant hyperthermia, a transfusion reaction, or a preexisting infection.
 - a. **Diagnosis and management** of a transfusion reaction are discussed in [Chapter 5](#).
 - b. **Malignant hyperthermia** is discussed in [Chapter 6](#).
 - c. **Preexisting infections** should be treated with empiric intravenous antibiotics. For patients with no systemic manifestations of sepsis, cefazolin alone is appropriate.
 2. **High fever (>39°C) in the first 24 hours** is commonly the result of a streptococcal or clostridial wound infection, aspiration pneumonitis, or a preexisting infection.
 - a. **Streptococcal wound infections** present with severe local erythema and incisional pain. Penicillin G (2 million units i.v. every 6 hours) or ampicillin (1–2 g i.v. every 6 hours) is effective therapy. Rarely, patients with a severe necrotizing clostridial infection present with systemic toxemia, pain, and crepitus near the incision. Treatment includes emergent operative débridement and metronidazole (500 mg i.v. every 6 hours) or clindamycin (600–900 mg i.v. every 8 hours).

- b. **Aspiration pneumonitis** (see [section III.C.3.d](#))
- 3. **Fever that occurs more than 72 hours after surgery** has a broad differential diagnosis, consisting of pneumonia, urinary tract infection, thrombophlebitis, wound infection, intraabdominal abscess, and drug allergy.
- b. **Diagnostic evaluation.** The new onset of fever or leukocytosis without an obvious source of infection requires a careful history and physical examination and selected laboratory tests. All intravenous sites should be examined as well.
 1. **Specific laboratory tests**
 - a. **CBC**
 - b. **Urinalysis**
 - c. **CXR**
 - d. **Gram stain/culture.** Cultures of the blood, sputum, urine, and/or wound should be dictated by the clinical situation.
 2. **Antibiotics.** Empiric antibiotics can be initiated, with therapy directed by clinical suspicion.
- 2. **Management of specific infectious etiologies.** [Table 7-13](#) summarizes antibiotic choices.

Table 7-13. Choice of antibiotics for specific postoperative infections

- a. **Wound infection** is diagnosed by local erythema, swelling, pain and tenderness, and wound drainage. Fever and leukocytosis are usually present but may be absent in superficial wound infections. The primary treatment is to open and pack the wound at the site of erythema and to allow drainage. The wound should be cultured at this time. If the infection is contained in the superficial tissue layers, antibiotics are not required. In the case of a clean procedure that did not enter the bowel, the usual pathogens are staphylococcal and streptococcal species. If local erythema is extensive, cefazolin (1 g i.v. every 8 hours) or oxacillin (1 g i.v. every 6 hours) can be administered. Wound infections in the perineum or after bowel surgery are more likely to be caused by enteric pathogens and anaerobes and should be treated with ceftazidime (1 g i.v. every 8 hours) or ceftazidime (1 g i.v. every 6 hours). More aggressive infections with involvement of underlying fascia require emergent operative débridement and broad-spectrum intravenous antibiotics.
 - b. **Respiratory infections.** Pneumonia is diagnosed by the presence of fever, leukocytosis, purulent sputum production, and an infiltrate on CXR. After Gram staining and culturing of the sputum, empiric antibiotics can be started. Pneumonias that occur in postoperative patients should be treated as nosocomial infections with ceftazidime (1–2 g i.v. every 8 hours) and gentamicin (1.0–1.5 mg/kg i.v. every 8 hours).
 - c. **Gastrointestinal infections** may present with fever, leukocytosis, and bloody diarrhea. Evaluation includes culturing the stool for *Clostridium difficile* and assaying for the *C. difficile* toxin. Initial therapy includes fluid resuscitation and antibiotic therapy ([Table 7-13](#)).
 - d. **Intraabdominal abscess or peritonitis** presents with fever, leukocytosis, abdominal pain, and tenderness. If the patient has generalized peritonitis, emergency laparotomy is indicated. If the inflammation appears to be localized, a CT scan of the patient's abdomen and pelvis should be obtained. The primary management of an intraabdominal abscess is drainage. In some circumstances, this can be performed percutaneously with radiologic guidance. In other situations, operative débridement and drainage are required. Empiric antibiotic therapy should cover enteric pathogens and anaerobes ([Table 7-13](#)).
 - e. **Genitourinary infections.** After the urine is cultured, simple lower-tract infections can be managed with trimethoprim-sulfamethoxazole (160 mg trimethoprim, 800 mg sulfamethoxazole p.o. every 12 hours) or ampicillin (1 g i.v. every 8 hours) for 3 days. Very ill patients or those with pyelonephritis should be treated with ampicillin and gentamicin (3–5 mg/kg in 2–3 divided doses) for 5–7 days.
 - f. **Prosthetic device–related infections** may present with fever, leukocytosis, and systemic bacteremia and may be very difficult to diagnose. Infection of prosthetic valves may present with a new murmur. MR scan or CT scanning may demonstrate infection of prosthetic vascular grafts with fluid or gas around the graft and development of a pseudoaneurysm. Management may require removal of the infected device and the use of long-term antibiotics. Patients who have an infected device left in place require intravenous antibiotic coverage as long as the graft is in place ([Table 7-13](#)).
 - g. **Catheter-related infections** are diagnosed by the presence of fever, leukocytosis, and systemic bacteremia. Local erythema and purulence may be present around central venous catheter insertion sites. Erythema, purulence, a tender thrombosed vein, or lymphangitis may be present near an infected peripheral intravenous line. Management includes removal of the catheter, culture of the catheter tip, and intravenous antibiotic coverage ([Table 7-13](#)).
 - h. **Fascial or muscle infections** may result from gross contamination of a surgical wound or from a previously infected wound. Fasciitis and deep-muscle infections present with hemorrhagic bullae that overlie the infected area, with rapidly progressive edema, erythema, pain, and crepitus. Fever, tachycardia, and ultimately cardiovascular collapse occur in rapid succession. Therapy includes emergent operative débridement, management of shock, and broad-spectrum antibiotics (including anaerobic coverage). Necrotizing fasciitis is a surgical emergency; death may result within a few hours of the development of symptoms.
 - i. **Viral infections** complicating operations are uncommon in immunocompetent patients.
 - j. **Fungal infections** (primarily with *Candida* species) occur most commonly after long-term antibiotic administration. In these patients, evaluation of persistent fever without an identified bacterial source should include several sets of routine and fungal blood cultures, removal of all intravenous catheters, and examination of the retina for *Candida endophthalmitis*. Therapy includes either amphotericin B or fluconazole ([Table 7-13](#)).
- F. DVT and PE**
1. **Diagnosis and treatment of DVT**
 - a. **Diagnosis**
 1. **Symptoms** of DVT vary greatly, although classically they include pain and swelling of the affected extremity distal to the site of venous obstruction. Signs of DVT on physical examination may include edema, erythema, warmth, a palpable cord, or calf tenderness with dorsiflexion of the foot (Homans' sign). Physical examination alone is notoriously inaccurate (<50%) in the diagnosis of DVT, and a high index of suspicion is often required to pursue further diagnostic measures.
 2. **Laboratory studies** may assist in the diagnosis and management of the patient with DVT. CBC, baseline prothrombin time and partial thromboplastin time (PTT), and chemistry-12 panel may suggest an underlying cause. Special hematologic testing generally is not required for postoperative DVT evaluation.
 3. **Noninvasive studies** of the venous system, most notably B-mode ultrasonography plus color Doppler (duplex scanning), have revolutionized the diagnosis and management of suspected DVT. Reported sensitivity and specificity of this test for the detection of proximal DVT are greater than 90%, with nearly 100% positive predictive value. Impedance plethysmography is similarly useful for the detection of all but asymptomatic proximal DVT. Both modalities are less reliable in the detection of infrapopliteal thrombi, and a negative study in symptomatic patients should be followed by repeat examination in 48–72 hours to evaluate for propagation of clot proximally. Patients in whom a negative study contrasts with a strong clinical suspicion may require contrast venography, the gold standard for diagnosis of DVT.
 - b. **Treatment**
 1. **Superficial thrombophlebitis** is treated effectively by local measures and poses little risk of PE. Elevation of the affected extremity, range-of-motion exercises, and elastic stockings enhance venous outflow, whereas heat improves arterial microcirculation, promoting clot lysis. Antiinflammatory agents (aspirin, nonsteroidal antiinflammatory drugs) also can be administered. Antibiotics are not indicated in this setting unless there is evidence of cellulitis.
 2. **DVT of the proximal veins** requires aggressive treatment to avoid potentially lethal PE and to decrease the incidence of postphlebotic venous insufficiency. Although not all isolated calf DVTs require treatment, they must be taken seriously, because up to 30% extend proximally and are at risk for embolization. If anticoagulation is withheld in these cases, serial noninvasive examinations are indicated.
 - a. **Heparin** is the traditional acute treatment for proximal DVT. It usually is administered i.v. at approximately 100 units/kg bolus (5,000–10,000 units) followed by a constant infusion (600–1,000 units per hour) targeted to an activated PTT of 50–80 seconds. Patients who receive heparin should have platelet determinations performed at least every 3 days to detect heparin-induced thrombocytopenia.
 - b. **Anticoagulation with warfarin** typically requires 1–3 days, during which time concurrent anticoagulation with heparin is necessary. Although the dosing regimen should be individualized, 5 mg warfarin can be given the first day that heparin is started, with a subsequent 5 mg per day for 1–2 days. Dosage thereafter should be directed toward obtaining an INR of 2.0–3.0. Duration of therapy for uncomplicated DVT in a patient with no remaining risk factors (i.e., immobility) is 6 months.
 2. **Diagnosis and treatment of PE**
 - a. **Diagnosis**
 1. **Symptoms** of PE are neither sensitive nor specific. Dyspnea, pleuritic chest pain, and cough are most common, and hemoptysis is encountered occasionally. Signs of PE most commonly include tachypnea and tachycardia. Patients with massive PE may experience syncope or cardiovascular collapse. PE should be considered in any postoperative patient with unexplained dyspnea, hypoxia, tachycardia, or dysrhythmia.
 2. **Laboratory studies.** Initial evaluation of patients with suspected PE must include noninvasive arterial oxygen saturation, ECG, and CXR. Findings

that are suggestive of PE include arterial oxygen desaturation, nonspecific ST- or T-wave changes on ECG, and atelectasis, parenchymal abnormalities, or pleural effusion on CXR. Such classic signs as S₁Q₃T₃ on ECG or a prominent central pulmonary artery with decreased pulmonary vascularity (Westermark's sign) on CXR are uncommon. ABG determination is a helpful adjunctive test; a decreased PaO₂ (<80 mm Hg), an elevated alveolar-arterial oxygen gradient, or a respiratory alkalosis may support clinical suspicion. Data that are obtained from these initial studies collectively may corroborate clinical suspicion but alone are neither sensitive nor specific for PE.

3. Imaging studies

- a. **V/Q scans** should be the primary screening test in patients with suspected PE. A scan that demonstrates one or more perfusion defects in the absence of "matched" ventilation defects is abnormal and may be interpreted as high, intermediate, or low probability for PE, depending on the type and degree of abnormality. V/Q scans alone are neither sensitive nor specific for PE, however, and their interpretation may be difficult in patients with preexisting lung disease, especially COPD. Nevertheless, "high-probability" scans are 90% predictive and suffice for diagnosis of PE in the appropriate clinical setting; they should prompt treatment. Likewise, a normal scan virtually excludes PE (96%). Scans of intermediate probability require additional tests, such as noninvasive lower-extremity duplex scanning or impedance plethysmography. If these tests are positive, anticoagulation is indicated in patients at low to moderate risk for bleeding. If these tests are negative in the setting of strong clinical suspicion, pulmonary angiography should be obtained.
- b. **Spiral CT scan** shows great promise as a routine screening test for PE. Although no large-scale studies comparing it to V/Q scan have been conducted, smaller studies have shown CT scan to be at least as reliable as V/Q scan in the definitive diagnosis of a PE. Although the reliability of the test is highly operator dependent, the utility of this technique is further enhanced by its ability to detect other pulmonary pathology and its potentially greater after-hours availability.
- c. **Pulmonary angiography** is the reference standard for the diagnosis of PE, but it is an invasive test with some element of risk. Use of this test should be reserved for (1) resolution of conflicting or inconclusive clinical and noninvasive data, (2) patients with high clinical suspicion for PE and extensive preexisting pulmonary disease in whom interpretation of V/Q scans is difficult, and (3) confirmation of clinical and noninvasive data in patients who are at high risk for anticoagulation or in unstable patients being considered for thrombolytic therapy, pulmonary embolectomy, or vena caval interruption.
- d. **MR angiography** holds great promise because it has been shown to have a sensitivity and specificity that compare well with pulmonary angiography but without the ionizing radiation or radiocontrast exposure.

b. Treatment

1. **Supportive measures** include administration of oxygen to correct hypoxemia and use of intravenous fluids to maintain BP. Hypotensive patients with high clinical suspicion of PE (i.e., high-risk patient, acute right heart failure, right ventricular ischemia on ECG) require immediate transfer to an ICU, where hemodynamic monitoring and vasoactive medications may be required.
2. **Anticoagulation with intravenous heparin** should be started immediately with a target-activated PTT of 50–80 seconds. Oral warfarin can be started concurrently while heparin is continued until a therapeutic prothrombin time is achieved. Anticoagulation should continue for 6 months unless risk factors persist or DVT recurs. Some studies have shown that low-molecular-weight heparin administered subcutaneously (1 mg/kg every 12 hours) may be as effective as intravenous heparin.
3. **Thrombolytic therapy** is not indicated in the routine treatment of PE in surgical patients because the risk of hemorrhage in individuals with recent (<10 days) surgery outweighs the uncertain long-term benefits of this therapy. Surgical patients with shock secondary to angiographically proved massive PE that is refractory to anticoagulation should be considered for either transvenous embolectomy or open pulmonary embolectomy. These aggressive measures rarely are successful.
4. **Inferior vena caval filter placement** is indicated when a contraindication to anticoagulation exists (i.e., active peptic ulcer disease, bleeding tendency, patients prone to falling), when a bleeding complication occurs while receiving anticoagulation, or when a DVT or PE recurs during anticoagulation therapy.

G. Complications of diabetes

1. **DKA** may occur in any diabetic patient who is sufficiently stressed by illness or surgery. DKA patients who require surgery should be afforded every attempt at correction of metabolic abnormalities before operation, although in cases such as gangrene, surgery may be essential for treatment of the underlying cause of DKA. DKA may occur without excessive elevation of the blood glucose. Management of this disorder should emphasize volume repletion, correction of acidosis and electrolyte abnormalities, and regulation of blood glucose with insulin infusion.
 - a. **Laboratory evaluation**
 1. **Blood glucose**
 2. **CBC**
 3. **Serum electrolytes**
 4. **Serum osmolality**
 5. **ABG**
 - b. **Restoration of intravascular volume** should be initiated with isotonic (0.9%) saline or lactated Ringer's solution without glucose. Patients without cardiac disease should receive 1 L or more of fluid per hour until objective evidence of normalization of intravascular volume is demonstrated by a urine output greater than 30 mL per hour and stabilization of hemodynamics. Invasive hemodynamic monitoring may be required to guide fluid replacement in some circumstances (i.e., CHF, suspected MI, and renal failure). Maintenance fluids of 0.45% NaCl with potassium (20–40 mEq/L) can be instituted when intravascular volume has been restored. Dextrose can be added to fluids when the blood glucose is less than 400 mg/dL.
 - c. **Correction of acidosis** with bicarbonate therapy is controversial but should be considered if the blood pH is less than 7.1 or shock is present. Two ampules (88 mEq NaHCO₃) of bicarbonate can be added to 0.45% NaCl and given during the initial resuscitation.
 - d. **Potassium replacement** should be instituted immediately unless hyperkalemia with ECG changes exists. In nonoliguric patients, replacement should begin with 30–40 mEq per hour KCl for serum potassium of less than 3.0; 20–30 mEq per hour KCl for serum potassium of 3.0–4.0; and 10–20 mEq per hour KCl for potassium of more than 4.0 mEq/L.
 - e. **Blood glucose** can be controlled with 10 units insulin as an intravenous bolus followed by insulin infusion at 2–10 units per hour to a target range of 200–300 mg/dL. When the blood glucose falls to 300–400 mg/dL, 5% dextrose must be added to the intravenous fluids. Therapy is guided by hourly blood glucose determinations.
2. **Nonketotic hyperosmolar syndrome** is characterized by severe hyperglycemia and dehydration without ketoacidosis. This occurs most often in elderly non-insulin-dependent diabetes mellitus patients with renal impairment and may be precipitated by surgical illness or stress. Laboratory findings include blood glucose that exceeds 600 mg/dL and serum osmolality of more than 350 mOsm/L. Therapy is similar to that for DKA but with two notable exceptions: (1) Fluid requirements are often higher, and replacement should be with 0.45% saline, and (2) total insulin requirements are less.

H. Hypertension

1. **Definition.** Postoperative hypertension should be defined by the patient's preoperative blood pressure. Patients with chronic hypertension have a shift in their cerebral autoregulatory system that may not allow for adequate cerebral perfusion at normotensive blood pressures. A reasonable goal of therapy for acute postoperative hypertension is within 10% of the patient's normal blood pressure.
2. **Treatment.** Before using antihypertensive drugs in the treatment of postoperative hypertension, it is essential to diagnose and treat secondary causes, such as pain, hypoxemia, hypothermia, and acidosis. Acute hypertension can be managed with clonidine (0.1 mg p.o. every 6 hours), hydralazine (10–20 mg i.v. every 6 hours), labetalol (10–20 mg i.v. every 10 minutes, to a total dose of 300 mg), or a nitroprusside drip (0.25–8.00 µg/kg per minute i.v.). In situations in which the patient is unable to take oral medications and i.v. medications are not appropriate, nitroglycerin paste (0.5–2.0 in. every 6 hours) can be used.

I. Thyroid-related complications

1. **Thyroid storm** is a medical emergency that may occur abruptly in any hyperthyroid patient who is subjected to severe illness or surgical stress.
 - a. **Diagnosis** is based on symptoms that include tachycardia, hypertension, fever, and mental status changes. Differential diagnosis in the perioperative period includes malignant hyperthermia, unrecognized pheochromocytoma, sepsis, and delirium tremens. Laboratory values, including thyroid function tests, are not specific for thyroid storm, and treatment should be instituted based on clinical suspicion.
 - b. **Treatment** in an ICU requires supportive measures, including cooling blankets, acetaminophen (aspirin increases free T₄), oxygen, and intravenous fluids. Antithyroid medication must be started promptly.
 1. **PTU** blocks organification of iodide in the thyroid and inhibits peripheral conversion of T₄ to triiodothyronine (T₃). This agent is administered as a 1-g p.o. (or per nasogastric tube) load, followed by 200 mg p.o. every 4 hours.
 2. **Iodine** acutely inhibits release of T₄ from the thyroid and also transiently blocks thyroid organification of iodide. Iodine can be given either as saturated solution of potassium iodide, 5 drops p.o. every 4 hours, or sodium iodide, 500 mg i.v. every 12 hours by continuous infusion, and should be started 1 hour after administration of PTU.
 3. **Propranolol** usually is given p.o., 20–80 mg every 6 hours, but may take 1 hour to be effective, with effects that last for 8 hours. More rapid and titratable control can be achieved with esmolol given as a 500-mg/kg intravenous loading dose over 1 minute followed by 50-mg/kg maintenance infusion. Maintenance rates of up to 200 mg/kg per minute are guided by close hemodynamic monitoring. Onset of action requires less than 5 minutes, and effects typically last 10–20 minutes after discontinuing the drug.

4. **Hydrocortisone** should be given 100 mg i.v. every 8 hours during thyroid storm to prevent Addisonian crisis and to inhibit peripheral conversion of T₄ to triiodothyronine.
2. **Myxedema coma** is a medical emergency with an overall mortality that approaches 50%. This condition may be encountered in hypothyroid patients after surgery or trauma. Clinical features include depressed level of consciousness or seizures, hypothermia, hypotension, hyponatremia, and hypoglycemia. Thyroid function tests may demonstrate decreased T₄, although this is nonspecific. If myxedema coma is suspected, treatment should begin immediately and include levothyroxine, 500 mg i.v.; hydrocortisone, 100 mg i.v. every 8 hours; and supportive measures. Improvement should be seen within 24 hours of the onset of therapy. Other causes of the patient's deterioration (i.e., sepsis) should be evaluated simultaneously.
- J. **Adrenal crisis.** Adrenal crisis is a life-threatening condition that results from inadequate adrenocortical response to stress. This condition may occur in patients with underlying adrenal insufficiency after surgery or trauma and presents as unexplained hypotension and tachycardia that are often unresponsive to fluid resuscitation and vasopressors. The diagnosis is supported by hyponatremia and hyperkalemia, as serum aldosterone levels are usually depressed.
1. **Diagnosis** requires a high clinical suspicion, and an adrenocorticotropic hormone (ACTH) stimulation test should be performed, although awaiting the results of the test should not delay therapy.
 2. **Treatment** should begin immediately and includes hydrocortisone sodium succinate, 100 mg i.v. every 8 hours, and isotonic fluid replacement. Response usually is dramatic and rapid and may provide a presumptive diagnosis until the ACTH stimulation test results return. As the patient's condition stabilizes, steroids are tapered to replacement doses (prednisone, 5 mg p.o. every morning and 2.5 mg p.o. every evening). Mineralocorticoid replacement (fludrocortisone, 0.1 mg per day p.o.) should begin with resumption of oral intake. Exogenous steroid supplementation should continue until hypothalamic-pituitary-adrenal axis recovery is confirmed by ACTH stimulation testing.

8 Vascular Access, Tubes, and Drains

Brice W. McKane, David M. Hovsepian, and Surendra Shenoy

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Venous Access Devices

Venous access devices provide the means to administer infusions (e.g., fluids, colloid, medications, and blood products) to the central or peripheral venous systems. They are also used to monitor intravascular volume and to draw blood samples. **Peripheral veins** can be accessed by direct needle puncture, with placement of a polyurethane cannula for short-term fluids or medication regimens. However, because peripheral veins are fragile and venous blood flows slowly, thrombosis often occurs within a few days. Some antibiotics, such as vancomycin, and most cancer chemotherapy agents are too astringent for peripheral veins and rapidly sclerose them. They should be infused into central veins, where higher blood flow provides rapid mixing.

Central venous access devices (CVADs) are used in patients who need prolonged infusions or chemotherapy administration, those who have exhausted their peripheral access sites, and those with the need for permanent venous access. CVADs can also be used to monitor central venous pressure, which can provide a reasonable estimate of intravascular volume. **Short-term CVADs**, such as triple-lumen catheters (e.g., Arrow-Howes multilumen, Arrow International, Inc., Reading, PA), are usually inserted at the bedside, often in the acute setting. Although they can be used for extended periods of time, weeks or even longer, they are at an increased risk for infection after only a few days. Catheter care protocols dictate regular changes, some as frequent as every 72 hours, to minimize the chances of infection. Hygiene at the site of entry plays a key role in prolonging the life of any peripheral or central line. **Long-term CVADs** are usually placed through a subcutaneous tunnel or into a subcutaneous pocket to lessen the chances of contamination and therefore require insertion in a sterile setting and in a nonacute situation.

I. Sites of CVAD insertion. The subclavian and jugular veins are the most common sites for CVAD insertion. Femoral veins can be used in emergent situations, but hygiene in the inguinal crease can be problematic. For elective central access, the right internal jugular vein access should be the route of choice. Subclavian vein access provides greater patient comfort and ease of nursing care; however, the risk of morbidity from pericatheter thrombosis or central venous stenosis may outweigh these benefits in certain patients. Moreover, the subclavian veins should not be used in patients who may require dialysis at some point in the future to preserve the venous outflow for possible creation of a dialysis fistula in the arm. Venous access is gained percutaneously using the Seldinger (“over-the-guidewire”) technique. Portable ultrasound units, such as the Site Rite (Dymax Corp, Pittsburgh, PA), aid in safe and reliable insertion of a needle into the vein, avoiding injury to nearby structures. Contrast injection through a peripheral i.v. line or fluoroscopic guidance using bone landmarks can also be used for subclavian vein access. If placement is not guided by fluoroscopy, a chest X-ray should be obtained after insertion.

II. Preoperative evaluation. Before placement of a CVAD, patients should be examined for previous central venous lines, the presence of a transvenous pacemaker, and signs of central venous obstruction. Active site infection, an open wound, a tracheostomy, and tumors of the head and neck can potentially influence the type and route chosen for catheter insertion. For example, women who have undergone axillary lymph node dissection during breast cancer surgery are prone to swelling on that side because of disruption of normal lymphatic drainage. CVAD insertion, therefore, should be done on the contralateral side, unless there is no other choice, to avoid the potential additional morbidity of venous thrombosis. Review of the pertinent laboratory tests should include coagulation parameters, the hematocrit, and the platelet count. If there are fewer than 50,000 platelets/mL, any patient who receives a tunneled catheter should receive 1 unit of single-donor platelets (or 6 units of random-donor platelets) within 2 hours of CVAD insertion. The international normalized ratio (INR) should be less than 2.25. For peripheral and nontunneled lines, a lower platelet count (25,000/mL) and a higher INR (<4.25) are acceptable because these are generally smaller catheters and bleeding is readily treatable by compressive dressing.

III. Types of central venous catheters. Five main types of CVADs are available: peripherally inserted central catheters (PICCs), short-term nontunneled central lines, intermediate-term nontunneled lines, tunneled catheters, and implanted ports.

- A. **PICCs.** PICCs are usually placed into an upper arm peripheral vein (antecubital) and then threaded centrally. Trained personnel (usually nurses) can place forearm or antecubital PICCs at the bedside, either in the hospital or in the home care setting. Veins above the elbow (basilic, cephalic, or brachial veins) are more likely to be patent for PICC insertion, as they are rarely damaged from previous i.v. lines. However, access to these veins usually requires radiologic guidance. Some PICCs only reach the shoulder (e.g., Landmark Midline, Menlo Care, Inc., Menlo Park, CA), whereas others (e.g., Cook PICC, Cook, Inc., Bloomington, IN) are long enough to be positioned in the right atrium. A PICC can remain in place for up to 6 months, but if access for that length of time is expected, a larger, more durable line, such as a Hohn catheter (Bard Access Systems, Salt Lake City, UT), may be preferable. A PICC is ideal for intermediate-term care, such as a several-week course of antibiotics. With PICCs, the risks of insertion-related complications such as pneumothorax are low. However, these catheters have some inherent disadvantages by virtue of their small size, including a somewhat limited ability to perform blood draws and a significant risk for thrombophlebitis of the relatively small veins through which they course (*JVIR* 11:1309–1314, 2000). One retrospective study determined that PICCs had a significantly higher incidence of phlebitis and catheter malfunction than centrally placed catheters. These complications occurred on average at 20 days, and, in 24% of these cases, the catheter could not be used for continued therapy (*Am J Surg* 176:208–211, 1998). Bedside placement charges for PICCs are substantially less than for other CVADs, making them attractive choices for many patient care settings. PICCs have the additional advantage of being placed on the arm, where the bacterial count is considerably lower than on the chest wall (10^5 fewer bacteria/cm²).
- B. **Short-term nontunneled central lines.** A wide variety of short-term options are available, with one, two, or three lumens and a large assortment of preset lengths and diameters. The advantages to short-term catheters are low cost, bedside placement and removal, and easy exchange of damaged catheters over a guidewire. Because they are only designed for short-term use, they have the disadvantages of less durable construction, more intensive flushing and dressing regimens, and a higher risk of dislodgment. These catheters should be used for treatments that last for a few days to weeks but are probably inappropriate for therapy lasting beyond that. Examples of these devices include the **triple-lumen catheter** (e.g., Arrow-Howes multilumen), the **Quinton dialysis catheter** (now **Mahurkar**, The Kendall Company, Mansfield, MN), and the **Hemocath pheresis catheter** (MedComp, Inc., Harleysville, PA).
- C. **Intermediate-term nontunneled central lines.** These CVADs are intended for longer-term treatments, such as antibiotics for bacterial endocarditis (usually 6–8 weeks) or short-course cancer chemotherapy. They are more rugged than the short-term catheters and can be used for up to 6 months. However, if access is needed for that length of time, a permanent catheter or port might be considered. The prototypical intermediate-term catheter is the **Hohn catheter**, which is available as a single-lumen [No. 5 French (Fr.)] and double-lumen (No. 7 Fr.) system. Although they can be placed at the bedside, they are more often placed in the radiology department using image guidance (ultrasound, fluoroscopy, venography) to minimize insertion failures, avoid complications, and ensure proper tip location. If damaged, they can be replaced over a guidewire, but if catheter-related sepsis or tract infection is suspected, a new access site should be chosen. Removal, as with all other central lines, consists of releasing the anchoring sutures, withdrawing the catheter, and applying gentle compression over the venotomy site for several minutes. Although some intermediate-term catheters have a silver-impregnated gelatin cuff (Vitacuff) at the point of skin entry, these provide only temporary antibiotic protection. They do not become incorporated into the tissues as the Dacron cuffs of permanent catheters do.
- D. **Tunneled catheters.** Tunneled catheters are used when indefinite venous access is needed. Indications include the need for prolonged hemodialysis or intravenous nutritional support (total parenteral nutrition) for extended periods of time. In comparison to nontunneled CVADs, these catheters are more durable

in construction, harder to dislodge, and less likely to develop tunnel infections because the Dacron anchoring cuff elicits an inflammatory reaction and seals the tract by fibrous tissue in-growth. By 3–4 weeks, the catheter becomes permanently fixed. Tunneled catheters are typically inserted in a surgical or radiologic suite using the Seldinger technique. The use of ultrasound localization has essentially eliminated the need for cutdowns in an adult patient, but occasionally one is needed for infants or small children. More creative approaches may be needed for those patients whose upper-extremity veins have been exhausted; among these are translumbar insertion (into the inferior vena cava) and transhepatic, intercostal, and lower-extremity vein access. Examples of the more common CVADs include the Hickman, Broviac, and Groshong catheters (all made by Bard Access Systems). The **Hickman catheter** is a silicone catheter with a Dacron cuff. It is available with one, two, or three lumens, with an optional Vitacuff. The **Broviac catheter** is similar to Hickman catheters but is smaller and intended for pediatric patients. The **Groshong catheter** is distinguished by a slit valve at the tip, which seals the catheter from the bloodstream. Unlike all other venous catheters, which require heparinized saline injected into the lumen(s) daily or weekly, the slit valve of the Groshong catheter only requires flushing with normal saline. This is particularly important for those patients who have a history of heparin allergy or heparin-induced thrombocytopenia.

E. Implanted venous ports. Ports were introduced in the early 1980s. They are used primarily for chronic therapy lasting longer than 6 months when only intermittent access is needed. A reservoir is inserted into a pocket underneath the skin, which only requires monthly flushes when not in use. Common indications include chemotherapy, intermittent access in frequently hospitalized patients (e.g., those with sickle cell disease), and for those in whom cosmesis is an important issue. Most models are similar in design, containing a silicone or polyurethane catheter connected to a metal or plastic reservoir that has a dense silicone septum for percutaneous needle access. Plastic ports are MR scan compatible (e.g., **MRI port**, Bard Access systems) and are as durable as those with a metal reservoir. The most common site of implantation is the upper chest, but there are smaller ports (e.g., **PASport**, Sims-Deltec, St. Paul, MN) that are designed for upper-extremity implantation. The septa must be traversed with a special noncoring (Huber) needle to prevent deterioration of the septum from repeated puncture. Large ports are designed for up to 2,000 accesses, whereas smaller, “low-profile” ports used in children or cachectic patients have septa that last for approximately 800–1,000 punctures.

The technique used to place ports is similar to that used for placement of tunneled CVADs. The internal jugular vein is the preferred route of access. The catheter is then tunneled from a subcutaneous pocket created in the infraclavicular fossa. Next, the catheter and port are assembled *in vivo*, although many models come preassembled, in which case the catheter must be tunneled up to the venotomy site and passed through a peel-away sheath into the vein. The incisions over the venotomy and the port sites are then closed, usually with resorbable monofilament suture (e.g., Monocryl, Ethicon, Inc., Somerville, NJ) or skin adhesive (e.g., Dermabond, Ethicon, Inc.), or both.

Ports are less prone to infection than other CVADs; however, frequent accessing minimizes this advantage because the usual source of infection is not contaminated infusate or hematogenous spread of bacteria but rather contamination that occurs because of a break in technique during access. A port is more prone to infection if the access needle is left in place for extended periods or if access is very frequent. Therefore, an implanted port is not a good option for someone who requires daily access (e.g., total parenteral nutrition). The port and its septum may be difficult to localize in some patients, particularly obese individuals whose pocket may have been created along a deep plane. This situation predisposes to extravasation of infusate caused by improper access or dislodgment of the needle.

IV. Catheter maintenance. Proper care of access sites and devices is crucial to their long-term function. Short-term and tunneled CVADs should have a sterile, occlusive transparent dressing placed over them, which is changed weekly by trained personnel (or patients themselves after instruction) using meticulous technique. More frequent dressing changes may be needed for those patients who are immune suppressed or otherwise predisposed to infection. Application of topical antibiotic beneath an occlusive dressing is not recommended, as it may provide a culture medium (moisture) for bacterial growth. Catheter lumens should be flushed on a regular basis to prevent thrombosis as outlined below:

A. Venous access device care

1. **Aspirate contents of catheter lumen**
2. **Saline flush** (10 mL normal saline)
3. **Administer drug, fluid, and so forth**
4. **Saline flush** (10 mL normal saline)
5. **Heparin lock per the following** (clamp while gently infusing the last 0.5 mL):
 - a. **Hickman catheter** (2.5 mL/lumen): heparin, 100 units/mL
 - b. **Implanted port** (5.0 mL/lumen): heparin, 100 units/mL
 - c. **Hohn catheter** (2.0 mL/lumen): heparin, 100 units/mL
 - d. **Plasmapheresis or dialysis catheter** (1.5 mL/lumen): heparin, 1,000 units/mL
 - e. **PICC** (2.5 mL/lumen): heparin, 100 units/mL
 - f. **Groshong catheter** (10 mL/lumen): normal saline

V. Complications. Early complications of CVAD placement are often due to injury to anatomic structures during blind needle-sticks and passage of guidewires and catheters. The availability of ultrasound to localize the vein and the use of fluoroscopy to guide catheter placement have greatly reduced complications such as pneumothorax and catheter tip malposition. Careful advancement of the guidewire also helps to avoid triggering an arrhythmia. Rare complications include hemothorax (puncture through a central or mediastinal vein), pericardial tamponade (right atrial perforation), and air embolism introduced through the tract or peel-away sheath. Trendelenburg positioning, Valsalva’s maneuver, or both serve to increase the intrathoracic pressure and may reduce the likelihood of air embolism. Late complications include infection, thrombosis, and pericatheter venous stenosis. Kinking or external compression may also occur and can induce catheter fragmentation, a rare sequela. The best way to address most of the major complications below is to work to make certain that they are avoided in the first place.

- A. **Pneumothorax.** The incidence of pneumothorax is low, typically less than 1–4%, depending on the experience of the operator. It is the most common early complication of subclavian vein access, especially when done blindly. In this setting, pneumothorax usually does not progress to a tension pneumothorax, and additional surgical intervention in the form of a tube thoracostomy is required in less than 50% of cases. Asymptomatic pneumothorax, less than 30% of the estimated hemithoracic volume, usually requires only observation and serial chest imaging. Increasing shortness of breath, rapid progression to a large pneumothorax initially, or signs of mediastinal shift should prompt insertion of a chest tube. For hemothorax, a large-bore chest tube through a lateral thoracostomy is usually needed. For a small, apical pneumothorax, a Heimlich valve (Becton-Dickinson and Company, Franklin Lakes, NJ) can be attached to an angiocath or small-bore drainage tube (e.g., No. 8 Fr.) placed anteriorly in the pleural space.
- B. **Bleeding.** Although rare, bleeding is the most common cause of access-related mortality. Mediastinal hematoma, hemothorax, or pericardial tamponade may result from perforation of the central venous system or the heart by the guidewire, sheath, or catheter. Mediastinal hematoma is usually self-limited unless the catheter tip itself is in the mediastinum and fluid infusion is initiated. Sudden decompensation during central CVAD insertion almost always indicates bleeding due to perforation. If hemothorax or pericardial tamponade develops, urgent intervention is needed. Fortunately, since the development of softer biomaterials, perforation and catheter erosions are extremely rare.
- C. **Arterial puncture.** Anatomic variations in the relative positions of the internal jugular vein and the carotid artery are well known. Troianos et al. demonstrated that the carotid artery lies posterior to the internal jugular vein in a significant percentage of patients, predisposing it to injury if both walls of the vein are traversed (*Anesthesiology* 85:43–48, 1996). The risk of arterial puncture can be significantly reduced using real-time ultrasound-guided cannulation, especially in infants and young children (*Anesthesiology* 91:71–77, 1999). Other factors, which predispose to arterial injury, include small venous diameter and relative positioning of the head, which can influence the location of the internal jugular vein. Return of pulsatile or bright-red blood from the introducer needle indicates probable arterial cannulation. The needle should be withdrawn and sufficient pressure applied to tamponade the arteriotomy without overstimulating the carotid body and causing a vagal response. If a carotid arteriotomy is dilated and a sheath is placed, there is a significant risk for cerebrovascular events. If arterial puncture is diagnosed after the dilator has been threaded, it should be kept in place and a vascular surgeon should be consulted.
- D. **Catheter tip malposition.** Fluoroscopy during catheter placement or a chest X-ray afterward documents catheter tip position and detects complications such as pneumothorax and mediastinal hematoma. The junction between the superior vena cava and the right atrium usually lies just below the right main-stem bronchus on the anteroposterior chest radiograph and is the ideal position for the catheter tip for upper-extremity catheters. If the tip is placed too low in the right atrium, large stiff catheters may contact the myocardium and induce an arrhythmia, precipitate an atrial thrombus, or perforate the chamber and cause pericardial tamponade. Conversely, catheters that are placed too high in the superior vena cava or in the brachiocephalic veins do not function well and have an increased risk of causing stenosis, thrombosis, and perforation, particularly if the insertion is from the left side (*Chest* 101:1633–1638, 1992). Similarly, lower-extremity CVADs function better when the tip is at or above the confluence of the iliac veins. Malpositioned catheters can often be safely repositioned under fluoroscopic guidance, but occasionally replacement under sterile conditions is needed.
- E. **Air embolism.** Venous air embolism is a rare but life-threatening complication that may occur during insertion, manipulation, or removal of a central venous catheter. Patients who are short of breath or are unable to lie flat are at increased risk for air embolism. One hundred cubic centimeters of air per second can enter the central venous system through a 14-gauge needle with a pressure gradient as low as 5 cm H₂O (*Ann Surg* 179:479–481, 1974). As mentioned earlier,

air embolism can largely be avoided by taking measures to increase the intrathoracic pressure during each of the steps at risk for this during catheter insertion or removal. Placing the patient into the Trendelenburg position and having him or her perform a Valsalva's maneuver are the two most effective ways to increase venous back pressure and decrease the risk of air embolism. Occlusive dressings should be applied over the exit site after removal of long-term catheters to prevent entry of air into the venous system through a well-formed catheter tract. The location of the embolism, volume of air, and speed of entry determine the cardiovascular, pulmonary, and other potential sequelae. A large sudden volume of air may generate an "air lock" in the right heart or at the level of the pulmonary microcirculation, eliminating forward blood outflow, which can result in rapid cardiovascular collapse. The early manifestations of this include tachycardia, hypotension, hypoxia, tachypnea, and a mill-wheel heart murmur. "Paradoxical" air embolism into the arterial circulation may occur through a patent foramen ovale or other right-to-left shunt. (*Crit Care Med* 28:1621–1625, 2000). The first step in managing patients who experience venous air embolism is to stop the entry of air. The patient should immediately be placed in the Trendelenburg position with right side up (to trap the air pocket) and be given supplemental oxygen. In some circumstances, it may be possible and desirable to attempt to aspirate the trapped air through a sheath or cannula.

- F. **Catheter dysfunction.** Occlusion is the most common cause of catheter dysfunction and is usually due to pericatheter thrombus or fibrin sheath formation. This may affect not only infusion but also the ability to draw blood from the catheter, which is not uncommonly the primary reason it was placed. The following infusion technique may alleviate the occlusive problem: (1) Instill 2 mg of tissue plasminogen activator (1 mg/mL). (2) Allow a 2-hour dwell time and then aspirate the lumen. If blood returns, discard the syringe and flush the lumen with heparinized saline ([Table 8-1](#)). (3) If no blood returns, repeat steps 1 and 2 once.

Table 8-1. Summary of surgical tubes

Other causes of catheter dysfunction include tip migration, accumulation of thrombotic or chemical particulate material within a reservoir, compression, fracture, and embolization. Catheters that are placed too medially via the subclavian approach are subject to compression by the coracoclavicular ligament, which attaches the clavicle to the first rib. With repeated arm motion, fatigue of the catheter material may ensue, resulting in catheter fracture and possibly embolization of the catheter fragment. Compression is seen as a "pinch-off" sign on an upright chest X-ray (*Radiology* 177:353–356, 1990). If compression exists, the catheter should be removed and a new catheter placed in a more lateral location. Catheter fracture may be suspected when a subclavian catheter suddenly fails to function, especially if there is pain or swelling in the infraclavicular fossa or an arrhythmia develops after infusion. The detached catheter fragment should be retrieved, which is usually easily done by radiologic techniques.

- G. **Extravasation.** Injury to the catheter, displacement of a port access needle, or separation of a catheter from its reservoir may lead to extravasation. Additionally, development of a fibrin sheath around a catheter may cause infusate to flow back along the catheter and into the tissues outside the vein. Extravasation may result in pain or swelling, or both, and the severity of tissue injury depends on the amount and type of infusate. Chemotherapy agents are particularly irritating and can produce substantial soft-tissue necrosis, requiring extensive débridement and, occasionally, reconstructive surgery.

H. Infections

1. **Risk factors.** Septicemia that results from central line infection is a significant cause of morbidity and mortality. Infection rates with CVADs vary from 17% to 34% (*Arch Surg* 133:1241–1246, 1998). The initial diagnosis, site and placement technique, usage pattern, site, and catheter care influence the risks of CVAD infection more than the type of device. Immunodeficient patients and patients with sickle cell anemia are at higher risk, although catheter use is much more prevalent in these individuals, too. The use of total parenteral nutrition also poses a risk for CVAD infection. It is an ideal culture medium that requires meticulous handling. Although a greater number of lumens is a theoretical risk for more frequent catheter infections, one retrospective review did not find this to be a significant factor (*Arch Surg* 133:1241–1246, 1998).
2. **Presentation.** Catheter infections may manifest with local, regional, or systemic signs.
 - a. **Local catheter-related infections.** Exit site infections may present with erythema and drainage locally. In the absence of systemic signs and negative blood cultures, treatment consists of routine antibiotics for skin flora and more frequent dressing changes and care. Catheter removal is required in only 10% of cases.
 - b. **Regional catheter infections.** Tunnel infections or pocket infections present with erythema, induration, and tenderness along the catheter tract or over the pocket, often with a clear demarcation of the affected area. Often, there are systemic or laboratory signs of bacteremia. If the diagnosis of a regional infection is made, the CVAD usually needs to be removed, and, in some cases, the tunnel or pocket may need to be débrided. Antibiotic treatment alone is rarely successful.
 - c. **Bacteremia or sepsis.** Sepsis is the most severe manifestation of a catheter-related infection. The causative organisms are gram-positive bacteria in 87% of cases, with *Staphylococcus epidermidis* being the most commonly isolated organism. Less frequently, gram-negative bacteria (e.g., *Pseudomonas*) are involved. Occasionally, fungi (typically *Candida*) are isolated. Sixty-eight percent of catheter infections are monomicrobial, and the remaining 32% are polymicrobial (*Arch Surg* 133:1241–1246, 1998). The ultraconservative approach dictates that all CVADs in septic patients are potentially infected and should be removed. This is often not practical, especially when access is limited and there are many other potential sources. Broad-spectrum antibiotics should be initiated and continued for a minimum of 10–14 days. If the fever resolves with the initiation of antibiotics, the catheter usually can be left in place unless evidence of a tunnel or pocket infection develops. If septicemia fails to resolve after the initiation of appropriate antibiotics, catheter removal is indicated. If fungemia is present, however, the catheter must be removed immediately and the tip cultured, with initiation of antifungal treatment.
3. **Thrombosis and stenosis.** The incidence of catheter-related venous thrombosis varies in the literature from as few as 2% of patients (*ASAIO J* 41:169–172, 1995) to as many as 63.5% (*Clin Cardio*, 16:26–29, 1993). Catheter size and biomaterial, venous entry site, and tip position contribute to thrombosis, as do associated infections and hypercoagulability. The cornerstone of treatment of catheter-related central venous thrombosis is the institution of systemic anticoagulation, because the risk of pulmonary embolism from upper-extremity venous thrombosis can be as high as 36% (*Curr Opin Pulm Med* 5:222–226, 1999). In severe cases, such as those that result in superior vena cava syndrome, catheter-directed thrombolytic therapy can be considered. In the absence of infection, CVADs should be left in place in asymptomatic or minimally symptomatic patients while anticoagulation is initiated to prevent the extension of thrombus. Rarely does catheter removal alter the course of symptom resolution, because thrombus is often present not only at the catheter insertion site but also in the vein(s) peripherally, which obstructs central venous flow with or without the catheter in place. Prophylactic use of low-dose warfarin has been advocated in patients with cancer, who often have associated hypercoagulable syndromes, to prevent catheter-related venous thrombosis (*Ann Intern Med* 112:423–429, 1990). Long-term CVADs are associated with a risk of venous stenosis as a result of local irritation or chronic thrombus formation. Catheters in the subclavian or innominate veins are at higher risk of producing symptomatic central venous stenosis than are those that are placed in the right internal jugular vein.

Vascular Access for Dialysis

The success of hemodialysis depends on the rate of blood flow through the dialyzer. Flow rates of between 350 mL and 450 mL per minute are required to provide adequate dialysis within a reasonable time frame (3–4 hours). The dialysis access is a port or site in the body that provides the necessary blood flow for dialysis. The access should be easy to cannulate and last for years with minimal maintenance. The incidence of complications, such as infection, stenosis, pseudoaneurysm formation, thrombosis, and outflow deterioration, should also be low. To date, no access type fulfills all of these criteria. In 1995, the **National Kidney Foundation–Dialysis Outcomes Quality Initiative** was established with the primary goal of improving the outcome and survival of patients with end-stage renal disease (ESRD). The initiative developed evidence-based clinical practice guidelines and determined that patient outcome is dependent on early identification of impending ESRD, protection of potential vascular access sites, and early dialysis access planning. Native vein arteriovenous (AV) fistulas have been shown to have the best 5-year patency rates, with a minimum requirement for intervention. In patients who require hemodialysis, but for whom an AV fistula is not an option, a prosthetic graft can serve as an alternative. Catheters for dialysis are restricted to the short term, or they are used as a last resort in patients who have exhausted all other permanent access options (*J Am Soc Nephrol* 7:523–535, 1996).

I. Preoperative evaluation

- A. **Timing.** Ideally, any patient with significant renal insufficiency should be referred for surgical evaluation approximately 1 year before the anticipated need for dialysis. This point is reached when the creatinine clearance is less than 25 mL per minute or the serum creatinine rises above 4 mg/dL. This provides ample opportunity and time for appropriate access planning, and efforts can be instituted to ensure preservation of the native veins for later AV fistula creation.
- B. **Preservation of access sites.** It is imperative to educate ESRD patients to protect their forearm veins from venipuncture and intravenous catheters. Likewise, hospital staff should be instructed to avoid damaging these essential veins. The nondominant arm is preferred for initial access creation. For routine venipuncture in patients who are waiting for dialysis access, the veins in the dorsum of the hand with frequent site rotation should be used. It is imperative to

note that subclavian vein cannulation may induce central venous stenosis, and this complication may preclude an entire arm from use in hemodialysis access; therefore, this practice should be avoided at all cost.

- C. **History and physical examination.** History should include a detailed assessment of the arterial, venous, and cardiopulmonary status to best determine the type of vascular access. It should also include any prior lines or surgery that could cause stenosis or occlusion of the arterial or venous systems. Even previous short-term catheters can play a significant role. Transvenous pacemakers; trauma to arms, chest, or neck with vascular injury; and radiation treatments to those areas may all limit vascular access for dialysis. Inquiry should also be made regarding coagulation disorders and hypercoagulability. Evidence of early cardiac dysfunction or volume overload indicates a patient at risk for congestive heart failure following fistula creation due to increased preload. Physical examination consists of evaluation of the peripheral vascular system and overall cardiovascular status. The arterial inflow is manifest by peripheral pulses and blood pressure. An Allen test should be performed to assess the integrity of the palmar circulatory arches. The venous system is evaluated for outflow adequacy by determining the presence of edema or identifying collateral veins over the chest wall and looking for residual scars from previous central venous catheters. A tourniquet, gravity, and gentle percussion are used to distend the forearm and upper arm veins. Their patency and continuity can be addressed by palpation and detection of a thrill. Cardiac auscultation is also important, paying special attention to outward signs of heart failure.
- D. **Diagnostic studies.** Renal failure is often secondary to systemic diseases, such as diabetes and hypertension, which can damage a number of organ systems. Hyperkalemia and acidosis are the most common electrolyte abnormalities seen in ESRD patients. Therefore, preoperative testing should include evaluation of serum electrolytes and glucose. Venography is indicated for patients with a history or physical findings that are suggestive of central venous stenosis or previous complicated vascular access. Ultrasound-guided vein mapping is useful in obese patients whose venous anatomy may not be easily delineated.
- E. **Placement.** Most procedures to create an AV fistula or graft, or to place a dialysis catheter, can be done on an outpatient basis. Local anesthesia and intravenous sedation and analgesia are usually sufficient for patient comfort. Native AV fistulas require a period of weeks to months to “mature” fully to a flow state that is adequate to sustain dialysis. Synthetic grafts require a period of 4–6 weeks before the material is incorporated into the surrounding subcutaneous tissues, the inflammation subsides, and the conduit is no longer porous. Therefore, patients require an alternative means of dialysis during this period, which is often the indication for a dialysis catheter.

II. Types of hemodialysis access. The need for dialysis access varies from patient to patient and may require temporary, intermediate, or permanent access. Temporary and intermediate access are most often provided by large-bore (e.g., No. 13.5 Fr.) central venous catheters. Dialysis access, which is intended to be permanent, on the other hand, is a surgically constructed conduit that is capable of delivering the highest blood flows possible, thereby optimizing hemodialysis.

- A. **Native vein AV fistulas (primary fistulas).** Native vein fistulas anastomose an artery, usually the radial or brachial artery, to an adjacent vein. One example is Brescia-Cimino fistula, an end-to-side anastomosis between the radial artery and cephalic vein. Another is the Gratz fistula, a brachial artery-to-cephalic vein anastomosis created above the elbow. Flow of arterial blood under pressure distends the outflow vein to produce a subcutaneous conduit. Interventions, such as ligation of tributaries of outflow veins and exercises to increase the flow to the extremity, may help accelerate early fistula maturation. Mature fistulas tend to have low rates of infection, stenosis, or bleeding and provide easy accessibility and good dialysis flows. The real limitation is the condition of the arterial and venous anatomy, because diseased vessels hinder normal maturation or may preclude the creation of a fistula altogether. Of the ESRD population in the United States, approximately 30% have AV fistulas as their permanent dialysis access (*Kidney Int* 58:2178–2185, 2000).
- B. **AV graft (AV shunt).** AV grafts are biologic or synthetic conduits that connect an artery and a vein, which are tunneled under the skin. They provide a readily accessible high-flow circuit. They are the principal alternative for those patients who are not candidates for primary fistulas or whose native fistula has failed. Occasionally, they are used as a first-choice procedure when life expectancy is limited. These fistulas are typically constructed between the brachial artery and the cephalic or brachial vein in the antecubital fossa and arranged in a loop configuration in the forearm. Upper arm grafts can also be created between the brachial artery and basilic or axillary veins. When all upper-extremity sites are exhausted, attention is turned to the lower extremity. Loop grafts typically connect the superficial femoral artery and femoral or saphenous veins. The most common graft material used in the construction of AV shunts is expanded polytetrafluoroethylene. This material allows in-growth of host tissue and formation of a pseudointimal lining. This lining resists infection and self-seals after needle puncture. Repeated puncture, however, weakens the integrity of the wall, and pseudoaneurysm formation is not uncommon in older grafts.
- C. **Central venous dialysis catheters.** Nontunneled central venous catheters, such as the **Quinton catheter**, are used as access devices for patients who need short-term hemodialysis. They are useful for patients in whom acute renal failure develops or when an established access fails and corrective action must be delayed for a short period. They can also be used for patients who cannot continue peritoneal dialysis because of acute infection or recent surgery.

Tunneled catheters (e.g., **Ash split catheter**, Bard Access Systems) have cuffs that anchor them to the subcutaneous tissues. They are used when the anticipated need for dialysis is for more than several weeks. They are often placed in ESRD patients awaiting fistula maturation or in patients who have lost all options for permanent access. They can also be used for individuals who are awaiting living-donor kidney transplantation or those with prolonged acute tubular necrosis in which renal function is expected to recover. Dialysis access catheters can be inserted at the time that they are needed, are ready for immediate use, and then are removed after the acute need has passed.

Most catheters have dual lumens with staggered tips. The catheter limb with the more proximal luminal opening (color code red: “arterial”) is used for inflow to the dialyzer, and the limb with the more distal luminal opening (color code blue: “venous”) is used to return the outflow from the dialyzer to the patient. Tunneled catheters are convenient to use and often preferred by patients; however, they are incapable of providing the high flows that are needed for optimal dialysis. They are also associated with a higher risk of infection and thrombosis than native AV fistulas and can result in direct injury to the central veins, which can have a devastating impact on future access options.

III. Complications. Complications related to AV access account for 19% of all hospitalizations in hemodialysis patients, and their management requires a multidisciplinary approach (*Kidney Int* 43:1091–1096, 1993).

- A. **Stenosis.** Pseudointimal hyperplasia within a synthetic graft or neointimal hyperplasia in a native AV fistula or in the outflow vein of a graft is the most common cause of shunt dysfunction. Stenoses are the underlying cause of thrombosis in approximately 85% of grafts (*J Vasc Surg* 26:373–380, 1997). Arterial inflow lesions are less common and can lead to low flow as well as elevated recirculation, and therefore distinction from an outflow lesion is not always straightforward. Elevated venous dialysis pressure, increased recirculation, abnormal physical findings, or decreasing blood flow necessitate fistulography and treatment of any underlying lesion(s) by angioplasty or surgical revision. Surgical intervention is recommended for long-segment stenoses, a stenosis of greater than 30% after angioplasty, or a stenosis that recurs in a short period of time.
- B. **Thrombosis.** Thrombosis that occurs within a month of placement is often due to anatomic or technical factors, such as a narrow outflow vein, misplaced suture, or graft kinking. Late thrombosis is generally the result of anastomotic or outflow venous stenosis. Prolonged hypotension during and after dialysis occasionally precipitates thrombosis, as can trauma from needle puncture or excessive compression after needle removal during hemostasis. Early thrombosis of native vein fistula often culminates in permanent loss of access. In contrast, late thrombosis usually results from inflow or outflow stenosis and is generally manageable by thrombectomy and surgical revision. Thrombosis of a prosthetic AV shunt can be treated surgically or percutaneously with a variety of techniques, including pharmacotherapy (lysis), mechanical clot maceration, and suction thrombectomy, either separately or in combination. Fistulography at the time of treatment usually reveals the precipitating cause, allowing immediate intervention. Surgical thrombectomy should be followed by fistulography to look for a stenosis that may not be fully appreciable during standard balloon thrombectomy. Long-term patency of AV grafts remains problematic. By 24 months, 96% of grafts require thrombectomy, angioplasty, or surgical revision; as a result, primary graft survival rates are quite low (*Am J Kidney Dis* 36:68–74, 2000). Secondary graft survival, that is, graft survival after intervention, was 65% at 1 year and 51% at 2 years (*Am J Kidney Dis* 36:68–74, 2000).
- C. **Infection.** Infections involving AV fistulas are, fortunately, rare. The major cause of access infections and bacteremia in dialysis patients is *Staphylococcus aureus*. In addition, the type of access is a major risk factor for infection, with a relative risk in comparison to primary fistulas of 1.29 for grafts and 7.64 for catheters (*ASAIO J* 46:S6–S12, 2000). Access infections usually respond to a prolonged course (6 weeks) of antibiotic therapy. If septic embolization occurs, the fistula should be revised or taken down. Infection of prosthetic AV grafts can be a difficult problem, and antibiotics as well as surgical intervention are frequently required. Initial treatment should cover gram-positive as well as gram-negative organisms, which can be followed by culture-specific antibiotic therapy. A superficial skin infection, not involving the graft, may respond to antibiotics alone. Focal graft infections can be salvaged with resection of the infected portion of the graft, but extensive infections and those that involve newly constructed, unincorporated grafts should be managed with complete excision. Evidence of bacteremia, pseudoaneurysm formation, or local hemorrhage should prompt graft removal, with placement of a new access at a different site.
- D. **Pseudoaneurysm and aneurysm formation.** A pseudoaneurysm is a contained leak into the soft tissues adjacent to a graft. They are most commonly the result of repeated trauma to the graft in the form of needle punctures and can grow quite large and even rupture through the skin. In the absence of infection, local repair of the defect or interposition of a new segment is indicated. Pseudoaneurysms rarely occur in AV fistulas and often develop because of outflow vein stenosis that should be corrected when repairing the aneurysm.
- E. **Arterial “steal” syndrome.** When an extremity distal to an AV access shows ischemic symptoms due to preferential blood flow into the access, the AV access is said to “steal” blood. Symptomatic steal is rare, occurring in approximately 1% of patients with distal AV accesses (*Surgery* 110:664–670, 1991). Although commonly seen in the early postoperative period, ischemia can develop at any time after the construction of a dialysis access. Upstream arterial stenoses can develop later, altering blood flow dynamics and causing a steal. Patients with diabetes, a history of prior access, or atherosclerotic disease are at highest risk. Patients with mild ischemia may complain of subjective coldness and paresthesias, but loss of sensation or motion does not develop. Physical examination may

demonstrate reduced skin temperature. Mild ischemia can be managed with symptom-specific therapy and frequent physical examinations. Failure of these symptoms to improve may require surgical correction with banding or ligation. Severe ischemia is a surgical emergency, and intervention should not be delayed because of the risk of irreversible nerve injury.

- F. **Venous hypertension** presents with swelling, skin discoloration, and hyperpigmentation in the limb that bears a functioning access. The most common cause is stenosis or obstruction in the outflow vein beyond the anastomosis. In chronic cases, ulceration and pain may develop. Management consists of correction of stenosis and occasionally ligation of the veins that are immediately distal to the access.
- G. **Congestive heart failure.** AV fistulas can significantly increase venous return to the heart. As a result, cardiac output and myocardial work increase, eventually leading to cardiomegaly and congestive heart failure in some patients. If heart failure develops, operative correction involves narrowing the proximal shunt or graft with either a prosthetic band or suture ligation. Occasionally, a new access must be constructed using smaller-diameter or tapered prosthetic material.

Management of Surgical Tubes and Drains

Surgical tubes are used to evacuate fluid from or to instill fluid into a body cavity, a visceral organ, or an organ with its own internal "circulation," such as the biliary tree and urinary tract. Placement and management are specific for each type of tube. To avoid misidentification of a tube or drain, a label should be attached immediately on placement, a description should be detailed in the procedure note, and careful instructions for use should be placed among the order sheets.

I. GI tubes

- A. **Nasogastric (NG) tubes.** NG tubes cross the nares and course down the esophagus into the stomach. Placement of an NG tube is contraindicated in the presence of documented or suspected facial bone fractures or nasopharyngeal obstruction. An **orogastric (OG) tube** can be placed as an alternative in these situations. Indications for placement of an NG or OG tube include decompression of the patient's stomach and small bowel, administration of medications or enteral nutrition, and gastric lavage. NG and OG tubes can be either single or double lumen. Double-lumen (e.g., **Argyle Salem Sump**, Sherwood Medical, St. Louis, MO) tubes are ideal for decompression because one port is open to air, which prevents suction from collapsing the tube against the stomach wall. Most tubes have a radiopaque stripe that is easily discerned on abdominal radiographs, and the terminal drainage hole is marked by a break in this stripe.

These tubes can be inserted at the bedside, and in adults the length of tube that is needed to reach the stomach is approximately 40–50 cm. The patient should be positioned upright or supine with the head of the bed at a 45-degree angle and the neck flexed; a small amount of water-based lubricant (e.g., Surgilube) should be used to facilitate placement. A topical local anesthetic gel, such as viscous lidocaine, may provide some relief from discomfort during passage of the tube, and further benefit may be gained by spraying the back of the throat with an anesthetic (e.g., Cetacaine). The patient is instructed to swallow as the tube is passed across the hypopharynx, down the esophagus, and into the stomach.

Tip location can be inferred by aspiration of gastric contents or by flushing the tube with 30–60 mL air and auscultating a rumbling sound in the left upper quadrant. If placed solely for decompression, an abdominal radiograph to confirm placement is not always necessary. However, before initiation or enteral feeding via an NG or OG tube, proper position must be confirmed radiographically. NG tubes that are placed at the time of surgery should have their position confirmed by palpation before abdominal closure.

Tube maintenance consists of flushing the tube with 30 mL saline or water frequently (as often as every 4 hours) to ensure patency. If a sump tube is used, 30 mL air should be injected through the air port as well. Tubes that occlude can usually be reopened by injection of several milliliters of carbonated soda or meat tenderizer through a small-bore (e.g., 1–3 mL) syringe.

Single-lumen tubes that are used for decompression should not be placed on continuous suction because they risk injuring the gastric mucosa and, as mentioned previously, do not work efficiently. They must be connected to low intermittent suction. Double-lumen (sump) tubes can be placed on gravity, continuous, or intermittent suction as the situation warrants. If needed for feedings, a soft single-lumen **Dobbhoff**-type tube (e.g., Entriflex, Sherwood Medical) should be used instead.

NG tubes have been associated with a variety of complications, the most devastating of which is the possibility of passage through the cribriform plate into the brain in a patient with a fracture of the nasopharynx. Large-bore NG and OG tubes compromise the function of the lower esophageal sphincter and have been associated with gastroesophageal reflux, esophagitis, esophageal stricture, and bronchial aspiration of gastric contents. Therefore, if used for drainage, they should not be capped off for extended periods of time. Perforations of the esophagus, stomach, or duodenum are rare but can have serious sequelae, usually mediastinitis or peritonitis. Necrosis of the nasal skin can result from prolonged pressure or traction on the tube. NG tubes may also cause sinusitis by blocking normal sinus drainage. Releasing the tape or anchoring suture and applying gentle traction can remove an NG tube.

- B. **Nasojejunal (intestinal) tubes.** When passed through the nares, nasojejunal tubes are indicated for administration of enteral nutrition or medication or for decompression of the small intestine.

1. **Feeding** nasojejunal tubes can be placed at the bedside or with fluoroscopic or endoscopic guidance to ensure passage through the pylorus and across the ligament of Treitz. Tube placement in the authors' ICU without radiologic assistance has been met with great success when using the following protocol:
 - a. **Administer metoclopramide i.v. over 1–2 minutes approximately 10 minutes before tube insertion.**
 - b. **If the NG tube or OG tube is in place, obtain aspirate, noting color and pH; clamp the NG/OG.**
 - c. **Set the hub of the stylet securely into the main port of the feeding tube.** Close the cap on the medication port.
 - d. **Flush the tube with approximately 10 mL tap water to check for patency or leaks and to activate the hydrophilic outer coating.**
 - e. **Elevate the head of the patient's bed as tolerated.**
 - f. **As with an NG or OG tube, insert into the nostril and advance to the nasopharynx and into the esophagus.** Flexion of the patient's neck and having the patient swallow facilitate passage of the tube into the esophagus.
 - g. **Advance the tube to 55–60 cm and auscultate over the epigastric area;** attempt to aspirate the gastric contents, comparing aspirate with previous NG aspirate if available. Gastric aspirate is often bilious in appearance. The pH may range from 1 to 7 depending on the use of antacid prophylaxis.
 - h. **Continue to advance the small-bowel feeding tube slowly with a gentle touch.** Infuse approximately 60 cc of air slowly starting at 70–75 cm to help open the pylorus. Never force the tube; if resistance is met, pull back and attempt to readvance. Continue to advance to the 100-cm mark. A vacuum should be present when the syringe is withdrawn with the tube in the small bowel.
 - i. **Aspirate and check the pH and color of the output when the tube is at the 100-cm mark.** The color of the small-bowel aspirate is generally yellow with a pH of 7+.
 - j. **When the tube is in position, remove the stylet and secure with tape to the nose.**
 - k. **If the NG tube is on suction before the procedure, restart the suction.**

Maintenance consists of flushing the tube with 20–30 mL saline or water before and after each use and up to every 4 hours when not in use. Enteral feedings cannot be given by bolus but rather must be administered as a slow continuous drip to avoid osmotic fluid shifts and diarrhea. Tubes that have occluded can usually be reopened, with injection of several milliliters of carbonated soda or meat tenderizer through a small-bore (e.g., 1- to 3-mL) syringe. Nasojejunal feeding tubes are removed at the bedside by releasing the tape or suture and applying gentle traction.

2. **Long (decompressive) intestinal tubes** are used to intubate the small bowel, most often for the management of small-bowel obstruction caused by postoperative adhesions, although an NG tube often suffices for this purpose (*Ann Surg* 206:126–133,1987). The tubes can be passed manually at the time of surgery, inserted endoscopically, or placed at the bedside. Patients with partial small-bowel obstruction need to be monitored frequently for intestinal strangulation or necrosis, including signs of fever, leukocytosis, tachycardia, and abdominal tenderness. The **Gowen decompression tube** (Wilson-Cook, Winston Salem, NC) offers the advantage of a sump lumen and is placed endoscopically in the distal duodenum or proximal jejunum, obviating the need for serial radiographs during initial advancement. Cantor and Abbott tubes are usually inserted at the bedside in a manner similar to that for an NG tube. The **Cantor tube** (Rush, Inc., Duluth, GA) has a balloon at the tip that is filled with 3 mL of mercury to weight it sufficiently for advancement along the GI tract through peristalsis. This action also occasionally serves to relieve partially obstructed bowel loops but offers no advantage over NG suction in the setting of intestinal ileus. Mercury should be handled with caution, as accidental inhalation can constitute a medical emergency. Perforation of the balloon and spillage of mercury into the small intestine, however, are not usually associated with toxicity. The **Miller-Abbott tube** (Rush, Inc.) is similar but has a weighted tip, which eliminates handling of the mercury.

Once one of these tubes has been placed in the patient's stomach, it should be taped to the forehead with 10–15 cm slack to allow forward motion. As the tube

advances, it should be periodically retaped with additional laxity. Serial abdominal radiographs should document the progression of the tube. Once in position, the tube can be placed to suction drainage. Water-soluble contrast can be injected through the tube to delineate the point of obstruction. Long tubes can also be removed at the bedside. The balloon should be deflated, and 10–15 cm of length can be withdrawn every 3–4 hours until the tip is in the patient's stomach to prevent intussusception during removal.

- C. **Sengstaken-Blakemore (S-B) tubes.** The S-B tube (Rush, Inc.) is a double-balloon catheter that is used to tamponade hemorrhage from gastroesophageal varices. It can be a lifesaving measure when sclerotherapy is unavailable or has failed and can serve to temporize until a transjugular intrahepatic portosystemic shunt (TIPS) shunt or surgical intervention can be performed. It should not be used in cases of a suspected Mallory-Weiss tear because of the risk of esophageal disruption. It is also ineffective for treating bleeding gastric varices or portal gastropathy because the gastric balloon only tamponades the gastric cardia.

Before an S-B tube is placed, the patient should be transferred to an intensive care unit, be intubated, and undergo gastric lavage. The integrity of the gastric and esophageal balloons should be confirmed before insertion. The tube can be passed through the patient's nose or via the mouth into the stomach. The gastric balloon is inflated with 100 cc of air, and the tube is pulled back until resistance is met, indicating that the gastric balloon is at the gastroesophageal junction. A portable radiograph is then obtained to document proper position. An additional 100–150 cc of air is injected into the gastric balloon, and the tube is affixed with tension using a 1- or 2-lb weight via a pulley system. The esophageal balloon is then inflated to between 24 and 45 mm Hg, and this pressure should be confirmed every 4 hours. An NG tube is placed into the esophagus above the esophageal balloon to aspirate oral secretions. A completion radiograph of the abdomen is obtained. After 24 hours, the weight should be released and the balloons deflated (sooner if signs of chest or abdominal discomfort develop). The tube is removed after an additional 24 hours if there is no recurrent bleeding. Complications of S-B tubes include recurrent hemorrhage after balloon deflation, esophageal perforation, tracheobronchial aspiration, and ischemic necrosis of the esophagus.

- D. **Nasobiliary tubes.** These tubes drain the common bile duct in patients who require decompression of the biliary tree. This provides a better pressure gradient than an internal drain, an advantage if rapid decompression is desired. They are generally soft silicone (Silastic) and are manufactured with a single lumen. They are inserted using endoscopic guidance and connected to gravity drainage. The tube also provides a means to perform diagnostic cholangiography and aid in visualization of the biliary tree during transhepatic drainage. Complications of nasobiliary tubes typically result from traumatic placement or malposition. When no longer needed, they can be removed by gentle traction.
- E. **Gastrostomy (G) and gastrojejunostomy (GJ) tubes.** G tubes are indicated for decompression of the stomach or for administration of enteral nutrition or medications. They enter the stomach through the anterior abdominal wall and can be placed surgically, endoscopically, or radiologically. G tubes for feeding typically have a single lumen. GJ tubes, on the other hand, may have one or two lumens to allow feeding into the proximal small bowel with or without gastric decompression, respectively. Enteral nutrition is generally administered in a bolus fashion through G tubes, whereas GJ tubes require continuous jejunal feeding to avoid diarrhea. Replacement requires a mature tract to avoid losing access. The length of time for tract maturation varies according to which type of tube is being used. Generally, it takes at least a week or two for a mature tract to form for silicone or rubber catheters and closer to 6 weeks for those made of polyurethane. The tube is best changed over a guidewire under fluoroscopic guidance to lessen the chance of malpositioning or loss of access. G and GJ tubes can be removed in the office or at the bedside. Most G sites close spontaneously in a day or two. Only rarely do they require operative closure at the fascial level. Complications are uncommon and are related to incorrect placement, hemorrhage, peritonitis, or local cellulitis.
- F. **Jejunostomy tubes.** These tubes are usually placed surgically through an incision in the anterior abdominal wall but can be placed percutaneously under fluoroscopic guidance if there are loops of jejunum that have become relatively fixed (e.g., prior surgery). They are used to provide enteral nutrition to patients with problems or risk factors for feeding into the stomach, such as a history of gastroesophageal reflux and aspiration. The tube is secured in the jejunum with either a purse-string suture or by imbricating the serosa of the jejunum over the tube to create a Witzel tunnel. As with other enteral feeding tubes, maintenance consists of flushing the tube with 20–30 mL of water or saline before and after each use and as often as every 4–6 hours when not in use. The tube can be removed with gentle traction once a mature tract has formed. Complications are related to dislodgment before tract maturation, bowel perforation by the catheter, and technical problems that result in traumatic or incorrect placement. Peritonitis is often the result. Intestinal obstruction can result from volvulus of the bowel around the tube, inspissated feeds, intussusception, or partial occlusion of the bowel by the catheter itself, especially if it is balloon tipped.
- G. **T tubes.** These are exteriorized T-shaped tubes placed into the bile duct often through or adjacent to the cystic duct following cholecystectomy and common bile duct exploration, biliary anastomosis after liver transplantation, or some other form of biliary surgery. T tubes are usually connected to gravity drainage and can be used later to perform cholangiography or provide access for the passage of interventional instruments. If there is no evidence of biliary obstruction, the tube can be removed with gentle traction once the tract has matured (usually several weeks). Complications of T tubes include cholangitis (tube malfunction in the setting of bile duct obstruction) leakage at the site of insertion, and biloma or abscess formation. Catheter fracture during removal is a very rare complication.
- H. **Cholecystostomy tubes.** Cholecystostomy tubes are indicated for patients with acute cholecystitis who are unfit for surgery or who require a period of defervescence before definitive surgery, specifically cholecystectomy (*Surg Endosc* 10:1185-1188, 1996). They are placed into the gallbladder via the anterior abdominal wall using either operative or radiologic technique. Gravity drainage suffices for resolution of the acute inflammation. If the bile is very viscous, patency of the tube can be optimized by irrigation with 3–5 mL of sterile saline once a day or more frequently as required. Patients with acute calculous cholecystitis ultimately require cholecystectomy or percutaneous stone extraction. Acalculous cholecystitis can be successfully treated by cholecystostomy drainage alone, and the tube can be removed when cholangiography has demonstrated patency of the cystic duct (*Surg Gynecol Obstet* 176:49–54, 1993).
- I. **Cecostomy tubes.** Historically, cecostomy tubes have been used for fecal diversion in patients with a large-bowel obstruction who are poor operative candidates or occasionally for perforated appendicitis. They are also advocated for children who have large-bowel dysmotility or fecal incontinence syndromes (*Endoscopy* 31:501–503; 1999). The cecum is drained through the anterior abdominal wall using either an operative or radiologic approach. The tube is left to gravity drainage and should be irrigated with 30 mL of saline periodically to prevent obstruction. If removal is desired, the tube can be taken out after the tract has healed for 2–4 weeks with gentle traction. Common complications include obstruction when the irrigation regimen is omitted or inadequate and peritonitis from a leak at the insertion site.
- J. **Rectal tubes.** Rectal tubes are indicated in bedridden patients who have diarrhea or frequent loose stools to prevent maceration of the skin of the perineum. The tubes can be placed at the bedside and connected to gravity drainage. They are available with or without a balloon tip. The tubes should not be left in place for more than a week to prevent pressure necrosis.

II. **Thoracostomy tubes** are used to drain fluid from the pleural or mediastinal space. The use of thoracostomy tubes is described in [Chapter 36](#).

III. Urinary catheters

- A. **Suprapubic catheters.** Suprapubic catheters are used to drain urine from the bladder in patients with bladder obstruction. They are placed through the anterior abdominal wall using either an operative or radiologic technique.
- B. **Balloon-tipped (Foley) catheters.** Foley catheters are silicone (Silastic) balloon-tipped catheters that are placed into the bladder through the urethra and left to gravity drainage. They can be used for relief of urinary retention, to measure urine output accurately, and to instill irrigant into the bladder. An angled-tip Coudet catheter can be used for patients with urethral narrowing due to prostatic enlargement or stricture. Viscous lidocaine injected into the urethra before insertion makes catheter placement less uncomfortable. If a catheter becomes obstructed, it should be irrigated with sterile saline and replaced as necessary. The most common complication of patients with an indwelling bladder catheter is urinary tract infection. Urinary tract infections should be treated with appropriate antibiotics and catheter change.
- C. **Nephrostomy tubes.** Nephrostomy tubes are discussed in [Chapter 40](#).

IV. **Surgical drains.** Catheters are also used to allow drainage of fluid or pus from a body cavity. Selection of the appropriate tube or drain for a particular situation is based on the type, viscosity, and volume of fluid to be evacuated.

- A. **Open drains.** Open drains are used to establish a tract from a body cavity to the skin surface. The most common open drain is the Penrose drain, made from 0.25- to 1.0-in. soft latex rubber tubing. Placed at the time of surgery, it acts as a wick and can effectively drain pus, serum, blood, or other fluid from virtually any body cavity. Dependent positioning facilitates drainage. The drain should be secured at the skin level to prevent retraction into the wound. A gauze sponge or ostomy appliance bag can be used to contain or collect drainage. Removal of this type of drain usually consists of withdrawal in increments during convalescence.
- B. **Closed suction drains.** Closed systems drain fluid into a collection container, sparing the skin from becoming macerated. They are placed at the time of surgery or postoperatively in the setting of a continued leak, particularly at the site of anastomotic breakdown. Large silicone (Silastic) catheters, such as the multi-side hole **Jackson-Pratt drain** (Allergiance Healthcare Corp, Waukegan, IL) and fluted **Blake drain** (Ethicon, Inc.), are the most commonly used closed systems that are placed intraoperatively, and they are usually attached to bulb suction. The output from the drain and the quality of the drainage fluid can be monitored easily. Analysis of the fluid may aid in the diagnosis of anastomotic leak. Closed suction drains are usually removed when the drainage has decreased to less than 30 mL in 8 hours. If the drain was placed after creation of a GI anastomosis, it is left in place until an oral diet has been resumed.

Potential complications include erosion of the drain into a surrounding structure, fracture of the drain during removal, and infection/superinfection.

- C. **Sump drains.** Sump drains are large-caliber, multilumen tubes that provide for continuous irrigation and aspiration. They are generally placed operatively for drainage of intraabdominal spaces that might benefit from continuous postoperative irrigation, from which large volumes of drainage and large particulate material are expected (e.g., pancreatic abscess).
- D. **Abscess drainage catheters.** Typically made of polyurethane or similar thin-walled polymer plastic, these drains are inserted percutaneously using ultrasound, fluoroscopic, or CT guidance. A wide variety of sizes and configurations are available. Gravity drainage usually suffices, but they can also be connected to suction. After a period of defervescence, contrast injection is performed to delineate the size of the abscess cavity and to look for connections with the GI tract or other organs. After drainage of an infected collection, the catheter can be removed once the output has decreased to 10 mL per day or less. A flushing regimen of 3–10 mL per shift or per day (depending on catheter size and fluid viscosity) may be needed to ensure continued performance. If the output drops off sharply despite flushing, or the patient fails to improve by laboratory and physical parameters, a CT scan should be obtained to assess the situation.

9 Minimally Invasive Surgery

Robert A. Underwood and Nathaniel J. Soper

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Introduction

Minimally invasive endoscopic surgery represents a rapidly advancing and expanding field of surgical access and therapy. Minimally invasive surgical techniques are now routinely used for many operations in the abdominal cavity and retroperitoneum (laparoscopy), thorax (thoracoscopy), and joint spaces (arthroscopy). Novel applications are continually being developed, such as axillary dissection, thyroidectomy, parathyroidectomy, saphenous vein harvest for coronary artery bypass, ligation of penetrating veins associated with varicosities, celiac plexus block for pain palliation, and anterior spine surgery (*Mastery of endoscopic and laparoscopic surgery*. Philadelphia: Lippincott Williams & Wilkins, 2000).

Laparoscopy and Thoracoscopy

I. Laparoscopy

- A. **Advantages** (compared to traditional laparotomy) include less pain, fewer systemic and wound-related complications, shorter hospital stay, quicker return to full activity and work, and potential overall cost savings.
- B. **Contraindications**
 1. **Absolute contraindications** include the inability to tolerate general anesthesia and uncorrectable coagulopathy.
 2. **Relative contraindications** include the following and require an increased level of laparoscopic experience:
 - a. **Prior abdominal surgery**, which may require alternative port locations to avoid intraabdominal adhesions and laparoscopic adhesiolysis to improve exposure.
 - b. **Peritonitis**, which may limit access secondary to adhesions.
 - c. **First- and third-trimester pregnancy**. The second trimester is the optimal time, but surgery should be undertaken in consultation with the mother's obstetrician. Transvaginal fetal monitoring is recommended. Close monitoring of end-tidal carbon dioxide is essential. Open insertion of the initial port and consideration of alternative port sites may be required to prevent iatrogenic injury to the gravid uterus. Later in pregnancy it is important to position the patient in a left lateral decubitus (Sims') position to avoid inferior vena cava compression and preserve venous outflow from the lower extremities.
 - d. **Severe cardiopulmonary disease**
 - e. **Massive abdominal distention**
- C. **Limitations inherent to laparoscopy**
 1. **Video imaging** of the operative field requires development of precise video-hand-eye coordination as well as clear communication between surgeon, assistant, and camera operator.
 2. **Monocular two-dimensional imaging** requires development of artificial three-dimensional skills by the laparoscopic surgeon.
 3. **Long rigid instruments** that are placed through ports fixed at the abdominal wall restrict movement, limit dexterity, decrease tactile sensation, and increase fatigue.
 4. **Magnification** provides good visualization in tight areas but also may lead to misidentification of structures and obscured visualization with only small amounts of bleeding.
- D. **Laparoscopic procedures**
 1. **Basic**. Diagnostic and staging laparoscopy, cholecystectomy, appendectomy, and inguinal and abdominal wall herniorrhaphy.
 2. **Advanced**. Gastric bypass for obesity, operations at the esophageal hiatus for gastroesophageal reflux and hiatal hernia, segmental bowel resection, gastrojejunostomy, gastrectomy, biliary bypass, common bile duct (CBD) exploration, adrenalectomy, nephrectomy, and splenectomy.
 3. **Anecdotal experience**. Laparoscopic procedures for peptic ulcer; esophagectomy; pancreatic resection; bladder resection, augmentation, or replacement; aortobifemoral bypass; anterior spinal fixation; thyroidectomy; parathyroidectomy; saphenous vein surgery; and lymph node dissection continue to expand the field of possible applications.
- E. **Technique of laparoscopy**
 1. **Consent** for laparoscopic procedures, similar to that for open operations, requires an understanding of the risks, benefits, and alternatives to the proposed laparoscopic procedure as well as the possibility of conversion to an open procedure at the discretion of the operating surgeon. It should be emphasized that long-term outcome studies for many of the minimally invasive procedures are not yet complete.
 2. **Patient preparation**. The patient is fasted overnight, and his or her stomach is decompressed with an orogastric tube. The need for preoperative mechanical bowel preparation is dictated by the procedure. A urinary catheter is placed while in the operating room, although this can be omitted if the operation involves only the upper abdomen, is expected to be brief, and the patient voids immediately preoperatively. An H₂-receptor antagonist is administered, and compression stockings are applied to the lower extremities. Because the operating table position is manipulated intraoperatively to displace intraabdominal organs by gravity, the patient must be secured to the table and padded to prevent iatrogenic compression injuries; a beanbag is helpful. A laparotomy tray should be readily available before the procedure is started.
 3. **Pneumoperitoneum**. A working space is created in the patient's abdomen using carbon dioxide via either a closed or open technique.
 - a. **Closed technique**. A Veress needle is placed most commonly at the umbilicus through a small skin-stab incision. Two serial clicks are heard as the needle penetrates the fascia and peritoneum, respectively. Aspiration and gravity injection of sterile saline (drop test) confirms proper intraabdominal placement. The abdominal cavity is insufflated via an automatic pressure-limited insufflator to 10–15 mm Hg, and pneumoperitoneum is percussed. After insufflation, the abdominal wall is stabilized manually, and the initial trocar and port are inserted blindly and away from all critical abdominal structures.
 1. **Elevated pressure** with low flow on insufflation usually indicates placement of the Veress needle into a closed space (e.g., pre- or retroperitoneal, within the omentum).
 - a. Verify that the port's insufflation valve is open.
 - b. Remove and reinsert the Veress needle and perform tests for placement.
 - c. If the **needle position is in doubt**, use an open insertion technique.
 2. **Return of blood, cloudy or bilious fluid, or enteric contents** after Veress needle placement mandates needle repositioning.
 3. **Gas embolism is life threatening**. With right ventricular outflow obstruction, expired end-tidal carbon dioxide falls, with concomitant hypotension and a "mill-wheel" heart murmur.
 - a. Stop insufflation and release the pneumoperitoneum.
 - b. Position the patient in a steep Trendelenburg position with the right side up to float the gas bubble up toward the right ventricular apex and away from the right ventricular outflow tract.
 - c. Aspirate air from the right ventricle through a central venous catheter.
 4. **Brisk bleeding** after trocar insertion warrants emergent conversion to open laparotomy. Do not remove the trocar until gaining proximal and distal control of the injured vessel.
 - b. **Open insertion** of the initial port uses a direct cutdown through the abdominal fascia. A Hasson (wedge-shaped) port is placed under direct vision and secured to the abdominal fascia with stay sutures.
 - c. **Alternatives to carbon dioxide pneumoperitoneum** have been advocated because of the potentially deleterious effects of hypercapnia. Alternative pneumoperitoneum gases, such as nitrous oxide, helium, and argon, have been evaluated experimentally. Increased intraabdominal pressure can occur with any insufflation gas. Compression of the vena cava can cause decreased renal blood flow and diminished urinary output. External abdominal wall lift devices are available to create a working space without pneumoperitoneum.
 4. **Port placement**. Location of ports has been standardized for most procedures, and several general rules for port placement have been established.
 - a. The **camera port** should be behind and between the surgeon's two operative ports to maintain proper orientation.
 - b. **Working ports** are placed lateral to the viewing port, with the operative field ahead. All ports should be at least 8 cm apart to avoid instrument

interference with one another. Ports should be approximately 15 cm from the operative field for the site to be reached comfortably by standard 30-cm instruments and to maintain a 1:1 ratio of hand-instrument tip movement. Local anesthesia, such as bupivacaine, is injected from skin to peritoneum before skin incision and trocar placement to reduce postoperative pain.

- c. **Port site fascial incisions** that are larger than 5 mm should be closed with permanent or long-term absorbable suture to avoid the risk of incisional herniation. Obesity may render fascial closure difficult and requires lengthening of the skin incision.

F. **Suturing and knot tying** are essential skills for the surgeon to master before attempting advanced laparoscopic procedures. One should remember and anticipate suturing when considering port placement.

1. **Extracorporeal knotting techniques** are recommended for more durable tissues that can tolerate the pulling through of the excess suture material (e.g., the gastric wall or esophageal crura). Extracorporeal techniques usually require more than 32-cm suture length and use a "knot pusher" to advance the throw down the port and make the knot snug.
2. **Loop ligatures** provide preformed sliding knots on a thin stylet to allow rapid ligation of structures. A grasping instrument is placed through the loop and grasps the tissue or vessel. The loop is then closed and tightened around the pedicle.
3. **Intracorporeal suturing** is preferred for delicate tissues, such as the intestine, bile duct, or esophagus. Sutures should be 8–10 cm long. Laparoscopic instrument tying is similar to that of open surgery. A variety of specific techniques have been developed to accomplish knot tying. The surgeon should be facile with at least one technique and be able to tie a square knot without undue tissue trauma or time.

G. **Postoperative management** for most laparoscopic procedures is similar to that of open procedures, although in-hospital and recuperative time are generally shorter.

1. **Urinary catheters and nasogastric tubes** can generally be removed in the operating room. This enhances patient comfort and encourages early ambulation.
2. **Drains and postoperative antibiotics** are used as needed.
3. **Analgesics** are usually administered as needed.
4. **Diet** is generally advanced on the first postoperative day, depending on the operative procedure.
5. **Physical activity** usually is not restricted, and patients can return to work in approximately 1 week.

II. Common laparoscopic procedures

A. Diagnostic laparoscopy

1. **Abdominal pain** can be evaluated in a minimally invasive and expeditious manner using laparoscopy, especially when there are multiple confounding differential diagnoses, such as right lower quadrant discomfort in the ovulating female patient who may have gynecologic or other causes of pain mimicking appendicitis. In one study of young women with a clinical diagnosis of appendicitis, laparoscopy avoided negative laparotomy in one-third of patients (*Br J Surg* 80:922, 1993). Other indications may include fever of unknown etiology, gastrointestinal bleeding of unknown location, and generalized peritonitis of unknown etiology. Diagnostic laparoscopy is most appropriately performed in the operating room, although intensive care unit (ICU) laparoscopy is feasible and reasonable in selected situations.
2. **Intraabdominal malignancy** may be more accurately staged by laparoscopy than by computed tomography (CT) scanning. Peritoneal spread of gastrointestinal and gynecologic tumors may be missed on CT scan but readily identified by laparoscopy. In one study of patients with pancreatic malignancy that was deemed resectable by preoperative transabdominal sonography and CT scan, laparoscopy detected metastases that precluded complete resection in 35% of patients (*Am J Surg* 151:76, 1986). The indications for laparoscopic staging of intraabdominal malignancies include obtaining tissue diagnosis, such as in the case of lymphomas in which needle biopsies are often nondiagnostic, confirming nodal involvement diagnosed on noninvasive studies, obtaining peritoneal lavage cytology, and assessing the abdominal cavity before major surgical resection. Laparoscopic ultrasonography allows the surgeon to visualize structures deep to organ surfaces for a more thorough intraoperative exploration. It also improves staging of malignancies and safely allows avoidance of unnecessary laparotomy in patients with unresectable intraabdominal malignancy (*J Am Coll Surg* 185:33, 1997).
3. Use of laparoscopy in **blunt and penetrating abdominal trauma** remains controversial because of low sensitivity for injuries to retroperitoneal structures and hollow viscera. Although the appropriate role for laparoscopy in the evaluation and management of trauma patients remains to be determined, reasonable algorithms for its use have been proposed. Hemodynamically stable patients with tangential penetrating wounds have avoided laparotomy when the fascia is proved to be intact by laparoscopy (*Arch Surg* 127:109, 1992). A few centers have evaluated blunt trauma laparoscopically, using local anesthesia and intravenous sedation in the emergency department. In one multicenter clinical trial of laparoscopy in blunt trauma, the predictive value of laparoscopic examination was 92%, which compared favorably to a predictive value of 72% for peritoneal lavage (*Ann R Coll Surg Engl* 70:153, 1988).

B. **Laparoscopic cholecystectomy** has been adopted rapidly around the world and is now recognized as the "gold standard" for the treatment of gallstone disease (*Arch Surg* 127S:917, 1992; *N Engl J Med* 330:409, 1994). By 1992, an estimated 80% of cholecystectomies in the United States were performed laparoscopically, and a National Institutes of Health Consensus Development Conference stated that laparoscopic cholecystectomy "provides a safe and effective treatment for most patients with symptomatic gallstones. Indeed, it appears to have become the procedure of choice for many of these patients" (*JAMA* 269:1018, 1992). Several randomized studies have demonstrated the benefits of laparoscopic cholecystectomy compared to open cholecystectomy (*Lancet* 340:1116, 1992; *Lancet* 343:135, 1994).

1. **Indications** for laparoscopic cholecystectomy are the same as for open cholecystectomy and include symptomatic cholelithiasis, symptoms that are attributable to a diseased gallbladder (including acalculous cholecystitis), and certain asymptomatic gallstones (e.g., in immunosuppressed patients, in *Salmonella typhi* carriers, and in those with porcelain gallbladder).
2. The **camera port** is placed at the umbilicus. One 10-mm midline epigastric port and two 5-mm right subcostal ports are generally used. With the gallbladder retracted over the liver by the assistant, the gallbladder infundibulum and porta hepatis become accessible to the operating surgeon. Antegrade dissection begins at the gallbladder neck to isolate the cystic artery and cystic duct. Clear dissection of Calot's triangle with identification of only the cystic duct and cystic artery terminating directly on the gallbladder constitutes a safe approach. Obtaining this "critical view" is always required and visualized before transection of any tubular structure.
3. **Intraoperative evaluation** of the CBD is performed for choledocholithiasis or bile duct injury or when the anatomy is in question intraoperatively. Indications for laparoscopic CBD evaluation include elevated preoperative serum liver enzymes, jaundice, pancreatitis, CBD diameter greater than 6 mm, and documented choledocholithiasis.
4. **CBD stones** can be managed laparoscopically. Most small stones can be cleared from the CBD via transcystic duct exploration. Alternatively, a laparoscopic choledochotomy and CBD exploration can be performed. Relaxation of the ampullary sphincter with 1–2 mg i.v. glucagon may allow stones to be flushed or pushed into the duodenum. Larger CBD stones can be retrieved with various basket devices through a flexible choledochoscope. Postoperative endoscopic retrograde cholangiography with sphincterotomy is another option to remove CBD stones.
5. **Complications**
 - a. **Bile duct injuries** have increased to approximately 0.5% in the era of laparoscopy, compared to the accepted rate of 0.1–0.2% for open cholecystectomy (*J Am Coll Surg* 180:101, 1995).
 - b. **No structure should be cut** before all structures are identified clearly and the safe view is obtained. The right hepatic artery may be mistaken for the cystic artery. A major bile duct, including the common hepatic duct or CBD, may be misidentified as the cystic duct.
 - c. **Conversion to an open procedure** is always appropriate if the operating surgeon is in doubt that the procedure can be completed safely via the laparoscopic approach. Elective conversion is never considered a complication.

C. **Laparoscopic appendectomy** (*Surg Clin North Am* 76:469, 1996; *Surg Endosc* 9:550, 1995)

1. **Indications** for laparoscopic appendectomy remain controversial. Many surgeons are adept at performing open appendectomy through a small McBurney's incision. The laparoscopic approach allows a more thorough abdominal examination, with visualization of the adnexa and other intraabdominal organs to exclude other causes of pain that may mimic appendicitis. It may be particularly useful with young female and obese patients.
2. Operation is performed through **ports at the umbilicus and left lower quadrant**, with an optional right-sided port if required. The appendix is elevated with a grasping forceps. If inflamed, the appendiceal stump can be divided with an endoscopic stapler or loop ligated. The appendix is placed in a plastic pouch to prevent contamination while it is removed through a port.
3. **Results** of randomized prospective studies show that laparoscopic appendectomy decreases postoperative pain and expedites hospital discharge compared to open appendectomy (*Surgery* 112:497, 1992; *Br J Surg* 79:818, 1992; *Ann Surg* 219:725, 1994; *Am J Surg* 169:208, 1995). Despite the shorter hospital stay, laparoscopic appendectomy appears to offer no overall medical cost savings secondary to higher costs for laparoscopic instrumentation (*Mastery of endoscopic and laparoscopic surgery*. Philadelphia: Lippincott Williams & Wilkins, 2000).

D. **Laparoscopic inguinal herniorrhaphy** (*J Am Coll Surg* 184:325, 1997)

1. Provides a tension-free prosthetic repair of the posterior aspect of the groin floor. **Indications** are controversial because of lack of long-term follow-up, but early results show recurrence rates that are at least equal to anterior open, tension-free mesh repairs. We believe that laparoscopic herniorrhaphies are most beneficial for bilateral or recurrent inguinal hernias and in patients who require rapid return to full activity.
 - a. **Bilateral hernias** require bilateral groin incisions for traditional open repairs, which may be incapacitating for active patients, who are therefore ideal candidates for laparoscopic repair.
 - b. **Recurrent hernias** that were previously repaired conventionally may benefit from laparoscopic rerepair through properitoneal virgin tissue rather than

through the scar tissue in the anterior aspect of the abdominal wall and groin.

- c. **Patients who may not comply** with limits on lifting or who wish to return to strenuous activity rapidly also are candidates for a laparoscopic repair, which does not rely on suture strength in the early postoperative period.
2. Most surgeons have adapted a **traditional Stoppa repair** (resurfacing the inguinal floor with prosthetic mesh) by using laparoscopic techniques.
 - a. The **transabdominal preperitoneal approach** is performed from within the peritoneal cavity with carbon dioxide insufflation. The peritoneum overlying the hernia is incised to enter the preperitoneal space. After hernia reduction, the inguinal floor defect is covered with mesh that is secured with staples. The peritoneum is then closed to diminish formation of adhesions between the mesh and bowel.
 1. The “**triangle of doom**” is the anatomic area between the spermatic vessels and the vas deferens as these structures approach the internal ring. Hidden beneath the peritoneum in the triangle are the external iliac artery and vein. Extreme care must be taken during dissection within the triangle and during placement of staples.
 2. **Neuralgia** may occur if the lateral femoral cutaneous nerve is injured. Staples lateral to the internal ring or on the iliopsoas muscle should be avoided.
 3. The **extraperitoneal technique** does not violate the peritoneum and may, therefore, increase safety and reduce the incidence of intraabdominal adhesions. A balloon is inflated in the preperitoneal space to dissect the peritoneum from the posterior surface of the abdominal wall and to create a working space in the groin. Mesh is laid between the fascial defect and peritoneum. This operation can be performed under general, spinal, epidural, or local anesthesia.
 3. **Results.** Large reports with short-term follow-up have shown an acceptable early recurrence rate (2–4%) and an early return to full physical activity when the laparoscopic Stoppa technique is used (*Surg Endosc* 7:155, 1993; *Surg Endosc* 7:115, 1993). Two prospective randomized trials comparing open and laparoscopic inguinal hernia repair have confirmed these findings with the transabdominal preperitoneal approach (*Arch Surg* 129:973, 1994; *Lancet* 343:1243, 1994; *N Engl J Med* 336:1541, 1997).
- E. **Laparoscopic abdominal wall hernia repair** (*Surg Endosc* 11:1:32, 1997)
 1. Although long-term follow-up is presently lacking, early indications are that this technique provides a safe and durable repair. **Indications** for repair of abdominal wall hernias using minimally invasive techniques are generally the same as those for traditional open repair. Indications include incisional, epigastric, umbilical, spigelian, and lumbar musculofascial defects. Repair is **contraindicated** in those patients with uncorrectable bleeding diatheses, in those with hostile abdomens containing dense adhesions that do not allow safe placement of ports and establishment of pneumoperitoneum, and in those who will not hemodynamically or respiratorily tolerate CO₂ pneumoperitoneum.
 2. **Patients with multiple abdominal wall defects**, such as the “Swiss cheese”-type finding following previous incision, are often better approached laparoscopically secondary to the ability to identify all defects clearly and to tailor the repair.
 3. Common to all techniques of abdominal wall hernia repair is transabdominal intraperitoneal placement of a **mesh-type prosthesis**.
 - a. **Polyethylene mesh, polytetrafluoroethylene (PTFE) sheet**, and combinations of the two, as well as special formulations using antiadhesion barriers such as hyaluronic acid methylcellulose, are most commonly used.
 - b. **Port placement** is variable and based on hernia defect location. Standard triangulation technique for working port placement should be used with as much distance as possible from the hernia defect. Thirty- or 45-degree laparoscopes are used for optimal viewing.
 - c. **All hernia sac contents must be reduced** before mesh placement.
 - d. **The mesh is sized appropriately** to allow generous coverage of the defined defects, with a minimum of 2-cm overlap at all edges. It is then inserted into the abdominal cavity through one of the port sites and secured with transfascial sutures, endoscopic fascial staples, or a combination of the two.
- F. **Laparoscopic antireflux procedures (LARS)** (*Curr Probl Surg* 36:10:765, 1999)
 1. **Indications** for gastric funduplications are the same as those for traditional open funduplications and include symptomatic gastroesophageal reflux disease (GERD) that is refractory to medical therapy and complications of GERD (ulcer, stricture, aspiration, or Barrett’s esophagus). One randomized prospective study showed that conventional open Nissen (360-degree) fundoplication resulted in more effective treatment of complicated GERD than did medical management with H₂-receptor antagonists (*N Engl J Med* 326:786, 1992).
 2. **Medical therapy** (H₂-receptor antagonist or proton pump inhibitors) for 8–12 weeks is warranted before an antireflux operation is considered. Long-term medication with prokinetic agents may also help to control symptoms.
 3. **Preoperative evaluation** may include radiographic imaging, endoscopy, pH monitoring, and esophageal manometry or impedance testing.
 - a. **Barium swallow** may demonstrate ulceration, stricture, hiatal hernia, or foreshortened esophagus. A markedly shortened esophagus requires a concomitant esophagus-lengthening procedure.
 - b. **Endoscopy** may show esophagitis or its complications and excludes esophageal malignancy or mucosal disease of the stomach or duodenum.
 - c. A 24-hour esophageal **pH study** helps to confirm the diagnosis of GERD and its severity by recording the frequency and duration of gastroesophageal reflux and correlating symptoms with reflux events.
 - d. **Esophageal manometry** documents lower esophageal sphincter pressure and rules out dysmotility of the proximal esophagus.
 4. The **patient is placed** in a modified dorsal lithotomy and reverse Trendelenburg position. The liver is retracted superiorly away from the diaphragmatic esophageal hiatus. The gastrohepatic ligament is incised and divided, and the esophageal hiatus is dissected to free the esophagus circumferentially. The anterior and posterior vagus nerves are identified and left in apposition with the esophagus. The short gastric vessels are divided to enhance mobility of the gastric fundus. The right and left crura of the diaphragm are reapproximated to prevent herniation through an enlarged hiatus. A “short floppy” Nissen fundoplication is performed by wrapping the gastric fundus posteriorly 360 degrees around the distal esophagus. The wrap is completed with a large bougie in place in the esophagus to help prevent formation of a tight wrap with consequent narrowing of the distal esophagus and postoperative dysphagia. The Toupet procedure is a 200- to 270-degree posterior fundoplication anchored to the crura and esophagus. This laparoscopic operation is preferable if the propulsive force of the esophagus is diminished based on manometric parameters.
 5. **Laparoscopic Nissen fundoplication** has demonstrated excellent short-term results (*Surg Laparosc Endosc* 1:138, 1991; *Surg Laparosc Endosc* 2:265, 1992; *Ann Surg* 220:472, 1994). More than 5,000 cases of LARS have been described in the literature since laparoscopic Nissen fundoplication was first reported in 1991, and long-term outcome studies are under way to document efficacy compared to conventional antireflux procedures. Generally, patients are discharged within 3 days, and more than 90% report complete relief of symptoms. To keep the outcomes of LARS in perspective, they should be compared with those of open antireflux operations. Splenic injury occurs in up to 8% of open cases, and the mortality ranges up to 1.4%. Pulmonary complications occur in between 1% and 6.5% of open operations. The length of hospital stay after open fundoplication varies with the institution and the type of health care system, but several studies suggest that hospital stay and costs are both greater for open operations. One prospective randomized trial that compared laparoscopic with open Nissen fundoplication revealed similar symptomatic outcomes but a hospital stay that was one-half as long as in the laparoscopic group. Long-term follow-up for the open procedures (>10-year follow-up) demonstrates a success rate of greater than 90% for the control of GERD symptoms. Early results of LARS have been encouraging, with perioperative rates of mortality and splenic injury both nearly zero. Conversion from a laparoscopic to an open procedure is higher early in a surgeon’s experience with the operation, but in most series the conversion rate is less than 5%. The operative time averages 1–3 hours, with a clear learning curve. In series from the United States, the duration of postoperative hospitalization averages less than 3 days, with anecdotal reports of performing outpatient LARS.
 6. **Respiratory and wound complications are uncommon** because of the minimally invasive nature of the access approach with diminished perioperative pain. Perioperative complications that require reoperation occur in less than 1% of patients and include perforation of the stomach or esophagus and migration of the fundoplication into the chest as a result of perioperative retching. Postoperative foregut symptoms are relatively common after antireflux surgery, whether performed with open or laparoscopic techniques. Dysphagia is a common problem early after operation but diminishes over time such that only 5–10% of all patients experience occasional mild dysphagia, and only 1–2% require esophageal dilatation. The so-called gas-bloat syndrome is a relatively uncommon finding, and most patients with postoperative bloating also had significant bloating before the operation. Many patients with GERD develop the habit of swallowing frequently to promote esophageal clearance of acid, leading to aerophagia. With a tight fundoplication, an inability to belch with bloating may occur but often resolves over time. Recurrent reflux symptoms are present in up to 5% of patients, and wrap disruption or migration either into the chest or down onto the stomach occurs in up to 7%. Most patients are satisfied with the operation, with overall satisfaction rates ranging from 87% to 100%. The quality of life is improved after LARS. Atypical symptoms of GERD, particularly chest pain and pulmonary symptoms, are less reliably resolved after LARS than are typical symptoms. The long-term outcome after laparoscopic posterior partial funduplications does not appear to be as good as after complete funduplications. The learning curve LARS requires 30–50 operations and is reflected by decreasing operative time and diminishing rates of complications and failed funduplications.
 7. **Failed funduplications can take several forms.** Postoperative dysphagia may be due to excessively tight closure of the esophageal hiatus, a tight wrap, or tension on the wrap, causing twisting of the lower esophagus. The most common fundoplication-associated complication after LARS is migration of the wrap through the hiatus into the mediastinum. This is often heralded by new-onset substernal or epigastric pain rather than reflux symptoms. The wrap itself may disrupt or migrate down onto the stomach (the so-called slipped Nissen), but this complication was more frequent after open Nissen fundoplication than after laparoscopic fundoplication. Fundoplication disruption or migration, or both, is associated with surgical inexperience, shortcomings of surgical technique, the presence of a large hiatal hernia, and the occurrence of diaphragmatic stressors, including perioperative vomiting. To prevent fundoplication failure, the surgeon must therefore carefully select patients, perform the operation with specific attention to full mobilization of the fundus and esophagus, and close the crura around the esophagus adequately. The management of fundoplication failures after LARS is complex. The presence of fundoplication failure is usually confirmed by a barium swallow, and this study also allows an assessment of the location of the gastroesophageal junction in relation to the esophageal hiatus. Occasionally, after multiple operations or in the presence of severe periesophageal mediastinal fibrosis, esophagectomy may be the best alternative

for a patient with failed fundoplication.

8. Finally, some patients have **postoperative symptoms** and are unhappy with the outcome despite an anatomically intact fundoplication. Occasional patients have reflux despite an intact fundoplication, and many patients have foregut symptoms that are independent of recurrent gastroesophageal reflux.

G. Laparoscopic colon resection

1. **Laparoscopic bowel resections** have been performed to treat malignant and benign disease, including diverticulitis, volvulus, rectal prolapse, and inflammatory bowel disease.
2. Using **laparoscopic techniques**, the colon is mobilized and the mesentery divided. The segment of colon can be exteriorized through a small incision (assisted by laparoscopy), resected, and anastomosed and then placed back into the abdominal cavity. Alternatively, resection and anastomosis can be performed totally intracorporeally.
3. **Patients are discharged** from the hospital when tolerating a regular diet well, typically within 4 days, a period that is generally shorter than that after open operation. However, longer operative times and the expense of the laparoscopic equipment offset potential savings resulting from shorter hospitalization.
4. **Malignancy.** Concern with adequacy of the *en bloc* resection when performed for malignancy and the possibility of tumor metastases following laparoscopic-assisted surgery have raised concern that this approach may not be appropriate for curable colon cancer and form the basis for the current recommendation that laparoscopic colectomy for malignancy be performed only within an approved study protocol by experienced laparoscopic surgeons. Multiinstitutional randomized clinical trials are currently under way comparing laparoscopic with open colectomy for resectable colon cancers.

H. Laparoscopic nephrectomy (*N Engl J Med* 324:1370, 1991; *J Urol* 154:479, 1995)

1. **Indications** include benign and malignant disease of the kidney.
2. **Retrograde catheters** are placed fluoroscopically in the ureters to avoid accidental injury. Five 11-mm ports are inserted in the flank. The ureter and kidney are dissected, and the ureter, renal artery, and renal vein are clipped and divided. The kidney is placed in an entrapment bag and is morcellated before it is removed through a port site.
3. The **mean hospital stay** is 4 days. Return to work and normal activities occurs within 3 weeks.

I. Laparoscopic adrenalectomy (*J Am Coll Surg* 183:1, 1996)

1. **Most laparoscopic adrenalectomies are performed** for small, benign adrenal tumors. Cushing's syndrome, pheochromocytoma, and asymptomatic adrenal lesions ("incidentalomas") have all been treated via laparoscopy. Large adrenal tumors (>6 cm) with a suspicion of malignancy are contraindications to the laparoscopic approach.
2. **Operation** is generally performed in the lateral decubitus position through four ports in the flank (two 5 mm and two 10/12 mm). The upper pole of the kidney is exposed, and the perinephric fat is dissected to expose the adrenal gland. The adrenal vessels are clipped and divided, and the organ is removed in an entrapment sac.
3. The **laparoscopic approach** results in decreased postoperative pain and quicker recovery. Patients resume eating the next day and are discharged on average by the third postoperative day.

J. Laparoscopic splenectomy (*Surg Endosc* 6:183, 1992; *Can J Surg* 41:28, 1998)

1. **Laparoscopic splenectomy has been performed for a variety of indications**, including immune thrombocytopenia, hemolytic anemia, lymphoma, and splenic artery aneurysm. Marked splenomegaly and uncontrollable bleeding diatheses are contraindications to laparoscopic splenectomy.
2. **Operation is performed** from a flank approach with the patient in a modified right lateral decubitus position. Through four 10-mm ports, the splenic flexure of the colon is mobilized, and the splenorenal ligament is divided. The splenic hilar vessels and short gastric vessels are isolated, ligated (via clips, staples, or sutures), and divided. Finally, the spleen is morcellated in an entrapment sac before the specimen is removed from the abdominal cavity.
3. **On average, patients resume a regular diet and are discharged from the hospital by the second postoperative day.** Patients normally return to work within 2 weeks of operation.

III. Thoracoscopy

A. Diagnostic thoracoscopy (*Surg Gynecol Obstet* 34:289, 1922)

1. **Video-assisted thoracoscopic surgery (VATS)** is performed in patients after thoracentesis and percutaneous pleural biopsy have failed to provide a diagnosis of suspected pleural disease. VATS frequently is used to diagnose malignancy in a solitary peripheral nodule. It is contraindicated in patients with extensive intrapleural adhesions or in those who are unable to tolerate single-lung ventilation.
2. **VATS is approximately 95% accurate** for diagnosis of pleural disease.

B. Therapeutic thoracoscopy

1. **VATS has been performed** for peripheral lung biopsy, closure of leaking blebs, parietal pleurodesis, pericardiectomy, and excision of mediastinal cysts. Fewer lobectomies, pneumonectomies, and esophagectomies have been performed owing to concern for adequacy of complete tumor resection, and therefore they should be considered investigational procedures at this time.
 - a. **Absolute contraindications** include extensive intrapleural adhesions or the inability to tolerate single-lung anesthesia.
 - b. **Relative contraindications** include previous thoracotomy, tumor involvement of the hilar vessels, and previous chemotherapy or radiotherapy for lung or esophageal tumors.
2. The **patient is placed** in the lateral decubitus or semioblique position. Thoracoscopy requires selective intubation to allow collapse of the ipsilateral lung and to create a working space within the thorax. For most procedures, three incisions are required. The thoracoscope is placed through a port in the seventh or eighth intercostal space in the midaxillary line. Working ports for instruments generally are at the fourth or fifth intercostal space in the anterior axillary line and posteriorly near the border of the scapula. The endoscopic stapler, electrocautery, or laser can be used for resection. A chest tube is generally placed through one of the port sites.
3. **Complications** include hemorrhage, perforation of the diaphragm, air emboli, prolonged air leak, and tension pneumothorax.
4. **Postoperative thoracoscopy management**
 - a. **Chest X-ray** is checked for residual air or fluid.
 - b. **Chest tubes**, if any, are usually removed in 1–2 days.
 - c. **Analgesia** is provided by patient-controlled anesthesia or orally administered medication as needed.
 - d. **Diet** is usually advanced by postoperative day 1.
 - e. **Physical activity** is as tolerated with a chest tube. Depending on the procedure and diagnosis, patients can return to work in approximately 1 week.

10 Wound Healing and Care

Sean McLean
Bruce Kraemer

Introduction

Acute Wound Healing

I. Physiology of the acute wound

II. Growth factors and cytokines

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Chronic Wound Healing

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Wound Care

I. Open wound care options

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Introduction

Wound healing is the normal body response to injury, various disease states, or aging. The goal of modern wound care is to promote the timely restoration of the body to a previous state of normal form and function. Wound healing is often classified as “normal” (acute wound healing) or “abnormal” (chronic wound healing). *Acute wound healing* is the normal, orderly process that occurs after a typical injury and requires minimal practitioner intervention. *Chronic wound healing* often necessitates a variety of interventions to correct and shift the healing process toward a more normal state of wound healing. An understanding of the basic processes as found in normal or acute wound healing allows for a better understanding of how alterations of these processes lead to abnormal wound healing and how interventions can result in restoration of the normal healing processes.

Acute Wound Healing

I. Physiology of the acute wound. The disruption of the integrity of tissues, whether surgical or traumatic, stimulates a series of events that attempt to restore the injured tissue to a normal state. The process of wound healing occurs in an orderly fashion and strikes a fine balance between repair and regeneration of tissue. Normal wound healing is affected by the extent of injury, tissue type, and the existence of comorbid conditions within the patient. The steps of wound healing can be grouped into early, intermediate, and late events.

- A. **Early wound healing** involves the establishment of hemostasis (day 1 of wounding) and the onset of inflammation (days 1–4 postwounding). With the onset of injury, blood vessels are disrupted, with subsequent hemorrhage. Severed blood vessels with any smooth muscles in their walls immediately constrict, and within minutes the coagulation cascade is initiated and produces the end-product fibrin, which plays an important role in the formation of clot and in wound healing. Fibrin, with its associated glycoproteins vitronectin and fibronectin, forms the initial matrix for early wound healing. In later phases of wound healing, the fibrin-formed matrix facilitates cell attachment and migration. It also serves as a reservoir for cytokines. With the production of fibrin, platelets are activated, and they bind to and aggregate on the fibrin lattice to form the clot that is necessary to achieve hemostasis. After aggregation, platelets release various cytokines, such as transforming growth factor (TGF)-beta, platelet-derived growth factor (PDGF), and basic fibroblast growth factor (bFGF), that will play a role in later phases of wound healing.

The **inflammatory phase** (days 1–4 postwounding) is the initial response of the body to injury. It is recognized at the skin level by the cardinal signs of inflammation—*rubor* (redness), *calor* (heat), *tumor* (swelling), and *dolor* (pain), which result from changes in the microcirculation. As hemostasis is established, leukocytes begin to migrate out of the intravascular space through gaps between endothelial cells. They migrate into the extravascular space and bind to the provisional wound matrix. The polymorphonuclear leukocyte (PMN) is the dominant inflammatory cell in the wound for the first 24–48 hours. PMNs phagocytize bacteria, foreign material, and damaged tissue. They also release cytokines that stimulate fibroblast and keratinocytes. Although the PMNs are not essential for wound healing, their absence can lead to a prolonged inflammatory phase, which delays or limits the later phases of healing.

The inflammatory phase progresses with the infiltration of **monocytes** into the wound. Monocytes migrate into the extravascular space through capillaries. Under the influence of cytokines and fibronectin, monocytes differentiate into macrophages. Macrophages are activated by several cytokines, including interleukin-2, and their migration is stimulated by components of the extracellular matrix (collagen, elastin, fibronectin), TGF-beta, and components of the complement cascade.

Macrophages are essential for normal healing because of their important role in the coordination of the healing process. They function to phagocytize bacteria, phagocytize damaged tissue, secrete enzymes for the degradation of tissue and extracellular matrix, and release cytokines for inflammatory cell recruitment and fibroblast proliferation. They are the predominant cell in the wound environment at 48–72 hours and remain within the wound for several days.

The inflammatory phase is a well-defined length of time in primarily closed wounds (approximately 4 days), but it continues indefinitely to the endpoint of complete epithelialization in wounds that close by secondary or tertiary intention. Foreign material or bacteria can change a normal healing wound to one of chronic inflammation. Persistent inflammation can be deleterious and hamper the healing process.

- B. **Intermediate wound-healing** events involve mesenchymal cell migration and proliferation, angiogenesis, and epithelialization. Two to four days after wounding, chemotactic cytokines (PDGF, TGF-beta) influence fibroblasts to migrate into the wound from undamaged tissue. Movement of cells occurs on the extracellular matrix, consisting of fibrin, fibronectin, and vitronectin. Cell migration is facilitated by matrix with a high level of hyaluronic acid and by the secretion of proteolytic enzymes to clear a path. Once present in the wound, fibroblasts begin to proliferate under the influence of many cytokines, including PDGF, TGF-beta, and insulinlike growth factor.

While the wound is infiltrated by mesenchymal cells, **angiogenesis** takes place to restore the vasculature that has been disrupted by the wound. It is stimulated by factors in the wound such as high lactate levels, acidic pH, and low oxygen tension. Cytokines and angiogenic factors such as TGF-alpha, TGF-beta, vascular endothelial growth factor, and FGF-2 also play an important signaling role in angiogenesis. These various signals activate endothelial cells to sprout from capillaries to form a vascular network. Through the modification of surrounding matrix and tube formation, the vascular system matures, resulting in fewer and larger vessels.

Epithelialization is the third critical aspect of intermediate wound-healing events. It restores the barrier between the wound and the external environment. Epithelialization of wounds occurs via the migration of epithelial cells from the edges of the wound and from remaining epidermal skin appendages. Migration and proliferation of epithelial cells is stimulated by epidermal growth factor (EGF). Migration of epithelial cells occurs at the rate of 1 mm per day in clean and open wounds. Primarily closed wounds have a contiguous epithelial layer at 24–48 hours.

- C. **Late wound-healing** events involve the deposition of collagen, other matrix proteins, and wound contraction. After being present in the wound, the primary function of the fibroblast becomes that of protein synthesis. Fibroblasts produce several proteins that are components of the extracellular matrix, including collagen, fibronectin, and proteoglycans. The cytokines TGF-beta, PDGF, FGF, and EGF stimulate synthetic activity that begins 3–5 days after wounding. Glucocorticoids inhibit protein production by fibroblasts.

Collagen is the main protein that is secreted by fibroblast. It provides strength and structure and facilitates cell motility in the wound. Collagen is synthesized at an accelerated rate for 2–4 weeks, greatly contributing to the tensile strength of the wound. Oxygen, vitamin C, alpha-ketoglutarate, and iron are important cofactors

for the cross-linkage of collagen fibers. If they are not present, wound healing may be poor.

Wound contraction is another aspect of late wound healing. *Wound contraction* is a decrease in the size of a wound without an increase in the number of tissue elements that are present. It involves movement of the wound edge toward the center of the wound. Wound healing is thought to be mediated by the myofibroblast, so named for its fibroblast and smooth-muscle cell-like properties. The cytokines TGF-beta and PDGF mediate contraction. It is differentiated from contracture, which is the pathologic and movement-limiting result of prolonged wound contraction across a joint. Wound contraction begins 4–5 days after wounding and continues for 12–15 days or longer if the wound remains open. Topical interferon-gamma inhibits wound contraction, and splints and topical dressings delay it.

- D. The **final wound-healing** event is scar remodeling. It begins at approximately 21 days after wounding. At the outset of scar remodeling, collagen synthesis is downregulated and the cellularity of the wound decreases. During scar remodeling, collagen is broken down and replaced by new collagen that is denser and organized along the lines of stress. As the wound matures, type III collagen is replaced with type I collagen. The number of cross-links between fibers also increases during remodeling. An increase in the number of collagen cross-links correlates directly with an increase in the bursting strength of the wound. By 6 months, the wound reaches 80% of the bursting strength of unwounded tissue. It is important to note that a well-healed wound never achieves the strength of unwounded tissue. This process reaches a plateau at 12–18 months, but it may last indefinitely.

II. Growth factors and cytokines are polypeptides that function to regulate the wound-healing response. They are produced by many cell types, including macrophages, platelets, and fibroblasts. They can function in an autocrine, paracrine, or endocrine fashion to stimulate cell growth, migration, proliferation, or protein production. The cloning and recombinant production of growth factors have led to the intense investigation of exogenous growth factors in the therapy of wounds.

- A. **PDGF** is a powerful chemoattractant and mitogen for fibroblasts, smooth-muscle cells, and inflammatory cells. It is produced by many cell types, including platelets, macrophages, and endothelial cells, and plays a role in many phases of wound healing. Recombinant PDGF has been shown experimentally to improve the breaking strength of incisional wounds and accelerate the normal sequences of wound healing.
- B. **Vascular endothelial growth factor (VEGF)** is a potent mitogen for endothelial cells and promotes angiogenesis.
- C. **TGF-alpha** is similar structurally to EGF. Produced primarily by keratinocytes and macrophages, TGF-alpha is a mitogen for keratinocytes and fibroblasts and serves as a proangiogenic factor.
- D. **TGF-beta** has five isoforms that may play many different roles. Members of the TGF-beta family can be produced by many cell types, including platelets, macrophages, fibroblasts, keratinocytes, and lymphocytes. TGF-beta stimulates the growth of fibroblasts and may stimulate or inhibit the growth of many other cell types. They also serve as chemoattractants for inflammatory cells and stimulate cells to produce extracellular matrix.
- E. **EGF** is produced by platelets early in wound healing. It promotes epidermal regeneration and epithelialization and serves as a chemoattractant for fibroblasts. Investigative efforts have shown that EGF may expedite wound healing.
- F. **FGF** represents a family of polypeptides. They are produced by fibroblasts, endothelial cells, smooth-muscle cells, and chondrocytes. FGFs may be sequestered in the extracellular matrix, where they are released, with degradation of the matrix. FGFs function as mitogens for cells of mesodermal or neuroectodermal origin and as angiogenic factors through the stimulation of endothelial cells.
- G. **Keratinocyte growth factor** is related to the FGF family. It is released by fibroblasts to stimulate keratinocytes.
- H. **Insulinlike growth factor** has two isoforms. It is released by platelets and stimulates an increase in collagen production by fibroblasts.

III. Timing of wound healing

- A. **Primary intention** occurs when the wound is closed by direct approximation of the wound margins or by placement of a graft or flap. Direct approximation of the edges of a wound provides the optimal treatment provided that the wound is clean, the closure can be done without undue tension, and the closure can occur in a timely fashion. Wounds that are less than 6 hours old are considered in the “golden period” and are less likely to develop into a chronic wound-healing state. At times, rearrangement of tissues is required to achieve this goal. Directly approximated wounds typically heal as outlined above provided that there is adequate perfusion of the tissues and no infection. Primary intention also describes the healing of wounds created in the operating room that are closed at the end of the operative period.
- B. **Secondary intention**, or spontaneous healing, occurs when a wound is left open and is allowed to close by epithelialization and contraction. Contraction is a myofibroblast (modified fibroblasts that have smooth-muscle cell-like contractile properties)–mediated process that aids in wound closure by decreasing the circumference of the wound. This method is commonly used in the management of wounds that are treated beyond the “golden period” (initial 6 hours) or the contaminated infected wounds with a bacterial count greater than 10^5 /g tissue. These wounds are characterized by prolonged inflammatory and proliferative phases of healing that continue until the wound has completely epithelialized or been closed by other means.
- C. **Tertiary intention, or delayed primary closure**, is a useful option for managing wounds that are too heavily contaminated for primary closure but appear clean and well vascularized after 4–5 days of open observation so that the cutaneous edges can be approximated at that time. During this period, the normally low PaO_2 at the wound surface rises, and the inflammatory process in the wound bed leads to a minimized bacterial concentration, thus allowing a safer closure than could be achieved with primary closure and a more rapid closure than could be achieved with secondary wound healing.

Chronic Wound Healing

I. Physiology of the chronic wound. A chronic wound is a wound that fails to heal in a reasonable amount of time as it relates to the etiology, location, and tissue type of the wound. Prolonged or incomplete healing occurs as a result of a disruption in the normal process of acute wound healing that leads to a poor anatomic and functional result. Most chronic wounds are slowed or arrested in the inflammatory or proliferative phases of healing and have marked increased levels of matrix metalloproteinases, which bind up or degrade the various cytokines and growth factors at the wound surface. Most often, there are definable reasons for the failure of these wounds to heal, and treatment of these causes, along with maximal medical management of the patient's medical problems, leads to a restoration of more normal healing processes. Therefore, treating a patient with a chronic wound involves investigation into the cause(s) of delayed healing and improving the intrinsic (within the wound itself) as well as extrinsic (systemic) patient factors that lead to poor wound healing.

- A. **Intrinsic or local factors** are abnormalities within the wound that prevent normal wound healing. These factors include the presence of a foreign body, necrotic tissue, repetitive trauma, hypoxia/ischemia, venous insufficiency, infection, growth factor deficit, excessive matrix protein degradation, and radiation. Factors that can be controlled by the surgeon include the blood supply to the wound, temperature of the wound environment, presence or absence of infection, hematoma or seroma, amount of local tissue trauma, and the technique and suture material that are used to close the wound. It should be noted that the technique and suture material are important only after other local factors have been addressed.
1. **Ischemia and hypoxia** are common contributing causes to nonhealing wounds. Atherosclerosis or local damage to vessels in the form of trauma or vasculitis cause ischemia and subsequent hypoxia in the wound. Hypoxia leads to impaired collagen synthesis, prevents fibroblast migration, and increases the susceptibility of the wound to infection. Essential to normal wound healing, molecular oxygen is needed for the hydroxylation reaction that cross-links collagen fibers. Molecular oxygen is also important in the host defense's ability to kill pathogens. Sick cell anemia may lead to vascular occlusion, with subsequent ischemia and tissue ulceration.
 2. **Infection** in the wound delays healing. Infection is considered to be present when the bacterial count of a quantitative tissue culture is greater than 10^5 organisms/g tissue. The critical factors in determining the susceptibility to infection are concentration of organisms, virulence, and host resistance. The host resistance can be impaired by diabetes, malnutrition, malignancy, steroids, or other immunosuppressive therapy. If allowed to persist, wound infections lead to increased tissue destruction and alter the effect of cytokines on wound healing. Clinical signs of infection are pyrexia, erythema, swelling, and purulence. Treatment must involve drainage, local débridement, and systemic antibiotics.
 3. The **presence of foreign bodies and necrotic tissue** can contribute to delayed wound healing. This occurs by prolonging the inflammatory phase of wound healing until the inciting factors are removed. Such factors also predispose a wound to infection. The combined presence of infection and a foreign body or devitalized tissue within the wound necessitates the removal of the latter by débridement before eradication of the infection can be achieved. Hematomas, seromas, devascularized bone and sequestrum are all factors that can increase susceptibility of a wound to infection.
 4. **Chronic venous insufficiency** leads to persistent venous hypertension and chronic edema in the lower extremities. These factors in turn lead to pericapillary fibrosis, tissue ischemia, and the liberation of superoxide radicals that are thought to result in delayed wound healing in extremities with chronic venous insufficiency.
 5. **Ionizing radiation** to the wound leads to abnormal wound healing. Early manifestations include erythema, edema, and hyperpigmentation, but the chronic effects of tissue ischemia, atrophy, and fibrosis cause radiation-exposed wounds to enter a chronic course.
 6. **Edema–acute swelling**, especially around joints, can lead to skin breakdown and full-thickness skin loss. Chronic swelling often leads to fibrofatty tissue deposition in underlying skin, which develops verrucous changes and at times irregular crevices and folds. Such skin is prone to breakdown and to development of infection. Infection leads to further lymphatic blockage and obliteration, and the problem gets chronically worse. Patients need to adopt an

aggressive program of limb elevation, external compression, and medical management of the edema.

7. The **microenvironment of the chronic wound** has been shown to be different from that of the acute wound in investigational settings. Studies have implicated a decrease in the endogenous levels of certain growth factors in wounds with impaired healing. Other investigators have established that an imbalance in the synthesis and degradation of extracellular matrix proteins is central to the establishment and maintenance of chronic wounds. This occurs through inadequate synthesis of extracellular matrix proteins, increased degradative enzymes, decreased regulation of degradative enzymes, or a combination of these causes.
- B. **Extrinsic or systemic factors** also contribute to abnormal wound healing. These factors are primarily linked to the underlying general health of the patient.
1. **Malnutrition** alters normal healing through the indirect and the direct effects of vitamin and mineral deficiency. An example is the patient with clinical or subclinical scurvy (vitamin C deficiency); these individuals produce inadequately hydroxylated collagen, thereby significantly weakening the healed wound.
 2. **Diabetes mellitus** is believed to affect healing adversely at every level and in every phase of the healing process. The lack of insulin (and its poorly understood trophic effects on healing tissues), hyperglycemia (by adversely affecting the migratory and phagocytic functions of inflammatory cells and proliferation of fibroblasts and endothelial cells), neuropathy, and the vascular disease that occurs in diabetic patients each contribute to poor healing.
 3. **Steroids and antineoplastic drugs** can diminish markedly the speed and quality of the healing process. The exact effects of steroids are not yet understood. Vitamin A, by poorly understood mechanisms, seems to cause a partial reversal of the detrimental effect of steroids on healing. Chemotherapeutic agents alter wound healing by decreasing mesenchymal cell proliferation and inducing a leukopenic state that reduces the inflammatory cells available for wound healing. Immunosuppression from AIDS or other diseases may also affect various phases of wound healing.
 4. **Smoking** contributes to delayed wound healing by causing cutaneous vasoconstriction, decreasing the oxygen-carrying capacity of hemoglobin, and contributing to atherosclerosis.
 5. **Collagen vascular diseases** often are accompanied by a vasculitic component, which needs to be controlled before healing can begin. The medicines that are used to treat collagen vascular diseases may impair cell migration and collagen deposition. Adjustment of dosage may lead to improved wound healing.
 6. **Cleansing agents** [such as chlorhexidine gluconate (Hibiclens) or povidone-iodine (Betadine)] **or chemicals** may impair wound healing by effecting cell migration.
 7. **Repetitive trauma**, intentional or otherwise, from shearing or pressure forces often leads to a failure in healing. Wound areas over pressure points often require stabilization of the overlying skin envelope with external taping or splinting.
 8. **Renal disease, liver disease—uremic patients** often heal their wounds more slowly.
 9. **Hematopoietic disorders.** Sickle cell disease with its high incidence of ankle wounds and leukoclastic and granulomatous processes, as well as mycoses fungoides, are conditions that are associated with poorly healing wounds. Maximal medical treatment for the underlying disorder is needed to effect meaningful healing.

II. Evaluation and management of the chronic wound

- A. **History and physical examination.** Evaluation and management of a chronic wound must begin with a thorough history and physical examination.
1. During the **history**, one must establish whether the wound is new or recurrent, how long it has been present, how it started, how quickly it developed, and whether it is improving or worsening. The patient must be questioned about existing comorbidities, with a focus on potential causes for immunosuppression (HIV, steroids, chemotherapy), undiagnosed diabetes mellitus, peripheral vascular disease, coronary artery disease, rheumatologic disorders, and radiation exposure. Smoking and alcohol use must also be documented.
 2. The **physical examination** must evaluate the size, depth, location, and tissues that are involved by the wound. Signs of infection (erythema, purulence, tenderness, warmth, and swelling) must also be evaluated, and a good pulse examination for extremity wounds must be performed. Appropriate serum chemistries may aid with the diagnosis of diabetes and renal or hepatic dysfunction. A complete blood cell count may indicate infection with elevated white cell count or white cell abnormalities. Radiography can be performed to determine underlying bony pathology. Doppler evaluation of the extremities should be carried out for suspected arterial insufficiency.
- B. **Management** of the chronic wound must focus on the optimization of host and local factors.
1. **Adequate nutrition** is necessary for appropriate wound healing. Sufficient calories, protein, vitamins, minerals, and water are necessary to prevent and aid in the healing of chronic wounds. Patients with severe malnutrition or whose gastrointestinal tract cannot be used should be placed on parenteral nutritional support.
 2. **Underlying factors** that affect wound healing—such as chemotherapy, steroids, alcohol consumption, cigarette smoking, and blood glucose levels—must be modified as necessary to aid in the wound-healing process.
 3. **Effective local wound care** is essential to the resolution of a chronic wound. Eradication of infection, aggressive débridement, and drainage of abscesses from the wound are important steps in local control.
 4. **Antibiotics.** Systemic antibiotics should be administered to treat active infection such as cellulitis. Topical antibiotics to the wound are effective in lowering the bacterial count.
 5. **Proper dressings** are an essential aspect of local care by helping to provide the appropriate environment for healing. Frequent wet-to-dry dressings are used when infection and drainage predominate. With the development of healthy granulation tissue, dressings should provide adequate protection and moisture to facilitate healing.
 6. **Edema control** is often necessary for wounds of the lower extremity due to venous insufficiency. Elevation and wrapping in an elastic bandage reduce edema and venous hypertension. Unna's boot, Jobst compression garments, and pneumatic compression devices can also be used.
- C. **Surgical therapy** may be necessary to aid in the healing of a chronic wound. This may occur through surgical débridement of infected or necrotic wounds and skin grafting on a healthy bed of granulation tissue. Revascularization procedures may be necessary to provide adequate blood flow to distal circulation that supplies a nonhealing/chronic wound.

III. Chronic wounds. Chronic wounds are a heterogeneous group. Chronic wounds that are associated with diabetes, pressure necrosis, and radiation therapy are frequently encountered, contributing to great disability within the population and causing a great burden on health care expenditures.

A. Diabetic foot ulcers

1. **Differential diagnosis.** The three most common causes of lower-extremity ulcers are arterial insufficiency, venous stasis, and diabetes mellitus. Venous stasis ulcers most often occur on the medial aspect of the patient's lower leg or ankle and are associated with the chronic edema and hyperpigmentation that are seen with venous insufficiency. Arterial insufficiency ulcers tend to occur distally on the tips of the patient's toes, but they can also occur at or near the lateral malleolus. The surrounding skin exhibits the thin, shiny, hairless characteristics that have been well described in these patients, and these individuals typically relate symptoms of claudication or rest pain. Peripheral pulses are diminished or absent. Diabetic ulcers are believed to be secondary to the severe neuropathy that is seen in these patients and to a lesser extent the vasculopathy as well (see the following). They are typically associated with very thick callus and most often occur on the patient's heels or on the plantar surface of the metatarsal heads.
2. **Causes**
 - a. **Neuropathy**
 1. **Peripheral neuropathy** is believed to be the most significant contributor to the development of lower-extremity ulcers in diabetic patients. The neuropathy impairs detection of injury from poorly fitting shoes or trauma, allowing further progression of tissue breakdown and infection. Diabetic motor neuropathy is also associated with abnormal weightbearing. The motor neuropathy results in abnormalities such as hammer toes or hallux valgus, which shifts weightbearing more proximally than normal on the metatarsal heads. Additionally, the dorsum of the toes at the posterior interphalangeal joints is often traumatized by ill-fitting shoes in patients with hammer toes.
 2. **Autonomic neuropathy** leads to failure of sweating and inadequate lubrication of the skin. Dry skin leads to mechanical breakdown that initiates ulcer formation. Autonomic neuropathy also contributes to failure of autoregulation in the microcirculation; therefore, arterial blood will shunt past capillaries into the venous blood flow. This reduces the nutritive blood flow to the skin in the diabetic foot and predisposes to ulcer formation.
 - b. **Ischemia.** The microvascular disease seen in diabetic patients also contributes to the development and progression of lower-extremity ulcers. These patients should be evaluated for proximal atherosclerotic disease that may be amenable to intervention, thus improving the chances for healing of the ulcer or for healing of an amputation.
3. **Evaluation and treatment**
 - a. **Examination.** The quality of the peripheral circulation, extent of the wound, and degree of sensory loss should be assessed. Web spaces should be examined for evidence of mycotic infection, which may lead to fissuring of the skin and subsequent infection. Mal perforans ulcers occur on the plantar surface of the metatarsals and extend to the metatarsal head, leaving exposed cartilage. Osteomyelitis of the phalanx or metatarsal is common.
 - b. **Clean wounds** are treated with conservative débridement and dressing changes, with careful trimming of the calluses and nails. Close follow-up is essential.
 - c. **Infected wounds.** The diagnosis of infection in a diabetic foot ulcer is a clinical one; wound cultures are unreliable unless derived from actual tissue samples. Plain films may show osteomyelitis or gas in the soft tissues. The progression of infectious processes in diabetic patients can occur with extreme rapidity, and thus these patients require hospitalization and aggressive wound care, with broad-spectrum antibiotics at initial presentation.

Abscess cavities must be completely drained and all dead tissue débrided; daily whirlpool treatments help cleanse the wound mechanically. Absolute nonweightbearing is crucial for healing.

- d. **Prevention** remains one of the single most important elements in the management of the diabetic foot. Meticulous attention to hygiene and daily inspection for signs of tissue trauma prevent the progression of injury. Podiatric appliances or custom-made shoes are helpful in relieving pressure on weightbearing areas and should be prescribed for any patient who has had neuropathic ulceration.

B. Leg ulcers

1. When **arterial ulcers** are suspected, a vascular evaluation should be instituted. Due to the tissue ischemia, antibiotics are often indicated with any signs of cellulitis.
 2. **Venous stasis ulcers** are one of the most common sources of leg ulcers and typically occur on the medial leg in the supramedial malleolar location. The patient often has a history of ulceration and associated leg swelling or of deep venous thrombosis. The wound may appear as a relatively superficial and extremely painful wound with irregular borders and an inflamed wound bed that bleeds fairly easily or as a partial-thickness wound with a dense, whitish-yellow fibrotic base. The wounds may be multiple and surrounded with a varying degree of inflammatory dermatitis. Cleansing the wound, providing a dressing that absorbs the wound drainage, leg compression, and leg elevation are the mainstays of treatment. After excluding significant ischemia, which can be present in up to 15% of patients, one can often use a multilayered compression dressing. This is changed on a weekly or biweekly basis until the wound heals. Aggressive medical treatment is often required to control the peripheral edema that in some patients is quite marked. More advanced methods of wound management such as skin grafting or the use of skin substitutes can be used, but only after the leg edema is controlled. External leg compression pumps and massage therapy are also useful in some patients. Wounds that are present for some time with increased drainage or pain are often infected. Treatment of infected wounds requires the addition of antibiotics.
- C. **Skin tears** are often seen in the elderly with skin that is markedly thinned and in the chronic steroid-using patient. The hypermobile skin with its poor subcutaneous connections is prone to rip under shearing forces, and patients often present with a flap of skin that is torn away from its wound bed. One should follow the principles that are outlined above, with the exception that the skin flap should be trimmed in its distant extent of obvious necrotic portions, but the remaining flap should be secured in place over the wound bed only to the extent that it does without tension. It is rare that such a wound should be sutured closed, because the flap will most likely necrose under the tension of the swelling that occurs over the following 2–4 days. Topical management of the area that is intentionally left open is often easily done with a hydrogel dressing (see Wound Care, [section I.D](#)).

D. Pressure ulcers

1. **Pathophysiology.** Prolonged pressure applied to soft tissue over bony prominences, usually caused by paralysis or the immobility that is associated with severe illness, predictably leads to ischemic ulceration and tissue breakdown. Muscle tissue seems to be the most susceptible. The particular area of breakdown depends on the patient's position of immobility, with ulcers most frequently developing in recumbent patients over the occiput, sacrum, greater trochanter, and heels. In immobile patients who sit for prolonged periods on improper surfaces without pressure relief, ulcers often develop under the ischial tuberosities. Pressure ulcers are described by stages ([Table 10-1](#)). Such wounds do not necessarily proceed through each one of these stages during formation but can present at the advanced stages. Likewise, as these wounds heal, they do not become a lesser stage despite their present depth (e.g., a nearly healed stage IV ulcer does not become a stage II ulcer but rather a healing stage IV ulcer, signifying that the tissues of the healing wound are abnormal). When a full-thickness injury to the skin has occurred, one cannot adequately stage the wound until the eschar is incised and the actual depth is determined.

Table 10-1. National Pressure Ulcer Advisory Panel classification scheme

2. **Prophylaxis**
 - a. **Skin care.** The patient's skin should be inspected and cleansed at least daily with application of moisturizers and barrier creams as necessary. Patients need to have a complete skin assessment and risk assessment done at the time of admission. All patients should be placed on the optimal support surface. Pressure-reducing support can be classified as static or dynamic. Static systems include mattresses filled with air, water, gel, or foam; dynamic support systems include low-air-loss mattresses and air-fluidized beds. Urinary and fecal continence needs to be maintained to prevent maceration and skin breakdown.
 - b. **Nutrition.** Nutritional deficits should be assessed and treated appropriately. Supplementation of vitamin C or vitamin A, or both, may be necessary if the patient is malnourished or is taking steroids.
 3. **Mobility.** Bedridden patients should be turned and repositioned with a minimum frequency of every 2 hours. Heel protectors, pillows, and foam wedges can be used to diffuse pressure from bony prominences across greater areas.
 4. **Treatment.** Most pressure ulcers heal spontaneously when pressure is relieved. This remains the most important factor in their healing. The healing process may require up to 6 months. Unless the patient was only temporarily immobilized, recurrences are common. Surgical management may include simple closure, split-thickness skin grafting, or musculocutaneous flap, but these measures should be reserved for well-motivated patients in whom a real reduction of risk factors for recurrence is possible. Healing of perineal and sacral wounds can be facilitated by urinary and fecal diversion, which reduces soiling and maceration of the wound bed.
- E. **Ionizing radiation.** Although ionizing radiation is a useful mode of cancer therapy, it produces detrimental local effects on tissue in the field of radiation and impairs normal wound healing. Radiation injures target and surrounding cells by direct damage to DNA or indirectly through free radicals or reactive species. Radiation has a negative impact on wound healing by harming the cellular elements that play a prominent role in various phases of wound healing. For instance, radiation alters wound healing by decreasing the proliferative capacity of cells, particularly those of endothelial, mesenchymal, and epithelial origin. Radiation also harms wound healing by decreasing the vascularity of the wound. This leads to the development of ischemia and hypoxia. Decreased vascularity occurs through the occlusion and thrombosis of small blood vessels and capillaries. In addition, the mechanisms for angiogenesis are limited due to poor endothelial cell proliferation.
- F. The **impaired blood supply** produced by radiation leads to a delay in healing through the creation of a hypoxic wound that lacks the molecular oxygen necessary for the cross-linkage of collagen fibers, for fibroblast stimulation, and for pathogen clearance. The result is a wound that is susceptible to bacterial invasion and deficit in collagen. With decreased levels of collagen, the wound lacks the key extracellular matrix component of the healing wound that allows the wound to mature appropriately through the phases of wound healing.
- G. **Radiation** also affects other cellular elements by harming keratinocyte proliferation and contributing to melanocyte destruction, which leads to failed epithelialization and hyperpigmentation in the wound. Total body irradiation leads to a total cessation of the proliferation of the cellular components of the bone marrow. Therefore, the inflammatory cells that are necessary for effective wound healing are unavailable, which prevents effective healing in such patients.
- H. The **early epithelial changes** that are characteristic of acute radiation injury include ulceration, edema, and sustained inflammation. Late changes include parenchymal degeneration, epithelial and dermal atrophy, decreased vascularity, fibrosis, and tissue necrosis. Like the healing wound, the skin has characteristic changes that are associated with radiation exposure. The skin is affected in a dose-dependent manner. Acute changes associated with the skin include erythema from dilation of blood vessels in the dermis, dry desquamation at moderate doses, or moist desquamation that occurs with the eradication of the cells of the epidermis. Late manifestations include hyper- or hypopigmentation, fibrosis of the skin and subcutaneous tissues, telangiectasia, sebaceous and sweat gland dysfunction, alopecia, and necrosis.
- I. The **timing of radiation therapy** as it relates to operative therapy has been an important aspect of oncologic care. The primary factors that determine the effects of preoperative radiation therapy on wound healing are the timing and dose. These vary from tissue to tissue. Postoperative radiation therapy has no effect on healing if it is administered 1 week after wounding. The intentional (surgical) wounding of a previously irradiated wound needs careful planning and consideration. Many of the cells in such an area have been permanently damaged; therefore, their proliferative capacity is decreased. In addition, the wound has decreased vascularity, which creates a relative state of hypoxemia. Furthermore, the dermis of such a wound is more susceptible to bacterial invasion. The combined factors place a previously irradiated area at extreme risk for abnormal wound healing if it is subjected to surgical intervention.
- J. It has long been realized for the above reasons that **radiation-damaged skin and wounds heal poorly**. Local measures that must be undertaken with wounds affected by radiation follow the same principles of good wound care. These measures include infection control through aggressive débridement and systemic antibiotics, topical antibiotics to promote epithelialization, moist dressings, and lubrication of dry skin. Optimal nutritional status must also be emphasized. Other methods, such as hyperbaric oxygen, have been used to increase the oxygen tension of irradiated wounds. Experimental therapies are currently under investigation. The use of the growth factors TGF-beta and PDGF have shown promise for treatment in radiation wound models in animals.

I. Open wound care options

- A. **Topical ointments.** Petroleum-based ointments that contain one or several antibiotics prevent adherence of dressings to the wound and, by maintaining moisture of the wound environment, accelerate epithelialization and healing of primarily approximated wounds.
- B. **Impregnated gauze.** Gauze that is impregnated with petrolatum is used for the treatment of superficial, partial-thickness wounds to maintain moisture, prevent excessive loss of fluid, and, in the case of Xeroform, provide mild deodorizing. It can also be used as the first layer of the initial dressing on a primarily closed wound. The use of this type of gauze is contraindicated when infection of the wound is suspected and inhibition of wound drainage would lead to adverse consequences.
- C. **Gauze packing.** The practice of packing an open wound with gauze prevents dead space, facilitates drainage, and provides varying degrees of débridement. The maximum amount of débridement is seen when the gauze is packed into the wound dry and removed after absorption and evaporation have taken place, leaving a dry wound with adherent gauze, which on removal also extracts superficial layers of the wound bed (dry-to-dry dressing). This dressing is seldom indicated. Wounds that are in need of great amounts of débridement usually benefit most from sharp débridement in the operating room or at the bedside; dry-to-dry dressings are painful and violate the principle of maintaining a moist environment for the wounds. Moist-to-dry dressings provide a much gentler débridement, are less painful, and can be performed with sterile normal saline or various additives. Dakin's solution [in full (0.5% sodium hypochlorite), half, or quarter strength] or acetic acid is used to pack infected open wounds when the antimicrobial action of these additives is desirable. Improvement of the foul odor that often emanates from drained abscesses and other infected open wounds is an added benefit when these additives are used.
- D. **Hydrogels.** These water- or glycerin-based gels (e.g., IntraSite) can be used in shallow or deep open wounds. The gel promotes healing by gently rehydrating necrotic tissue, facilitating its débridement, and absorbing exudate that is produced by the wounds, as well as maintaining a moist wound environment. A nonadherent, nonabsorbent secondary dressing is applied over the gel; dressings should be changed every 8 hours to 3 days, depending on the condition of the wound.
- E. **Hydrocolloids.** These occlusive, adhesive wafers provide a moist and protective environment for shallow wounds with light exudate. They can remain in place for 3–5 days and can be used under compression dressings to treat venous stasis ulcers.
- F. **Alginates.** Complex carbohydrate dressings composed of glucuronic and mannuronic acid, derived from brown seaweed, are formed into ropes or pads that are highly absorbent (e.g., Kaltostat). Alginates are absorbable and are useful for the treatment of deep wounds with heavy exudate because they form a gel as they absorb wound drainage.
- G. **Adhesive films.** These plastic membranes (e.g., Tegaderm) are self-adhering and waterproof yet permeable to oxygen and water vapor. They are appropriate for partial-thickness wounds, such as split-thickness skin graft donor sites or superficial abrasions. They can also be used as secondary dressings on wounds that are being treated with hydrocolloids or alginates.
- H. **Collagen-containing products.** A number of collagen-containing products are available in powder, sheet, or fluid form. They are available as pure collagen, typically types 1 and 3, or combined with other materials such as calcium alginate (Fibracol). Some wounds respond better to collagen than to other dressing materials.
- I. **Hydrofibers** represent a newer dressing category of strands that are some of the most absorptive materials available to pack in a heavily draining wound.
- J. **Growth factors.** Human recombinant PDGF is the only U.S. Food and Drug Administration–approved clinically available growth factor. Topically applied to a granulating wound, it promotes granulation tissue formation, angiogenesis, and epithelialization. A saline-moistened gauze dressing is applied daily at midday to help keep the wound bed moist. Although initial approval was for the treatment of diabetic plantar foot ulcers, most uses of the drug are on other wound types. EGF is presently in clinical trials for the treatment of venous stasis ulcers.
- K. **Skin substitutes.** Cultured dermis and epidermis are available clinically for treatment of wounds as 5-cm² circular sheets (Apligraf). Wounds must have a good granulating wound bed and low bacterial counts, and hemostasis must be meticulous. Trials are undergoing on the use of topically applied quick-frozen fibroblasts on wounds.

II. Wound closure materials and techniques

- A. **Skin adhesives.** Topically applied adhesives (Dermabond) can be used to maintain skin edge alignment in wounds that are clean and can be closed without tension and are in areas that are not subject to motion or pressure.
- B. **Steri-Strips.** Skin tapes are the least invasive way to close a superficial skin wound; however, because they provide no eversion of wound edges, the cosmetic result may be suboptimal. Additionally, skin tapes tend to loosen if moistened by serum or blood and, therefore, are seldom appropriate for all but the most superficial skin wounds in areas of minimal or no tension. Their most frequent use is in support of a skin closure after suture or staple removal.
- C. **Suture**
 1. **Needles.** Curved needles are designed for use with needle holders, whereas straight (Keith) needles can be used with or without a holder. Two types are in common usage: circular (tapered, noncutting) and triangular (cutting). Cutting needles are preferable for closure of tough tissue, such as skin, and noncutting needles are preferable for placing sutures in delicate tissues, such as blood vessels or intestine.
 2. **Suture material.** Several characteristics differentiate the various suture materials. They include
 - a. **Absorbable versus nonabsorbable.** Among the absorbable materials, wide variability is found with regard to tensile strength, rate of absorption, and tissue reaction.
 - b. **Monofilament versus braided.** Braided suture has better handling characteristics than does monofilament suture, but the interstices between the braided strands that compose the suture are easily colonized by bacteria and thus pose an infection risk.
 - c. **Natural versus synthetic.** Characteristics of the commonly used materials are summarized in [Table 10-2](#) and [Table 10-3](#).

Table 10-2. Characteristics of absorbable sutures

Table 10-3. Characteristics of nonabsorbable sutures

3. **Staples** allow for quick closure. In areas of lower cosmetic sensitivity, such as the thick skin of the back or anterior abdominal wall, staples may produce cosmetic results approximating those of sutures. They are particularly useful for closure of scalp wounds.
- D. **Skin suture technique**
 1. **Basic surgical principles apply:** closure without tension, elimination of dead space, aseptic technique, and (when closing skin) eversion of the skin margins. A dog-ear occurs when unequal bites are taken on opposing sides of a wound or incision, causing the tissue to bunch up as the end of the wound is approached. This can be prevented by carefully aligning the wound at the time of deep tissue closure (elimination of dead space) with interrupted absorbable sutures and by taking equal bites of tissue on both sides of the wound.
 2. **Suture removal.** Suture scars occur when stitches are left in place too long, allowing epithelialization of the suture tracts. This complication can be minimized by timely suture removal. Facial sutures should be removed at days 3–5; elsewhere, days 7–10 are appropriate. These guidelines should be modified for the individual patient. Application of skin tapes after suture removal provides further support ([Fig. 10-1](#)).

Fig. 10-1. Suture techniques. **A:** Simple interrupted stitches. **B:** Simple running stitch. **C:** Running locked stitch. **D:** Subcuticular stitch. **E,F:** Interrupted vertical mattress stitch. **G,H:** Interrupted horizontal mattress stitch.

III. Care of wounds in the emergency room

- A. **History and physical examination.** A careful history and physical examination of the whole patient should be performed, with attention to the time and mechanism of injury, initial treatments given, and prior or associated injuries. Medical, surgical, and immunization history and all known medication allergies should be documented. It is critically important that all injuries be identified, with appropriate prioritization of administered treatment plans. Careful neurologic and vascular examination should be performed distal to the site of injury and before administration of any anesthetics that could limit a later assessment.
- B. **Anesthesia.** Lidocaine (Xylocaine) in concentrations from 0.5% to 2.0% is generally chosen for its rapidity of action (1–2 minutes). If longer duration is desired, bupivacaine (Marcaine) can be used; however, it may require up to 10 minutes to full onset. A 50-50 mixture of 1% lidocaine and 0.25% bupivacaine provides a rapid and reasonably long-acting local anesthetic to improve hemostasis and prolong the effect of the anesthetic. This mixture should not be used to treat wounds on distal extremities (nose, earlobes, fingers, toes, or penis) because the profound vasoconstriction may lead to ischemic tissue loss. Whenever local anesthetics are used, care should be taken to avoid intravascular injection by aspirating before infiltration. The maximum safe amount of anesthetic that can be administered to the patient should be calculated before starting treatment. Patients with an allergy to amide anesthetics should be treated with ester anesthetics. The addition of sodium bicarbonate to lidocaine in a 1:9 ratio adjusts the acidic pH of the anesthetic so that its administration is less painful.
- C. **Wound cleansing.** After adequate anesthesia is administered, the wound and surrounding skin should be cleansed in a gentle fashion. This is best accomplished with a standard wound cleansing solution (e.g., Safclens, Shurclens, etc.). It should be remembered that many standard scrub solutions are extremely toxic to all living cells; thus, they should never be used to wash the wound itself. A good rule to follow is that one should never place a solution in a wound that one would not place in one's own eye. Wounds are best irrigated with saline or lactated Ringer's solution with pressures of 8–15 psi. An 18- or 19-gauge intravenous catheter or needle on a 35- to 60-mL syringe provides 8 psi irrigating pressure, which is adequate force to irrigate most wounds. Battery-powered irrigation systems, available for portable use, deliver pressures of up to 15 psi and are easier to use when irrigating the wound with several liters of fluid. Abrasions should be scrubbed carefully with a gloved hand during cleansing to remove foreign material that might lead to traumatic skin tattooing.
- D. **Wound hemostasis and exploration.** Direct pressure, elevation, and even the use of a blood pressure cuff as a tourniquet are effective means to limiting blood loss in the emergency setting. The use of electrocautery, suture ligation, or hemostat clamping of a bleeding site is best done by the practitioner who is familiar with the anatomy of the area because major nerves often lie adjacent to major arteries, and any imprecision can lead to an iatrogenic injury that is worse than the initial trauma. Wounds should be explored carefully for foreign bodies and to determine the extent of injury. Multiplane X-ray views of the soft tissues of the wounded area can prove to be useful in locating radiopaque objects. In the presence of difficult-to-locate or numerous foreign bodies, the wound can best be explored in the operating room.
- E. **Débridement.** Traumatic breaks of the skin are often irregular, and the force of impact leaves a zone of surrounding skin and underlying tissue injury that is often best treated by judicious sharp débridement. All foreign material and devitalized tissue must be removed before wound closure is attempted. The goal of débridement is obtaining a clean wound with a bleeding skin margin that overlies healthy, viable tissue.
- F. **Wound closure.** The decision to close a wound depends largely on the amount of contamination present and the amount of time that the wound has been open. Wounds that are older than 6–8 hours, puncture wounds, human bites, and wounds with gross infection should not be closed, with the possible exception of facial wounds, for which the superior vascular supply can often overcome otherwise major contamination. At a microscopic level, wounds with more than 10^5 bacteria per gram of tissue are considered too heavily contaminated to close safely. Dog and cat bites often can be closed safely after thorough irrigation and débridement, with administration of appropriate antibiotics.
- G. **Additional considerations**
1. **Tetanus prophylaxis.** Tetanus is a potentially fatal disorder that is characterized by uncontrolled spasms of the voluntary muscles. It is caused by the neurotoxin of the anaerobic bacterium *Clostridium tetani*. A tetanus-prone wound has one or more of the following characteristics: (1) more than 6 hours old; (2) deeper than 1 cm; (3) contaminated by soil, feces, or rust; (4) stellate configuration (burst-type injury with marked soft-tissue injury); (5) caused by missile, crush, burn, or frostbite; (6) contains devitalized or denervated tissue; and (7) caused by an animal or human bite (*Emerg Med Clin North Am* 10:531, 1992). Current recommendations for tetanus prophylaxis are summarized in [Table 10-4](#).

Table 10-4. Summary of Immunization Practices Advisory Committee recommendations for tetanus prophylaxis in routine wound management

2. **Antibiotics.** Antibiotic use does not allow closure of a wound that would otherwise be left open to heal secondarily, and it is not a substitute for good wound cleansing and débridement. Antibiotics should be chosen based on the indication (prophylactic or therapeutic), the location and age of the wound, and the mechanism of injury. In addition, one should consider the likely pathogen(s) that are most involved under the circumstances. Prophylactic antibiotics are indicated for immunocompromised patients and those with prosthetic heart valves or other permanently implanted prostheses. Prophylactic antibiotics should also be used when intestinal or genitourinary tract contamination is present, when an infection is likely to develop, or when an infection has potentially disastrous consequences (*Surg Clin North Am* 77:3, 1997). For wounds that are likely to become infected, obtaining good wound cultures at the time of injury helps to better target the specific organism(s) early that failed to respond to initial broad-spectrum antibiotic treatment.
- H. **Bites.** The treatment of a bite wound beyond the basic treatment of copious irrigation and débridement is most dependent on the source of the bite.
1. **Human bites** typically occur during interpersonal conflict. Because the wound often seems relatively trivial, such as a small puncture wound or a laceration in a patient who is very upset or intoxicated, the patient may delay seeking treatment, which increases the likelihood of the injury becoming infected. A particularly troublesome bite is a small skin injury that is seen over the metacarpophalangeal joint of a patient who punched someone else in the mouth and sustained a tooth cut of the skin overlying the fistful knuckle. Such injuries often require operative joint irrigation and parenteral antibiotics. Unintentional bites of the lip or tongue that are sustained in a fall or during a seizure may also occasionally come to the attention of a surgeon. The oral flora of humans include *Staphylococcus*, *Streptococcus*, anaerobic bacteria, *Eikenella corrodens*, and anaerobic gram-negative rods; antibiotic coverage should be directed initially toward these organisms.
 2. **Mammalian animals.** As infection is the most common complication of domestic animal bites, these bites should be considered contaminated and their immediate closure deferred. Infections that are caused by dog bites are usually polymicrobial, and pathogens include viridans streptococci, *Pasteurella multocida*, *Bacteroides*, *Fusobacterium*, and *Capnocytophaga*. The oral flora of the domestic cat is believed to be less complex, with *P. multocida* being found in up to 60% of wounds that are caused by cat bites. Local laws require the confinement of animals to ensure that they do not manifest rabies. Rabies, a routinely fatal disease of the central nervous system, is caused by the rabies virus, which is a member of the rhabdovirus group and contains a single strand of RNA. Thanks in large part to an intensive immunization program, the incidence of rabies in the United States has been reduced greatly, to approximately five cases per year. Today, the major risk comes from wild animal bites. The recommendations for rabies prophylaxis and treatment are summarized in [Table 10-5](#).

Table 10-5. Rabies postexposure prophylaxis treatment guide

3. **Snake bites.** Ten percent of snakes in the United States are venomous. Determining whether a venomous snake caused the bite is critical in the early management of bite injuries. Pit vipers usually leave two puncture wounds, whereas nonvenomous snakes generally leave a characteristic U-shaped bite wound. Poisonous snake venom contains many polypeptides that are damaging to human tissues, including phospholipase A, hyaluronidase, adenosine triphosphatase, 5-nucleotidase, and nicotinic acid dehydrogenase. The degree of envenomation and the time from injury determine the clinical manifestation. Immediate signs of envenomation include regional edema, erythema, and intense pain at the site of the bite. Systemic manifestations can ensue rapidly especially with greater envenomation. The hematocrit and platelet count may fall, with concomitant elevation of the prothrombin time, partial thromboplastin time, and bleeding times. Without treatment, severe envenomation may lead to pulmonary edema, peripheral vascular collapse, direct cardiotoxicity, and acute renal failure. Coral snake venom is less toxic locally but can lead to profound neurologic sequelae. Early symptoms include nausea, euphoria, salivation, paresthesias, ptosis, and muscle weakness leading to respiratory arrest.

Treatment is most successful if administered promptly. Extremity wounds should be immobilized and a tourniquet applied proximal to the bite site to minimize the spread of the venom. Although small amounts of venom can be removed by suction through small incisions over the bite wound, a wider surgical excision of the bite removes even more provided that it can be done in a timely fashion. Polyvalent antivenin may help to neutralize the venom and should be administered intravenously as soon as possible after more severe bites or when systemic symptoms are noted. The species-specific dose of antivenin should correspond to the perceived severity of the envenomation. Larger doses of antivenin carry the risk of serum sickness. Shock is treated with circulatory support. Broad-spectrum antibiotics and tetanus prophylaxis are also indicated.

4. Spider bites

- a. The **black widow (*Latrodectus mactans*)** is found throughout the United States and prefers to inhabit dry, dark crevices. The female is distinguished by her shiny black body and a red hourglass mark on the abdomen. The actual bite may cause little pain, and victims often do not recall the event. The venom, a neurotoxin, causes muscular rigidity. Chest pain from muscular contraction follows upper-extremity bites, whereas lower-extremity bites may cause rigidity of the abdominal wall. Patients who present with abdominal wall rigidity, which might typically suggest an acute abdominal emergency, lack associated abdominal tenderness. Intense muscular spasms and pain are usually self-limiting and require no specific treatment. Severe cases may progress to respiratory arrest, which, along with shock, accounts for the observed mortality of approximately 5%. Therapy consists of respiratory and circulatory support, broad-spectrum antibiotics, narcotic analgesia, and muscle relaxants. Antivenin (*L. mactans*) is indicated for the very young or old and for patients with severe illness.
- b. The **brown recluse (*Loxosceles reclusa*)** is found throughout the central and southern United States, most often inhabiting dark, moist environments. It is 10–15 mm long, with a light tan to brown color, a flat body, and a violin-shaped band over the head and chest area of the back. Brown recluse venom is very locally toxic, containing hyaluronidase and other elements that lead to coagulation necrosis of the area around the wound. Systemically, hemolysis with hemoglobinuria, hemolytic anemia, and renal failure may develop. Pain at the time of the bite is an inconsistent symptom; however, several hours after the bite, a characteristic lesion is seen, with a central zone of pale induration surrounded by an erythematous border. By this time, pain is severe. After approximately 1 week, a black eschar develops, which soon sloughs, leaving an ulcer that may continue to enlarge, with extensive necrosis of the underlying fat and subcutaneous tissues. Systemic illness most often occurs in children, with fever, malaise, nausea, and vomiting. Therapy is supportive, and mortality is rare. Excision of the wound should be deferred until the ulcer is well demarcated; broad-spectrum antibiotics are recommended.

11 Critical Care

T. Philip Chung and J. Perren Cobb

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Introduction

Patients are admitted to intensive care units (ICUs) because of either the presence or the risk of organ dysfunction. This chapter focuses on the three most common types of organ failure that are responsible for surgical ICU admissions: respiratory, cardiovascular, and renal failure. Also included are sections on sedation and analgesia, patient monitoring and transport, dosages of commonly used drugs, and prophylaxis against stress-induced upper gastrointestinal hemorrhage.

Sedation and Analgesia

A common neurologic manifestation of severe acute illness is altered mentation, which can span the spectrum from delirium to coma. The pathophysiology is poorly understood but may involve catecholamine excess and abnormalities of cerebral blood flow. The intensivist must also treat emotional distress and pain, which frequently accompany operations, procedures, dressing changes, and the insertion of catheters and tubes. Skillful control of pain and agitation in the ICU minimizes the threat of the patient to him- or herself or to the staff and allows for the orderly conduct of resuscitative efforts.

I. Control of agitation. The most frequently used agents are **benzodiazepines** delivered intravenously, potent inducers of sedation, anxiolysis, and amnesia. The precise mechanism of action of benzodiazepines has not been fully elucidated, but it appears to be mediated through gamma-aminobutyric acid, an inhibitory neurotransmitter. **Midazolam** has a short half-life (20–60 minutes) and therefore a rapid onset and offset of action. It is used in bolus doses of 0.5–2.0 mg (0.03 mg/kg) i.v. every 15–20 minutes to provide anxiolysis and sedation and to minimize compromise in cardiopulmonary function. For long-term (continuous) sedation, a longer-lived agent is recommended. **Lorazepam** is less lipophilic than midazolam and therefore has a longer time-to-onset of effect, making it less ideal for rapid sedation, and a prolonged duration with an elimination half-life of 10–20 hours. Bolus doses of lorazepam, 1–2 mg per hour i.v., to achieve the desired level of sedation with subsequent continuous infusion at 0.5–2.0 mg per hour is usually effective. Titration of sedation and communication are simplified by the use of an objective scoring system, such as the modified Ramsay scale ([Table 11-1](#)). Effective doses of either midazolam or lorazepam may be higher in patients who are tolerant, especially those who have taken similar agents previously or who consume alcohol regularly. In contrast, patients older than age 50 years or those with preexisting cardiopulmonary, hepatic, or renal dysfunction are particularly susceptible to these agents and their metabolites. In more debilitated patients, therefore, the initial doses of midazolam and lorazepam should be reduced to avoid overdose (manifested clinically as cardiopulmonary collapse). Flumazenil antagonizes the pharmacologic actions of benzodiazepines. Because patient sensitivity to these agents varies widely, immediate access to cardiopulmonary resuscitative equipment must always be available.

Table 11-1. Modified Ramsay sedation scale

Propofol is a nonbenzodiazepine sedative-hypnotic with an extremely short onset and offset of action that is usually delivered as a continuous infusion. It does not accumulate to the degree that midazolam or lorazepam does, resulting in a shorter length of sedation after discontinuation. Although high cost limits its widespread use, propofol is used routinely by many intensivists with excellent results. Prospective, randomized controlled trials are under way comparing the cost effectiveness of propofol to benzodiazepines for continuous sedation.

Haloperidol is also used short term to treat agitation, especially if it is accompanied by delirium. The combination of haloperidol and a benzodiazepine (midazolam or lorazepam) is particularly effective because smaller doses of each agent can be used, usually avoiding the extrapyramidal side effects of haloperidol. The initial dose of haloperidol in these settings is 1.5–2.5 mg i.v. every 6 hours for 1–3 days. Patients who receive haloperidol should have their electrocardiographic (ECG) QT interval measured every 8 hours; the drug should be discontinued if the QT interval increases by more than 50% of baseline or exceeds 450 msec (*J Intensive Care Med* 3:195, 1988).

II. Control of pain. Intravenous narcotics are used liberally in the surgical ICU. Morphine is administered most commonly because of its low cost and familiarity. Fentanyl is a synthetic opiate that is more potent and lipophilic than morphine. It has a half-life of 30–60 minutes due to its rapid redistribution. Unlike morphine, fentanyl does not cause histamine release and therefore is less likely to cause hypotension. Morphine and fentanyl can be administered either as a bolus or continuous infusion (see [Chapter 6](#)). Patient-controlled analgesia is useful for patients who are alert enough to dose themselves. Because all narcotics can cause respiratory depression and hypotension, care in the use of these agents is particularly important in patients with poorly compensated cardiopulmonary dysfunction (smaller than usual initial doses are safer). Thoracic or lumbar epidural catheters are usually well tolerated, decrease the need for intravenous narcotics, and can

substantially improve compliance with respiratory therapy.

III. For patients who require **long-term sedation and analgesia**, a prospective, randomized controlled study demonstrated that daily interruption of sedation to wakefulness produced decreased time on mechanical ventilation and shorter ICU stays [*N Engl J Med* 342(20):1477, 2000].

Acute Respiratory Failure

I. Etiology. Respiratory failure results from inadequate exchange of oxygen and carbon dioxide. This is due to failure of the mechanical ventilatory apparatus (as in neuromuscular disease, inspiratory muscle fatigue, and airway obstruction), which results in hypoventilation and, thereby, hypercapnia and hypoxemia. It may also result from failure of the gas-exchange mechanism [as in asthma, chronic obstructive pulmonary disease, acute respiratory distress syndrome (ARDS) (*Crit Care Med* 26:369, 1998), pneumonia, pulmonary embolism, and pulmonary edema], resulting in ventilation-perfusion mismatch. Alternatively, it may be due to a combination of these events. Although determining the etiology is important therapeutically and prognostically, the early treatment of respiratory failure by mechanical positive-pressure ventilation is the same, regardless of etiology.

II. Diagnosis. Signs or symptoms of respiratory impairment (e.g., tachypnea, dyspnea, or mental status changes) should prompt analysis of arterial blood gases (ABGs). A hemoglobin saturation of less than 92% corresponds to a PaO₂ of less than 60 mm Hg, which seriously compromises tissue oxygenation. An acute rise in PaCO₂ to greater than 50 mm Hg with a pH of less than 7.35 (respiratory acidosis) implies a significant imbalance between carbon dioxide production and elimination (alveolar ventilation). Both of these laboratory abnormalities are consistent with respiratory insufficiency. In a patient with the aforementioned clinical signs of respiratory impairment, a diagnosis of respiratory failure is made.

III. Treatment

- A. **Airway management.** Securing and maintaining a patent airway are essential. The most common obstruction of the airway in a patient with an altered sensorium is the tongue. This is corrected easily by the chin-lift or jaw-thrust maneuver or by placing an oropharyngeal or nasopharyngeal airway. If uncertainty exists about whether the airway is patent or protected from aspiration, endotracheal (ET) intubation is indicated.
1. **Oral and nasal ET intubation** (see also [Chapter 28](#)). In most ICUs, intubation trays complete with all the necessary tools are readily available. For oral and nasal routes, preoxygenation by use of a bag-valve-mask apparatus, suction, topical anesthetics, adequate sedation and muscle relaxation, and an appropriately sized ET tube are required. The oral route is usually the most expeditious, but it requires a deeper level of patient sedation and significant operator skill with a laryngoscope. The nasal route can be used only when the patient is breathing spontaneously; significant skill is needed to direct the tip of the ET tube blindly past the vocal cords and into the trachea. Once the tube is in the trachea and the adequacy of bilateral ventilation has been established using auscultation or a carbon dioxide indicator, a chest X-ray (CXR) is required to document correct ET tube position.
 2. **Tracheostomy** should be considered in the presence of severe maxillofacial injury to ensure an adequate airway or if prolonged (>2 weeks) oral or nasal intubation is anticipated. Tracheostomy provides a more secure airway, improved patient comfort and oral hygiene, increased patient mobility, and enhanced secretion removal.
 3. **Cricothyroidotomy** is useful in emergency situations when attempts to ventilate by bag-valve-mask and ET tube are unsuccessful. The technique is straightforward: Extend and surgically prepare the patient's neck, and make a midline incision using a No. 11 blade through the cricothyroid membrane. Hold the wound open by twisting the handle of the knife blade 90 degrees or spreading with a hemostat. A tracheal hook through the wound is frequently useful to stabilize the cricoid bone. Intubate the trachea with a small tracheostomy or ET tube (typically size 6 for an adult).
 4. **Complications.** Immediate complications include passage of the ET tube into either the esophagus or the tissue surrounding the trachea. The latter can lead to hemorrhage, pneumothorax, pneumomediastinum, subcutaneous emphysema, and injury to the recurrent laryngeal nerve. Delayed complications include hemorrhage, which rarely may result from erosion of the tube into a prominent vessel (usually the brachiocephalic artery). This is treated by immediate orotracheal intubation, removal of the tracheostomy tube, insertion of the surgeon's finger into the tracheostomy site, and anterior compression of the brachiocephalic artery against the clavicle. ET tube cuff pressures should be monitored frequently and kept below capillary filling pressures (i.e., <25 mm Hg) to prevent tracheal ischemia, which, if untreated, can lead to tracheomalacia or tracheal stenosis.
- B. **Oxygen therapy.** The objective of supplemental oxygen administration is to increase the relative concentration of oxygen in the alveoli. This is accomplished most commonly by delivering oxygen through a nasal cannula, simple face mask, or face mask with a reservoir ([Table 11-2](#)). The inspired oxygen concentration varies depending on the percentage of entrained air: The more air that is entrained (with an ambient oxygen concentration of 0.21), the lower the fraction of inspired oxygen (FIO₂). When the required FIO₂ is high (>0.60), a high air-flow system with oxygen enrichment via a jet-mixing or Venturi apparatus is used, delivered by a tight-fitting mask with a reservoir (to minimize entrainment). Whenever possible, inspired oxygen should be humidified to prevent drying of the airways and respiratory secretions.

Table 11-2. Oxygen delivery systems

- C. **Mechanical ventilation** is indicated for the treatment of respiratory failure. Critical physiologic values that are useful in making the decision to implement mechanical ventilation are listed in [Table 11-3](#). The goal of treatment is to improve alveolar ventilation and oxygenation and to reduce the work of breathing while other therapies are instituted to treat underlying disease processes.

Table 11-3. Guidelines for elective intubation and mechanical ventilation

1. **Modes of mechanical ventilation** (*N Engl J Med* 330:1056, 1994; *Chest* 104:1833, 1993) can be divided into volume-limited and pressure-limited modes (compared in [Table 11-4](#)). The key to the differences between these modes lies in understanding the relationship of pressure to volume [i.e., pulmonary compliance, in which compliance equals the change in volume divided by the change in pressure ($C = \Delta V / \Delta P$)]. The goal of volume-limited modes is to deliver a set tidal volume to the patient at a rate that ensures adequate alveolar ventilation; airway pressures vary depending on compliance. In contrast, the goal of pressure-limited modes is to deliver a set airway pressure; tidal volume varies depending on compliance.

Table 11-4. Modes of ventilation

a. Volume-limited modes

1. **Assist-control (A/C) ventilation** delivers a preset tidal volume at a set rate. As the machine senses each inspiratory effort by the patient, it delivers the set tidal volume. If the patient's respiratory rate is below the machine's set rate, ventilator-initiated breaths are delivered to make up the difference between the set rate and the patient's. A/C ventilation minimizes the work of breathing because the ventilator assists all breaths (hence, the term *full support*); however, for this reason, this mode is uncomfortable if the patient's breaths are dyssynchronous with those delivered by the ventilator. Respiratory alkalosis from hyperventilation may develop in agitated patients.
2. **Intermittent mandatory ventilation (IMV)**, like the A/C mode, delivers a preset tidal volume at a set rate. IMV differs from A/C for spontaneously breathing patients because the ventilator in the IMV mode does not assist spontaneous respiratory efforts. The tidal volume during these breaths thus is determined entirely by the strength of the patient's respiratory effort. To prevent stacking of mechanical breaths on top of spontaneous breaths,

positive-pressure breaths interjected by the ventilator are triggered by the patient's spontaneous efforts, allowing synchronization with the patient's ventilatory pattern. The gas for spontaneous breathing during IMV is provided by activation of either a flow-by valve in continuous-flow systems or a demand valve that opens a reservoir of fresh gas during inspiration. With demand-valve systems, often there is a significant lag between the start of the inspiratory flow (generated by the patient) and arrival of fresh gas in the trachea. During this lag, the patient expends work in the effort to breathe, which may potentiate respiratory muscle fatigue (*Am Rev Respir Dis* 134:902, 1986). This mode may be more comfortable than A/C for alert, robust patients because it allows unassisted breaths; however, for those with weak respiratory muscles, it is less comfortable because it makes them work harder for a given minute ventilation. Most ventilators allow low levels of pressure support [see [section III.C.1.b\(2\)](#)] to aid spontaneous ventilation and reduce the work imposed by these demand valves.

b. Pressure-limited modes

1. **Pressure-control ventilation** delivers a preset inspiratory pressure (as opposed to tidal volume) at a set rate. This mode is used in patients with poor (low) lung compliance in whom high inspiratory pressures develop when they are ventilated with the more traditional modes described previously. Thus, the advantage of this mode is that it allows the physician to set the peak airway pressure and thereby minimize barotrauma. The disadvantage is that the tidal volume varies depending on compliance. Sudden development of an increase in airway resistance (coughing, thick secretions, a kink in the ET tube, a Valsalva's maneuver), for example, increases airway pressures and decreases tidal volumes to dangerously low levels. For this reason, patients who are ventilated using this mode must be more heavily sedated and closely monitored.
2. **Pressure-support ventilation** delivers a preset inspiratory pressure but at no set rate. Constant inspiratory pressure continues until the inspiratory flow of gas falls below a predetermined level and the exhalation valve opens. Thus, tidal volumes are generated only when the patient is breathing spontaneously. This allows the patient to maintain control of inspiratory and expiratory time and thus tidal volume; as a result, this mode is by far the most comfortable for spontaneously breathing patients. The disadvantages of pressure-support ventilation are (1) that all ventilation depends on patient effort, and (2) that sudden increases in airway resistance, as with pressure-control ventilation, decrease tidal volumes. Small amounts (5–8 cm H₂O) of pressure-support ventilation are used routinely to overcome the resistance to air flow provided by the ET tube and the inspiratory demand valves of the ventilator (*Am Rev Respir Dis* 139:513, 1989).

c. Other modes of ventilation

1. **High-frequency ventilation** uses substantially faster rates (60–300 per minute) and smaller tidal volumes (2–4 mL/kg) than do conventional modes. The result is a relative decrease in diaphragmatic excursion, lung movement, and airway pressures. Physical mechanisms that are responsible for gas movement are complex and incompletely understood; they include convective flow, pendelluft, and diffusion (*Crit Care Med* 22:S24, 1994). High-frequency jet ventilation is used most frequently for mechanical ventilatory support during bronchoscopic, laryngoscopic, and upper airway surgery because it can be delivered by jets of air through a thin tube placed in the trachea. High-frequency oscillatory ventilation is under investigation as a means of ventilating adult patients with ARDS (*Crit Care Med* 25:937, 1997). These modes may also be useful in patients with severe damage of only one lung (using independent lung ventilation), bronchopleural fistulas, or profound respiratory failure that is unresponsive to other modes of ventilation.
2. **Biphasic positive airway pressure** is a newer form of ventilation that is delivered by means of a tight-fitting nasal mask (no ET tube) that allows for independent control of positive inspiratory and expiratory pressures. It is most useful as a bridge to aid respiratory efforts in patients with mild to moderate respiratory insufficiency of short duration [e.g., asthma or chronic obstructive pulmonary disease exacerbations or pulmonary edema (*Am J Respir Crit Care Med* 151:1799, 1995; *Chest* 105:229, 1994)].

2. Ventilator management

- a. **Choice of ventilator mode.** One of the most important duties of the intensivist is to match the needs of the patient with the appropriate ventilator mode. This is accomplished most easily by considering the advantages and disadvantages of each mode ([Table 11-4](#)). Failure to do so results in patient agitation and a significant waste of patient energy spent “fighting the ventilator.” Typically, the best mode for the paralyzed patient with relatively good lung function is A/C ventilation. If the patient has severe pulmonary dysfunction with very low lung compliance and resultant high peak airway pressures, pressure-control ventilation may be better suited to avoid barotrauma. Most awake patients benefit from IMV plus pressure support. This combination can provide the full spectrum of ventilatory support, allowing several physiologic advantages: control of the number of spontaneous breaths, control of the degree of ventilator support for spontaneous breaths, lower intrathoracic pressures, exercise and maintenance of respiratory muscle function and coordination, and lower levels of sedation. As a result, this is a common combination of modes used to wean patients off the ventilator.
 - b. **FIO₂** should be adjusted to ensure adequate arterial oxygenation, which is a blood hemoglobin saturation of 92% in lighter-skinned individuals and 95% in darker-skinned patients (*Chest* 97:1420, 1990). The lowest possible FIO₂ (ideally <0.40) should be used to achieve these levels of arterial saturation to prevent pulmonary oxygen toxicity and retrolental fibroplasia (in neonates). Warming and humidifying the inspired gas prevents drying of secretions and heat loss and promotes mucociliary clearance.
 - c. **Tidal volume.** Tidal volume size was studied in a multicenter, prospective randomized trial, which demonstrated improved survival in patients who were ventilated with lower tidal volume (6 mL/kg predicted body weight) than suggested historically (10–15 mL/kg): 31.0% versus 39.8%, respectively ($p = .007$) [*N Engl J Med* 342(18):1301, 2000]. As a result of this important study, the tidal volume is adjusted when possible to maintain peak airway pressures at less than 30 cm H₂O to minimize barotrauma but more than 20 cm H₂O to minimize atelectasis.
 - d. **Ventilatory rate.** Once the tidal volume has been determined, the rate is chosen (typically 8–16 breaths per minute) to provide adequate minute ventilation (the product of rate and tidal volume). The rate is adjusted to optimize arterial pH and PaCO₂; an end-tidal CO₂ monitor is frequently useful in this regard.
 - e. **Inspiratory-expiratory (I/E) ratio.** The normal I/E ratio is 1:2–1:3. Longer expiratory times allow patients with obstructive lung disease (high compliance) to exhale fully and prevent stacking of breaths. In contrast, longer inspiratory times, which decrease peak airway pressures, are useful in patients with low pulmonary compliance. Inverse ratio ventilation takes advantage of breath stacking, using I/E ratios from 1:1 to 4:1. Used only in patients with severe, consolidating lung disease, inverse ratio ventilation is believed to improve gas exchange by progressive alveolar recruitment [mean airway pressures are higher, keeping a larger number of alveoli open for a greater percentage of the respiratory cycle (*Crit Care Med* 23:224, 1995)]. Inverse ratio ventilation is used most commonly with pressure-control ventilation.
 - f. **Positive end-expiratory pressure (PEEP)** increases functional residual capacity, increases lung compliance, and improves ventilation-perfusion matching by opening terminal airways and recruiting partially collapsed alveoli. Five-centimeter H₂O PEEP is considered physiologic; higher levels are used when hypoxemia is moderate to severe. PEEP significantly increases intrathoracic pressure and therefore decreases cardiac output (CO), reduces venous return to the heart, increases airway pressure, and alters pulmonary vascular resistance. PEEP levels of greater than 15 cm H₂O significantly increase the risk of barotrauma and spontaneous pneumothorax. PEEP applied to the spontaneously ventilating patient without inspiratory ventilatory support is called **continuous positive airway pressure (CPAP)**.
3. **Sedation and neuromuscular paralysis.** Sedation is often necessary in mechanically ventilated patients to control anxiety, allow the patient to rest, and synchronize breathing of the patient with the ventilator (*Am Rev Respir Med* 147:234, 1993; *Chest* 104:566, 1993). Paralysis rarely is necessary but is useful in patients with severe respiratory failure because it increases pulmonary compliance by decreasing the elastic recoil of the chest wall. A list of commonly used sedatives and paralytics and their dosages is included in [Chapter 6](#) (see [Table 6-2](#) and [Table 6-3](#)).

4. Complications

- a. **ET tube dislodgment and patient self-extubation** can produce a medical emergency characterized by life-threatening hypoxia and hypercarbia in those who are profoundly ill. For this reason, restraint of the patient's upper extremities is frequently required. In contrast, a period of observation is often indicated in patients who are weaning from ventilation because a surprisingly large number of these individuals remain successfully extubated. If the patient shows any signs of persistent respiratory distress, however, he or she should be re-intubated immediately.
- b. **ET tube cuff leaks** should be suspected when there is an unexplained decrease in the returned expired volume associated with a fall in airway pressure. Because cuff leaks increase the risk of aspiration and decrease the efficiency of ventilation, the tube should be changed urgently.
- c. **Respiratory distress** may occur suddenly during mechanical ventilation and is due either to an acute change in the patient's status or ventilator malfunction. The first priority is to disconnect the ventilator and switch to bag ventilation using 100% oxygen to ensure adequate ventilation and oxygenation. Increased airway pressures indicate obstruction of the tube with secretions or a kink in the tube, bronchospasm, pneumothorax, or migration of the ET tube into a main-stem bronchus. Check the ET tube for patency and suction through it; if there is a partial obstruction, use large-volume saline lavage to clear the tube. If the obstruction is complete, remove the ET tube and re-intubate the patient. Listen closely for any change in breath sounds that are consistent with a pneumothorax or new lung consolidation or pleural fluid collection. A less common but important cause of respiratory distress is pulmonary embolism.

Check the ventilator's function and, if normal, return the patient to the ventilator, making any needed changes in ventilator settings to ensure adequate ventilation and oxygenation. The results of an ABG and CXR frequently are helpful in identifying a cause of the change in respiratory status.

- d. **Barotrauma** from very high peak airway pressures (>50 cm H₂O) can lead to subcutaneous emphysema, pneumomediastinum, and pneumothorax (*Crit Care Med* 23:223, 1995). Whereas subcutaneous emphysema and pneumomediastinum usually are benign, a pneumothorax that develops while a patient is on positive-pressure ventilation is at high risk for becoming a tension pneumothorax and thus usually is treated emergently (tube thoracostomy).

- e. **Oxygen toxicity** refers to lung damage from high levels of intraalveolar oxygen. The precise mechanism is not known but probably involves oxidation of cell membranes and generation of toxic oxygen radicals. An FIO_2 of 0.40 or less is considered safe even for long periods. Although experimental data demonstrate that microscopic damage to alveoli occurs after only a few hours of an FIO_2 of 1.0 in animals, convincing studies in patients are lacking. It appears prudent, however, to keep the FIO_2 at 0.60 or less whenever possible, often using higher levels of PEEP (8–12 cm H_2O) to help wean down the FIO_2 (*New Horizons* 1:504, 1993). Another adjunct that is useful for patients with severe hypoxemia is inhaled nitric oxide (NO), especially in the setting of pulmonary hypertension. NO is an endogenous, ubiquitous second messenger, with potent vasorelaxing properties. Available as a supplemental gas that can be entrained with inhaled gases, NO diffuses across the alveolar membrane into pulmonary vascular smooth muscle, resulting in vasodilation and increased local blood flow. This selective effect of increased perfusion in ventilated alveoli is believed to contribute to improved ventilation-perfusion matching and gas exchange. Results of a randomized, double-blinded, phase II study of NO in patients with ARDS indicated that NO provided modest improvements in oxygenation but no improvement in mortality or the number of days alive off mechanical ventilation (*Crit Care Med* 26:15, 1998).
5. **Weaning off mechanical ventilation.** Although there are exceptions (e.g., immediate extubation of a healthy patient with normal lungs after general anesthesia), discontinuing mechanical ventilation usually requires several steps, collectively called *weaning*. In general, hemodynamic instability or a high work of breathing (e.g., minute ventilation >15 L per minute) is a contraindication to weaning. Weaning of the FIO_2 to 0.40 or less and PEEP of 5 cm H_2O or less is accomplished first (see preceding section). Most relatively healthy patients tolerate rapid reduction in the IMV or A/C rate over several hours because their mental status improves after general anesthesia. Weaning parameters ([Table 11-5](#)) are measured as soon as the patient is awake and alert enough to cooperate (*N Engl J Med* 324:1445, 1991). In contrast, the patient who has needed prolonged ventilatory support may require several days to weeks to wean because of marginal respiratory muscle strength and time required for the injured lungs to recover. The optimal strategy to wean patients continues to be a topic of debate. The results of controlled clinical trials indicate that the method of weaning ventilator support is most likely of little consequence for patients who have been on mechanical ventilatory support for 2 weeks or less, as the primary determinant of weaning success is simply resolution of the pathology that induced respiratory failure (*N Engl J Med* 335:1864, 1996; *N Engl J Med* 332:345, 1995; *Am J Respir Crit Care Med* 150:896, 1994). One successful weaning protocol that is useful in medical as well as surgical ICUs has been published (*Crit Care Med* 25:567, 1997). To aid spontaneous respiratory efforts, many intensivists add pressure-support ventilation, which allows fine adjustment in the degree of support provided by the ventilator (see [section III.C.2.a](#)). As lung mechanics improve, the amount of pressure support is decreased gradually. Others use short periods of unassisted spontaneous ventilation with small amounts of CPAP (CPAP trials) to test muscle strength and recovery of pulmonary function.

Table 11-5. Guidelines (weaning parameters) for assessing withdrawal of mechanical ventilation

Shock

Shock is manifested by hypotension and occurs when either the supply of or the ability to use oxygen and other nutrients is insufficient to meet metabolic demands. If left uncorrected, shock inevitably leads to the death of cells, tissues, organs, and, ultimately, the patient. Understanding the pathophysiology of shock depends on an appreciation of the relationship of blood pressure, specifically, mean arterial pressure (MAP), to CO and systemic vascular resistance (SVR): MAP is directly proportional to CO and SVR. Because CO is equal to stroke volume times heart rate, and stroke volume is directly proportional to preload, afterload, and myocardial contractility, MAP is directly proportional to heart rate, preload, afterload, and contractility. Compensatory changes for systemic hypotension include the release of catecholamines, aldosterone, renin, and cortisol, which act in concert to increase heart rate, preload, afterload, and contractility.

I. Classification and recognition of shock ([Table 11-6](#)). The morbidity and mortality of circulatory shock are related not only to the underlying cause but also to the depth and duration of circulatory compromise. Early recognition and prompt intervention are therefore critical.

Table 11-6. Clinical parameters in shock

- A. **Cardiogenic shock** results from inadequate CO due to either intrinsic cardiac failure (e.g., acute myocardial infarction, valvular stenosis, regurgitation or rupture, ischemia, arrhythmia, cardiomyopathy, or acute ventricular septal defect) or extrinsic processes that interfere with cardiac performance (e.g., pericardial tamponade, massive pulmonary embolism, or tension pneumothorax). Patients typically are peripherally vasoconstricted, tachycardic, and oliguric. Jugular venous pressure typically is elevated.
- B. **Hypovolemic shock** results from loss of circulating blood volume (preload usually at least 20%) caused by acute hemorrhage, fluid depletion, or dehydration; these three usually are distinguishable from one another by history. Like the patient with cardiogenic shock, these individuals typically are peripherally vasoconstricted, tachycardic, and oliguric. In contrast, however, jugular venous pressure is low with hypovolemic shock.
- C. **Distributive shock. Septic shock** results from systemic infection and decreased myocardial afterload. Typically, sepsis is manifested by a hyperdynamic state consisting of tachycardia, vasodilation with decreased cardiac filling pressures, decreased SVR, and increased CO; however, some patients present with hypodynamic septic shock with decreased CO and hypoperfusion. **Neurogenic shock** results from interruption of the spinal cord at or above the thoracolumbar sympathetic nerve roots, which also produces loss of sympathetic tone to the vascular system.

During sepsis, vasodilation occurs secondary to vasoactive mediators released in response to infection. During neurogenic shock, vasodilation occurs secondary to loss of sympathetic tone. The cardiovascular response is the same: Patients are typically peripherally vasodilated (warm extremities), tachycardic, and oliguric. Jugular venous pressure is low.

II. Treatment.

- A. **Interventions common to all types of shock.** The goal of therapy is to improve the delivery of oxygen to the peripheral tissues. Because oxygen delivery is the arithmetic product of arterial oxygen saturation (SaO_2), hemoglobin concentration, and CO, each of these parameters should be optimized.
1. **SaO_2 .** Administer supplemental oxygen, secure or provide an adequate airway, and check for adequate, bilateral ventilation. Pulse oximetry (SaO_2) that exceeds 92% should allow adequate delivery of oxygen at the periphery; however, this should be maximized in the acute setting.
 2. **Hemoglobin concentration.** The hemoglobin concentration must be adequate to deliver oxygen to the tissues. One study has indicated that for most critically ill patients a transfusion trigger of hemoglobin, 7 g/dL, is appropriate, aiming to keep it at 7.0–9.0 g/dL (*N Engl J Med* 340:409, 1999). A tube of blood should be sent early to the blood bank for cross-matching to avoid delays in blood availability for transfusion.
 3. **CO.** The ECG tracing provides direct information about heart rate and several indirect clues about stroke volume. The atrial contraction provides approximately 25% of ordinary CO so that the atrioventricular dyssynchrony observed in atrial fibrillation or third-degree atrioventricular block predictably causes significant impairment of CO. Tachyarrhythmias decrease diastolic ventricular and coronary artery filling times. When severe (e.g., heart rate ≥ 140 beats per minute), tachycardia predictably impairs preload, stroke volume, and CO. When treating tachycardia per se, it is imperative to distinguish between tachycardia as a compensatory response (e.g., sinus tachycardia secondary to hypovolemia) and tachycardia as a cause of shock (e.g., ventricular tachycardia). With the exception of the patient in pulmonary edema, *all* patients in circulatory shock should initially receive 10–20 mL/kg of a balanced salt solution, such as lactated Ringer's solution (normal saline is not a balanced salt solution). The pace of volume infusion should reflect the depth of circulatory shock. To achieve rapid infusion rates, short, large-bore intravenous catheters (e.g., 14 gauge) in an antecubital vein are best. If this is not possible, a No. 8.5 French (Fr.) cordis (Swan-Ganz introducer) inserted into a central vein is effective. The stopcocks should be removed from the venous lines to reduce flow resistance and deliver *warmed* fluids. Hypothermia is aggravated by rapid infusion of room-temperature crystalloid and refrigerated blood, thereby impairing the ability to unload oxygen from hemoglobin in the periphery and compromising all enzymatic processes, especially coagulation.

4. To assess the adequacy of resuscitation, peripheral pulses and urine output should be evaluated. Palpable pedal pulses or urine output that exceeds 1 mL/kg per hour usually indicates a cardiac index of greater than 2 L/m² per minute. These two simple techniques can be used to estimate cardiac performance in many patients. Patients who do not improve with initial resuscitative measures may require invasive hemodynamic monitoring [pulmonary artery (PA) catheterization; see Routine Monitoring of the Critically Ill Patient, [section V](#)]. All shock victims should be monitored with an indwelling bladder catheter. Metabolic acidosis, identified by an ABG determination and serum electrolytes, reflects the depth of circulatory compromise and the adequacy of resuscitation. Infusion of sodium bicarbonate should be reserved for patients with a pH of less than 7.1, because the sodium bicarbonate may actually worsen intracellular pH as the bicarbonate is converted to CO₂ at the tissue level.

B. Specific therapy

1. **Cardiogenic shock.** It is critical to distinguish shock caused by intrinsic myocardial dysfunction from that which results from extrinsic processes that interfere with venous return to the heart. Diagnosis may require echocardiography and cardiac catheterization. Management is directed toward maintaining adequate myocardial perfusion and CO with volume expansion and vasopressors, inotropes, or chronotropes ([Table 11-7](#)). Initial treatment often is guided by central venous pressure (CVP) measurements or, in severe cases, PA catheter “wedge” pressure, while the precipitating cause of compromise is identified and treated. Mechanical support with intraaortic balloon counterpulsation may be necessary before and during recovery from definitive surgical treatment (see [Chapter 35](#)). The two common thoracic processes that compromise venous return and cause pump failure are tension hemothorax or pneumothorax and pericardial tamponade (see [Chapter 28](#)).

Table 11-7. Vasoactive drugs and their specific actions

2. **Hypovolemic shock.** Therapy focuses on control of ongoing volume loss and restoration of intravascular volume. External hemorrhage should be controlled by direct pressure. Internal hemorrhage may be difficult to detect and control without further diagnostic tests or surgical intervention. The degree of volume deficit ([Table 11-8](#)) determines the type and volume of resuscitative fluid. Patients with blood losses of up to 20% of their circulating blood volume can be resuscitated using crystalloid solutions alone, typically lactated Ringer's solution. However, because salt solutions equilibrate with the interstitial space, volume replacement with these solutions alone requires three times the estimated volume deficit. Patients in whom diaphoresis, ashen facies, and hypotension develop have lost 30% or more of their blood volume and require urgent transfusion of blood. Individuals with severe dehydration often have profound metabolic and electrolyte abnormalities. Fluid administration should be modified once laboratory analysis of serum electrolytes is completed. With adequate volume resuscitation, vasoconstrictors and vasoactive agents can usually be avoided.

Table 11-8. Physiologic changes in hypovolemic shock

3. Distributive shock

- a. **Septic shock.** Management of septic shock requires vigorous volume resuscitation, often coupled with judicious administration of a vasoconstrictor (*Ann Intern Med* 120:771, 1994). Dopamine, phenylephrine, and norepinephrine commonly are used to increase vascular tone and mean aortic pressure. Although septic shock is typified by a high CO, myocardial contractility paradoxically is decreased, whereas heart chambers typically are dilated. To maintain CO, heart rate usually is increased. Septic patients who fail to achieve rapid hemodynamic stability with fluids and small doses of vasoconstrictors may require insertion of a PA catheter to optimize cardiac performance. Typically, higher filling pressures are needed (pulmonary capillary wedge pressure of 14–18 mm Hg) to optimize performance in the dilated, septic heart. Ultimately, successful management depends on treatment of the underlying sepsis.
- b. **Neurogenic shock.** As with septic shock, the initial intervention in neurogenic shock is volume infusion. A peripheral vasoconstrictor, such as phenylephrine or norepinephrine, is administered to increase vascular tone if hypotension is refractory to volume infusion alone. Because patients with spinal shock tend to equilibrate body temperature with their environment, fluids and ambient room temperature must be kept warm.

Routine Monitoring of the Critically Ill Patient

I. Temperature monitoring. Critically ill patients are at increased risk of temperature disorders as a result of debility, impaired voluntary control of temperature, frequent use of sedative drugs, and high predisposition to infection. All critically ill patients should have core temperatures measured at least every 4 hours, usually with a rectal thermometer, which is more reliable than the oral or axillary routes.

II. ECG monitoring. Continuous ECG monitoring with computerized arrhythmia detection systems is standard in most ICUs. Aside from providing rapid assessment of heart rate and rhythm, continuous monitoring allows for rapid detection of dysrhythmias, thereby increasing the likelihood of successful resuscitation. Treatment of common cardiac dysrhythmias is detailed in [Chapter 7](#).

III. CVP monitoring. Central venous catheters provide access to measure CVP and to administer vasoactive drugs and total parenteral nutrition. For techniques of catheter insertion, refer to [Chapter 42](#).

IV. Arterial pressure monitoring

A. **Indirect arterial pressure measurement** with a sphygmomanometer is performed at least hourly.

B. **Direct arterial pressure measurement** with intraarterial catheters offers continuous measurement of arterial pressures and waveforms and easy, painless access for ABG measurement. Arterial cannulation is warranted in patients with hemodynamic instability and in those who require frequent blood gas analyses. The most common sites of insertion are the radial, femoral, dorsalis pedis, and axillary arteries. The radial artery usually is chosen because of its accessibility and generally good collateral blood flow. Axillary and femoral artery catheterization should be avoided in infants, because occlusion may cause extremity ischemia and subsequent deformity.

1. **Insertion technique.** For radial artery cannulation, place the dorsal aspect of the patient's forearm on an arm board with his or her wrist slightly extended over a gauze roll. For femoral cannulation, the patient should be fully supine. Use sterile technique and a standard surgical preparation; standard insertion kits are generally available. After administration of a local anesthetic, arterial cannulation is performed using one of three techniques.
 - a. **Direct insertion of a catheter** over a needle inserted at a 30-degree angle. Once a flash of blood is obtained, the needle is advanced 1–2 mm so that the needle and the cannula are entirely within the lumen before the cannula is advanced over the needle and retrograde up the arterial lumen. The needle is then withdrawn.
 - b. Using the **Seldinger technique**, the artery can be reached with a needle, through which a guidewire can be advanced into the vessel. After needle removal, the catheter can be advanced over the wire into the lumen. This technique is particularly useful for femoral arterial cannulation.
 - c. **Surgical cutdown** is occasionally required if percutaneous attempts have failed.
2. **Complications.** Arterial cannulation carries a low risk of complication. Thrombosis or clot formation in the artery occurs, most commonly in the smaller peripheral arteries, but usually recanalization occurs after catheter removal. The extremity distal to the catheter should be assessed frequently, and the catheter should be removed immediately if there is evidence of ischemia. Infectious complications include local cellulitis and bacteremia, which may result from catheter colonization or from contamination of the fluid-filled monitoring system. The insertion site should be inspected frequently and flush systems changed every 72 hours. Local infection and bacteremia generally resolve after catheter removal. Other complications include emboli from the catheter tip, hematoma at the insertion site, and hemorrhage from an open system.

V. PA catheterization. PA (also called *Swan-Ganz*) catheters are used to determine cardiac filling pressures, left and right ventricular function, changes in hemodynamic status, and responses to treatment with fluid and cardioactive agents.

A. **Insertion technique.** A variety of catheter constructions are available. The most commonly used construction is a four-lumen catheter. In this device, one lumen allows for balloon inflation, a second (at the tip) allows for PA pressure measurements and blood sampling, another (30 cm from the tip) is for right atrial measurements, and a fourth contains a thermistor used to measure core body temperature and CO by thermodilution. Five-lumen (so-called VIP) catheters also are available for additional central venous access. Other modifications include lumens that allow for cardiac pacing, continuous mixed venous oxygen saturation measurement, and right ventricular ejection fraction measurement.

Continuous ECG, blood pressure monitoring, and peripheral intravenous access are required. First, establish central venous access with a No. 8.5 Fr. introducer-type catheter (see [Chapter 42](#)). The connection ports of the PA catheter are then handed off to an assistant, who connects these ports to pressure transducers, flushes the catheter lumens with saline, and tests the balloon (1.5 cc of air recommended for a No. 7 Fr. catheter) to ensure that the inflated balloon completely covers the catheter tip. To establish the reliability of the PA pressure waveform, look at the waveform while doing the following:

- Flush the lumen (to rule out dampening).
 - Shake the tip of the catheter (pressure oscillations should be readily apparent).
 - Place the distal tip of the catheter at the level of the right atrium (to ensure zeroing).
- B. Place and expand the sterile protective sleeve over the catheter. Insert the catheter through the introducer to 15–20 cm; there should be no resistance, and the pressure tracing should be typical of CVP ([Fig. 11-1](#)). Inflate the balloon completely; again, there should be no resistance and no change in the pressure waveform. While watching the waveform, advance the catheter smoothly. The position of the catheter tip is determined by recognition of the characteristic waveforms ([Fig. 11-1](#)) and by the depth of insertion (the distance between black marks on the catheter is 10 cm). The catheter is advanced until a PA “wedge” tracing is obtained (typically at 40–60 cm). This occurs when the balloon occludes the distal PA segment; with absent flow, pressures rapidly equilibrate, and the pressure tracing from the distal opening measures left atrial pressure. When the balloon is deflated, the PA pressure tracing should return. Slowly reinflate the balloon; if PA occlusion occurs at less than 1.5 cc, deflate the balloon and withdraw the catheter 1–2 cm. Repeat this maneuver until the wedge tracing occurs with 1.5-cc inflation volume. Expand the protective sleeve over the exposed length of the catheter and secure it to the introducer. This keeps the enclosed catheter sterile for subsequent manipulations. Confirm placement of the catheter and assess for pneumothorax with a CXR.

Fig. 11-1. Representation of the characteristic pressure waveforms recorded as the pulmonary artery catheter is advanced through the right atrium (RA) and right ventricle (RV) into the pulmonary artery (PA) and to the pulmonary capillary wedge (PCW) position. ECG, electrocardiogram. (From Cummins RO, ed. *Textbook of advanced cardiac life support*. Dallas: American Heart Association, 1994, with permission. Copyright © American Heart Association.)

Do not inflate the balloon beyond the volume that achieves wedging, and *do not* leave the balloon inflated for longer than 10 seconds. *Never* withdraw the catheter with the balloon inflated. Frequent movement of the catheter tip with changes in patient position and fluid status requires that the physician reestablish correct positioning of the catheter at least once a day.

C. **Complications** that are associated with central venous access are described in [Chapter 8](#). PA catheter balloon rupture exposes the patient to the risk of air and balloon fragment emboli. **Balloon rupture** should be suspected when air inflated into the balloon does not return; the diagnosis is confirmed if blood can be aspirated from the balloon port. If either of these occurs, the catheter should be removed immediately. **Pulmonary infarction**, caused by peripheral migration of the catheter tip with persistent wedging of the tip in the PA, can be avoided by careful monitoring of PA waveforms and daily CXR to document catheter position. **PA perforation** presents with hemoptysis, typically after balloon inflation. Management of this serious complication includes placement of the patient with his or her involved side (known from that day's CXR) dependent and emergent thoracic surgical consultation. **Thrombus** and central venous occlusion can occur and are rare causes of pulmonary embolism. Atrial and ventricular **arrhythmias** occur commonly during insertion of PA catheters and usually are self-limited. The most frequent persistent cardiac rhythm disturbance is catheter-induced right bundle-branch block; hence, PA catheters are relatively contraindicated in patients with complete left bundle-branch block. Catheter position should be assessed if catheter-induced ectopy persists; the catheter should be removed at any sign of hemodynamic compromise secondary to ventricular dysrhythmia.

VI. Respiratory monitoring

- A. **Pulse oximetry** provides quantitative, continuous assessment of SaO₂ and closely correlates with SaO₂ obtained by ABG determination. Probe malposition, motion, hypothermia, vasoconstriction, and hypotension may result in poor signal detection and unreliable measurements. Nail polish, dark skin, and elevated serum lipids falsely lower the SaO₂ measurement, whereas elevated carboxyhemoglobin or methemoglobin falsely raises the measurements. Despite these caveats, pulse oximetry should be used in critically ill patients with respiratory insufficiency or failure.
- B. **Capnography** provides quantitative, continuous assessment of expired CO₂ concentrations. Measurements of expired end-tidal PCO₂ (ETCO₂) are less reliable than oximetry in critically ill patients. However, the gradient between PaCO₂ and ETCO₂ measurements can be used to follow trends. A rise in ETCO₂ can indicate a decrease in alveolar ventilation or an increase in CO₂ production, as is seen with overfeeding, sepsis, fever, exercise, or acute increases in CO. A fall in ETCO₂ indicates either an increase in alveolar ventilation or an increase in dead space, as is seen with massive pulmonary embolism or air embolism, ET tube or main-stem bronchus obstruction, ventilator circuit leak, or a sudden drop in CO.

Upper Gastrointestinal Hemorrhage Prophylaxis

Patients in the ICU are at increased risk for stress-induced mucosal ulceration and resultant GI hemorrhage. Risk factors include head injury (Cushing's ulcers), burns (Curling's ulcers), requirement for mechanical ventilation, previous history of peptic ulcer disease, use of nonsteroidal antiinflammatory drugs or steroids, or the presence of shock, renal failure, portal hypertension, or coagulopathy. Strong data exist to support the use of drugs to maintain mucosal integrity in these patients at increased risk. In an evidence-based review of discordant metaanalyses, H₂-receptor antagonists (cimetidine, ranitidine, famotidine) were found to reduce significantly the incidence of clinically important GI bleeding in critically ill patients (*JAMA* 275:308, 1996). **Sucralfate**, a cytoprotective agent that does not alter gastric pH, is also used because of reports suggesting that alteration of gastric pH may predispose to bacterial colonization and increased risk of ventilator-associated pneumonia. However, in a multicenter, randomized, blinded, placebo-controlled trial (*N Engl J Med* 388:791, 1998), mechanically ventilated patients who received **ranitidine** (50 mg i.v. every 8 hours) had significantly lower rates of clinically important GI hemorrhage than those treated with sucralfate (1 g p.o. or per nasogastric tube every 6 hours). No significant difference was found between the rates of ventilator-associated pneumonia in the two groups. (See [Chapter 13](#) for a discussion of the management of patients with GI hemorrhage.)

Renal Dysfunction

I. **Etiology and diagnosis.** Renal dysfunction commonly presents as progressive oliguria in the setting of increased renal function indices [blood urea nitrogen (BUN) and serum creatinine]. This can progress to renal failure and anuria (urine output <100 mL per day) that require renal replacement therapy (approximately 5% of all ICU admissions). Renal insufficiency can also present as polyuria when decreased renal tubular function (fluid resorption) is not coupled with decreased glomerular filtration (“high-output” renal failure). Traditionally, the etiology of renal dysfunction has been divided into prerenal, intrarenal, and postrenal causes (*N Engl J Med* 334:1448, 1996). A careful history and review of the medical record are critical to making the correct diagnosis.

- A. **Prerenal.** The glomerular and tubular function of the kidneys is normal, but clearance is limited as a result of decreased renal blood flow. This is the most common cause (40–70%) of renal insufficiency in the surgical ICU, frequently as a result of perioperative hypotension or intraoperative manipulation (e.g., suprarenal aortic cross-clamping). The rise in the BUN typically is greater than that of the serum creatinine (BUN to creatinine ratio >20). The concentrating ability of the kidneys is normal, and, thus, the urine osmolality (>500 mOsm) and fractional excretion of sodium (FE_{Na} >1) are normal.
- B. **Intrarenal.** Tubular injury is most often caused by ischemia or toxins. Nephrotoxins commonly encountered by ICU patients include aminoglycosides, intravenous radiopaque contrast agents, amphotericin, and chemotherapeutic drugs (e.g., cisplatin). Those with preexisting renal disease or diabetes are particularly

susceptible. Intravenous hydration with saline-containing solutions (0.9% or 0.45% saline) before and during the administration of nephrotoxins should be used to decrease the incidence of renal insufficiency in patients at risk (*N Engl J Med* 331:1416, 1994). Because the concentrating ability of the tubules is compromised, the urine osmolality is low (<350 mOsm) and the FE_{Na} is greater than 1. Urinalysis and microscopic analysis of the urinary sediment may yield additional information about tubular pathology.

- C. **Postrenal.** Bilateral obstruction of urinary flow can be caused by direct intraoperative injury or manipulation, prostatic hypertrophy, coagulated blood, or extrinsic compression (e.g., tumors). Urinary catheter malfunction must always be ruled out, typically by flushing the catheter with sterile saline. Ultrasound examination of the urinary system is used to rule out hydronephrosis.
- D. **Abdominal compartment syndrome** results from massive tissue (bowel) edema within the abdominal compartment or retroperitoneal hemorrhage, frequently as a complication of severe trauma. Increased intraabdominal pressure decreases renal perfusion and retards renal venous and urinary outflow, inducing renal injury by a combination of pre-, intra-, and postrenal insults. Assessment of urinary bladder pressure via a Foley catheter serves as an indirect but accurate measure of intraabdominal pressure (*J Trauma* 45:597, 1998). Pressure greater than 25 cm H₂O demands intervention, typically reexploration (convert mm Hg to cm H₂O by multiplying mm Hg by 1.3).

II. Treatment

- A. **Supportive measures.** Initial therapy should be directed at minimizing ongoing renal injury by optimizing renal perfusion and discontinuing potentially nephrotoxic agents. The former is usually accomplished by judicious volume resuscitation. In the event that fluid resuscitation does not improve low urine output (<0.5 mL/kg per hour), measurement of CVP or pulmonary capillary wedge pressure can be used to guide fluid resuscitation and optimization of CO. Although frequently advocated, the use of low-dose dopamine or diuretics (furosemide, mannitol) to increase urine output per se has not been shown to improve either renal function or outcome. The dosages or medication eliminated by the kidney should be adjusted for the degree of renal insufficiency. The treatment of electrolyte (hyperkalemia) and acid-base disorders (metabolic acidosis) that accompany renal failure are covered in [Chapter 4](#).
- B. **Renal replacement therapy.** Indications include complications of renal dysfunction that fail medical management, such as hypervolemia, uremia (pericarditis or encephalopathy), severe acidemia, refractory hyperkalemia, or platelet dysfunction. Decisions about when and how to initiate renal replacement therapy remain the subject of controversy and ongoing clinical trials (*New Horizons* 3:760, 1995). Both types of dialysis discussed in the following sections require placement of a large dialysis catheter into a central vein.
 - 1. **Intermittent.** Because peritoneal dialysis is usually impractical in the surgical ICU, intermittent hemodialysis is the method of choice. Some hemodynamic impairment usually ensues as a result of rapid, large shifts of fluid from the intravascular compartment through the dialysis filter. This is usually well tolerated, but it can induce hemodynamic deterioration (hypotension or dysrhythmias) by decreasing myocardial preload. Judicious use of a vasopressor medication, such as phenylephrine, may be helpful in this regard.
 - 2. **Continuous** (*N Engl J Med* 336:1303, 1997). Continuous venovenous hemodialysis (CVVHD) is used in patients with preexisting hemodynamic instability, usually in the setting of shock. CVVHD decreases the rate of fluid shifts and, thus, improves hemodynamic stability while permitting more precise control of fluid and electrolyte repletion. The disadvantage of this type of dialysis is that CVVHD requires constant systemic anticoagulation to prevent clotting of blood in the filter and continuous, sophisticated nursing surveillance.

Transport of the Critically Ill Patient

Organ decompensation and machine malfunctions occur with disproportionately high frequency during transport of critically ill patients. Adherence to the following guidelines helps to minimize these events.

I. Transport only stable patients. The only exception to this rule is the patient who requires immediate, life-saving, operative, or therapeutic intervention.

II. Recognize cul-de-sacs. Elevators and X-ray suites are places where lines and tubes commonly are dislodged, where patient deterioration can go unrecognized, and where it is physically difficult to get to the patient. Meticulous attention to catheters, lines, and tubes prevents dislodgment and malfunction.

III. Maintain airway patency. If the airway or adequacy of spontaneous ventilation is questionable, intubate the patient before transport. If the intubated patient is agitated or combative, administer sedative or neuromuscular blocking agents before moving him or her.

IV. Ensure adequate alveolar ventilation and oxygenation. Significant variation may be seen from baseline blood gases during the change from mechanical to manual (bagged) ventilation. Ensure that the patient is adequately ventilated with a bag, and maintain satisfactory SaO₂ before you leave the safe environment of the ICU. Remember that PEEP-dependent patients commonly require a special bag equipped with a PEEP valve.

V. Check the oxygen tank. The 2-ft-high oxygen cylinders that are most commonly used for transport hold 600 L of oxygen. When the tank is fully charged to 2,200 lb per sq in., approximately 1 hour of oxygen is available when the flow rate is set at 10 L per minute.

VI. Enlist enough help to push the bed, intravenous poles, and other equipment; ventilate the intubated patient; commandeer elevators; and clear pathways. Notify the receiving facility of your imminent arrival and of the urgency of your patient's needs.

VII. Travel with the monitors you need. (e.g., ECG, arterial pressure, oximeter), but realize that malfunctions and erroneous data are common. A hand on the femoral pulse frequently is the most rapid and reliable source of information.

VIII. Have backup equipment and personnel close at hand. The travel bag should contain a tight-fitting mask, an oral airway, and vasoactive drugs (the usual triad is epinephrine, atropine, and lidocaine) and must move with the patient. Familiarize yourself with its contents. If the patient is dependent on an antiarrhythmic agent, vasopressor, or other drug that must be infused continuously, do not move the patient without two working intravenous lines in place.

Commonly Used Drugs

[Table 11-9](#) lists drugs that are commonly used in the ICU and their dosages.

Table 11-9. Drugs commonly used in the intensive care unit

12 Esophagus

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Disorders of the Esophagus

I. Hiatal hernia

II. GER

III. Barrett's esophagus

IV. Esophageal carcinoma

V. Esophageal perforation

VI. Esophageal diverticula

VII. Functional esophageal disorders

VIII. Esophageal strictures

IX. Benign esophageal neoplasms

X. Caustic ingestion

XI. Complications of esophageal surgery

Disorders of the Esophagus

I. Hiatal hernia. The distal esophagus normally is held in position by a fusion of the endothoracic and endoabdominal fasciae at the diaphragmatic hiatus called the *phrenoesophageal membrane*. A hiatal hernia is present when a lax or defective phrenoesophageal membrane allows protrusion of the stomach up through the esophageal hiatus of the diaphragm.

- A. **Epidemiology.** A hiatal hernia is the most common abnormality reported on upper gastrointestinal (GI) radiographic barium study. An estimated 10% of the adult population in the United States have a hiatal hernia. The condition occurs most commonly in women in their fifth and sixth decades. Most hiatal hernias are asymptomatic; however, an estimated 5% of patients with a hiatal hernia have symptoms that are related to persistent gastroesophageal (GE) reflux disease.
- B. **The type of hiatal hernia** is defined by the location of the GE junction and the relationship of the stomach to the distal esophagus.
 1. In **type I** or **sliding** hiatal hernia, the phrenoesophageal membrane is intact but lax, thereby allowing the distal esophagus and gastric cardia to herniate through the esophageal hiatus. The GE junction is therefore located above the diaphragm. This is the most common type and is usually asymptomatic.
 2. A **type II** or **paraesophageal** hiatal hernia occurs when a focal defect is present in the phrenoesophageal membrane, usually anterior and lateral to the esophagus, which allows a protrusion of peritoneum to herniate upward alongside the esophagus through the esophageal hiatus. The GE junction remains anchored within the abdomen, whereas the greater curvature of the stomach rolls up into the chest alongside the distal esophagus. Eventually, most of the stomach can herniate. Because the stomach is anchored at the pylorus and cardia, however, the body of the stomach undergoes a 180-degree organoaxial rotation and ends up as an upside-down, intrathoracic stomach when it is herniated.
 3. **Type III** represents a **combination** of types I and II. This type is more common than is a pure type II and is characterized by herniation of the greater curvature of the stomach and the GE junction into the chest. Patients thus affected are exposed to the problems and risks of both types of hernias.
 4. A **type IV** hiatal hernia is defined as the condition in which abdominal organs other than or in addition to the stomach herniate through the hiatus. Typically, these hernias are large and contain colon or spleen in addition to the stomach within the chest.
- C. **Symptoms and complications** in patients with **sliding** (type I) hiatal hernias are related to associated GE reflux (GER; see [section II](#)). **Paraesophageal and combined** (types II, III, and IV) hernias frequently produce postprandial pain or bloating, early satiety, breathlessness with meals, and mild dysphagia related to compression of the distal esophagus by the adjacent herniated stomach. The herniated gastric pouch is susceptible to volvulus, obstruction, and infarction and can develop ischemic ulcers with frank or occult bleeding (secondary to longitudinal ulcers, termed *Cameron's ulcers*), perforation, or gangrene.
- D. **Diagnosis and evaluation**
 1. **Chest X-ray.** The finding of an air-fluid level in the posterior mediastinum on the lateral X-ray suggests the presence of a hiatal hernia. Differential diagnosis includes mediastinal cyst, abscess, or a dilated obstructed esophagus (as is seen in end-stage achalasia).
 2. A **barium swallow** confirms the diagnosis and defines any coexisting esophageal abnormalities, including strictures or ulcers, and is the diagnostic study of choice. The positions of the GE junction and proximal stomach define the type of hiatal hernia.
 3. **Esophagogastroduodenoscopy (EGD)** is indicated in patients with symptoms of reflux or dysphagia to determine the degree of esophagitis, presence of a stricture, Barrett's esophagus, or a coexisting abnormality. EGD also establishes the location of the GE junction in relation to the hiatus. A sliding hiatal hernia is present when 2 cm or more of gastric mucosa is present between the diaphragmatic hiatus and the mucosal squamocolumnar junction.
 4. **Esophageal manometry** to evaluate esophageal motility is warranted in patients who are being considered for operative repair.
- E. **Management**
 1. **Asymptomatic sliding hernias** require no treatment.
 2. **Patients with sliding hernias and GER with mild esophagitis** should undergo an initial trial of medical management.
 3. **Patients who fail to obtain symptomatic relief with medical therapy or who have severe esophagitis** should undergo esophageal testing to determine their suitability for an antireflux procedure and hiatal hernia repair.
 4. **Patients who do not experience reflux but have symptoms related to their hernia** (chest pain, intermittent dysphagia, or esophageal obstruction) should undergo hiatal hernia repair.
 5. **All patients who are found to have a type II, III, or IV hiatal hernia and who are operative candidates should be considered for repair.** Medically treated patients with a paraesophageal hernia, even when asymptomatic, have nearly a 30% incidence of death from the development of a catastrophic complication (*J Thorac Cardiovasc Surg* 53:33, 1967). Operative repair can be performed through either an abdominal or thoracic approach and consists of reduction of the hernia, resection of the sac, and closure of the hiatal defect. In combined (type III) hernias, the esophagus frequently is shortened, and therefore a thoracic approach is preferred.
 6. **Paraesophageal hiatal hernias** are associated with a 60% incidence of GER. Furthermore, the operative dissection may lead to postoperative GER in previously asymptomatic patients. Therefore, an antireflux procedure should be performed at the time of hiatal hernia repair.

II. GER

- A. **Prevalence.** GER is a normal event after a meal and during belching. Normally, refluxed gastric juice is cleared rapidly from the distal esophagus. Symptoms of heartburn and excessive regurgitation are relatively common in the United States, however, occurring in approximately 7% of the population on a daily basis and in 33% at least once a month. Often, these individuals have X-ray evidence of a hiatal hernia. Reflux and hiatal hernia are not necessarily related, and each can occur independently.
- B. **Pathophysiology** in GER relates to abnormal exposure of the distal esophagus to refluxed stomach contents. In 60% of patients, a mechanically defective lower-esophageal sphincter (LES) is responsible for the GER. The sphincter function of the LES depends on the integrated mechanical effect of the sphincter's intramural pressure and length and the length of esophagus exposed to intraabdominal positive pressure. Other etiologies of GER are inefficient esophageal clearance of refluxed material, fixed gastric outlet obstruction, functional delayed gastric emptying, increased gastric acid secretion, and inappropriate relaxation of the LES.
- C. The classic **symptom** of GER is posturally aggravated substernal or epigastric burning pain that is readily relieved by antacids. Additional common symptoms include regurgitation or effortless emesis, dysphagia, and excessive flatulence. Atypical symptoms may mimic laryngeal, respiratory, cardiac, biliary, pancreatic, gastric, or duodenal disease.
- D. **Diagnosis and evaluation**
 1. **Contrast radiography (upper GI)** demonstrates spontaneous reflux in only approximately 40% of patients with GER. However, it documents the presence or absence of hiatal hernia; can demonstrate some complications of reflux, such as esophageal stricture and ulcers; and is an appropriate initial study. The study should include a full view of the esophagus as well as a complete evaluation of the stomach, pylorus, and duodenum.
 2. **EGD** is indicated in patients with symptoms of GER to evaluate for esophagitis and the presence of Barrett's changes. **Esophagitis** is a pathologic diagnosis, but an experienced endoscopist can readily distinguish the more advanced stages. Four general grades of esophagitis occur.
 - a. **Grade I:** Normal or reddened mucosa
 - b. **Grade II:** Superficial mucosal erosions and some ulcerations
 - c. **Grade III:** Extensive ulceration with multiple, circumferential erosions with luminal narrowing; possible edematous islands of squamous mucosa present,

producing the so-called cobblestone esophagitis

d. **Grade IV:** Fibrotic peptic stricture, shortened esophagus, columnar lined esophagus

3. **Esophageal manometric testing** is appropriate in the patient with reflux symptoms once surgery is being considered. Manometry defines the location and function of the LES and helps to exclude achalasia, scleroderma, and diffuse esophageal spasm from the differential diagnosis. Characteristics of a manometrically abnormal LES are (1) a pressure of less than 6 mm Hg, (2) an overall length of less than 2 cm, or (3) an abdominal length of less than 1 cm. Values below these are abnormal, and a patient with one or more of these abnormal values has a 90% probability of having reflux. Manometry also assesses the adequacy of esophageal contractility and peristaltic wave progression as a guide to the best antireflux procedure for the patient.
 4. **Esophageal pH testing** over a 24-hour period is regarded as the gold standard in the diagnosis of GER. It is now used mainly when the data from the remainder of the evaluation are equivocal and diagnosis of reflux is in doubt. The 24-hour pH testing can be performed on an outpatient or ambulatory basis: The patient has an event button to record symptoms and keeps a diary of body position, timing of meals, and other activities. This allows correlation of symptoms with simultaneous esophageal pH alterations. A score is derived based on the frequency of reflux episodes and the time required for the esophagus to clear the acid. Score values that fall outside two standard deviations from the mean of values obtained from normal volunteers are considered abnormal. This test has a 90% sensitivity and a 90% specificity for diagnosing or excluding reflux (*J Thorac Cardiovasc Surg* 79:656, 1980).
 5. A **gastric emptying study** can be useful in evaluating patients with reflux and symptoms of gastroparesis.
- E. **Complications.** Approximately 20% of patients with GER have complications, including esophagitis, stricture, or Barrett's esophagus. Other less common complications include acute or chronic bleeding and aspiration.

F. Treatment

1. **Medical treatment** aims to reduce the duration and amount of esophageal exposure to gastric contents and to minimize the effects on the esophageal mucosa.
 - a. Patients are instructed to remain upright after meals, avoid postural maneuvers (bending, straining) that aggravate reflux, and sleep with the head of the bed elevated 6–8 in.
 - b. **Dietary alterations** are aimed at maximizing LES pressure, minimizing intragastric pressure, and decreasing stomach acidity. Patients are instructed to avoid fatty foods, alcohol, caffeine, chocolate, peppermint, and smoking and to eat smaller, more frequent meals. Obese patients are instructed to lose weight, avoid tight-fitting garments, and begin a regular exercise program. In addition, anticholinergics, calcium channel blockers, nitrates, beta-blockers, theophylline, alpha-blockers, and nonsteroidal antiinflammatory medications may exacerbate reflux and should be replaced with other preparations or reduced in dosage if possible.
 - c. **Pharmacologic therapy** is indicated in patients who do not improve with postural or dietary measures. The goals are to lower gastric acidity or enhance esophageal and gastric clearing while increasing the LES resting pressure.
 1. **Antacids** neutralize stomach acidity and thus raise intragastric pH. Many patients self-medicate with these agents for many years before seeking professional attention.
 2. **H₂-receptor antagonists** lower gastric acidity by decreasing the amount of acid that the stomach produces. Proton pump inhibitors act by selective noncompetitive inhibition of the H⁺/K⁺ pump on the parietal cell and are more effective than H₂ antagonists in healing esophagitis (*Aliment Pharmacol Ther* 4:145, 1990).
 3. **Prokinetic agents**, such as metoclopramide (dopaminergic antagonist), can decrease GER by increasing the LES tone and accelerating esophageal and gastric clearance.
 - d. **Endoluminal suturing** is a recently developed approach to the treatment of GER that is appealing because it can be performed without an incision. It involves the use of a sewing machine mounted on a standard gastroscope to create an "internal plication" of the stomach just below the LES (*Gastrointest Endosc* 53:416, 2001). Criteria for patient selection and long-term efficacy have yet to be established.
2. **Surgical treatment** should be considered in patients who have symptomatic reflux, manometric evidence of a defective LES, and failure to achieve relief with maximal medical management. Alternatively, surgical therapy should be considered in symptomatic patients who have achieved relief with medical therapy but to whom the prospect of a lifetime of medicine is undesirable (i.e., because of cost, side effects, inconvenience, or compliance). Surgical treatment consists of either a transabdominal or transthoracic antireflux operation to reconstruct a competent LES and a crural repair to maintain the reconstruction in the abdomen.
 - a. A **transabdominal approach** is preferred in most patients, except when a shortened esophagus is present because the shortened esophagus cannot be adequately pulled into the abdomen for the fundoplication. A shortened esophagus should be suspected when a stricture is present and in those who have had a failed antireflux procedure. The transabdominal approach is recommended for those with a coexisting abdominal disorder, a prior thoracotomy, or severe respiratory disorder.
 1. **Nissen fundoplication** is the most commonly performed procedure for GER. It consists of a 360-degree fundic wrap via open or laparoscopic technique. Long-term results in several series of open procedures are excellent, with 10-year freedom from recurrence of more than 90%. Short-term results of the laparoscopic approach are as good as the open-repair results for relief of GER symptoms, with concomitant shorter hospital stay, better respiratory function, and decreased pain postoperatively (*Br J Surg* 87:873, 2000). The full fundoplication in this repair is very effective at preventing reflux but, compared to the other procedures, is associated with a slightly higher incidence of an inability to vomit, gas bloating of the stomach, and dysphagia. During surgery, care must be taken to ensure that the wrap is short, loose, and placed appropriately around the distal esophagus to minimize the incidence of these complications.
 2. The **Hill posterior gastropexy** anchors the GE junction posteriorly to the median arcuate ligament and creates a partial or 180-degree imbrication of the stomach around the right side of the intraabdominal esophagus. Hill recommended using intraesophageal manometry during placement of the sutures to achieve a pressure of 50 mm Hg in the distal esophagus. With this technique, the incidence of recurrent hiatal hernia is low and the incidence of recurrent or persistent reflux is variable.
 3. The **Toupet fundoplication** is a 210-to 270-degree posterior wrap, with the wrapped segment sutured to the crural margins and to the anterolateral esophageal wall.
 - b. A **transthoracic approach** is recommended in patients with esophageal shortening or stricture, coexistent motor disorder, obesity, coexistent pulmonary lesion, or prior antireflux repair.
 1. **Nissen fundoplication** can be done via a transthoracic approach, with results similar to those obtained with a transabdominal approach.
 2. The **Belsey Mark IV repair** consists of a 240-degree fundic wrap around 4 cm of distal esophagus. In cases of esophageal neuromotor dysfunction, it produces less dysphagia than may accompany a 360-degree (Nissen) wrap. Furthermore, the ability to belch is preserved, thereby avoiding the gas-bloat syndrome that may occur after a complete (360-degree) wrap. Careful 10-year follow-up demonstrates a good long-term result in 85% of patients (*J Thorac Cardiovasc Surg* 53:33, 1967).
 3. **Collis gastroplasty** is a technique that is used to lengthen a shortened esophagus. To minimize tension on the antireflux repair, a gastric tube is formed from the upper lesser curvature of the stomach in continuity with the distal esophagus. The antireflux repair then is constructed around the gastroplasty tube. A gastroplasty should be considered preoperatively in patients with obvious or subtle esophageal shortening, such as those with gross ulcerative esophagitis or stricture, failed prior antireflux procedure, or total intrathoracic stomach (*Ann Surg* 206:473, 1987). However, in many of these patients the esophagus can be adequately mobilized to allow more than 3 cm of intraabdominal esophagus and thereby avoid the need to lengthen the esophagus. Development of an angled endoscopic stapler has made laparoscopic Collis gastroplasty technically feasible.

III. Barrett's esophagus is defined as a metaplastic transformation of esophageal mucosa. The metaplastic epithelium may be of either gastric or intestinal type. To qualify for the term *Barrett's*, the metaplastic epithelium either must extend at least 3 cm above the normal GE junction or, in shorter segments, must have intestinal-type metaplasia on biopsy. The columnar epithelium of Barrett's esophagus may replace the normal squamous epithelium circumferentially, or it may be asymmetric and irregular. Barrett's esophagus evolves as a result of chronic GER.

- A. **Prevalence.** Barrett's esophagus is diagnosed in approximately 2% of all patients undergoing esophagoscopy and in 10–15% of patients with esophagitis. Autopsy studies suggest that the actual prevalence is much higher because many patients are asymptomatic and remain undiagnosed. Most patients diagnosed with Barrett's esophagus are middle-aged white men.
- B. **Symptoms** are a result of GER. Approximately 50% of patients with endoscopically proven Barrett's have associated heartburn, 75% have dysphagia, and 25% have bleeding (*Ann Surg* 198:554, 1983).
- C. **Pathologic subtypes.** Three pathologic types of metaplastic columnar epithelium may occur together or independently.
 1. The **modified fundic or body type** is the least common and contains parietal and chief cells identical to those in the gastric body mucosa.
 2. The **junctional type** has mucous glands and resembles the gastric cardia.
 3. The **specialized or intestinal type** has goblet cells scattered among the columnar epithelial cells, is the most common type, and has a high association with the subsequent development of adenocarcinoma.
- D. **Diagnosis.** The diagnosis of Barrett's esophagus may be suggested on X-ray by the presence of a hiatal hernia (associated with 80% of Barrett's esophagus) with esophagitis and an esophageal stricture. Confirmation of the diagnosis requires endoscopy, with careful correlation between the endoscopic appearance and histologic findings of mucosal biopsies.
- E. **Complications**

1. **Esophageal ulceration and stricture** are more likely to occur in patients with Barrett's esophagus than in those with GER alone. This most likely reflects the more severe nature of the GER in patients with Barrett's esophagus.
 - a. **Barrett's ulcers** are distinctly different from the common erosions seen in esophagitis in that they penetrate the metaplastic columnar epithelium in a manner similar to that seen in gastric ulcers. They occur in up to 50% of patients with Barrett's esophagus and, like gastric ulcers, can cause pain, bleed, obstruct, penetrate, and perforate.
 - b. **Benign strictures** occur in 30–50% of patients with Barrett's esophagus. The stricture is located at the squamocolumnar junction, which in Barrett's esophagus is not the esophagogastric junction. The strictures secondary to Barrett's are located in the middle or upper esophagus, unlike peptic strictures not associated with Barrett's esophagus, which usually occur in the distal esophagus.
2. **Dysplasia.** The metaplastic columnar epithelium of Barrett's esophagus is prone to development of areas of dysplasia that can be detected only by biopsy. Pathologically, dysplasia is graded as low or high grade, with high grade being pathologically indistinguishable from carcinoma *in situ*.
3. **Malignant degeneration** from benign to dysplastic to malignant epithelium has been demonstrated in Barrett's esophagus. Low-grade dysplasia is present in 5–10% of patients with Barrett's esophagus and can progress to high-grade dysplasia and malignancy.
4. **Adenocarcinomas** of the gastric type that arise within the esophagus above the normal GE junction are characteristic of malignant degeneration in Barrett's esophagus. The risk of development of adenocarcinoma in Barrett's esophagus is 50–100 times that of the general population. In several long-term series, the incidence of malignant degeneration in Barrett's esophagus has been estimated at between 1 in 50 and 1 in 400 patient-years of follow-up.

F. Treatment

1. Uncomplicated Barrett's esophagus in **asymptomatic** patients requires no specific therapy, but endoscopic surveillance and biopsy should be performed at least annually. Neither medical nor surgical treatment of reflux has been demonstrated to reverse the columnar metaplasia of Barrett's esophagus. However, elimination of reflux with an antireflux procedure may halt progression of the disease, heal ulceration, and prevent stricture formation.
2. Uncomplicated Barrett's esophagus in **symptomatic** patients should be treated using the same principles that apply to patients with GER without Barrett's esophagus. In addition, they should have annual surveillance endoscopy with biopsy.
3. **Barrett's ulcers** usually heal with medical therapy. Frequently, 8 weeks of treatment with an H₂-receptor antagonist or proton pump inhibitor is necessary to achieve complete healing. Recurrence of ulcers is common after discontinuation of therapy. Ulcers that fail to heal despite 4 months of medical therapy are an indication for rebiopsy and antireflux surgery.
4. **Strictures** associated with Barrett's esophagus usually are successfully managed with periodic esophageal dilation combined with medical management. Recurrent or persistent strictures warrant an antireflux operation combined with intraoperative stricture dilation. After surgery, several dilations can be required to maintain patency during the healing phase. Rarely, undilatable strictures require resection.
5. **Dysplasia** on biopsy of Barrett's esophagus indicates that the patient is at risk for the development of adenocarcinoma.
 - a. **Low-grade dysplasia** requires frequent (every 3–6 months) surveillance esophagoscopy and biopsy. Medical therapy for GER is recommended in these patients, even when asymptomatic.
 - b. **High-grade dysplasia** is pathologically indistinguishable from carcinoma *in situ* and is an indication for esophagectomy. Patients who undergo esophagectomy for high-grade dysplasia have up to a 22–73% chance of having an unidentified focus of invasive carcinoma present in the resected esophagus. Cure rates of nearly 100% can be expected in patients with cancer that is limited to the mucosa who undergo esophagectomy (*J Thorac Cardiovasc Surg* 108:813, 1994).
6. **Adenocarcinoma** in patients with Barrett's esophagus is an indication for esophagogastrectomy. Early detection offers the best opportunity to improve survival after resection, which overall is 20% at 5 years.

IV. Esophageal carcinoma

- A. **Epidemiology.** Carcinoma of the esophagus represents 1% of all cancers in the United States and causes 1.8% of cancer deaths. The two principal histologies are adenocarcinoma and squamous cell carcinoma.
 1. **Risk factors** for squamous cell esophageal cancer include African-American race, alcohol and cigarette use, tylosis, achalasia, caustic esophageal injury, Plummer-Vinson syndrome, nutritional deficiencies, and ingestion of nitrosamines and fungal toxins. Geographic location also represents a risk factor, likely as a result of local dietary customs, with a high incidence noted in certain areas of China, South Africa, Iran, France, and Japan.
 2. **Risk factors** for adenocarcinoma of the esophagus include Caucasian race, chronic reflux symptoms, and Barrett's esophagus.
- B. **Pathology**
 1. **Squamous cell carcinoma** was previously the most common cell type. It tends to be multicentric and most frequently involves the middle third of the esophagus.
 2. **Adenocarcinoma** now comprises more than 50% of esophageal carcinomas and is the carcinoma with the greatest rate of increase in the United States. It is less likely to be multicentric; however, it commonly has extensive proximal and distal submucosal invasion. Adenocarcinoma most commonly involves the distal esophagus.
 3. **Less common malignant esophageal tumors** include small cell carcinoma, melanoma, leiomyosarcoma, and, rarely, other sarcomas, lymphoma, or esophageal involvement by metastatic cancer.
- C. Most patients with early-stage disease are asymptomatic or may have symptoms of reflux. **Symptoms** of esophageal cancer are dysphagia, odynophagia, and weight loss. Symptoms that are suggestive of unresectability include hoarseness, abdominal pain, persistent back or bone pain, hiccups, and respiratory symptoms (cough, aspiration pneumonia), which suggest possible esophagorespiratory fistula formation. Approximately 50% of presenting patients have unresectable lesions or distant metastasis, which is largely responsible for the generally poor prognosis.
- D. The **diagnosis** is suggested by barium swallow and confirmed with esophagoscopy and biopsy or brush cytology.
- E. **Staging.** A system for staging esophageal cancer allows assignment of patients to groups with similar prognosis, helps determine if local or systemic therapy is needed, and allows comparison of response to different types of therapy ([Table 12-1](#)). Evaluation for lymph node and distant-organ metastatic disease is often accomplished with CT scan. Whole body imaging with positron emission tomographic scanning has shown great promise, particularly for detection of metastatic disease (*Ann Thorac Surg* 64:770, 1997). Endoscopic ultrasonography is more accurate in determining the depth of wall invasion and involvement of peritumoral lymph nodes. Upper and midesophageal lesions require bronchoscopy to evaluate the airway for involvement by tumor.

Table 12-1. TNM (tumor, node, metastasis) staging system for esophageal cancer

F. Treatment

1. **Surgical resection** remains a mainstay of curative treatment for vigorous patients with localized disease. It offers the best opportunity for cure and provides substantial palliation when cure is not possible. Resection and esophageal replacement also offer reliable relief of dysphagia. Overall 5-year survival is 20–30%, with higher rates for patients with lower stages of disease (*J Thorac Cardiovasc Surg* 105:265, 1993).
 - a. **Options for resection** include a standard transthoracic esophagectomy, a transhiatal esophagectomy, or an *en bloc* esophagectomy. Total esophagectomy with a cervical esophagogastric anastomosis and subtotal resection with a high intrathoracic anastomosis have become the most common resections and produce the best long-term functional results as well as the best chance for cure. Esophagogastrectomy with anastomosis to the distal half of the esophagus is seldom used because troublesome postoperative reflux symptoms are common.
 - b. **Options for esophageal replacement** include stomach, colon, and jejunum.
2. **Neoadjuvant therapy** with preoperative chemotherapy (cisplatin, vinblastine, and 5-fluorouracil) and 4,500-cGy radiotherapy has been shown to improve survival (*J Thorac Cardiovasc Surg* 105:265, 1993; *N Engl J Med* 335:462, 1996). This approach has not yet become the standard of care and is an area of ongoing investigation.
3. **Radiotherapy** is used worldwide for attempted cure and palliation of patients with squamous cell esophageal cancer that is deemed unsuitable for resection. Five-year survival is 5–10%. Palliation of dysphagia is successful temporarily in 80% of patients but rarely provides complete long-term relief. Combination therapy that involves radiation and concurrent administration of 5-fluorouracil with mitomycin C or cisplatin has been suggested to improve results and has replaced radiation alone in most current protocols.
4. The goal of **palliative treatment** is the relief of obstruction and dysphagia.
 - a. **Radiotherapy and chemotherapy** work best in patients with squamous cell carcinoma, particularly when it is located above the carina. Adenocarcinoma is less responsive to radiation, and the acute morbidity of external-beam irradiation to the epigastric area in terms of nausea and vomiting is substantial.
 - b. **Esophageal bypass procedures** have been tried, but the morbidity and mortality of such procedures are excessive.
 - c. **Intraluminal prostheses** have been developed to intubate the esophagus and stent the obstruction. Self-expanding wire-mesh stents, often with a soft silicone (Silastic) coating, have been used with greater ease of insertion and satisfactory results. None of these prostheses allow normal swallowing, and,

in most cases, little more than a pureed diet can be tolerated. The two methods for insertion are (1) peroral (push technique) and (2) via a laparotomy (pull technique). Of these, the peroral route is associated with fewer complications. Potential complications include perforation, erosion or migration of the tube, and obstruction of the tube by food or proximal tumor growth.

- d. **Endoscopic laser techniques** can restore an esophageal lumen successfully 90% of the time with only a 4–5% perforation rate.

V. Esophageal perforation

A. **Causes** of esophageal perforation may be either intraluminal or extraluminal and are associated with a 20% mortality.

1. Intraluminal causes

- Instrumental injuries** represent 75% of esophageal perforations and occur during endoscopy, dilation, sclerosis of esophageal varices, transesophageal echocardiography, or tube passage. The common sites are the normal anatomic sites of narrowing in the cervical and distal esophagus. Tumors that are located at the cardia pose a particular risk for perforation during dilation, as a result of fixation and angulation of the esophagus at the GE junction with this condition.
- Foreign bodies** can cause acute perforation or commonly take a more indolent course, with late abscess formation in the mediastinum or development of an empyema.
- Ingested caustic substances**, such as alkali chemicals, can produce coagulation necrosis of the esophagus and perforation.
- Cancer of the esophagus** may lead to perforation.
- Barotrauma** induced by external compression (e.g., Heimlich maneuver), forceful vomiting (Boerhaave's syndrome), seizures, childbirth, or lifting can produce esophageal perforation. Almost all of these perforations occur in the distal esophagus on the left side.

2. Extraluminal causes

- Penetrating injuries** can occur from stab wounds or, more commonly, gunshot wounds.
- Blunt trauma** may produce an esophageal perforation related to a rapid increase in intraluminal pressure or compression of the esophagus between the sternum and the spine.
- Operative injury** to the esophagus during an unrelated procedure occurs infrequently but has been reported in association with thyroid resection, anterior cervical spine operations, proximal gastric vagotomy, pneumonectomy, and laparoscopic fundoplication procedures.

B. **Signs and symptoms** of esophageal perforation typically begin with dysphagia, pain, and fever and progress to leukocytosis, tachycardia, respiratory distress, and shock if left untreated. Cervical perforations may present with neck stiffness and subcutaneous emphysema, and an intrathoracic perforation should be suspected in patients with chest pain, subcutaneous emphysema, dyspnea, and a pleural effusion (right pleural effusion in proximal perforations, left effusion in distal perforations). Intraabdominal perforations usually present with peritonitis.

C. The **diagnosis** of esophageal perforation is suggested by pneumomediastinum, pleural effusion, pneumothorax, atelectasis, and soft-tissue emphysema on chest X-ray or mediastinal air and fluid on CT scan and is followed with a water-soluble or dilute barium contrast esophagography. Contrast studies carry a 10% false-negative rate for esophageal perforations. Because esophagoscopy is used primarily as an adjunctive study and can miss sizable perforations, any discoloration or submucosal hematoma should be considered highly suspicious for perforation after trauma to the posterior mediastinum. Whenever an esophageal perforation is suspected, diagnosis and treatment must be prompt because morbidity and mortality increase in proportion to their delay.

D. **Principles of management** include (1) adequate drainage of the leak, (2) intravenous antibiotics, (3) adequate fluid resuscitation, (4) adequate nutrition, (5) relief of any distal obstruction, (6) diversion of enteric contents past the leak, and (7) restoration of GI integrity. Initially, patients are kept on nothing-by-mouth status, a nasogastric tube is placed carefully in the esophagus or stomach, and they receive intravenous hydration and broad-spectrum antibiotics.

E. **Definitive management** generally requires operative repair, although a carefully selected group of nontoxic patients with a locally contained perforation may be observed. Patients with an intramural perforation after endoscopic procedures or dilation have a characteristic radiographic finding of a thin collection of contrast material parallel to the esophageal lumen without spillage into the mediastinum. Management with a nasogastric tube and antibiotics almost always is successful in these patients.

- Cervical and upper thoracic perforations** usually are treated by cervical drainage alone or in combination with esophageal repair.
- Thoracic perforations** should be closed primarily and buttressed with healthy tissue, and the mediastinum should be drained widely. Even in operations on perforations that are more than 24 hours old, primary mucosal closure usually is possible. When primary closure is not possible, options include wide drainage alone or in combination with resection or exclusion and diversion in cases of severe traumatic injury to the esophagus.
- Abdominal esophageal perforations** typically result in peritonitis and require an upper abdominal midline incision to correct.
- Perforations associated with intrinsic esophageal disease** (e.g., carcinoma, hiatal hernia, or achalasia) require addressing the perforation as described previously and surgically correcting the associated esophageal disease concomitantly.

VI. **Esophageal diverticula** are acquired conditions of the esophagus found primarily in adults. They are divided into traction and pulsion diverticula based on the pathophysiology that induced their formation.

- Pharyngoesophageal (or Zenker's) diverticulum** is a pulsion diverticulum and is the most common type of symptomatic diverticulum. **Typical symptoms** are progressive cervical dysphagia, cough on assuming a recumbent position, and spontaneous regurgitation of undigested food that may produce episodes of choking and aspiration. **Diagnosis** with barium swallow should prompt **surgical correction** with cricopharyngeal myotomy and diverticulectomy or suspension. Notably, almost all patients with pharyngoesophageal diverticula have GER, which is thought to produce cricopharyngeal dysfunction and the resultant diverticulum.
- Traction or midesophageal or parabranchial diverticulum** occurs in conjunction with mediastinal granulomatous disease. Symptoms are rare, but, when they are present, operative excision of the diverticulum and adjacent inflammatory mass usually corrects the problem. On rare occasions, these diverticula present with chronic cough from an esophagobronchial fistula.
- Epiphrenic or pulsion diverticulum** can be located at almost every level but typically occurs in the distal 10 cm of the thoracic esophagus. It is also a pulsion diverticulum and develops as a herniation of the mucosa through the muscular layers of the esophagus on the right side as a result of functional or mechanical esophageal obstruction. Many patients are asymptomatic at the time of diagnosis, and, in those who are symptomatic, it is difficult to determine whether the symptoms stem from the diverticulum or from the underlying esophageal disorder.
 - The **diagnosis** is made with a contrast esophagogram; however, endoscopic examination and esophageal function studies are essential in defining the underlying pathophysiology. In advanced disease, the diagnosis can be confused with achalasia owing to the dependency of the diverticulum and the lateral displacement and narrowing of the GE junction.
 - Surgical treatment** is recommended for patients with progressive or incapacitating symptoms that are associated with abnormal esophageal peristalsis; it consists of diverticulectomy or diverticulopexy, with an extramucosal esophagomyotomy. The myotomy extends from the neck of the diverticulum down to the stomach. When the diverticulum is associated with a hiatal hernia and reflux, a concomitant nonobstructive antireflux procedure (Belsey Mark IV) is recommended. Any associated mechanical obstruction must be corrected.

VII. **Functional esophageal disorders.** This category includes a diverse group of disorders involving esophageal skeletal or smooth muscle.

- Motor disorders of esophageal skeletal muscle** result in defective swallowing and aspiration. Potential causes can be classified into five major subgroups: neurogenic, myogenic, structural, iatrogenic, and mechanical. Most causes of oropharyngeal dysphagia are not correctable surgically. However, when manometric studies demonstrate that pharyngeal contractions, although weak, are still reasonably well coordinated, cricopharyngeal myotomy can provide relief.
- Motor disorders of esophageal smooth muscle** can be subdivided into primary dysmotilities and disorders that involve the esophagus secondarily and produce dysmotility.

1. Primary dysmotility

- Achalasia** is rare (1/100,000 population) but is the most common primary esophageal motility disorder. It typically presents between the ages of 35 and 45 years. Chagas' disease, caused by *Trypanosoma cruzi* and seen primarily in South America, can mimic achalasia and produce similar esophageal pathology. Achalasia is a disease of unknown etiology, characterized by loss of effective esophageal body peristalsis and failure of the LES to relax with swallowing, with resultant esophageal dilatation. LES pressure is often (but not invariably) elevated. The characteristic pathology is a deficiency of, or changes in, the ganglia of Auerbach's plexus. Three clinical stages have been outlined and are characterized by symptomatology and diameter of the esophagus: stage I (esophagus <4 cm), stage II (esophagus 4–7 cm), and stage III (esophagus >7 cm).
 - Symptoms** include progressive dysphagia, noted by essentially all patients, regurgitation immediately after meals (>70%), odynophagia (30%), and aspiration with resultant bronchitis and pneumonia (10%). Some patients experience chest pain due to esophageal spasms.
 - The **diagnosis** is suggested by a chest X-ray, which often shows a fluid-filled, dilated esophagus and absence of a gastric air bubble. A **barium esophagogram** demonstrates tapering ("bird's beak") of the distal esophagus and a dilated proximal esophagus. The bird's beak deformity is not specific for achalasia and can be seen in any process that narrows the distal esophagus (e.g., benign strictures or carcinoma). **Esophageal manometry** is the definitive diagnostic test for achalasia. Characteristic manometric findings include the absence of peristalsis, mirror-image contractions, and limited or absent relaxation of the LES with swallowing. Endoscopy should be performed to rule out benign strictures or malignancy,

so-called pseudoachalasia.

3. **Medical treatment** is aimed at decreasing the LES tone and includes nitrates, calcium channel blockers, and endoscopic injection of botulinum toxin (blocks acetylcholine release from nerve terminals) in the area of the LES. Surgical treatment with a **modified Heller esophagomyotomy** has been shown to produce excellent results in 95% of patients, compared with only 65% excellent results using forceful **pneumatic bougienage** (*Gut* 30:299, 1989). Many esophageal surgeons favor extending the myotomy onto the stomach and a concomitant antireflux procedure with the esophagomyotomy to avoid the two major causes of operative failure: (1) an incomplete myotomy due to inadequate mobilization of the esophagogastric junction, and (2) late stricture due to GER disease caused by the incompetent LES, combined with the inability of the aperistaltic esophagus to evacuate refluxed material. **Video-assisted thoracoscopic approaches** have been tried, with early results showing a higher incidence of postoperative GER compared to the open procedure (*Ann Thorac Surg* 56:680, 1993). More recently, **laparoscopic esophagomyotomy** combined with a partial fundoplication has been reported and is rapidly being adopted as the primary surgical option of most centers (*Surg Endosc* 14:746, 2000).
 - b. **Vigorous achalasia** is a term used to describe a variant of achalasia in which patients present with the clinical and manometric features of classic achalasia and diffuse esophageal spasm. These patients have spastic pain and severe dysphagia, likely because of residual disordered peristalsis that is ineffective in overcoming the nonrelaxed LES. Treatment is the same as for classic achalasia, except that consideration should be given to performing a longer (to the aortic arch) esophagomyotomy. With relief of the obstruction caused by the nonrelaxing LES, the pain usually disappears.
 - c. **Diffuse esophageal spasm** is characterized by loss of the normal peristaltic coordination of the esophageal smooth muscle. This results in simultaneous contraction of segments of the esophageal body.
 1. The primary **symptom** is severe spastic pain, which can occur spontaneously and at night. Additionally, dysphagia, regurgitation, and weight loss are common.
 2. The **diagnosis** is confirmed with esophageal manometry, which usually demonstrates spontaneous activity, repetitive waves, and prolonged, high-amplitude contractions. Characteristic broad, multi-peaked contractions with or without propagation are seen, and normal peristaltic contractions also may be present. Intravenous injection with the parasympathomimetic bethanechol (Urecholine) can provoke pain and abnormal contractions.
 3. **Treatment** with calcium channel blockers and nitrates can reduce the amplitude of the esophageal contractions but usually is not beneficial. Surgical treatment consists of a long esophagomyotomy, extending from the stomach to the aortic arch, and often a concomitant antireflux procedure.
 - d. **Nutcracker esophagus** refers to a condition characterized manometrically by prolonged, high-amplitude peristaltic waves associated with chest pain that may mimic cardiac symptoms. Treatment with calcium channel blockers and long-acting nitrates has been helpful. Esophagomyotomy is of uncertain benefit.
2. **Secondary dysmotility** represents the esophageal response to inflammatory injury or systemic disorders, such as scleroderma, multiple sclerosis, or diabetic neuropathy. Inflammation can produce fibrosis that can lead to partial or complete loss of peristalsis and esophageal contractility. The most common cause of secondary dysfunction is the reflux of gastric contents into the esophagus.
- a. **Progressive systemic sclerosis**, or scleroderma, has esophageal manifestations in 60–80% of patients, and often the esophagus is the earliest site of GI involvement. It is characterized by atrophy of the smooth muscle of the distal esophagus, deposition of collagen in connective tissue, and subintimal arteriolar fibrosis. Normal contractions are present in the striated muscle of the proximal esophagus.
 - b. In a subset of patients with severe, long-standing GER disease, **complications**, including erosive esophagitis and stricture formation, occur as a result of the combination of a hypotensive incompetent LES and poor esophageal emptying secondary to low-amplitude, disordered peristaltic contractions. Intensive medical treatment of the reflux is essential before operation. Most surgeons prefer a Collis gastroplasty and Belsey antireflux procedure for these patients because of the presence of esophageal shortening and weak peristalsis.

VIII. Esophageal strictures are either benign or malignant, and the distinction is critical. **Benign strictures** are either congenital or acquired.

- A. **Congenital webs** are the only true congenital esophageal strictures. They represent a failure of appropriate canalization of the esophagus during development and can occur at any level. An imperforate web must be distinguished from a tracheoesophageal fistula, although a perforate web may not produce symptoms until feedings become solid.
- B. **Acquired strictures**
 1. **Esophageal rings or webs** occur at all levels in relation to the etiology of the webbing process. An example is **Schatzki's ring**, which occurs in the lower esophagus at the junction of the squamous and columnar epithelium. A hiatal hernia is always present, and the etiology is presumed to be GER. Esophagitis is rarely present. Treatment generally consists of medical management of reflux with periodic dilation for symptoms of dysphagia.
 2. **Strictures** of the esophagus can occur from any esophageal injury, including chronic reflux, previous perforation, infection, or inflammation.
- C. **Symptoms** from a stricture consist of progressive dysphagia to solid food and usually begin when the esophageal lumen narrows beyond 12 mm.
- D. **Evaluation and treatment** of a stricture begins with the categorical **exclusion of malignancy**. The diagnosis usually is made with barium swallow. Esophagoscopy is essential to assess the location, length, size, and distensibility of the stricture and to obtain appropriate biopsies or brushings. Because a peptic stricture secondary to reflux always occurs at the squamocolumnar junction, biopsy of the esophageal mucosa below a high stricture should demonstrate columnar mucosa. If squamous mucosa is found, the presumptive diagnosis of a malignant obstruction should be made, although strictures due to Crohn's disease, previous lye ingestion, or monilial esophagitis are among alternative diagnoses. Most strictures are amenable to **dilation**, and this relieves the symptoms. Attention is then directed at correcting the underlying etiology. **Resection** can be required for recurrent or persistent strictures or if malignancy cannot be ruled out.

IX. Benign esophageal neoplasms are rare, although probably many remain undetected. The most common lesion is a stromal tumor (leiomyoma), followed by polyps. Less common lesions are hemangioma and granular cell myoblastoma.

- A. **Clinical features** depend primarily on the location of the tumor within the esophagus. **Intraluminal** tumors, such as polyps, cause esophageal obstruction, and patients present with dysphagia, vomiting, and aspiration. **Intramural** tumors, such as leiomyomas, usually are asymptomatic but, if large enough, can produce dysphagia or chest pain.
- B. **Diagnosis and evaluation** usually involve a combination of barium swallow, esophagoscopy, and perhaps CT scanning or MR scan studies.
- C. **Treatment** for all symptomatic or enlarging tumors is surgical removal. Intraluminal tumors can be removed successfully via endoscopy, but if large and vascular, they should be resected via thoracotomy and esophagotomy. Intramural tumors, such as leiomyomas, can usually be enucleated from the esophageal muscular wall without entering the mucosa. This is performed via a video-assisted thoracoscopic or open thoracotomy approach. Laparoscopic resection may be appropriate for distal lesions.

X. Caustic ingestion. Liquid alkali solutions (e.g., **Drano** and **Liquid-Plumr**) are responsible for most of the serious caustic esophageal and gastric injuries and can produce necrosis in both organs. Acid ingestion is more likely to produce isolated gastric injury.

- A. **Initial management** is directed at hemodynamic stabilization and evaluation of the airway and extent of injury.
 1. **Airway compromise** can occur from burns of the epiglottis or larynx and may require tracheostomy.
 2. **Fluid resuscitation** and **broad-spectrum antibiotics** should be instituted.
 3. **Vomiting should not be induced**, but patients should be placed on **nothing-by-mouth** status and given an **oral suction** device.
 4. **Steroids** are of no proven benefit.
- B. **Evaluation** with water-soluble contrast esophagography and gentle esophagoscopy should be done early to assess the severity and extent of injury and rule out esophageal perforation or gastric necrosis.
- C. **Management**
 1. **Without perforation**, management is supportive because acute symptoms generally resolve over several days.
 2. **Perforation, unrelenting pain, or persistent acidosis** mandate surgical intervention. A transabdominal approach is recommended to allow evaluation of the patient's stomach and distal esophagus. If necrotic, the involved portion of the patient's stomach and esophagus must be resected, and a cervical esophagostomy must be performed. A feeding jejunostomy is placed for nutrition, and reconstruction is performed 90 or more days later.
 3. **Late problems** include the development of **strictures** and an increased risk of **esophageal carcinoma** (1,000 times that of the general population).

XI. Complications of esophageal surgery. Esophageal surgery is fraught with potential complications, and consistently good results require meticulous attention to operative technique.

- A. **Postthoracotomy complications** can include atelectasis and respiratory insufficiency, pneumonia, atrial fibrillation, wound infections, and persistent postoperative pain.
- B. **Complications related to an esophageal anastomosis** consist primarily of leak or stricture.
 1. **Anastomotic leaks** are managed according to the size of the leak, the location of the anastomosis, and the clinical status of the patient.

- a. **Cervical** anastomotic leaks usually can be managed by opening of the incision and drainage. Occasionally, the leak tracks below the thoracic inlet into the mediastinum, necessitating wider débridement and drainage. If a major leak occurs, esophagoscopy should be performed to rule out a significant ischemic injury to the stomach. If present, the anastomosis should be taken down, and a cervical esophagostomy should be performed. The necrotic portion of the stomach should be resected, and the remaining stomach should be returned to the abdomen, with placement of a gastrostomy and feeding jejunostomy. Reconstruction with residual stomach or the colon is done at a later date.
 - b. **Intrathoracic** anastomotic leaks are associated with a high mortality. Small, well-drained leaks can be treated conservatively, but large or poorly drained leaks require operative exploration.
2. **Strictures** usually are the result of a healed anastomotic leak, relative ischemia of the anastomosis, or recurrent cancer. Most can be dilated successfully.
- C. **Complications of antireflux repairs** generally result from **preoperative** failure to recognize a confounding abnormality, such as poor gastric emptying or weak esophageal peristalsis, or **operative** miscalculations that result in too tight a fundoplication or excessive tension on the repair. Most of these complications require operative revision.
1. **Postoperative dysphagia** can result from a fundoplication that is too long or tight, a misplaced or slipped fundoplication that is positioned around the stomach rather than the distal esophagus, or a complete fundoplication in the setting of poor esophageal contractile function. It also can result from operative distortion of the GE junction, excessive narrowing of the diaphragmatic hiatus, or disruption of the crural closure with herniation of an intact repair into the chest.
 2. **Persistent or recurrent reflux** after surgery suggests an inadequate or misplaced fundoplication, disruption of the fundoplication, or herniation of the repair into the chest (*Ann Surg* 229:669, 1999).
 3. **Breakdown of an antireflux repair** usually is recognized by a gradual recurrence of symptoms. Most commonly, disruption of a repair is due to inadequate mobilization of the cardia and excessive tension on the repair.
 4. **Gas bloating** or gastric dilation can occur if the fundoplication is too tight or if there is unrecognized gastric outlet obstruction or delayed gastric emptying.

Disorders of the Stomach

I. Treatment of peptic ulcer disease

II. Gastric adenocarcinoma

III. Primary gastric lymphoma (PGL)

IV. Benign gastric tumors

V. Postgastrectomy syndromes

Disorders of the Stomach

I. Treatment of peptic ulcer disease has changed dramatically over the past two decades with the development of antisecretory drugs (H₂ blockers and proton pump inhibitors) and recognition of the role of *Helicobacter pylori* in its pathogenesis [*Gastroenterology* 118:(2 Suppl 1), 2000]. Intractability and recurrence of disease in adequately treated patients are quite rare, with surgical management reserved primarily for complications of ulcers (bleeding, perforation, gastric outlet obstruction). Cigarette smoking is a clear risk factor for **ulcerogenesis**, whereas the role of alcohol is less clear; diet and caffeine do not appear to be important. The use of nonsteroidal antiinflammatory drugs must be determined when evaluating a patient with peptic ulcer disease.

- A. **Symptoms** of uncomplicated peptic ulcer disease include chronic, intermittent, gnawing, or burning epigastric pain. In the case of duodenal ulcer, ingestion of food may alleviate the discomfort, whereas it may be exacerbated in the case of gastric ulcer.
- B. **Differential diagnosis** is broad and includes acute cholecystitis, pancreatitis, reflux esophagitis, and gastric cancer.
- C. **Esophagogastroduodenoscopy (EGD)** is more sensitive and specific than contrast examination for peptic ulcer disease (*Ann Intern Med* 101:538, 1984) and permits biopsy, which is critical in the evaluation of gastric ulcers to rule out malignancy as well as antral biopsies for *H. pylori*. Right upper quadrant ultrasonography and serum amylase level are helpful in ruling out other causes of upper abdominal pain.
- D. ***H. pylori* infection** is associated with more than 90% of duodenal ulcers and 75% of gastric ulcers. The **cod liver oil (CLO) test** is a rapid urease test performed on antral tissue obtained during endoscopy that is fast and relatively inexpensive; **histology** is considered the gold standard. Noninvasive testing for infection includes **serum immunoglobulin** for *H. pylori* and the **labeled urea breath test**, which is the test of choice for confirming eradication. **Treatment** involves a 2-week course of triple therapy (omeprazole-amoxicillin-clarithromycin or omeprazole-metronidazole-clarithromycin). When *H. pylori* is eradicated, the 12-month recurrence rate of duodenal ulcer is less than 5%, compared to 15% with maintenance H₂-blocker therapy (*Arch Intern Med* 155:1958, 1995).
- E. **Fasting serum gastrin level** should be obtained in patients with recurrent ulcer after adequate treatment, multiple ulcers, ulcers in unusual locations (such as the second and third portions of the duodenum), and complications of ulcers that require surgical therapy to rule out Zollinger-Ellison syndrome.
- F. **Classification of peptic ulcers** is based on location and has important treatment ramifications.
 1. **Duodenal ulcers** occur most frequently in the first portion of the duodenum and are associated with **acid hypersecretion**. Gastric metaplasia is common in the surrounding mucosa. Because there is minimal risk of malignancy as compared to gastric ulcers, excision of duodenal ulcer is not usually an important part of surgical strategy.
 2. **Type I gastric ulcer** (55%) occurs near the junction of the parietal and antral mucosa on the lesser curvature where the inner oblique muscle is absent (usually near the incisura) and is associated with **normal acid secretion**. Surgical treatment of choice for intractable or complicated benign type I gastric ulcer is antrectomy with Billroth I reconstruction (recurrence rate 3%). Truncal vagotomy (TV) does not confer additional benefit. Alternatively, wedge excision of the ulcer with parietal cell vagotomy (PCV; in which antral and pyloric vagal innervation is preserved) can be performed, although this can be technically difficult and is associated with a higher recurrence rate.
 3. **Type II gastric ulcer** (25%) is located in the gastric body in combination with a duodenal ulcer and is associated with **acid hypersecretion**. Antrectomy (including the gastric ulcer) and truncal vagotomy with either Billroth I or II reconstruction is the procedure of choice for intractable type II gastric ulcer. Wedge excision of the gastric ulcer with PCV is also acceptable.
 4. **Type III gastric ulcer** (15%) is prepyloric and is also associated with **acid hypersecretion**. Treatment is similar to that of type II gastric ulcer, but wedge excision with PCV is associated with a higher recurrence rate.
 5. **Type IV gastric ulcer** (5%) is located on the lesser curve near the gastroesophageal junction and is associated with **normal acid secretion**. Excision of the ulcer may require a generous distal gastrectomy. Acid reduction procedure is not necessary.
- G. **Complications of peptic ulcer disease** usually require surgical evaluation.
 1. **Hemorrhage** is the leading cause of death due to peptic ulcer disease, with a 5–10% mortality. Evaluation and management begin with aggressive resuscitation and correction of any coagulopathy followed by EGD. Although spontaneous cessation of bleeding occurs in 70% of patients, endoscopy is especially warranted in individuals who present with hypotension, hematemesis, melena, age older than 60 years, multiple medical comorbidities, and inability to clear gastric blood with lavage, as these patients have a higher risk of recurrent bleeding. Individuals with endoscopic findings of a visible vessel or active bleeding are also at high risk, and endoscopic thermal coagulation with or without epinephrine injection is mandated in these cases (*JAMA* 262:1369, 1989). Repeated episodes of bleeding, continued hemodynamic instability, an ongoing transfusion requirement of more than 6 units of packed red blood cells over 24 hours, and more than one unsuccessful endoscopic intervention are **indications for surgery**.
 - a. **Bleeding duodenal ulcers** are usually located on the posterior duodenal wall within 2 cm of the pylorus and typically erode into the gastroduodenal artery. Bleeding is controlled by duodenotomy and oversewing of the bleeding vessel. Patients who have failed medical therapy, have presented with shock, or have had other medical comorbidities but are otherwise stable should have a concomitant acid-reducing procedure, most commonly truncal vagotomy with pyloroplasty if this can be performed expeditiously. Occasionally, PCV can be considered in young, otherwise healthy, hemodynamically stable patients. Alternatively, some surgeons advocate *H. pylori* eradication rather than vagotomy after control of bleeding to reduce the risk of recurrent bleeding without the morbidity of vagotomy (*Br J Surg* 77:1004, 1991).
 - b. **Bleeding gastric ulcers** are optimally treated by wedge resection to eliminate the bleeding site and exclude malignancy, with a concomitant acid-reduction procedure for type II and type III (high-acid) gastric ulcers in stable patients (truncal vagotomy with pyloroplasty). If wedge resection of the ulcer cannot be performed due to its juxtapyloric location, a gastrotomy or pyloroplasty incision is made, the bleeding site is oversewn, and multiple biopsies are taken. Partial gastrectomy or antrectomy is associated with high mortality and morbidity in the emergent setting and should be viewed with caution.
 2. **Perforated peptic ulcer** typically presents with sudden onset of severe abdominal pain but can present less dramatically, particularly in hospitalized, elderly, and immunocompromised patients. The resulting peritonitis is often generalized but can be localized when the perforation is walled off by adjacent viscera and structures. Examination reveals low-grade fever, tachycardia, abdominal wall rigidity, and leukocytosis. Abdominal X-ray reveals free subdiaphragmatic gas in 80–85% of cases. **Nonoperative treatment of perforated duodenal ulcer** can be considered in cases in which the perforation has been present for more than 24 hours, the pain is well localized, there is no evidence of ongoing extravasation on upper GI water-soluble contrast studies, and the patient is reliable and can be examined frequently (*N Engl J Med* 320:970, 1989). All patients with perforation should undergo aggressive fluid resuscitation and have broad-spectrum antibiotics instituted before **surgical management**.
 - a. **Perforated duodenal ulcers** are best managed by simple omental patching and peritoneal débridement followed by *H. pylori* eradication. An acid-reducing procedure (preferably PCV) should be added in stable patients if the patient is known to be *H. pylori* negative or has failed medical therapy.
 - b. **Perforated gastric ulcers** are best treated by wedge resection to eliminate the perforation and exclude malignancy, with a concomitant acid-reduction procedure in stable patients (truncal vagotomy with pyloroplasty or PCV) with type II and type III gastric ulcers. If wedge resection of the ulcer cannot be performed due to its juxtapyloric location, multiple biopsies of the ulcer are taken and omental patching is performed. Partial gastrectomy or antrectomy is associated with high mortality and morbidity in the emergent setting and is rarely indicated.
 3. **Gastric outlet obstruction** can occur as a chronic process due to fibrosis and scarring of the pylorus and duodenum from chronic ulcer disease or as a consequence of acute inflammation superimposed on previous scarring of the gastric outlet. **Patients present** with recurrent vomiting of poorly digested food, dehydration, and hypochloremic alkalosis. **Initial management** consists of aggressive correction of volume and electrolyte abnormalities, nasogastric suction, and intravenous antisecretory agents. Parenteral nutrition is occasionally required. EGD is necessary to evaluate the nature of the obstruction and to rule out malignant etiology. Acute obstruction typically resolves in 3–4 days. One-third of patients have significant improvement with initial management, but eventually 75% of patients require surgical intervention.
 4. **Indications for surgical therapy** include persistent obstruction after 7 days of nonoperative management and recurrent obstruction. The goals of operative management are to relieve obstruction and reduce acid secretion. PCV with gastrojejunostomy may be the treatment of choice, as peripyloric scarring, inflammation, and edema may make pyloroplasty difficult in the case of truncal vagotomy with pyloroplasty and make closure of the duodenal stump difficult

in truncal vagotomy with antrectomy with Billroth II reconstruction. **Endoscopic balloon dilatation** results in immediate symptomatic improvement in 80% of patients, but fewer than 40% have sustained improvement at 3 months (*Gastrointest Endosc* 43:98, 1996), and it is reserved for patients who are poor operative risks.

II. Gastric adenocarcinoma is the second most common cause of cancer mortality worldwide but only the seventh most frequent cause of cancer death in the United States (13,500 deaths per year). Its incidence in the United States has decreased dramatically over the past 60 years (33/100,000 in 1965 to 7/100,000 at present) for reasons that are incompletely understood, possibly related to changes in diet or food storage. Interestingly, proximal adenocarcinomas make up an increasing proportion of newly diagnosed cancers. The overall 5-year survival rate in the United States is 15%.

- A. **Risk factors** for gastric cancer include *H. pylori* infection, male gender, chronic atrophic gastritis, pernicious anemia, adenomatous gastric polyps, Ménétrier's disease, and previous gastric resection. Unlike colorectal carcinoma, the majority of gastric cancers do not arise from adenomatous polyps. Up to 12% of gastric ulcers may be cancerous.
- B. **Signs and symptoms** of gastric cancer include abdominal pain, unexplained weight loss, anorexia, early satiety, anemia, or upper GI bleeding, but none of these is sensitive or specific. Similarly, the physical examination is usually nonspecific, occasionally revealing evidence of advanced or metastatic disease, such as abdominal mass, ascites, jaundice, temporal wasting, enlarged supraclavicular node (Virchow's node), infiltration of the umbilicus (Sister Mary Joseph's node), a mass in the pelvic cul-de-sac (Blumer's shelf), or enlarged ovaries on pelvic examination (Krukenberg's tumor).
- C. **Diagnosis** is typically made by **EGD**, which permits visualization of the tumor, determination of its size and location, and biopsy and brushing of suspicious lesions. **Screening examination** by endoscopy or contrast studies is not cost effective for the general U.S. population given the low incidence and high cost but may be warranted in high-risk individuals, such as patients more than 20 years post-partial gastrectomy, those with pernicious anemia or atrophic gastritis, or immigrants from endemic areas (Russia, Asia).
- D. **Staging** is important in selecting the appropriate treatment ([Table 13-1](#)). Abnormal findings on physical examination and chest X-ray may indicate metastatic spread.

Table 13-1. TNM (tumor, node, metastasis) staging of gastric cancer

1. **CT scan of the abdomen and pelvis** may show ascites, metastatic spread to other organs, and lymphadenopathy. However, it is not uncommon for tumors to be either understaged due to failure to detect small peritoneal, omental, or hepatic metastases or incorrectly determined to be unresectable due to involvement of contiguous organs when evaluated by CT alone (*Gut* 41:314, 1997).
 2. **Endoscopic ultrasonography** is superior to CT in delineating the depth of tumor invasion in the gastric wall and adjacent structures and identifying perigastric lymphadenopathy. Lesions that are confined to the mucosa or submucosa are known as **early gastric cancer** and account for 5% of gastric cancer cases. Tumors that are limited to the mucosa rarely metastasize to regional lymph nodes (1–4%) and are thus potentially amenable to endoluminal resection or local excision as opposed to radical resection.
 3. **Laparoscopy** significantly enhances the accuracy of staging, leading to a change in the preoperative staging in up to 58% of patients, with upstaging more common than downstaging (*Surg Endosc* 11:1159, 1997), resulting in a change in management of up to 40% (*Endoscopy* 31:3427, 1999). Laparoscopy may prevent unnecessary laparotomy or allow more accurate selection of those patients with locally advanced disease for investigational neoadjuvant therapies. It can be performed as a separate procedure or as part of a planned curative resection.
- E. **Surgery** is indicated for potentially curable tumors and for palliation of patients who present with bleeding, obstruction, or perforation due to locally advanced, incurable cancers. The abdomen is thoroughly explored via upper-midline or Chevron incision to rule out metastatic disease and to determine the extent and mobility of the primary tumor.
1. **Curative resection** is undertaken if the cancer appears to be confined to the primary tumor and the regional lymph nodes. The primary tumor is resected in its entirety with a normal stomach tissue margin of 5–7 cm, with adequacy of margins confirmed by frozen section. *En bloc* resection of adjacent structures, including the transverse colon, pancreas, liver, or spleen, may be required to obtain negative margins. As such, most surgeons instruct their patients to undergo a mechanical and antibiotic bowel prep preoperatively.
 - a. **The optimal extent of lymphadenectomy** is controversial. D1 lymphadenectomy entails removal of perigastric nodes; a D2 lymphadenectomy includes removal of left gastric, hepatic, splenic, and celiac nodes. Even wider (D3 and D4) resections are practiced in Japan when involvement of those nodes is suspected, but they are rarely performed in the West. D2 dissection may improve accuracy of staging and offers radical local clearance; it was shown to have survival benefit in patients with stage II and IIIA disease [*Ann Surg* 228(4):449, 1998] but was found to be associated with increased morbidity and mortality with no survival benefit in another study (*N Engl J Med* 340:908, 1999).
 - b. **Reconstruction** is usually performed with an antecolic Roux-en-Y or Billroth II gastrojejunostomy, theoretically avoiding anastomosis in the retroperitoneal bed, where recurrence leading to anastomotic obstruction can occur. Retrocolic anastomosis, however, is associated with improved dependent gastric emptying and is preferred for resections for nonmalignant disease.
 2. **Palliative resection** may prevent bleeding, obstruction, and perforation in patients with metastatic or otherwise unresectable cancer. Alternatively, if gastric outlet obstruction is the primary symptom of an unresectable tumor, laparoscopic gastrojejunostomy can be performed at the time of staging laparoscopy, as it affords lower morbidity than open surgery. If laparotomy has been performed, however, open resection of the primary cancer results in a longer period of symptom palliation than does bypass alone (*Surgery* 88:476, 1980).
- F. **Adjuvant therapy**
1. **Adjuvant chemotherapy** has not shown any consistent benefit in disease-free interval or survival (*JNCI* 85:1839, 1993).
 2. **Neoadjuvant chemotherapy** may reduce tumor size, enhancing the chance for resectability. Other theoretical benefits include the elimination of delayed administration of therapy due to prolonged postoperative recovery, prevention of nontherapeutic gastrectomy when disease is rapidly progressive during treatment, and administration of therapy when measurable disease is present to assess response, allowing treatment to continue only in those patients who respond to the regimen. Response to neoadjuvant chemotherapy has been associated with markedly improved survival after curative resection in phase II trials [*Ann Surg* 229(3):303, 1999], warranting further investigation in the context of clinical trials.
 3. **Postoperative radiotherapy** with concomitant 5-fluorouracil may decrease locoregional recurrence in patients with advanced disease, but its use postoperatively and as a component of neoadjuvant treatment remains investigational [*Cancer* 86(9):1657, 1999].
 4. **Hyperthermic intraperitoneal chemoperfusion with mitomycin C** was associated with improved peritoneal recurrence rates and long-term survival in patients with locally advanced cancer in a prospective randomized trial (*Cancer* 85:529, 1999) and is currently being investigated.

III. Primary gastric lymphoma (PGL) accounts for fewer than 5% of gastric neoplasms. PGL, however, accounts for two-thirds of all primary GI lymphomas. It is usually B-cell, non-Hodgkin's lymphoma. *H. pylori* has been shown to be associated with PGL, particularly with mucosa-associated lymphoid tissue tumors, and its eradication has been demonstrated to result in clinical regression of these tumors [*Ann Oncol* 10(6):637, 1999]. Patients are typically in their 50s, and presentation is similar to that of gastric adenocarcinoma, with pain (68%), weight loss (28%), and bleeding (28%) most common. Most PGLs are located in the distal stomach.

- A. **Diagnosis of PGL** can be difficult. **Physical examination** is nonspecific and may reveal adenopathy, abdominal mass, or hepatosplenomegaly. **EGD** typically shows superficial stellate ulcers with extensive gastric wall infiltration. Endoscopic biopsy has a sensitivity of only 80% because obtaining adequate tissue can be difficult. The cod liver oil (CLO) test should be performed to assess *H. pylori* status. GI contrast studies cannot reliably differentiate lymphoma from adenocarcinoma. Occasionally, percutaneous CT-guided fine-needle biopsy can yield the diagnosis. Failure to establish a definitive pathologic diagnosis is an indication for surgery.
- B. **Staging** via physical examination, CBC, lactic dehydrogenase, chest and abdominopelvic CT scan, and bone marrow biopsy that reveals no evidence of extragastric tumor is adequate to exclude metastatic disease. As with adenocarcinoma, endoscopic ultrasonography and laparoscopy can be helpful in more accurately determining staging information. Modified Ann Arbor stage IIIIE and stage IV lymphoma must be ruled out, as their treatment is nonsurgical.
- C. **Optimal treatment of PGL** has not been determined, as there are no large prospective trials with adequate numbers of patients. Some centers advocate gastric resection as the treatment of choice for stage IE or IIE PGL [*Gastroenterology* 119(5):1191, 2000], whereas others suggest reserving resection for those in whom chemotherapy fails [*Cancer* 88(9):1979, 2000].
 1. **Surgery** provides a good chance for cure for stage IE and IIE PGL and is the most effective treatment for complications such as bleeding, perforation, obstruction, or fistula formation. Additionally, resection affords accurate tissue diagnosis, staging, and grading and may prevent the development of the aforementioned complications during or after additional treatment. Of note, microscopically positive margins do not have a negative impact on survival if adjuvant treatment is given.
 2. **Chemotherapy** consists of cyclophosphamide-doxorubicin-vincristine-prednisone or cyclophosphamide-nitrogen

mustard–vincristine–procarbazine–prednisone and is used primarily for stage III and IV disease and as adjuvant treatment in patients with a high probability of recurrence.

3. **Radiotherapy** as adjuvant treatment is controversial but may improve survival if positive margins or gross disease remain after surgery (*Ann Surg* 195:196, 1982).

IV. Benign gastric tumors account for fewer than 2% of all gastric tumors. They are usually located in the antrum or corpus. Presentation can be similar to that of peptic ulcer or adenocarcinoma, and diagnosis is made by EGD or contrast radiography.

- A. **Gastric polyps** are classified by histologic findings. Endoscopic removal is appropriate if the polyp can be completely excised.
 1. **Hyperplastic polyps** are regenerative rather than neoplastic and constitute 75% of gastric polyps. Risk of malignant transformation is minimal.
 2. **Adenomatous polyps** are the second most common gastric polyp and are neoplastic in origin. The incidence of carcinoma within the polyp is proportionate to its size, with polyps of greater than 2 cm having a 24% incidence of malignancy. Patients with familial adenomatous polyposis have a 50% incidence of gastroduodenal polyps and require endoscopic surveillance. Surgical resection with a 2- to 3-cm margin of gastric wall can often be performed laparoscopically and is required if endoscopic excision is not possible.
- B. **Gastrointestinal stromal tumors (GIST)** are most commonly found in the stomach and are usually benign (90%), although the true benign or malignant nature of the lesion is established by the lack of adjacent organ invasion or metastasis. Bleeding may arise from the erosion of the overlying mucosa, or these tumors may present secondary to mass effect. Endoscopy reveals an extraluminal mass with overlying normal or umbilicated mucosa. Treatment can be accomplished by wedge resection with a 2- to 3-cm histologically negative margin of gastric wall, although formal gastric resection may be required, depending on the location of the tumor.

V. Postgastrectomy syndromes are caused by changes in function as a consequence of gastric operations. Clearly defining the syndrome that is present in a given patient is critical to developing a rational treatment plan (*Surg Clin North Am* 72:445, 1992). Most are treated nonoperatively and resolve with time. They may occur in up to 20% of patients who undergo gastric surgery, depending on the extent of resection, disruption of the vagus nerves, status of the pylorus, type of reconstruction, and presence of mechanical or functional obstruction. Denervation of the proximal stomach impairs its receptive relaxation and accommodation functions, resulting in a rapid rise in intragastric pressure, with consequent accelerated emptying of liquids. Because emptying of solids is normally regulated by antral innervation and function, distal stomach denervation usually results in an increased rate of emptying of solids as well.

- A. **Dumping syndrome** is thought to result from the rapid emptying of a high osmolar carbohydrate load into the small intestine, caused by the loss of reservoir capacity (due to vagotomy or resection) and the loss of pylorus function (by resection or pyloroplasty), and occurs most commonly after Billroth II reconstruction.
 1. **Early dumping** occurs within 30 minutes of eating and is characterized by nausea, epigastric distress, explosive diarrhea, and vasomotor symptoms (dizziness, palpitations, flushing, diaphoresis). It is presumably caused by rapid fluid shifts in response to the hyperosmolar intestinal load and release of vasoactive peptides from the gut.
 2. **Late dumping** symptoms are primarily vasomotor and occur 1–4 hours after eating. The hormonal response to high simple carbohydrate load results in hyperinsulinism and reactive hypoglycemia. Symptoms are relieved by carbohydrate ingestion.
 3. **Treatment** is primarily nonsurgical and results in improvement in all but 1% of patients over time. Meals should be smaller in volume but increased in frequency, liquids should be ingested 30 minutes after eating solids, and simple carbohydrates should be avoided. Use of the long-acting somatostatin analogue octreotide (25–100 µg s.c. before meals) results in significant improvement and persistent relief in 80% of patients when behavioral modifications fail [*Clin Endocrinol* 51(5):619, 1999]. If reoperation is necessary, conversion to Roux-en-Y gastrojejunostomy is usually successful. Other surgical options include conversion to Billroth I (gastroduodenostomy) and jejunal segment interposition.
- B. **Alkaline reflux gastritis** is most commonly associated with Billroth II gastrojejunostomy and requires operative treatment more often than do other postgastrectomy syndromes. It is characterized by burning epigastric pain, nausea, and bilious emesis. The latter does not relieve the pain and is not associated with meals. Bile, not pancreatic enzymes or alkalinity, is the culprit that damages gastric mucosa (*Ann Surg* 180:648, 1995).
 1. **Endoscopy** reveals inflamed, beefy-red, friable gastric mucosa and can rule out recurrent ulcer as a cause of symptoms. Bile reflux into the stomach is occasionally seen. Enterogastric reflux can be confirmed by hepatic iminodiacetic acid scan, in which radiolabeled nucleotide is administered intravenously and excreted into the bile. Mechanical obstruction is absent, distinguishing alkaline reflux gastritis from loop syndrome.
 2. **Nonoperative therapy** consists of feeding patients ursodeoxycholic acid and is only occasionally effective.
 3. **Surgery** is the only proven treatment and consists of diversion of bile from the gastric mucosa. The creation of a long-limb (45-cm) Roux-en-Y effectively eliminates alkaline reflux [*Gastroenterol Clin North Am* 23(2):281, 1994]. However, symptoms are not invariably alleviated, and complications of operation include delayed gastric emptying, marginal ulceration, and the Roux stasis syndrome. Other authors advocate conversion to Billroth I gastroduodenostomy with biliary diversion using choledochojejunostomy via a 30- to 35-cm Roux-en-Y limb (*Arch Surg* 132:245, 1997).
 4. **Roux stasis syndrome** may occur in up to 30% of patients after Roux-en-Y gastroenterostomy (*Am J Surg* 155:490, 1988) and is characterized by chronic abdominal pain, nausea, and vomiting that is worsened by eating. It results from a functional obstruction due to disruption of the normal propagation of pacesetter potentials in the Roux limb from the proximal duodenum and altered motility in the gastric remnant. Near-total gastrectomy to remove the atonic stomach can improve gastric emptying and is occasionally useful in patients with refractory Roux stasis. Use of an “uncut” Roux-en-Y reconstruction (*Surg Gynecol Obstet* 166:69, 1988) may preserve normal pacemaker propagation and prevent the development of the syndrome.
- C. **Loop syndromes** result from mechanical obstruction of either the **afferent** or **efferent** limb of the Billroth II gastrojejunostomy. The location and etiology of the obstruction are investigated by plain abdominal X-rays, CT scan, upper GI contrast studies, and endoscopy. Relief of the obstruction may require adhesiolysis, revision of the anastomosis, or, occasionally, bowel resection.
 1. **Afferent loop syndrome** can be caused acutely by bowel kink, volvulus, or internal herniation, resulting in severe abdominal pain and nonbilious emesis within the first few weeks after surgery. Lack of bilious staining of nasogastric drainage in the immediate postoperative period is concerning for this complication. Examination may reveal a fluid-filled abdominal mass, and laboratory findings may include elevated bilirubin or amylase. **Duodenal stump blowout** may result from progressive afferent limb dilatation, producing peritonitis, abscess, or fistula formation. In the urgent setting, jejunostomy can effectively decompress the afferent limb. A more **chronic form** of afferent loop syndrome results from partial mechanical obstruction of the afferent limb and causes postprandial right upper quadrant pain relieved by bilious emesis that is not mixed with recently ingested food. Stasis can lead to bacterial overgrowth and subsequent bile salt deconjugation in the obstructed loop, causing **blind loop syndrome** (steatorrhea, vitamin B₁₂, folate, and iron deficiency) by interfering with fat and vitamin B₁₂ absorption.
 2. **Efferent loop syndrome** results from intermittent obstruction of the efferent limb of the gastrojejunostomy, presenting as abdominal pain and bilious emesis months to years after surgery, behaving like a proximal small-bowel obstruction.
- D. **Postvagotomy diarrhea** has an incidence of 20% after truncal vagotomy and is thought to result from alterations in gastric emptying and vagal denervation of the small bowel and biliary tree. The diarrhea is typically watery and episodic. Treatment includes antidiarrheal medications (loperamide, diphenoxylate with atropine, cholestyramine) and decreasing excessive intake of fluids or foods that contain lactose. Symptoms usually improve with time, and surgery (placement of a 10- to 15-cm antiperistaltic jejunal segment 100 cm distal to the gastrojejunostomy) is rarely indicated.
- E. **Nutritional disturbances** can occur in up to 30% of patients after gastric surgery, either as a result of functional changes or postgastrectomy syndromes. Prolonged **iron, folate, vitamin B₁₂, calcium, and vitamin D deficiencies** can result in anemia, neuropathy, dementia, and osteomalacia. These can be prevented with supplementation.

14 Small Intestine

Matthew G. Mutch and David C. Linehan

Disorders of the Small Intestine

I. Small-bowel obstruction

II. Infectious diseases of the small bowel

III. Meckel's diverticulum

IV. Neoplasms

V. Disturbances in small-bowel physiology

VI. Crohn's disease

Disorders of the Small Intestine

I. **Small-bowel obstruction** is one of the most common clinical problems that is faced by general surgeons.

A. Etiology

1. **Adhesions** are the most common cause of small-bowel obstruction, accounting for 50–70% of all cases. Adhesions can be acquired from previous intraabdominal surgery or an intraabdominal inflammatory process. An adhesive obstruction develops in approximately 5% of patients with previous abdominal surgery during their lifetime. Congenital bands can also cause intestinal obstruction in the pediatric population.
2. **Incarcerated external hernias** (e.g. inguinal, femoral, umbilical, incisional, or parastomal) are the second most common cause of small-bowel obstructions, and they are the most common cause in patients with no history of abdominal surgery. Internal hernias (e.g., paraduodenal, mesenteric defects, and foramen of Winslow hernias) may also cause obstruction.
3. **Neoplasms.** Primary small-bowel tumors can cause intraluminal obstruction, whereas metastatic or intraabdominal tumors can cause obstruction by external compression.
4. **Intussusception** of the small bowel can result in obstruction and ischemia of the intussuscepting segment of bowel. Tumors, polyps, or enlarged mesenteric lymph nodes may serve as lead points for the intussusception.
5. **Crohn's disease** can cause obstruction secondary to acute inflammation during a flare or from a chronic stricture.
6. **Volvulus** is often caused by adhesions or congenital anomalies, such as intestinal malrotation. It is a more common cause of large-bowel obstruction.
7. **Gallstone ileus.** Intense inflammation of the gallbladder results in fistulization of the biliary tree and the duodenum or small bowel that allows gallstones to migrate into the gastrointestinal (GI) tract. Larger stones can then become lodged in the small bowel, most commonly in the terminal ileum or ileocecal valve resulting in obstruction.
8. **Strictures** secondary to ischemia, inflammation, radiation therapy, or surgical trauma.
9. **External compression** from tumors, abscess, hematomas, or other fluid collections.
10. **Foreign bodies**, such as bezoars.
11. **Meckel's diverticulum** can produce volvulus, intussusception, or Littre's hernia.
12. **Cystic fibrosis** can cause chronic partial obstruction of the distal ileum and right colon as a result of inspissated, meconiumlike gastrointestinal contents.

B. Classifications

1. **Mechanical obstruction** is the blockage of the intestinal lumen by an intrinsic or extrinsic lesion. The resulting obstruction can be partial, allowing some distal passage of gas or fluid, or complete, with total occlusion of the lumen. **Simple obstruction** implies that there is no vascular compromise of the involved bowel and that the occlusion has occurred at only one site. A **strangulated obstruction** means that the involved bowel has vascular compromise, which can ultimately lead to gangrene of the intestinal wall. A closed-loop obstruction is when a segment of bowel is occluded proximally as well as distally. Differentiating between partial and complete, simple or strangulated, can have a significant impact on therapy.
2. **Paralytic ileus** implies failure of peristalsis secondary to a neurogenic disturbance. It can be caused by recent abdominal surgery, electrolyte disturbances, peritonitis, trauma, systemic infections, bowel ischemia, and medications.

C. Diagnosis

1. **Signs and symptoms** of a small-bowel obstruction depend on the location, etiology, duration, and classification of the obstruction as well as the physiologic condition of the patient. Typically, patients present with abdominal pain, vomiting, obstipation, abdominal distention, and failure to pass flatus. Proximal small-bowel obstructions tend to be associated with bilious vomiting early in the course, little abdominal distention, and variable amounts of pain. For more distal obstructions, vomiting tends to be less, occurring later in the course, and can be thick and feculent. Abdominal distention typically increases the more distal the obstruction, and the pain, characterized as crampy, colicky, and intermittent, is poorly localized. Obstipation occurs after the segment of bowel distal to the obstruction is evacuated of gas and feces; thus, obstipation usually occurs later the more proximal the obstruction. Bowel sounds are variable in that they may be either hypoactive or hyperactive. Temperature, heart rate, blood pressure, and urine output may all be normal until the patient becomes progressively dehydrated. The longer a patient has a bowel obstruction, the greater are the chances of developing a complication. As time goes on, hypovolemia progresses secondary to impaired intestinal absorption, increased intestinal secretion, and vomiting, which can ultimately lead to shock and multiorgan dysfunction syndrome. As dehydration progresses, the chance of a simple obstruction converting to a strangulated obstruction increases. The preoperative diagnosis of a strangulated obstruction cannot be made or excluded reliably by any known clinical or laboratory parameter (*Am J Surg* 145:176, 1983). However, findings of complete mechanical small-bowel obstruction associated with well-localized constant pain or peritoneal signs, fever, tachycardia, hypotension, and leukocytosis should lead to a high index of suspicion of strangulated bowel. This should then lead to urgent operative intervention. Hematemesis, gross or occult, or bloody bowel movements should also raise the level of suspicion for strangulated bowel.
2. **Physical examination** should begin with inspection of the abdomen. A varying amount of distention will be present, depending on the site of obstruction. A thorough search for surgical scars and hernias should be performed. Visible and audible peristaltic waves may be present. Palpation should determine the location and severity of tenderness and evaluate for the presence of peritoneal signs. Localized pain, guarding, or peritonitis is suggestive of ischemic bowel. Auscultation may or may not reveal bowel sounds. Digital rectal examination may reveal the presence of a rectal mass or impacted stool. Signs of dehydration, nutritional status, and overall physiologic health should be sought.
3. **Laboratory evaluation.** Laboratory values are usually normal in the early stages of a small-bowel obstruction and therefore are of little use in making the diagnosis. As the obstruction progresses, patients tend to become dehydrated. Laboratory tests may reveal decreased levels of sodium, chloride, and potassium, accompanied by increased levels of bicarbonate, blood urea nitrogen (BUN), creatinine, and rising hematocrit. Serum amylase, released from small-bowel mucosa, can be elevated. A mild leukocytosis (WBC >10,000 cells/ μ L) is not unusual. A WBC count of greater than 15,000 cells/ μ L or metabolic acidosis should raise the suspicion of strangulation.
4. **Radiologic evaluation**
 - a. **Supine and upright abdominal plain films** that reveal dilated, distended loops of small bowel with air-fluid levels and paucity of gas in the colon and rectum are characteristic findings of a small-bowel obstruction. These findings may be absent in early obstructions, proximal obstructions, or closed-loop obstructions or when fluid-filled bowel contains little gas. In a complete small-bowel obstruction, there tends to be a paucity of colonic gas. On the other hand, a partial small-bowel obstruction has dilated loops of small bowel with variable amounts of colonic gas. Thumb-printing, loss of mucosal pattern, and gas within the bowel wall or intrahepatic branches of the portal vein are suggestive of a strangulated obstruction. The presence of free intraabdominal air on an upright chest X-ray or right lateral decubitus plain film of the abdomen indicates perforation of a hollow viscus. The findings of air in the biliary tree and a radiopaque gallstone in the right lower quadrant are suggestive of gallstone ileus. Paralytic ileus appears as gaseous distention that is uniformly distributed throughout the stomach, small intestine, and colon.
 - b. **Contrast studies** (small-bowel follow-through or water-soluble contrast enema) can be helpful in localizing the specific site of obstruction and provide information regarding the etiology of the obstruction. For patients who have small-bowel obstruction with no history of abdominal surgery or no identifiable hernia or who are failing to improve with nonoperative therapy, a small-bowel follow-through can be particularly helpful. For very distal small-bowel obstructions or colonic obstructions, a water-soluble contrast enema can enhance the diagnosis and management of these obstructions. The use of barium should be avoided in the setting of acute bowel obstruction because bowel perforation with its leakage into the peritoneal cavity causes an intense inflammatory reaction, which can significantly increase morbidity and mortality. However, computed tomography (CT) has better sensitivity and specificity for the diagnosis of a small-bowel obstruction.
 - c. **Abdominal/pelvic CT** is an excellent imaging modality for diagnosing small-bowel obstruction. It has the ability to localize and characterize the obstruction as either partial or complete. It can give information regarding the cause of obstruction (e.g., malignancy, inflammatory process, or extrinsic causes) and the presence of other intraabdominal pathology. Evidence suggests that CT scanning can improve the preoperative diagnosis of bowel with

vascular compromise (*AJR Am J Roentgenol* 154:99, 1990; *J Comput Assist Tomogr* 17:386, 1993). CT findings of thickened bowel wall, mesenteric edema or vascular engorgement, and pneumatosis are all suggestive of strangulated bowel.

D. Differential diagnosis

1. **Paralytic ileus** typically has many of the same signs and symptoms as a small-bowel obstruction. A thorough history and physical examination (e.g., narcotic use, psychiatric medications, hypokalemia, gas throughout the entire GI tract on plain films) can help differentiate ileus from obstruction.
2. **Colonic obstruction** can easily be confused with a distal small-bowel obstruction, especially if the ileocecal valve is incompetent. This is a situation in which a contrast enema is helpful.
3. **Mesenteric vascular occlusion** can produce colicky abdominal pain, especially after meals. Acute occlusion often presents with marked leukocytosis and severe abdominal pain out of proportion to physical findings. Angiography confirms the diagnosis.
4. **Intestinal pseudo-obstruction** occurs in association with a variety of disorders and presents with signs and symptoms of intestinal obstruction without evidence of an obstructing lesion. Treatment is directed at the underlying disease.

E. Treatment

1. **Partial small-bowel obstruction**, when discriminated from complete obstruction, can be treated expectantly as long as the patient continues to pass flatus and is clinically stable. The cornerstone of treating any type of bowel obstruction is adequate fluid resuscitation to achieve a urine output of at least 0.5 mL/kg per hour. The patient is placed on nothing-by-mouth status, all electrolyte abnormalities are corrected, and a nasogastric or long intestinal tube is placed for GI decompression. Studies have shown no significant difference in outcome between patients who are managed with nasogastric tubes and those managed with long intestinal tubes (*Ann Surg* 206:126, 1987; *Am J Surg* 170:366, 1995). During this trial of nonoperative management, it is imperative that the patient undergo serial abdominal examinations every 4–6 hours and that daily abdominal plain films are obtained. If signs and symptoms of strangulation or conversion to complete obstruction develop or the patient fails to improve with 48–72 hours of tube decompression, a laparotomy is indicated. In patients with a bowel obstruction secondary to an incarcerated hernia, attempts to reduce the hernia can be made with mild sedation and gentle force. After successful reduction, the patient should be monitored carefully for evidence of bowel infarction or perforation. Other situations that may warrant a trial of nonoperative therapy include early postoperative obstruction, multiple prior episodes of obstruction, multiple previous abdominal operations with extensive adhesions, abdominal irradiation, Crohn's disease, and abdominal carcinomatosis. If tube decompression does not produce marked clinical and radiologic improvement within 24–48 hours, surgery must be strongly considered.
2. **Complete bowel obstructions** are often associated with strangulation and are therefore managed with prompt surgical intervention. Mortality that is associated with gangrenous bowel can approach 30% if surgery is delayed beyond 24–36 hours. Once again, fluid and electrolyte resuscitation with tube decompression is the first step in managing small-bowel obstructions.
 - a. **Fluid replacement** should begin with an isotonic saline solution (0.9% NaCl). Potassium should be added only after adequate urine output (0.5 mL/kg per hour) is achieved. Bicarbonate should be given if the arterial pH is less than 7.1. Serum electrolyte values, hourly urine output, and central venous pressure should be monitored to assess adequacy of resuscitation. Monitoring of arterial blood pressure and measurement of serial arterial blood gases should be used when indicated. Intravenous antibiotics should be given if strangulation is suspected.
 - b. **Operation.** After fluid resuscitation has been initiated and antibiotics have been administered, an exploratory laparotomy is performed. In the majority of cases, this is done through a midline incision. However, in the case of an incarcerated inguinal or femoral hernia, a standard groin incision can be used. During the exploration, adhesions are lysed, any gangrenous bowel is resected, and the source of obstruction is identified. The viability of adjacent or compromised bowel must be assessed; various methods include inspection of bowel color, palpation or Doppler ultrasound of arterial pulsations, and visualization of peristalsis in the bowel after several minutes of being wrapped in warm saline-soaked packs. Intravenous fluorescein injection followed by inspection of the bowel under ultraviolet (Wood's) light can also be used. A second-look operation should be planned if any doubt exists about the viability of the remaining bowel at the end of the initial operation or at any time in the postoperative period. In cases in which the obstructing lesion cannot be resected, a bypass procedure should be performed. Placement of gastrostomy tube for postoperative decompression should be considered in cases that are expected to have a prolonged ileus.
3. **Prognosis.** The postoperative mortality from nonstrangulating obstruction is 2%. Obstructions that are associated with strangulated bowel carry a mortality of 8% if surgery is performed within 36 hours of the onset of symptoms. Mortality can approach 30% if operation is delayed beyond 36 hours.

II. Infectious diseases of the small bowel

- A. **Acute gastroenteritis** can range from annoying GI dysfunction to a fulminant, life-threatening process. Diarrhea and vomiting that are associated with waves of crampy abdominal pain are the typical presenting symptoms. However, nausea, fever, malaise, myalgia, and headache can also occur. Diarrhea is defined as abnormally frequent evacuation of watery stools. Small-bowel diarrhea typically does not contain blood, pus, or mucus, which can be helpful in differentiating it from that of colonic origin.
 1. **Traveler's diarrhea.** More than 16 million people travel annually to developing countries, and approximately one-third develop diarrhea. Virtually all of these cases are of infectious etiology and are acquired through the ingestion of fecally contaminated food or water. Up to 70% of cases are caused by enterotoxigenic *Escherichia coli*. Other causes include nontyphus *Salmonella*, *Shigella*, *Giardia*, rotavirus, and parasites. Patients typically have four to five loose stools a day that are associated with crampy abdominal pain; 2–10% may have fever and bloody diarrhea. The illness is self-limiting and usually lasts for 3–5 days. Treatment is aimed at preventing dehydration and alleviation of symptoms, with agents such as bismuth–subsalicylate diphenoxylate and loperamide. If the illness is more severe (fever or bloody stools), such antibiotics as trimethoprim-sulfamethoxazole (160 mg trimethoprim, 800 mg sulfamethoxazole), norfloxacin (400 mg), or ciprofloxacin (500 mg) should be given twice a day.
 2. **Food poisoning** is most often caused by *Salmonella*, *Staphylococcus aureus*, *Clostridium perfringens*, or *Yersinia enterocolitica*. In 20–40% of cases, no identifiable cause is found. Waterborne disease outbreaks usually are caused by *Giardia lamblia*, *Helicobacter*, Norwalk virus, or rotavirus. Diarrhea and vomiting begin after 8–48 hours and usually resolve within 24–72 hours. Fever is uncommon. Pharyngitis occurs in 50% of children and 10% of adults with *Yersinia* infection. Antibiotics are not used except in fulminant cases.
- B. **Chronic infection.** Diarrhea that persists for longer than 2–3 weeks can be due to infection with *Y. enterocolitica*, *Clostridium difficile*, *Cryptosporidium*, or other parasites. Tuberculosis enteritis usually affects the colon and ileocecal area and can cause obstruction, hemorrhage, or malabsorption.
- C. **Viral gastroenteritis.** Rotavirus predominantly affects children at 6–24 months. Norwalk virus affects mostly older children and adults. Treatment consists of fluid and electrolyte replacement.

III. Meckel's diverticulum

- A. **Location.** Meckel's diverticulum is the most common congenital anomaly of the gastrointestinal tract. It occurs when the intestinal portion of the vitelline or omphalomesenteric duct fails to obliterate during the fifth or sixth week of fetal development. Following the “rule of twos,” incidence is 2%, male-female incidence is 2:1, it is located 2 ft from the ileocecal valve, the base is typically 2 in. in length, and it often contains two types of mucosa. It is a true diverticulum that contains all layers of the bowel wall and is located on the antimesenteric border of the bowel. As mentioned previously, it can contain heterotopic mucosa, such as gastric (62%), pancreatic (6%), combined gastric and pancreatic (5%), jejunal (2%), and Brunner's gland (2%), although up to 50% contain normal ileal mucosa.
- B. **Diagnosis.** The vast majority of Meckel's diverticulum is asymptomatic and is diagnosed incidentally at surgery or during an autopsy. Two-thirds of patients who manifest symptoms present before the age of 2 years. Signs and symptoms occur only after a complication of Meckel's has occurred.
 1. **Clinical presentation**
 - a. **Bleeding** in the form of melena or bright red blood per rectum is the most common presenting symptom (30–50%). It tends to be painless and episodic. The source is typically a peptic ulcer on the adjacent normal ileal mucosa caused by the acid-secreting gastric mucosa that lines the diverticulum.
 - b. **Intestinal obstruction** due to volvulus of the small bowel around a fibrous band connecting the diverticulum to the anterior abdominal wall, intussusception, or incarcerated internal hernia (Littre's) is the second most common presentation (30–35%).
 - c. **Acute Meckel's diverticulitis** occurs in 20% of symptomatic patients and is often mistaken for acute appendicitis. It is caused by intraluminal obstruction of the diverticulum, which leads to inflammation, edema, ischemia, necrosis, and perforation.
 2. **Radiologic evaluation.** The preoperative diagnosis of Meckel's diverticulum is very difficult. Therefore, it requires a high index of suspicion and the clinical signs and symptoms to help focus the radiologic evaluation.
 - a. A **technetium-99m sodium pertechnetate (“gastric” or “Meckel's”) scan** is able to identify the ectopic gastric mucosa that is present within the diverticulum. The sensitivity of the scan can be increased by the administration of an H₂-receptor antagonist. The Meckel's scan can be useful when a patient with a symptomatic but undiagnosed Meckel's diverticulum presents with GI bleeding of unknown etiology.
 - b. **Contrast studies**, such as small-bowel follow-through or enteroclysis, occasionally may detect the presence of the diverticulum. They may also be helpful in detecting a small-bowel intussusception.
 - c. **Bleeding scans** and **angiography** can be helpful in finding the site of hemorrhage during episodes of active bleeding. Recognition of the embryonic blood supply of the Meckel's diverticulum can aid in identifying it during angiography.
- C. **Differential diagnosis.** The differential diagnosis depends on the presenting complication of the Meckel's diverticulum. Because of the variable location of the ileum, one must consider the entire litany of intraabdominal pathology when these patients present with an inflammatory or obstructive process. The following is

a brief list of inflammatory processes that have similar presentations as Meckel's diverticulitis.

1. **Acute appendicitis** presents with signs and symptoms that are indistinguishable from those of acute Meckel's diverticulitis, and the two often can be distinguished at operation.
2. **Diverticulitis.** Sigmoid or even right colon diverticulitis has the same presentation as acute appendicitis or Meckel's diverticulitis.
3. **Crohn's disease.** Rectal bleeding may occur, especially in patients with colonic involvement, but it is not the main presenting symptom, thus distinguishing it from Meckel's diverticulum.

D. Treatment

1. **Surgical resection** is indicated in the symptomatic patient. Patients with bleeding that results from the presence of ectopic gastric mucosa should undergo a segmental small-bowel resection. This procedure removes the diverticulum as well as the bleeding ileal ulcer. For patients who present with obstruction, either from a fibrous band connecting the diverticulum to the anterior abdominal wall or from intussusception, simple diverticulectomy can be performed. If the base of the diverticulum is wide or there is concern for the viability of the surrounding bowel, a segmental resection should be performed. In the presence of acute diverticulitis, the affected segment of bowel should be resected.
2. **Incidental diverticulectomy** remains controversial. Morbidity and mortality associated with the presence of a Meckel's diverticulum are extremely low. Current recommendations are to leave the diverticulum alone if there is no evidence of ectopic tissue (e.g., localized thickening of the diverticulum) and if the orifice is wide. Resection of the diverticulum should be performed if there is evidence of ectopic mucosa, if the orifice is narrow, or if it is suspected that the patient's symptoms are due to the diverticulum. If patients are undergoing surgery for presumed appendicitis and the appendix is found to be normal, the terminal ileum must be inspected for a Meckel's diverticulum. If present, the diverticulum should be resected.

IV. Neoplasms of the small intestine occur infrequently and account for fewer than 25% of all GI neoplasms, benign and malignant. Tumors of the small intestine present insidiously with vague, nonspecific symptoms. Most benign tumors remain asymptomatic and are diagnosed incidentally during a laparotomy or at the time of an autopsy. On the other hand, the majority of malignant tumors eventually become symptomatic. However, symptoms tend to occur at late stages of the disease. The clinical presentation of symptomatic small-bowel neoplasms includes abdominal pain, obstruction, and hemorrhage. Patients with malignant tumors tend to have some degree of weight loss, which can be associated with an abdominal mass or even perforation of the tumor.

A. Benign tumors are ten times more common than malignant tumors.

1. **Adenomas.** The three types of small-bowel adenomas are simple tubular adenomas, villous adenomas, and Brunner's gland adenomas. They can occur sporadically as solitary lesions or in association with familial polyposis syndrome or Gardner's syndrome. Symptomatic lesions can cause fluctuating pain secondary to intermittent obstruction, intussusception, or bleeding. The duodenum is the most common site for all three types of adenomas to occur. Tubular adenomas have a low malignant potential and can be treated with complete endoscopic polypectomy. Villous adenomas have significant malignant potential. If the polyp can be completely excised endoscopically, this is adequate treatment. If complete resection is not possible, transduodenal excision with adequate margins is needed. Adenomas of the jejunum or ileum should be resected with a segmental bowel resection. Brunner's tumors represent hyperplasia of exocrine glands of the first part of the duodenum and have no malignant potential. Therefore, endoscopic resection is adequate for symptomatic lesions.
2. **Leiomyomas** arise from the mesenchymal cells of the small bowel and are the most common benign neoplasm of the small intestine. They grow submucosally and project into the lumen of the small bowel. On a small-bowel contrast study, they appear as a smooth, eccentric filling defect with intact, normal-appearing mucosa. Histopathologic examination is needed to distinguish benign from malignant stromal tumors. Therefore, once a lesion is detected, treatment consists of a segmental small-bowel resection.
3. **Hamartomas** arise in patients with Peutz-Jeghers syndrome. This is an autosomal dominant inherited syndrome of mucocutaneous melanotic pigmentation and multiple gastrointestinal polyps. Surgery is indicated only for obstruction, intussusception, or bleeding. At surgery, all polyps larger than 1 cm should be resected.
4. **Lipomas, neurofibromas, and fibromas** can cause symptoms that require operation. **Endometriosis**, appearing as puckered, bluish-red, serosally based nodules, can become implanted on the small bowel.

B. Malignant tumors

1. **Adenocarcinoma** is the most common malignant small-bowel tumor. Most tumors occur in the duodenum (periampullary region), and their frequency decreases as one progresses distally along the small bowel. Risk factors for the development of adenocarcinoma include villous adenomas, polyposis syndromes, and Crohn's disease. Patients often remain asymptomatic for long periods of time, and up to 80% of patients have distant metastases at the time of diagnosis. The presenting symptoms depend on the location of the primary tumor. Periampullary tumors can present with painless jaundice, duodenal obstruction, or bleeding. Jejunal or ileal tumors tend to present with abdominal pain and weight loss, which may be due to the progressive obstruction of the bowel lumen. Contrast studies, endoscopy with or without retrograde cholangiopancreatography, and computed tomography can be used to make the diagnosis. Treatment consists of segmental resection of the small bowel and its adjacent mesentery. Any adherent structures should be resected *en bloc* if possible. Tumors of the terminal ileum should be resected along with the right colon. For carcinomas of the duodenum, a Whipple procedure is usually required. Overall 5-year survival is 38% for all patients with a small-bowel adenocarcinoma. The survival can reach 63% for node-negative disease and decreases to 50% for node-positive disease (*Am J Surg* 173:237, 1997). In patients with metastatic disease at the time of diagnosis, survival of more than 6 months is rare.
2. **Gastrointestinal stromal tumors (leiomyosarcomas)** account for 10–20% of small-bowel neoplasms. These tumors can arise from any one of the mesodermal-derived components of the small-intestinal wall, such as the muscular, nervous, connective tissue, fat, or vascular elements. As was mentioned previously, the distinction between benign and malignant lesion can only be made histologically. This may prove to be difficult and is based on the number of mitotic figures per high-powered field (mf/hpf). GI stromal tumors can be located anywhere throughout the small intestine, and their distribution is proportional to the length of each anatomic division (jejunum > ileum > duodenum). These tumors grow extraluminally and cause symptoms late in their course. Because of their vascular nature, the tumors can outgrow their blood supply, leading to tumor necrosis, which can result in hemorrhage into either the peritoneum or the lumen of the bowel. The management of GI stromal tumors is surgical resection. Wide *en bloc* surgical resection to obtain tumor-free margins must be performed for curative therapy. Extensive lymph node resection is unnecessary because these tumors have a low potential for lymphatic spread. Patients with metastatic disease should also undergo resection of the primary tumor to prevent complications of bleeding or obstruction. Adjuvant chemotherapy and radiation therapy have not been shown to be effective in the treatment of GI stromal tumors. However, a recent report has shown metastatic GI stromal tumors to respond to ST1571, an inhibitor of tyrosine kinase activity. These tumors express a proto-oncogene named *c-kit*, whose gene product is a growth factor receptor with tyrosine kinase activity. Inhibition of this receptor has shown radiographic and histologic regression of metastatic lesions (*N Engl J Med* 344:1052, 2001). The benefit of resection of isolated pulmonary or hepatic lesions is unknown, but an aggressive approach is warranted in certain selected patients. Histologic grade, determined by the number of mf/hpf, and tumor size are the most important predictors of survival. Overall, 5-year survival is 40–50%. This improves to 60–80% after surgical resection of low-grade tumors (<10 mf/hpf) and falls to less than 20% for high-grade tumors (>10 mf/hpf).
3. **Primary small-bowel lymphomas** account for approximately 15% of all small-bowel neoplasms. Fifteen percent of GI lymphomas occur in the small intestine, with the stomach being the most common site. Virtually all small-bowel lymphomas are non-Hodgkin's, B-cell lymphomas. Lymphomas can arise *de novo* in the lymphoid tissue of the small bowel or in association with a preexisting systemic condition such as celiac disease, Crohn's disease, or several forms of immunosuppression (pharmacologic immunosuppression or HIV). The presentation of these patients can range from fatigue, malaise, weight loss, and abdominal pain to obstruction, bleeding, or perforation. Imaging studies, such as CT scans or small-bowel contrast studies, can help with the diagnosis, but surgery is frequently required for histologic confirmation of the diagnosis. Treatment of lymphoma that is localized to the small bowel involves wide resection of that segment of intestine and its associated mesentery. To stage the tumor accurately, the liver should be biopsied and the periaortic lymph nodes should be sampled. For widespread disease, resection of the affected small bowel should be performed to prevent complications. The role of adjuvant chemotherapy and radiotherapy remains controversial. Five-year survival for patients with fully resected disease approaches 80%, but individuals with more advanced disease usually die within a year of surgery.
4. **Metastases** from cutaneous melanoma as well as carcinomas of the cervix, kidney, breast, and lung can spread to the small bowel. Small-bowel metastases are found in 50% of patients dying from malignant melanoma. Palliative surgical resection is performed for lesion-causing symptoms, such as obstruction or bleeding.

C. Carcinoid tumors arise from the enterochromaffin cells of the gastrointestinal tract. Eighty-five percent of carcinoid tumors are found in the appendix. Thirteen percent are found in the small intestine, with ileum harboring the vast majority of them. Finally, 2% are found in the rectum. Small-bowel carcinoid tumors tend to be much more aggressive than their appendiceal or rectal counterparts. Most patients do not manifest signs or symptoms of the tumor until late in the course. Carcinoid tumors can produce symptoms locally by causing intestinal obstruction, pain, or bleeding, or they can produce systemic symptoms of the carcinoid syndrome. The tumor causes an intense mesenteric desmoplastic reaction that can lead to shortening and fibrosis of the mesentery. This reaction can cause obstruction due to kinking or narrowing of the bowel. The presence of the carcinoid syndrome implies the presence of hepatic metastases. For tumors smaller than 2 cm in size, only 2% will have metastasized, whereas 80% of tumors larger than 2 cm will have spread.

1. **Carcinoid syndrome** is a spectrum of symptoms that is caused by the oversecretion of active hormones produced by the tumor. The presence of the syndrome implies that hepatic metastases are present, because it is the hepatic tumor burden that is releasing the hormones into the systemic circulation. The clinical hallmarks of the syndrome are diarrhea and flushing of the face, neck, and upper chest, which can last for seconds to minutes. Tachycardia, hypotension, and bronchospasm can also occur. If the carcinoid syndrome is long-standing, it can lead to right heart endocardial and valvular fibrosis. The

hormonal mediators that are responsible for the syndrome are unclear. Serotonin, bradykinin, substance P, histamine, dopamine, and prostaglandins have all been implicated. The diagnosis of carcinoid syndrome is confirmed by finding increased urinary excretion of 5-hydroxyindoleacetic acid, a metabolite of serotonin.

2. Treatment

- a. The management of carcinoid tumors is **surgical**. The type of surgery depends on the location and size of the tumor. The entire small bowel should be inspected because 30% of the time synchronous lesions are present. Jejunal and ileal tumors should be treated with segmental resection including the adjacent mesentery. Small tumors (<1 cm) that are located in the third or fourth portions of the duodenum can either be locally excised or can be included in a segmental resection. For large tumors and periampullary tumors, a Whipple procedure should be performed. In the presence of locally advanced disease with involvement of adjacent organs or peritoneum, aggressive surgical resection should be undertaken. This can help delay the occurrence of the mesenteric desmoplastic reaction or hepatic metastases and the carcinoid syndrome. Solitary and accessible liver lesions should be resected. Adjuvant cytotoxic chemotherapy is of little benefit. Palliation for unresectable hepatic metastases can sometimes be achieved with hepatic artery embolization, radiofrequency ablation, or chemotherapy (*World J Surg* 22:309, 1998).
 - b. The somatostatin analogue **octreotide** offers excellent palliation of carcinoid syndrome in patients with unresectable disease. Octreotide decreases the concentration of circulating serotonin and urinary 5-hydroxyindoleacetic acid (5-HIAA) and can relieve the diarrhea and flushing in 90% of patients.
 - c. Carcinoids are slow-growing tumors, and **prognosis** depends on the stage of the tumor. The overall 5-year survival is 60%. Patients with local disease that is completely resected have a normal life expectancy. For patients with resectable node-positive disease, median survival is 15 years. Median survival drops to 5 years with unresectable intraabdominal disease, and it is 3 years for patients with hepatic metastases.
- D. **Peritoneal carcinomatosis** is defined as diffuse studding of the peritoneal, mesenteric, and bowel surfaces by tumor nodules. Common neoplasms that are associated with peritoneal carcinomatosis include carcinomas of the pancreas, stomach, ovaries, and colon. The use of staging laparoscopy in pancreatic cancer before laparotomy enhances the sensitivity for detecting peritoneal carcinomatosis and hepatic metastases, which are frequently missed by more conventional staging modalities. Surgical treatment is generally palliative, given the extremely poor prognosis.

V. Disturbances in small-bowel physiology

- A. **Motility** of the small bowel is controlled by the interaction of intrinsic electromechanical characteristics of the smooth muscle, the myenteric nerve plexus, the central nervous system, and various GI hormones. During times of feeding, small-bowel motility aids in the digestion and absorption of nutrients. It promotes contact of the chyme with the various digestive enzymes and gut mucosa as well as delivering the nutrients to the portion of the intestine that is responsible for their absorption. During the fasting state, the intrinsic electrical activity of the migrating motor complex serves a “housekeeper” function by clearing the intestinal lumen of residual food debris, bacteria, and desquamated cells. Disorders in small-bowel motility can be primary (e.g., congenital defect in the nerve or muscle development) or acquired as result of surgical intervention, medications, or systemic disease.
1. **Primary hypomotility disorders** are a dysfunction of the small bowel's intrinsic nervous system or musculature. This can result from a congenital absence or an acquired absence due to systemic diseases such as scleroderma, lupus, diabetes mellitus, amyloidosis, or muscular dystrophy. The clinical presentation can be that of a small-bowel obstruction or ileus. Radiographically, there is air/gas throughout the entire GI tract, with particular distention of the small bowel. The treatment for primary hypomotility is medical rather than surgical. Medical therapy includes the use of prokinetic drugs (e.g., metoclopramide or erythromycin) and manipulation of the diet.
 2. **Postvagotomy syndromes** occur after vagotomy with or without gastric resection. These syndromes include **gastric atony**, **dumping**, and **diarrhea**. **Gastric atony** can cause symptoms of nausea, abdominal pain, bloating, and vomiting after meals, which is typically worse with solids than liquids. Initial treatment is with promotility agents. If medical management fails, surgical resection of the remaining gastric remnant is indicated. **Dumping syndrome** is a sequela of the loss of normal gastric and pyloric function. Its treatment is dietary modification, such as voiding high concentrated carbohydrates and not eating and drinking simultaneously. **Postvagotomy diarrhea** results from rapid transit of food through the GI tract. Management is the same diet modifications that are used to treat dumping syndrome.
- B. **Water absorption.** Under normal circumstances, approximately 7–10 L of fluid enters the small intestine each day. Of this fluid, 2 L is derived from oral intake, 1 L from saliva, 2 L from gastric secretion, 2 L from pancreatic secretion, 1 L from bile, and 1 L from small-intestinal secretion. Only 600–1,500 mL fluid reaches the large bowel, indicating a net absorption of fluid. Alterations in small-bowel permeability, tonicity of enteric substances, or rate of transit can result in diarrhea and large volume losses.
- C. **Electrolyte absorption.** The majority of electrolyte absorption occurs in the small intestine. The most important electrolytes absorbed are sodium, chloride, and calcium. Sodium absorption occurs through passive diffusion, countertransport with hydrogen, and cotransport with chloride, glucose, and amino acids. Chloride is absorbed in exchange for bicarbonate, which accounts for the alkalinity of the luminal contents. Calcium is actively absorbed in the proximal small intestine by a process that is stimulated by 1,25-dihydroxyvitamin D₃. Emesis, diarrhea, obstruction, and small-bowel ostomy effluent can result in impaired small-bowel electrolyte absorption.
- D. **Nutrient absorption.** All physiologically significant nutrient absorption occurs in the small intestine.
1. **Carbohydrates**, in the form of starch, sucrose, lactose, and maltose, are the main source of caloric nutrition. The average Western adult consumes 400 g carbohydrates a day, which yields 1,600 kcal. Absorption of carbohydrates occurs after they have been digested by amylase from saliva glands followed by carbohydrate-specific enzyme hydrolysis at the brush border membrane of the small-intestinal mucosa. Patients with carbohydrate malabsorption, such as lactose intolerance, may present with watery diarrhea, hyperflatulence, bloating, and cramping abdominal pain. Lactose intolerance is diagnosed by a trial of lactose-free diet, the hydrogen breath test, or the lactose absorption test. Intestinal biopsies and measurement of disaccharidase activity identify patients with specific disaccharidase deficiencies.
 2. **Protein** is absorbed in the small intestine as amino acids. It is digested into amino acids in the stomach by pepsin and in the duodenum by pancreatic proteolytic enzymes. Protein malabsorption becomes clinically apparent only when it is severe enough to produce hypoalbuminemia that leads to peripheral edema and ascites. Quantitative stool protein determination is performed by placing the patient on a protein diet of 100 g per day. Normal fecal protein levels should be less than 2.5 g per day.
 3. **Fat absorption.** After digestion by lingual lipase, gastric lipase, and pancreatic lipase, fat is absorbed as micelles, which are formed with the aid of bile salts. Malabsorption of fat is the most frequently encountered malabsorption syndrome. Symptoms include weight loss, diarrhea, and steatorrhea. Diagnostic testing includes fecal fat determination and D-xylose analysis. Qualitative fecal fat analysis is performed with Sudan black staining and quantitative 72-hour fecal fat determination. For accurate results, the patient should be placed on a fixed fat diet (usually 100 g per day) beginning 24 hours before stool collection is initiated. Malabsorption is diagnosed when more than 6 g fecal fat is present in 24 hours. The D-xylose test, in which 25 g D-xylose is given orally followed by collection of urine for 5 hours with determination of D-xylose excretion, is a screening study for the presence of diffuse small-bowel mucosal disease.
 4. **Vitamin or mineral malabsorption.** Deficiencies in the fat-soluble vitamins A, D, E, and K may result from steatorrhea or resection of the ileum. Diseases of the terminal ileum can cause malabsorption of bile acids and vitamin B₁₂. Chronic anemia can result from malabsorption of iron and vitamin B₁₂. The Schilling test is used to diagnose vitamin B₁₂ malabsorption, although it is inaccurate in patients with pancreatic insufficiency.
- E. **Specific disorders of small-intestinal physiology**
1. **Medical conditions** of the small bowel that are associated with malabsorption include celiac sprue, lactose intolerance, radiation enteritis, Crohn's disease, Zollinger-Ellison syndrome, VIPoma (vasoactive intestinal polypeptide-producing tumor), tuberculosis, and occlusive vascular disease. Diseases of the pancreas and liver can result in gut malabsorption. Pancreatic insufficiency from chronic pancreatitis or excessive pancreatic resection can result in fat and protein malabsorption. Presentation includes weight loss, steatorrhea, and abdominal pain. An abnormal glucose tolerance test, glucosuria, and a low serum calcium level are also suggestive of pancreatic insufficiency. Hepatitis and cirrhosis are the most common hepatic diseases that can cause malabsorption. In these conditions, the number of bile salts in the enterohepatic circulation are decreased, which leads to malabsorption of fats, calcium, and the fat-soluble vitamins A, D, E, and K.
 2. **Surgical conditions associated with malabsorption**
 - a. **Partial or total gastrectomy** can lead to a variety of metabolic abnormalities and intestinal malabsorption. Deficiencies in total body fat, protein, calcium, fat-soluble vitamins, and vitamin B₁₂, as well as anemia, can develop. A Billroth I reconstruction is less likely to cause malabsorption than a Billroth II reconstruction, which is associated with a 20% incidence of steatorrhea. Vitamin B₁₂ deficiency results from the loss of production of intrinsic factor by the gastric mucosa and presents as megaloblastic anemia. Treatment consists of oral iron, calcium, and vitamin supplements. Medium-chain triglycerides and dipeptides may help if fat and protein malabsorption are present.
 - b. **Vagotomy** affects small-bowel motility, the mixing and reservoir functions of the stomach, pancreatic and biliary secretions, and gallbladder emptying. After truncal vagotomy and antrectomy, diarrhea develops in 15–20% of patients. The frequency of stools typically decreases over time, and only 1–2% of patients ultimately require long-term antidiarrheal therapy. However, with a highly selective vagotomy the incidence of diarrhea is approximately 1%. The diarrhea is a result of impaired biliary or pancreatic function, increased fecal excretion of bile salts and acids, and gastric dumping. Antispasmodics, such as codeine, diphenoxylate hydroxylate, or loperamide, along with dietary changes help to ameliorate postvagotomy diarrhea.
 - c. **Enteric fistulas** can cause significant syndromes of malabsorption either by bypass of intestine or by large loss of fluid, electrolytes, and nutrients. **Gastrocolic fistulas** cause malabsorption by diverting ingested food into the colon, which results in nutrients bypassing the small bowel. Also, the fistula

enables the colonic flora to reflux into the stomach, allowing bacterial overgrowth of the stomach and small bowel. The effects of an enteric fistula depend on the location, daily output, and organs involved. Low-output fistulas (<200 mL per day) seldom cause malnutrition. Proximal, high-output fistulas (>1,000 mL per day) frequently result in significant malnutrition. Biliary and pancreatic fistulas can lead to major derangements of fat, protein, and starch absorption. In cases of biliary fistulas, the bile can be collected and re-fed to the patient.

- d. **Short-bowel syndrome** after massive small-bowel resection results in serious nutritional and absorptive abnormalities when up to 3 m of functional small intestine remains. The goals of therapy are to recover from the inciting event, maintain nutritional status, maximize enteral nutritional absorption, and prevent complications. All patients should be started on total parenteral nutrition (TPN). Individuals with less than 100 cm of small bowel will most likely require lifelong TPN. Patients with 1–3 m of small intestine can expect to rely on oral food intake to obtain the complete nutritional requirements. Medical therapy that is directed at increased dietary fiber, inhibition of bowel secretion and motility, reduction of gastric hypersecretion, binding of bile salts, and prevention of bacterial overgrowth can help to maximize intestinal absorption of nutrients and minimize the complications of malnutrition.
- e. **Blind loop syndrome** encompasses any process that causes stasis of small-bowel contents and subsequent overgrowth of bacteria. Strictures, diverticula, fistulas, or poorly emptying (dysfunctionalized) segments of small bowel (e.g., a narrowed afferent loop of a Billroth II or Roux-en-Y reconstruction) can cause a blind loop syndrome. Inflammatory bowel disease, scleroderma, and pseudo-obstructive syndromes may also lead to bacterial overgrowth. Patients present with steatorrhea, diarrhea, megaloblastic anemia, and malnutrition. Bacteria deconjugate bile salts, leading to fat and vitamin B₁₂ malabsorption. Carbohydrate absorption and protein absorption are also impaired. Treatment consists of broad-spectrum antibiotics and antidiarrheal drugs. Surgical treatment of underlying anatomic causes is indicated when medical therapy gives adequate relief of symptoms.

VI. Crohn's disease is an idiopathic, chronic, granulomatous inflammatory disease that can affect any part of the GI tract from the lips to the anus. It is a slowly progressive disease that is characterized by episodes of exacerbation and remission, and there is no known cure at this time.

A. **Epidemiology.** The etiology of Crohn's disease is unknown. However, there is a definite genetic component because Crohn's disease is 17–35 times more likely to develop in patients with a positive family history than in the general population. Smoking has been shown to increase risk for the development and recurrence of Crohn's disease. Other environmental components, such as infection and diet, have been inconclusively examined. The incidence ranges from 2.7 to 5.0 per 100,000, and the age distribution tends to be bimodal (ages 15–29 years and 55–70 years).

B. **Gross and microscopic pathology** are the keys to distinguishing Crohn's disease from ulcerative colitis, which is necessary because the management of these two diseases is distinctly different.

1. **Disease distribution.** The distal ileum is the most common site involved in the disease (75% of patients). In 15–30% of patients, disease is limited only to the small bowel. Disease that occurs in the small bowel and colon is found in 40–60% of patients, with only 25–30% having disease confined to the colon. The duodenum is involved in 1–7% of cases, and 3% of patients have anorectal disease only. Skip lesions occur in 15% of patients.
2. **Gross appearance.** Externally, bowel that is involved with Crohn's disease is thickened and rigid, has "fat creeping" onto the antimesenteric border, and is associated with a shortened fibrotic mesentery. The mucosal lesions include pinpoint hemorrhages, aphthous ulcers, deep linear fissures, and, ultimately, cobblestoning. It is common for these lesions to occur segmentally along the intestine versus being continually involved.
3. **Microscopic appearance.** Crohn's disease is characterized by full-thickness, transmural inflammation of the bowel wall. The inflammation begins adjacent to the crypts, which leads to the development of aphthous ulcers, linear fissures, and crypt abscesses. The transmural involvement can lead to sinus tracts and fistulas between crypt abscesses and adjacent segments of bowel. Granulomas are found in the bowel wall in 40–60% of patients, and they are detected in mesenteric lymph nodes in 25%.

C. Diagnosis

1. Clinical presentation

- a. **Diarrhea** occurs in more than 90% of patients and usually is not bloody when only the small bowel is involved. However, if the colon is involved, up to one-third of patients have bloody diarrhea. The diarrhea is a result of mucosal inflammation with decreased absorption and increased secretion as well as the loss of bile salts, which manifests as steatorrhea.
- b. **Abdominal pain** typically is intermittent, crampy, worse after meals, relieved by defecation, and poorly localized. Pain that becomes constant and localized signals parietal peritoneal inflammation.
- c. **Weight loss** occurs as a result of malabsorption, decreased oral intake, protein-losing enteropathy, and steatorrhea. Vitamin and mineral deficiencies and growth retardation occur in children with Crohn's disease.
- d. **Constitutional signs and symptoms**, such as malaise, fever, and anemia, are common. Abdominal fullness or a mass caused by thickened bowel, a phlegmon, or an abscess may be palpable. Patients can also present with a fever of unknown origin.
- e. **Anorectal disease** is a frequent manifestation of Crohn's disease, and it may precede development of intestinal symptoms by several years. Anorectal lesions of Crohn's disease are characterized as recurrent nonhealing anal fissures, large ulcers, complex anal fistulas, and perirectal abscesses. Other features that are suggestive of anorectal Crohn's disease are multiplicity of lesions, lateral fissures, deep ulcers of the perianal skin and anal canal, anal stricture, and multiple fistulas. Thus, patients with recurrent or atypical anorectal pathology of unclear etiology should be investigated for Crohn's disease.
- f. **Extraintestinal manifestations.** Lesions such as pyoderma gangrenosum, erythema nodosum multiforme, and aphthous stomatitis can involve the skin. Ocular lesions include conjunctivitis, iritis, and uveitis. Arthritis, ankylosing spondylitis, and hypertrophic osteoarthropathy can also develop, as can sclerosing cholangitis, which can lead to cirrhosis.

2. A thorough **physical examination** with special attention to the abdominal and anorectal areas is performed. No physical signs are pathognomonic for Crohn's disease, but the presence of peritoneal signs or an undrained perirectal abscess needs to be ruled out.

3. **Laboratory evaluation** is nonspecific and variable. A modestly elevated WBC count can indicate active disease; marked elevation suggests an abscess or other suppurative complications. Anemia due to iron, vitamin B₁₂, or folate deficiency is common. Thrombocytosis can occur in active disease. Elevated sedimentation rate and hypoalbuminemia can be present.

4. **Imaging studies** are indicated when establishing the diagnosis of Crohn's disease in a patient who requires a major change in medical therapy or when a complication develops and surgical intervention is required.

- a. **Contrast X-rays**, which include **small-bowel follow-through**, **enteroclysis**, and **water-soluble contrast enema**, are very valuable in the diagnosis and surveillance of Crohn's disease. **Air-contrast studies** give better mucosal detail and can detect aphthous ulcers, but single-contrast ("full-column") studies are more useful for detecting fistulas, stricture, and tumors. Small-bowel studies are helpful because they give an idea of the extent of disease, and contrast enemas can help determine whether the colon is involved.
- b. **Direct endoscopic visualization** of accessible areas is better than contrast X-ray at defining the presence, extent, and severity of mucosal disease. However, contrast X-rays are better at identifying fistulas and strictures. Colonoscopy can detect the presence of ulcers, deep fissures, and cobblestoning, and it allows for biopsies. The presence of granulomas is diagnostic for Crohn's disease. Colonoscopy is also important for cancer surveillance, because these patients have a 4–20 times increased risk of developing adenocarcinoma. In patients with active disease, great care needs to be taken during colonoscopic examination, as excess force or air insufflation can lead to perforation.
- c. **CT scan** is useful to identify abscesses and areas of focal inflammation and to assess bowel wall thickening.
- d. **Nuclear medicine** studies, such as **indium-111-tagged granulocytes** or **technetium-99-labeled phagocytes**, can be used to identify the location, extent, and severity of intestinal inflammation. However, these studies are of limited clinical use and are rarely used.

D. **Complications** of Crohn's disease include intestinal obstruction, abscess, fistula, stricture, perforation, and perirectal abscess and fistula. Toxic colitis or toxic dilatation of the ileum is a surgical emergency that can occur in these patients. Patients also have an increased risk of cancer in areas of small bowel or colon involved in the disease process. Surgically bypassed loops of intestine are a particularly high risk.

E. Differential diagnosis

1. **Ulcerative colitis.** Patients with Crohn's disease generally have less severe diarrhea, usually without gross blood. Perianal lesions, nonconfluent skip lesions, transmural involvement, large ulcers and fissures, involvement of other portions of the intestinal tract often with rectal sparing, and the presence of granulomas all help to differentiate Crohn's disease from ulcerative colitis. Patients who cannot be clearly differentiated are diagnosed with indeterminate colitis.
2. **Appendicitis.** Acute right lower quadrant abdominal pain due to inflammation of the distal ileum in Crohn's disease can mimic acute appendicitis.
3. **Ileitis** due to *Shigella* species, amebas, *Giardia* species, or *Yersinia* species can mimic Crohn's disease, and the diagnosis is made by stool culture.
4. **Intestinal lymphoma** often appears as diffuse involvement with masses in the bowel wall on X-rays, whereas Crohn's disease shows more localized involvement of the ileum with ulcers and strictures. Open surgical biopsy may be required to make the diagnosis.
5. **Intestinal tuberculosis** most commonly involves the cecum and ileum and resembles Crohn's disease clinically and pathologically.
6. Other diseases, such as **ischemic enteritis**, **diverticulitis**, **pseudomembranous colitis**, **irritable bowel syndrome**, and **carcinoma**, can also mimic Crohn's disease.

F. Treatment

1. **Medical management.** Crohn's disease has no cure. Therefore, treatment is aimed at control of the disease. The goals of medical management are to reduce bowel inflammation, provide symptomatic relief, and correct nutritional disturbances. Therapeutic recommendations depend on the disease location, severity, and complications.

- a. **Adequate nutritional intake** is essential in the treatment of disease flares and in the maintenance phase. Whenever possible, enteral feeds should be

continued. A low-residue, high-protein, milk-free diet can often provide adequate nutritional support for patients with mild to moderate disease flares. Supplementation with iron, calcium, magnesium, folate, zinc, and vitamins B₁₂, D, and K may be necessary. Nutritional requirements often need to be individualized given the disease location and history of surgery. Patients with bile salt deficiency due to ileal disease benefit from substitution of medium-chain triglycerides for dietary fat. Those with ileal resection or extensive small-bowel disease are prone to oxalate nephrolithiasis and should receive a low-oxalate, high-calcium diet. Patients with severe or unresponsive disease should be started on TPN and placed on complete bowel rest. Healing rates for Crohn's fistulas that are treated with TPN and bowel rest can approach 75%. Long-term TPN may be indicated for patients with short-gut syndrome.

2. **Medical therapy** is divided into **acute** and **maintenance phases**. Mild to moderate disease can be treated with an oral aminosalicylate (sulfasalazine, 3–6 g per day, or mesalamine, 3.2–4.8 g per day). For ileal or colonic disease, metronidazole, 500 mg three times a day, can be added. Metronidazole is also helpful for treating perianal disease. Maintaining nutritional intake is very important and can usually be met with oral intake. In patients with severe disease, steroid therapy should be initiated provided that an active infection or abscess has been excluded (prednisone, 20–40 mg/kg per day p.o.; methylprednisolone, 30 mg/kg i.v. every 6 hours; or hydrocortisone, 50–100 mg i.v. every 6 hours). Response to therapy should become evident within 7 days of its initiation. For patients with a poor response to therapy or a severe flare, maintaining nutritional support may require TPN and bowel rest. Compelling data have shown that infusions of growth hormone and a monoclonal antibody to tumor necrosis factor-alpha can be used to treat moderate to severe active disease (*N Engl J Med* 342:1633, 2000; *N Engl J Med* 337:1029, 1997). Once a patient has recovered from an acute flare of the disease, the medical regimen should be simplified to prevent long-term complications of the medications and recurrence of the disease. Steroids should be tapered as soon as possible.
3. **Surgical therapy**
 - a. **Surgical therapy** is not curative; therefore, indications for surgery include the correction of a complication, treatment of disease that is unresponsive to medical therapy, and prevention of carcinoma. Complications include obstruction (e.g., stricture, inflammation, or adhesion), perforation, enteric fistula, intraabdominal abscess, fulminant or toxic colitis, carcinoma, GI bleeding (rare), or growth failure due to persistent disease in children. Toxic colitis, free perforation, or failures of medical therapy with signs of peritonitis require emergent operations. However, most of the time an acute situation can be treated medically to allow for surgical intervention in a more semielective or elective setting. For example, abscesses can be drained percutaneously; fistulas can be “cooled down” with medical therapy, nutritional support, and bowel rest; and bowel obstructions can be initially managed with GI decompression, n.p.o. status, intravenous fluids, and medical therapy. Thus, recognizing the need for surgery and determining the timing of operation are the cornerstones to successful surgical management of patients with Crohn's disease. Approximately 70–80% of all patients with Crohn's disease require surgery at some point in their lifetime.
 - b. **Surgical procedures.** The most important principle in the surgical management of Crohn's disease is treatment of the specific indication for surgery while preserving as much bowel length as possible in order to try to prevent short-gut syndrome. It has become clear that the resection to histologically negative margins does not reduce the likelihood of disease recurrence. Therefore, resection should be limited only to grossly diseased bowel. The normal-appearing bowel should be primarily anastomosed. Diseased bowel is characterized by creeping mesenteric fat, bowel wall thickening, strictures, bowel and mesenteric foreshortening, enlarged mesenteric lymph nodes, inflammatory masses, abscesses, or adherent bowel loops. Minimal amounts of mesentery should be resected to retain maximal blood flow to the remaining bowel. Specific issues that require special consideration are gastroduodenal disease, multiple skip lesions, and chronic fibrotic strictures. Gastroduodenal Crohn's disease usually presents surgically as obstruction, which should be treated with resection when possible; otherwise, gastrojejunostomy bypass should be performed. When multiple segments of bowel are grossly involved, these short segments can be resected and primarily anastomosed. Preservation of intestinal length is the goal. For strictures of less than 10 cm in quiescent bowel, strictureplasty is the procedure of choice. Either a Heineke-Mikulicz or a Finney strictureplasty can be used. Short strictures that are within reach of an endoscope can be amenable to balloon dilation. In patients with matted bowel masses that are adherent to vital structures or those who are at high surgical risk, intestinal bypass with or without defunctionalizing the involved segment is acceptable. However, the risk of recurrence in the excluded segment of bowel is high.
 - c. **Appendectomy.** Patients who are being explored for presumed acute appendicitis and are found to have Crohn's disease of the distal ileum should have the appendix removed if the cecum is not inflamed. The terminal ileum should not be disturbed. If the cecum is involved with disease, appendectomy is not performed.
 - d. **Perianal disease.** The anorectal glands in the anterior and posterior midline are the source of most fistulas and perirectal abscesses in Crohn's disease. Preservation of the sphincter complex is the cornerstone of the surgical management of perianal disease. Examination under anesthesia with rigid proctoscopy is necessary to evaluate the extent of disease fully. Medical therapy with sitz baths, antibiotics, and control of diarrhea can be tried initially if no abscess is present. Adequate external drainage of abscesses, often using setons, drains, and unroofing of the fistulous tract, is mandatory.
- G. **Complications of surgery** include leaks and fistulas at the anastomotic sites and sepsis related to intraabdominal abscesses and wound infections. Urolithiasis and gallstones are common after intestinal resection for Crohn's disease. Overall, operative mortality is less than 5%.
- H. **Prognosis.** Crohn's disease is a panintestinal disease and currently has no cure. A high rate of Crohn's disease recurrence is found at intestinal anastomoses (50–75% at 15 years), and in 90% of patients recurrence ultimately develops despite resection. The rate of reoperation is 75% at 15 years. Reoperation after strictureplasty is required in one-third of patients by 10 years. Despite the incurable nature of Crohn's disease, 80–85% of patients who require surgery are able to lead normal lives. The mortality is 15% at 30 years, and the disease tends to “burn out.”

15 Acute Abdominal Pain and Appendicitis

David J. Finlay and Gerard M. Doherty

Acute Abdominal Pain

I. Background and anatomy

II. Types of abdominal pain

III. History

IV. Medical history

V. Medications

VI. Organ system review

VII. Physical examination

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Diagnosis and Treatment of Specific Disorders

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Acute Abdominal Pain

Evaluation of the patient with acute abdominal pain requires a careful history and physical examination by a skilled physician in conjunction with selective diagnostic testing.

I. Background and anatomy

A. Definition of the acute abdomen

1. **Recent or sudden onset of unexpected abdominal pain** (usually within 24–72 hours of presentation).
2. **Frequently, associated gastrointestinal (GI) signs and symptoms.** Differential diagnosis includes a variety of intra- and extraperitoneal processes. Acute abdomen does not invariably signify the need for surgical intervention.

B. Anatomy of the peritoneum

1. The **peritoneum** is a continuous layer with visceral and parietal components that develop separately and have separate nerve supplies.
 - a. The **visceral peritoneum** is innervated by the autonomic nervous system; innervation is bilateral, and visceral pain is usually midline and vague.
 - b. The **parietal peritoneum** is innervated by somatic nerves that supply the abdominal wall. Innervation is unilateral via the spinal somatic nerves; parietal pain, therefore, often lateralizes to one or more abdominal quadrants (e.g., inflamed appendix producing parietal peritoneal irritation).
2. The **embryologic origin** of the affected organ determines the level of visceral pain in the abdominal midline.
 - a. **Foregut-derived structures** (stomach to second portion duodenum, liver and biliary tract, pancreas, spleen) present with epigastric pain.
 - b. **Midgut-derived structures** (second duodenum to proximal two-thirds of the transverse colon) present with periumbilical pain (e.g., early appendicitis—visceral irritation).
 - c. **Hindgut-derived structures** (distal transverse colon to anal verge) present with suprapubic pain.

II. Types of abdominal pain

A. Visceral pain

is usually deep, dull, and poorly localized (e.g., vague periumbilical pain of the midgut).

1. It is **triggered** by inflammation, ischemia, and geometric changes such as distention, traction, and pressure.
2. It **signifies** intraabdominal disease but not necessarily the need for surgical intervention.

B. Parietal pain

is sharp, severe, and well localized.

1. It is **triggered** by irritation of the parietal peritoneum by an inflammatory process (e.g., chemical peritonitis from perforated peptic ulcer or bacterial peritonitis from acute appendicitis). It may also be triggered by mechanical stimulation, such as with a surgical incision.
2. It is **associated with** physical examination findings of peritonitis and frequently **signifies** the need for surgical treatment.

C. Referred pain

arises from a deep visceral structure but is superficial at the presenting site ([Fig. 15-1](#)).

Fig. 15-1. Frequent sites of referred pain and common causes.

1. It **results from** central neural pathways in the spinal cord that are common to the somatic nerves and visceral organs.
2. **Examples include** biliary tract pain (referred to the right inferior scapular area) as well as diaphragmatic irritation from any source, such as subphrenic abscess (referred to the ipsilateral shoulder).

III. History.

Pain that is severe and persists for longer than 6 hours should prompt a thorough surgical evaluation. Key elements in the history are as follows:

A. Onset and duration of pain

1. **Sudden onset of pain** (within seconds) suggests perforation or rupture [e.g., perforated peptic ulcer or ruptured abdominal aortic aneurysm (AAA)]. Infarction, such as myocardial infarction or acute mesenteric occlusion, can also present with sudden onset of pain.
2. **Pain that begins rapidly and accelerates** within minutes may result from several sources.
 - a. **Colic syndromes**, such as biliary colic, ureteral colic, and small-bowel obstruction (SBO)
 - b. **Inflammatory processes**, such as acute appendicitis, pancreatitis, and diverticulitis
 - c. **Ischemic processes**, such as mesenteric ischemia, strangulated intestinal obstruction, and volvulus
3. **Pain that is more gradual in onset and increases** in intensity over several hours may be caused by one of the following:
 - a. **Inflammatory conditions**, such as appendicitis and cholecystitis
 - b. **Obstructive processes**, such as nonstrangulated bowel obstruction and urinary retention
 - c. **Other mechanical processes**, such as ectopic pregnancy and penetrating or perforating tumors

B. Character of pain

1. **Colicky pain** is severe, sharp pain that is accompanied by intervals when the pain is less severe or absent.
 - a. It **occurs secondary to hyperperistalsis of smooth muscle** against a mechanical site of obstruction (e.g., SBO, renal stone).
 - b. **An important exception is biliary colic**, in which pain tends to be constant.
2. **Pain that is sharp, severe, and persistent and increases in intensity** over time suggests an infectious or inflammatory process (e.g., appendicitis).

C. Location of pain

1. **Pain caused by inflammation of specific organs** may be localized [e.g., right upper quadrant (RUQ) pain caused by acute cholecystitis].
2. **Careful attention must be given to radiation of pain.** The pain of renal colic, for example, may begin in the patient's back or flank and radiate to the ipsilateral groin, whereas the pain of a ruptured aortic aneurysm or pancreatitis may be referred to the patient's back.

D. Alleviating and aggravating factors

1. **Patients with diffuse peritonitis** describe worsening of pain with movement (i.e., parietal pain); therefore, the pain is ameliorated by lying still.
2. **Patients with intestinal obstruction** have visceral pain and usually experience a transient relief from symptoms after vomiting.

E. Associated symptoms

1. **Nausea and vomiting** frequently accompany abdominal pain and may hint at its etiology. Vomiting that occurs after the onset of pain may suggest appendicitis, whereas vomiting before the onset of pain is more consistent with the diagnosis of gastroenteritis or food poisoning. The sequence as well as the character of the emesis should be documented. Biliary emesis suggests a process distal to the pylorus.
 2. **Fever or chills** suggest an inflammatory or an infectious process, or both.
 3. **Anorexia** is present in the vast majority of patients with acute peritonitis.
- F. **Thorough menstrual history in women**
1. **Pelvic inflammatory disease (PID)** typically occurs early in the cycle and may be associated with a vaginal discharge.
 2. **Ectopic pregnancy** must be considered in any woman of childbearing age with lower abdominal pain, especially if accompanied by a history of amenorrhea.
 3. **Ovarian cysts** can cause sudden pain by enlarging or rupturing. The timing in relation to the menstrual cycle is key. A ruptured follicular cyst pain occurs at midcycle (i.e., mittelschmerz), whereas the pain of a ruptured corpus luteum cyst develops around the time of menses.
 4. **Abdominal pain that occurs monthly** suggests endometriosis.

IV. Medical history

- A. It is important to obtain a good **surgical history**. Previous abdominal surgery in a patient with colicky abdominal pain may suggest intestinal obstruction secondary to adhesions.
- B. **Other illnesses** may place a patient at risk for certain abdominal events.
 1. Patients with a history of **peripheral vascular disease** or **coronary artery disease and abdominal pain** may have an AAA or mesenteric ischemia.
 2. Patients with a history of **cancer** may present with bowel obstruction from recurrence.
 3. **Major medical problems** are important to recognize early in the patient who may require urgent surgical exploration.

V. Medications

- A. **Nonsteroidal antiinflammatory medications**, such as aspirin or ibuprofen, place patients at risk for the complications of peptic ulcer disease, including bleeding and perforation.
- B. **Corticosteroids** may mask classic signs of inflammation, such as fever and peritoneal irritation, making the abdominal examination less reliable.
- C. **Antibiotics** consumed by patients may aid or hinder diagnosis.
 1. Patients with **peritonitis** may have decreased pain.
 2. Patients who have **diarrhea and abdominal pain** may have antibiotic-induced pseudomembranous colitis caused by *Clostridium difficile*.
 3. Beware of the **elderly patient** on immunosuppressants or antibiotics.

VI. Organ system review must be carried out to exclude various extraabdominal causes of abdominal pain.

- A. **Diabetic patients** or patients with known coronary artery disease or peripheral vascular disease who present with vague epigastric symptoms may have myocardial ischemia as the cause of the abdominal symptoms.
- B. **Right lower lobe pneumonia** may present as RUQ pain in association with cough and fever.

VII. Physical examination

- A. **Overall appearance** should be assessed.
 1. Patients with **diffuse peritonitis** appear acutely ill and tend to lie quietly on their side with their knees drawn toward their chest.
 2. Patients with **colic** tend to be restless and unable to find a comfortable position. Patients with ureteral colic may writhe in pain and walk around the examination room.
 3. Patients who are **jaundiced** may have biliary obstruction from periampullary cancer, a biliary stone, or ductal stricture.
 4. Patients who appear **weak and lethargic** may be septic.
- B. **Vital signs** are important indicators of a patient's overall condition.
 1. **Fever** suggests the presence of inflammation or infection. Marked fever (>39°C) suggests an abscess, cholangitis, or pneumonia.
 2. **Hypotension or tachycardia, or both**, may indicate hypovolemia or sepsis.
- C. The **abdominal examination** should be carried out thoroughly and systematically.
 1. **The patient's abdomen should be inspected** for distention, surgical scars, bulges, and areas of erythema.
 2. **Auscultation** may reveal the high-pitched, tinkling bowel sounds of obstruction or the absence of sounds due to ileus from diffuse peritonitis.
 3. **Percussion of the patient's abdomen** may reveal the tympanitic sounds of distended bowel in intestinal obstruction or the fluid shift that is characteristic of ascites. Percussion is also a useful tool for the delineation of localized tenderness and peritoneal irritation (deep palpation or rebound is usually unnecessary to determine peritoneal irritation).
 4. **Palpation of the patient's abdomen** should be performed with the patient in a supine position and with his or her knees flexed, if necessary, to relieve pain.
 - a. **Begin the examination at a point remote from the site of pain.**
 - b. **Areas of tenderness and guarding should be noted.** Rebound tenderness is not a very reliable sign of peritonitis. The presence of involuntary guarding (localized or diffuse) due to muscular rigidity from underlying peritoneal irritation is often a better sign of peritonitis. Peritonitis may also be elicited by rocking the patient's pelvis or shaking the bed to create friction between the abdominal wall and peritoneal viscera.
 - c. **Pain out of proportion to physical examination findings** may suggest mesenteric ischemia.
 - d. **A thorough search for hernias**, including femoral hernias, must be carried out.
 - e. **Any palpable masses should be noted.**
 5. **Rectal examination** should be performed routinely in all patients with abdominal pain.
 - a. **Tenderness or mass on the right pelvic side wall** is sometimes seen in appendicitis.
 - b. A **mass in the rectum** may indicate obstructing cancer. Important details are the fraction of circumference involved, tumor mobility, and distance from the anal verge.
 - c. The **presence of occult blood in the stool specimen** may indicate GI bleeding from peptic ulcer disease. Beware of the positive Hemoccult following rectal examination.
 6. **Pelvic examination** must be performed in all women of childbearing age who present with lower abdominal pain.
 - a. **Cervical discharge** and overall appearance of the cervix should be noted.
 - b. **Bimanual examination should be performed** to assess cervical motion tenderness, adnexal tenderness, or the presence of adnexal masses.
 7. **Specific physical examination findings** should be sought in the appropriate clinical setting.
 - a. **Murphy's sign** is inspiratory arrest while maintaining continuous pressure in the RUQ. Seen in acute cholecystitis, Murphy's sign reflects the descent of an inflamed gallbladder with inspiration. When the inflamed gallbladder makes contact with the examiner's hand, the patient experiences pain, causing the inspiratory arrest. A sonographic Murphy's sign may be elicited during ultrasound (US) palpation of the gallbladder with the US probe tip (this is secondary to direct pressure and is less reliable).
 - b. The **obturator sign** reflects inflammation adjacent to the internal obturator muscle (as is sometimes seen in appendicitis); it may also be present with an obturator hernia. While the patient is in a supine position with the knee and hip flexed, the hip is internally and externally rotated. The test is positive if the patient experiences hypogastric pain.
 - c. The **iliopsoas sign** is seen when an adjacent inflammatory process irritates iliopsoas muscle. It is classically observed in retrocecal appendicitis. The patient's thigh is usually already drawn into a flexed position for relief. The test is best performed with the patient lying on the left side. With the knee flexed, the thigh is hyperextended. The test is positive if the patient experiences pain on the right side with this maneuver.
 - d. **Rovsing's sign** may also be seen in acute appendicitis. Indicative of an inflammatory process in the right lower quadrant (RLQ), Rovsing's sign is RLQ pain from percussion in the left lower quadrant.

VIII. Laboratory evaluation. Selective use of laboratory tests in an appropriate clinical setting is often essential to the diagnosis.

- A. A **complete blood count with cell count differential** is important in the assessment of surgical conditions and should be done in every patient with acute abdominal pain.
 1. **White blood cell (WBC) count elevation** may indicate the presence of an infectious source (e.g., ruptured appendix).
 2. **Left shift on the differential** to more immature forms is often helpful because this may indicate the presence of an inflammatory source even if the WBC count is normal.

3. **Hematocrit elevation** may be due to volume contraction from dehydration. Conversely, a low hematocrit may be due to occult blood loss.
- B. An **electrolyte profile** may reveal clues to the patient's overall condition.
 1. A **hypokalemic, hypochloremic, metabolic alkalosis** may be seen in patients with prolonged vomiting and severe volume depletion. The hypokalemia reflects the potassium-hydrogen ion exchange at the cellular level in an effort to correct the alkalosis.
 2. **Elevation of the blood urea nitrogen or creatinine** is also indicative of volume depletion.
- C. **Liver function tests** may be obtained in the appropriate clinical setting.
 1. A **mild elevation of transaminases** (<2× normal), alkaline phosphatase, and total bilirubin is sometimes seen in patients with acute cholecystitis.
 2. A **moderate elevation of transaminases** (>3× normal) in the patient with acute onset of RUQ pain is most likely due to a common bile duct (CBD) stone. Elevation of the transaminases often precedes the rise in total bilirubin and alkaline phosphatase in patients with acute biliary obstruction (e.g., from a CBD stone).
 3. **Markedly elevated transaminases** (i.e., >1,000 IU/L) in the patient without pain are more likely due to hepatitis or ischemia.
- D. **Pancreatic enzymes** (i.e., amylase and lipase) should be obtained if the diagnosis of pancreatitis is considered. It is important to note that the degree of enzyme elevation does not correlate with the severity of the pancreatitis.
 1. **Mild degrees of hyperamylasemia** may be seen in several situations, such as intestinal obstruction.
 2. **Elevation of lipase** usually indicates pancreatic parenchymal damage.
- E. **Urinalysis** with microscopic examination is helpful in assessing urologic causes of abdominal pain.
 1. **Bacteriuria, pyuria, and a positive leukocyte esterase** usually suggest a urinary tract infection (UTI). Recurrent UTI in males should always elicit an evaluation for etiology, as this is unusual.
 2. **Hematuria** is seen in nephrolithiasis and/or renal as well as urothelial cancer.
- F. **Beta-human chorionic gonadotropin** must be obtained in any woman of childbearing age. A positive urine result should be quantitated by serum levels.
 1. A low level (<4,000 mIU) is seen in **ectopic pregnancy**.
 2. Levels above 4,000 mIU indicate **intrauterine pregnancy** (i.e., one that should be seen on US).

IX. Diagnostic imaging. Radiologic evaluation of the patient with abdominal pain is a key element in the workup. However, it must be emphasized that the use of the various modalities available should be very selective to avoid the unnecessary cost and possible morbidity associated with some modalities. For instance, radiographic studies are not indicated in most young adult patients who present with classic signs and symptoms of acute appendicitis.

- A. **Plain abdominal films** often serve as the initial radiologic evaluation.
 1. **Films should be obtained** in the supine and erect positions.
 2. **Free intraperitoneal air is best visualized** on an upright chest film with both hemidiaphragms exposed.
 - a. **If the patient is unable to assume an upright position**, a lateral decubitus film, with the patient's left side down, should be obtained.
 - b. **Free air may not be detectable** in up to 20% of cases of a perforated viscus.
 3. The **bowel gas pattern** should be assessed with regard to dilatation, air-fluid levels, and the presence of gas throughout the small and large intestine.
 - a. In **SBO**, one sees small-bowel dilatation (valvulae conniventes), air-fluid levels, and a paucity of gas in the colon or in the segment of bowel distal to the obstruction. The absence of air in the rectum suggests complete obstruction (beware of the presence of colonic gas following rectal examination).
 - b. A **sentinel loop** (i.e., a single, dilated loop of bowel) may be seen adjacent to an inflamed organ (as in pancreatitis) and is due to localized ileus in that segment.
 4. **Calcifications** should be noted.
 - a. **The vast majority of urinary stones (90%) are visible** on plain films, whereas only 15% of gallstones are calcified and, therefore, visible on plain films.
 - b. **Calcifications in the region of the pancreas** may indicate chronic pancreatitis.
 - c. **Fecalith in the RLQ may suggest appendicitis** in the appropriate clinical setting.
 - d. **Calcification in the wall of the aorta** may suggest an AAA.
 - e. The most common calcifications seen in the abdomen are "**phleboliths**" (benign calcifications of the pelvic veins). Phleboliths can be distinguished from renal stones by their central lucency, which represents the lumen.
 5. The **presence of gas** in the portal or mesenteric venous systems, **intramural gas** in the GI tract, or **gas in the biliary tree** (in the absence of a surgical enteric anastomosis) may indicate an ominous condition in the appropriate clinical setting.
- B. **US** may provide diagnostic information in some conditions. This modality has the advantages of being portable, relatively inexpensive, and free of radiation exposure. Unfortunately, US visibility is limited in settings of obesity, bowel gas, and subcutaneous air.
 1. **RUQ US** is particularly useful in biliary tract disease.
 - a. **Gallstones** can be detected in up to 95% of patients.
 - b. Findings suggestive of **acute cholecystitis** include gallbladder wall thickening of greater than 3 mm, pericholecystic fluid, or a stone in the neck of the gallbladder.
 - c. **Dilatation of the CBD** (>8 mm, or larger in elderly patients), indicative of obstruction, may be detected.
 - d. **Sonographic Murphy's sign** may be present, indicating gallbladder inflammation.
 2. **Pelvic or transvaginal US** is particularly useful in women in whom ovarian pathology or an ectopic pregnancy is suspected.
 3. US is also sometimes used in the evaluation of **RLQ pain**.
 - a. It may be helpful in the **diagnosis of appendicitis**, particularly in the pediatric population or in nonobese adults.
 - b. It should be used in **selected cases only** given the institutional experience with this technique.
- C. **Contrast studies**, although rarely indicated in the acute setting, may be helpful in some situations.
 1. In most instances, a **water-soluble contrast agent** (e.g., Hypaque) should be used to avoid possible barium peritonitis.
 2. **Contrast enema** is particularly useful in differentiating adynamic ileus from distal colonic obstruction.
- D. **Computed tomographic (CT) scanning** may provide a thorough evaluation of the patient's abdomen and pelvis in a relatively short period. Oral and intravenous contrast should be administered if not specifically contraindicated. CT scanning is the best single radiographic study in the patient with unexplained abdominal pain. It is of particular benefit in certain situations, including
 1. In **patients in whom an accurate history cannot be obtained** (e.g., demented or obtunded patients or those in whom the history is atypical)
 2. In **patients with abdominal pain, leukocytosis, and examination findings that are worrisome** but not definitive for peritoneal irritation
 3. When there is acute abdominal pain in **a patient with a chronic illness** (e.g., Crohn's disease)
 4. For **evaluation of retroperitoneal structures** (e.g., stable patients with suspected leaking AAA)
 5. In **patients with a history of intraabdominal malignancy**
- E. **Radionuclide imaging studies** have few indications in the acute setting.
 1. **Biliary radiopharmaceuticals**, such as hepatic 2,6-dimethyliminodiacetic acid or disopropyliminodiacetic acid, may be used to evaluate filling and emptying of the gallbladder in a patient with suspected acute cholecystitis or a nondiagnostic US examination. Nonfilling of the gallbladder implies cystic duct obstruction and may indicate acute cholecystitis in the appropriate clinical setting. This test is especially valuable in the diagnosis of acalculous cholecystitis.
 2. **Radioisotope-labeled red blood cell (RBC) or WBC scans** are sometimes helpful in delineating sites of bleeding or inflammation, respectively.
 3. **Technetium-99m pertechnetate** may be used to detect a Meckel's diverticulum because this isotope is concentrated in the ectopic gastric mucosa frequently seen within the diverticulum.
- F. **Invasive radiologic techniques** may have a role in some situations, including
 1. **Angiographic diagnosis of suspected mesenteric arterial occlusion**
 2. **Diagnosis and therapy of acute GI bleeding**

X. Differential diagnosis of acute abdominal pain ([Fig. 15-1](#))

- A. **Upper abdomen**
 1. **Perforated peptic ulcer**
 2. **Acute cholecystitis**
 3. **Acute pancreatitis**
- B. **Lower or midabdomen**
 1. **Acute appendicitis**
 2. **Acute diverticulitis**
 3. **Intestinal obstruction**
 4. **Mesenteric ischemia**
 5. **Ruptured AAA**
- C. **Other causes**

1. **Gynecologic and obstetric**
 - a. **PID**
 - b. **Ectopic pregnancy**
 - c. **Ruptured ovarian cyst**
2. **Urologic**
 - a. **Nephrolithiasis**
 - b. **Pyelonephritis or cystitis**
3. **Nonsurgical causes**
 - a. **Myocardial infarction, especially in diabetic patients**
 - b. **Gastroenteritis**
 - c. **Pneumonia**
 - d. **Diabetic ketoacidosis**

Diagnosis and Treatment of Specific Disorders

I. Specific diseases presenting as an acute abdomen

- A. **Acute appendicitis.** The most common surgical procedure performed on an emergency basis is an appendectomy. Acute appendicitis develops in approximately 10% of the population in Western countries. The maximal incidence occurs in an individual's teens and 20s. The male-female ratio of approximately 2:1 gradually declines after age 25 until the gender ratio is equal.
1. **Pathophysiology.** Appendiceal obstruction is the most common initiating event of appendicitis. Hyperplasia of the submucosal lymphoid follicles of the appendix accounts for approximately 60% of obstructions (most common in teens), whereas in older adults and children the fecalith is the most common etiology (35%). Obstruction of the appendiceal lumen is followed by increased intraluminal pressure secondary to continued mucosal secretion and bacterial overgrowth. As the pressure increases, the appendiceal wall becomes thinned and lymphatic and venous obstruction occurs. Necrosis and perforation develop when the arterial flow to the appendix is compromised.
 2. **Diagnosis.** The diagnosis of acute appendicitis is made by clinical evaluation. Although laboratory tests and imaging procedures can be helpful, they are of secondary importance.
 - a. **Clinical presentation**
 1. **Symptoms.** Appendicitis typically begins with progressive, persistent midabdominal discomfort caused by obstruction and distention of the appendix, which stimulates the visceral afferent autonomic nerves (T8-10 distribution). This is followed by anorexia and a low-grade fever (<38.5°C). As distention of the appendix increases, venous congestion stimulates intestinal peristalsis, causing a cramping sensation that is soon followed by nausea and vomiting. Ninety percent of patients are anorexic, 70% become nauseated and vomit, and 10% have diarrhea. Once the inflammation extends transmurally to the parietal peritoneum, the somatic pain fibers are stimulated, and the pain localizes to the RLQ. Peritoneal irritation is associated with pain on movement, mild fever, and tachycardia. One-fourth of patients present initially with localized pain and no prior visceral symptoms. The onset of symptoms to time of presentation is usually less than 24 hours for acute appendicitis and averages several hours.
 2. **Physical examination**
 - a. **The examination begins** by assessing the patient's abdomen in areas other than the area of suspected tenderness. Location of the appendix is variable. However, the base usually is found at the level of the S1 vertebral body, lateral to the right midclavicular line at **McBurney's point** (two-thirds of the distance from the umbilicus to the anterosuperior iliac spine). The presence of pain in the RLQ during gentle finger percussion in the LLQ (**Rovsing's sign**) indicates peritoneal irritation. The degree of direct tenderness is appreciated. The degree of muscular resistance to palpation parallels the severity of the inflammatory process. Cutaneous hyperesthesia is often present overlying the region of maximal tenderness. Exacerbation of pain with passive stretching of the iliopsoas muscle (positive psoas sign) implies the presence of local inflammation in the area of the muscle (e.g., retrocecal appendicitis). A pelvic appendix may produce hypogastric pain with passive internal rotation, a positive obturator sign.
 - b. **Rectal examination** is performed to evaluate the presence of localized tenderness or an inflammatory mass in the pararectal area. It is most useful for atypical presentations that are suggestive of a pelvic or retrocecal appendix.
 - c. **In women, a pelvic examination is performed** to assess the presence of cervical motion tenderness and adnexal pain or masses.
 - d. **A palpable mass in the RLQ suggests a periappendiceal abscess or phlegmon.**
 3. **Unusual presentations of appendicitis**
 - a. **When the appendix is retrocecal or behind the ileum**, it may be separated from the anterior abdominal peritoneum, and abdominal localizing signs may be absent. Irritation of adjacent structures can cause diarrhea, urinary frequency, pyuria, or microscopic hematuria.
 - b. **When the appendix is located in the pelvis**, it may simulate acute gastroenteritis with diffuse pain, nausea, vomiting, and diarrhea. The diagnosis may be suspected if tenderness is present on digital rectal examination.
 - c. **Appendicitis is the most common nongynecologic surgical emergency during pregnancy.** The incidence of appendicitis during pregnancy is 1:1,500–2,000 per pregnancy. It occurs in the pregnant and the nonpregnant patient with equal frequency. Appendicitis is evenly distributed throughout the trimesters, but the incidence of perforation is highest in the third trimester.
 - i. **Appendicitis must be suspected in any pregnant woman with abdominal pain.** The gravid uterus displaces the appendix superiorly and laterally toward the RUQ ([Fig. 15-2](#)), thereby complicating the diagnosis of appendicitis. Separation of the visceral and parietal peritoneum due to the enlarging uterus limits localization of the pain by decreasing the somatic component of the pain. In addition, nausea and vomiting can be incorrectly attributed to the morning sickness that is common in the first trimester.
 - ii. **Operation is indicated in a pregnant patient as soon as the diagnosis of appendicitis is suspected.** Death of the fetus occurs in 1.5–3.0% of pregnant patients with uncomplicated appendicitis, but this figure can reach 35% when complicated by perforation and diffuse peritonitis. A negative laparotomy carries a risk of fetal loss of up to 3%.
 - b. **Laboratory evaluation.** Complete blood cell count, serum electrolytes, and urinalysis should be obtained preoperatively for patients with suspected appendicitis. A serum pregnancy test also must be performed for all ovulating women.
 1. **Complete blood cell count.** A leukocyte count of greater than 10,000 cells/μL with polymorphonuclear cell predominance (>75%) is common in the child and young adult with appendicitis. The total number of WBCs and the proportion of immature forms increase if there is appendiceal perforation. In older adults, the leukocyte count and differential are normal more frequently than in younger adults, as high as 10%. Pregnant women normally have an elevated WBC count that can reach 15,000–20,000 as their pregnancy progresses.
 2. **Urinalysis** is abnormal in 25–40% of patients with appendicitis. Pyuria, albuminuria, and hematuria are common. Large quantities of bacteria suggest UTI as the cause of abdominal pain. If the urinalysis shows more than 20 WBCs per high-power field or more than 30 RBCs per high-power field, it suggests UTI. Significant hematuria should prompt consideration of ureteral stones.
 3. **Serum electrolytes, blood urea nitrogen, and serum creatinine** are obtained to identify and correct electrolyte abnormalities caused by dehydration secondary to vomiting or poor oral intake.
 - c. **Radiologic evaluation.** Diagnosis of appendicitis usually can be made without radiologic evaluation. In complex cases, however, the following imaging can be helpful.
 1. **Abdominal X-rays are not helpful in diagnosing appendicitis.** One study demonstrated an appendicolith on only 1.14% of the plain films performed on patients with surgically proved appendicitis. Other suggestive radiologic findings include a distended cecum with adjacent small-bowel air-fluid levels, loss of the right psoas shadow, scoliosis to the right, and gas in the lumen of the appendix. A perforated appendix rarely causes a pneumoperitoneum.
 2. **US** is most useful in women of childbearing age and in children because other causes of abdominal complaints can be demonstrated. Findings associated with acute appendicitis include an appendiceal diameter of greater than 6 mm, lack of luminal compressibility, and presence of an appendicolith. The perforated appendix is more difficult to diagnose and is characterized by loss of the echogenic submucosa and the presence of a

Fig. 15-2. Changes in location and direction of the appendix during pregnancy. The normal and postpartum position of the base of the appendix is medial to McBurney's point. At the fifth month, the appendix is at the level of the umbilicus and iliac crest. (Adapted from Baer JL, et al. *JAMA* 1932;98:1359.)

loculated periappendiceal or pelvic fluid collection. The quality and accuracy of US are highly operator dependent. In women, ovarian pathology may be identified or excluded.

3. **Abdominal CT scan** is generally performed only in complex cases or in patients with atypical presentations. It is superior to US in these cases (*Am J Surg* 179:379, 2000). CT findings of appendicitis include a distended thick-walled appendix with inflammatory streaking of surrounding fat, a pericecal phlegmon or abscess, an appendicolith, or RLQ intraabdominal free air that signals perforation. CT scan is particularly useful in distinguishing between periappendiceal abscesses and phlegmon.
 4. **Barium enema (BE)** is rarely used to diagnose acute appendicitis. It may establish luminal patency of the appendix and may show mass effects on the colonic wall characteristic of appendicitis. If barium contrast completely fills the appendix, appendicitis is unlikely (10–20% of normal appendixes do not fill). BE may be helpful in differentiating right colonic or terminal ileal mucosal disease that may simulate appendicitis, such as inflammatory bowel disease. However, this test must be avoided in patients with toxic colitis.
 - d. **Diagnostic laparoscopy** is most useful among ovulating women with an equivocal examination for appendicitis. In this subgroup, one-third of women prove to have primary gynecologic pathology. The appendix may also be removed via the laparoscopic approach. Therefore, some surgeons advocate an initial laparoscopic approach in all ovulating women with suspected appendicitis.
3. **Differential diagnosis**
- a. **Gastrointestinal diseases**
 1. **Gastroenteritis** is characterized by nausea and emesis before the onset of abdominal pain, along with generalized malaise, high fever, diarrhea, and poorly localized abdominal pain and tenderness. Although diarrhea is one of the cardinal signs of gastroenteritis, it can occur in patients with appendicitis. Diarrhea is most common in young children. Additionally, the WBC count is less frequently elevated ([Table 15-1](#)) in patients with gastroenteritis.

Table 15-1. Factors differentiating appendicitis from nonsurgical illness

2. **Mesenteric lymphadenitis** usually occurs in patients younger than 20 years old and presents with middle, followed by RLQ, abdominal pain, but without rebound tenderness or muscular rigidity. Nodal histology and cultures obtained at operation can identify etiology, most notably *Yersinia* species, *Shigella* species, and *Mycobacterium tuberculosis*. Mesenteric lymphadenitis is known to be associated with upper respiratory tract infections.
 3. **Meckel's diverticulitis** presents with symptoms and signs indistinguishable from those of appendicitis, but it characteristically occurs in infants.
 4. **Perforated peptic ulcer disease, diverticulitis, and cholecystitis** can present clinical pictures similar to those of appendicitis.
 5. **Typhlitis** is characterized by inflammation of the wall of the cecum or terminal ileum. It is most commonly seen in immunosuppressed children who are being treated with chemotherapy for leukemia and can also occur in HIV-positive patients. Although management of typhlitis is usually nonsurgical, it is difficult to distinguish preoperatively between typhlitis and appendicitis in susceptible children.
- b. **Urologic diseases**
 1. **Pyelonephritis** causes high fevers, rigors and costovertebral pain, and tenderness. Diagnosis is confirmed by urinalysis with culture.
 2. **Ureteral colic.** Passage of renal stones causes flank pain radiating into the groin but little localized tenderness. Hematuria suggests the diagnosis, which is confirmed by intravenous pyelography or noncontrast CT. Abdominal plain films frequently show renal stones.
 - c. **Gynecologic diseases**
 1. **PID** can present with symptoms and signs indistinguishable from those of acute appendicitis, but the two often can be differentiated on the basis of several factors ([Table 15-2](#)). Cervical motion tenderness and milky vaginal discharge strengthen a diagnosis of PID. In patients with PID, the pain is usually bilateral with intense guarding on abdominal and pelvic examinations. Transvaginal US can be used to visualize the ovaries and to identify tubo-ovarian abscesses.

Table 15-2. Factors differentiating pelvic inflammatory disease from appendicitis

2. **Ectopic pregnancy.** A pregnancy test should be performed in all female patients of childbearing age presenting with abdominal complaints.
 3. **Ovarian cysts** are best detected by transvaginal or transabdominal US.
 4. **Ovarian torsion.** The inflammation surrounding an ischemic ovary often can be palpated on bimanual pelvic examination. These patients can have a fever, leukocytosis, and RLQ pain consistent with appendicitis. A twisted viscus, however, differs in that it produces sudden acute intense pain with simultaneous frequent and persistent emesis. This diagnosis can also be confirmed on US.
4. **Treatment**
- a. **Preoperative preparation.** Intravenous isotonic fluid replacement should be initiated to achieve a brisk urinary output and to correct electrolyte abnormalities. Nasogastric suction is helpful, especially in patients with peritonitis. Temperature elevations are treated with acetaminophen and a cooling blanket. **Anesthesia should not be induced in patients with a temperature higher than 39°C.** Preoperative antibiotic coverage is begun.
 - b. **Antibiotic therapy.** Broad-spectrum antibiotic coverage is initiated preoperatively to assist in controlling local and general sepsis and to reduce the incidence of postoperative wound infection. For acute appendicitis, this typically consists of a second-generation cephalosporin (cefotetan or cefoxitin). In patients with acute nonperforated appendicitis, therapy should not continue for more than 24 hours. Many studies have shown that a single preoperative dose is sufficient. Antibiotic therapy in perforated or gangrenous appendicitis should be continued for 3–5 days.
 - c. **Appendectomy.** With very few exceptions, the treatment of appendicitis is appendectomy. Patients with diffuse peritonitis or questionable diagnosis should be explored through a midline incision. The mortality after appendectomy is high in elderly patients. Equivocal diagnosis of appendicitis in this frail patient population warrants increased diagnostic effects before emergent appendectomy (*Ann Surg* 233:4, 2001). In most patients, a transverse incision (e.g., Davis-Rockey, Fowler-Weir) provides the best cosmetic appearance and allows easy extension medially for greater exposure ([Fig. 15-3](#)). The external and internal oblique and transversus abdominis muscle layers may be split in the direction of their fibers. After entering the peritoneal cavity, Gram stain and cultures are obtained if purulence is present. Once the cecum is identified, the anterior taenia can be followed to the base of the appendix. The appendix is gently delivered into the wound, with any surrounding adhesions broken carefully. If the appendix is normal on inspection (occurring in 5–20% of explorations), it is removed, and appropriate alternative diagnoses are entertained. The cecum, sigmoid colon, and ileum are carefully inspected for changes indicative of diverticular (including Meckel's diverticulum), infectious, ischemic, or inflammatory bowel disease (e.g., Crohn's). Evidence of mesenteric lymphadenopathy is sought. In women, the ovaries and fallopian tubes are inspected for evidence of PID, ruptured follicular cysts, ectopic pregnancy, or other pathology. Bilious peritoneal fluid suggests perforation either of a peptic ulcer or the gallbladder.

Fig. 15-3. Incisions for appendectomy include the gridiron incision (McBurney), transverse incision (Davis-Rockey, Fowler-Weir), and midline incision. (Adapted from Sabiston DC Jr, ed. *Atlas of general surgery*. Philadelphia: WB Saunders, 1994.)

- d. **Laparoscopic appendectomy.** Laparoscopic management of appendicitis has evolved from a purely diagnostic to a therapeutic modality. The surgical literature to date shows little or no patient benefit over the traditional open procedure. Laparoscopic appendectomy is more costly and offers no decrease in the length of stay or complications, with a slight decrease in wound infection but a slight increase in intraabdominal infection (*Am J Surg* 179:375, 2000).
- e. **Drainage of appendiceal abscess.** Management of appendiceal abscesses remains controversial. Patients initially seen when symptoms are subsiding with a well-localized periappendiceal abscess can be treated with systemic antibiotics and considered for percutaneous US- or CT-guided catheter drainage, followed by elective appendectomy 6–12 weeks later (*Radiology* 163:23, 1987). This strategy is successful in more than 80% of patients. The appendix must be removed because the patient has a 60% risk of developing appendicitis again within 2 years. Alternatively, immediate appendectomy

can shorten the duration of the illness and is as safe as catheter drainage and delayed appendectomy. Anatomically obscure or multiloculated appendiceal abscesses should be cultured and evacuated surgically. Appendectomy should be performed at this initial operation, with the wound left open. Systemic antibiotics are administered for at least 5 days or until the leukocyte count and temperature have returned to normal.

f. **Incidental appendectomy** is removal of the normal appendix at laparotomy for another condition. Although one study showed an increased risk of wound infection in patients older than 50 years of age undergoing routine cholecystectomy with incidental appendectomy, most studies demonstrate no evidence of adverse consequences after incidental appendectomy. The appendix must be easily accessible through the present abdominal incision, and the patient must be clinically stable enough to tolerate the extra time needed to complete the procedure. Because most cases of appendicitis occur in a patient's teens and 20s, the benefit of incidental appendectomy decreases substantially once a person is older than 30 years of age. Crohn's disease involving the cecum, radiation treatment to the cecum, immunosuppression, and vascular grafts or other bioprostheses in place are contraindications for incidental appendectomy because of the increased risk of infectious complications or appendiceal stump leak.

5. Complications of acute appendicitis

- Perforation** is accompanied by severe pain and fever. It is unusual within the first 12 hours of appendicitis but is present in 50% of patients younger than age 10 years and older than age 50 years. Acute consequences of perforation include fever, tachycardia, generalized peritonitis, and abscess formation. Treatment is appendectomy, peritoneal irrigation, and broad-spectrum intravenous antibiotics for several days.
- Postoperative wound infection risk** can be decreased by appropriate intravenous antibiotics administered before skin incision. The incidence of wound infection increases from approximately 3% in cases of nonperforated appendicitis to 4.7% in patients with a perforated or gangrenous appendix. Primary closure in the setting of perforation is unchanged from delayed closure (4.6%) and is not recommended (*Surgery* 127:136, 2000). Wound infections are managed by opening, draining, and packing the wound to allow healing by secondary intention. Intravenous antibiotics are indicated for associated cellulitis or systemic sepsis.
- Intraabdominal and pelvic abscesses** occur most frequently with perforation of the appendix. Postoperative intraabdominal and pelvic abscesses are best treated by percutaneous CT- or US-guided aspiration. If the abscess is inaccessible or resistant to percutaneous drainage, operative drainage is indicated. Antibiotic therapy can mask but does not treat or prevent a significant abscess.
- Other complications**
 - Pyelophlebitis** is septic portal vein thrombosis, usually is caused by *Escherichia coli*, and presents with high fevers, jaundice, and eventually hepatic abscesses. CT scan demonstrates thrombus and gas in the portal vein. Prompt treatment (operative or percutaneous) of the primary infection is critical, followed by broad-spectrum intravenous antibiotics.
 - Enterocutaneous fistulas** from a leak at the appendiceal stump closure occasionally require surgical closure, but most close spontaneously.
 - SBO** is four times more common after surgery in cases of perforated appendicitis than in uncomplicated appendicitis.

B. Acute cholecystitis

- Most patients provide an **antecedent history of biliary colic** (i.e., epigastric or RUQ pain after consumption of a fatty meal).
- Typical presentation** is epigastric or RUQ pain, nausea, and vomiting 4–6 hours after consumption of a meal.
- Examination** is characterized by fever, RUQ tenderness, and a positive Murphy's sign.
- Laboratory examination** reveals leukocytosis (with left shift) and a slight elevation of liver function tests (not always present).
- Helpful **radiographic studies** include US (may show the presence of gallstones, gallbladder wall thickening, pericholecystic fluid, and sonographic Murphy's sign) and disopropyliminodiacetic acid scan (shows nonfilling of the gallbladder).
- It is important to consider the full spectrum of biliary tract disease.**

C. Acute pancreatitis

- The most common cause is alcohol consumption**, followed by gallstones; other causes include post–endoscopic retrograde cholangiopancreatography, certain medications, and hypertriglyceridemia.
- Typically, it presents as **severe epigastric pain** that radiates to the patient's back.
- Examination** is characterized by epigastric tenderness with varying degrees of tachycardia, fever, and hypotension, depending on the severity of the attack.
- The **spectrum of severity** ranges from mild edema around the pancreas to pancreatic necrosis with infection.
- The severity of the attack and the prognosis may be estimated by various indicators (**Ranson's criteria**).
- Laboratory examination** is characterized by elevation of amylase, lipase, and serum transaminases, none of which correlate with severity.
- Plain film** may reveal sentinel loop or pancreatic calcifications; CT scan with intravenous contrast is indicated in severe cases to determine the presence of pancreatic necrosis or fluid collection (optimal results are obtained after 3 days).

D. Perforated peptic ulcer

- Duodenal ulcers** are more common than gastric ulcers.
- Is associated with the chronic use of **nonsteroidal antiinflammatory medications**.
- Most patients provide a **history compatible with peptic ulcer disease**.
- Typically **presents as sudden onset of severe epigastric pain** that eventually involves the patient's entire abdomen.
- Physical examination** is remarkable for diffuse abdominal tenderness, rigidity, and peritoneal signs.
- Plain films** usually, but not always, reveal free intraperitoneal air.
- Therapy** consists of fluid resuscitation, intravenous antibiotics, and emergent surgical exploration.

E. Intestinal obstruction

- SBO**
 - The most common cause is **adhesions from previous surgery**; others include hernias, cancer, intussusception, and volvulus.
 - It usually presents as sharp, crampy periumbilical pain** with intervening pain-free periods; associated symptoms include nausea and vomiting.
 - Examination** is marked by abdominal distention, high-pitched or tinkling bowel sounds, and a variable degree of abdominal tenderness.
 - Plain abdominal films** reveal dilated loops of small bowel, air-fluid levels, and paucity of gas in the colon. Proximal SBO, however, may not be associated with dilated bowel loops on plain films and often requires a contrast study for diagnosis.
- Large-bowel obstruction**
 - It may be caused by cancer, diverticulitis, volvulus, or impaction.**
 - Presenting symptoms** include constipation, abdominal distention, and varying degrees of abdominal pain.
 - Examination** may reveal abdominal distention or a mass on rectal examination.
 - Plain abdominal films** may reveal colonic distention. An enema with opaque contrast (e.g., Hypaque) is often necessary to rule out the presence of a mass (e.g., obstructing colon, rectal cancer).
 - The **risk of perforation** increases as the cecal diameter exceeds 12–13 cm.

F. Mesenteric ischemia

- It may result from superior mesenteric artery thrombosis from severe vascular disease or from superior mesenteric artery occlusion by embolus** (e.g., atrial fibrillation).
- It presents as sudden onset of severe, constant abdominal pain with associated emptying of bowel contents** (vomiting and diarrhea).
- Examination** may reveal **pain out of proportion** to physical findings.
- Laboratory studies** are marked by leukocytosis and acidosis (secondary to accumulation of lactate).
- Angiography** may confirm the diagnosis; however, radiologic studies are not indicated if the patient is found to have peritonitis on physical examination.

G. Ruptured AAA

- It presents as sudden onset of abdominal pain** with varying degrees of radiation to the patient's flank or back, or both.
- Patients with free intraabdominal rupture** rarely, if ever, survive until hospital arrival; those with contained rupture or leak may present in shock.
- Examination** is marked by the presence of a tender, pulsatile abdominal mass.
- Plain films** may reveal calcification in the aortic wall; CT scan is the gold standard for diagnosis (only performed in hemodynamically stable patients).
- Patients with hypotension from a known aneurysm** should be taken emergently to the operating room without further diagnostic studies. Anesthesia induction should be delayed until the patient is prepped and draped to avoid intubation-induced hypotension.

II. Surgical options

- Exploratory laparotomy** is mandatory in any patient in whom a surgically correctable cause of acute abdomen is suspected or diagnosed.
 - Diffuse peritonitis** (e.g., from a perforated viscus)
 - Acute, high-grade intestinal obstruction**
 - Ischemic or necrotic bowel**
- Laparoscopy** may be used as a diagnostic and a therapeutic tool in patients with a variety of acute abdominal conditions. It has the advantage of enabling a more thorough examination of the peritoneal cavity than is obtainable through a small (e.g., RLQ) surgical incision. Laparoscopy is not as useful in the visualization of retroperitoneal structures. Settings in which laparoscopy may be particularly beneficial include
 - Suspected but equivocal appendicitis** (e.g., RLQ pain in a young woman, in whom gynecologic pathology often confounds the diagnosis); laparoscopy

may be diagnostic and therapeutic because laparoscopic appendectomy can usually be carried out in patients with uncomplicated appendicitis.
2. **Critically ill patients** in whom a negative laparotomy may cause great morbidity.

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The Washington Manual of Surgery

16 Colon, Rectum, and Anus

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Disturbances of Physiology

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Disturbances of Physiology

I. Normal colon function

- A. **Water absorption.** Normal ileal effluent totals 900–1,500 mL per day, and stool water loss amounts to only 100–200 mL per day. Under maximum conditions, the colon can absorb 5–6 L of fluid a day, and only when large-bowel absorption is less than 2 L per day does an increase in fecal water content result in diarrhea. The majority of colonic absorption takes place in the right colon.
- B. **Electrolyte transport.** Sodium and chloride absorption occur by active processes in exchange for potassium and bicarbonate. Although absorption of nutrients is minimal in the colon, passive absorption of short-chain fatty acids produced by colonic bacteria can account for up to 540 kcal per day and provide much of the energy needed for electrolyte transport in normal colons.
- C. **Motility patterns of the colon** allow for mixing of intestinal content and for elimination and, in part, are due to the balance of excitatory parasympathetic input and inhibitory sympathetic input. A patient's emotional state, amount of exercise and sleep, amount of colonic distention, and hormonal milieu may all affect the three patterns of colonic motility.
 1. **Retrograde movements** occur mainly in the right colon. These contractions prolong the exposure of luminal contents to the mucosa and thereby increase the absorption of fluids and electrolytes.
 2. **Segmental contractions**, the most commonly observed motility pattern, represent localized simultaneous contractions of the longitudinal and circular colonic musculature in short colonic segments.
 3. **Mass movements** occur three to four times a day and are characterized by an antegrade, propulsive contractile wave involving a long segment of colon.
- D. **Microflora.** One-third of the dry weight of feces is normally composed of bacteria. Anaerobic *Bacteroides* species are most prevalent (10^{11} /mL), although *Escherichia coli* has a titer of 10^9 /mL. Bacteria produce much of the body's vitamin K. Endogenous colonic bacteria also suppress the emergence of pathogenic microorganisms. Antibiotic therapy can alter the endogenous microflora, resulting in changes in drug sensitivity (warfarin) or infectious colitides due to pathogenic microbial overgrowth (*Clostridium difficile* colitis).
- E. **Colonic gas** (200–2,000 mL per day) is composed of (1) swallowed oxygen and nitrogen; and (2) hydrogen, carbon dioxide, and methane produced during fermentation by colonic bacteria. Hydrogen and methane are combustible gases and may explode when electrocautery is used for polypectomy or biopsy. Adequate bowel cleansing (mannitol is not an appropriate agent in this circumstance because it is a substrate for bacterial fermentation) is therefore necessary before the use of intracolonic electrocautery.

II. Disorders of colonic physiology

- A. **Constipation** is difficult to define and is often used by patients to describe a number of different defecatory symptoms (infrequent bowel movements, difficult or painful movements, etc.). Constipation is generally defined clinically as one or fewer stools per week.
 1. **Etiologies** include medications (narcotics, anticholinergics, antidepressants), hypothyroidism, hypercalcemia, dietary factors (low fluid intake, high cheese intake), decreased exercise, and neurologic disorders (e.g., Parkinson's disease and multiple sclerosis). Abnormalities of pelvic floor function (obstructed defecation), such as paradoxical puborectalis muscle function or intussusception of the rectum (internal or external rectal prolapse), may result in constipation, as can idiopathic delayed transit of feces through the colon (colonic inertia).
 2. **Change in bowel habits** is a common presentation of colorectal neoplasia and should initially be evaluated with colonoscopy or proctoscopy with barium enema. If this is negative and the patient fails to respond to a trial of fiber supplementation and increased fluid intake, a colon transit study is indicated. On day 0, the patient ingests a capsule containing indigestible, radiopaque rings, and abdominal plain X-rays are obtained on day 3 and day 5. Persistence of the rings in the proximal colon on both films indicates delayed colonic transit. When the rings persist in the rectosigmoid region, functional anorectal obstruction (obstructed defecation) may be present, often from a pelvic floor abnormality. This may be evaluated with cine defecography or anorectal manometry, or both.
 3. **Treatment** of colonic inertia initially is conservative and includes **laxatives, stool softeners, increased exercise, and avoidance of predisposing factors**. If these prove unsuccessful and obstructed defecation has been ruled out, the patient may benefit from surgical therapy. In patients with long-standing, debilitating symptoms refractory to nonoperative measures, **total abdominal colectomy with ileosigmoid or ileorectal anastomosis** may prove curative.
- B. **Colonic pseudo-obstruction** (Ogilvie's syndrome), a profound colonic ileus without evidence of mechanical obstruction, is a diagnosis of exclusion. It most commonly occurs in critically ill or institutionalized patients. Initial treatment after confirmation of the absence of a fixed mechanical obstruction is conservative. Nasogastric decompression, rectal tube placement, an aggressive enema regimen (e.g., cottonseed and docusate sodium enema), correction of metabolic disorders, and discontinuation of medications that decrease colonic motility are the cornerstones of initial management. Rapid cecal dilation or a cecal diameter of 9–12 cm on plain abdominal X-rays requires prompt colonoscopic decompression. This is initially successful in 70–90% of cases, with a recurrence rate of 10–30% (which is usually also amenable to colonoscopic decompression). Laparotomy should be performed in patients with peritonitis, and nonviable bowel should be resected often with proximal fecal diversion.
- C. **Volvulus** is the twisting of an air-filled segment of bowel about its mesentery and accounts for nearly 10% of bowel obstruction in the United States.
 1. **Sigmoid volvulus** accounts for 80–90% of all volvulus and is most common in elderly or institutionalized patients and in patients with a variety of neurologic disorders. It is an acquired condition resulting from sigmoid redundancy with narrowing of the mesenteric pedicle.
 - a. **Diagnosis** is suspected when there is abdominal pain, distention, cramping, and obstipation. **Plain films** often show a characteristic **inverted-U**, sausage-like shape of air-filled sigmoid. If the diagnosis is still in question and gangrene is not suspected, water-soluble **contrast enema** usually shows a **bird's beak deformity** at the obstructed rectosigmoid junction and can be therapeutic.
 - b. In the absence of peritoneal signs, **treatment** involves **sigmoidoscopy** with the placement of a rectal tube beyond the point of obstruction. If a sigmoidoscope does not reach the obstruction, colonoscopic decompression may be useful. If **peritonitis** is present, the patient should undergo laparotomy and **Hartmann's procedure** (sigmoid colectomy, end-descending colostomy, and defunctionalized rectal pouch). An alternative in the stable patient without significant fecal soilage of the peritoneal cavity is sigmoidectomy, on-table colonic lavage, and colorectal anastomosis with or without proximal fecal diversion (loop ileostomy).
 - c. **Recurrence rate** after sigmoidoscopic decompression is approximately 40% once the rectal tube is removed. Therefore, if the patient's medical condition allows, an elective sigmoid colectomy with primary anastomosis should be performed after mechanical bowel preparation.
 2. **Cecal volvulus** occurs in a younger population than does sigmoid volvulus and is thought to occur secondary to congenital failure of retroperitonealization of the cecum.
 - a. **Diagnosis.** Presentation is similar to that of distal small-bowel obstruction, with nausea, vomiting, abdominal pain, and distention. Plain films show a

kidney-shaped air-filled cecum with the convex aspect extending into the left upper quadrant. A **Hypaque enema** may be performed, which shows a tapered cutoff in the ascending colon.

- b. **Management** involves urgent laparotomy and right hemicolectomy. Ileocolostomy is preferred; otherwise, ileostomy and mucous fistula are performed if concerns of patient stability or bowel viability exist. Cecopexy has also been advocated. This involves elevation of a retroperitoneal flap and suture of the cecum to this flap to eliminate the pathologic condition predisposing the patient to cecal volvulus. This technique is often a formidable undertaking in the setting of the acutely dilated and fragile cecum.
3. **Transverse volvulus** is rare and has a similar clinical presentation to sigmoid volvulus. Diagnosis is made based on the results of plain film and Hypaque enema. Endoscopic decompression has been reported, but operative resection is usually required.

D. Diverticular disease

1. **General considerations.** Colonic diverticula are **false diverticula** in which mucosa and submucosa protrude through the muscularis propria. This occurs at the mesenteric side of the antimesenteric taenia, where arterioles penetrate the muscularis. The **sigmoid colon** is most commonly affected, perhaps owing to decreased luminal diameter and increased luminal pressure. Diverticula are associated with a low-fiber diet and are rare before age 30 years (<2%), but the **incidence increases with age** to a 75% prevalence after age 80.

2. Complications

- a. **Infection (diverticulitis).** Microperforations can develop in long-standing diverticula, leading to fecal extravasation and subsequent peridiverticulitis.
 1. **Presentation** is notable for left lower quadrant pain (which may radiate to the suprapubic area, left groin, or back), fever, altered bowel habit, and urinary urgency. Physical examination varies with severity of the disease, but the most common finding is localized left lower quadrant tenderness. The finding of a mass suggests an abscess or phlegmon.
 2. **Computed tomographic (CT) scan** is the diagnostic modality of choice. It visualizes thickened bowel wall as well as abscess formation, which may be drained percutaneously under CT guidance. Ultrasonography can also yield the diagnosis. Neither sigmoidoscopy nor contrast enema is recommended in the initial workup of diverticulitis, owing to the risk of perforation or barium or fecal peritonitis, respectively.
 3. **Treatment** is tailored to symptom severity.
 - a. **Mild diverticulitis** can be treated on an outpatient basis with a clear liquid diet and broad-spectrum oral antibiotics for 7–10 days.
 - b. **Severe diverticulitis** is treated on an inpatient basis, with complete bowel rest, intravenous fluids, narcotic analgesia using meperidine, and broad-spectrum parenteral antibiotics. If symptoms improve within 48 hours, a high-fiber diet is resumed, and antibiotics are given orally when the fever and leukocytosis resolve. Colonoscopy or water-soluble contrast study must be performed after 4–6 weeks to rule out colon cancer, inflammatory bowel disease, or ischemia as a cause of the segmental inflammatory mass. If resection is planned after a particularly severe initial attack of diverticulitis (abscess requiring drainage, fistula formation) or recurrent diverticulitis, clear liquids and parenteral nutritional supplementation are used for 2–3 weeks until operation to allow for a one-stage procedure.
 4. The lifetime likelihood of **recurrence** is 30% after the first episode and more than 50% after the second episode of diverticulitis. Therefore, **resection** is considered 4–6 weeks after treatment of a severe initial attack of diverticulitis or after treatment of the first recurrence if the condition of the patient permits.
 5. **Elective resection** for diverticulitis usually consists of a sigmoid colectomy. The proximal resection margin is through uninfamed bowel, but there is no need to resect all diverticula in the colon. The distal margin extends distal to normal, pliable rectum, even if this means dissection beyond the peritoneal reflection. Recurrent diverticulitis after resection is most frequently related to inadequate distal margin of resection.
- b. **Generalized peritonitis** is rare and results if diverticular perforation leads to widespread fecal contamination. Patients present with diffuse severe abdominal pain with voluntary and involuntary guarding; they require emergent laparotomy. In most cases, resection of the diseased segment is possible (**two-stage procedure**), and a Hartmann's procedure is performed. The colostomy can then be taken down at a separate operation in the future. Colostomy closure is not a trivial procedure, with rates of morbidity of 20–30% (bleeding, anastomotic leak, abscess) and a mortality of 2–3%. An alternative in the management of the stable patient undergoing urgent operation for acute diverticulitis without significant fecal contamination is sigmoidectomy, on-table colonic lavage, and colorectal anastomosis with or without proximal fecal diversion (loop ileostomy).
- c. **Diverticular abscess** is usually identified on CT scan. A **percutaneous drain** should be placed under **CT guidance**. This avoids immediate operative drainage, allows time for the inflammatory phlegmon to be treated with intravenous antibiotics, and turns a two-stage or three-stage procedure into a one-stage procedure. A low pelvic abscess may be drained into the rectum via a transanal approach.
- d. **Fistula** may occur between the colon and other organs, including the bladder, vagina, small intestine, and skin, owing to diverticulitis. Diverticulitis is the most common cause of enterovesical fistulas, which present more commonly in men, secondary to the interposition of the uterus in women. Colovaginal and colovesical fistulas usually occur in women having previously undergone hysterectomy. Colocutaneous fistulas are uncommon and are usually easy to identify. Coloenteric fistulas are likewise uncommon and may be entirely asymptomatic or result in corrosive diarrhea.
 1. **Presentation of enterovesical fistula** involves frequent **urinary tract infections** and often is unsuspected until **fecaluria** or **pneumaturia** is noted. Air in the uninstrumented bladder on CT scan suggests the diagnosis. Lower endoscopy, barium enema, intravenous pyelography, and cystoscopy often fail to demonstrate the fistula. A colovaginal fistula is usually suspected based on the passage of air or gas per vagina. The fistula may be difficult to identify on physical examination or the previously mentioned tests. Both fistulas may be identified by the ingestion of charcoal and the presence of charcoal in the urine or on a tampon inserted into the vagina.
 2. **Immediate treatment** of the inflammatory mass adjacent to the bladder is as previously described for severe diverticulitis. Colonoscopy is performed after 4–6 weeks to rule out other possible etiologies, including cancer or inflammatory bowel disease. After several weeks of antibiotics, sigmoid resection is performed after preoperative placement of temporary ureteral catheters. Ureteral catheters can be very helpful in identifying the distal ureter in the inflammatory pericolonic mass, thereby shortening the operative time, and in identifying ureteral injuries. Usually, the fistula tract can be broken using finger fracture, and the bladder defect does not typically require repair. A Foley catheter is left in place for 7–10 days to allow this defect to heal. A colovaginal fistula is managed in a similar fashion. It may be helpful to interpose omentum between the colorectal anastomosis and the bladder or vaginal defect.

E. **Acquired vascular abnormalities** are more common in elderly patients than in younger individuals. Most cases of massive lower gastrointestinal (GI) hemorrhage stop spontaneously, but surgery is required in 10–25% of cases.

1. **Diverticulosis.** The media of the perforating artery adjacent to which the colonic diverticula protrude may become attenuated. If this erodes at the neck or dome of the diverticulum, massive intraluminal bleeding can result. This arterial bleeding usually is bright red and is not associated with previous melena or chronic blood loss. Bleeding generally occurs from the proximal colon. Urgent resection of the affected colonic segment should be considered in patients with active ongoing bleeding (>6 units packed RBCs per 24 hours). Elective resection of the affected colonic segment should be performed in patients with recurrent bleeding or in those in whom recurrent bleeding may be poorly tolerated.
2. **Angiodysplasias** are small arteriovenous fistulas resulting from mucosal venule dilation followed by precapillary sphincter incompetence. They rarely occur before age 40 years and are more common (80%) in the right colon. Diagnosis can be made by colonoscopy or angiographic features (early filling of a dilated venule).
3. **Massive lower GI bleeding** is defined as hemorrhage distal to the ligament of Treitz that requires more than 3 units of blood in 24 hours. **Management** consists of simultaneously restoring intravascular volume and identifying the site of bleeding so that treatment may be instituted.
 - a. **Resuscitation** is performed using a combination of isotonic crystalloid solutions and packed RBCs as needed.
 - b. **Diagnosing the site of bleeding** is more important initially than identifying the cause. A nasogastric tube may be placed to rule out an upper GI source of bleeding. Return of bilious fluid effectively eliminates an upper GI source. Proctoscopy is helpful to rule out anorectal causes of bleeding. The choice of localizing study depends on the estimate of bleeding rate.
 1. **Nuclear scan** using technetium-99m sulfur colloid or tagged RBCs can identify bleeding sources with rates in excess of 0.5–1.0 mL per minute. Tagged RBC scan can identify bleeding up to 24 hours after isotope injection, which may be important in patients who bleed intermittently. Approximately 50% of patients do not have their bleeding site identified until more than 6 hours after injection. Although these scans can accurately demonstrate ongoing bleeding, they do not definitively identify the anatomic source of bleeding; hence, planning a segmental gastrointestinal resection based on this study is not entirely reliable.
 2. **Mesenteric angiography** should be performed in the patient with a positive nuclear medicine bleeding scan with massive lower GI bleeding to identify definitively the anatomic source of bleeding. Angiography can localize bleeding exceeding 1.0 mL per minute and has the benefit of either therapeutic vasopressin infusion (0.2 units per minute) or embolization, which together are successful in stopping the bleeding in 85% of cases. This is advantageous in converting an emergent operation in an unstable patient with unprepared bowel to an elective one-stage procedure. Provocative angiography may also be useful in the patient with recurrent bleeding without a localized source. It is performed by administration of heparin to induce bleeding followed by mesenteric angiography to localize the source. It is an infrequently performed procedure in selected patients with recurrent massive lower GI bleeding.
 3. **Colonoscopy** is not effective in high rates of bleeding because of poor visualization and higher complication rates. With slower bleeding after the administration of an adequate bowel preparation over 2 hours, colonoscopy offers the therapeutic advantages of injecting vasoconstrictive agents (epinephrine), vasodestructive agents (alcohol, morrhuate, sodium tetradecyl sulfate), or thermal therapy (laser photocoagulation, electrocoagulation, heater probe coagulation) to control bleeding.
 4. In the rare patients who continue to bleed with no source identified, **laparotomy** should be considered. Intraoperative small-bowel enteroscopy may

be performed if a source is not obvious at the time of exploration. If a source is still not identified, **total colectomy with ileorectostomy or end ileostomy** is performed. This is associated with an incidence of recurrent bleeding of less than 10%, but the mortality of patients who rebleed is 20–40%. It is very uncommon for a patient to undergo resection for lower GI bleeding without a localized source.

4. **Ischemic colitis** results from many causes, including thrombosis, embolization, iatrogenic ligation after abdominal aortic aneurysm repair, thromboangiitis obliterans, and polyarteritis nodosa. It is idiopathic in the majority of patients. Patients are usually elderly and present with low abdominal pain localizing to the left and melena or hematochezia. The rectum often is normal on proctoscopy, owing to its dual vascular supply. Contrast enema may show thumb printing that corresponds to submucosal hemorrhage and edema. Diagnosis depends on colonoscopy and biopsy. Disease is present most frequently at the watershed area of the splenic flexure (although it may occur anywhere in the colon). Once the diagnosis is made, bowel rest is instituted. In the presence of bowel necrosis or peritonitis, emergent resection is performed, usually with proximal fecal diversion. If these are not present but there is fever or an elevated white blood cell count, observation and intravenous antibiotics are used. Observation alone with repeat colonoscopy after 2–5 days can be sufficient in the absence of these findings. Long-term observation is required, as patients may develop focal colonic strictures, particularly if the ischemic episode was severe. The strictures typically present in the descending colon and must be differentiated from a neoplastic process. Segmental resection with anastomosis is curative.
5. **Radiation proctocolitis** results from pelvic irradiation for uterine, cervical, bladder, prostate, or rectal cancers. Risk factors include dose greater than 6,000 centigray (cGy), vascular disease, diabetes mellitus, hypertension, and advanced age. The early phase occurs within days to weeks; mucosal injury, edema, and ulceration develop, with associated nausea, vomiting, diarrhea, and tenesmus. The late phase occurs within weeks to years, is associated with tenesmus and hematochezia, and consists of arteriolitis and thrombosis with subsequent bowel thickening and fibrosis. Ulceration with bleeding, stricture, and fistula formation may occur. Treatment is often difficult and frustrating for both the patient and surgeon. Medical treatment may be successful in mild cases, using stool softeners, steroid enemas, and topical 5-aminosalicylic acid products. Application of 4% formalin to affected mucosa may be efficacious if these measures fail in patients with transfusion-dependent rectal bleeding. With stricture or fistula, thorough evaluation is required to rule out locally recurrent disease. Strictures may be treated by endoscopic dilation but often recur. Surgical treatment consists of a diverting colostomy and is reserved for medical failures, recurrent strictures, and fistulas. Proctectomy is rarely required and is usually associated with unacceptable morbidity and mortality.

III. Dysfunction of the anorectum

A. Normal anorectal function

1. The **rectum functions as a capacitance organ**, holding 650–1,200 mL, whereas daily stool output is 250–750 mL.
2. The **anal sphincter mechanism** allows defecation and continence. The internal sphincter (involuntary) accounts for 80% of resting pressure, whereas the external sphincter (voluntary) accounts for 20% of resting pressure and 100% of squeeze pressure. The internal and external sphincters are contracted at rest.
3. **Defecation** has four components: (1) mass movement of feces into the rectal vault; (2) rectal-anal inhibitory reflex, wherein distal rectal distention causes involuntary relaxation of the internal sphincter; (3) voluntary relaxation of the external sphincter mechanism; and (4) increased intraabdominal pressure.
4. **Continence** requires normal capacitance, normal sensation at the transition zone above the dentate line, puborectalis function for solid stool, external sphincter function for fine control, and internal sphincter function for resting pressure. The puborectalis maintains the anorectal angle, and contraction prevents solid-stool passage.

B. Incontinence is the inability to control elimination of rectal contents.

1. **Etiologies** include (1) **mechanical defects**, such as sphincter damage from obstetric trauma, treatment of abscess or fistula by fistulotomy through a significant portion of the external sphincter in the anterior quadrant, and scleroderma affecting the external sphincter; (2) **neurogenic defects**, including pudendal nerve injury due to birth trauma or lifelong straining and systemic neuropathies such as multiple sclerosis; and (3) **stool content–related causes** (e.g., diarrhea and radiation proctitis).
2. **Evaluation** includes visual and digital examination observing for gross tone or squeeze abnormalities. **Anal manometry** quantitatively measures parameters of anal function, including resting and squeeze pressure, sphincter length, and minimal sensory volume of the rectum. **Electromyography** can be used to determine innervation abnormalities but has been supplanted by a combination of anorectal manometry, pudendal nerve latency testing, and endoanal ultrasound.
3. **Treatment** depends on the type and severity of the defect. Minor **mechanical anal sphincter defects** are treated using dietary fiber to increase stool bulk and biofeedback. Major anal sphincter defects require anal sphincter reconstruction in which the external sphincter is circularized and the perineal body is reconstructed using the overlapping muscle repair. Pneumatic artificial anal sphincters may be used in patients without a reconstructible native anal sphincter. Transferred muscle with implantable pacemakers (dynamic graciloplasty) have been used in the past for this severe problem but have fallen out of favor. **Neurogenic defects** may be managed with biofeedback in mild cases. Severe denervations of an intact anal sphincter may be managed with sacral nerve stimulation.

C. Obstructed defecation (pelvic floor outlet obstruction) presents with symptoms of chronic constipation and straining with bowel movements.

1. **Physiologic evaluation** includes (1) **defecography** to evaluate maintenance of fixation of the posterior rectum to the sacrum as well as relaxation of the puborectalis, and (2) **colonic transit study** to distinguish colonic inertia from anorectal malfunction.
2. **Anal stenosis** is a rare cause of obstructed defecation and presents with frequent thin stools and bloating. The most common etiologies include scarring after anorectal surgery (rare), chronic laxative abuse, radiation, recurrent anal ulcer, Crohn's disease, and trauma. Initial treatment is anal dilation, although advanced cases are treated with advancement flaps of normal perianal skin.
3. **Pelvic floor abnormalities**
 - a. **Nonrelaxation of puborectalis** results in straining and incomplete evacuation. Diagnosis requires a normal colonic transit time and persistent puborectalis distortion on defecography. Biofeedback is the treatment of choice.
 - b. **Internal intussusception** (see [section III.D.1](#)).
 - c. **Problems associated with obstructed defecation** include **fecal impaction** and **stercoral ulcer** (mucosal ulceration due to pressure necrosis from impacted stool), which is treated with enemas, increased fiber, and stool softeners. **Descending perineum syndrome** occurs when chronic straining causes pudendal nerve stretch and subsequent neurogenic defect. **Rectocele** results from a weak, distorted rectovaginal septum that allows the anterior rectal wall to bulge into the vagina. Attempts at correction of any of these conditions without addressing the underlying pathology are doomed to failure.

D. Abnormal rectal fixation occurs to varying degrees, leading to internal or external prolapse of the full thickness of the rectum. Rectal mucosal prolapse is a prolapse of only rectal mucosa and is usually easily differentiated by physical examination.

1. **Internal intussusception (internal rectal prolapse)** presents with mucous discharge, hematochezia, tenesmus, and constipation. Proctoscopy is performed and can demonstrate a **solitary rectal ulcer** at the lead point of the internal prolapse. **Treatment** initially is increased bulk, stool softeners, and glycerin suppositories. **Indications for surgery** are chronic bleeding, impending incontinence, and lifestyle-changing symptoms. Surgical options are controversial. The most frequent procedure is transabdominal rectopexy (suture fixation of the rectum to the presacral fascia) and anterior resection of the sigmoid colon if constipation is a prominent feature of the patient's complaints.
2. **External rectal prolapse** is protrusion of full-thickness rectum through the anus. Symptoms include pain, bleeding, mucous discharge, and incontinence. Examination shows a prolapsed mass with concentric mucosal rings in distinction from prolapsed hemorrhoids (rectal mucosal prolapse), with which they may be confused but which show deep radial grooves. **Risk factors** include increased age, female gender, institutionalization, antipsychotic medication, previous hysterectomy, and spinal cord injury. **Evaluation includes barium enema or colonoscopy** to rule out malignancy and **manometry and electromyography** to determine sphincter function. With a **normal sphincter**, surgical strategy is to shorten the redundant rectosigmoid colon and reestablish posterior sacral attachment. In patients able to tolerate a general anesthetic and abdominal exploration, the procedure of choice is as for an internal prolapse. The lowest recurrence rates (7–8%) are obtained with transabdominal rectopexy with or without an anterior resection of the sigmoid colon (Goldberg-Frykaman procedure). **Incontinent patients** regain continence in 60–70% of cases, and final continence may not be evident for up to a year after surgical correction of the prolapse. Patients unable to withstand a general anesthetic or major abdominal procedure may be managed with a perineal proctectomy (modified Altmeier procedure). Although very well tolerated by the patient, it has a higher recurrence rate (~20%). This is also an attractive procedure for the rare young man with external rectal prolapse in whom potency is a concern.

E. Hemorrhoids are vascular and connective tissue cushions that exist in three columns in the anal canal: right anterolateral, right posterolateral, and left lateral. **Internal hemorrhoids** are above the dentate line and thus covered with mucosa. These may bleed and prolapse, but they do not cause pain. **External hemorrhoids** are below the dentate line and covered with anoderm. These do not bleed but may thrombose, which causes pain and itching, and secondary scarring may lead to skin tag formation. Hard stools, prolonged straining, increased abdominal pressure, and prolonged lack of support to the pelvic floor all contribute to the abnormal enlargement of hemorrhoidal tissue.

1. **Internal hemorrhoids** are **graded** as first degree (bleed only), second degree (bleed and prolapse but reduce spontaneously), third degree (bleed and prolapse and require manual reduction), and fourth degree (bleed and are incarcerated).
2. **Treatment** is medical for first-degree and most second-degree hemorrhoids. It includes increased dietary fiber and water, stool softeners, and avoidance of straining during defecation. **Refractory second- and third-degree hemorrhoids** may be treated in the office by elastic ligation. The ligation must be 1–2 cm above the dentate line, staying away from the rectum and sphincter to avoid pain and infection. One quadrant is ligated every 2 weeks in the office, and the patient is warned that the necrotic hemorrhoid may slough in 7–10 days, with bleeding noted at that time. Aspirin or other nonsteroidal antiinflammatory drugs

taken during this postoperative period increase the risk of bleeding with sloughing. Patients on anticoagulation should be treated with excisional hemorrhoidectomy instead of elastic ligation (see following section). Severe sepsis may occur after banding in immunocompromised patients or those who have had full-thickness rectal prolapse ligated by mistake. Patients present with severe pain, fever, and urinary retention within 12 hours of ligation. This life-threatening disorder should be treated with immediate removal of rubber bands accompanied by broad-spectrum intravenous antibiotics.

3. **Excisional hemorrhoidectomy** is reserved for large third- and fourth-degree hemorrhoids, mixed internal and external hemorrhoids, and thrombosed, incarcerated hemorrhoids with impending gangrene. The procedure is performed with the patient in the **prone flexed position**, and the mucosal edges are closed, leaving the anoderm open. Complications include a 10–50% incidence of urinary retention, bleeding, infection, sphincter injury, and anal stenosis from taking too much mucosa at the dentate line.
 4. **Stapled hemorrhoidectomy** is currently being evaluated as an alternative to traditional excisional hemorrhoidectomy. This procedure is performed by a circumferential excision of redundant rectal mucosa approximately 5 cm superior to the dentate line using a specially designed circular stapler. Preliminary results have shown a decrease in postoperative pain and a quicker return to normal activity. Randomized prospective trials are ongoing.
 5. **Acutely thrombosed external hemorrhoids** are treated by excision outside the mucocutaneous junction, which can be done in the office or emergency room with the wound left open. If the thrombosis is more than 48 hours old, the patient is treated with nonsurgical management.
- F. **Anal fissure** is a split in the anoderm. Ninety percent occur posteriorly, and 10% occur anteriorly. Symptoms include tearing pain with defecation (which lasts for hours afterward) and blood, usually on the toilet paper. Manometry and digital rectal examination demonstrate increased sphincter tone and muscular hypertrophy in the distal one-third of the internal sphincter. Repeated tearing results in a chronic anal ulcer that usually is accompanied by an external skin tag. Differential diagnosis includes Crohn's disease (often in the lateral location), tuberculosis, anal cancer, abscess or fistula, cytomegalovirus, herpes simplex virus, chlamydia, and syphilis. **Ninety percent heal with medical treatment** that includes increased fiber, stool softeners, and sitz baths. Topical nitroglycerin 0.2–0.5% has also been shown to promote healing. If surgery is required, lateral internal anal sphincterotomy is 90% successful. Recurrence and minor incontinence occur in fewer than 10% of patients.

Infections

I. Colitis

- A. **Pseudomembranous colitis** is an acute diarrheal illness resulting from toxins produced by overgrowth of *C. difficile* after antibiotic treatment (especially the use of clindamycin, ampicillin, or cephalosporins). Antibiotics already have been stopped in one-fourth of cases, and symptoms can occur up to 6 weeks after the cessation of antibiotic use. **Diagnosis** is made by measurement of toxin or culture of bacteria in one of at least three stool samples. Treatment begins with stopping the offending antibiotic followed by oral or intravenous metronidazole (250 mg p.o. every 6 hours or 500 mg i.v. every 8 hours). Oral (but not intravenous) vancomycin (500 mg p.o. every 6 hours) is an alternate, but more expensive, therapy. For severe cases in patients unable to take oral medications, vancomycin enemas may be useful. Recurrence after treatment is 20%, but this disease usually responds to retreatment. Rarely, pseudomembranous colitis presents with severe peritoneal irritation and colonic distention with **toxic megacolon** or **perforation**. Emergency laparotomy with total colectomy and end-ileostomy is required.
- B. **Amebic colitis** results from invasive infection by the protozoan *Entamoeba histolytica*, which is spread by the fecal-oral route. It is most commonly encountered in patients who have traveled abroad. The cecum usually is affected with small ulcers that may perforate or form an inflammatory mass or **ameboma**. **Diagnosis** is made by examining stool for ova and parasites, which is 90% sensitive in identifying the trophozoites. **Treatment** is oral metronidazole (250 mg p.o. every 6 hours) and iodoquinol. Surgical treatment is reserved for perforation or for ameboma refractory to treatment.
- C. **Actinomycosis** is an abdominal infection that most commonly occurs around the cecum after appendectomy owing to the anaerobic gram-positive *Actinomyces israelii*. An inflammatory mass often is present with sinuses to the skin that can drain **sulfur granules**. **Diagnosis** is confirmed by anaerobic culture (the organism may take up to a week to isolate), and **surgical drainage** combined with penicillin or tetracycline is required.
- D. **Neutropeic enterocolitis** after chemotherapy occurs most commonly in the setting of acute myelogenous leukemia after cytosine arabinoside therapy. Patients present with abdominal pain, fever, bloody diarrhea, distention, and sepsis. The cecum often dilates, and there may be pneumatosis. **Initial treatment** includes bowel rest, total parenteral nutrition, and broad-spectrum intravenous antibiotics. Laparotomy with colectomy and ileostomy is required if peritonitis develops. If further chemotherapy is required, elective right colectomy is considered.
- E. **Cytomegalovirus colitis** presents with bloody diarrhea, fever, and weight loss. It affects 10% of patients with acquired immunodeficiency syndrome (homosexuals are more commonly affected) and is the most common cause for emergent abdominal surgery in patients with acquired immunodeficiency syndrome. Ganciclovir is the treatment of choice, with emergent colectomy with ileostomy reserved for toxic megacolon.

II. Infection of the anorectum

A. Anorectal abscess

1. **Cryptoglandular abscess** results from infection of the anal glands in the crypts at the dentate line. Because the glands penetrate the internal sphincter, the initial abscess is in the intersphincteric space. Then infection can spread (1) superficially to the external sphincter into the perianal space, (2) through the external sphincter into the ischioanal space (which in turn may connect posteriorly via the deep postanal space, resulting in a horseshoe abscess), or (3) deep to the external sphincter into the supralelevator space.
 - a. **Diagnosis** usually is obvious, with severe anal pain and a palpable, fluctuant mass.
 - b. **Treatment** is surgical drainage, with the skin incision kept close to the dentate line to avoid the possible creation of a long fistula tract. Intersphincteric abscesses are drained by an internal sphincterotomy. Perianal and ischioanal abscesses are drained through the perianal skin, often with the use of small (No. 10–14 French) mushroom catheters to minimize incision length and subsequent scarring. Antibiotic therapy is not necessary unless the patient (1) is immunocompromised, (2) is diabetic, (3) has extensive cellulitis, or (4) has valvular heart disease. Immunocompromised patients may present with anal pain without fluctuance because of the paucity of leukocytes. The painful indurated region must still be drained, and the underlying tissue must undergo biopsy and culture.
 - c. **Outcome from drainage alone** shows that 50% of patients are cured, and 50% develop a chronic fistula. We do not advocate fistulotomy at the initial operation because the internal opening may not be evident, and a complicated fistulotomy may result in sphincter injury.
2. **Necrotizing anorectal infection (Fournier's gangrene)** occurs rarely but can result in massive, life-threatening tissue destruction. Patients present with systemic toxicity and perianal pain. There may be crepitation and extensive necrosis under relatively normal skin. Synergistic flora (including clostridial and streptococcal species) of anorectal and urogenital origin may be involved. Immediate wide surgical débridement of all nonviable tissue and intravenous antibiotics are mandatory. Early treatment is critical, but mortality is still approximately 50%.
3. **Fistula in ano** represents the chronic stage of cryptoglandular abscess but also may be due to trauma, Crohn's disease, tuberculosis, cancer, and radiation.
 - a. Patients **present** with persistent purulent perianal drainage from the external and the internal openings of the fistula. The location of the internal opening along the dentate line is approximated by using **Goodsall's rule**: Fistulas with external openings anterior to a transverse plane through the anal canal penetrate toward the dentate line in a radial direction, whereas fistulas posterior to that plane curve so that the internal opening is in the posterior midline. One exception is fistulas in which the anterior opening is more than 3 cm from the anal margin; these tend to have an internal opening in the posterior midline.
 - b. **Treatment** is fistulotomy with curette or cauterization of the granulation in the fistula tract and healing by secondary intention. Transsphincteric fistulas are incised only if there is adequate puborectalis and external sphincter external to the tract and kept open by marsupialization. Horseshoe fistulas have a high recurrence rate and require bilateral counterdrainage with mushroom catheters and fistulotomy of the posterior tract. High complicated fistulas involving the majority of the external sphincter cannot be incised. Seton placement allows slow scarring of the fistula tract but also has a 17% incontinence rate. We prefer primary drainage, with delayed repair using an endorectal advancement flap.

B. Rectovaginal fistula

1. The **diagnosis** of rectovaginal fistula is suspected from a history of passing flatus or stool per vagina. Diagnosis is usually made via speculum or anoscopic examination but occasionally administration of methylene blue enema, which can contaminate a vaginal tampon, is necessary.
 2. **Classification** and etiology depend on location. Low fistulas are due to obstetric injuries, foreign body penetration, or Crohn's disease. Mid fistulas are due to ischioanal abscess, Crohn's disease, radiation, or surgical excision of an anterior rectal tumor. High fistulas are due to diverticulitis, operative injury, radiation, or Crohn's disease. Carcinoma of the rectum, cervix, or vagina may result in fistulas at any level.
 3. **Treatment** depends on the cause. Fistulas due to abscesses are counterdrained through the perianal skin, and the fistula is observed. Fistulas due to obstetric injury are observed for 3 months before operative repair is attempted. Low and mid fistulas are treated with endorectal advancement (sliding) flap after full mechanical and antibiotic bowel preparation. High fistulas are best treated transabdominally with resection of the affected segment of rectum. Nonhealing fistulas are biopsied to rule out cancer. Operative repair of fistulas due to radiation rarely heal and should not be undertaken because the tissue is permanently damaged. Advancement flaps for fistulas due to perineal Crohn's disease are often successful if the Crohn's disease is in remission.
- C. **Pilonidal disease** occurs secondary to infection of a hair-containing sinus in the postsacral intergluteal fold 5 cm superior to the anus. Patients present with pain, swelling, and drainage when the sinuses become infected. The disease is most prevalent in men in the second and third decades of life. Symptoms are

distinguished from perianal abscess by the lack of anal pain, the more superior location of the fluctuant mass, and the presence of midline cutaneous pits. Treatment is incision, drainage, and curettage, allowing secondary closure when acutely inflamed. The disease tends to recur, however, and once the active inflammation has resolved, the sinus can be excised electively, with primary closure and a higher chance of cure. In extensive disease, excision of the sinus followed by a Z-plasty to obliterate the intergluteal cleft has been described.

- D. **Hidradenitis suppurativa** is an infection of the apocrine sweat glands and mimics fistula in ano except that involvement is external to the anal verge. The treatment of choice is wide incision of the involved skin.
- E. **Pruritus ani** is a common symptom often secondary to dietary factors (e.g., coffee, alcohol) or to such local factors as excessive washing. Treatment is directed toward proper diet and hygiene. Persistent symptoms prompt an investigation for possible hemorrhoids, villous tumors, or prolapse. Children should be evaluated for pinworms, which, if found, are treated with piperazine citrate.
- F. **Proctitis and anusitis** present as a bloody mucous discharge with tenesmus. They may be inflammatory, owing to ulcerative colitis or Crohn's disease, or secondary to radiation. Treatment includes increased dietary fiber, hydrocortisone suppositories, and such antispasmodics as belladonna and opium suppositories.
- G. **Condyloma acuminatum** is an anorectal and urogenital wart caused by infection with human papilloma virus. The disease is most commonly transmitted with anal intercourse and presents with visible perianal growth, often accompanied by pruritus, anal discharge, bleeding, and pain. Common treatments include topical bichloroacetic acid, 25% podophyllin application, and excision with electrocoagulation under local anesthesia. Smoke generated with coagulation contains viable organisms and must be completely evacuated.

Inflammatory Bowel Disease

I. General considerations

- A. **Ulcerative colitis** is an inflammatory process of colonic mucosa characterized by alterations in bowel function, most commonly bloody diarrhea with tenesmus. The disease always involves the rectum and may extend continuously to the proximal colon. Patients often have abdominal pain, fever, and weight loss, which correlate to the degree of intestinal inflammation. The disease has a male predominance. As the duration of the inflammation increases, pathologic changes progress. Initially, mucosal ulcers and crypt abscesses are seen. Later, mucosal edema and pseudopolyps (islands of normal mucosa surrounded by deep ulcers) develop, and the end-stage pathologic changes show a flattened, dysplastic mucosa. The lumen is normal in diameter.
- B. **Crohn's disease** is a transmural inflammatory process that can affect any area of the GI tract from the mouth to the anus. The disease has a segmental distribution with areas of normal mucosa interspersed between affected areas of bowel. Common symptoms include diarrhea, abdominal pain, nausea and vomiting, weight loss, and fever. There can be signs of an abdominal mass or of perianal fistulas on physical examination. The disease has a female predominance. Common pathologic changes include fissures and fistulas, transmural inflammation, and granulomas. Grossly, the mucosa shows aphthoid ulcers that often deepen over time and are associated with fat wrapping and bowel wall thickening. As this progresses, the bowel lumen narrows and obstruction or perforation may result.
- C. **Indeterminate colitis** is a term used for cases in which the pathologic pattern does not fall clearly into one or the other aforementioned patterns. This can be due either to inadequate tissue biopsy or to a truly indeterminate form of disease.
- D. **Extraintestinal manifestations** of inflammatory bowel disease are common with ulcerative colitis and with Crohn's disease. Musculoskeletal, skin, eye, blood, and hepatic abnormalities predominate.
- E. In the **chronic phase of inflammatory bowel disease**, pathologic patterns of ulcerative colitis differ from those of Crohn's disease. Ulcerative colitis shows dry granular mucosa, which may harbor severe dysplasia or malignancy. In Crohn's disease, there is bowel wall scarring with loss of reservoir function. The mucosa is less involved.

II. Ulcerative colitis

A. Indications for surgery

1. **Active disease unresponsive to medical treatment.** Failure to wean from high-dose steroids after two successive tapers prompts evaluation for surgery.
 2. The **risk of malignancy** is related to extent and duration of disease but not intensity of disease. The risk increases by 1% per year after 10 years of disease. Colonoscopy is performed 7–10 years after the diagnosis and every 1–2 years thereafter with random and specific biopsies. If dysplasia is found resection is recommended.
 3. **Severe bleeding** that does not respond adequately to medical therapy requires resection for control.
 4. **Toxic megacolon** initially is treated with bowel rest, antibiotics, steroids, and avoidance of contrast enemas, antidiarrheals, and morphine. If the patient develops worsening sepsis or peritonitis, abdominal colectomy is performed.
- B. **Surgical management** aims at removing the colorectal mucosa while maintaining bowel function as much as possible. Because the disease is localized to the rectum and colon, curative resection is possible. Sphincter-sparing procedures are preferred because they preserve the functions of continence and defecation. However, they are associated with higher postoperative complication risk. Anal sphincter function is assessed with manometry to ensure normal function before contemplating a sphincter-sparing procedure in a patient medically able to undergo the operation.
1. **Restorative proctocolectomy (ileal pouch–anal anastomosis)** maintains fecal stream through the anal sphincter mechanism and is the operation of choice in most patients. An abdominal colectomy is carried out to a level 3 cm above the levators. At this point, the colon may be transected and removed, and a mucosal proctectomy is performed transanally, leaving the sphincters, the levators, and the distal 3 cm of rectal muscularis *in situ*. A distal ileal pouch is constructed over a distance of 15 cm in a J or S configuration, pulled through the sphincters, and sutured to the dentate line. A stapled anastomosis leaving a 2- to 3-cm cuff of anal canal mucosa technically is easier but requires long-term surveillance of the residual mucosa. A protecting loop ileostomy is constructed and is taken down 3 months later, after healing of the distal anastomosis. **Complications** include increased stool frequency (5–7 daily), nocturnal soiling in 20–55% of patients, and pouchitis, an intermittent inflammatory process that typically responds to metronidazole in 28% of patients. Pouch capacity increases over time, and eventually the need to empty the pouch may be decreased to an average of four to five times a day.
 2. **Total proctocolectomy and ileostomy** are performed in patients who have perioperative sphincter dysfunction or incontinence and in high-risk patients who would not tolerate potential postoperative complications. Most patients do well with a well-placed **Brook ileostomy** that has a spigot configuration and empties into a bag appliance in an uncontrolled fashion. A **Kock pouch** or continent ileostomy does not empty spontaneously, does not require a permanent appliance, and requires cannulation six to eight times daily. These are more difficult to construct and prone to obstruction. This alternative is occasionally offered to patients who desire continence or who have severe skin allergies, making ileostomy appliances problematic.

III. Crohn's disease is a chronic disease that is not surgically curable. Surgery should therefore be performed only for complications of the disease, with the foreknowledge that recurrence is common and the awareness of the segmental nature of the disease. Complications of acute disease include perforation, fistulas, and phlegmon. Chronic disease results in a fibrotic phase in which strictures may form. When a patient presents with a complication requiring surgery, all attempts should be made to prepare the patient so that a single operation will suffice. With complications of acute disease, this may require bowel rest with total parenteral nutrition, antibiotics, antiinflammatory medications, and, when indicated, percutaneous drainage of abscesses.

- A. **Surgical management** of Crohn's disease involves resection of the diseased segment of intestine responsible for the complications requiring surgery. Resection is bounded by grossly normal margins; no attempt is made to obtain microscopically negative margins because outcome and recurrence are unaffected by this. If significant intraabdominal infection or inflammation is encountered during surgery, a proximal ostomy is created to allow complete diversion of intestinal contents and resolution of the initial process. If no infection or inflammation is encountered, normal-appearing bowel can be primarily anastomosed.
- B. **Small-intestinal Crohn's disease** is covered in [Chapter 14](#).
- C. **Colonic Crohn's disease** often requires operation after a shorter duration of symptoms than is typical for patients with either small-intestinal or ileocolic Crohn's disease. Perforation can occur without dilation of the colon, secondary to thickening of the colonic wall. Surgical options include total abdominal colectomy with ileorectal anastomosis (which may be protected by a proximal loop ileostomy), total abdominal colectomy with an end ileostomy and maintenance of the rectum as a Hartmann's pouch, or abdominal perineal proctocolectomy with permanent end-ileostomy. Sphincter-preserving surgery is not an option secondary to the risk of recurrence. Rarely, colonic strictures can occur in an isolated segment, causing obstruction. Resection should be considered, although dilation has been described in situations in which surveillance of the proximal colon is not possible. Strictureplasty to salvage part of the colon should be avoided because the colon has no critical physiologic function.
- D. **Rectal Crohn's disease** rarely occurs in isolation. Once the rectum has become so fibrotic that it loses its reservoir capacity, proctectomy should be considered. Precise dissection along the rectal wall should minimize complications in comparison to patients treated for rectal cancer, in whom surrounding tissue must be included in the resection specimen.
- E. **Anal disease** occurs in 35% of patients with Crohn's disease. Conservative therapy with antiinflammatory agents, immunosuppressive agents, and metronidazole is the mainstay of treatment for anal Crohn's disease. Perianal sepsis should be controlled with local drainage (see Infections, [section II.A](#)).
- F. **Ileostomy construction**

1. **Content of the effluent from an ileostomy** is high volume and liquid and contains proteolytic enzymes. It is crucial to maintain a stoma appliance that protects the surrounding skin.
2. **Stoma construction** of either a loop ileostomy or end-ileostomy should include eversion of the end with three-point sutures. This results in a spigot configuration. Precise apposition of mucosa and skin prevents serositis and obstruction.
3. **Ileostomy care** requires special attention to avoid dehydration and obstruction. The patient is encouraged to drink plenty of fluids and to use antidiarrheal agents as needed to decrease output volume. Patients should be warned to avoid fibrous foods, such as whole vegetables and citrus fruits, because these may form a bolus of indigestible solid matter that can obstruct the stoma. Irrigating the ostomy with 50 mL warm saline from a Foley catheter inserted beneath the fascia, in combination with intravenous fluids and nasogastric decompression, may relieve obstruction and dehydration. If the obstruction is not due to food blockage, water-soluble contrast enema may be diagnostic and therapeutic.

Neoplastic Disease

- I. The **etiology** of colorectal neoplasia has genetic and environmental components.
 - A. **Familial cancer syndromes** account for 10–15% of colorectal cancers. One in 200 people carry high-risk alleles that cause inherited colorectal cancer.
 1. **Familial adenomatous polyposis (FAP)**
 - a. FAP is an **autosomal dominant disorder** clinically diagnosed when a patient has more than 100 adenomatous polyps in the large bowel or when a member of an FAP family has an adenomatous polyp detected. FAP patients account for approximately 1% of all patients with colorectal neoplasia. The average age of diagnosis of an FAP patient is 29 years, and the average age of diagnosis of colorectal cancer is 39 years.
 - b. **The genetic defect in FAP** is an alteration in the adenomatous polyposis coli (APC) gene. Ten to 20% of FAP patients have acquired the abnormality by spontaneous mutation.
 - c. **FAP syndromes**
 1. **Familial polyposis.**
 2. **Gardner's syndrome** includes epidermal inclusion cysts and bone osteomas.
 3. **Turcot's syndrome** includes brain tumors (usually glioma or medulloblastoma).
 - d. **Extracolonic manifestations**
 1. **Upper GI neoplasms** include hyperplastic fundic polyps and duodenal adenomas. Pancreaticoduodenectomy (Whipple procedure) is performed when cancer or villous adenoma is found.
 2. **Desmoid tumors** are the only incapacitating, lethal extraintestinal manifestations of FAP and can form dense fibrous tissue in the mesentery, leading to vascular encroachment.
 - a. **Treatment** has been attempted with chemotherapy [doxorubicin (Adriamycin)], radiation, tamoxifen, and sulindac (200 mg p.o. b.i.d. has been reported to cause tumor regression).
 - b. **Operative therapy** is generally inadequate and should be reserved only for decompression of obstruction or to relieve incapacitating symptoms. Pseudocapsules form around desmoid tumors, and recurrence is likely unless wide margins are obtained beyond the pseudocapsule.
 3. **Epidermoid cysts** are only of cosmetic significance.
 - e. **Screening of patients** from FAP kindreds may be done by DNA analysis for mutated APC gene product from peripheral blood. Given the large size of the APC gene (~10 kb) and the genotypic variations resulting in the phenotypic expression of the syndrome, direct genetic testing is impractical unless the genetic defect within the affected kindred is known. Protein truncation assay is the most widely used noninvasive modality to screen at-risk populations. It will likely replace ophthalmologic examination for congenital hypertrophy of the retinal pigmented epithelium (highly suggestive of the disease) as the screening modality of choice. Screening colonoscopy is also a viable modality commencing when the at-risk patients reach the age of 10 years and periodically thereafter. As with other familial cancer syndromes, genetic testing and risk assessment are probably best done in conjunction with a trained genetic counselor.
 - f. **Surgical management** involves removing the tissue at risk for malignancy.
 1. **Total proctocolectomy** with ileal reservoir and ileoanal anastomosis is becoming the operation of choice in patients with FAP. The operative morbidity with regard to incontinence and pouchitis is slightly better in FAP patients than in patients with ulcerative colitis.
 2. **Colectomy and ileorectostomy** are considered if there is relatively little rectal involvement (<50 polyps). There is a long-term cancer risk of 2–40%, and vigilant surveillance with biannual proctoscopy must be carried out. Sulindac, 150 mg p.o. b.i.d., and vitamin C may have a beneficial effect in decreasing the number and size of residual polyps. This operation is not recommended for patients who will not be compliant with lifelong follow-up due to the risk of cancer in the remaining stump.
 3. **Proctocolectomy and ileostomy** remove all tissue at risk but leave a permanent ileostomy and are generally a less desirable option.
 2. **Hereditary nonpolyposis colon cancer (HNPCC)**
 - a. HNPCC is an **autosomal dominant disorder** without a specific phenotypic marker and accounts for up to 10–15% of colorectal neoplasia. In these kindreds, there is also an increased incidence of endometrial, breast, and prostate cancer. The most widely accepted criteria for diagnosis are the **Amsterdam criteria**:
 1. **At least three relatives with histologically verified colorectal cancer**, one of which is a first-degree relative of another
 2. **Involvement of at least two generations**
 3. **At least one colorectal cancer diagnosis before the age of 50 years**
 - b. The **genetic basis of HNPCC** is instability in microsatellite regions (i.e., short repetitive sequences located throughout the genome) of DNA. This is caused secondary to a loss of fidelity in DNA repair known as a *replication error*. Alterations in the mismatch repair genes hMSH2, hMLH1, and, to a lesser extent, hPMS1 and hPMS2 account for the majority of patients with HNPCC.
 - c. Clinically, patients with HNPCC have a predominance of **right-sided cancer**. Approximately 20% have a synchronous cancer, and 25% develop a metachronous cancer. In light of these findings, subtotal colectomy is the recommended **surgical treatment** for HNPCC patients. Tumors are more often poorly differentiated, mucin-producing, or signet-ring-type cancers than in patients with sporadic colon cancer. Despite these findings, patients with HNPCC do better than those with sporadic colon cancer of the same pathologic stage.
 - d. **Genetic screening** is currently available for many of the specific mutations that cause HNPCC. Although no definite consensus on surveillance exists, we recommend colonoscopy every 1–2 years beginning at age 18 years or at least 5 years before the age at diagnosis of the youngest family member with cancer.
 - B. **Sporadic cancers** account for approximately 85% of colorectal neoplasia. Although no inherited genetic mutation can be identified, first-degree relatives of patients with colorectal cancer have a three- to ninefold increase in the risk of developing the disease. Overwhelming evidence suggests that colorectal carcinomas develop from precursor adenomas and are associated with an increasing number of genetic mutations. A single genetic mutation in the germ line of a patient may cause an adenoma to develop. Further mutation in either a tumor-suppressor gene or oncogene is responsible for further development of the adenoma and eventually the transformation to neoplasia. Genes implicated in this journey from normal epithelium to carcinoma include *K-ras*, *DCC*, and *p53*.
 - C. **Environmental factors** have also been proposed to play a significant role in the etiology of colorectal neoplasia. Dietary factors that have been shown to increase cancer risk include a diet high in unsaturated animal fats and highly saturated vegetable oils. Increased fiber decreases cancer risk in those on a high-fat diet. Epidemiologic studies indicate that people from less industrialized countries have a lower risk of colorectal cancer. This appears to be associated with a diet that is lower in processed foods and higher in natural fiber. This survival benefit disappears in people who immigrate to the United States.
 - D. **Premalignant conditions.** The incidence of cancer in ulcerative colitis with pancolitis is 1% per year after 10 years or 10% after 20 years, and the risk of cancer being present when biopsies show dysplasia is 30%. The cancers tend to be more advanced (~35% are stage III or IV) when they present. Crohn's disease also has an increased cancer risk, which is as high as 7% at 20 years (although this might be primarily in patients who have excluded segments of bowel). There is a high cancer incidence at stricture sites, and biopsies are performed at the time of stricturoplasty. Squamous cell cancers and adenocarcinomas can develop in chronic fistula tracts.

II. Detection

- A. **Tests of occult blood** are of unclear benefit. In the guaiac reaction, a colorless phenol in guaiac gum is oxidized in the presence of a peroxidase catalyst into a blue quinone. In addition to hemoglobin, other dietary components (e.g., meat and some vegetables) catalyze this reaction, leading to false positives. The positive predictive value is only approximately 2.5%. Large-scale screening studies with stool guaiac demonstrate that the test may decrease mortality by identifying cancers at an earlier stage; however, its cost effectiveness is still in question.
- B. **Diagnosis** relies on good history and physical examination in patients at risk. Rectal examination can identify cancers up to 8 cm above the dentate line (20% of colorectal cancer). Proctosigmoidoscopy can be performed in the office with a single enema preparation and reaches cancers up to 20–25 cm. Cancers distal to

the splenic flexure (50% of colorectal tumors) are seen by flexible sigmoidoscopy after a one-to two-enema preparation. Air-contrast (double-contrast) barium enema is 90% sensitive for polyps greater than 1 cm in diameter. If perforation is an increased possibility, water-soluble contrast should be used instead of barium because of the devastating results of barium peritonitis. Colonoscopy is the most accurate method for detecting polyps less than 1 cm in diameter. The complication rate is less than 0.2%.

C. Imaging techniques to assess invasion or metastases

1. **CT scan** identifies liver metastases as well as adrenal, ovarian, pelvic, and lymph node metastases (in rectal cancer, CT scan also identifies rectal wall invasion with 60–90% accuracy and detects lymph nodes >1 cm in 75% of cases). A CT with intravenous contrast also demonstrates the integrity of the urinary tract. Combined CT scan and angiogram (angio-computed tomography) is approximately 95% sensitive in detecting liver metastases. CT scan is also useful in detecting recurrent disease postoperatively in a patient with a rising carcinoembryonic antigen (CEA) level.
 2. **MR scan** yields information similar to that obtained from the CT scan and has little or no advantage except for clarifying questionable liver lesions detected by CT. This is due to the increased sensitivity of MR scan for detecting liver metastases.
 3. **Fluorodeoxyglucose positron emission tomography (PET)** detects differences in the metabolism of fluorodeoxyglucose. It is the most sensitive test in detecting recurrent carcinoma in a postoperative patient with a rising CEA level. PET scan allows differentiation of metabolically active tumor from scar tissue (which is not active metabolically), which often appear similar on CT scan.
 4. **Transrectal ultrasonography** is accurate in detecting depth of invasion in rectal cancer. T1 lesions are limited to the mucosa and submucosa, T2 lesions are limited to the muscularis propria, T3 lesions penetrate the rectal wall, and T4 lesions invade adjacent structures. It may also detect regional adenopathy.
 5. **Monoclonal antibodies** tagged with a radioactive isotope that recognizes CEA or cell membrane features of colon cancer allow tumor imaging and hold promise for therapeutic destruction of colorectal carcinomas in the future.
- D. **Surveillance** is the periodic complete examination of a patient with known increased risk. **Screening** is the limited examination of a population with the goal of detecting patients with increased risk.
1. **Screening** of the general population is of unproved cost effectiveness. The American Cancer Society recommends yearly digital rectal examination and testing for fecal occult blood past age 40 years. Air-contrast barium enema with proctoscopy or total colonoscopy should be offered to patients at the age of 50 years. These should be repeated every 10 years as long as normal or if the patient is not at high risk for colorectal neoplasia. Another screening algorithm calls for flexible sigmoidoscopy at age 50 years with repeat imaging every 3–5 years. Indications for colonoscopy in this algorithm are detection of fecal blood or polyp on sigmoidoscopy.
 2. **High-risk individuals** should be in a surveillance program. Previous cancer or polypectomy increases the risk of metachronous cancer by a factor of 2.7–7.7. Routine surveillance has been shown to reduce the incidence of metachronous cancer, although its influence on survival is unknown. High-risk patients are those with (1) ulcerative colitis of greater than 10 years' duration, (2) Crohn's disease with stricture, (3) a history or family history of polyps or cancer, or (4) a family history of FAP or HNPCC. Our surveillance algorithm calls for initial or perioperative colonoscopy followed by yearly examination until no lesions are detected, examination every 3 years until no lesions are detected, and then examination every 5 years.

III. Polyps

A. Nonadenomatous polyps

1. **Peutz-Jeghers syndrome** is an autosomal dominant condition characterized by hamartomatous polyps of smooth muscle throughout the GI tract and mucocutaneous pigmentation. It is associated with increased risk of malignancy in multiple organs and with a 2–13% incidence of GI malignancy. Symptoms include bleeding or obstruction secondary to intussusception. Treatment is polypectomy for polyps greater than 1.5 cm in diameter. When symptomatic polyps result in surgery, all polyps should be removed via intraoperative polypectomy and endoscopy. Surveillance colonoscopy and esophagogastroduodenoscopy (EGD) are recommended every 2 years as well as periodic screening for breast, cervical, testicular, ovarian, and pancreatic cancer.
 2. **Juvenile polyps** are cystic dilations of glandular structures in the lamina propria without malignant potential that may result in bleeding or obstruction. There are two peaks in incidence of isolated juvenile polyps: in infants and at age 25 years. They are the most common cause of GI bleeding in children and should be treated with polypectomy. **Multiple polyposis coli** (diffuse juvenile polyps) is an autosomal dominant syndrome characterized by multiple juvenile polyps and increased risk for GI malignancy. These patients are considered for total abdominal colectomy or proctocolectomy with ileal pouch–anal anastomosis.
 3. **Hyperplastic polyps** show epithelial dysmaturity and hyperplasia and are the most common colorectal neoplasm (ten times greater than adenoma). They have no malignant potential. Most are less than 0.5 cm in diameter and rarely need treatment.
- B. **Adenomas** are benign neoplasms with unrestricted proliferation of glandular epithelium within the colonic mucosa but with no invasion of the basement membrane. It is generally accepted that adenomas are precursors to colorectal cancer in the majority of cases. The degree of differentiation decreases as a polyp becomes more like a cancer. *Severe atypia* refers to malignant cells in a polyp that have not invaded the muscularis mucosae (formerly known as *carcinoma in situ*). Adenomatous polyps fall into three broad categories.
1. **Tubular adenomas** are usually pedunculated and account for 65–80% of adenomas. They have a 5% risk of carrying cancer.
 2. **Tubulovillous adenomas** account for 10–25% of adenomas. They have a 22% risk of carrying cancer.
 3. **Villous adenomas** are usually sessile and account for 5–10% of adenomas. They have a 40% risk of carrying cancer.
- C. **Treatment** consists of colonoscopic removal. Pedunculated polyps have a stalk of less than 1.5 cm and are removed using the cautery snare. Semisessile and sessile polyps have stalks or pedicles greater than 1.5 cm and may require piecemeal extraction. The site of incomplete removal should be marked with 0.1 mL India ink for possible later intraoperative or repeat colonoscopic identification. In certain instances, surgery must be considered.
1. **The risk of metastatic cancer** in regional lymph nodes is 1% in a completely excised, pedunculated polyp in which cancer invades only the head of the polyp. These cases may be treated with either colectomy or polypectomy with close follow-up.
 2. **Sessile polyps** containing cancer and high-risk pedunculated polyps (with invasion into the base) require colectomy. Risk of lymphatic spread in these patients is approximately 10%, with a 10% local recurrence rate if there is (1) lymphatic invasion, (2) vascular invasion, (3) poor differentiation, or (4) close margin.
 3. **Colonoscopic complications** include perforation and bleeding. Conservative therapy for perforation with intravenous antibiotics and bowel rest is possible if sepsis and peritonitis are absent. Otherwise, urgent laparotomy with colonic repair with or without proximal diversion (loop ileostomy) is performed. An alternative is segmental colonic resection with primary anastomosis and is especially attractive if the perforated segment harbors intraluminal pathology. Bleeding may be immediate or delayed by 7–10 days. Angiography or colonoscopy is performed, depending on the rate of bleeding, followed by surgery if needed. Most postcolonoscopy bleeding abates spontaneously.
- D. **Villous adenoma of the rectum** can present with watery diarrhea and hypokalemia. The risk of cancer in lesions greater than 4 cm with induration is 90%, and transrectal ultrasonography should be used to determine thickness and possible invasion. **Treatment** is by transanal local excision, using epinephrine injected submucosally to aid dissection of the tumor away from the rectal wall and followed by closure of the mucosa with suture. If the adenoma is circumferential, a mucosal proctectomy and coloanal anastomosis are performed.

IV. Colon cancer

- A. The **incidence** of colorectal cancer in the United States has been stable since the 1950s, with 131,000 new cases (94,000 colon and 37,000 rectal) each year and 58,000 deaths each year. It is the third most lethal cancer in men and women, with a slight female predominance in colon cancer and male predominance in rectal cancer. There is a 5% lifetime risk; 6–8% occur before age 40 years, and incidence increases steadily after age 50.
- B. **Subacute presentation** of colon cancer depends on the location of the lesion. **Right colon** lesions occasionally cause hematochezia, but more often bleeding is occult, causing anemia and fatigue. **Left colon** lesions more often cause crampy abdominal pain, altered bowel habit, or hematochezia. Approximately 50% of patients with other symptoms complain of weight loss, but weight loss is almost never the sole manifestation of a colorectal tumor.
- C. **Acute presentation.** Left colon cancer presents as large-bowel obstruction with inability to pass flatus or feces, abdominal pain, and distention in fewer than 10% of cases. Rarely, colon cancer presents as perforation with focal or diffuse peritonitis or as a fistula with pneumaturia or feculent vaginal discharge. These symptoms may be difficult to distinguish from those of diverticulitis. Metastatic disease is usually asymptomatic but may present with jaundice, pruritus, and ascites or with cough and hemoptysis.
- D. **Diagnosis and staging**
 1. **Once the diagnosis is suspected based on history, physical examination, or screening tests**, every attempt should be made to obtain biopsy of the primary lesion and rule out synchronous cancer (3–5%). Colonoscopy to the cecum or flexible sigmoidoscopy and barium enema are acceptable. In patients presenting with obstructive symptoms, diagnosis is suggested on plain abdominal films, and water-soluble contrast is performed to assess the degree and level of obstruction.
 2. **Staging studies** to look for distant metastases include chest X-ray and abdominal CT scan. Serum CEA is a glycoprotein present in embryonic and fetal tissue but absent in normal adult colonic mucosa. Rising CEA after resection is suggestive of recurrence, but nonspecific elevation occurs in smokers and patients with cirrhosis, pancreatitis, renal failure, and ulcerative colitis. Elevated CEA is correlated with tumor recurrence and, when elevated preoperatively, is associated with higher postoperative recurrence rate regardless of the stage of the original tumor.
- E. **Surgical treatment**

1. **Bowel preparation** is critical to minimize the titers of *E. coli* and *Bacteroides fragilis*, which are the primary sources of infection. Mechanical cleansing consists of Fleet Phospho-Soda (mono and dibasic sodium phosphate), which acts as a purgative when given in adequate volume (45 mL), and is accompanied by large volumes (24 oz) of clear liquids. Two doses are given, at 12 p.m. and at 6 p.m., the day before surgery. Patients with cardiac or renal failure or severe hypertension on sodium restriction should be given these doses cautiously because hypocalcemia, hyperphosphatemia, hyponatremia, and acidosis may occur. An alternate mechanical bowel preparation includes an isotonic lavage solution (GoLYTELY) containing polyethylene glycol (an osmotic purgative) in a balanced salt solution. Early on the preoperative day, 4 L is given orally within a 4-hour period, accompanied by a clear liquid diet. Antibiotics are required to decrease the wound infection rate from 30% to less than 10%. We give oral neomycin (1 g p.o. at 1 p.m., 2 p.m., and 11 p.m.) on the preoperative day and broad-spectrum intravenous antibiotics immediately before surgery.
 2. **Operative technique** begins with a thorough exploration that includes palpation of the liver. The colonic segment is ligated to prevent intraluminal spread. The main segmental vessels are then ligated and divided, and *en bloc* resection of colon and any adherent structure is carried out, including small bowel, ovaries, uterus, or kidney. If curative resection is not possible, palliative resection should be attempted, and if this cannot be done, bypass should be performed. For right colon cancer, resection includes the distal 10 cm of terminal ileum to the transverse colon, taking the ileocolic, right colic, and right branch of the middle colic vessels. A transverse colon lesion is resected with either an extended right colectomy or a transverse colectomy, taking only the middle colic vessels. Left colon lesions require dividing the inferior mesenteric artery (IMA) at its origin, retaining the arc of Riolan. If multiple carcinomas are present, or if a colon carcinoma with multiple neoplastic polyps is present, then a subtotal colectomy is performed. The specimen margin is inspected in the operating room to ensure at least a 2-cm margin (5 cm for poorly differentiated tumors). Laparoscopic colectomy has been described for all forms of colorectal disease and, although safe, remains controversial. If laparoscopic colectomy is performed, it should be guided by the same principles as a standard open colectomy.
 3. **Emergency operations** are undertaken without bowel preparation and have a higher incidence of wound infection. For obstruction, right colectomy still can be performed with primary anastomosis and no diversion. Options with a left colon cancer include (1) resection with colostomy and either mucous fistula or Hartmann's pouch, (2) resection with intraoperative lavage and primary anastomosis, (3) resection with primary anastomosis and proximal diverting ileostomy, (4) subtotal colectomy and ileosigmoidostomy, and (5) colostomy with staged resection of the tumor in an unstable patient with markedly dilated colon. In all but the subtotal colectomy, the proximal colon must be evaluated in the postoperative period for synchronous cancer.
- F. **Staging and prognosis.** The **Dukes' classification** is the most widely used and useful staging system and is directly related to the American Joint Committee on Cancer TNM staging categories. It identifies the depth of invasion of the tumor (T), regional lymph node status (N), and presence of distant metastases (M). Dukes' A, or stage I, tumors do not involve the muscularis and have a 90% 5-year survival. Dukes' B (stage II) tumors penetrate the muscularis and have a 60–80% 5-year survival. Dukes' C (stage III) tumors involve lymph nodes and have a 20–50% 5-year survival. Dukes' D (stage IV) tumors have distant metastases and a 5-year survival of less than 5%. Unfavorable characteristics include poor differentiation, mucinous or signet-ring pathology, venous or perineural invasion, bowel perforation, aneuploid nuclei, and elevated CEA.
- G. **Adjuvant chemotherapy** using 5-fluorouracil and either levamisole or leucovorin increases survival and disease-free survival in patients with stage III (Dukes' C) colon cancer. Chemotherapy has not been shown to offer a survival benefit to patients with stage II (Dukes' B) cancer, and adjuvant treatment in this population is controversial.
- H. **Follow-up** is crucial in the first 4 years after surgery, when 90% of recurrences occur. Yearly colonoscopy is recommended. CEA tests may be checked, and rising levels prompt a CT scan, a chest X-ray, and possible PET scan evaluation to detect recurrence.

V. Rectal cancer

- A. The **pathophysiology** of rectal cancer differs from that of colon cancer in light of several anatomic factors: (1) confinement of pelvis and sphincters, making wide excision impossible; (2) proximity to urogenital structures and nerves, resulting in high levels of impotency in men; (3) dual blood supply and lymphatic drainage; and (4) transanal accessibility.
- B. **Diagnosis and staging**
1. **Local aspects.** Digital rectal examination can give information on the size, fixation, ulceration, local invasion, and lymph node status. Rigid sigmoidoscopy and biopsy are crucial to measure precisely the distance to the dentate line and to obtain a tissue diagnosis. Flexible sigmoidoscopy is not accurate at judging length of tumor from the dentate line (which in turn determines the operative procedure to be performed) and so is not a useful diagnostic modality when staging rectal cancer. Transrectal ultrasonography is accurate in assessing depth of invasion. Although lymph nodes are well visualized by transrectal ultrasonography, their presence does correlate with the risk of their containing tumor deposits.
 2. **Regional aspects.** Pelvic CT, MR scan, and transrectal ultrasound can yield information on local extension of tumor toward the bony pelvis. Pelvic examination is necessary to assess the possible fixation of the tumor to adjacent genitourinary structures. Cystoscopy may be required in some men to evaluate extension into the prostate or bladder.
 3. **Distant spread** is evaluated (as with colon cancer) with chest X-ray, abdominal CT, and serum CEA. PET scanning is frequently helpful in identifying recurrent disease.
- C. **Surgical treatment goals** are to remove cancer with adequate margins and perform an anastomosis only if there is good blood supply, absence of tension, and normal anal sphincters. If any of these conditions cannot be met, the entire rectum must be removed and the patient left with a permanent colostomy.
1. Mechanical and antibiotic **bowel preparation** is the same as that for colon cancer.
 2. The **stoma sites on the abdominal wall should be marked** for possible colostomy on the left side or midline, avoiding bony prominences and scars and staying inside the rectus muscle at the summit of a fat fold. The right lower quadrant should also be marked in the event that a temporary loop ileostomy is necessary. Stoma sites should be marked even if the surgeon is anticipating performing an anastomosis.
 3. **Positioning and preparation.** If the patient has had previous pelvic surgery or the cancer is suspected to involve the bladder or ureter, ureteral stents should be placed after induction of anesthesia. The patient is placed in the dorsal lithotomy position, giving access to the abdomen and perineum. A nasogastric tube, Foley catheter, and No. 34 French mushroom rectal catheter are placed, and the rectum is irrigated until clear with warm saline before instilling 100 mL povidone-iodine (Betadine).
 4. **Operative technique.** The patient's abdomen is explored through a midline incision. The left colon is mobilized using the embryonic fusion plane, reflecting the ureter and gonadal vessels laterally. The IMA is ligated at the aorta. The inferior mesenteric vein is ligated with the left colic artery. The colon is transected at the descending and sigmoid junction with a purse-string suture, and an end-to-end anastomosis stapler anvil is placed in the proximal segment. Rectal dissection then proceeds posteriorly through the avascular presacral fascia, laterally through the vascular lateral ligaments, and finally anteriorly, with preservation of the seminal vesicles or vagina. Dissection continues distally well beyond the tumor so that transection allows at least a 2-cm distal margin.
 5. **Surgical options** at this point depend on the height of the lesion, the condition of the sphincters, and the condition of the patient. An abdominoperineal resection is performed for tumors in the lower one-third of the rectum or if sphincter function is questionable or destroyed. Low anterior resection using an intraluminal stapler is the operation of choice when technically possible for tumors of the proximal two-thirds of the rectum. Hartmann's resection is chosen if there is preoperative obstruction, sepsis, or intraoperative instability. Coloanal anastomosis with temporary proximal stoma may be elected in patients with benign neoplasms or carpet villous adenomas low in the rectum, for management of radiation-induced rectal injury, and for selected rectal cancers in the lower third of the rectum.
 6. **Complications**
 - a. **Impotence** occurs in 50% of men and must be discussed preoperatively. The sites of nerve injury are the IMA origin, the presacral fascia, the lateral ligaments, and anteriorly at the level of the vagina and seminal vesicles. Prosthesis may be considered 1 year after surgery, once the pelvis is shown to be free of recurrence and the patient has had appropriate time to adapt to changes in body image.
 - b. **Leakage** at the anastomosis occurs in up to 20% of patients, typically on postoperative days 4–7. Fever, increased or changed drain output, or abdominal pain during this period should prompt in-depth physical examination and CT scan evaluation. Intravenous antibiotics and bowel rest are usually sufficient, but laparotomy and fecal diversion are necessary for large leaks. Anastomotic leaks are more common in young muscular men, possibly due to the power of their sphincter mechanisms, which act as a postoperative obstruction. These patients therefore undergo digital sphincter dilation intraoperatively, at the conclusion of the case, and every few days postoperatively until normal bowel function has returned.
 - c. **Massive presacral venous bleeding** can occur at the time of resection. This is controlled either with metal thumbtacks driven into the sacrum or by packing the pelvis for 24–48 hours.
 7. **Obstructive rectal cancer** requires emergent laparotomy on an unprepared bowel. The type of procedure depends on whether presurgical adjuvant therapy is considered. A decompressing transverse colostomy can be made through a small upper midline incision. This may be a blowhole type if the colon is massively dilated, or it may be a loop colostomy over a rod. If preoperative radiotherapy is not given, options include Hartmann's resection, total colectomy with ileorectostomy, and low anterior resection protected by proximal diversion.
 8. **Colostomy construction technique** depends on whether the goal is decompression or diversion.
 - a. A **decompressing colostomy** vents the distal and proximal bowel limbs while maintaining continuity between the limbs. A blowhole is used for massively dilated colon. The anterior wall of the transverse colon is sewn in two layers to the abdominal fascia and the skin. A loop colostomy is formed by bringing a loop of colon through the abdominal wall and suspending it on an ostomy rod. This colostomy also diverts for 6 weeks.
 - b. **Diverting colostomies**, such as end colostomy and mucous fistula, are used for distal resection or perforation so that the distal limb is completely separated from the fecal stream. All colostomies are matured in the operating room. If a stoma rod is used, it is removed 1 week after surgery.

- c. **Complications** include necrosis, stricture, and herniation. If the stoma becomes dusky, an anoscope is inserted. If necrosis does not extend below the fascia, it can be observed safely; otherwise, urgent revision is performed.
- D. **Adjuvant therapy** for rectal cancer should routinely be considered because the overall 5-year survival of the disease is only 50%, and the local recurrence rate is 20–30%.
1. **Preoperative radiotherapy** has been shown to increase 5-year survival and decrease local recurrences in all stages in a multicenter, prospective randomized trial and is now the standard of care (*N Engl J Med* 336:980, 1997). Two dosage options that are biologically similar may be considered: (1) 2,000 cGy over 5 days preoperatively followed by immediate operation or (2) 4,500 cGy over 5 weeks followed by a 7-week waiting period to allow for tumor shrinkage. Postoperative therapy has been advocated by some if the adequacy of the resection is in doubt and the patient did not receive preoperative radiotherapy. Postoperative radiotherapy is associated with a higher risk of complications, including injury to the small intestine and the colorectal anastomosis.
 2. **Neoadjuvant chemoradiation**, including chemotherapy with a 5-fluorouracil-based regimen, results in a modest survival benefit and decreased local recurrence over radiation therapy alone in patients with stage II and stage III disease.
- E. **Nonresectional therapy** is indicated in some early-stage cancers, patients with poor operative risk, and those with widespread metastases. Options include transanal excision, electrocoagulation, endocavitary radiation, cryotherapy, and laser vaporization. Most recently, we have used combined external-beam and endocavitary radiation as definitive treatment of favorable but invasive rectal cancers.
- F. If the patient has **incurable cancer** and a life expectancy of less than 6 months, external beam radiation with or without chemotherapy combined with laser destruction, dilation, or stenting the rectum can prevent obstruction. If life expectancy exceeds 6 months, resectional therapy is attempted.
- G. The major cause of **locally recurrent rectal cancer** is a positive margin on the pelvic side wall (radial margin). Recurrences tend to occur within 18 months and grow back into the lumen, presenting with pelvic pain, mass, and rectal bleeding or a rising CEA level. Diagnosis is confirmed by examination and biopsy as well as CT or PET scan. Treatment is not highly satisfactory, and there is a 10–20% palliation rate. If chemoradiation has not been given previously, it is given at this point, and, if distant metastases are again ruled out, low anterior resection, abdominoperineal resection, or pelvic exenteration (resection of rectum and urinary bladder) is performed based on whether the sphincters and the genitourinary organs are involved.

VI. Other colorectal tumors

- A. **Lymphoma** is most often metastatic to the colorectum, but primary non-Hodgkin's colonic lymphoma accounts for 10% of all GI lymphomas. The GI tract is also a common site of non-Hodgkin's lymphoma associated with human immunodeficiency virus. The most common presenting symptoms include abdominal pain, altered bowel habit, weight loss, and hematochezia. Biopsies are often not diagnostic because the lesion is submucosal. Workup is similar to that for colon cancer but should include a bone marrow biopsy and a thorough search for other adenopathy. Treatment is resection with postoperative chemotherapy. Intestinal bypass, biopsy, and postoperative chemotherapy should be considered for locally advanced tumors.
- B. **Retrorectal tumors** usually present with postural pain and a posterior rectal mass on physical examination and CT scan.
1. The **differential diagnosis** includes congenital, neurogenic, osseous, and inflammatory masses.
 2. **Diagnosis** is suspected based on CT scan and physical findings. Biopsy should not be performed. Formal resection should be undertaken.
- C. **Carcinoid tumor**
1. **Colonic carcinoid** accounts for 2% of GI carcinoids. Lesions less than 2 cm in diameter rarely metastasize, but 80% of lesions greater than 2 cm in diameter have local or distant metastases, with a median survival of less than 12 months. These lesions are treated with local excision if small and with formal resection if greater than 2 cm.
 2. **Rectal carcinoid** accounts for 15% of GI carcinoids. As with colonic carcinoids, lesions less than 2 cm in diameter have low malignant potential and are well treated with transanal or endoscopic resection. Rectal carcinoids greater than 2 cm in diameter are malignant in 90% of cases. Treatment of large rectal carcinoids is controversial, but low anterior resection or abdominoperineal resection is probably warranted.

VII. Anal neoplasms

- A. **Tumors of the anal margin**
1. **Squamous cell carcinoma** behaves like cutaneous squamous cell carcinoma, is well differentiated and keratinizing, and is treated with wide local excision with chemoradiation if large.
 2. **Basal cell carcinoma** is a rare, male-predominant cancer and is treated with local excision.
 3. **Bowen's disease** is intraepidermal squamous cell carcinoma. It is rare and usually slow growing. Treatment is wide local excision.
- B. **Anal canal tumors**
1. **Epidermoid carcinoma** is nonkeratinizing and derives from the anal canal 6–12 mm above the dentate line.
 - a. **Epidermoid cancer** has a female predominance and usually presents with an indurated, bleeding mass. On examination, the inguinal lymph nodes should be examined specifically because spread below the dentate line passes to the inguinal nodes. Diagnosis is made by biopsy, and 30–40% are metastatic at the time of diagnosis.
 - b. **Treatment** involves chemoradiation following the **Nigro protocol**: day 1 (3,000-cGy external-beam radiation; mitomycin C, 15 mg/m² i.v.; and 5-fluorouracil, 1,000 mg/m² every 24 hours by continuous infusion over 4 days) and day 30 (repeat 5-fluorouracil 4-day infusion). Surgical treatment is reserved for locally persistent or recurrent disease only. The procedure of choice is abdominoperineal resection.
 2. **Adenocarcinoma** is usually an extension of a low rectal cancer and has a poor prognosis.
 3. **Melanoma** accounts for 1–3% of anal cancers and is more common in Caucasians in the fifth and sixth decades of life. Symptoms include bleeding, pain, and a mass, and the diagnosis is often confused with that of a thrombosed hemorrhoid. At the time of diagnosis, 38% of patients have metastases. Treatment is wide local excision, and 5-year survival is less than 20%.

17 Pancreas

Keith D. Amos and Jeffrey A. Drebin

Disorders of the Pancreas

I. Acute pancreatitis

II. Chronic pancreatitis

III. Exocrine pancreatic cancer

IV. Congenital abnormalities

V. Exocrine pancreatic insufficiency

VI. Cystic diseases

Disorders of the Pancreas

I. Acute pancreatitis

A. **Pathophysiology.** Acute pancreatitis is an inflammatory process of variable clinical severity in which pancreatic injury ranges from mild edema to pancreatic and peripancreatic necrosis. Mechanisms of pathogenesis, which are not mutually exclusive, include the following:

1. **Secretion** into an obstructed duct
2. **Bile reflux** into the pancreatic duct
3. **Duodenal reflux** into the pancreatic duct
4. **Intracellular protease activation**

B. **Causes.** Biliary disorder (gallstones) accounts for approximately 40% of cases, alcohol accounts for approximately 40%, and drugs (definite association: isoniazid, estrogens; probable association: thiazides, furosemide, sulfonamides, tetracycline, corticosteroids) account for approximately 5%. Iatrogenic causes, particularly endoscopic retrograde cholangiopancreatography (ERCP), may account for another 5% of cases. Other less common causes include scorpion stings, hypertriglyceridemia (especially types I and V), hypercholesterolemia, hypercalcemia, infectious diseases (mumps; orchitis; Coxsackie virus B; Epstein-Barr virus; cytomegalovirus; rubella; hepatitis A, B, and non-A, non-B; *Ascaris* species; and *Mycoplasma pneumoniae*), tumors, trauma, and idiopathic factors.

C. Diagnosis

1. **Clinical presentation.** Patients typically complain of upper abdominal pain, often radiating to the back. Tenderness is usually limited to the upper abdomen but may be associated with signs of diffuse peritonitis. Occasionally, irritation from intraperitoneal pancreatic enzymes results in impressive peritoneal signs, simulating other causes of an acute abdomen. Nausea, vomiting, and a low-grade fever are frequent, as are tachycardia and hypotension secondary to hypovolemia. Asymptomatic hypoxemia, renal failure, hypocalcemia, and hyperglycemia are evidence of severe systemic effects. Rarely, peripheral manifestations (e.g., subcutaneous fat necrosis or pancreatic arthritis) can be associated with acute pancreatitis. Subcutaneous fat necrosis is not necessarily associated with a worse prognosis, but when multiple sites are involved, mortality is high.

2. Laboratory studies

a. **Serum amylase** is the single most useful test. Levels rise within 2–12 hours of symptoms and may return to normal over the following 2–5 days. Persistent elevations of levels for longer than 10 days indicate complications, such as pseudocyst formation. Normal levels can indicate resolution of acute pancreatitis, pancreatic hemorrhage, or pancreatic necrosis. There is no correlation between amylase level and etiology, prognosis, or severity.

Nonpancreatic causes of hyperamylasemia include renal failure, salivary gland disease, cirrhosis, hepatitis, common bile duct stones, acute cholecystitis, penetrating peptic ulcers, intestinal obstruction, afferent loop syndrome, diabetic ketoacidosis, lung cancer, pancreatic cancer, parotid tumors, ruptured ectopic pregnancy, endometritis, ruptured graafian follicle, and ovarian cysts and cancer. Amylase isoenzymes help differentiate between hyperamylasemia due to acute pancreatitis and other sources.

b. **Serum lipase** generally is considered more specific for pancreatic disease and remains elevated longer, returning to normal after 3–5 days.

c. **Urinary amylase** is increased in acute pancreatitis (usually >5,000 IU per 24 hours).

d. **Amylase-creatinine clearance ratio above 5** indicates acute pancreatitis. The test is unreliable in burns and diabetic ketoacidosis. It has not been shown to be any more accurate than serum or urinary amylase.

e. **Serum calcium** levels may fall as a result of complexing with fatty acids (saponification) produced by activated lipases.

f. **Discrimination between interstitial and necrotizing pancreatitis.** Some authors have demonstrated the usefulness of elevated serum levels of C-reactive protein and lactate dehydrogenase for early detection of necrotizing pancreatitis. Other markers associated with necrotizing pancreatitis include elevated serum levels of neutrophil-elastase, interleukin-6, and alpha-macroglobulin.

g. **Trypsin-like immunoreactivity** is more specific to the pancreas than is amylase measurement but is limited in availability. Elevated levels of trypsinlike immunoreactivity have been found in most cases of acute pancreatitis (see [section V.B.2.d](#)).

3. **Radiology.** No single radiographic technique provides a perfect index of acute pancreatitis.

a. **Plain films** can show pancreatic calcifications (best seen obliquely 15 degrees in either direction from either the supine or prone position), gallstones, focal areas of small-bowel dilatation, segmental ileus (sentinel loop sign), ascites, and the presence of upper abdominal masses. Chest radiographs may reveal right or left pleural effusions, basilar atelectasis, or an elevated left hemidiaphragm.

b. **Ultrasonography** sensitivity for pancreatitis ranges from 62% to 95%, whereas its specificity is greater than 95%. The pancreas is not visualized in up to 40% of patients due to overlying bowel gas.

c. **Computed tomographic (CT) scan** is superior to ultrasonography in evaluating the pancreas and is not limited by bowel gas. Contrast-enhanced CT scan has a sensitivity of 90% and specificity of close to 100% for acute pancreatitis and also can provide prognostic information by assessing the amount of glandular necrosis. CT findings include parenchymal enlargement and edema, necrosis, blurring of fat planes, peripancreatic fluid collections, bowel distention, and mesenteric edema.

d. **ERCP** is not indicated routinely in the evaluation of patients during an attack of acute pancreatitis. **Indications for ERCP** are as follows:

1. **Preoperative evaluation** of patients with suspected traumatic pancreatitis, to determine whether the pancreatic duct is disrupted
2. Patients with **suspected biliary pancreatitis and severe disease** who are not clinically improving by 24 hours after admission, so that endoscopic sphincterotomy and stone extraction may be performed
3. **Patients older than age 40 years with no identifiable disease** to rule out occult common bile duct stones, pancreatic or ampullary carcinoma, or other causes of obstruction
4. **Patients younger than age 40 years** who have had cholecystectomy or have experienced more than one attack of unexplained pancreatitis

e. **Percutaneous transhepatic cholangiography** is typically used in situations in which ERCP cannot be performed due to clinical, technical, or anatomic restrictions and in which documented common bile duct obstruction is present.

D. **Prognosis** can be estimated by Ranson's criteria ([Table 17-1](#)).

Table 17-1. Ranson's criteria

E. Complications

1. **Necrotizing pancreatitis** refers to loss of pancreatic microcirculation due to edema resulting in the death of significant portions of the pancreas; it develops in 10–20% of cases. The prognosis of necrotizing pancreatitis is linked closely to the extent of necrosis.

2. **Infected pancreatic necrosis** occurs in 5–10% of cases. Pancreatic infections are responsible for more than 80% of deaths. Gram-negative organisms are more common than gram-positive organisms (typically *Pseudomonas*, *Escherichia coli*, *Klebsiella*, *Proteus*, *Enterobacter*, and *Staphylococcus*). With the use of antibiotic prophylaxis, fungal infections are becoming increasingly more common.

3. **Acute pseudocyst** (see [section VI.A](#)).

F. **Treatment**

1. **Supportive care**

- a. **Bowel rest** (nothing by mouth; total parenteral nutrition if prolonged course).
- b. **Interactive volume resuscitation** with isotonic fluids (0.9% NaCl or lactated Ringer's solution). Urinary output is monitored with a Foley catheter.
- c. **Analgesia**. Narcotics usually are required for pain relief. Meperidine (Demerol) is preferred to morphine because some believe it has less spasmodic effect on the sphincter of Oddi.
- d. **Central hemodynamic monitoring (central venous pressure)** is useful to guide care in critically ill patients.
- e. **Respiratory monitoring** of supplementary oxygen and arterial blood gases should be done every 12 hours for the first 3 days in moderately severe pancreatitis to assess oxygenation and acid-base status. Hypoxemia is extremely common, even in mild cases of acute pancreatitis. Pulmonary complications occur in up to 50% of patients.
- f. **Alcohol withdrawal prophylaxis** is important during alcoholic pancreatitis.
- g. **Stress ulcer prophylaxis**, such as H₂-blockers, antacids, and proton pump inhibitors.
- h. **Antibiotics** are not effective in preventing septic complications in mild to moderate cases of pancreatitis. However, in several prospective, randomized trials, patients with severe pancreatitis who received prophylactic antibiotics had a significantly lower rate of septic complications (12% vs. 30%).
- i. **Nasogastric tube decompression** has not been shown to change the course of pancreatitis significantly, but it may be useful in the setting of intractable vomiting.
- j. **Somatostatin analogues** are not useful in treating acute pancreatitis.
- k. **Peritoneal dialysis** has not reduced mortality but might prevent early complications of severe pancreatitis (see [section I.F.2.c](#)).

2. **Surgical treatment**

- a. **Diagnostic uncertainty**. Exploratory laparotomy may be indicated if the diagnosis of pancreatitis is uncertain. Distortion and inflammation can make exploration of the right upper quadrant difficult; as many as 30% of common bile duct stones are missed. Intraoperative cholangiography is mandatory after cholecystectomy for choledocholithiasis.
- b. **Gallstone-induced pancreatitis**. Eradication of the biliary disease almost always prevents recurrent acute pancreatitis. Choledocholithiasis is found in only 25% of cases because gallstones frequently produce pancreatitis due to transient obstruction of the ampulla of Vater and then pass spontaneously. Cholecystectomy should be carried out as soon as possible after recovery to prevent recurrent attacks of pancreatitis. During open or laparoscopic cholecystectomy, intraoperative cholangiography is mandatory.
- c. **Pancreatic drainage and débridement**. For infected necrotizing pancreatitis, percutaneous drainage is futile. Wide débridement (necrosectomy) supplemented by either open packing or closed drainage is effective. Closed continuous lavage is hypothesized to reduce further infection and necrosis by cleansing the region of the devitalized tissue and the other vasoactive and toxic substances. However, controversy exists regarding the usefulness of closed lavage versus débridement and open packing of the retroperitoneum. At our institution, the preference is for wide débridement and packing, frequent débridement in the operating room, and eventual closure over large drains.
- d. **Pancreatic resection**. Because severe pancreatitis often leads to necrosis of large areas of the pancreas and peripancreatic fat, some have attempted to gain control of fulminant disease by resecting much or even all of the gland (partial or total pancreaticoduodenectomy). Radical resection procedures place greater stress on already severely ill patients; pancreaticoduodenectomy or total pancreatectomy is associated with very high mortality.

II. **Chronic pancreatitis**

A. **Etiology**. In the Western hemisphere, alcohol abuse accounts for more than 75% of cases. Causes for the remaining 25% include idiopathic, metabolic [hypercalcemia, hypertriglyceridemia (types I and V), hypercholesterolemia, hyperparathyroidism, cystic fibrosis], drugs, trauma, and congenital abnormalities (sphincter of Oddi dysfunction or pancreas divisum). Chronic pancreatitis is characterized by diffuse scarring and strictures in the pancreatic duct and is often seen in association with endocrine or exocrine insufficiency. A history of recurrent acute pancreatitis is present in some but not all patients with chronic pancreatitis.

B. **Diagnosis**

1. **History and physical examination**. A complete history and physical examination should be complemented by the appropriate investigative studies. It is not possible to rely on clinical detection of known stigmas of pancreatic disease for the diagnosis of chronic pancreatitis (e.g., pancreatic insufficiency) because substantial glandular destruction must occur before secretory function is lost. Clinical examination should focus on a careful search for manifestations of hyperlipidemia, nutritional deficiencies, or signs of alcohol abuse. Physical findings include weight loss proportional to the severity of anorexia as well as steatorrhea. Tenderness of the upper abdomen is common. An enlarged pancreas occasionally is palpable, especially in a thin person, but the findings of a mass may indicate the presence of a pseudocyst. Occasional findings include jaundice secondary to stricture of the common bile duct, enlarged spleen secondary to thrombosis of the splenic vein, ascites secondary to a pancreaticoperitoneal fistula, or gastric succussion splash secondary to duodenal obstruction.
 - a. **Abdominal pain**. Upper midepigastic pain radiating to the back is the cardinal symptom and is present in 95% of cases. Early episodes mimic an attack of acute pancreatitis and may last for several hours to days. As the disease progresses, the attacks tend to be more frequent and prolonged.
 - b. **Malabsorption** due to exocrine insufficiency requires destruction of more than 90% of the pancreas parenchyma. This can result in weight loss, steatorrhea, asthenia, metabolic bone disease, clotting dysfunction, and vitamin deficiency, including A, D, E, K, and B₁₂.
 - c. **Steatorrhea** occurs when lipase output by the pancreas is less than 10% of normal.
 - d. **Endocrine deficiency**. Pancreatic endocrine function should always be assessed when there is evidence of exocrine insufficiency. The islets of Langerhans have a greater resistance to injury by inflammation and fibrosis than do the exocrine tissues; most patients who develop diabetes already have pancreatic exocrine insufficiency and steatorrhea. Deficient insulin and glucagon secretion results in brittle diabetes mellitus.
2. **Laboratory tests**
 - a. **Amylase and lipase levels** are elevated in acute pancreatitis but rarely are useful in chronic pancreatitis.
 - b. **Pancreatic stimulation tests** (see [section V](#)).
 - c. **Pancreatic endocrine function**. Fasting and 2-hour postprandial blood glucose levels or glucose tolerance tests may be abnormal in 14–65% of patients with early chronic pancreatitis and in up to 90% of patients when calcifications are present.
 - d. **Fecal fat** (see [section V](#)).
 - e. **Liver function tests** may suggest biliary obstruction from cicatricial narrowing of the lower common bile duct.
3. **Radiology studies**
 - a. **Plain films** of the abdomen show diffuse calcification of the pancreas in approximately 30% of patients with relatively early stages of chronic pancreatitis and in 50–60% of cases of advanced disease.
 - b. **Ultrasonography** is approximately 60% sensitive for the diagnosis of parenchymal or ductal disease.
 - c. **CT scan** is approximately 75% sensitive for diagnosis of parenchymal or ductal disease.
 - d. **ERCP** is 85–90% sensitive. It allows for evaluation of ductal anatomy (see [section II.B.4.a](#)). Brushings and biopsies of suspicious lesions can be performed as well as therapeutic intervention, including stenting and sphincterotomy.
 - e. **Ultrasonography- or CT-guided fine-needle biopsies** may be useful in cases in which chronic pancreatitis cannot be distinguished from pancreatic cancer. By itself, however, a single biopsy finding of inflammatory changes is not sufficient to rule out carcinoma.
4. **Distinguishing between chronic pancreatitis and pancreatic cancer**
 - a. **ERCP signs suggestive of cancer**
 1. **Abrupt complete obstruction of an otherwise normal main pancreatic duct**
 2. **Encasement of a long segment of main pancreatic duct with normal ductal appearances on either side**
 3. **Parenchymal filling of necrotic tumor areas**
 4. **Double-duct sign of invasion of main pancreatic duct and common bile duct by periampullary cancer**
 - b. **Cytology** from pancreatic juice, duodenal aspirate, or fine-needle aspiration (CT-guided, ultrasonography-guided, or ERCP brushings and biopsies). Percutaneous biopsy should be reserved to prove the diagnosis in patients with apparent unresectable disease.
 - c. **Carbohydrate antigen (CA) 19-9** is often elevated in pancreatic cancer, but it rarely detects small tumors and may also be elevated in pancreatitis. This substance is not useful in screening asymptomatic patients. Carcinoembryonic antigen, tissue polypeptide antigen, and CA 125 may be elevated in cystic pancreatic neoplasms but are not useful in periampullary carcinomas.

C. **Complications**

1. **Common bile duct obstruction**

- a. **Etiology** may be transient obstruction from pancreatic inflammation and edema or from stricture of the intrapancreatic common bile duct.
- b. **Strictures**
 1. **Often long and smooth** (2–4 cm in length)
 2. **Three percent to 29% of chronic pancreatitis**

3. **Must be distinguished from pancreatic carcinoma** (see [section II.B.4](#))
 2. **Duodenal obstruction**
 - a. **Mechanisms**
 1. **Acute pancreatic inflammation and edema**
 2. **Chronic fibrotic reaction**
 3. **Pancreatic pseudocyst**
 4. **Pancreatic cancer**
 - b. **Approximately 75% will resolve** with nonsurgical management within 3–4 weeks.
 3. **Pancreatic fistulas** occur by disruption of the pancreatic duct.
 - a. **Pancreatic ascites** is the result of disruption of the pancreatic duct anteriorly.
 - b. **Pancreatic pleural effusion** is the result of disruption of the pancreatic duct posteriorly to the retroperitoneal space and tracking cephalad to the posterior mediastinum and pleural spaces.
 - c. **Pancreaticocentric fistulas** result in draining of a pancreatic abscess cavity or pseudocyst into the stomach, duodenum, transverse colon, or biliary tract. They are often asymptomatic but may become infected or result in hemorrhage.
 - d. **Pseudocyst** (see [section VI.A](#)).
 4. **Splenic vein thrombosis** results in left-sided portal hypertension and gastric varices. It can present with hematemesis, melena, anemia, abdominal pain, or splenomegaly. Splenectomy results in cure in 90% of patients.
 5. **Pancreatic carcinoma.** Chronic pancreatitis has been suggested in some studies to increase the risk of pancreatic carcinoma by a factor of two or three.
- D. **Treatment**
1. **Medical**
 - a. **Malabsorption or steatorrhea** (see [section V](#)). There is some evidence that adequate supplies of oral pancreatic enzyme supplementation can improve pain control.
 - b. **Diabetes** initially can be responsive to careful attention to overall good nutrition and dietary control; however, oral hypoglycemic agents or insulin therapy often are ultimately required. There is some propensity to hypoglycemic attacks. Diabetic ketoacidosis is not commonly seen except after major pancreatic resections.
 - c. **Analgesia.** Narcotics usually are required for pain relief. Meperidine is recommended because it has less spasmodic effect on the sphincter of Oddi than does morphine. Narcotic dependency is a frequent complication of therapy and correlates with higher mortality.
 - d. **Abstinence** from alcohol and withdrawal prophylaxis are of paramount importance.
 - e. **Cholecystokinin antagonists and somatostatin analogues** have been considered for treatment of chronic pancreatitis, but early studies have shown disappointing or only short-lived effects. However, **somatostatin analogues** may be of use in the management of pancreatic fistulas.
 - f. **Tube thoracostomy or repeated paracentesis** may be required for pancreatic pleural effusions or pancreatic ascites. Approximately 40–65% of patients respond to nonsurgical management within 2–3 weeks.
 2. **Surgical**
 - a. **Principles**
 1. **Indications for surgery** include severe intractable pain, multiple relapses, inability to rule out neoplasm, and complications (pseudocyst, obstruction, fistula, infections, and portal hypertension).
 2. **Surgical candidates.** Patients must have a trial of nonsurgical therapy, including abstinence. They must be willing and able to care for themselves after surgery, which may include an apancreatic state.
 3. **Choice of procedure.** Surgical therapy must allow for drainage of the pancreatic duct and resection of the diseased pancreas. Choice of procedure is made based on structural changes found on ERCP, CT scan, and ultrasonography and those found during surgery. Drainage procedures are preferable because they allow for preservation of pancreatic tissue and function but require a dilated pancreatic duct of at least 8 mm.
 - b. **Drainage procedures**
 1. **The Puestow procedure** is longitudinal side-to-side pancreaticojejunostomy. It is indicated in patients with functionally significant strictures along the pancreatic duct (chain of lakes on pancreatograms) and with ducts of at least 8 mm in diameter.
 2. **The Duval procedure** comprises resection of the body and tail of the pancreas with retrograde Roux-en-Y pancreaticojejunostomy. It allows for drainage, antegrade through the ampulla of Vater and retrograde through the Roux limb, and has a lower patency rate than does the Puestow procedure. It is rarely performed except in patients with a single focal stricture secondary to trauma.
 - c. **Pancreatectomy**
 1. **Distal subtotal pancreatectomy (Child's procedure).** This technique is used for disease lateralized to the tail of the gland, cysts in the body and tail, severe disease in the body and tail with ductal obstruction at the neck of the gland, and previous ductal injury from blunt abdominal trauma with fracture of the pancreas and stenosis of the duct at the midbody level.
 2. **Total pancreatectomy** is performed only as a last resort in patients whose previous operations have failed and who appear capable of managing an apancreatic state.
 3. **Pancreaticoduodenectomy (Whipple procedure)** is indicated in cases in which the pancreatitis disproportionately involves the head of the pancreas, the pancreatic duct is of small diameter, or in which cancer cannot be ruled out in the head of the pancreas. For chronic pancreatitis, the pylorus-preserving technique is advocated. The use of vagotomy is controversial. This procedure, although more technically demanding, results in a higher degree of pain relief than does the distal resection.
 - d. **Combined resectional and drainage procedures**
 1. **The Berger procedure** is a duodenum-preserving resection of a portion of the pancreatic head. This operation preserves a small amount of pancreatic tissue within the C-loop of the duodenum and also in front of the portal vein. A jejunal loop is anastomosed to the proximal and the larger distal stumps of the remaining pancreas. This procedure should not be performed in the presence of cancer.
 2. **The Frey procedure** is a longitudinal pancreaticojejunostomy that is performed with excision of peripancreatic duct tissue that is in the region of an inflammatory mass of the pancreas.
 - e. **Celiac plexus block.** Although transiently effective in some cases, nerve ablation procedures have a minimal role in the management of pain in patients with chronic pancreatitis due to poor long-term results.

III. Exocrine pancreatic cancer

A. **Incidence and epidemiology.** Pancreatic cancer is the fifth and most common cause of cancer death in the United States and accounts for approximately 3% of all cancers. It is associated with smoking, benzidine and beta-naphthylamine exposure, alcohol, diet, recent-onset diabetes, chronic pancreatitis, and family history. Approximately 80% of pancreatic carcinomas are ductal cell adenocarcinomas, 4% are giant-cell carcinomas, 3% are adenosquamous carcinomas, 2% are mucinous carcinomas, 1% are cystadenocarcinomas, 1% are acinar cell adenocarcinomas, and the remaining 9% are unclassified. Seventy percent of pancreatic cancers occur at the head, 20% in the body, and 10% in the tail. Other periampullary tumors, such as carcinomas of the distal bile duct, duodenum, and ampulla of Vater, are less common and constitute approximately one-third of resectable periampullary cancers.

B. Diagnosis

1. **History and physical examination**
 - a. **Abdominal pain.** Patients present with a typically progressive midepigastic dull ache that often radiates to the back. It is present in 80% of patients, especially if the tumor involves body or tail of the pancreas.
 - b. **Weight loss** occurs in approximately 70% of patients. Pancreatic cancer must be ruled out in patients older than 50 years who present with vague abdominal pain and weight loss.
 - c. **Obstructive jaundice and pruritus** commonly are associated with cancer of the pancreatic head.
 - d. **Weakness, fatigue, malaise, anorexia, and vague constitutional symptoms** present in 30–40% of patients.
 - e. **Cholangitis** develops in approximately 10% of patients.
 - f. **Courvoisier's sign** (painless jaundice with a palpable gallbladder) usually is associated with pancreatic head tumors, periampullary carcinoma, and primary bile duct tumors.
 - g. **Virchow's node** (left supraclavicular) and **Sister Mary Joseph's node** (umbilical) indicate metastatic disease.
 - h. **Trousseau's migratory thrombophlebitis** is a superficial thrombophlebitis that can be associated with pancreatic cancer.
2. **Laboratory tests.** None is singularly diagnostic for pancreatic cancer.
 - a. **Elevated serum bilirubin**
 - b. **Elevated alkaline phosphatase**
 - c. **Mild elevations of serum glutamic-oxaloacetic transaminase and serum glutamate pyruvate transaminase**
 - d. **Tumor markers** (see [section II.B.4](#))
3. **Radiologic studies**

- a. **Plain films** of the abdomen usually are of little benefit.
 - b. **CT scan.** In periampullary carcinoma, dilatation of intrahepatic and extrahepatic ducts as well as the main pancreatic duct can be seen. Tumors larger than 2 cm generally can be detected.
 - c. **ERCP** (see [section II.B.4.a](#)).
 - d. **Upper gastrointestinal series.** This test can be of use in evaluating patients with duodenal and gastric outlet obstruction.
 - e. **Percutaneous (CT- or ultrasonography-guided) needle biopsies** are a highly accurate method for preoperative diagnosis (sensitivity is 60–90%, and specificity is close to 100%). A negative biopsy does not rule out carcinoma, however, and any biopsy risks seeding the peritoneal cavity. Therefore, most surgeons believe that preoperative tissue diagnosis should be obtained only to avoid diagnostic laparotomy if the disease appears to be unresectable.
- C. **Others.** Ultrasonography is not as sensitive as CT scan and is limited by the presence of bowel gas. MR scan may provide as much information as does CT scan but is costly, is limited in availability, and takes longer. Endoscopic ultrasonography can assess tumor size as well as portal and mesenteric vascular involvement and obtain tissue samples. Staging laparoscopy may be useful in identifying small liver metastasis and peritoneal implants (see [section III.D.2](#)).
- D. **Treatment**
1. **Nonsurgical.** Patients preoperatively found to have unresectable disease or are considered to be poor surgical candidates may be offered palliative nonsurgical treatment. Biliary bypass can be performed by percutaneous drainage or with endoscopic stent placement. Gastric outlet obstruction can be palliated with a percutaneous gastrostomy tube.
 2. **Surgical.** Initially, exploratory laparotomy or laparoscopy is performed to assess whether disease is resectable. We advocate staging laparoscopy with intraoperative Doppler flow ultrasonography and biopsies to assess the primary tumor, local invasion, peritoneal implantation, and metastases to lymph nodes and the liver. Available data suggest that this technique, combined with preoperative spiral CT scanning, can reduce the incidence of unresectable disease at laparotomy to less than 10%.
 - a. **Pancreaticoduodenectomy (Whipple procedure)** consists of *en bloc* resection of the head of the pancreas, distal common bile duct, duodenum, jejunum, and gastric antrum. Pylorus-sparing pancreaticoduodenectomy has been advocated by some, but there are no data demonstrating improved survival or lower morbidity. Some reports demonstrate some benefit of an extended lymphadenectomy, including nodes from the celiac axis to the iliac bifurcation and nodes from the portal vein and superior mesenteric artery. There has been a sharp decline in morbidity and mortality in specialized centers, with a 30-day mortality of less than 5%.
 - b. **Palliative bypass.** If the disease is considered unresectable at the time of surgery, palliative choledochojejunostomy or gastrojejunostomy can be performed to palliate or prevent biliary and gastric outlet obstruction.
 - c. **Postoperative considerations.** Delayed gastric emptying, pancreatic fistula, and wound infections are the three most common complications of the pancreaticoduodenectomy. Up to one-fourth of patients require a nasogastric tube for longer than 10 days, but delayed gastric emptying almost always subsides with conservative treatment. Drains generally are removed when output decreases. Some surgeons prefer to continue with drainage until the patient is eating. Randomized studies have not demonstrated that the perioperative use of octreotide (a somatostatin analogue) can decrease pancreatic fistulas in patients undergoing pancreaticoduodenectomy.
 3. **Radiotherapy and chemotherapy.** The use of adjuvant therapy has been investigated in a prospective randomized trial of surgery alone versus surgery plus adjuvant radiotherapy and chemotherapy; the 2-year actuarial survivals were found to be 18% and 43%, respectively. Patients in the radiotherapy and chemotherapy arm were given a total radiation dose of 40 Gy and 500 mg/m² of 5-fluorouracil (intravenous bolus).
- E. **Prognosis.** Surgical resection can increase survival. Overall 5-year survivals are 5–20% for patients after resection. In patients with small tumors, negative resection margins, and no evidence of nodal metastases, the 5-year survival is as high as 40%. Mean survival for unresectable locally advanced disease is 3–10 months, and for hepatic metastatic disease, survival is 6 months.

IV. Congenital abnormalities

- A. **Pancreatic divisum.** Failure of the ventral and dorsal primordia to fuse results in separation of the ducts of Wirsung and Santorini. The duct of Wirsung may measure no more than 1–2 cm in length, whereas the duct of Santorini becomes the main ductal system and drains via the minor papilla. Pancreas divisum is detected by ERCP, and the incidence is estimated to be 5%. Approximately 25% of affected patients develop pancreatitis secondary to obstruction or stenosis. However, the association between pancreatitis and pancreas divisum is controversial. Sphincteroplasty and cholecystectomy is the procedure of choice for symptomatic patients.
 - B. **Heterotopic (accessory) pancreas.** Typical locations for ectopic pancreatic tissues include the stomach, duodenal or ileal wall, Meckel's diverticulum, and the umbilicus. Less common sites include the colon, appendix, gallbladder, omentum, and mesentery. Most ectopic pancreatic tissue is functional; islet tissue is most often present in the stomach and duodenum. Heterotopic pancreas may result in pyloric stenosis, disruption of peristalsis, peptic ulcers, or neoplasms.
 - C. **Annular pancreas.** Malrotation of the ventral primordium during the fifth week results in a thin flat band of normal pancreatic tissue surrounding the second part of the duodenum. The pancreatic tissue may be completely free from the duodenum or may invade the muscularis deeply. The annular pancreas usually contains a duct that connects to the main pancreatic duct. Annular pancreas may cause duodenal obstruction *in utero*, resulting in hydramnios. Approximately one-half of affected patients do not have symptoms until late in adulthood, however, and complaints usually relate to duodenal obstruction. Treatment of choice is duodenoduodenostomy for symptomatic patients.
- D. **Other common pancreatic anomalies**
1. **Variations of pancreatic ducts**
 2. **Vascular anomalies**
 3. **Aplasia-hypoplasia**
 4. **Pancreatic cysts**
 5. **Cystic fibrosis**

V. Exocrine pancreatic insufficiency

- A. **Etiology**
 1. **Pancreatic disease** (acute and chronic pancreatitis)
 2. **Pancreatic resection**
 3. **Cystic fibrosis**
- B. **Diagnosis**
 1. **History and physical examination**
 - a. **Malabsorption and steatorrhea** are the most common complaints associated with exocrine pancreatic insufficiency.
 - b. **Family history** (particularly pancreatic disease or cystic fibrosis) and social history (particularly alcohol consumption and smoking) are relevant.
 - c. **Clinical examination** should focus on a careful search for the following signs:
 1. **Manifestations of hyperlipidemia**
 2. **Nutritional deficiencies**
 3. **Signs of alcohol abuse**
 2. **Laboratory tests**
 - a. **A 72-hour fecal collection for estimation of daily fecal fat** is the most sensitive and specific stool test for exocrine insufficiency. Fat absorption has the most clinical significance, although fewer problems are associated with protein malabsorption. Carbohydrate malabsorption probably is unimportant. Malabsorption occurs only if more than 90% of pancreatic exocrine function is lost. In an individual ingesting 100 g fat per day, excretion of more than 7 g fat in a 24-hour period suggests pancreatic insufficiency. Neutral fat (Sudan stain) suggests pancreatic disease, whereas split fat suggests small-bowel disease.
 - b. **The secretin-cholecystokinin test** is the most sensitive (80%) but poorly tolerated test. Pancreatic juice from duodenum is collected after hormonal stimulation with secretin and cholecystokinin. Duodenal fluid normally has more than 80 mEq/L HCO₃⁻ and HCO₃⁻ output greater than 15 mEq per 30 minutes.
 - c. **The para-aminobenzoic acid (PABA) excretion (bentiromide) test** is approximately 70% sensitive. Ingested bentiromide is cleaved by chymotrypsin in PABA, which is excreted into the urine. If exocrine pancreatic insufficiency is present, less than 50% of the normal amount of PABA is excreted.
 - d. **Trypsin-like immunoreactivity testing** is approximately 50% sensitive but more specific than the PABA excretion test. Low serum levels of trypsin indicate exocrine pancreatic insufficiency.
 - e. **Pancreolauryl test.** Ingested fluorescein dilaurate is broken down by pancreatic esterase and excreted in the urine. Low levels of urinary fluorescein suggest exocrine insufficiency.
 - f. **Triolein breath test.** Breath concentrations of carbon dioxide 14 are measured after ¹⁴C-triolein is ingested. A peak carbon dioxide 14 concentration of less than 3.5% of the given triolein dose per hour suggests pancreatic insufficiency. The test is rapid and simple.
- C. **Treatment**
 1. **Principles.** Pancreatic enzyme supplements are extracted from animal pancreata and are used to treat malabsorption. Supplements also have been

advocated in alleviation of pain in chronic pancreatitis, but their efficacy is not well established.

2. **Dietary.** Patients should be given a 3,000- to 6,000-calorie, low-fat (<50 g) diet. At least 400 g carbohydrates and 100 g protein are recommended.
3. **Pharmacology.** Pancreatin is available as an enteric-coated tablet (Pancrex V Forte) to prevent the inactivation of the preparation in acid. The coating is designed to dissolve in pH greater than 6 and typically is accomplished in the distal small bowel. Pancreatin also is formulated as microsphere caplets (Pancrease MT, Creon, Nutrizym GR) to prevent the pyloric retention occasionally seen with the tablet forms. This preparation is preferred for most patients because it is approximately 20% more effective than the enteric-coated tablets. In circumstances in which the stomach secretes less acid and empties rapidly (e.g., in partial gastrectomies, vagotomies, and gastroenterostomies), uncoated capsules of pancreatin (Pancrex V and Cotazym) are reasonable choices.
4. **Dosages.** Pancreatin should be taken during each meal rather than before or after eating. The dosages and preparation chosen depend on the diet and the extent of the pancreatic insufficiency. For adults, a starting dosage of one tablet of pancrelipase (Pancrease) with each meal is reasonable, and dosages can be adjusted accordingly until appropriate clinical response is achieved (e.g., control of steatorrhea). In certain circumstances, such as cystic fibrosis, as many as 30 tablets per day may be needed.

VI. Cystic diseases

A. Pancreatic pseudocysts

1. **General considerations.** A true cyst has an epithelial lining and does not communicate with the pancreatic ducts. A pseudocyst has no epithelial lining but communicates with the pancreatic ducts and thus contains elevated pancreatic enzyme concentrations. It is important to distinguish pseudocysts from tumors. An acute pancreatic fluid collection follows in approximately 25% of patients with acute pancreatitis. It is characterized by acute inflammation, cloudy fluid, a poorly defined cyst wall, and necrotic but sterile debris. This may resolve spontaneously, and, in fact, it is likely that most "pseudocysts" that resolve are of this type. By definition, a fluid collection in the first 4 weeks is known as an *acute fluid collection*; after 4 weeks, it becomes an *acute pseudocyst*.
2. **Causes.** Pseudocysts develop after disruption of the pancreatic duct with or without proximal obstruction; they usually occur after an episode of acute pancreatitis. In children, most pseudocysts arise as a complication of blunt abdominal trauma.
3. **Diagnosis**
 - a. **Clinical presentation**
 1. The most common complaint is **recurrent or persistent upper abdominal pain**.
 2. Other symptoms include **nausea, vomiting, early satiety, anorexia, weight loss, and jaundice**.
 3. Physical examination is significant for **upper abdominal tenderness** and occasionally reveals an abdominal mass.
 - b. **Laboratory tests**
 1. **Amylase.** Serum concentrations are elevated in approximately one-half of cases. Urinary levels of amylase are elevated in up to 80% of cases.
 2. **Liver function tests** occasionally are elevated and, therefore, are not of diagnostic use.
 3. **Cystic fluid analysis** is discussed in [section VI.B.2.a.\(1\)](#).
 - c. **Radiologic studies**
 1. **CT scan** is the radiographic study of choice for initial evaluation of pancreatic pseudocysts and is twice as sensitive as ultrasonography in detection of pseudocysts. CT scan findings that determine prognosis include the following:
 - a. **Pseudocysts smaller than 5 cm** usually resolve spontaneously.
 - b. **Pseudocysts with wall calcifications** generally do not resolve.
 - c. **Pseudocysts with thick walls** tend to be resistant to spontaneous resolution.
 2. **Ultrasonography** detects approximately 85% of pseudocysts. The false-positive rate is 8.3%, and the false-negative rate is 8.5%. Its use is limited by obesity and bowel gases, but it may be used in follow-up studies once a pseudocyst has been identified by CT scan.
 3. **ERCP** allows for the determination of pancreatic duct anatomy and influences therapeutic intervention. Approximately one-half of pseudocysts have ductal abnormalities identified by ERCP, such as proximal obstruction, stricture, or communications with the pseudocyst.
4. **Complications**
 - a. **Infection** is reported in 5–20% of pseudocysts.
 - b. **Hemorrhage** results from erosion into surrounding visceral vessels and occurs in approximately 7% of cases. Most common arteries are the splenic (45%), gastrointestinal (18%), and pancreaticoduodenal (18%) arteries.
 - c. **Obstruction.** Compression can occur anywhere from the stomach to the colon. The arteriovenous system also can be subject to compression, including the vena caval and portal venous system. Hydronephrosis can result from obstruction of the ureters. Biliary obstruction can present as jaundice, cholangitis, and biliary cirrhosis.
 - d. **Rupture** occurs in fewer than 3% of cases. Approximately one-half of patients can be treated nonsurgically, with total parenteral nutrition and symptomatic paracentesis or thoracentesis. Rupture is occasionally associated with severe abdominal pain and presents as a surgical emergency.
5. **Treatment** depends on symptoms, age, pseudocyst size, and the presence of complications.
 - a. **Nonoperative.** If the pseudocyst is new, asymptomatic, and without complications, the patient can be followed with serial CT scans or ultrasonography to evaluate size and maturation of the pseudocyst. The majority of pseudocysts larger than 6 cm require surgery.
 - b. **Percutaneous drainage** can be considered for patients in whom the pseudocyst does not communicate with the pancreatic duct (pseudopseudocysts) and for those who cannot tolerate surgery. The overall success rate is 81%, but the procedure may lead to pancreatic fistula at the drain tract. Duration of drainage ranges from several days to several months (usually 3–4 weeks).
 - c. **Excision** can be necessary in patients with symptomatic immature pseudocysts associated with complications or in those with mature distal pancreatic pseudocysts. Resection can require a Whipple procedure (pancreaticoduodenectomy) for pseudocysts at the head of the pancreas (rarely indicated) or distal pancreatectomy for pseudocysts in the body or tail of the pancreas.
 - d. **External drainage** is indicated when the pseudocyst is infected and without a mature wall. External drainage with a catheter usually results in pancreaticocutaneous fistula, which often closes spontaneously. Some authors advocate somatostatin to aid in closure.
 - e. **Internal drainage.** Cyst-enteric drainage is the procedure of choice in uncomplicated pseudocysts requiring surgical treatment. Options include Roux-en-Y cystojejunostomy, loop cystojejunostomy, cystogastrostomy, and cystoduodenostomy. It is always important to obtain a biopsy of the cyst wall to rule out cystic neoplasm. The recurrence rate after these procedures is approximately 10%. There is a slightly higher incidence of recurrence and postoperative complications with the cystogastrostomy than with the Roux-en-Y cystojejunostomy. At our institution, we favor the Roux-en-Y cystojejunostomy in most patients.

B. Pancreatic cysts

1. **Histopathologic classification.** Serous cystic neoplasms tend to have a low malignant potential, as opposed to mucinous tumors, which imply a latent tendency or overt malignancy.
 - a. **Serous cystadenoma** is frequently located at the pancreatic head.
 - b. **Mucinous cystadenoma** is usually located in the pancreatic body or tail.
 - c. **Other cystic neoplasms**
 1. **Cystadenocarcinoma**
 2. **Acinar cell cystadenocarcinoma**
 3. **Cystic choriocarcinoma**
 4. **Cystic teratoma**
 5. **Angiomatous neoplasms**
2. **Diagnosis**
 - a. **Laboratory tests**
 1. **Cystic fluid analysis**
 - a. **Carcinoembryonic antigen** is elevated in mucinous cysts and is low in serous cysts and pseudocysts.
 - b. **CA 125** is elevated in malignant cysts, low in pseudocysts, and variable in mucinous cystic neoplasms and serous cystadenoma.
 - c. **CA 19-9** is nondiscriminatory.
 - d. **Amylase** generally is high in pseudocysts and low in cystic tumors.
 - e. **Lipase** generally is high in pseudocysts and low in cystic tumors.
 - f. **Fluid viscosity** is elevated in mucinous tumors and low in pseudocysts and serous cystic tumors.
 2. **Percutaneous cytology** is useful in determining malignant tumors but limited in discriminating between pseudocysts and serous cystadenoma.
 3. **Percutaneous wall biopsy.** Full-thickness biopsy allows for discrimination between pseudocysts (which lack epithelial lining) and cystic neoplasms. It also can discriminate between serous (cuboidal epithelium) and mucinous (tall columnar epithelium and goblet cells). However, neoplastic cysts may have areas of cyst wall denuded of epithelium, which mimic pseudocysts.
 - b. **Radiographic characteristics of cystic neoplasms**
 1. **Sharply circumscribed and encapsulated**
 2. **Multiple septations and cystic cavities**

3. **Central calcifications with stellate pattern and multiple tiny cysts**, suggesting benign serous cystadenoma
4. **Peripheral calcifications and larger cysts**, suggesting mucinous cystadenoma
5. **Ultrasonography: can reveal mixed echogenic fluid with septations**
6. **Angiography: can show irregular tumor vessels, hypervascularity, and atrioventricular shunting**
3. **Treatment relies on total extirpation.** Extent of resection depends on location and extent of the tumor. Aggressive therapy is warranted even in the presence of metastases. The role of adjuvant therapy has yet to be determined.
4. **Prognosis.** With curative resection, 5-year survival of mucinous cystadenocarcinoma is greater than 60%.
5. **Miscellaneous cystic lesions**
 - a. **True pancreatic cysts are congenital**, not premalignant, and usually are asymptomatic.
 - b. **Hydatid cysts** are managed as with other intraperitoneal hydatid cysts.
 - c. **von Hippel-Lindau syndrome** can involve cystic lesions of the pancreas and nonfunctional islet cell pancreas tumors.

18 Surgical Diseases of the Liver

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Introduction

An elucidation of the internal anatomy of the liver coupled with advances in surgical critical care over the last two decades has made major hepatic surgery safer and more effective. This section reviews conditions affecting the liver that require, or benefit from, surgical intervention including portal hypertension. Discussion of indications for, and techniques of, hepatic transplantation is reserved for a subsequent chapter.

Resection of the Liver

I. Hepatic resection is used to treat a variety of benign or malignant neoplasms and some chronic diseases, such as cystic disorders. Resection of up to 80% of the liver is possible, with long-term recovery, with the remaining liver regenerating rapidly, to a normal liver volume in the noncirrhotic patient. By the third postoperative week, liver function tests, including serum albumin and bilirubin levels as well as the prothrombin time, usually reflect restored physiology.

A. Nomenclature. Although hepatic resections have become more common, more effective, and safer, the nomenclature remains redundant and confusing. In an effort to standardize descriptions, a modified nomenclature for hepatic structures and resections has been introduced by the International Hepato-Pancreato-Biliary Association (*HPE* 2:333, 2000) and embraced by hepatobiliary specialists. This system is predicated on the surgical anatomy of the liver, with the internal hepatic divisions delineated by the arterial and biliary anatomy.

- 1. Internal anatomy: first division.** The liver is divided into two almost equally sized *hemilivers*; the plane between hemilivers is the *midplane* of the liver and runs from the gallbladder fossa to the inferior vena cava. Each hemiliver is supplied one hepatic arterial branch, one bile duct, and one portal vein, referred to as the *right and left bile ducts*, and so forth. A resection of a hemiliver is termed a *hemihepatectomy* or *hepatectomy*; for example, right hepatectomy.
- 2. Internal anatomy: second division.** Further divisions of the liver are based on the internal course of the hepatic artery and bile duct. These structures retain a high order of bilateral symmetry, whereas the portal vein does not. Its asymmetry results from retained portions from the fetal circulation. The liver is thus divided into four nearly equal **sections**: the right anterior and posterior sections and the left medial and lateral sections. A vessel supplying a section is a sectional vessel (e.g., the right anterior sectional artery). The liver resection involving one of these domains is termed a **sectionectomy**.
- 3. Internal anatomy: third division.** The liver is further subdivided into segments numbered I–IX. These are the same as originally described by Couinaud. Resection of a segment is termed a *segmentectomy*.

B. Perioperative management of hepatic resections. Preoperative management involves correction of anemia and coagulopathy, the administration of vitamin K in the presence of jaundice, optimization of nutrition, and administration of proper prophylactic antibiotics. **Operative conduct** for major liver resection consists of wide exposure thorough a bilateral subcostal incision. The vascular pedicles to the side of the liver to be resected are isolated. The same is done for the hepatic vein(s) draining the part of the liver to be transected. During transection, maintenance of a low central venous pressure (<5 mm Hg) and placement of the patient in the Trendelenburg position reduce blood loss. A small amount of positive end-expiratory pressure (5 cm H₂O) is used to prevent air embolism. Vascular control can be augmented by intermittent occlusion of all hepatic inflow (Pringle maneuver) to total vascular occlusion. Postoperatively, hypoglycemia may result in the absence of supplemental glucose, and therefore frequent accuchecks should be obtained. Hyperbilirubinemia is unusual but may occur and persist for days to weeks. Hypoprothrombinemia develops but usually is not critical; if necessary, fresh frozen plasma is infused to keep the International Normalized Ratio (INR) at less than 1.5. Albumin infusion may be necessary to keep the serum albumin above 2 g/dL. The most common complication from liver resection is intraabdominal abscess. Treatment usually consists of percutaneous drainage, although rarely some collections require an operative intervention. Another procedure-specific complication is a bile leak from the cut surface of the liver or a damaged biliary duct. This may manifest as a bile fistula or a *biloma*, a localized collection of bile. This can usually be managed by percutaneous drainage. Liver failure may result from insufficient residual functional hepatic parenchyma after extensive resections.

Diseases of the Liver

I. Liver tumors

A. Benign tumors

- 1. Hamartoma** is normal liver tissue in an abnormal arrangement. It is usually tiny and of no clinical significance. An exception is mesenchymal hamartoma, which can present as a rapidly growing mass in a child and rarely in adults. Hamartomas may be solitary or multiple, with a firm and nodular gross appearance. Typically, they are white lesions 1–5 mm in diameter, but they may be larger. Resection may be indicated based on growth or obstructive symptoms; however, except for mesenchymal hamartomas, this is a very uncommon indication for resection.
- 2. Hemangioma** is the most common benign liver tumor, being reported in up to 7% of autopsies. It is also a form of hamartoma rather than neoplasm. Two forms predominate. The more frequently seen capillary form is often multiple, asymptomatic, and usually of no clinical significance. Cavernous lesions, on the other hand, are generally solitary, large, and often symptomatic. A description of their qualities follows.
 - a. Pathology.** Size ranges from 1 to 2 cm up to the volume of a hemiliver or more. They are spherical, soft, and easily compressible. On sectioning, they are purple in color and spongelike. Microscopically, vascular spaces are lined with flat cuboidal cells and may show evidence of thrombotic congestion. Malignant degeneration does not occur.
 - b. Presentation.** Most hemangiomas are asymptomatic and are identified incidentally. Approximately 60% of those with large lesions are symptomatic, presenting as hepatomegaly, often in conjunction with upper abdominal fullness or penetrating abdominal or pleuritic pain. This discomfort may be positional. Obstructive symptoms [jaundice (rare), early satiety, nausea] reflect compression of other structures. High-output cardiac failure can occur, especially in the pediatric population. These lesions can also cause sequestration of platelets and clotting factors that may result in a consumptive coagulopathy or purpura. Women are more frequently symptomatic (10:1 ratio) due to the larger average size of their lesions on presentation. Hemorrhage from free rupture into the peritoneal cavity is an extremely rare occurrence but can be precipitated by needle biopsy.
 - c. Diagnosis.** Laboratory abnormalities are rare. As these are vascular tumors, diagnostic biopsy is unnecessary and should be discouraged, as it can cause severe and even fatal hemorrhage. Ultrasound is highly sensitive but not specific and can identify an homogenous hyperechoic mass with pathognomonic compressibility. Contrast computed tomography (CT) scans show a low-density area with characteristic peripheral enhancement in the early phase. The specificity of CT scanning has rendered arteriography less useful. The most specific and sensitive tests are MR scan and red cell–tagged radionuclide scan.
 - d. Treatment** consists of observation alone for most hemangiomas. One rare exception is the asymptomatic patient with a large mass who is at significant risk for a traumatic event. The preferred treatment for symptomatic lesions is surgical extirpation. Hemangiomas can usually be enucleated under vascular control (intermittent Pringle maneuver). Formal anatomic resection (e.g., hemihepatectomy) is used when the tumor has largely replaced a distinct anatomic unit. Regression after low-dose radiation therapy or embolization in select cases has been described but should be reserved for large unresectable lesions or for a patient unfit for surgery. In the rare case of spontaneous hemorrhage, control with vascular interventional embolization can temporize until a definitive surgical approach can safely occur after resuscitation.
- 3. Adenoma.** An association with all the synthetic estrogen and progesterone preparations is known (*Toxicol Environ Health* 5:231, 1979). With the introduction of oral contraceptive medication, the incidence of adenomas markedly increased in the 1960s and 1970s but has stabilized since, probably due to modifications of these medications with decreased estrogen contents.
 - a. Pathology.** Grossly, adenomas are soft fleshy tumors with smooth surfaces. Microscopically, they are made up of monotonous sheets of hepatocytes

containing glycogen. Portal triads are absent, and the vessels are thin walled. The lesions are separated from normal liver by a capsule.

- b. **Clinical manifestations.** Most cases present as a mass in the liver, often palpable. Approximately 33% of patients present with intraperitoneal bleeding, and others present with abdominal pain without rupture. Asymptomatic tumors noted either radiographically or at laparotomy are less frequent presentations. Rupture may occur during pregnancy due to rapid growth under the influence of estrogens. These lesions are premalignant.
 - c. **Diagnostic studies.** Liver serology and alpha-fetoprotein (AFP) levels usually are normal. Ultrasonography or CT scan can identify lesions but cannot differentiate an adenoma from a malignant lesion. Some advocate needle biopsy as a method of differentiating benign from malignant lesions, although the accuracy is less than 100%. Technetium sulfur colloid scan is useful for distinguishing adenoma from focal nodular hyperplasia (FNH). The latter registers warm or hot, based on the uptake of sulfur by the abundant Kupffer cells, whereas the adenoma, which has few or no Kupffer cells, is cold. Hemangiomas can be confused with these entities and can be differentiated by a tagged red blood cell scan as previously described.
 - d. **Treatment.** Some small, asymptomatic lesions regress with cessation of oral contraceptives. Lesions that do not regress should be excised unless they are small and centrally located or are multiple and dispersed throughout the liver. Resection is indicated for large, painful, or ruptured lesions or those larger than 5 cm in diameter. An anatomic resection is usually performed, although no normal liver tissue need be taken. Recurrence after resection has not occurred.
4. **Focal nodular hyperplasia (FNH)** represents a reaction to an injury rather than a true neoplasm. Although an association with oral contraceptives has been suggested, the correlations are much lower than are those for adenomas. FNH frequently occurs in women who are not on oral contraceptives, as well as in men.
- a. **Pathology.** The lesions usually are solitary and small and often are peripherally located near the edge of the liver. Histologically, they are composed of fibrous bands and normal liver tissue. The overall appearance resembles a regenerating nodule with a central stellate scar.
 - b. **Clinical manifestations.** The large majority of lesions are found incidentally at laparotomy. Less commonly, patients present with a palpable mass or pain. Hemorrhage is not characteristic and has not been reported.
 - c. **Diagnostic studies.** Liver function tests, AFP levels, and hepatitis B serology usually are normal. The lesions are isodense on ultrasonography and CT examination. Angiography, however, can demonstrate a hypervascular tumor with large arteries and evidence of arteriovenous shunting in 85% of cases. There is a characteristic stellate distribution of the blood vessels and a central scar on CT scan. The lesions are warm or hot on technetium sulfur colloid scan, as previously described.
 - d. **Treatment.** Elective resection is not indicated in asymptomatic patients when studies differentiate FNH from adenoma or malignant lesions. Oral contraceptives should be stopped. There is no contraindication to pregnancy with this lesion.
- B. **Malignant tumors of the liver** are classified as primary epithelial carcinomas [hepatocellular carcinoma (HCC), hepatoblastoma, and cholangiocarcinoma], mesenchymomas, mixed tumors, and sarcomas. Secondary tumors (metastases) are far more prevalent in Western countries.
1. **Hepatoma or HCC**
 - a. **Incidence.** There are wide geographic variations in the incidence of HCC. The annual incidence in the United States is approximately 2–4 per 100,000 population, with the tumor representing fewer than 2% of all malignancies. In contrast, some areas in Africa and Asia have incidences as high as 60 per 100,000 population. Although HCC occurs in all age groups, it is diagnosed mainly in the fifth and sixth decades. In Africans and Asians, however, the peak age is between 20 and 40 years. There is a 3:1 male predominance.
 - b. **Etiology.** A variety of etiologic factors have been implicated. Because almost every type of experimentally induced cirrhosis transforms into carcinoma, a strong association exists between cirrhosis and the development of primary hepatic carcinoma. Malignant tumors of the liver occur in 4.5% of cirrhotic patients and in up to 10% when hemochromatosis is the inciting factor. Conversely, approximately 70–85% of primary HCC arises in the setting of cirrhosis.
 - c. **Viral hepatitis** has a strong correlation with HCC. Serologic markers for hepatitis B virus, or antibody to hepatitis C virus, are detected in almost all patients with HCC in endemic areas. The question remains as to whether the viruses themselves are oncogenic or whether they lead to chronic active hepatitis with ultimate progression to cirrhosis and, finally, cancer. The evidence for the association between hepatitis C and HCC, although weaker, appears to be established, and is germane given that infection with hepatitis C is now more common than with hepatitis B. Hepatitis C viral RNA has been demonstrated within HCC tumor extracts. Other etiologic factors that have been implicated include the aflatoxin of the mold *Aspergillus flavus*, parasite infestation with the liver fluke *Clonorchis sinensis*, polyvinylchloride, anabolic steroids, and metabolic disorders, such as alpha₁-antitrypsin deficiency and hemochromatosis.
 - d. **Pathology.** Gross patterns of HCC include nodular (aggregate of clusters of nodules), massive (single large mass), and diffuse (widespread fine nodular pattern) types. The right hemiliver is involved more frequently than the left, except for the fibrolamellar variant, of which 75% of reported cases have been in the left lobe. Microscopically, the formation of giant cells is a feature of HCC. HCC cells frequently invade venous branches, causing vascular dilatation, which contributes to the nodular appearance of the liver. Fibrolamellar carcinoma has a distinctive feature of abundant fibrous stroma arranged in thin parallel bands.
 - e. **Clinical manifestations are vague.** Eighty percent of patients experience weight loss and weakness. Approximately 50% (75% with fibrolamellar carcinoma) present with abdominal pain that usually is dull, persistent, and in the epigastrium or right upper quadrant. Acute severe abdominal pain has been associated with intraperitoneal hemorrhage due to rupture of a necrotic nodule or erosion of a blood vessel. This occurs infrequently (10% of patients) but is often lethal.

Physical examination may reveal an enlarged but nontender liver. Splenomegaly as a result of portal hypertension may be present. Ascites is present in nearly half the patients but usually is mild. As it is often difficult to distinguish HCC from nodular areas of cirrhosis, a clinical picture with rapid acceleration of symptoms suggests carcinoma. For instance, hypoglycemic intervals or amelioration of preexisting diabetes also suggests superimposed HCC.

f. **Diagnostic studies**

1. **Laboratory.** HCC usually, but not always, is associated with increases in alkaline phosphatase levels as well as elevations of transaminases and 5'-nucleotidase. Serum bilirubin usually is normal. AFP appears in the serum and is elevated in 75% of affected African patients but in only 30% of patients in the United States.
2. **Radiologic studies**
 - a. **Ultrasonography** is noninvasive and relatively inexpensive and can be highly accurate in the detection of HCC. The diagnostic accuracy is enhanced when ultrasound findings are coupled with concomitant AFP elevations.
 - b. **CT scan** with contrast medium is useful in localizing HCC. Its sensitivity is enhanced with lipiodol (a lipid lymphographic agent that is selectively retained in HCC) when this is injected 2 hours before the scan.
 - c. **MR scan** has the advantages of CT scan and can also delineate vascular anatomy with precision.
3. **Needle biopsy.** Laparoscopic or percutaneous biopsies are techniques for obtaining a tissue diagnosis. Normal coagulation parameters and a platelet count should be assured before percutaneous biopsy. A tissue diagnosis is not required before an attempt at surgical extirpation if other diagnostic modalities favor HCC as the diagnosis. Biopsy may be required by the medical oncologist in the setting of advanced disease to guide chemotherapy.
- g. **Treatment.** The only curative therapy is surgical resection. The size and location of the HCC dictate the extent of hepatectomy. A macroscopic margin of 1 cm generally is regarded as adequate, although this may vary according to the size of the lesion. In some cases, resection entails a hemihepatectomy, although lesser liver-sparing segmental "anatomic" resections are preferred and have satisfactory results (*JACS* 187:471, 1998) This is especially true in cases in which cirrhosis, increased vascularity, and associated morbidities pose a prohibitive risk for a major hepatic resection. Repeat hepatic resection for recurrence has been demonstrated to be safe and effective in selected lesions.
 1. **Liver transplantation** is an option in HCC that is unresectable by standard methods. Strict criteria for number and size of tumors exist in most centers. Donor shortage with advancement of disease while on the waiting list, a high recurrence rate, reactivation of hepatitis, and enhancement of tumor growth by immunosuppressive therapy have hampered wide application of this modality.
 2. **Neither radiotherapy nor chemotherapy is curative.** The combination of chemotherapy and surgery is currently being studied. Unresectable lesions or recurrences have been palliated with ablative techniques and are also used for metastatic tumors as described in the following section.
 3. Ablative therapies are effective treatment for small HCCs and are the treatment of choice in patients who are not candidates for surgery because of advanced cirrhosis or other morbidities. **Percutaneous ethanol injection** has been reported to produce as good results as liver resection in lesions less than 3 cm. Ethanol is injected into the tumor in one or staged procedures depending on the size of the tumor. **Transarterial chemoembolization** combines devascularization of the tumor with administration of chemotherapy and is a competing therapy to percutaneous ethanol injection. **Radiofrequency ablation (RFA)** places RF electrodes in tumors and destroys them by heating the tumors to 60–100 °C. It is the newest modality and can also be performed percutaneously. All of the ablative modalities have been used to treat tumors in patients who are on transplantation waiting lists.
- h. **Prognosis.** Unresected HCC has a very poor prognosis. Patients rarely survive beyond 4 months after diagnosis. Five-year survival after curative resection is 35–50%. The fibrolamellar variant has a more favorable prognosis. However, intrahepatic recurrence is common, occurring after 60–70% of resections. Indicators of poor outcomes include larger tumors, vascular invasion, and serum AFP levels greater than 2,000. In addition, resection in the setting of cirrhosis also demonstrates decreased long-term survival when compared to noncirrhotics (*Ann Surg* 229:790, 1999). Liver transplantation for extensive disease has achieved similar 5-year survival rates but may have a much higher cure rate for smaller tumors.

2. **Metastatic neoplasms.** Metastatic disease of the liver is almost 20 times more common than primary carcinoma in the United States. Colorectal primaries frequently present with metastases confined primarily within the liver. The metastatic lesion is usually more aggressive with accelerated growth kinetics than its extrahepatic primary. Operative resection is the only potentially curative approach but is only applicable in roughly 25% of patients who present with liver metastases. There have been successful curative extirpations of other metastatic lesions of breast, neuroendocrine, and melanoma derivation, although indications for such operations are limited.
 - a. **Clinical manifestations.** Most patients are asymptomatic; however, with advanced disease, symptoms include fatigue, anorexia, weight loss, dull pain, fullness in the epigastrium or a palpable mass, ascites, jaundice, and fever. With a carcinoid primary tumor, liver metastases are responsible for the carcinoid “flushing” syndrome, as serotonin byproducts are excreted directly into the systemic circulation from the liver without hepatic degradation.
 - b. **Diagnostic studies.** Although elevations in the serum alkaline phosphatase, gamma-glutamyl transpeptidase, lactate dehydrogenase, serum glutamic oxaloacetic transaminase, and 5'-nucleotidase levels may occur, they are neither sensitive nor specific for liver metastases. Serum AFP should be assessed to rule out a hepatoma and is usually negative. In a patient with colorectal cancer, a **carcinoembryonic antigen (CEA) level** in excess of 5 ng/mL is the most sensitive blood test for detecting metastases. An elevation should prompt an abdominal CT scan, or preferably a **positron emission tomogram (PET)**, for more precise staging.
 1. **CT scan** with arterial contrast enhancement is a sensitive preoperative imaging study, although T1-weighted MR scan is comparable. However, small surface lesions frequently are missed with all preoperative imaging studies. Subsequently, intraoperative ultrasonography with palpation is the most sensitive detector of liver metastasis.
 2. **Positron emission tomography** is the most sensitive test for liver metastases as well as extrahepatic metastases. It often detects lesions in patients with elevated CEA and normal CT scan. It is based on the principle that metabolically active tumor cells have an increased avidity for glucose. Fluorodeoxyglucose is taken up by tumor cells but not metabolized so that it accumulates in them. This glucose analogue-linked positron emitter is injected, and uptake is measured by nuclear scintigraphy. This technique is also extremely valuable in determining if tumor has recurred at the primary site, a condition that would preclude metastasectomy. Higher rates of resection and short-term survival have been reported following screening with this modality (*Ann Surg* 233:293, 2001).
 - c. **Treatment of colorectal metastases.** Three conditions must be met for consideration of curative resection. First, control of the primary disease must be assured. Second, there should be no evidence of systemic disease. Third, the metastatic lesion must be suitable for local hepatic resection. When local factors include fewer than four tumors, and a 1-cm margin can safely be obtained, a formal, anatomic liver resection should be performed. Outcomes are not as good if these parameters are not adhered to. For small solitary lesions that can safely be removed, a wedge resection rather than a formal hepatectomy should be performed. The survival rates for adequate wedge resections and hepatic lobectomies are comparable.
 1. **Timing of resection** is variable. If, during an uncomplicated initial colon resection, disease amenable to a small wedge resection or a resection of segments II and III is identified, it can be performed without added morbidity. However, in lesions requiring more substantial resections, these are often performed after recovery from the primary operation. There is a trend toward performing both procedures simultaneously.
 2. **Hepatic resection** for colorectal cancer, when the preceding criteria are met, has a 30–35% 5-year survival rate and a 20% 10-year survival rate. Repeat hepatic resection for recurrent disease is safe and may be indicated in some patients, yielding similar results to those of initial hepatic resection procedures. Adjuvant chemotherapy after liver resection is actively under investigation.
 3. Because the primary site of failure following hepatic resection for this disease is the liver itself, efforts are necessary to control microscopic disease regionally. There is renewed enthusiasm for the use of regional chemotherapy via **hepatic arterial infusion devices** in the setting of metastatic colorectal disease. A recent well-designed, randomized prospective study demonstrated better 2-year survival and decreased hepatic recurrence rates after local hepatic infusion of chemotherapy and systemic therapy than after systemic treatment alone (*N Engl J Med* 341:2039, 1999).
 4. The ablative technologies, **cryotherapy** and **RFA**, have also been used to treat unresectable metastatic tumors. RFA is a much more convenient therapy and associated with fewer postoperative complications. There is great interest in this modality, but its effectiveness is unproven.

II. Hepatic abscess. Liver abscesses may originate from bacterial, parasitic, or fungal pathogens. Bacterial abscesses predominate in the United States, whereas amebic (parasitic) abscesses are more common in younger age groups and in endemic areas.

A. Pyogenic abscesses in the liver

1. **Etiology.** Pyogenic liver abscesses generally occur secondary to other sources of bacterial sepsis in the abdomen, the two main sources from biliary and portal vein sepsis. Direct spread from biliary infections, such as empyema of the gallbladder; from cholangitis; or after biliary tract surgery or instrumentation is uncommon but may be life threatening. Bacterial abscesses from portal venous spread of infection from appendicitis, diverticulitis, or other enteric sources also are uncommon and may be difficult to diagnose. “Cryptogenic” hepatic abscesses have no identified primary infection and today are the most common abscesses. Other sources for hepatic contamination are distant infections, such as pneumonia, upper urinary tract infection, or endocarditis. Other rare bacterial liver abscess etiologies include superinfection of amebic or hydatid cyst lesions in the liver. The abscesses are solitary in approximately half of cases and curiously occupy the right hemiliver more frequently.
2. **Diagnosis** depends on clinical suspicion and is often delayed. Constitutional **symptoms** include fever, malaise, rigors, and weight loss. **Signs** include right upper quadrant or right costovertebral angle tenderness and a consistent, intermittent spiking fever. Jaundice is uncommon in the presence of the primary abscess, although it may accompany the associated primary biliary tract focus. The signs and symptoms typically reflect the nature of the primary bacterial source rather than the hepatic abscess. Useful imaging studies include ultrasonography or CT examination of the liver.
3. **Treatment** of bacterial liver abscess includes systemic antibiotic therapy and percutaneous drainage of the abscess cavity. The drain is removed when the cavity has collapsed. Drainage may be impossible, particularly if there are multiple small abscesses. This specific group represents a disproportionate percentage of the mortality associated with liver abscess. Operative drainage is used either when the patient requires laparotomy for management of his or her primary disease, such as appendicitis or cholecystitis, or when abscess drainage is not technically feasible via percutaneous techniques. Empiric antibiotic treatment should include coverage for bowel flora (gram negatives and anaerobic bacteria); once an isolate has been identified, appropriate treatment should be modified for the identified pathogens. An aggressive approach to antibiotic therapy should continue for at least 1 week beyond the resolution of abscesses that can be imaged and beyond any overt evidence of systemic illness.

B. Amebic abscess should be considered in every case of solitary hepatic abscess.

1. **Etiology.** Amebic abscess is caused by the organism *Entamoeba histolytica*, which travels from the bowel to the liver via the portal venous system. The abscesses are most frequently solitary in the right hemiliver, and the contents are often necrotic, with a pasty consistency. The most concentrated area of amebic infiltration is at the edges of the abscess cavity.
2. **Signs and symptoms** of amebic abscess are similar to those of pyogenic abscess but may appear more indolent. Patients less frequently have chills, and symptoms may go on for a longer period before detection. Serologic tests for amebic infestation are positive in nearly 100% of affected patients. Again, the imaging modalities that are most useful are CT scan and ultrasound of the liver.
3. **Management** requires systemic metronidazole (750 mg p.o. t.i.d. or 500 mg i.v. every 6 hours for 14 days). If there is no improvement in clinical systems within 48 hours of beginning antibiotics, a complicated amebic abscess must be considered.
4. **Complications** can include bacterial superinfection, erosion into surrounding structures, or free rupture into the peritoneal cavity. Percutaneous or operative drainage may then be necessary. Although, mortality is infrequent in uncomplicated cases, these complications carry a considerable mortality (as high as 40%).

III. Hepatic cysts can be divided into nonparasitic cysts and echinococcal cysts.

A. Nonparasitic cysts generally are benign. They can be solitary or multiple and typically are small and asymptomatic, often identified on imaging for other issues. Asymptomatic cysts require no treatment regardless of size. Large cysts may be symptomatic because of increased abdominal girth or compression of adjacent structures; there may be vague pain. Bleeding, infection, or obstructive jaundice can occur but are infrequent. Such cysts can be unroofed operatively by either an open approach or, more recently, by laparoscopy. Infected cysts are treated in a similar manner to hepatic abscesses. If the cyst contains bile, communication with the biliary tree is assumed. It should be excised or drained with the closure of the biliary communication. Alternatively, a Roux-en-Y cyst-jejunostomy can be created for internal drainage.

Polycystic kidney disease sometimes is accompanied by **polycystic liver disease**, which usually is asymptomatic. If symptomatic, the symptoms generally are attributable to hepatomegaly from numerous cysts. Liver function is rarely impaired by the gross displacement of parenchyma by these massive cystic cavities. Symptomatic polycystic liver disease has been treated by drainage of the superficial cyst into the abdominal cavity with fenestration of deeper cysts into the superficial cyst cavities. Liver resection with retention of the least cystic areas of hepatic parenchyma is more effective. Malignant cystic lesions, cystadenoma or cystadenocarcinoma, can rarely occur in the liver. These lesions are distinguished from simple cysts by the presence of a mass or septa. They are treated by resection.

B. Echinococcal cysts are the most common hepatic cystic lesions in areas outside the United States. Approximately 80% of hydatid cysts are single and in the right hemiliver. The most common presenting symptoms and signs are right upper quadrant abdominal pain and palpable hepatomegaly. Imaging by nuclear

medicine scan, ultrasonography, or CT scan can demonstrate the abnormality. The cyst must not be aspirated because aspiration can cause spillage of the organisms and spread the disease throughout the abdominal compartment. Useful laboratory tests might include liver function tests, the results of which may be mildly elevated. In addition, a peripheral eosinophilia is often detected. Serologic tests include indirect hemagglutination and Casoni's skin test, each of which is 85% sensitive.

Treatment is primarily operative. Operation is required for symptomatic and asymptomatic cysts, unless the asymptomatic cyst wall is completely circumferentially calcified. Small superficial or pendulous cysts may be excised. Larger cysts should be unroofed and the contents removed piecemeal. The abdomen is explored, and before unroofing, the cyst is isolated and excluded from the peritoneal cavity by sewing a sterile i.v. bag directly to the cyst wall peripherally and then exteriorizing it from the abdominal wound. Next, the cavity is aspirated with a closed system. If there is no evidence of bile in the cyst aspirate, a scolecolidal agent is instilled into the cavity. Choices include hypertonic saline, 80% alcohol, or 0.5% cetrimide. A second cycle is performed after 5 minutes. In patients with biliary communication, only hypertonic sodium chloride (10–20%) or 0.5% sodium hypochlorite should be used as a scolecolidal agent because other agents may damage communicating bile ducts. The cavity is then managed by external drainage or, more frequently, by obliteration by either closing the liver parenchyma or by packing with omentum. Pericystectomy is excision of the cyst in the plane between the host pericyst and liver tissue. The advantage of this technique is that there is no residual cavity. It can be a bloody procedure, but it is becoming the procedure of choice in endemic areas where these lesions are more frequently encountered. Formal right or left hepatectomy is another alternative, particularly for larger multiple cysts, but is rarely necessary. For those cysts with demonstrated communication with a biliary radical, drainage should be achieved with a Roux-en-Y hepaticoenterostomy. Postoperative therapy with praziquantel, mebendazole, or albendazole has been advocated to prevent recurrence. In patients with disseminated echinococcosis or in those with systemic illnesses that prevent operative intervention, these agents are the cornerstone of therapy.

IV. Portal hypertension is the sequela from either pre-, intra-, or posthepatic mechanical obstruction of the portal venous system.

A. The **most common cause of portal hypertension** in the United States is *intrahepatic* obstruction of portal venous flow from cirrhosis (most commonly due to alcoholic, but also nutritional, postnecrotic, or biliary, causes); other intrahepatic portal venous obstruction can be due to hepatic fibrosis from hemochromatosis, Wilson's disease, or congenital fibrosis. *Prehepatic* portal venous obstruction due to congenital atresia or portal vein thrombosis is far less common. *Posthepatic* obstruction (Budd-Chiari syndrome) is associated with lymphoreticular malignancy or a hypercoagulable state such as polycythemia vera. The prognosis is poor; side-to-side portacaval shunt, mesoatrial shunt (if the inferior vena cava is obstructed), or transplantation is recommended if no primary malignancy can be identified. The remainder of this section focuses on the management of portal hypertension caused by intrahepatic portal vein obstruction, primarily cirrhosis.

B. **The manifestations of liver disease with portal hypertension result from hepatic insufficiency and the mechanical effects of portal venous hypertension.**

1. **Hepatic insufficiency may result in hepatic encephalopathy**, which is a syndrome of disordered consciousness in patients with decreased hepatocellular function. The specific toxin that causes the encephalopathy has not yet been delineated. Potential agents include multiple toxins that normally are cleared by the liver, such as ammonia, gamma-aminobutyric acid, mercaptans, and short-chain fatty acids. Factors associated with progressive hepatic encephalopathy can include volume shifts due to medical treatment or renal failure, the use of medications that act on the central nervous system (such as benzodiazepines or opioids), protein load due to hemorrhage of blood in the GI tract, high dietary protein intake, worsening hepatocellular function, or following the creation of central portosystemic shunts.
2. **Manifestations of increased pressure** in the portal vein include increased blood flow through collateral venous beds and increased interstitial tissue hydrostatic pressure. The latter causes ascites. The collateral venous beds, which develop in cirrhosis, bypass the liver by connecting the portal circulation to the systemic circulation. The most clinically significant sites are those at the gastroesophageal junction that connect the left gastric vein (a part of the portal circulation) to the esophageal veins (systemic circulation), resulting in esophageal and gastric varices. Other common collaterals develop when a recanalized umbilical vein collateralizes to the abdominal wall veins or a superior hemorrhoidal vein collateralizes to middle and inferior hemorrhoidal veins, or they develop via the veins of Retzius, numerous small veins in the retroperitoneum that connect the retroperitoneal viscera to the posterior abdominal wall. Left-sided (**sinistral**) portal hypertension can be caused by isolated splenic vein thrombosis. This is most often caused by adjacent pancreatitis. Thrombosis results in increased pressure in the splenic vein at the distal end of the pancreas and the development of collaterals through the short gastric vessels and gastric mucosa back to the liver, thus bypassing the thrombosed splenic vein segment. This segmental area of portal hypertension typically causes gastric varices without esophageal varices. Cure is achieved by splenectomy.
3. **Mechanisms of ascites and edema** are salt and water retention by the kidneys, decreased plasma oncotic pressure, and increased lymphatic flow from increased portal venous hydrostatic pressure. Although the ascites can be massive, they are rarely life threatening unless complications, such as umbilical hernia with erosion, infection and ascitic leak, respiratory embarrassment, or spontaneous bacterial peritonitis, occur. Spontaneous bacterial peritonitis presents as abdominal pain, fever, ileus, and worsening encephalopathy; however, the signs are subtle, and diagnosis may be difficult. Diagnosis is made by paracentesis and is likely when ascitic fluid contains more than 250 polymorphonuclear leukocytes/ μL and if a single organism is cultured. Bacterial cultures should be collected in blood culture bottles; the most common organisms are *Escherichia coli*, pneumococci, and streptococci.

C. **Management of portal hypertension**

1. **Elective reduction of portal venous pressure** can be accomplished by a variety of venous shunting procedures or hepatic replacement with liver transplantation. The objective of these procedures is to reduce the pressure in the portal vein and thus decrease the flow through collateral venous beds. In addition, central shunts are effective in reducing ascites. Excellent results have been reported over the last decade with the use of surgical shunts in patients with preserved hepatic function (Childs' A or B). Liver transplantation is the only treatment that addresses and reverses the primary defect of increased hepatic resistance to portal venous flow. This modality is preserved for those patients with chronic or acute hepatic failure.
 - a. **Liver transplantation** should be considered for any patient with cirrhosis who has survived an episode of variceal hemorrhage and who has manifestations of end-stage liver disease (see [Chapter 29](#)). Patients who are likely candidates for liver transplantation probably should not undergo elective portosystemic shunt operations, if possible, because such shunts may make subsequent liver transplantation more technically demanding.
 - b. **Total portosystemic shunt** operations include the end-to-side, side-to-side, portacaval, mesocaval, and central splenorenal shunts. The end-to-side operation is the prototype and is the simplest portacaval shunt to perform. It involves disconnection of the portal venous circulation from the liver and connection of the intestinal end of the portal vein to the inferior vena cava. This bypasses all the portal circulation from the liver to the systemic circulation, thus decompressing any collateral venous beds. The other variations of this operation include a side-to-side portacaval anastomosis as well as anastomoses at sites more distant from the liver, such as mesocaval H-graft and central splenorenal shunts. The physiologic effects of all these shunts are similar because flow through the hepatic limb of the portosystemic shunt is nearly always away from the liver and toward the anastomosis. Side-to-side or mesoatrial shunts are required in patients with Budd-Chiari syndrome to decompress the hepatic limb of the portal vein. If a shunt is constructed, immediate and permanent protection from visceral bleeding occurs as the venous collateral beds are decompressed. The primary complication of these operations is hepatic encephalopathy due to reduced hepatic blood flow. Attempts have been made to construct smaller side-to-side grafts to partly maintain hepatic flow and reduce the incidence of encephalopathy.
 - c. **Selective shunts** were developed to decrease the pressure in a portion of the portal circulation while disconnecting from the remainder of the portal circulation. The classic procedure is the distal splenorenal (Warren) shunt, which involves dividing the splenic vein and anastomosing the splenic end of the vein to the left renal vein. The left gastric vein, right gastroepiploic vein, and veins in the splenocolic ligament are then all divided. This excludes the gastrosplenic portal circulation from the remainder of the portal circulation and selectively decompresses the gastroesophageal varices. This operation is more difficult and time consuming than the total shunt procedures, and it is appropriate only for elective treatment of patients with gastroesophageal varices and a history of bleeding. It is contraindicated in patients with ascites that cannot be controlled medically. Hepatic encephalopathy occurs less commonly with this approach. Over time, the shunt tends to become less selective as alternative collateral pathways develop that reconnect the main portal venous circulation to the gastrosplenic portal venous system; varices may redevelop at that time. Another variation of a selective shunt that is less widely adopted diverts flow from the left gastric vein to the inferior vena cava by autogenous saphenous vein graft (Inokuchi shunt).
 - d. **The Sugiura procedure** may occasionally be used in patients with recalcitrant variceal hemorrhage. In this procedure, the spleen is removed, the stomach and lower esophagus are devascularized, and the esophagus is transected and reanastomosed with a circular stapling device to disrupt the esophageal venous plexus. This procedure is used uncommonly in the United States but is popular in the Far East.
 - e. The development of **transjugular intrahepatic portosystemic stent-shunts (TIPSS)** provides a nonsurgical alternative in the treatment of portal hypertension. TIPSS is a minimally invasive alternative to open surgical procedures that can temporarily and effectively reduce portal hypertension. It involves the intrahepatic placement of an expandable metallic stent between branches of the hepatic and portal venous circulation. Technical success rates approach 95%, with short-term success in controlling acute variceal hemorrhage observed in more than 80% of patients. Although TIPSS is clearly indicated in acute variceal bleeding that cannot be successfully managed with standard medical therapy (see the following section), TIPSS stenoses and occlusions limit the long-term usefulness of this modality for the elective treatment of patients with well-preserved liver function. Some reports indicate that the primary 1-year patency rates for TIPSS are as low as 25–66%, whereas long-term patency rates are excellent for surgical shunts (approaching 100%).
2. **Acute management of bleeding esophageal varices** is a complex problem that requires multidisciplinary support of the bleeding patient. Because up to

one-third of patients affected with bleeding esophageal varices die during the initial hospitalization for GI bleeding, an aggressive approach is required. Many experience a warning or “herald” episode of lesser severity before life-threatening exsanguination. Therefore, all patients with known or suspected esophageal varices and active GI bleeding should immediately be admitted to the intensive care unit for resuscitation and monitoring. Endotracheal intubation to protect the airway, prevent aspiration, and facilitate the safe performance of endoscopy and other procedures is nearly always indicated. Vascular access, including short, large-bore peripheral lines, and invasive monitoring of central venous pressure and arterial pressure should be achieved. Urinary bladder catheterization and initial volume resuscitation should ensue. Then all patients should have emergent upper endoscopy to document the source of hemorrhage. Because up to 50% of patients with known esophageal varices have upper GI hemorrhage from an alternative source such as gastric or duodenal ulcer, a thorough endoscopy is required. Once varices are identified as the source, several therapeutic options exist and should be implemented.

- a. **Endoscopic sclerotherapy or banding** is performed at the patient's bedside in the intensive care unit at initial endoscopy. Sclerotherapy can be effective in controlling primary hemorrhage and obliterating varices after primary hemorrhage is controlled. Recurrent bleeding occurs in as many as 50% of patients. Although endoscopic sclerotherapy is widely accepted as the procedure of choice for the management of bleeding esophageal varices, new endoscopic treatment modalities have emerged. Endoscopic variceal ligation (banding) is equally effective in controlling active variceal bleeding and is associated with fewer complications and a lower rebleeding rate than endoscopic sclerotherapy. In addition, intravariceal injection of cyanoacrylate (a substance that undergoes instantaneous polymerization after injection) may effectively complement traditional sclerotherapy.
 - b. **Intravenous vasopressin** can be used simultaneously with other treatments. It can decrease flow through the esophageal varices and control hemorrhage but is less effective than sclerotherapy. The standard dosage is 100 units of vasopressin in 250 mL D5W at 0.3 units per minute initially, increasing up to a maximum dosage of 0.9 units per minute to control bleeding. The significant side effects of vasopressin include myocardial, mesenteric, or cutaneous ischemia due to arterial constriction. This severely limits the use of vasopressin in patients with concomitant vascular or coronary artery disease. Some controlled trials have demonstrated that nitroglycerin vasodilators reduce the systemic side effects of vasopressin and should therefore be used in combination with this agent. Newer alternatives include terlipressin (Glypressin), a synthetic analogue of vasopressin that appears to be equally effective but with fewer side effects, and octreotide (a long-acting analogue of somatostatin), which reduces splanchnic blood flow without inducing systemic changes.
 - c. **Balloon tamponade** is useful for temporizing variceal bleeding while more definitive therapy is planned. The specially designed balloon catheters include the Sengstaken-Blakemore tube, the Minnesota tube, and the Linton tube. Each has a gastric balloon; the Sengstaken-Blakemore and Minnesota tubes also have an esophageal balloon. For safe and effective use of these devices, the balloons must be carefully placed according to the manufacturer's directions. The position of the gastric balloon in the stomach must always be confirmed by X-ray before inflation because balloon inflation in the esophagus can be disastrous. Balloon pressure must be maintained as directed by the manufacturer to avoid the complications of mucosal ulceration and necrosis. This is not definitive therapy and should be used only as a temporizing measure.
 - d. **TIPSS** can be used in the acute management of patients with variceal bleeding. Once the patient has been stabilized enough to travel to an interventional radiology suite, the TIPSS procedure may provide acute decompression of the esophageal varices and thus control of bleeding that has been recalcitrant to endoscopic sclerotherapy. This may be particularly useful as a bridge to transplantation for appropriate candidates.
 - e. **Emergency portacaval shunt** generally is reserved for patients in whom other measures fail. This operation carries significant in-hospital mortality and risk of hepatic encephalopathy, particularly because it is used in the group of patients that has failed on other treatments and required multiple blood transfusions, often due to poor hepatic reserve. Only the technically simpler central portacaval shunts (end-to-side or side-to-side) should be used in the emergency setting because the other shunts require more dissection and operative time. Portacaval shunting gives better results when used as the initial treatment in all patients without preliminary nonsurgical management and multiple transfusions. Excellent results have been obtained in some centers in the setting of alcoholic hepatitis/cirrhosis with bleeding. However, most centers do not manage bleeding patients in this manner primarily.
 - f. **After acute variceal hemorrhage is controlled**, the patient should be treated with chronic esophageal variceal sclerotherapy/banding. After initial control has been achieved, a second session is scheduled for 7–10 days later. A third treatment is scheduled approximately 1 month after the initial bleed and then at 3-month intervals thereafter until all varices are eliminated. This treatment regimen can decrease the rebleeding rate from 80% to 50% and has a similar 25% reduction in the overall mortality in randomized trials. Patients who fail on chronic sclerotherapy because of rebleeding should be considered for elective shunting procedures or liver transplantation, depending on their degree of hepatic reserve.
3. **Management of ascites** must be cautious and gradual to avoid sudden changes in systemic volume status, which can precipitate hepatic encephalopathy, renal failure, or death.
 - a. **Salt restriction** is the initial treatment measure. Sodium intake should be limited to 1,000 mg per day. Stricter limitations are unpalatable, and most patients are not compliant.
 - b. **Diuretic therapy** should be gradually applied in patients in whom ascites is not controlled by salt restriction. Weight loss should rarely be more than 500 g per day to avoid significant side effects. Spironolactone is the initial diuretic of choice at 25 mg p.o. b.i.d. This dosage may be increased to a maximum of 150 mg p.o. q.i.d. Furosemide (20 mg per day p.o. initially) may be added if spironolactone fails to initiate diuresis. Volume status must be monitored closely by daily weight check and frequent examinations during initial furosemide treatment.
 - c. **Paracentesis** is useful in the initial evaluation of ascites, when spontaneous bacterial peritonitis is included in the differential diagnosis, and to provide an acute decompression of tense ascites. Up to 5 L ascites can be removed safely if the patient has peripheral edema, the fluid is removed over 30–90 minutes, and oral fluid restriction is instituted to avoid hyponatremia. Paracentesis can be used to provide acute relief of symptoms of tense ascites, including respiratory compromise, impending peritoneal rupture through an ulcerated umbilical hernia, or severe abdominal discomfort.
 - d. A **peritoneal venous shunt** can be used in the minority of patients who have ascites refractory to all medical therapy. The LeVeen shunt, constructed of plastic, is placed with one end in the peritoneal cavity and the other end in the subclavian vein. The Denver shunt is a modification that includes a subcutaneous manual pump that can be used to transfer ascites intermittently from the abdomen to the subclavian vein. These shunts are particularly useful in patients with tense ascites and umbilical hernias because they can provide decompression of the ascites during the perioperative period of the hernia repair. The main complication of peritoneal venous shunting is disseminated intravascular coagulation, which can be fulminant after shunt placement and requires shunt occlusion but rarely occurs if the peritoneal cavity is lavaged first. Shunts are **contraindicated** in patients with bacterial peritonitis, recent variceal hemorrhage, elevated bilirubin levels, hepatorenal syndrome, or existing coagulopathy. The shunts tend to clog with time and are used very uncommonly today.
 4. **Control of hepatic encephalopathy** requires the limitation of dietary protein intake and the use of lactulose and oral antibiotics.
 - a. **Dietary changes** should be initiated first. Dietary protein should be eliminated while adequate nonprotein calories are administered. After clinical improvement, a 20-g-per-day protein diet may be administered, with increasing protein allowances of 10 g per day every 3–5 days if encephalopathy does not recur.
 - b. **If the encephalopathy is not controlled** by diet alone, **oral agents** can be added.
 1. **Lactulose** is a nonabsorbed synthetic disaccharide that produces an osmotic diarrhea, altering intestinal flora. The oral dosage is 15–45 mL b.i.d. to q.i.d. The dosage then is adjusted to produce two to three daily soft stools. Alternatively, a lactulose enema can be prepared with 300 mL lactulose and 700 mL tap water administered two to four times daily.
 2. **Useful oral antibiotic preparations** include neomycin (1 g p.o. every 4–6 hours or 1% retention enema every 6–12 hours) and metronidazole (250 mg p.o. every 8 hours). The oral antibiotics are used as second-line agents to lactulose because, although they are equally effective, the neomycin carries some risk of ototoxicity and nephrotoxicity, and metronidazole carries some risk of neurotoxicity, albeit with prolonged administration.

19 Biliary Surgery

Charles M. Vollmer, Jr. and Steven M. Strasberg

Biliary Disorders

I. Cholelithiasis and its complications

II. Acalculous cholecystitis and other biliary infections

III. Sclerosing cholangitis

IV. Choledochal cysts

V. Biliary tree tumors

VI. Bile duct injuries

Biliary Disorders

I. Cholelithiasis and its complications

A. **Incidence.** Approximately one-tenth of the adult population harbors gallstones, with the female incidence twice that of males. In some countries (e.g., Sweden, Chile) and among certain ethnic groups (e.g., Pima Indians) the incidence of gallstones may approach 50%. Cholesterol gallstones account for 85% of all stones, with pigment stones comprising the rest.

B. Risk factors

1. Cholesterol gallstones

- There is a steady **increase in prevalence with age**.
- Gender.** Cholelithiasis is more common in women at all ages, the female to male ratio being 2:1 (25% vs. 12% at age 60 years). Factors contributing to the increased risk in females include pregnancy and the use of oral contraceptives.
- Obesity** is an independent factor affecting the prevalence of gallstone disease by a factor of three. Bile supersaturated with cholesterol leads to increased gallstone formation and disease, particularly in the obese population.
- Western diet.** The prevalence of cholesterol gallstones is higher in Western societies and is increasing in Japan with changes in societal dietary habits. Stones are rare among vegetarians.

2. Pigment stones (contain calcium bilirubinate)

- Black stones.** Risk factors include hemolytic disorders, cirrhosis, and ileal resection. Black stones also contain calcium carbonate and sulfate.
- Brown stones** are associated with biliary stasis and infection (particularly *Klebsiella*). The stones are made up to a large degree of bacterial cell bodies. Brown stones also contain calcium palmitate.

C. **Natural history.** Understanding the natural history of cholelithiasis is central to the rational management of asymptomatic patients with gallstones (*Hepatology* 16:820, 1992). People with gallstones can be divided into three groups: those who are asymptomatic, those with symptoms, and those who develop complications. There is generally a stepwise progression in this spectrum. The majority of people with gallstones never become symptomatic. Of the pool of asymptomatic patients with gallstones, only 1–2% per year will manifest symptoms of biliary colic. It is unusual (<0.5% per year) for an asymptomatic patient to develop complicated gallstone disease without first suffering symptoms. Complications develop in 3–5% of patients with symptomatic biliary colic per annum. These include acute cholecystitis, choledocholithiasis, cholangitis, pancreatitis, and gallstone ileus.

D. Asymptomatic gallstones

1. **Diagnosis.** By definition, the diagnosis of asymptomatic gallstones is incidental.

- Incidental gallstones** in the asymptomatic patient. They are discovered on routine studies performed on healthy patients. Historically, this usually consisted of radiopaque stones found on routine chest X-ray or an abdominal series. Presently, incidental gallstones are discovered more frequently during obstetric ultrasonography.
 - Incidental gallstones in the symptomatic patient.** Gallstones that are discovered in a patient who is being investigated for a symptom not due to stones present a common problem. In some patients, it is obvious that the symptom is not due to the incidentally discovered gallstones, but in others, the situation is not as evident. Common abdominal symptoms, such as dyspepsia, bloating, eructation, or flatulence *without associated pain*, are probably not derived from gallstones. Patients who have abdominal pain that is likely from other causes, such as irritable bowel syndrome, gastritis, peptic ulcer disease, or reflux disease, are more vexing. Clinical criteria must be used to determine whether the symptoms are due to the gallstones (see [section I.E.1](#)), and proper workup of these associated problems should proceed before assigning the patient's symptoms to cholelithiasis.
 - Gallstones** discovered incidentally at laparotomy. Exploration of the patient's abdomen at laparotomy sometimes discloses the presence of otherwise unexpected gallstones. When discovered, the preoperative history and physical examination must be reviewed during surgery to determine whether the gallstones truly were asymptomatic.
2. **Management of asymptomatic gallstones.** There is no role for prophylactic cholecystectomy in most patients with asymptomatic gallstones, with a few exceptions. As a result there is no need to screen for gallstones.
- Diabetes mellitus.** Prophylactic cholecystectomy in the diabetic patient with gallstones is debatable. There is no evidence that these patients are more likely than other asymptomatic patients to become symptomatic with biliary colic, or even to experience more severe complications without first presenting with biliary colic. Thus, there appears no reason to treat diabetic patients while they are asymptomatic. However, because the consequences of complications are more severe in this population, it is recommended that *symptomatic* diabetic patients have prompt surgery.
 - High cancer risks.** Patients with a porcelain gallbladder, the rare occurrence of a calcified gallbladder wall (note wall versus stones), should have cholecystectomy because of a higher risk of malignancy (approximately 25%). North and South American Indians and European–American Indian admixed populations have an increased risk of gallstones and gallbladder cancer. In this subgroup, prophylactic cholecystectomy may be warranted given the 3–5% development of cancer in patients with asymptomatic cholelithiasis.
 - Children with gallstones** have a relative indication for cholecystectomy due to the generalized difficulty in declaring and interpreting symptoms in this population.
 - Gallstones discovered at laparotomy.** Controversy continues about the management of gallstones discovered at laparotomy. The literature is conflicting with regard to the incidence of biliary symptoms after surgery in patients in whom the gallbladder is not removed, as well as the incidence of longer recovery time and perioperative complications in patients who do have incidental cholecystectomy (“cholecystectomy *en passant*”). A clear-cut role for prophylactic cholecystectomy for treatment of asymptomatic stones discovered at major abdominal surgery has yet to be demonstrated. Scenarios in which cholecystectomy *en passant* is contraindicated include concomitant placement of vascular grafts in the abdomen, when the cholecystectomy procedure is likely to be hazardous, when the patient is unstable from the primary procedure, or when long extensions of incisions are required to gain exposure of the gallbladder.
 - Patients debilitated with spinal cord trauma** have an increased incidence of asymptomatic, symptomatic, and complicated cholelithiasis. However, multiple reports have recently concluded that the magnitude of risk does not warrant prophylactic cholecystectomy in this population (*J Am Coll Surg* 189:274, 1999).

E. Symptomatic gallstones

1. **Diagnosis** depends largely on correlating symptoms with evidence of gallstones by diagnostic imaging.

- Biliary colic** is the classic presentation. This may present in a very characteristic way but may also be confused with other gastrointestinal (GI) maladies. Typical biliary colic is characterized by its periodicity, location, severity, and timing. **Periodicity:** The pain comes in distinct attacks between which the patient is well. **Location:** The typical location is in the epigastrium or right upper quadrant. **Severity:** Pain generally is intense causing the patient to cry or restrict breathing and has been described as equivalent to labor pain. Frequently, it is so severe that it prompts the patient to seek professional care immediately, and parenteral narcotics may be necessary for control. Meperidine is favored over morphine. **Timing:** The attack is usually within hours of eating a meal; often, it awakens the patient from sleep. Other related symptoms include back pain, left upper quadrant pain, nausea, and vomiting, but it is important to understand that these usually occur in addition to, rather than in place of, the characteristic symptoms as described. The pain is often atypical, lacking one or more of the typical features. The less typical the pain, the more another cause should be suspected.
- Physical signs.** There are few abdominal findings during an attack of biliary colic. Right upper quadrant tenderness may be present. The more profound tenderness present with acute cholecystitis differentiates it from biliary colic. Jaundice is not typical.
- Diagnostic imaging** is used to confirm the presence of gallstones, as well as the absence of complications. Sonographic diagnosis is based on the demonstration of echogenic structures in conjunction with posterior acoustic shadows. There usually is little or no associated gallbladder wall thickening or other evidence of acute cholecystitis. The biliary ducts must be assessed for evidence of dilatation or choledocholithiasis.

- d. **Differential diagnosis** includes acute cholecystitis, peptic ulcer disease, gastroesophageal reflux, irritable bowel syndrome, and diseases based in the thorax, such as inferior wall myocardial ischemia or right lower lobe pneumonia. Appropriate auxiliary testing is dictated by clinical suspicion of these entities.

2. Management

- a. **Laparoscopic cholecystectomy** is the appropriate treatment for the vast majority of patients with symptomatic gallstones.
1. **Laparoscopic cholecystectomy** is performed following standard laparoscopic principles. During this procedure, great care must be taken to conclusively identify the cystic structures. The complication rate from this procedure is low, and the patient's recovery and return-to-work times are excellent.
 - a. **Indications.** Approximately 95% of patients are candidates for the laparoscopic approach. Contraindications include the presence of generalized peritonitis, cholangitis, concomitant diseases that prevent use of a general anesthetic, or the patient's refusal of open cholecystectomy should urgent conversion be required. Local inflammation in Calot's triangle, as may be the case in subacute cholecystitis, can prevent complete visualization of the appropriate structures and negates a safe dissection.
 - b. **Critical view technique.** To prevent biliary injury, the cystic duct and cystic artery must be conclusively identified before transection. To achieve proper identification, Calot's triangle must be dissected free of fat as well as fibrous and areolar tissue and, importantly, the lower end of the gallbladder needs to be exposed off of the liver bed. A complete dissection demonstrates only two structures (the cystic duct and artery) entering the gallbladder. This constitutes the "critical view of safety" (*J Am Coll Surg* 180:113, 1995).
 - c. **Intraoperative cholangiography** is indicated in the following situations: patients with known choledocholithiasis, a history of jaundice, a history of pancreatitis, a large cystic duct and small gallstones, any abnormality in preoperative liver function tests, or dilated biliary ducts on preoperative sonography. An absolute indication is for confirmation of the ductal anatomy during laparoscopic cholecystectomy whenever the critical view is not achieved by dissection. **Laparoscopic ultrasound** is an alternative method for the detection of common bile duct stones showing equivalent accuracy and decreased operative time and cost (*J Gastrointest Surg* 2:50, 1998).
 - d. **Complications.** Laparoscopic cholecystectomy appears to be associated with an increased incidence of bile duct injury rising from 0.1–0.3% to open cholecystectomy to 0.5–0.8% using the laparoscopic technique (*Surgery* 128:668, 2000). Some reports indicate that unretrieved gallstone spillage can be the source of infrequent but serious long-term complications.
 2. **Open cholecystectomy** is performed in the minority of patients who have contraindications to laparoscopic cholecystectomy or who require conversion from laparoscopic cholecystectomy because of inability to complete the laparoscopic procedure or when necessary in conjunction with a laparotomy for another operation (e.g., the Whipple procedure). The gallbladder is dissected free of the hepatic bed from the top down after identification but before division of the cystic structures. This allows definitive demonstration of the cystic duct and cystic artery and prevents damage to the remaining biliary tree or hepatic arterial circulation. As biliary and arterial anatomy is variable, the conclusive identification of the cystic duct and artery is required before each is divided. Drains are infrequently necessary, most often placed when the plane of dissection atypically travels into liver parenchyma, raising the possibility of leakage from a biliary radical.
 - b. **Medical dissolution** of gallstones can sometimes be effected with oral bile acid therapy, but given the proven effectiveness of laparoscopic cholecystectomy, there is virtually no current application of medical dissolution, except possibly in the ideal patient (very small cholesterol gallstones in a nonobese patient with a functioning gallbladder). Development of gallstones after gastric bypass for morbid obesity is very common and may largely be prevented by bile acid therapy. The current optimal bile acid therapy for dissolution of gallstones is ursodeoxycholic acid, 10–15 mg/kg per day.
 - c. **Extracorporeal shock wave lithotripsy** is used to fragment single stones that are 10–20 mm in diameter. This allows for complementary dissolution therapy and passage of smaller fragments into the intestine. This therapy is now historical for gallstones.

F. Complications of cholelithiasis

1. **Acute cholecystitis** is an inflammatory complication of cholelithiasis that involves the gallbladder with a variable degree of severity. As in biliary colic, acute cholecystitis is initiated by obstruction of the cystic duct by an impacted gallstone. Although the onset and character of the resulting pain resemble that of biliary colic, it is unremitting and may persist for several days. In a limited number of cases, the cystic duct remains obstructed, and one of the complications of acute cholecystitis may develop. These include empyema, gangrene, or contained or free perforation of the gallbladder with abscess formation.
 - a. **Diagnosis** of cholecystitis depends on the constellation of symptoms, signs, and demonstration of characteristic findings with diagnostic imaging.
 1. **The symptoms** of acute cholecystitis are similar to, but more severe than, biliary colic. Unlike the time-limited pain of biliary colic, the right upper quadrant pain is persistent. Progression of the inflammatory process results in inflammation of the parietal peritoneum, and patients are more reluctant to move. Systemic complaints such as anorexia, nausea and vomiting are common. Rigors may occur but are uncommon. Associated signs of acute cholecystitis are fever (often absent), local tenderness and, in some cases, signs of peritoneal irritation in the right upper quadrant or even more diffusely. **Murphy's sign**—inspiratory arrest during deep palpation of the right upper quadrant—is characteristic of acute cholecystitis. This is most informative when the acute inflammation has subsided and direct tenderness is absent. Mild jaundice may be present. Severe jaundice, however, is rare and suggests the presence of common bile duct stones, cholangitis, or obstruction of the common hepatic duct. Intense pericholecystic inflammation due to a large impacted stone in Hartmann's pouch is termed **Mirizzi's syndrome** and may also cause high bilirubin levels.
 2. **Laboratory abnormalities** may include leukocytosis (typically 12,000–15,000 cells/ μ L, but often the white blood cell count is normal). Complications of cholecystitis, such as gangrene, perforation, or cholangitis, are suggested by an extremely high white blood cell count, more than 20,000 cells/ μ L. Liver function tests, including a serum bilirubin (usually <3 mg/dL), alkaline phosphatase, and serum amylase, also may be abnormal. However, fever and leukocytosis are often absent, and only the persistence of right upper quadrant pain suggests the diagnosis of acute cholecystitis.
 3. **Confirmation with diagnostic imaging**
 - a. **Ultrasonography** is not only the most sensitive and specific test for diagnosing cholelithiasis but also is effective in evaluating patients with suspected acute cholecystitis. Ultrasonographic findings indicative of acute cholecystitis are gallbladder wall thickening and the presence of pericholecystic fluid collections. A sonographic Murphy's sign has also been described (which, in this case, means direct tenderness over the gallbladder when compressed by the ultrasound probe). In one metaanalysis, the sensitivity and specificity of sonography for evaluating patients with suspected gallstones were 0.88–0.90 and 0.97–0.98, respectively. For the diagnosis of acute cholecystitis, the sensitivity was 0.91 and specificity 0.79 (*Arch Intern Med* 154:2579, 1994).
 - b. **Radionuclide cholescintigraphy** occasionally is useful in providing additional information in cases that are not well-defined by ultrasonography. Scintigraphic scanning is done using derivatives of iminodiacetic acid [hepatic 2,6-dimethyliminodiacetic acid (HIDA), paraisopropyl-iminodiacetic acid (PIPIDA), or disopropyliminodiacetic acid] and enables visualization of the biliary system. Concentration of the radionuclide in the bile by the liver allows for a demonstration of flow from the liver into the common hepatic duct, filling or nonfilling of the gallbladder, and emptying of the gallbladder and biliary tree into the duodenum. Because these tests depend on hepatic excretion of bile, they are often not useful when the serum bilirubin exceeds 3 mg/dL. A completely normal test result is gallbladder filling within 30 minutes. Nonfilling after 4 hours' observation in the appropriate clinical setting is good evidence of acute cholecystitis. Administration of morphine may increase the sensitivity and specificity of the test by encouraging spasm of the sphincter of Oddi and thereby enhancing gallbladder distention.
 - c. **Computed tomographic (CT) scanning** occasionally is performed to evaluate the patient with abdominal pain and acute illness. CT scan can demonstrate gallstones, although it is less sensitive for these than is ultrasonography. It may also indicate evidence of acute cholecystitis, including gallbladder wall thickening, pericholecystic fluid and edema, and emphysematous cholecystitis (air in the gallbladder or gallbladder wall).
 - b. **Management of acute cholecystitis**
 1. **Initial management** for patients with acute cholecystitis includes hospitalization, intravenous fluid resuscitation, and parenteral antibiotics. The antibiotic regimen should be appropriate for the gram-negative rods and anaerobes that are typical of bowel flora. Traditional regimens have been (1) a third-generation cephalosporin with good anaerobic coverage, (2) a third-generation cephalosporin combined with metronidazole, or, less frequently, (3) an aminoglycoside with metronidazole. However, the recent introduction of fourth-generation cephalosporins with broad coverage has provided a simple, powerful alternative. Although enterococci are frequently cultured from the gallbladder in acute cholecystitis, it is not necessary to cover these organisms separately because rarely are they a solitary pathogen.

In most patients, acute cholecystitis resolves with nonsurgical treatment. However, nonsurgical management must be reassessed frequently and should include monitoring of the patient's fever curve, physical examination, and laboratory values. If the patient is deteriorating or not improving, alterations in treatment must be made. These include changing the antibiotic regimen, percutaneous cholecystostomy, and even operative cholecystectomy or cholecystostomy.

2. **Patients with acute cholecystitis** should have cholecystectomy as definitive treatment. Controversy surrounds the timing of operation. Because most cases of acute cholecystitis resolve with antibiotic therapy, two approaches are available. Cholecystectomy can be performed either early in the course of the illness or alternatively after some delay, usually 6–8 weeks.
 - a. **Early cholecystectomy** has the advantage of resolving the illness more quickly than does antibiotic therapy. The laparoscopic approach in particular must be done early in the course of the disease, when there is a minimum of inflammation around the base of the gallbladder. This operation is best performed within 48 hours after the onset of symptoms and in the same fashion as previously described for symptomatic gallstones. However, the rate of conversion to open cholecystectomy is higher during acute cholecystitis. Approximately 80% of attempts at

laparoscopic cholecystectomy during acute cholecystitis are successful. Open cholecystectomy is also an option in acute cholecystitis and can probably be performed safely somewhat later (up to 72 hours) after the onset of the illness.

- b. **Delayed cholecystectomy** (interval cholecystectomy) can be performed after the patient recovers from the acute cholecystitis with conservative treatment. The operation should then be delayed 2–3 months to allow resolution of the acute inflammation around the base of the gallbladder. This is occasionally the only option in patients who initially seek medical attention after 3–4 days of continuous symptoms of acute cholecystitis. The cholecystectomy can be performed via either laparoscopic or open techniques.
 - c. **Cholecystostomy** should be performed in patients who have acute cholecystitis and who are failing systemic therapy but are not candidates for cholecystectomy because of severity of illness or concomitant medical problems (*Arch Surg* 134:727, 1999). Cholecystostomy can be performed operatively or percutaneously. The latter is less invasive and allows drainage of the gallbladder, which almost uniformly resolves the episode of acute cholecystitis. After resolution of the acute episode, the patient can eventually undergo either cholecystectomy or percutaneous stone extraction and removal of the cholecystostomy tube. Nonoperative stone removal is reasonable in elderly or debilitated patients who cannot have a general anesthetic.
2. **Choledocholithiasis** generally is due to gallstones that have passed from the gallbladder through the cystic duct into the common duct. In Western countries, stones rarely originate in the hepatic or common ducts, where they can become lodged and obstruct the biliary tree, causing symptoms. However, these “primary stones” are more prevalent in the Orient.
- a. **The most common manifestation** of choledocholithiasis is jaundice (with bilirubin typically <10 mg/dL). Biliary colic is common. On physical examination, signs are usually limited to icterus. Ultrasonography is useful and usually demonstrates associated gallstones in the gallbladder and bile duct dilation. The bile duct stones are seen in only 20–50% of cases. Frequently, gallstones in the lower common bile duct cannot be demonstrated by ultrasonography because of overlying bowel gas. The diagnosis often is confirmed by endoscopic retrograde cholangiopancreatography (ERCP) or percutaneous transhepatic cholangiography (PTC), which can opacify the biliary tree and demonstrate the intraductal stones.
 - b. **Management** of choledocholithiasis depends on the available expertise. Unless they are elderly or have prohibitive concomitant medical problems, all patients should have cholecystectomy. All jaundiced patients and those known to have many, large, or intrahepatic stones should have preoperative ERCP to rule out malignancy and to retrieve the stones. At centers with experienced laparoscopic biliary surgeons, after uncomplicated choledocholithiasis is diagnosed by transabdominal ultrasonography, laparoscopic cholecystectomy may be performed, with laparoscopic attempts to clear the common bile duct of stones using fluoroscopic cholangiography, biliary balloon catheters, stone baskets, or direct laparoscopic common bile duct exploration. This approach requires an institutional commitment to equipment and expertise that is not universally available. If the patient's common bile duct cannot be cleared of stones during the laparoscopic operation, the patient should subsequently have ERCP with sphincterotomy and clearance of the common bile duct. It is possible that the bile duct cannot be cleared by either laparoscopic exploration or ERCP exploration. In this circumstance, open reoperation might be required, but this is exceedingly rare in experienced hands.

An alternative strategy in centers where this expertise is not available includes a preoperative ERCP with sphincterotomy in all patients who are at high risk of common bile duct stones or in whom common bile duct stones have been demonstrated. ERCP with sphincterotomy carries a 1% risk of mortality and 10% risk of morbidity, principally acute pancreatitis. The patient can have laparoscopic cholecystectomy later. If the ERCP was unsuccessful at clearing the common bile duct, the patient may require conversion to open cholecystectomy if the duct cannot be cleared employing the laparoscopic approach. The choice of strategy depends on available expertise.

3. **Acute cholangitis** is an infection of the biliary tree that is potentially life-threatening. **Charcot's triad**, the combination of fever, jaundice, and right upper quadrant pain, remains the hallmark of this disease but is present in only 50–70% of patients. A spectrum of diseases is present, ranging from subclinical illness to acute toxic cholangitis. This condition may present with the advanced symptoms of **Reynolds' pentad** (Charcot's triad with hemodynamic instability and mental status changes). Cholangitis is typically associated with partial or complete obstruction of the biliary tree and concomitant infection. Therefore, investigation of the biliary tree is mandatory to demonstrate and relieve the underlying etiology of the obstruction. Biliary tract conditions associated with cholangitis include choledocholithiasis, benign and malignant strictures, biliary-enteric anastomoses, and indwelling tubes or stents. Invasive procedures such as T-tube cholangiography, ERCP, or PTC are also associated with cholangitis, and therefore patients are usually treated with antibiotics before these procedures. Ultrasonography or CT scan may reveal biliary dilatation, but definitive diagnosis is made by ERCP or PTC. These studies are superior in that they are both diagnostic and therapeutic, demonstrating the level of obstruction and allowing culture of bile, removal of stones or indwelling foreign bodies, and placement of drainage catheters if necessary.

Treatment. Initial management of cholangitis includes intravenous antibiotics appropriate for the coverage of the most commonly cultured organisms: *Escherichia coli*, *Klebsiella pneumoniae*, *Streptococcus faecalis*, and, less commonly, *Bacteroides fragilis*. In patients with acute toxic cholangitis or in patients who fail to respond to antibiotic therapy, emergency decompression of the biliary tree is required. This is typically accomplished by endoscopic sphincterotomy and nasobiliary drainage, although percutaneous transhepatic drainage is also effective. If decompression by these less invasive means is not available or possible, operative intervention to decompress the biliary tree is indicated. In such an unstable patient, operative intervention should be limited to insertion of a T tube in the common bile duct. Definitive operative therapy for benign or malignant biliary tract strictures should be deferred until a later date. Cholangitis in patients with indwelling tubes or stents generally requires that the stent be removed and replaced.

4. **Gallstone ileus** is a rare complication that results from obstruction of the intestine, usually the lower small bowel, by a large gallstone. The gallstone generally erodes from the gallbladder into the duodenum and travels through the small bowel until it reaches the ileum, where it cannot pass because of the small caliber of this part of the bowel. The usual presentation is a small-bowel obstruction. Air in the biliary tree is explained by retrograde passage of GI air through the cholecystoenteric fistula. Treatment is exploratory laparotomy and management of the small-bowel obstruction by removal of the gallstone from the bowel through an enterotomy made in healthy intestine. Rarely, segmental small-bowel resection is needed because of erosion of the stone. The entire bowel should be searched diligently for other stones.
5. **Biliary pancreatitis** is caused by passage of gallstones from the gallbladder through the common bile duct with distal impaction. Although the mechanism remains unclear, passage of a stone in this way can cause an episode of acute pancreatitis (see [Chapter 17](#)). Management of the biliary tract disease in this entity includes ERCP with sphincterotomy if the patient has severe acute pancreatitis that may be caused by an impacted stone at the ampulla. Otherwise, ERCP in the acute setting probably is not indicated. Once the acute episode of pancreatitis has resolved, the gallbladder should be removed while the patient is still hospitalized, or within 2 weeks, to prevent recurrent acute biliary pancreatitis as an outpatient. Preoperative ERCP is unnecessary because most of the time the causative stone has passed, but an operative cholangiogram should *always* be done during the cholecystectomy to prove that this has indeed occurred.

II. Acalculous cholecystitis and other biliary infections

- A. **Acalculous cholecystitis** typically occurs in the face of concomitant acute systemic illness, such as extensive burns or major trauma, or after significant abdominal or thoracic operations. It is also associated with prolonged dependence on parenteral nutrition, with episodes of systemic sepsis, or during multiple organ system failure.
 1. **Symptoms and signs** depend largely on the patient's concurrent medical conditions. Alert patients typically complain of right upper quadrant or diffuse upper abdominal pain and tenderness. White blood cell count, bilirubin, aspartate aminotransferase, and alkaline phosphatase levels may be elevated. Signs and symptoms typical of acute cholecystitis may not be evident due to sedation or altered consciousness in patients with systemic illness. Elevations of the alkaline phosphatase or bilirubin levels warrant further investigation in these clinical circumstances.
 2. **Diagnostic imaging** is essential in establishing the diagnosis of acalculous cholecystitis. Ultrasonography is inexpensive and can be done at the bedside in the critically ill patient. It can demonstrate the typical findings of acalculous or calculous cholecystitis, including gallbladder wall thickening, pericholecystic fluid, or abscess formation in the right upper quadrant. Limitations include overlying bowel gas or concomitant abdominal wounds or dressings. Therefore, false-negative outcomes are possible. Abdominal CT scan is as sensitive as ultrasonography for this condition and provides more complete imaging of the remainder of the abdominal cavity from the lung bases to the pelvis. This modality can reveal other intraabdominal problems that may be a part of the differential diagnosis, particularly in postoperative patients. CT scan generally does not suffer from the same limitations mentioned previously for transabdominal ultrasound. However, its principal disadvantage is that it requires transfer of the patient to the radiology suite, which may be a prohibitive risk in the critically ill person. Therefore, the two modalities are complementary. In difficult cases, percutaneous cholecystostomy may be both diagnostic and therapeutic.
 3. **Management of acalculous cholecystitis** must be tailored to the individual patient. Definitive treatment includes urgent cholecystectomy. However, most affected patients are not fit enough to tolerate a major abdominal operation. In these patients, PTC is the procedure of choice; it resolves the cholecystitis in nearly 100% of patients and generally is well tolerated. Additional principles of management include initiation of systemic antibiotics, maintenance of nothing-by-mouth status, and treatment of the associated illnesses that have placed the patient at risk for this condition. Ultimately, cholecystectomy is performed if the patient recovers from the primary disease process.
- B. **Oriental cholangiohepatitis**, also known as **recurrent pyogenic cholangitis**, is endemic to the Far East. It is usually due to infestation of the biliary tree with

parasites (*Ascaris lumbricoides*, *Clonorchis sinensis*) that cause stasis, bacterial overgrowth, and brown stone formation. Typical findings include multiple intrahepatic and extrahepatic biliary ductal stones, strictures, and repeated bacterial infections of the biliary tract. Strictures may be found throughout the biliary tree but are more common in the main hepatic ducts. The left duct is more frequently and severely affected than is the right.

1. **Symptoms and signs** are those of recurrent cholangitis. The patients are typically young, thin, and present with right upper quadrant pain, fever, and jaundice. The episodes can vary in severity, from chronic subclinical illness leading gradually to generalized symptoms of hepatic insufficiency and malnutrition, to severe acute suppurative cholangitis with hypotension, alteration in mental status, acidosis, and even death when not recognized and treated in a timely fashion.
2. **Diagnostic imaging** demonstrates the typical diffuse biliary findings and multiple stones.
 - a. **Ultrasonography** can show biliary obstruction, stones in the biliary tree, pneumobilia from infection with gas-forming organisms, liver abscesses, or, occasionally, strictures. Ultrasound is very good at demonstrating intrahepatic stones by their characteristic posterior acoustic shadowing. Conversely, ultrasonography is not sensitive for demonstrating strictures, although some may be evident with this test.
 - b. **CT scan** can demonstrate findings similar to ultrasonography. It is also valuable in delineating the hepatic anatomy and judging the amount of hepatic parenchyma that has been preserved in more advanced disease. In addition, it can help guide parameters for potential liver resection.
 - c. **ERCP and PTC** are the mainstays of biliary imaging for Oriental cholangiohepatitis. As is the case with the more conventional cholangitis, these studies can allow for not only the diagnosis of biliary obstruction and definition of the level of biliary strictures and stones, but also can provide the opportunity for therapeutic decompression of the biliary tree in the acute setting.
3. **Management of Oriental cholangiohepatitis** is palliation of the biliary strictures and the provision of wide biliary drainage. During an acute episode of cholangitis, temporary percutaneous or endoscopic drainage must be achieved to alleviate the sepsis. General systemic supportive care, including intravenous antibiotics and generous hydration are indicated. In occasional patients with known Oriental cholangiohepatitis and documented lower duct strictures, acute surgical intervention with drainage of the common bile duct by T-tube placement is appropriate.

In the chronic setting, the multiple biliary strictures and stones must be addressed, and this generally requires multiple percutaneous or endoscopic approaches to the biliary tree with attempts at dilation. When these eventually fail, the patient should have cholecystectomy and wide biliary drainage above the level of obstruction, such as a choledochoduodenostomy or a hepaticojejunostomy emptying into a Roux-en-Y limb. When using this technique, the blind end of the limb should be sutured to the peritoneal surface of the abdominal wall and marked with a radiopaque ring to provide a portal of entry for future approaches to the biliary-enteric anastomosis. In occasional cases, liver resection is appropriate if the damaged area is completely or mostly confined to one portion of the liver.

III. Sclerosing cholangitis is a cholestatic disorder of unknown cause characterized by progressive fibrous obliteration of the biliary tract. Almost 70% of patients are men, and many are in the fifth decade of life. An association with inflammatory bowel disease exists. Sclerosing cholangitis is present in 1–5% of those with inflammatory bowel disease. However, the incidence of inflammatory bowel disease in patients with sclerosing cholangitis is greater, quoted as between 25% and 75%.

A. Pathology

1. **Liver.** The most consistent change is the obliteration of macroscopic bile ducts. Other findings include cholestasis; periductal fibrosis; inflammation with accumulation of plasma cells, lymphocytes, and polymorphonuclear leukocytes; obliterative cholangitis; and secondary biliary sclerosis.
2. **Bile ducts.** Concentric fibrosis of the duct walls with patchy chronic inflammatory infiltrate results in encroachment of the lumen, which may be diffuse or segmental. In most patients, both intrahepatic and extrahepatic ductal segments are involved. The hepatic duct bifurcation is typically the area most severely involved (*Ann Surg* 200:54, 1984).

B. Diagnosis

1. **Clinical manifestations.** There are no pathognomonic signs. Although patients can be asymptomatic for up to 15 years, prolonged disease ultimately leads to progressive hepatic failure. The condition is characterized by relapses and remissions, with quiescent periods that can be significant. Jaundice with pale stools and dark urine forms the initial clinical picture. With advanced disease, pain in the right upper quadrant, pruritus, fatigue, and weight loss often accompany the jaundice. Cholangitis with pyrexia and rigors may ultimately occur. Physical examination commonly reveals jaundice, hepatomegaly, and splenomegaly.
2. **Diagnostic studies**
 - a. **Radiologic findings.** The procedure of choice is ERCP. In some instances, the intrahepatic biliary tree may not be well visualized, and transhepatic cholangiography is complementary. The most common finding is a diffuse and irregular narrowing of the entire biliary tree. The strictures are short and annular, giving a beaded appearance. In progressive disease, the strictures become confluent, and diverticula of the ducts appear. Although cholangiography is the gold standard, scintigraphy, sonography, and CT scan also are useful in making the diagnosis.
 - b. **Biochemical studies.** The alkaline phosphatase level is almost always elevated, usually out of proportion to the bilirubin level. Serum transaminases may be mildly elevated. Most patients are negative for hepatitis B surface antigen.
3. **Management.** Symptomatic improvements have been reported with the use of various drugs aimed at reversing a presumed autoimmune etiology. These include corticosteroids, immunosuppressive agents, methotrexate, and D-penicillamine. However, none of these alter the natural history of the disease. Sclerosing cholangitis has been effectively palliated with endoscopic or percutaneous dilation of strictures. Placement of stents after dilation can be a valuable preoperative adjunct. However, some form of operative intervention is required for most patients.
 1. **Resection or bypass of localized strictures.** The role of nontransplant surgery in this disease is limited. Occasionally, however, the disease is focused in the extrahepatic bile ducts, and a “dominant stricture” exists that can be excised or bypassed by hepaticojejunostomy. The candidates should be ideal. It should only be done in those with good residual liver function.
 2. **Extensive, diffuse stricture disease** with end-stage cirrhosis is an indication for orthotopic liver transplantation. If the patient has undergone a previous decompressive operation, transplantation is technically more challenging but certainly not contraindicated. Accordingly, decompressive procedures are performed less commonly now than in the past, and patients are referred and listed for transplantation sooner.
 3. **Sclerosing cholangitis** is a risk factor for cholangiocarcinoma; therefore, close surveillance of patients is needed. The diagnosis is difficult because cholangiocarcinomas also masquerade as strictures. An elevated CA 19-9 may be helpful in diagnosing malignancy. Rapid clinical deterioration is also highly suggestive. Suspected malignancy is an indication for resection or transplantation even in the absence of end-stage liver disease.
4. **Prognosis.** Many patients have a course that progresses to cirrhosis and liver failure despite early palliative interventions. Liver transplantation likely improves survival and quality of life. Overall, a 75% 10-year survival is expected.

IV. Choledochal cysts

- A. **General considerations.** Choledochal cysts are congenital dilations that may occur anywhere along the course of the biliary tract. They are more frequently identified in women (3:1 ratio) and those of Asian descent. Sixty percent are diagnosed before the age of 10. Diagnosis and treatment are essential because the cysts predispose to jaundice, choledocholithiasis, cholangitis, portal hypertension, and cholangiocarcinoma.
- B. **Diagnosis** is made with ultrasound and/or CT scan. An apparent increase in incidence of these lesions has paralleled advances and use of these modalities. Further delineation of the cyst should be obtained with specific biliary imaging such as ERCP.
- C. **Anatomic classification.** These are not cystic lesions per se but rather dilations of the bile duct. A classification scheme has identified five distinct types. Type I cysts are saccular, localized, or fusiform dilations of the common bile duct and are by far the most common (65–90%). Type II cysts are rare, isolated diverticula of the common bile duct. Type III cysts, otherwise termed **choledochoceles**, are localized dilatations within the intraduodenal part of the common bile duct. Most lesions thought to be choledochoceles are, in fact, duodenal duplications. Type IV cysts are characterized by multiple cystic areas of the biliary tract, both inside and outside of the liver. Finally, type V cysts are single or multiple lesions based only in the intrahepatic portion of the tract.
- D. **Etiology.** The majority of cysts are associated with an unusually long common channel—that is, bile and pancreatic ducts join much higher than usual (“anomalous biliary-pancreatic junction”). This is believed to be etiologic. An alternative embryologic theory proposes abnormal canalization of the developing ductal system. Type II and III cysts are likely developmental anomalies similar to diverticula in other areas of the GI tract.
- E. **Presentation.** The “classic” triad of right upper quadrant pain, jaundice, and a palpable abdominal mass is actually present only a minority of the time. Pain may mimic biliary colic. Neonates frequently present with biliary obstruction, whereas older youths suffer from jaundice and abdominal pain. Rarely, pancreatitis or duodenal obstruction can be caused by a choledochocoele. Findings of cirrhosis, sepsis from cholangitis, or free intraperitoneal rupture occur infrequently as late complications.
- F. **Treatment** is primarily surgical, as medical therapies are not successful. Cholangiocarcinoma develops in up to 30% of cysts and usually presents in the fourth decade of life. Cyst excision with a Roux-en-Y hepaticojejunostomy reconstruction is the treatment of choice for types I and IV. Simple excision of the rare type II cyst has been performed. Local cyst excision with sphincteroplasty through a transduodenal approach is effective for type III disease. Caroli’s disease can be treated with hemihepatectomy when confined to one side of the intrahepatic tract. More often, bilateral disease is present with associated liver damage and mandates orthotopic liver transplantation.

V. Biliary tree tumors

- A. **Benign tumors of the bile ducts.** Benign tumors, usually adenomas, are rare and arise from the ductal glandular epithelium. They are characteristically polypoid and rarely are larger than 2 cm. Most are found adjacent to the ampulla, with the common bile duct being the next most common site.
1. **Most patients present with intermittent obstructive jaundice**, often accompanied by right upper quadrant pain. This presentation may be confused for choledocholithiasis. The lesions often are detected by intraoperative cholangiography, choledochoscopy, or ultrasonography. Lesions near the ampulla may be visible via endoscope.
 2. Despite some authors' suggestions to this effect, the **precancerous character of these benign lesions remains controversial**. Given the rarity of these tumors, it has been difficult to substantiate claims supporting either position.
 3. **Treatment** should involve complete resection of the tumor with a margin of duct wall. High recurrence rates have been reported after simple curettage of the polyps. Lesions situated at the ampulla can usually be managed by transduodenal papillotomy or wide local excision.
- B. **Malignant tumors of the bile ducts.** Cholangiocarcinoma arises from the bile duct epithelium and can occur anywhere along the course of the biliary tree. Cholangiocarcinoma is a rare malignancy and is associated with a relatively poor prognosis. Although these tumors metastasize infrequently, when they do intrahepatic and peritoneal patterns predominate. Frequently, their proximity to critical structures, such as the hepatic artery or portal vein, precludes curative resection.

Cholangiocarcinoma has been classified into three main types according to anatomic location: intrahepatic, extrahepatic upper duct (also called **hilar** or **Klatskin tumor**), and extrahepatic lower duct. This simple classification dictates preferred surgical treatment.

The **median age of onset** is approximately 60 years, and men are afflicted more frequently than are women. Associations exist with primary sclerosing cholangitis, choledochal cysts, intrahepatic stones, and certain parasitic infestations.

1. **Diagnosis.** Jaundice, followed by weight loss and pain, are the most frequently encountered symptoms.
 - a. **MR scan** is an all-purpose investigation for cholangiocarcinoma. It provides cholangiography, can demonstrate the tumor and its relationship to key vessels, and detect intrahepatic metastases. It is rapidly supplanting older diagnostic approaches.
 - b. **Ultrasonography** can demonstrate bile duct masses and bile duct dilatation and provide rudimentary information on the extent of tumor involvement within the liver. Duplex ultrasonography also can demonstrate the relationship to critical vascular structures. Some reports indicate that ultrasound is more than 80% accurate in predicting portal vein involvement.
 - c. **CT scan** may also be helpful in delineating the mass and defining its relation to the liver. Advanced scanners can provide similar information to MR scan. Both investigations are not needed.
 - d. **ERCP** is the most valuable diagnostic tool for cholangiography of lower duct tumors. Distal lesions may be indistinguishable from pancreatic carcinoma on preoperative evaluation, and the distinction is often not made until final pathologic analysis. It is also valuable for upper duct tumors, but if obstruction is complete the upper limit of the tumor cannot be delineated. Since the advent of MR cholangiography, its role is increasingly becoming therapeutic—that is, to decompress the liver preoperatively.
 - e. **PTC** has been used when ERCP cannot delineate the tumor completely. Under PTC guidance, fine-needle aspiration cytology can provide a tissue diagnosis in approximately 75% of patients.
2. **Assessment of tumor resectability.** The following findings garnered from preoperative assessment exclude consideration of a curative resection: (1) bilateral intrahepatic bile duct spread of the disease, or multifocal disease on cholangiography; (2) involvement of the main trunk of the portal vein (except in unusual circumstances); (3) bilateral involvement of hepatic arterial or portal venous branches; and (4) a combination of vascular involvement with cholangiographic evidence of extensive contralateral ductal spread.

Intraoperative findings of lymph node or omental/peritoneal metastases also preclude resection. Staging laparoscopy may be useful in discovering these factors before formal laparotomy. Vascular involvement is not an absolute contraindication to resection. It is possible to resect and reconstruct portal venous segments. Arterial reconstructions are more difficult. Due to its proximity to the hilar confluence, the caudate lobe should always be resected *en bloc* with a mass in this vicinity.

3. **Treatment.** Resection remains the primary treatment for cholangiocarcinoma and provides the only opportunity for cure.

Intrahepatic tumors are best treated with hepatic resection. However, these tumors often present at an advanced stage, often with bilateral hepatic spread, and only 15–20% are resectable.

4. Resection of **hilar** tumors includes the bile duct bifurcation and the caudate lobe, with subsequent biliary reconstruction using a Roux-en-Y hepaticojejunostomy. More extensive disease involving structures confined to one hemiliver often mandate ipsilateral hemihepatectomy.

Some lesions are situated in the middle of the extrahepatic bile duct and may be approached with an excision of the supraduodenal extrahepatic bile duct, cholecystectomy, and portal lymphadenectomy. These lesions may be clinically indistinguishable from a tumor of gallbladder origin.

In contrast to more proximal tumors, approximately 80% of **lower duct** tumors are resectable. In the absence of local extension or distant metastases, pancreaticoduodenectomy is necessary for definitive resection. Survival data are also more favorable: The 5-year survival rate ranges from 17% to 39%. Tumors derived from the bile duct have a slightly better prognosis than those of pancreatic origin in the same region, probably reflecting a more favorable biologic behavior of the former.

Adjuvant external beam radiation and chemotherapy have been advocated but remain investigational at this time. Clinical trials are limited by the paucity of eligible patients. The most efficacious chemotherapeutic agent to date is 5-fluorouracil, with paltry responses of up to 15%.

5. **Palliation** for patients with disseminated or unresectable disease involves surgical, radiologic, or endoscopic biliary decompression. When unresectability is demonstrated preoperatively or at staging laparoscopy, decompression should be by endoscopic or percutaneous stents that are fully internal, if possible. When unresectable disease is encountered at laparotomy, internal biliary drainage is best achieved by choledochojejunostomy. Intrahepatic bypasses are occasionally performed.
- C. **Carcinoma of the gallbladder**
1. **General considerations.** Carcinoma of the gallbladder, like cholangiocarcinoma, is an uncommon cancer (5,000 per year in the United States) with a poor prognosis. Approximately one-third of these tumors are diagnosed incidentally during a cholecystectomy, and cancer is found in approximately 1% of all cholecystectomy specimens. The incidence increases with age, peaking at 70–75 years, and has a 3:1 female to male ratio. There is a strong correlation with gallstones (95%). Histologically, nearly all are adenocarcinomas and concomitant cholecystitis is frequently present. Porcelain gallbladder, or calcification and wall thickening from chronic irritation, carries a risk of cancer of approximately 25%.

The tumor spreads primarily by direct extension into segments IV and V of the liver, adjacent to the gallbladder fossa. The cancer also spreads via the lymphatics along the cystic duct to the common bile duct. Only a small percentage of patients have noninvasive lesions that are resectable for potential cure.

2. **Treatment.** Most gallbladder cancers have invaded into the liver or extend into the porta hepatis before diagnosis. For such patients, palliative procedures, such as decompression of the proximal biliary tree or bypass of duodenal obstruction, are available, but not curative. When gallbladder carcinoma is limited to the gallbladder and contiguous liver, surgical attempts at cure are appropriate. This entails radical resection with portal lymph node dissection.
 - a. **In situ and early disease** confined to the gallbladder wall (i.e., not penetrating the muscularis) may be treated by radical cholecystectomy alone, which includes the gallbladder and the gallbladder bed of the liver. Invasion through the muscularis of the gallbladder or the presence of lymph node metastases requires more radical resection. Depending on the extent of local invasion, extirpation may range from wedge resection of the liver adjacent to the gallbladder bed to resection of 75% of the liver. Due to the close proximity of structures emerging from the porta hepatis, hepatic wedge resection in this area may be difficult, leading some to recommend segmental liver resection (usually segments IV and V). Dissection (in continuity) of the portal, paraduodenal, and hepatic artery lymph nodes should accompany the liver resection. Survival advantages have been demonstrated after radical resection. Because of the aggressive nature of this malignancy, adjuvant chemoradiation is often recommended, but no proof of efficacy is available.
 - b. **Extensive liver involvement or discontinuous metastases** preclude surgical resection as a reasonable option. Patients thus affected often are

symptomatic, with pain, jaundice, nausea and vomiting, and weight loss. Jaundice may be palliated by percutaneous or endoscopically placed biliary stents. Duodenal obstruction can be surgically bypassed if present. Radiotherapy can decrease tumor bulk and temporarily relieve obstruction, but no survival benefits have been demonstrated. As with most malignancies of hepatobiliary origin, there are no effective chemotherapeutic agents.

VI. Bile duct injuries

- A. **General considerations.** Although problematic during the era of open cholecystectomy, injuries to the biliary system presently have a renewed emphasis due to their increased frequency in the era of laparoscopic cholecystectomy. These injuries are usually associated with litigious action, as they can be morbid, psychologically devastating, and even deadly. Although it is the most frequent, cholecystectomy is not the only causative factor. Trauma and iatrogenic injury from biliary instrumentation or other operations on the upper GI tract also contribute.
1. **Generalized risk factors** for injury during cholecystectomy include (1) lack of training or experience, especially when operating in the face of acute edema, (2) local factors of acute or chronic inflammation, (3) technique of cholecystectomy, and (4) unusual anatomy, particularly an aberrant right hepatic duct. The same conditions also predispose toward injuries to the hepatic vasculature. Prevention is the best approach to this problem and can be ensured by dissecting the aforementioned "critical view of safety" or by performing cholangiography before definitive transection of any structures when the anatomy is unclear. Most injuries are not recognized during the original surgery, but when they are, they are usually identified by cholangiography, bile in the field, an open lumen, or hemorrhage.
 2. Once the injury is identified, **the surgeon** should proceed directly to open exploration and repair only if qualified and comfortable with complex techniques in hepatobiliary surgery or to control life-threatening hemorrhage. If not prepared to perform a definitive repair, a drain should be placed in the right upper quadrant, and the patient should be immediately referred to a specialist in hepatobiliary surgery.
- B. **Classification.** A classification scheme has been proposed and is now widely accepted (*J Am Coll Surg* 180:101, 1995). Type A injuries are cystic duct leaks or leaks from small ducts in the liver bed. Type B and C injuries involve an aberrant right hepatic duct. Type B represents an occluded segment, whereas Type C involves open drainage from the proximal draining duct not in continuity with the common duct. Type D injuries are *lateral* injuries to the extrahepatic bile ducts. Type E injuries (I–V) are derived from the Bismuth classification of common bile duct injuries and represent circumferential injury (transections or occlusions) at various levels of the common bile duct.
- C. **Presentation and diagnosis** depend on the type of injury. Abdominal pain, fever, sepsis, and bilious-cutaneous fistulas are common for type A injuries, although these usually manifest initially with vague symptoms such as anorexia or failure to thrive. Type B injuries usually present long after operation with cholangitis from obstruction of a hepatic drainage segment. Type C and D injuries present with bile peritonitis or sepsis from an ongoing leak. Type E injuries are often immediately apparent at operation. When not initially recognized, they present with jaundice and often pain. Combinations of diagnostic techniques are often necessary to fully delineate the injury and include CT scans, ultrasound, hepatobiliary scintigraphy, fistulograms, ERCP, and PTC.
- D. **Management**, again, depends on type and timing of presentation. Most type A injuries can be successfully managed with ERCP and sphincterotomy, stenting, or both. Occlusive lesions require decompression of the proximal drainage systems via PTC. In general, if an injury requires operative repair and the patient is stable, it should be performed within the first few days after the original insult while inflammation is at a minimum. However, this is most often not possible due to delayed diagnosis, and longer-term temporization is required. Control of sepsis, percutaneous drainage, and adequate nutrition should be optimized before definitive repair which should be performed months henceforth.
- E. **Operative repair**, when indicated, is best achieved by means of a Roux-en-Y hepaticoenterostomy, in which the bile duct is dissected and débrided back to viable tissue. All bile ducts must be accounted for, and adequate blood supply must be apparent for each. A mucosa-to-mucosa anastomosis constructed with fine absorbable suture and no tension is desired. Excellent long-term outcomes have been described, with anastomotic stricture the most common, yet infrequent, complication (*Arch Surg* 134:604, 1999). Rarely, liver transplantation may be necessary for those patients who suffer profound hepatic insufficiency secondary to the injury.

20 Spleen

Alliric I. Willis and Jeffrey F. Moley

Introduction

Indications for Splenectomy and Procedure

I. Hematologic disorders

II. Cysts, tumors, and abscesses

III. Splenectomy

Introduction

The spleen has multiple hematologic and immunologic functions. It receives as much as 5% of the cardiac output through its blood supply from the splenic artery and the short gastric arteries and is drained by corresponding veins. Histologically, the spleen is comprised of 80% red pulp, predominately red blood cells (RBCs), and 20% white pulp, predominately macrophages and lymphocytes. As many as 20% of normal patients may have accessory splenic tissue, which can be found between the stomach and pancreas. Splenic functions include blood filtering, removal and destruction of aged and deformed RBCs, RBC and platelet storage, phagocytosis of bacteria (contains 25% of fixed tissue macrophages in the body), and production of immunoglobulins IgG and IgM (largest producer of IgM) as well as opsonins such as tuftsin and properdin. Nontraumatic indications for splenectomy include hematologic disorders, cysts, tumors, and abscesses.

Indications for Splenectomy and Procedure

I. **Hematologic disorders** are the most common nontraumatic indication for splenectomy ([Table 20-1](#)).

Table 20-1. Splenectomy for thrombocytopenia

- A. **Immune (idiopathic) thrombocytopenic purpura (ITP)**, a syndrome of thrombocytopenia caused by antiplatelet antibody (IgG)-mediated destruction of platelets, is the most common hematologic indication for splenectomy.
1. **Acute ITP.** Ninety percent of pediatric cases but less than 10% of adult cases of ITP. Characterized by rapid onset of thrombocytopenia after viral infection. Spontaneous remission occurs in 90% of cases within 6–12 months.
 2. **Chronic ITP.** A more indolent form of thrombocytopenia that may be present for years. More than 90% of adult cases of ITP. Three-to-one female-to-male preponderance. Spontaneous remission is rare.
 3. **Clinical findings** of ITP can include easy bruising, gingival bleeding, epistaxis, and menorrhagia. Intracranial hemorrhage, although rare, is more common in children and is associated with high mortality. Symptoms are associated with platelet counts of less than 50,000 cells/ μ L, but spontaneous bleeding is rare when platelet counts are greater than 30,000 cells/ μ L. Splenomegaly is rare.
 4. **Laboratory evaluation** reveals thrombocytopenia on peripheral smear. Bone marrow examination is usually normal. Platelet-associated IgG autoantibodies can be detected in 90% of patients but can also occur in other thrombocytopenic states.
 5. **Differential diagnosis** includes drug-induced antiplatelet antibodies and secondary thrombocytopenias from systemic lupus erythematosus, chronic lymphocytic leukemia, and lymphoma. The treatment of human immunodeficiency virus-associated thrombocytopenia is similar to chronic ITP.
 6. **Treatment of ITP.** Ninety percent of children younger than 10 years of age require no therapy. No specific treatment is required for patients with platelet counts of greater than 50,000/ μ L who have minimal or no symptoms. Avoidance of even minor trauma is necessary. Platelet transfusion is indicated in acute emergencies, when bleeding must be stopped immediately.
 - a. **Corticosteroids.** Prednisone, 1–2 mg/kg per day p.o., should be initiated on diagnosis and as initial treatment for relapse in symptomatic patients. Response rates are 60–80% in acute ITP but less than 25% in chronic ITP. Treatment may be required for 2 weeks before a response is seen. Platelet count rises secondary to an increase in platelet production, although platelet-associated IgG levels decrease with treatment as well. Steroids are tapered when the platelet count exceeds 30,000–50,000/ μ L and symptoms are minimal. For severe thrombocytopenia and active bleeding, intravenous methylprednisolone (1 g per day i.v. for 3 days) followed by prednisone may be indicated.
 - b. **Splenectomy** remains the principal treatment for ITP and is indicated for inadequate response to steroids, relapse after an initial remission, and disease that has lasted for more than 1 year. Intracranial hemorrhage requires emergency splenectomy. Steroids are continued during the postoperative period until the platelet count reaches 100,000/ μ L and are then tapered gradually. Successful remission occurs in up to 80% of patients overall and in over 90% of patients who have a good initial response to steroid therapy.
 - c. **Immunosuppressive therapy** should be considered for patients who fail steroids and splenectomy (more frequently male patients). Vincristine, 1–2 mg per week i.v. for 4–6 weeks, can produce prolonged increases in platelet count by preventing phagocytes from destroying the antibody-coated platelets. In addition, cytotoxic agents, such as azathioprine (150 mg per day p.o. for 6 months) or cyclophosphamide (1–2 mg/kg per day p.o. for 2–3 months) can also be used. Treatment is effective in up to 65% of patients, although complete remissions are rare.
 - d. **Danazol**, 200 mg p.o. t.i.d., can gradually increase the platelet count by decreasing the number of IgG receptors on phagocytes. Side effects include water retention, virilization, hepatotoxicity, and “paradoxical hemorrhage” due to increased fibrinolysis.
 - e. **Intravenous immune globulin (IVIG)**, 0.4 g/kg per day i.v. for 5 days or 1 g/kg i.v. for two daily doses, can provide a transient, rapid increase in the platelet count. The proposed mechanisms of action include blockade of Fc receptors on phagocytes, which thereby attenuates platelet removal, or modulation of both B- and T-lymphocyte function via antiidiotypic antibodies present in the pooled gamma globulin, which results in decreased platelet-associated IgG production. IVIG is used in patients with acute bleeding, in children to defer splenectomy or reduce steroid usage, and preoperatively in patients who do not respond to steroid therapy.
- B. **Thrombotic thrombocytopenic purpura (TTP)** is a rare syndrome characterized by thrombocytopenia, microangiopathic hemolytic anemia, fever, neurologic abnormalities, and renal dysfunction, although any of the latter three findings may be absent at the time of presentation. TTP is characterized by widespread microvascular thrombosis of arterioles and capillaries. TTP is a medical emergency, and therapy should be instituted immediately. Platelet transfusion may exacerbate symptoms and is contraindicated.
1. **Daily total plasma exchange using fresh frozen plasma as replacement fluid** is the mainstay of treatment of TTP. The response rate exceeds 80%, and survival is 90%. It is hypothesized that plasma exchange may remove a harmful circulating factor or replace a deficient factor or both. Therapy should be continued until the platelet count, serum lactate dehydrogenase, and neurologic status have normalized.
 2. **Prednisone**, 1 mg/kg per day p.o., or methylprednisolone, 1 mg/kg per day i.v., and **aspirin**, 325 mg per day p.o., are usually added to plasma exchange regimens.
 3. **Patients** with refractory disease and those who require prolonged plasmapheresis may benefit from **vincristine**, 1–2 mg per week i.v., until a response is seen (maximum dose 15 mg). **IVIG**, 0.4 g/kg per day i.v. for 5 days, can also be effective.
 4. **Splenectomy** has been reported to occasionally lead to remission in patients with an inadequate response to plasma exchange (*Ann Hematol* 70:231, 1995). Splenectomy may also play a role in preventing relapse in patients who are in remission. The spleen is a major site of endothelial damage in TTP, and its removal abolishes the endothelial injury-induced production of platelet-activating von Willebrand factor fragments. Plasma exchange should be continued for several days postoperatively after a response is observed.
 5. **Hemolytic-uremic syndrome (HUS)** is a closely related disease (initially described in children) wherein renal dysfunction is the primary manifestation. The syndromes are part of a spectrum often termed *TTP-HUS*, and treatment is similar for both.
- C. **Hemolytic anemias** result from increased destruction of RBCs ([Table 20-2](#)).

Table 20-2. Splenectomy in hemolytic anemias

- Hereditary spherocytosis** is the most common congenital hemolytic anemia related to an abnormality in erythrocyte structure. A defect in the membrane protein spectrin leads to small, spherical, and relatively rigid erythrocytes that are unable to deform adequately to traverse the splenic microcirculation, resulting in their sequestration and destruction in the splenic red pulp. Inheritance is typically autosomal dominant, although up to 25% of cases may be sporadic.
 - Presentation** can include jaundice due to hemolysis, splenomegaly, mild to moderate anemia, and pigment gallstones (30% of adults, rare in children younger than 10 years old). Spherocytes are present on the peripheral smear with microcytic anemia and reticulocytosis. The osmotic fragility test is positive, and the direct Coombs' test is negative.
 - Splenectomy**, which alleviates hemolysis and corrects the anemia, is indicated in nearly all patients with hereditary spherocytosis. Splenectomy should not be performed in routine cases before 6 years of age due to the increased risk of postsplenectomy sepsis. Indications for earlier splenectomy include aplastic crisis, growth failure, and severe anemia. Cholecystectomy also should be performed at the time of splenectomy if cholelithiasis is present.
 - Hereditary elliptocytosis** is an autosomal dominant disorder in which an intrinsic cytoskeletal defect is present, causing up to 90% of circulating erythrocytes to be elliptical. Most patients are asymptomatic, but a minority may have symptomatic hemolytic anemia. Splenectomy removes the site of hemolysis and corrects the anemia by prolonging RBC survival.
 - Autoimmune hemolytic anemia** may be classified by the temperature at which autoantibodies react with RBC surface antigens.
 - Warm autoimmune hemolytic anemia** results from splenic sequestration and destruction of RBCs coated with autoantibodies (usually IgG) that interact optimally with their antigens at 37°C. Anti-IgG antiserum causes agglutination of the patient's RBCs (positive direct Coombs' test). Etiologies include chronic lymphocytic leukemia, non-Hodgkin's lymphoma, collagen vascular disease, and drugs (especially alpha-methyl-dopa and beta-lactamases), although most cases are idiopathic. Presentation is typical of all hemolytic anemias, and primary treatment is directed against the underlying disease. If unsuccessful, the mainstay of therapy is corticosteroids. Nonresponders or patients requiring high steroid doses benefit from splenectomy 60–80% of the time.
 - Cold autoimmune hemolytic anemia** is characterized by fixation of C3 to IgM antibodies that bind RBCs with greater affinity at temperatures approaching 0°C. Hemolysis occurs either immediately by intravascular complement-mediated mechanisms or via removal of C3-coated red cells by the liver. Splenectomy has no therapeutic benefit.
 - Sickle cell disease** is due to homozygous inheritance of the S variant of the hemoglobin beta chain and is usually associated with autosplenectomy secondary to repeated vasoocclusive episodes. Splenectomy may be indicated, however, for treatment of acute splenic sequestration crisis, hypersplenism, symptomatic splenomegaly, and splenic abscess.
 - Thalassemias** are hereditary anemias caused by a defect in hemoglobin synthesis wherein an insufficient amount of a hemoglobin polypeptide chain is produced. Thalassemias are named for the deficient peptide chain and are classified further as thalassemia major or minor, depending on whether the patient is homozygous or heterozygous for the abnormal globin gene.
 - Beta-thalassemia major (Cooley's anemia)** is a homozygous dysfunction of both beta-globin genes. Signs and symptoms include severe anemia, jaundice, hepatosplenomegaly, enlargement of the head, and gallstones. Primary treatment frequently includes iron chelation therapy to combat the iron overload from repeated transfusions. In severe cases, splenectomy both removes the primary site of extravascular hemolysis, thereby resulting in decreased transfusion needs, and cures the discomfort associated with massive splenomegaly.
 - Beta-thalassemia minor** results when a single abnormal beta-globin is inherited. It typically requires no treatment.
 - Pyruvate kinase deficiency** is a rare autosomal recessive enzymatic deficiency that results in lowered erythrocyte production of adenosine triphosphate and hemolytic anemia of variable severity. Splenectomy may improve the anemia, but the response may not be complete.
- D. Hypersplenism** is increased normal splenic function. Signs may include anemia, thrombocytopenia, or leukopenia, for which there is usually compensatory bone marrow hyperplasia. Improvement can occur after splenectomy.
- Primary hypersplenism is a diagnosis of exclusion.** It is a very rare disease with marked female predominance. If an exhaustive workup proves negative, splenectomy is indicated and usually results in substantial improvement. These patients rarely respond to corticosteroids.
 - Secondary hypersplenism** refers to splenic overactivity associated with a known cause of splenomegaly. The most common etiologies are congestive splenomegaly and neoplastic disorders. Primary therapy is always aimed at the underlying disorder.
 - Portal hypertension** is usually the result of cirrhosis or portal or splenic vein obstruction. Approximately 60% of patients with cirrhosis develop splenomegaly, and 15% develop hypersplenism, with thrombocytopenia being the primary manifestation. The transjugular intrahepatic portosystemic shunt procedure may be an effective alternative to surgery in this patient population because splenectomy does not improve overall survival, and risk for perioperative complications is high (*Am J Gastroenterol* 91:134, 1996). Splenic vein thrombosis causes hypersplenism in addition to gastric and esophageal varices. Splenectomy is the therapy of choice when splenic vein thrombosis results in variceal bleeding.
 - Leukemias** are lymphoproliferative conditions that can present with cytopenia, splenomegaly, and lymphadenopathy. Splenectomy in symptomatic patients with chronic myelogenous leukemia, chronic lymphocytic leukemia, and hairy cell leukemia can palliate the cytopenia, decrease the risk of hemorrhage, and relieve symptoms caused by splenomegaly or splenic infarcts, but it has no effect on the natural course of disease.
 - Inflammatory and infiltrative conditions** that cause hypersplenism include Felty's syndrome and Gaucher's disease. Felty's syndrome consists of the triad of rheumatoid arthritis, splenomegaly, and granulocytopenia. Splenectomy is effective in a small subset of patients with severe granulocytopenia, significant recurrent infections, and chronic leg ulcers. Gaucher's disease is an autosomal recessive disorder of glycolipid metabolism secondary to a deficiency in beta-glucocerebrosidase. Splenectomy is effective in correcting the cytopenias associated with the disease and symptoms related to splenomegaly. Bone pain and neurologic abnormalities are unaffected.
 - Agnogenic myeloid metaplasia (myelofibrosis)** is a myeloproliferative disorder characterized by bone marrow fibrosis, leukoerythroblastosis, and extramedullary hematopoiesis. The latter results in splenomegaly, which can become quite painful, as well as hypersplenism with resulting cytopenias. Initial management may include transfusions, steroids, cytotoxic agents, androgens, and splenic irradiation. When hypersplenism is severe and refractory, splenectomy eliminates the symptoms of splenomegaly and induces a remission in the hemolytic anemia. It can be complicated by postoperative hypercoagulability. The progression of the disease is not altered, and cytopenia requiring transfusion frequently recurs. Adequacy of bone marrow hematopoiesis must be ascertained before splenectomy is considered.
- E. Lymphoma**
- Staging** for Hodgkin's and non-Hodgkin's lymphoma is based on the extent of disease and presence of symptoms. Patients are classified as A if asymptomatic; B if fevers, weight loss, or night sweats are present; E if extralymphatic involvement (stomach, small intestine) is present; and S if the spleen is involved.
 - Stage I:** localized to a single lymph node or nodal group
 - Stage II:** more than one nodal group on the same side of the diaphragm
 - Stage III:** more than one nodal group on both sides of the diaphragm
 - Stage IV:** spread to organs other than lymph nodes or spleen
 - Hodgkin's disease** generally presents with cervical adenopathy and spreads in a predictable fashion among nodal groups. Stage IA and stage IIA disease are typically treated with radiation. Stage IVA and all stage B patients are treated with chemotherapy with either a regimen of mechlorethamine, vincristine, procarbazine, and prednisone or a regimen of doxorubicin (Adriamycin), bleomycin, vinblastine, and dacarbazine. Stage IIIA disease is amenable to either treatment modality, whereas patients with bulky mediastinal adenopathy require multimodality therapy. Initial staging is done with computed tomographic (CT) scan and lymphangiography, which can predict nodal involvement in 90% of cases. **Staging laparotomy** is indicated when the assigned preoperative stage and treatment may be changed based on operative findings. The operation consists of a thorough abdominal exploration, splenectomy, liver biopsy, bone marrow biopsy, and multiple lymph node samplings. In the past, laparotomy revised the patient's stage upward in 25–30% of cases and downward in 10% of cases. However, due to continued improvements in radiologic staging and salvage chemotherapy in patients with radiation-treated supradiaphragmatic disease who later present with previously undetected abdominal disease, staging laparotomy is now performed infrequently.
 - Non-Hodgkin's lymphomas** have a less predictable pattern of spread and are more variable in clinical course. Because the disease is usually advanced at the time of diagnosis, staging laparotomy is rarely indicated. Splenectomy is occasionally indicated for secondary hypersplenism or massive splenomegaly to improve symptoms and cytopenia. However, disease progression remains unaltered.

II. Cysts, tumors, and abscesses

- Splenic cysts** are uncommon and can be parasitic or nonparasitic. Most are located in the lower pole in a subcapsular position.
 - Parasitic cysts comprise more than two-thirds of splenic cysts worldwide but are rare in the United States.** The majority are hydatid cysts caused by

Echinococcus species. They are typically asymptomatic but may rupture or cause symptoms due to splenomegaly. Primary treatment options include splenectomy or resection of the roof of the cyst with splenic salvage, which may be performed via laparoscopy. High-risk patients can be treated with mebendazole, 10 mg/kg per day p.o. for 2 weeks, and cyst aspiration, followed by injection with hypertonic saline solution and reaspiration.

2. **Nonparasitic cysts** can be true cysts or pseudocysts.
 - a. **Pseudocysts lack an epithelial lining** and comprise more than two-thirds of nonparasitic cysts. They typically result from traumatic hematoma formation, with subsequent absorption. Symptomatic splenic pseudocysts may present with left upper quadrant pain radiating to the shoulder. Pseudocysts smaller than 5 cm can be followed safely with ultrasonography and often resolve spontaneously. Larger cysts may rupture and require excision with splenorrhaphy or partial excision and marsupialization. Splenectomy is indicated if splenic salvage is not possible. Percutaneous aspiration is associated with a high incidence of infection and reaccumulation and is not indicated.
 - b. **True cysts have an epithelial lining** and are most often congenital. Other rare true cysts include epidermoid and dermoid cysts. They are typically asymptomatic and found during radiologic examinations or surgery. Generally, no treatment is needed. For cysts that become symptomatic, splenic salvage can be attempted for cysts less than one-half the size of the organ; otherwise, splenectomy is indicated.
- B. **Primary splenic tumors are extremely rare.** Splenic metastases from breast, melanoma, and lung primary tumors are found in up to 7% of cancer patients at the time of autopsy.
 1. **Vascular tumors are the most common primary splenic neoplasm.** The majority are benign hemangiomas or lymphangiomas requiring no treatment. Neoplasms causing splenomegaly are an indication for splenectomy. Hemangiosarcomas usually involve the liver and are not curable.
 2. **Hamartomas** are rare benign tumors composed mainly of vascular elements. Treatment is similar to that for other benign splenic neoplasms.
 3. **Splenic lymphoma** is localized to the spleen and surrounding retroperitoneal lymph nodes without peripheral involvement. Typically, the neoplasm is discovered during operation for splenomegaly of unknown origin. Adjuvant chemotherapy or radiation may be beneficial.
- C. **Splenic abscesses are rare but potentially lethal.** Approximately two-thirds are due to seeding from a distant bacteremic focus, most commonly endocarditis or urinary tract infection. Other etiologies include posttraumatic events, hemoglobinopathies, and contiguous spread (*World J Surg* 14:513, 1990).
 1. **Fever is present in nearly all cases.** Abdominal discomfort and splenomegaly occur in one-half of all patients.
 2. **Radiologic evaluation.** CT scan and ultrasonography are the best diagnostic modalities for splenic abscess. CT scan typically shows an image of low homogeneous density with edges that do not intensify with intravenous contrast.
 3. **Treatment.** Antibiotic therapy should be instituted immediately after obtaining blood cultures. Nearly 60% of identified infectious agents are aerobes, with one-half being *Staphylococcus* and *Streptococcus* species. Unilocular abscesses may be amenable to treatment by percutaneous drainage and antibiotics. Splenectomy in combination with postoperative antibiotics represents definitive therapy. Fungal abscesses, however, can often be treated with antifungal agents alone without splenectomy or drainage.

III. Splenectomy

A. Preoperative and perioperative preparation

1. **Vaccinations**
 - a. **Polyvalent pneumococcal vaccine**, Pneumovax, covers 85–90% of pneumococcal types and should be administered at least 2–3 weeks preoperatively to all patients older than 2 years old. The vaccine should be repeated in 6 years after splenectomy. Prevnar is a pneumococcal vaccine approved for use in infants and children from 6 weeks to 2 years old.
 - b. **Meningococcal vaccine** can be given as a one-time vaccination to patients older than 2 years old.
 - c. **Haemophilus influenzae type B conjugate vaccine** should be considered if the patient did not receive it during infancy.
 - d. **Influenza vaccination** should be considered.
 2. **Transfusion antibodies** are present in many patients undergoing splenectomy, which may complicate cross-match for blood products. Platelets are transfused after the splenic artery is clamped to prevent their consumption.
 3. **Steroids** are continued at stress doses if needed.
 4. **Preoperative splenic artery embolization** can be a beneficial adjunct in high-risk cases, such as splenectomy for massive splenomegaly secondary to myelofibrosis (*Am Surgeon* 10:602, 1988).
- B. **Splenectomy** can be performed using a traditional open approach (via midline, subcostal, paramedian, or transverse incision) or using laparoscopic techniques. Laparoscopic splenectomy has become increasingly more common for removal of spleens, as it may provide reduced morbidity while being no less efficacious in removal of the spleen. Massive splenomegaly is no longer contraindicated laparoscopically, as hand-assisted laparoscopy makes this approach possible. Ongoing hemorrhage from splenic trauma and portal hypertension are contraindications for the laparoscopic approach. The patient is placed in the right lateral decubitus position on a beanbag. An ultrasonic scalpel coagulator may facilitate division of the short gastric vessels. An entrapment sack is used to remove the spleen either intact by extending an abdominal port incision or via morcellation if an intact specimen is not required for pathologic diagnosis. Depending on the degree of splenomegaly, the splenic vessels may be ligated before or after mobilization of the organ. All approaches should conclude with a search for **accessory spleens**. These are found in up to 10–20% of patients (most often in the splenic hilum), and failing to remove them can result in an unsuccessful operation when splenectomy is performed for a hematologic indication. Accessory spleens should be left in place, however, when splenectomy is performed secondary to trauma to preserve splenic immunologic function. Drainage of the splenic fossa is considered if there has been extensive manipulation of the tail of the pancreas and for detection of postoperative hemorrhage in patients with coagulation disorders.

C. Complications of splenectomy

1. **Early complications** occur within 60 days of operation.
 - a. **Atelectasis is the most common postoperative complication** (20–40%) and can be prevented by intraoperative removal of diaphragmatic irritants (blood, irrigation solution) and good postoperative pulmonary toilet.
 - b. **Thrombocytosis** is common after splenectomy. Platelet count frequently remains at 400,000/ μ L and may reach 2–3 million/ μ L in the immediate postoperative setting. Although the degree of thrombocytosis does not correlate with an increased risk of thrombotic complications, aspirin is usually given when the platelet count rises above 1 million/ μ L. Granulocytosis is also observed frequently, eventually shifting into a lymphocytosis and monocytosis.
 - c. **Subphrenic abscesses** are rare and are more likely to occur with concomitant gastrointestinal tract surgery. Presentation may be clinically subtle in patients receiving systemic steroids.
 - d. **Pancreatitis and pancreaticocutaneous fistulas** occasionally occur secondary to pancreatic injury (1–3%).
 - e. **Gastric perforation** may result from direct intraoperative injury during division of the gastrosplenic ligament or from loss of collateral blood supply to the greater curvature of the stomach on division of the short gastric vessels. Some surgeons prefer postoperative nasogastric decompression until gastric motility returns to try to prevent ischemia associated with gastric distention.
2. **Overwhelming postsplenectomy infection (OPSI)** is a late complication that may occur throughout a patient's lifetime. Patients present with nonspecific flulike symptoms rapidly progressing to fulminant sepsis, consumptive coagulopathy, bacteremia, and ultimately death within 12–48 hours in asplenic or hyposplenic individuals. The majority of cases occur within the first 2 years of splenectomy.
 - a. **Overall incidence of OPSI is 3.8%.** In children, the incidence is 4%, with subsequent mortality of 45%. In adults, the corresponding values are 1.9% and 60% (*Surg Clin North Am* 63:1313, 1983).
 - b. **Encapsulated bacteria**, especially *Streptococcus pneumoniae* (>50%), *H. influenzae* type B, and *Neisseria meningitidis*, are the most commonly involved organisms. The loss of splenic reticuloendothelial cells (which have the unique ability to clear particulate antigen in the absence of antibody), loss of opsonin synthesis, and decrease in the lymphocyte T4-T8 ratio limit the ability of asplenic patients to respond appropriately to infection.
 - c. **Successful treatment of OPSI requires prompt recognition** and is generally supportive. Initial treatment is high-dose third-generation cephalosporins.
 - d. **Daily prophylactic antibiotics** have been recommended after operation in all children younger than age 5 years and in immunocompromised patients (*Surg Clin North Am* 61:135, 1981) because these patients are unlikely to produce adequate antibody in response to pneumococcal vaccination. Some authorities recommend lifelong prophylaxis with penicillin in all splenectomized patients (*Surg Clin North Am* 63:1313, 1983). **All patients who have had splenectomy should be educated about the risk of OPSI and the need for early physician consultation** in the event that fever or other prodromal symptoms should occur.

21 Fistulas, Short-Bowel Syndrome, and Bariatric Surgery

Ketan M. Desai and J. Christopher Eagon

Gastroenterologic Disorders

I. A fistula

II. Short-bowel syndrome

III. Radiation enteritis

IV. Bariatric surgery

Gastroenterologic Disorders

I. A fistula is a communication between two epithelialized surfaces. Fistulas may be categorized according to anatomy, output, or etiology.

A. Anatomy

1. **External fistulas** are the most common fistulas and connect an internal organ system with the skin (e.g., enterocutaneous fistula).
2. **Internal fistulas** connect two hollow structures of the same or different organ systems, without external communication (e.g., colovesical fistula). In some cases, these are created intentionally (e.g., gastroenterostomy and choledochojejunostomy).
3. **Proximal fistulas** are located in the upper gastrointestinal (GI) tract. They are usually associated with high outputs (3 L per day or more) and severe symptoms and sequelae, and they often have a poor prognosis.
4. **Distal fistulas** arise in the distal ileum, colon, or rectum. They are often associated with fewer complications than are proximal fistulas, and they often close with nonoperative treatment.
5. **Simple fistulas** have a single tract, whereas complicated fistulas have multiple and varied tracts connecting one or more organs.

B. Output

1. **High-output fistulas** drain more than 500 mL per day.
2. **Low-output fistulas** drain less than 500 mL per day.

C. Etiology

1. **Abdominal surgical procedures** are the leading cause of fistula formation, accounting for 67–80% of cases. The risk is greatest for operations performed for inflammatory bowel disease, ischemia, malignancy, or extensive intestinal adhesions. Dissection of diseased bowel may result in unrecognized bowel perforation, devascularization, and serosal disruption. Anastomotic disruption, leak, and perianastomotic abscess also are common causes. Malnutrition significantly increases the risk of fistula formation.
 2. **Inflammatory bowel disease.** Fistulas typically occur where the inflammatory disease is worst, usually in the lower GI tract. Internal fistulization is especially common with Crohn's enteritis.
 3. **Diverticular disease** causes fistulas when localized abscesses drain into adjacent organs. Common examples include colovesical and colovaginal fistulas. Internal fistulas should be suspected in patients with diverticular disease who exhibit persistent and recurrent sepsis.
 4. **Malignancy.** Fistulas form as cancers perforate or invade adjacent structures. Healing does not occur if cancer is present, and surgical resection is the only means of cure.
 5. **Radiation enteritis** predisposes to fistula formation after surgery, regardless of the timing of exposure.
 6. **Trauma.** Fistula formation occurs more frequently after penetrating trauma than after blunt trauma. Unrecognized enterotomies as well as injuries that are repaired amid contamination may be prone to fistula formation. Viscus rupture caused by blunt trauma may go unrecognized, and subsequent abscess formation and drainage into adjacent structures result in fistula formation. This most commonly occurs after rupture of the duodenum, colon, or pancreas into the retroperitoneum.
 7. **Congenital fistulas.** Tracheoesophageal, rectovaginal, and vitellointestinal duct fistulas manifest shortly after birth. Rarely, a Meckel's diverticulum or a patent omphalomesenteric duct presents later in life as a *de novo* enterocutaneous fistula.
 8. **Other causes of GI fistulas** include foreign body, vascular insufficiency, and amebiasis.
- D. **Prevention.** Identification of high-risk individuals, meticulous surgical technique, and proper use of perioperative antibiotics are important. Thorough preoperative bowel preparation significantly reduces the risk of fistula formation.
- E. **Pathophysiology.** Fistula-associated complications may be life-threatening and require rapid intervention to avoid morbidity and mortality. The overall mortality for all fistulas is 5–20%.

1. Loss of GI contents

- a. **Hypovolemia.** High-output fistulas may discharge large volumes of fluid, which cannot be adequately replaced by enteral means, leading to dehydration and intravascular volume depletion.
- b. **Acid-base and electrolyte abnormalities.** Loss of large fluid volumes and associated electrolytes, acids, and bases results in metabolic derangements. Severity is directly correlated with the quantity of fistula output.

2. **Malnutrition** is caused by insufficient caloric intake to meet increased metabolic demands associated with fistula formation, such as sepsis. Additionally, substantial portions of the GI tract may be functionally excluded from contact with enteric contents, with ensuing malabsorption, leading to alterations in carbohydrate, fat, and protein metabolism; hypovitaminosis; and mineral deficiency.

3. **Sepsis** is the main determinant of mortality from a fistula. Sepsis accompanies a large percentage of fistulas and is caused by seeding of the fistula tract by organisms indigenous to the bowel. A septic focus diminishes the potential for healing.

F. Initial management

1. **Fluid resuscitation** to correct hypovolemia and ensure adequate tissue perfusion is a priority. Once the fluid deficit is corrected, accurate measurement of ongoing fluid losses and prompt replacement are essential to maintain euvolemia. Intravenous fluid administration is typically necessary because attempts at enteral replacement result in increased fluid loss from the fistula. Serum electrolytes and acid-base status must be followed closely and abnormalities corrected. Fluid replacement composition depends on the site of the fistula in the GI tract and the quantity of fluid loss, and content must be tailored to meet the specific replacement demands (see [Table 4-1](#)). High-output gastric fistulas occasionally may require addition of hydrochloric acid. Biliary and pancreatic fistula effluent is hypertonic and associated with large bicarbonate and sodium losses.
2. **Complete bowel rest** is instituted in the initial management of patients with fistulas. This may reduce fistula drainage and may simplify the evaluation and stabilization of the patient. Nasogastric suction has not proved beneficial except in the presence of distal obstruction.
3. **Nutritional support** should be instituted within 24 hours of diagnosis. Early, aggressive parenteral nutritional therapy has dramatically decreased mortality from fistulas from 58% to 16% (*Am J Surg* 108:157, 1964).
 - a. **Enteral feeding**, if possible, is the primary method of choice to provide nutrients. Patients with colcutaneous and ileal low-output fistulas, including the terminal ileum or cecum, may be safely treated with enteral feeding. Adequate nutrient absorption requires at least 4 feet of functional intestinal tract. Standard enteral formulas are sufficient in most cases; however, if the available bowel is short, elemental feeding may maximize absorption. In cases of proximal fistula, such as esophageal, gastric, duodenal, and proximal jejunal sites, enteral feeding can be given below the fistula if distal enteral access is available (i.e., feeding jejunostomy tube).
 - b. **Parenteral nutrition** is the secondary modality available to provide adequate calories and nutrients along with complete bowel rest. Indications include intolerance to enteral nutrition, jejunal and ileal high-output fistulas, and proximal fistulas if distal enteral access is not possible. The benefit of GI tract absorption is lost. However, providing adequate nutrition parenterally is vital when the GI tract cannot be used.
4. **Sepsis must be controlled early.**
 - a. **Intraabdominal abscess** should be excluded in each patient with a GI fistula by abdominopelvic computed tomographic scan. Abscess drainage, whether percutaneous or open, is a therapeutic priority.
 - b. **Intravenous antibiotics** are indicated when infection is present. Antimicrobial therapy is directed at bowel flora and should consist of treatment against gram-negative bacteria and anaerobes. Continuous bacterial seeding from the GI tract promotes persistent and recurrent sepsis, which is an indication for operation if uncontrolled with appropriate antimicrobial therapy.
 - c. **Infected wounds** are adequately opened and packed to allow complete drainage, débridement, and healing by secondary intention. Frequent dressing changes may be required.
5. **Fistula drainage** is controlled by wound management and pharmacotherapy.
 - a. **Dressings** may be used for low-output fistulas to absorb drainage fluid. However, prolonged contact of enteric contents with a surrounding wound or the skin may impede healing and cause skin breakdown. Intubation of matured fistula tracts may be beneficial. A suction or sump drainage system for

high-output fistulas is preferred.

- b. **Randomized prospective trials** have demonstrated the efficacy of octreotide, 150 µg s.c. t.i.d., in decreasing fistula output (*Lancet* 2:672, 1987). In addition, H₂-receptor antagonists reduce gastric and duodenal fistula output and provide stress ulceration prophylaxis; however, their efficacy has not been proved.
6. **Skin protection is instituted promptly.** Irritation and excoriation of skin surrounding the site of fistula drainage are common, can be very painful, complicate wound management, and may become secondarily infected. Skin surrounding a fistula is protected with a barrier device or powder [e.g., DuoDerm (ConvaTec, Princeton, NJ)]. The skin should be examined and cleansed frequently. An enterostomal therapist is helpful in managing complex wounds.
- G. **Fistula assessment.** In the initial 4–6 weeks, 50–70% of fistulas close. The frequency of closure may not necessarily be related to the quantity of initial fistula output. The difficult decision on how long to wait for spontaneous closure depends on individual circumstances and complexity of the underlying illness. There has been a trend toward allowing longer periods for fistula closure with improved home intravenous therapy, parenteral nutrition, and use of somatostatin analogue. If spontaneous closure does not appear likely, surgical therapy is indicated. The anatomy of the fistula is defined to assist planning of the appropriate surgical procedure.
 1. **Contrast radiography** is most commonly used. Fistulography may be performed in mature fistula tracts (usually after 7–10 days) and usually provides good visualization of all tracts and sites of communication with the GI tract. If the fistula tract is not mature, fistulography may be contraindicated. Oral contrast studies can demonstrate contrast extravasation through the fistula and are most valuable to assess internal fistulas and distal obstruction. Contrast enema is the study of choice for rectal or colonic fistulas. Pyelography and cystography may be used in cases that involve the urinary tract.
 2. **Endoscopy** (i.e., esophagogastroduodenoscopy, colonoscopy, or cystoscopy) aids in the assessment of coexistent disease in the organ from which the fistula arises, such as peptic ulceration, inflammatory bowel disease, and cancer. Fistula openings may be difficult to identify by endoscopy.
 3. **Abdominopelvic computed tomographic scan** is the study of choice to evaluate for abscess. Its value is limited when abscess is not strongly suspected.
- H. **Surgical treatment** is indicated when fistulas fail to heal with nonoperative measures and when sepsis cannot be controlled. Common conditions under which fistulas fail to close include malignancy, radiation, obstruction distal to the fistula, inflammation, foreign body, and epithelialization of the fistula tract. The goals of surgery are to eradicate the fistula tract and to restore epithelial continuity of the associated organ systems.
 1. **Gastric fistulas.** High-output gastric fistulas arise from anastomotic breakdown or ulcer perforation and require surgical repair. Most low-output gastric fistulas close spontaneously (e.g., gastrostomy closure after removal of gastrostomy tubes). In cases that do not close, primary repair or serosal patch placement usually is successful.
 2. **Duodenal fistulas** are caused by breakdown of an anastomosis, trauma, or, less commonly, peptic ulceration. The high enzyme content of the effluent can cause severe skin excoriation. Most duodenal fistulas close spontaneously with conservative measures. If the fistula is secondary to inflammatory bowel disease, chances for spontaneous closure are poor. When surgical intervention is required, primary closure of small duodenal wall disruptions may be performed, but duodenal stricture is associated with primary closure of large defects. Close proximity of the defect to the ampulla may also prevent primary closure. In these cases, duodenal wall integrity may be restored by serosal patch using another portion of bowel. Alternatively, a Roux-en-Y duodenoenterostomy may be performed to divert duodenal output into the bowel.
 3. **Biliary and pancreatic fistulas** almost always close with nonsurgical therapeutic measures, which may include percutaneous interventional or endoscopic drainage techniques. Octreotide therapy may be beneficial.
 4. **Small-bowel fistulas** are typically cured with bowel resection and primary reanastomosis. In rare severe cases, a temporary diverting enterostomy may be necessary. For internal fistulas, if the openings are in close proximity, the involved region can be resected in continuity.
 5. **Large-bowel fistulas** generally are associated with high spontaneous closure rates. Fluid and electrolyte abnormalities are rare because outputs tend to be low. However, sepsis rates may be greater. If surgical closure is required, an adequate mechanical bowel preparation is important. Primary closure, as opposed to resection with primary reanastomosis, depends on associated conditions, the nutritional status of the patient, and the location and complexity of the lesion.
 6. **Gastrostomy or enterostomy feeding tubes** placed at the time of definitive repair may facilitate postoperative management. Antibiotic therapy and nutritional support should continue into the postoperative period.

II. Short-bowel syndrome

- A. **Etiology and pathophysiology.** Short-bowel syndrome is characterized by dehydration, electrolyte derangements, acidic diarrhea, steatorrhea, malnutrition, and weight loss. Congenital anomalies leading to short-bowel syndrome include intestinal atresia, midgut volvulus with intestinal necrosis, and necrotizing enterocolitis. In middle-aged adults, inflammatory bowel disease and trauma are the leading causes of massive intestinal resection. In the elderly, prominent causes include mesenteric ischemia, strangulated hernia, and extensive resection for malignancy.
- B. **In the adult,** the length of the small bowel varies from 300 to 600 cm, which correlates directly with body surface area. Several factors determine the severity of short-bowel syndrome, including the extent of resection, the part of the GI tract removed, the type of disease necessitating the resection, the presence of coexistent disease in the remaining bowel, and the adaptability of the remaining bowel. Generally, resection resulting in less than 120 cm of intact bowel results in short-bowel syndrome. Resection of up to 50% of the small bowel in adults can be tolerated without serious complications, and up to 70% resection can be tolerated if the terminal ileum and cecum are preserved. Infants may survive after resection of up to 85% of bowel owing to the enhanced ability of the bowel to adapt and grow with the child. Loss of the ileocecal valve results in rapid emptying of enteral contents into the colon and reflux of colonic bacterial flora into the small bowel. Because of its specialized absorptive function, resection of the ileum is also not well tolerated. However, the entire jejunum can be resected without serious adverse nutritional sequelae.
- C. **Adaptation.** The distal small intestine has the greatest adaptive potential and can assume many of the absorptive properties of the proximal GI tract. Cellular hyperplasia and bowel hypertrophy occur over a 2- to 3-year period, increasing the absorptive surface area. Fat absorption is the metabolic process most likely to be permanently impaired; other functions adjust and normalize fairly well. Bowel motility also changes, with the more proximal intestine developing motility patterns similar to the normal ileum.
- D. **Fluid and electrolyte response.** Of the 8–10 L of chyme presented daily to the small intestine, only 1–2 L are delivered into the colon. Significant quantities of electrolytes are absorbed in this process. With short-bowel syndrome, this physiology is altered. Strict intake and output records as well as close monitoring of serum electrolytes are critical in the early management of patients with short-bowel syndrome. The pH of enteric drainage should be monitored, whether from fistulas, stoma, or feces.
- E. **Malabsorption and malnutrition**
 1. **Gastric hypersecretion,** seen in the early postoperative period, can persist for prolonged periods. Increased acid load may injure distal bowel mucosa, leading to hypermotility and impaired absorption. The severity of hypersecretion correlates directly with the extent of bowel resection. This generally is more pronounced after jejunal resection than after ileal resection. Loss of an intestinal inhibitory hormone has been implicated.
 2. **Cholelithiasis.** Altered bilirubin metabolism after ileal resection increases risk for pigment gallstones secondary to a decreased bile salt pool, which causes a shift in the cholesterol saturation index. Chronic total parenteral nutrition (TPN) also predisposes to increased risk of cholelithiasis.
 3. **Hyperoxaluria and nephrolithiasis.** Excessive fatty acids within the colonic lumen bind intraluminal calcium. Unbound oxalate, normally made insoluble by calcium binding and excreted in the feces, thus is absorbed readily, resulting in hyperoxaluria and calcium oxalate urinary stone formation.
 4. **Diarrhea and steatorrhea** are caused by rapid intestinal transit, presence of hyperosmolar enteric contents in the distal bowel, disruption of the enterohepatic bile acid circulation, and bacterial overgrowth. Fat absorption is most severely impaired by ileal resection. The delivery of bile acids into the colon produces a reactive watery diarrhea that may be severe. Unabsorbed fats in the colon further inhibit absorption and stimulate secretion of water and electrolytes.
 5. **Intestinal microflora.** Loss of the ileocecal valve permits reflux of colonic bacteria into the small bowel. Intestinal dysmotility further promotes bacterial colonization. Bacterial overgrowth and change in the indigenous microbial population result in pH alteration and deconjugation of bile salts, with resultant malabsorption, fluid loss, and decreased vitamin B₁₂ absorption. Infectious diarrhea, bacterial or viral, is a major cause of morbidity.
- F. **Effects on intestinal motility.** Normal motility depends on the quantity and concentration of enteric contents in the bowel lumen. Trophic factors also play a role. In short-bowel syndrome, these parameters are altered. Because the ileum has the greatest capacity for absorption of fluid and electrolytes and for postresection adaptation, short-bowel syndrome symptoms are most likely to occur after ileal resection.
- G. **Early postoperative management**
 1. **Fluids and electrolytes.** The primary goal is to stabilize the metabolic, respiratory, and cardiovascular parameters related to fluid shift and sepsis that frequently accompany massive small-bowel resection. All fluid losses should be strictly accounted for and replaced.
 2. **Ileus** may be prolonged because of deranged motility patterns and changes in intraluminal milieu. Parenteral nutrition should be provided until GI tract function resumes. If ileus persists for an unduly prolonged period, mechanical obstruction or sepsis should be ruled out. Short-bowel syndrome ileus generally resolves more quickly in children than in adults.
 3. **Gastric hypersecretion.** H₂-receptor antagonists or proton pump inhibitors reduce the hypersecretion response and protect against peptic ulceration. Antacids neutralize acid on contact and should be administered for nasogastric aspirate pH of less than 5.
 4. **Nutritional support** should be instituted early to maintain positive nitrogen balance and to promote wound healing and adaptation of the remaining bowel. Enteral nutrition provides a positive trophic effect to the bowel mucosa and should be started as soon as possible. Feeding tubes placed at laparotomy can

be very helpful. Even if caloric goals are not met, enteral formula stimulates the remaining intestine and facilitates adaptation. Initial feeds should be low volume, low fat, and isosmotic and then advanced as tolerated. Elemental feeding may be required in severe cases of short-bowel syndrome.

H. Long-term management

1. **Diarrhea** has many causes in short-bowel syndrome. Frequently, dietary modification improves symptoms. H₂-receptor antagonists reduce acid production and the volume of enteric contents. Chelating resins, such as cholestyramine, reduce intraluminal bile salts and subsequent diarrhea but affect the available systemic bile salt pools. Antisecretory medications, such as loperamide and somatostatin analogue, may be beneficial. Low-dose oral narcotics, such as diphenoxylate hydrochloride and atropine (Lomotil) or codeine, are efficacious but addictive. Bacterial overgrowth should be evaluated by stool culture, and prophylactic antimicrobials, such as metronidazole, should be administered as needed.
 2. **Nutritional support.** Vitamins, trace elements and minerals, and essential fatty acids should be parenterally administered until adequate enteral absorption is established. If the patient is unable to assume an oral dietary regimen, parenteral vitamin and mineral supplementation is warranted. The absorption of fat-soluble vitamins A, D, E, and K is especially likely to be compromised. Vitamin B₁₂ and calcium absorption are also affected by altered fat absorption and should be supplemented. Chronic TPN can be administered nightly to permit normal daily life.
 3. **Late complications**, mostly secondary to metabolic derangements, are common. Anastomotic leak, fistula, stricture, and obstruction can occur well beyond the early postoperative period. Massive bowel resection typically is undertaken in the presence of soiling, necrosis, and abscess. Late obstruction (partial or complete) is fairly common, and reoperative rates are high. Other problems include nephrolithiasis, cholelithiasis, nutritional deficiency (e.g., anemia, bone disease, coagulopathy), liver dysfunction, TPN-related complications, and central venous catheter-related problems (e.g., sepsis and thrombosis).
- I. **Surgical therapy.** Various surgical procedures have been described for the management of short-bowel syndrome, although they have not been widely adopted. Most applications are in the pediatric age group. Most important is prevention by minimizing the extent of initial bowel resection.
1. **Slowing of intestinal transit.** If intestinal transit times are slowed, enteric contents have a greater opportunity to be absorbed.
 - a. An **antiperistaltic segment** is created by reversing a segment of the intestinal tract; counterpulsation then acts to slow transit time. The antiperistaltic effect, however, has been shown to decrease with time.
 - b. The **recirculating loop** has poor results because of a high incidence of stasis enteritis.
 - c. **Colonic interposition** provides a slower intrinsic rate of motility and a larger luminal diameter. Results are limited.
 - d. **Intestinal pacing** in the distal GI tract produces retrograde peristalsis, thereby slowing transit times. This procedure is experimental.
 - e. **Increased intestinal surface area.** A tapering and lengthening procedure (tapering enteroplasty) may double bowel length (*J Pediatr Surg* 15:145, 1980). It is technically difficult to perform, however, especially when extensive adhesions or mesenteric thickening is present. Results have been moderate.
 - f. **Intestinal transplantation.** Future advances in immunosuppression and preservation techniques may make isolated bowel transplantation, which is still somewhat controversial, a more attractive alternative in some patients, especially those with massive resection and virtually no remaining bowel.

III. Radiation enteritis

- A. **Etiology and pathophysiology.** Approximately 2–5% of patients who undergo abdominopelvic external beam radiation, typically for the treatment of malignancies, have radiation-induced bowel damage. Toxicity depends on dose. Small bowel is most susceptible, followed by colon and, lastly, rectum. Radiation-induced injury causes an obliterative endarteritis, resulting in chronic tissue ischemia, followed later by ischemic fibrosis and vascular and lymphatic ectasia. The presence of preexisting vascular disease (diabetes, hypertension, and cardiovascular disease) increases risk. Risk is also greater in thin patients, during concomitant chemotherapy with damage to replicating mucosa, and when prior abdominal surgery has left fixed loops of bowel in the pelvis.
- B. **Acute radiation injury**, seen within hours or days of radiation delivery, causes depletion of actively proliferating cells in the bowel mucosa. A leukocytic infiltrate with crypt abscess formation follows. Symptoms include periumbilical abdominal pain, diarrhea, nausea, and vomiting. Contrast and endoscopic studies reveal edematous bowel mucosa. Therapy revolves around treatment of symptoms and avoidance of further injury. A 10% reduction in radiation dose usually prevents further episodes of acute radiation enteritis.
- C. **Chronic radiation injury** may be observed decades after radiation exposure. Bowel wall thickening, neovascularization, and telangiectasia may progress slowly. Fibrous bands and strictures form secondary to mesenchymal injury. More commonly, preexisting radiation damage impairs the ability of the bowel to heal after some other primary insult, such as inflammation, malignancy, or surgical manipulation.
- D. **Clinical presentation** is highly variable and depends on the bowel segment involved.
 1. **Small-bowel enteritis symptoms** range from nausea, vomiting, and abdominal pain to obstruction, perforation, and fistula formation. Oral contrast studies delineate the extent of disease. Findings include bowel wall thickening, decreased peristalsis, and stricture.
 2. **The colon and rectum** are more resistant to radiation injury. Nonetheless, radiation therapy commonly is targeted in these areas by external beam or endocavitary delivery. **Symptoms** include rectal bleeding, diarrhea, tenesmus, abdominal pain, and constipation. Proctosigmoidoscopy reveals edematous, inflamed, and friable mucosa. Barium enema can rule out fistula or stricture.
- E. **Medical management** is aimed at controlling symptoms by dietary modification and medication. Low-fat, low-residue, and lactose-free diets are recommended. In severe cases, bowel rest and TPN may be indicated. Agents that reduce bowel motility may help in mild cases. Antispasmodic and bile salt chelating agents also are used. Prostaglandin synthesis inhibition with aspirin has been shown to reduce symptoms in a prospective randomized trial (*Lancet* 2:942, 1975). Corticosteroids, in combination with TPN, are believed to reduce the inflammatory reaction, thereby alleviating symptoms. Radiation coloproctitis is treated with sitz baths, steroid retention enemas, and stool-bulking agents. If ulcerative lesions are identified, biopsy should be performed to rule out new or recurrent malignancy.
- F. **Surgical therapy** is indicated for partial or complete obstruction, bowel perforation, fistula, and bleeding. Those without an identified lesion do not benefit from surgery. Poor healing associated with irradiated intestine must be taken into account. A thorough history of the original malignancy and the timing and total dose of radiation received guide surgical management. A careful GI workup identifies the location and extent of disease. TPN should be provided if nutritional status is poor. Preoperative ureteral stent placement often is desirable.
 1. **Intraoperative efforts** are individualized and revolve around the pathologic lesion. Adhesions should be anticipated and lysis avoided except where absolutely necessary. Enteral bypass may be a better alternative than resection in rare cases. Adequate blood supply to the anastomotic margin must be ensured. Frozen section of this area has been advocated to ensure absence of disease. If there is any doubt, the bowel should be resected to briskly bleeding edges. A feeding enterostomy tube can usually be placed in the distal GI tract to aid in postoperative management. Postoperative gastric decompression is important, and oral intake should proceed cautiously.
 2. **Surgical indications** for colonic disease include perforation, obstruction, fistula, and radiation proctitis. Bleeding is usually secondary to mucosal telangiectasia, and noninvasive local control should be attempted. Colonic or abdominoperineal resection with diversion may be necessary. Diversion of the GI tract as a primary treatment modality does provide symptomatic relief, but it has not been shown to alter the course of radiation-induced injury to the colon and rectum, nor does it stop bleeding. Early diversion with later definitive repair is the therapeutic standard in severe cases.

IV. Bariatric surgery

- A. **Obesity**, a disease of modernization, is a major health epidemic and is common in the United States. The primary cause of obesity is related to dietary factors, but genetic predisposition is important. Rarely, endocrine dysfunction is the cause. **Morbid obesity** is defined as a body mass index (weight in kg/height in m²) greater than 40, or, roughly, 100 pounds greater than ideal body weight.
- B. **Complications of morbid obesity are significant.** Morbidly obese young men have 12 times the mortality risk of the matched general population. Of the many medical sequelae associated with obesity, nearly all are reversible on resolution of the obese state.
 1. **Cardiopulmonary effects.** Systemic hypertension is the most common complication associated with morbid obesity. Coronary atherosclerosis is 10 times more prevalent in obese populations than in those matched for age, gender, and other risk factors. Patients are also 10 times more likely to experience sleep apnea than are the nonobese. Decreased chest wall compliance results in hypoventilation or pickwickian syndrome, with resultant hypoxia and acidosis, leading to pulmonary hypertension and eventual right-sided heart failure.
 2. **Non-insulin-dependent diabetes mellitus (NIDDM)** is secondary to increased peripheral resistance to insulin. On reversal of the obese state, diabetes resolves in two-thirds of patients. Diabetic microvascular disease leads to cardiac, renal, and ocular problems.
 3. **Degenerative joint disease** from mechanical overbearing of the joint synovial surface results in degeneration, inflammation, and disabling pain.
 4. **Hiatal hernia and gastroesophageal reflux** are secondary to increased intraabdominal pressure.
 5. **Cholelithiasis** is three times more common than in the general population.
 6. **Thromboembolic disorders** arise from venous insufficiency, leading to stasis and thrombosis. Thrombophlebitis is very common, and risk for pulmonary embolism is high.
 7. **Endocrine dysfunction.** Obese females often develop amenorrhea and menometrorrhagia, with associated hirsutism and breast atrophy, whereas obese males sometimes have feminization syndromes caused by excessive estrogen production by adipocytes.
 8. **Psychosocial problems.** Low self-esteem and depression make recovery from the obese condition very difficult.
 9. **Other complications** include fat-induced liver disease, pseudotumor cerebri (benign intracranial hypertension), complications of pregnancy, and gout.
- C. **Medical treatment**
 1. **Medical therapy** can consist of low-calorie diets, behavior modification, exercise, and pharmacotherapy. These therapies may produce temporary reductions

in weight, but medical management is difficult, with extremely high relapse rates after 1–2 years. Newer medications that suppress appetite and promote weight loss may be used in conjunction.

2. **Psychological assessment** is necessary to treat underlying depression and low self-esteem as well as to prepare patients for surgical therapy.

D. **Bariatric surgery** was performed in the 1970s, but initial poor surgical results and unacceptable long-term complication rates caused interest to wane. The development and improved success of newer surgical procedures have resulted in renewed enthusiasm and led to a National Institutes of Health Consensus Development Conference on morbid obesity (*Am J Clin Nutr* 55:615S, 1992).

1. **Indications.** Patients with a body mass index of greater than 40, or greater than 35 with associated comorbidities, who have failed intensive efforts at weight control using medical means are candidates for bariatric surgery.

2. **Benefits** of surgery are related to the reversal of the disease processes associated with severe obesity. Hypertension resolves in 50% and improves in another 25% of patients. NIDDM is reversed in two-thirds of cases, and the incidence of new-onset diabetes is decreased by a factor of 30 compared with untreated controls.

3. **Bariatric surgical procedures** rely on one or more of several effects on GI motor function. Gastric restriction produces a small gastric reservoir, thereby producing early satiety and so reducing oral intake. Malabsorption of varying degrees is induced by bypass of selected portions of the proximal small bowel.

a. **Vertical banded gastroplasty** is associated with a low incidence of metabolic derangement because it maintains GI tract continuity. An end-to-end anastomosis (EEA) stapler is used to create a window in the midbody of the stomach. A vertical staple line then is placed from the angle of His to the window to partition the stomach, leaving a small reservoir. A mesh band is then positioned through the hole, around the lesser curve, to restrict the size of the passage for the food bolus. Complications are rare and include breakdown of the suture line and erosion of the mesh band into the gastric wall, resulting in **perforation**. Metabolic complications and malnutrition are less frequent than with procedures that include a malabsorptive component. Mortality is less than 1%. Weight loss is approximately 45% of excess body weight at 1 year.

b. **Roux-en-Y gastric bypass (RYGBP)** is currently the most popular procedure. A 30-mL proximal gastric pouch is created either by transection or by using a stapling device. A 1-cm diameter anastomosis is then performed between the pouch and a Roux-en-Y limb of small bowel. This results in a small reservoir, a small passage for pouch emptying, and bypass of the duodenum and proximal jejunum. The length of the Roux limb directly correlates with the degree of postoperative weight loss. Mortality is 1%, most commonly secondary to a leak at the gastrojejunostomy, which occurs in 2–4% of patients. Late complications include incisional hernia, stomal stenosis, and ulcer. Nutritional complications include folate, vitamin B₁₂, iron, and calcium deficiency. Weight loss is 70% of excess weight at 1 year. Reversal of NIDDM occurs rapidly, often within 2 weeks. Dumping syndrome occurs in most patients, but is desirable, and reinforces dietary behavior modification.

c. **Jejunioileal bypass** is a purely malabsorptive procedure. At one time the most frequently performed weight reduction procedure, it has a high rate of long-term complications, including diarrhea, bypass or stasis enteritis (characterized by abdominal pain and distention), excessive flatulence, electrolyte abnormalities, cholelithiasis, hepatic dysfunction, and nephrolithiasis secondary to hyperoxaluria. The long-term mortality rate is 10%. As a result, this procedure is no longer performed for obesity, and those with severe sequelae should be reversed and converted to a gastric bypass.

E. Laparoscopic bariatric surgery

1. **Indications** for bariatric surgery are the same as those for traditional open procedures. Patients with a body mass index of greater than 40, or greater than 35 with associated comorbidities, are candidates for laparoscopic surgery. The National Institutes of Health consensus conference confirmed that surgical therapy is a successful treatment option for severe obesity. However, it should be considered only if nonsurgical means of weight loss have failed.

2. **Preoperative medical evaluation** similarly includes input from the primary care physician, dietitian, and psychiatrist or psychologist, with special attention to the patient's weight history, dietary habits, motivation, social history, and evidence of comorbid medical conditions.

3. **Contraindications** to laparoscopic bariatric surgery vary by institution. A body mass of index of greater than 60, previous upper abdominal surgery, evidence of an excessively large liver, and the presence of a ventral hernia are relative contraindications.

4. **Gastric stapling operations** rely on the concept of restricting the volume of oral intake. The vertical banded gastroplasty, RYGBP, and the adjustable gastric banding are procedures amenable to a laparoscopic approach. RYGBP appears to be most beneficial in producing long-term weight loss. Patients who have undergone RYGBP tend to have dumping, which motivates them to avoid sweets and other high-calorie foods.

5. **Laparoscopic RYGBP** is a technically challenging operation. The patient is initially positioned supine in reverse Trendelenburg with the left lateral segment of the liver retracted superiorly, thereby exposing the gastroesophageal junction. Dissection is carried out between the left gastric artery and the lesser curvature 4 cm distal to the gastroesophageal junction and on the greater curve at the angle of His to create windows into the lesser sac. A Baker tube is inserted into the stomach and the balloon inflated with 15 mL of saline to size the proximal gastric pouch. A gastrotomy is created directly over the balloon. The anvil of a 21-mm EEA stapler is passed into the gastric lumen via a second gastrotomy created in the distal stomach. The post of the anvil is passed through the upper gastrotomy and secured in place with a purse string suture. The stomach is then transected distal to the anvil and the lower gastrotomy is oversewn. The jejunum is then measured 30 cm distal to the ligament of Treitz and transected. Seventy-five to 150 cm of distal jejunum is used for the Roux limb and a functional end-to-side jejunojunctionostomy is created. The Roux limb is passed into the lesser sac through a defect created in the transverse mesocolon. After the creation of an enterotomy in the proximal Roux limb, the base of the EEA stapler is inserted in an antegrade fashion. The spike of the stapler is passed through the antimesenteric border of the Roux limb, mated to the post of the anvil, and fired. The Roux limb enterotomy is stapled closed, and the gastrojejunostomy is tested for a leak by instillation of 40 mL of methylene blue via a Baker tube.

6. **Complications** in the immediate postoperative period are similar to open gastric bypass and include wound infection, bleeding, deep venous thrombosis, pulmonary embolism, anastomotic leakage, myocardial infarction, and a 1–2% mortality. Laparoscopic bariatric surgery may prove to be beneficial in decreasing the incidence of a number of these complications. Delayed complications are less common than in open surgery and include anastomotic stricture and rare incisional hernias.

22 Occlusive Arterial Disease

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Introduction

Stenosis or occlusion of branches of the arterial tree can generate a variety of clinical syndromes. The specific symptoms and severity depend on the downstream organ(s), the degree of stenosis, the chronicity of obstruction, and the availability of collateral circulation. The vast majority of occlusive disease is secondary to atherosclerotic change of the arterial intima. This is, in general, a progressive disorder, influenced to a large degree by genetic predisposition. However, the evolution of the disease for most individuals can be modified by changes in environmental factors, particularly diet and exercise. Atherosclerotic disease is a systemic illness, and although symptomatic disease may predominate in one organ, subclinical disease, particularly of the coronary arteries, is generally present. In fact, 50% of the mortality associated with peripheral arterial reconstructions for atherosclerotic disease is cardiac in nature. Other less common causes of occlusive disease include **fibromuscular dysplasia**, **radiation-induced vascular injury**, and the **vasculitides** (e.g., Takayasu's arteritis and Buerger's disease).

Acute Arterial Occlusion of the Extremity

Symptoms of acute arterial insufficiency occur abruptly. The presentation generally includes the **five Ps of acute ischemia: pain, pallor, pulselessness, paresthesias, and paralysis**; some add poikilothermy to the list. The level of occlusion may be localized by the absence of pulses and the level of coolness of the limb. If adequate collateral circulation is not present, irreversible changes may appear as early as 4–6 hours after onset. Therefore, priority must be given to restoration of blood flow within this time period. These symptoms may be nonspecific or difficult to assess in obtunded patients, especially those with chronic arterial disease. Once the occlusive process has begun, regardless of its cause, vasospasm and propagation of thrombus distal to the site of initial occlusion can contribute to further ischemia.

I. Etiology

- A. The most common cause of acute arterial insufficiency is **embolization**.
 1. **Cardiac sources** account for more than 70% of emboli and usually are the result of mural thrombi that develop due to cardiac aneurysms or arrhythmias, such as atrial fibrillation. Other cardiac causes of emboli include valvular heart disease, prosthetic heart valves, bacterial endocarditis, and atrial myxoma.
 2. **Arterial-arterial emboli** can result from ulcerated atheroma or aneurysms, although embolization from abdominal aortic aneurysms is distinctly rare. The blue-toe syndrome occurs in patients with microemboli from unstable proximal arterial plaques and is characterized by the presence of intact pulses and painful, ischemic lesions in the distal extremity. Atheroemboli in the lower extremity secondary to plaque disruption by catheters can occur. The severely diseased distal aorta in some of these patients is evident on arteriography and has been termed **shaggy aorta**.
 3. **Venous-arterial emboli** (paradoxical emboli) can result from an intracardiac shunt (e.g., patent foramen ovale) or intrapulmonary arteriovenous malformations (e.g., Osler-Weber-Rendu syndrome).
 4. Occasionally, it is difficult to discern whether a person with **advanced atherosclerotic disease** has had an embolus or whether an already compromised vessel has undergone acute thrombosis. This is particularly true in patients without arrhythmias or prior myocardial infarction. The presence of contralateral pulses and no past history of claudication may help in making this differentiation.
- B. **Direct arterial trauma** is frequently obvious but may initially be occult and may result in arterial stenosis or occlusion only after an intimal flap or arterial wall hematoma progresses sufficiently to cause symptoms. Careful initial examination will avoid this pitfall. Arterial compromise can also occur in the setting of compression by joint dislocations (e.g., knee), bone fragments (e.g., tibial plateau fracture), or compartment syndrome.
- C. **Other causes** of acute ischemia include arterial thrombosis, aortic dissection, venous outflow occlusion, and low-flow state.

II. Diagnosis and evaluation

- A. If **physical examination** demonstrates clear evidence of embolization, definitive therapy should not be delayed. If there is a concern that the occlusive process may be thrombotic, however, arteriography may be indicated. Angiographically, embolic occlusions can be distinguished from thrombotic occlusions by their occurrence just distal to vascular bifurcations and by the concave shadow formed at the interface with the contrast. In select cases, thrombolysis may be a useful adjunct to define underlying occlusive disease. In general, patients with acute ischemia unrelated to trauma should be considered to have coexistent cardiac disease. All patients should have an electrocardiogram and chest X-ray performed. After limb revascularization a transesophageal echocardiogram can be useful in diagnosing a cardiac source. Laboratory determinations usually are of little benefit in the early assessment but are important in the later management of the patient.
- B. **Patients who present with penetrating trauma, long bone fractures, or joint dislocations** may have vascular injuries. Hard signs of arterial injury include diminished or absent pulses distal to an injury, ischemia distal to an injury, visible arterial bleeding from a wound, a bruit at or distal to the site of injury, or the presence of a large, expanding, or pulsatile hematoma. Patients with penetrating injuries who display clear signs of arterial injury need urgent surgical intervention without preoperative angiography. Soft signs of injury include the anatomic proximity of a wound to a major vessel, injury to an anatomically related nerve, unexplained hemorrhagic shock, or a moderately sized hematoma. In those with only soft signs, a careful documentation of pulses by Doppler pressures distal to the injury should be undertaken with comparison to the contralateral limb. A difference of greater than 10–20% should be an indication for arteriography or exploration. In certain situations, duplex scan of the injured area can be useful in the diagnosis of intimal flap, pseudoaneurysm, or arterial or venous thrombi, or both.

III. Management

- A. Once a diagnosis of acute arterial ischemia due to emboli or thrombi is made, **heparin** should be administered immediately. An intravenous bolus of 80 units/kg

followed by an intravenous infusion of 18 units/kg per hour is usually satisfactory. Partial thromboplastin time (PTT) should be maintained between 60 and 80 seconds.

1. **Surgical therapy**, such as embolectomy, should be performed as soon as possible in patients with an obvious embolus. **Embolectomy** can be done under local anesthesia if the patient cannot tolerate general anesthesia. Once the artery is isolated, a Fogarty catheter is passed proximally and distally to extract the embolus and associated thrombus. In some cases, intraoperative thrombolysis may be necessary because distal vessels may be thrombosed beyond the reach of the Fogarty catheter. Distal patency can be proved, if necessary, with an intraoperative arteriogram, depending on the status of distal vessels and pulses after embolectomy. In the leg, if adequate distal perfusion is not established and an angiogram demonstrates distal thrombus, the distal popliteal artery may be explored, with selective passage of the embolectomy catheter into the anterior tibial, posterior tibial, and peroneal arteries. The arteriotomy can be closed with a patch graft if there is arterial narrowing. Bypass grafting may also be required if significant preexisting arterial disease in the affected segment is discovered.
 2. **High-dose heparinization** (20,000-unit initial bolus followed by 2,000–4,000 units per hour) may be used in patients who are at extremely high risk for surgery and who have advanced ischemia with a nonviable extremity. The goal is to preserve as much tissue as possible by limiting progressive thrombosis.
 3. **Thrombolytic therapy** may be useful in patients with clearly viable extremities in whom thrombosis is the likely underlying cause of their acute ischemia. In general, the fresher the thrombus, the more successful the thrombolysis. Thrombolysis can frequently identify the underlying stenosis, which may be treated by balloon angioplasty/stent or by surgical means.
 - a. **Urokinase** was the agent of choice until 1999, when the U.S. Food and Drug Administration withdrew the drug from the market because of production concerns. Reteplase and, to a lesser degree, alteplase have been used as replacements. Optimal dosage strategies have not been formalized, but infusion rates between 0.25 and 1.00 units per hour have been reported, with complication rates of less than 10%. Repeat arteriography is performed to gauge the results of treatment. Duration of therapy is generally between 4 and 16 hours but may extend for as long as 30 hours.
 - b. The use of **glycoprotein IIb/IIIa inhibitors** may also have a role in combination with a thrombolytic agent. One study chose a dose of 0.25 mg/kg abciximab in combination with reteplase without an increase in complications.
 - c. During this procedure, the patient is usually monitored in the **intensive care unit (ICU)**. Thrombin time, fibrinogen level, fibrin degradation product level, PTT, and complete blood count are followed closely to limit the risk of hemorrhage. In general, the likelihood of serious hemorrhagic complications increases when fibrinogen levels drop below 100 mg/dL and the PTT rises above three to five times normal. Once the artery is open, the patient can be managed either with systemic anticoagulation or with surgical intervention (i.e., operative arterial reconstruction, balloon angioplasty).
 4. **Percutaneous aspiration thromboembolectomy** is an investigative technique and may prove useful as an adjunct to thrombolysis by decreasing clot volume. Various devices are currently undergoing clinical trials.
- B. In the setting of trauma, **operative exploration** should be performed in any limb that is ischemic or if arteriography demonstrates a significant intimal flap or other pathology. In the presence of coexistent neurologic or orthopedic injuries, it is essential to reestablish arterial flow first, either by direct repair or bypass grafting. At the conclusion of the orthopedic repair, the arterial repair should be reexamined to ensure that it has been correctly fashioned to the final bone length. In cases of joint dislocation, reduction of the dislocation should be accomplished first because this may alleviate the need for arterial reconstruction.
1. **Intraoperatively**, it is essential to obtain proximal and distal control of the injured artery before exploring the hematoma or wound. Ideally, when repairing an artery, an end-to-end anastomosis is preferable. A few centimeters of the artery can usually be mobilized proximally and distally to accomplish this reapproximation. However, the uninjured leg or other potential vein harvest site should be prepared in case a conduit is required. It is preferable to use autologous tissue in this setting. If concomitant venous injuries are identified, these should be repaired as well. In addition, if injuries to the great vessels are suspected, the patient's chest should be prepared because a median sternotomy or thoracotomy may be required. A completion arteriography can help to document and ensure distal flow, especially if significant spasm is present and distal pulses are not readily palpable.
 2. In general, **injuries to the subclavian, axillary, brachial, femoral, superficial femoral, profunda femoral, and popliteal arteries should be repaired**. The radial or ulnar artery may be ligated if the other vessel is intact and functioning. Similarly, isolated injuries to the tibial arteries may be ligated if a tibial artery is intact.

IV. Complications

- A. **Reperfusion injury** occurs after reestablishment of arterial flow to an ischemic tissue bed, which can lead to further tissue death. It results from the formation of oxygen-free radicals, which directly damage the tissue and cause white blood cell accumulation and sequestration in the microcirculatory system. This process, termed the **no-reflow phenomenon**, tends to prolong the ischemic interval because it impairs adequate nutrient flow to the tissue despite the restoration of axial blood flow. Currently, there is no proved therapy that limits reperfusion injury.
- B. **Myoneuropathic syndrome** occurs when the by-products of ischemic muscle, including potassium, lactic acid, myoglobin, and creatinine phosphokinase, are released into the systemic circulation after reperfusion. The electrolyte and pH changes can trigger dangerous arrhythmias, and precipitation of these toxic metabolites in the renal tubules can cause renal failure. The likelihood that a patient will develop myoneuropathic syndrome relates to the duration of ischemia and the muscle mass at risk. In an attempt to combat this, some surgeons clamp the femoral vein before revascularization and perform a transverse venotomy after lower-extremity arterial inflow is established. The first 250–500 mL blood is discarded or aspirated to an autotransfusion system, which removes the hyperkalemic and acidotic plasma and allows return of the red blood cells. Aggressive hydration, diuresis promotion with mannitol (25 g i.v.), and intravenous infusion of bicarbonate [2–3 ampules 7.5% NaHCO₃ (44.6 mEq/ampule) in 1 L D5W] to keep urine pH above 6.5 are helpful.
- C. **Compartment syndrome** results when prolonged ischemia causes cell membrane damage with leakage of fluid into the interstitium. The edema results in extremely high intracompartmental pressures, particularly in the lower extremity. When these intracompartmental pressures exceed capillary perfusion pressure, further muscle and nerve necrosis ensues. A four-compartment **fasciotomy** should be performed when there is a concern for the possible development of a leg compartment syndrome. Fasciotomy should be routine in any patient with more than 6 hours of lower-extremity ischemia or in the presence of combined arterial and venous injuries. Leg fasciotomies usually are performed through two incisions, one anterolateral and another posteromedial. The skin is left open, to be closed either secondarily or by skin graft at a later time.
- D. **Catheter-related complications** can occur early or late. Early complications result from arterial wall trauma secondary to the balloon-tipped catheter and include arterial perforation and rupture, intimal dissection, and pseudoaneurysm formation. A late catheter-related complication is the development of accelerated atherosclerosis in the embolectomized vessel, probably due to endothelial denudation and medial injury.

V. **Follow-up care** usually is directed at treating the underlying cause of the obstruction. Patients with mural thrombi or arrhythmias require long-term anticoagulation. The in-hospital mortality associated with embolectomy is as high as 20–30%, mostly due to coexistent cardiac disease.

Chronic Arterial Occlusive Disease of the Extremity

The **lower extremities are most frequently affected by chronic occlusive disease**, although upper-extremity disease can occur. The principal early symptom of arterial occlusive disease is claudication, which is usually described as a cramping pain or heaviness in the affected extremity that occurs after physical exertion. Claudication is relieved by rest but recurs predictably with exercise. Lower-extremity occlusive disease is subdivided into three anatomic sections based on symptoms and treatment options. Aortoiliac occlusive disease, or “inflow disease,” affects the infrarenal aorta and the common and external iliac arteries. Femoral-popliteal occlusive disease, or “outflow disease,” affects the common femoral, superficial femoral, and popliteal arteries. Finally, tibial-peroneal disease, or “runoff disease,” affects the vessels distal to the popliteal artery.

I. Clinical presentation

- A. **Aortoiliac disease** presents with symptoms of lower-extremity claudication, usually of the hip, thigh, or buttock. It may coexist with femoral-popliteal disease, contributing to more distal symptoms as well. The symptoms usually develop gradually, although acute worsening of symptoms may suggest the acute thrombosis of a diseased vessel. These patients ultimately develop incapacitating claudication but not rest pain unless distal disease is present as well. **Leriche's syndrome** is a constellation of symptoms in men that results from the gradual occlusion of the terminal aorta. Symptoms include sexual impotence, extreme leg fatigue with exercise, leg musculature atrophy, trophic changes of the feet, and leg pallor. In contrast to the male predominance in chronic peripheral vascular disease (PVD), isolated aortoiliac disease affects women and men equally.
- B. Patients with **femoral-popliteal and tibial disease** present with claudication of the lower extremity usually most prominent in the calves. More severe impairment of arterial flow can present as rest pain. Rest pain is a burning pain in the distal foot, usually worse at night or when the leg is elevated, and is relieved by placing the leg in a dependent position. Examination of the chronically ischemic extremity reveals decreased or absent distal pulses and trophic changes that include thickening of the nails, loss of leg hair, shiny skin, and ulceration that appears at the tips of the toes.
- C. **Symptomatic arterial occlusive disease of the upper extremity is relatively rare**.
 1. The **proximal subclavian artery** is most commonly affected by atherosclerotic disease, followed by axillary and brachial arteries. These patients typically present with arm claudication or finger-hand ischemia or necrosis. Occasionally, ulcerated plaques of the innominate or subclavian arteries can be a source of embolization to the upper extremity.

2. **Vertebral steal** can result when an occlusive subclavian artery lesion is located proximal to the origin of the vertebral artery. The arm's demand for blood is supplied by reversed flow in the ipsilateral vertebral artery, which shunts blood from the posterior cerebral circulation. Most patients with proximal subclavian lesions are completely asymptomatic. Others may present with arm ischemia or posterior circulation symptoms, such as drop attacks, ataxia, sensory loss, or diplopia, occurring after exercise of the affected limb.

II. Diagnosis

- A. The diagnosis of chronic arterial occlusive disease is concerned with determining the presence of significant **flow-limiting lesions** and distinguishing the disease from mimics such as arthritis, gout, and neuromuscular disorders. Furthermore, the degree of arterial flow limitation and the impact of reduced tissue perfusion are integral to the diagnostic procedures and determination of appropriate therapy.
- B. For patients presenting with lower-extremity symptoms, it is essential to examine the femoral and distal pulses at rest and after exercise. The **absence of femoral pulses is indicative of aortoiliac disease**, although some patients with aortoiliac disease have palpable pulses at rest that are lost after exercise. Audible bruits may also be appreciated over the lower abdomen or femoral vessels.
- C. **Noninvasive testing** can quantify flow through larger vessels and can quantify tissue perfusion.
 1. **Segmental arterial Doppler readings with waveforms** should be performed in all patients with suspected symptomatic arterial disease. The ankle-arm index (AAI; the ratio of the BP in the leg to that in the arm) allows one to quantify the degree of ischemia. Patients without vascular disease have an AAI of more than 1.0, patients with claudication have an AAI of less than 0.6, and patients with rest pain and severe ischemia have an AAI of less than 0.4. Waveform changes help localize the site of significant disease.
 2. **Transcutaneous measurement** of local tissue oxygenation has been developed to attempt to quantify the physiologic derangements of ischemia. However, the usefulness of this test in the general vascular patient has not been established.
- D. **Digital subtraction arteriography** is the gold standard with which to evaluate the arterial tree before planned revascularization. A typical digital subtraction arteriography of the lower extremities includes images of the infrarenal aorta, renal, iliac, femoral, tibial, and pedal vessels.

III. Management

- A. Intermittent claudication by itself is not an indication for **surgical intervention** because it has a benign course in most patients. In patients presenting with claudication alone, 70–80% remain stable or improve and 10–20% worsen. Only 5–10% of patients develop gangrene and are at risk for limb loss. Therefore, first-line treatment of patients with claudication should be medical therapy. Essentially, there are three indications for surgical intervention.
 1. **Limb salvage** is the goal of surgery in patients with ischemic rest pain or tissue loss, including frank distal gangrene. Multilevel femoral-popliteal-tibial disease is the typical distribution in these patients. When combined significant aortoiliac disease and distal disease are present in a patient with a threatened limb, however, an inflow (aortoiliac) procedure should be performed first.
 2. **Peripheral atheroembolization** from aortoiliac ulcerated plaques, even if there is little or no history of claudication, is an indication for exclusion and bypass or endarterectomy of the aortoiliac system.
 3. **Incapacitating claudication** that jeopardizes a patient's livelihood or influences his or her quality of life indicates disease likely to respond well to treatment. These patients should adhere to a program of risk factor reduction as well.
- B. **Medical therapy** is available for those patients with symptoms who are not candidates for surgical intervention. However, no medical therapy is available to halt the progression completely or reverse the changes of advanced atherosclerotic disease.
 1. **Risk factor modification** is the single most important intervention to reduce the impact of advanced atherosclerotic disease. Control of hypertension and serum glucose, cessation of smoking, management of lipid disorders, attainment of ideal body weight, and regular exercise should be the goals.
 2. Evidence is now accumulating that **aspirin** alone or combined with dipyridamole may have an impact on the natural progression of claudication due to atherosclerotic disease. Further, because many of these patients have concomitant coronary artery or cerebrovascular disease, lifelong therapy with 81 or 325 mg aspirin daily may reduce the risk of myocardial infarction or stroke.
 3. **Clopidogrel and ticlopidine** are chemically related antiplatelet agents that have shown some benefit in reducing vascular death or the need for revascularization in some recent studies in patients with intermittent claudication. However, the evidence is not as strong as that for aspirin.
 4. **Pentoxifylline** is a rheologic agent thought to reduce blood viscosity and allow for improved flow through small arterioles and stenotic arteries. Although the drug is frequently prescribed, its benefit is unpredictable and generally quite small. It may be best suited for patients with severe disease not amenable to reconstruction.
 5. **Cilostazol** is a type III phosphodiesterase inhibitor and the newest agent available for treatment of claudication. Given at 50 mg or 100 mg twice a day, it increases walking distances when compared to placebo and pentoxifylline. Early postmarketing studies suggest that the drug is safe in most patients, although its use is contraindicated in those with class III–IV heart failure due to theoretical concerns of toxicity.
- C. **Preoperative care** of patients with PVD includes a complete arterial evaluation. In addition to angiographic evaluation of the symptomatic arterial tree, patients undergo screening for associated cardiac, renal, cerebrovascular, and pulmonary disease so that any correctable lesions can be addressed. Myocardial complications account for the majority of early and late deaths; therefore, patients with questionable myocardial function may require more extensive cardiac evaluation. Screening for carotid disease should also be performed and include a history of stroke or transient ischemic attack (TIA) and carotid auscultation. On the day before surgery involving the abdominal aorta, patients are kept on a liquid diet, hydrated well, and given a mechanical bowel preparation. Parenteral antibiotics are started 1–2 hours preoperatively and are continued for 24 hours after surgery.
- D. **Endovascular surgical therapy** is discussed in more detail in [Chapter 24](#). Endovascular techniques are most successful in the aortoiliac system. They have been attempted in the distal vascular beds, with poor long-term results. There may be a very limited role for angioplasty of short-segment distal occlusions in high-risk patients who are facing limb loss.
- E. **Open surgical therapy**
 1. **Aortoiliac occlusive disease**
 - a. **Aortobifemoral grafting** is the treatment of choice in **good-risk patients**. This procedure may be performed through a transperitoneal or a retroperitoneal approach. Results at our institution suggest that the retroperitoneal approach reduces postoperative complications, especially ileus. Although a number of grafts are available for use as replacement conduit, the knitted polyethylene terephthalate (Dacron) prosthesis is the most commonly used. A distal endarterectomy may be performed in conjunction with a bypass to improve outflow. Similarly, if significant renal artery disease is identified preoperatively, renal revascularization by endarterectomy or bypass grafting can be performed at the time of aortic reconstruction. Results are excellent, with reported patency rates of up to 95% at 5 years.
 - b. **Femorofemoral, ilioliac, or iliofemoral bypasses** are alternatives in high-risk patients with **unilateral iliac disease**. Patency rates are lower than those for aortobifemoral grafts.
 - c. **Axillobifemoral bypass** is an adequate alternative for high-risk patients who need revascularization. This **bypass avoids an intraabdominal procedure** and the need for cross-clamping the aorta. Patency rates are significantly worse than for aortobifemoral bypass, although for patients with a short life expectancy (<5 years), this is a reasonable alternative.
 - d. **Aortoiliac endarterectomy** may be considered for patients who have disease localized to the distal aorta and common iliac vessels. Advantages include avoiding prosthetic material and better flow into the hypogastric arteries. This procedure should not be done in patients with aneurysmal changes of the aorta, a completely occluded aorta to the level of the renal arteries, or extension of disease into the external iliac vessels.
 2. **Femoral, popliteal, and tibial occlusive disease**
 - a. In patients with above-knee occlusion, an **above-knee femoral-popliteal bypass** is indicated. In patients who have disease below the knee, a more **distal bypass** may be performed to the **below-knee popliteal, posterior tibial, anterior tibial, or peroneal arteries**. In extreme cases, if all tibial vessels are occluded, **pedal vessels** may serve as suitable outflow vessels. These grafts usually originate from the common femoral artery, although a more distal vessel may be used if the inflow into that vessel is adequate.
 - b. Best results are obtained with the use of autologous vein as a **conduit**. The greater saphenous is the vein of choice. The lesser saphenous vein or arm veins may provide good alternatives. These autologous grafts can be used either **in situ** or **reversed**. The advantages of the *in situ* bypass are (1) the vein's nutrient supply is left intact, and (2) the vein orientation allows for a better size match (the large end of the vein is sewn to the large common femoral artery, and the small end is sewn to the distal vessel). The reversed-vein bypass advantage is that endothelial trauma is minimized because valve lysis is not necessary.
 - c. When autologous vein is not available, **umbilical vein and cryopreserved vein grafts** can be used. Comparable patency rates have also been achieved using polytetrafluoroethylene as above-knee grafts. In extreme cases, prosthetic grafts can be bypassed to the tibial vessels, but with inferior results. An alternative is to combine the prosthetic graft with a vein graft as a composite or to use a small cuff of vein (Miller cuff) or patch angioplasty (Taylor patch) just at the distal anastomosis. These modifications are believed to improve patency by improving compliance match at the distal anastomosis.
 - d. **Endarterectomy** plays a negligible role as a sole procedure in an extremity, except in cases of isolated short-segment stenosis of the superficial femoral artery. Endarterectomy may have a role in patients in whom there is limited vein available or in the presence of an infected field. In these patients, a superficial femoral artery endarterectomy may obviate the need for a prosthetic graft.
 - e. **Sympathectomy** sometimes is used as an adjunct to vascular reconstructions and involves division of the sympathetic chain and L3-5 ganglia. With the

development of **percutaneous alcohol ablation** of the sympathetic ganglia, this procedure is becoming more popular. Although it does not increase blood supply to the muscles, it does result in maximal dilation of small arterioles and collaterals, providing increased blood flow to the skin and subcutaneous tissues. The effect is short lived, lasting only 4–6 weeks, but occasionally sympathectomy relieves rest pain and helps in the healing of small ulcerations.

- f. **Amputation** is reserved for patients with gangrene or persistent painful ischemia not amenable to vascular reconstruction. These patients have severe coexistent vascular disease, and the survival of patients undergoing major amputations is 50% at 3 years and 30% at 5 years.
 1. **Level of amputation** is determined clinically. Important factors include the level necessary to remove all the infected tissue and the adequacy of the blood supply to heal the amputation at a given level. A general principle is to preserve the length of the extremity because this improves the patient's opportunity for rehabilitation. In some cases, revascularization before amputation enables a more distal amputation to heal adequately.
 2. **Digital amputations** are performed commonly in diabetic patients who develop osteomyelitis or severe foot infections.
 3. **Transmetatarsal amputations** usually are performed when several toes are involved in the ischemic process or after previous single-digit amputations.
 4. **Syme's amputation** involves the removal of the entire foot and calcaneus while preserving the entire tibia. It is rarely appropriate for PVD.
 5. **Below-knee amputation (BKA)** is the most common type of amputation performed for patients with severe occlusive disease.
 6. **Above-knee amputation (AKA)** heals more easily than BKA and is useful in older patients who do not ambulate.
 7. **Hip disarticulation** rarely is performed for PVD.

3. Upper-extremity occlusive disease

- a. For **proximal subclavian disease**, the choice of bypass procedure depends primarily on the patency of the ipsilateral common carotid artery.
 - b. If the ipsilateral common carotid artery is patent, **carotid-subclavian bypass** is performed through a supraclavicular approach using a prosthetic graft (avoid vein grafts). Subclavian artery transposition to ipsilateral carotid artery is another good alternative if anatomically feasible, and it avoids the use of a bypass conduit.
 - c. If the ipsilateral carotid artery is occluded, **subclavian-subclavian bypass** may be performed. This is an extraanatomic approach using a longer-segment prosthetic graft, and the patency is thereby diminished somewhat.
4. **Intraoperative anticoagulation** is employed during most vascular reconstructions. Generally, unfractionated heparin (100–150 units/kg) is administered intravenously shortly before cross-clamping and supplemented with 50 units/kg until the cross-clamps are removed. Reversal with protamine administration is generally done to reduce needle hole bleeding.

F. Postoperative care

1. For **open aortic procedures**, **early** postoperative care is usually administered in the ICU, where frequent hemodynamic and hematologic measurements are performed and identified conditions treated. Assessment of distal pulses should be done intraoperatively, immediately after reconstruction and regularly thereafter. In uncomplicated cases, the patients usually are extubated the day of surgery or on postoperative day 1. The patients are kept well hydrated for the first 2 postoperative days, after which third-space fluid begins to mobilize and diuresis ensues. Fluid management frequently is guided by Swan-Ganz catheter pressure monitoring. Antibiotics are continued for 24 hours postoperatively. A nasogastric tube is kept in place until any ileus resolves. Patients are instructed not to sit with the hips flexed at greater than 60 degrees for the first 72 hours after graft placement, although ambulation as early as possible is encouraged.
2. For **distal bypass grafts**, **pulses should be assessed frequently** for the first 24 hours and then several times a day. Antibiotics are continued for 48 hours postoperatively or longer, if infected ulcers warrant such treatment. **Ambulation as early as possible** is encouraged in patients without tissue necrosis. In patients who are unable to ambulate immediately, **physical therapy** can help increase strength in the limb and prevent contracture. Sitting with the hips flexed to 90 degrees is discouraged in any patient with a femoral anastomosis. Patients should be discharged with instructions to keep their legs elevated to help resolve the edema that develops in the revascularized extremity. Staples are left in place for 2–3 weeks because these patients frequently have delayed wound healing.
3. **Perioperative antithrombotic therapy** should include **aspirin** (81–325 mg per day) for all prosthetic infrainguinal reconstructions starting preoperatively. Also, any bypass distal to the knee should likely include aspirin. **Dipyridamole** (75 mg 3 times daily) may be added, although the increment of additional benefit is unknown. In patients sensitive to aspirin, clopidogrel (75 mg per day) may be substituted.
4. **Postoperative oral anticoagulation** has a more limited role. Due in part to the increased risk of hemorrhage, anticoagulation with warfarin (International Normalized Ratio 2.0–3.0) is generally limited to grafts considered to be at a high risk for thrombosis. There may be some benefit to administration of **dextran 40** (0.5 mL/kg per hour i.v.) for up to 72 hours postoperatively.
5. For **amputations**, weightbearing is delayed for 4–6 weeks. Some advocate the use of stump shrinkers to aid the maturation of the stump. In all cases, early consultation with a physical therapist is recommended. The role of the physical therapist is essential in maintaining strength in the limb, preventing contracture, and rehabilitating the patient once a prosthesis is fitted. In addition, as soon as the patient is ready, he or she should be fitted for a prosthesis and ambulation training should begin. Rehabilitation rates (ability to walk without assistance) for patients undergoing major amputation are 60% and 30%, respectively, for patients with a unilateral BKA or AKA. For those with bilateral amputations, rehabilitation rates drop to 40% for patients with bilateral BKA and 10% for patients with bilateral AKA.
6. **Long-term follow-up for distal bypass grafts** should consist of arterial Doppler examinations every 3 months for the first 18 months, then every 6 months for a year, and then yearly. Less frequent follow-up is necessary for aortoiliac bypasses. Significant reduction in the AAI or flow velocities predicts pending graft failure, and such grafts should be studied further by arteriography. Intervention to repair or revise stenosed grafts results in much higher long-term patency than repairing or replacing occluded grafts.

G. Complications

1. **Early** complications occur in approximately 5–10% of patients after aortic surgery and frequently relate to preoperative comorbid disease. Myocardial infarction, congestive heart failure, pulmonary insufficiency, and renal insufficiency are most common. Complications related directly to the aortic reconstruction include hemorrhage, embolization or thrombosis of the distal arterial tree, microembolization, ischemic colitis, ureteral injuries, impotence, paraplegia, and wound infection. Late complications include anastomotic pseudoaneurysm or graft dilatation, graft limb occlusion, aortoenteric erosion or fistula, and graft infections.
2. In **distal revascularizations**, most of the early complications are also related to comorbid conditions. Early graft thrombosis (within 30 days of surgery) most often results from technical errors, hypercoagulability, inadequate distal runoff, and postoperative hypotension. Technical errors are responsible for more than 50% of early graft failures and include graft kinks, retained valve leaflets, valvulotome trauma, intimal flaps, significant residual arteriovenous fistulas, and the use of an inadequate conduit (i.e., small vein).

Extracranial Cerebrovascular Disease

Atherosclerotic occlusive disease of the extracranial carotid artery is a major risk factor for stroke. Stroke is the primary cause of disability and the third most common cause of death in the United States, with more than 160 new strokes per 100,000 people occurring each year. The initial mortality from stroke is between 20% and 30%. Of patients who survive the initial event, one-third function normally, one-third recover with mild deficits, and one-third can no longer function normally. A smaller percentage of patients require total custodial care. Of patients who survive, approximately 50% die of recurrent stroke within 5 years.

I. Presentation

- A. The **clinical presentation** of patients with symptomatic occlusive disease is a neurologic deficit. Extracranial vascular occlusive disease can result in lateralizing and global symptoms. However, many patients may have an asymptomatic stenosis that is identified by a health care provider based on auscultation of carotid bruits or screening Doppler study.
- B. **Lateralizing ischemic events** can result in aphasia (motor or receptive), combined sensory and motor deficits, and various visual disturbances. Deficits such as these are usually associated with the anterior cerebral circulation (i.e., the internal carotid artery and its branches).
 1. **TIA**s are transient hemispheric neurologic deficits that may last from several seconds to hours but fewer than 24 hours. TIAs that occur in rapid succession, interspersed with complete recovery but with progressively smaller intervals between attacks (**crescendo TIAs**) carry a high risk of progression to a permanent neurologic deficit and must be evaluated emergently.
 2. **Amaurosis fugax**, or temporary monocular blindness, described as a shade coming down over the eye, results from emboli lodging in the ophthalmic artery. Funduscopic examination can demonstrate these cholesterol plaques, commonly known as **Hollenhorst plaques**, as they traverse the retina.
 3. A **reversible ischemic neurologic deficit** is a longer-lasting neurologic deficit that can last for up to 7 days but ultimately resolves completely.
 4. If the neurologic deficit is fixed and persists beyond 7 days, it is considered a completed **stroke**. In addition, some patients may present with a neurologic deficit that fluctuates, gradually worsening over a period of hours or days while the patient is under observation. This situation is considered a stroke in evolution and, like crescendo TIAs, needs prompt treatment.
- C. **Global ischemic events** are manifested by symptoms such as vertigo, dizziness, perioral numbness, ataxia, or drop attacks. These usually are associated with

interruption of the brainstem or posterior circulation (i.e., the vertebrobasilar system).

II. Pathophysiology and epidemiology. Ischemic events in patients with extracranial vascular disease can be the result of emboli or a low-flow state. Although it can be clinically significant, disease of the vertebral arteries usually remains asymptomatic. On the other hand, even asymptomatic significant occlusive carotid arterial disease carries with it a doubling of baseline stroke risk. Once a significant carotid lesion results in an ipsilateral lateralizing cerebral event, the risk of stroke may be as high as 26% over 2 years.

III. Diagnosis. A careful neurologic examination is performed before obtaining any diagnostic studies. Imaging of the carotid arterial system attempts to classify the degree of stenosis because of the prognostic implications. Due to methodologic differences in calculating the percentage stenosis encountered in different studies, there is some disagreement about exact cutoff percentages. However, in general, there are essentially four levels of stenosis (with approximate percentages): mild (<50%), moderate (50–69%), severe (70–99%), and occluded (100%).

- A. A variety of noninvasive and invasive diagnostic studies are available. **Color-flow duplex scanning** uses real-time B-mode ultrasound and color-enhanced pulsed Doppler flow measurements to determine the extent of the carotid. This is the initial screening test for carotid disease. The reliability of this study depends in large part on the abilities of the vascular technicians and requires validation. In the setting of a validated vascular laboratory, treatment of most carotid lesions can be instituted based on ultrasound/duplex alone.
- B. **Arteriography** remains the gold standard for the diagnosis of cerebrovascular disease. Unlike the duplex scanning, however, arteriography remains a painful procedure with inherent risks, such as dye allergy, renal toxicity, and stroke (2–4% of patients). Because of these risks and improvements in duplex ultrasonography, carotid arteriography is generally limited to patients with technically inadequate duplex ultrasonography or for verification of carotid occlusion.
- C. Although **MR angiography** is a highly sensitive technique for the evaluation of patients for symptomatic cerebrovascular disease, its precision remains inferior to that of conventional angiography.

IV. Management

- A. **Medical therapy.** It is important to make every effort to modify risk factors to prevent progression of carotid occlusive disease. Control of hypertension, cessation of smoking, management of lipid disorders, attainment of ideal body weight, and regular exercise should be undertaken. No drug therapy has been shown to reduce the risk of stroke in patients with asymptomatic carotid disease. Medical management in symptomatic patients is focused primarily on the use of antiplatelet agents, specifically aspirin. Aspirin is effective in reducing stroke and stroke-related deaths as well as myocardial infarctions. Low doses (80 mg per day) are as efficacious as higher doses (1,200 mg per day). Other antiplatelet agents, such as dipyridamole and ticlopidine (250 mg p.o. b.i.d.), are no better than aspirin alone. Anticoagulation with heparin sodium is beneficial in patients who have cardiac emboli. In evolving strokes, heparin may be useful in preventing progression of thrombus. The major contraindication to heparinization is a recent hemorrhagic brain infarct; therefore, a computed tomographic (CT) scan should be obtained before heparin is given.
- B. **Surgical therapy.** Surgical management is the treatment of choice for extracranial cerebrovascular disease and has been documented to reduce stroke. However, it should be performed only in centers of excellence in which the combined stroke and mortality rate is less than 3%.
 1. **Indications**
 - a. **Asymptomatic patients with greater than 60% stenosis.**
 - b. **Symptomatic patients with greater than 70% stenosis.**
 - c. **Symptomatic patients with greater than 50% stenosis** who have an **ulcerated lesion** or whose **symptoms persist while they are on aspirin.**
 - d. **Selected patients with stroke in evolution.** Operation is performed to restore normal blood flow to allow recovery of ischemic brain tissue that is nonfunctional yet metabolically alive. Surgical candidates have mild to moderate neurologic defects and no evidence of hemorrhage on CT scan. The timing of surgery in these cases is controversial.
 - e. **Selected patients with completed strokes.** Operation is performed in the hope of reducing stroke recurrence, which is 7–8% per year with nonsurgical therapy. Patients with a mild deficit and greater than 70% stenosis or with greater than 50% stenosis and an ulcer are candidates for surgery. In addition, patients with a moderate deficit, a lesion greater than 70%, and a contralateral occluded carotid artery are surgical candidates. The timing of surgery in these cases is debatable. A prudent approach is to wait 4–6 weeks to minimize the risk of postoperative hemorrhage.
 - f. Rarely, **endarterectomy** is performed on patients with completely occluded carotid arteries. Candidates for surgery include patients who have undergone endarterectomy who develop immediate postoperative thrombosis or symptoms; patients who are asymptomatic and have had a bruit disappear while under observation; patients who have had a recent occlusion and have fluctuating or progressive symptoms; and patients who are symptomatic and have a new internal carotid occlusion that can be operated on within 2–4 hours of the onset of symptoms.
 2. **Carotid endarterectomy** has been performed for more than 40 years and is the most commonly performed vascular operation. A beneficial outcome to the patient depends on meticulous technique, resulting in perioperative event rates (stroke and death) of less than 3%.
 - a. **Anesthesia** for carotid endarterectomy can be general endotracheal anesthesia, regional cervical block, or local anesthesia. The choice of anesthesia depends on a combination of patient factors and surgeon expertise. No single method of anesthesia has proved superior.
 - b. During **dissection and mobilization of the common carotid artery and its branches**, it is important to proceed with gentle dissection and minimal manipulation of the carotid bulb to prevent embolization from the atherosclerotic plaque.
 - c. After **systemic heparinization** of the patient, the carotid artery is occluded, and a longitudinal **arteriotomy** is made from just proximal to the plaque in the common carotid artery to just beyond the distal extent of the plaque in the internal carotid artery.
 - d. **Placement of tubing to shunt blood from the common carotid artery** around the operative field to the internal carotid artery during endarterectomy is a controversial practice. Although some surgeons “always shunt,” and a few “never shunt,” most surgeons “selectively shunt” based on preoperative patient factors (e.g., the presence of contralateral internal carotid artery occlusion) or intraoperative neurologic assessment.
 1. For patients undergoing **anesthesia for carotid endarterectomy** with local or regional anesthesia, intraoperative neurologic assessment can be as simple as having the patient squeeze a noise toy in the contralateral hand after carotid occlusion and answering a few simple questions.
 2. For patients under **general anesthesia**, several different methods have been used to assess cerebral perfusion or neurologic function after clamping of the carotid artery. These include transcranial Doppler measurements and intraoperative electroencephalogram (EEG) monitoring.
 - e. The **plaque** is carefully separated from the media and removed. The carotid endarterectomy is closed using a running suture, and if the internal carotid artery is small, a patch angioplasty can be performed.
 3. **Postoperative care**
 - a. **Immediately** after endarterectomy, neurologic function and BP alterations should be monitored. Hypertension and hypotension are common after endarterectomy and may cause neurologic complications. The extremes of BP should be treated with either sodium nitroprusside or phenylephrine (Neo-Synephrine) to keep the systolic BP between 140 and 160 mm Hg (slightly higher in chronically hypertensive patients). The wound should be examined for hematoma formation. Aspirin is resumed in the immediate postoperative period. In patients with reactive platelets, dextran 40 (up to 20 mL/kg per 24 hours for up to 72 hours) may be started intraoperatively and continued into the early postoperative period. Anaphylaxis can occur with dextran use; therefore, a hapten (Promit, 15% dextran 1) is given before the start of infusion to bind antibodies to dextran.
 - b. **Patient follow-up.** A baseline duplex scan is obtained 2–3 weeks after the procedure and again at 6 months. After that, patients can be followed yearly. Patients who can tolerate aspirin are given 325 mg per day.
 4. **Complications**
 - a. **Stroke** rates must be low (3%) to make operative management of cerebrovascular disease reasonable, especially in asymptomatic patients.
 - b. **Myocardial infarction** remains the most common cause of death in the early postoperative period. As many as 25% of patients who undergo endarterectomy have severe, correctable coronary artery lesions.
 - c. **Cranial nerve injuries** occur in 5–10% of patients who undergo carotid endarterectomy. The most commonly injured nerve is the hypoglossal, followed by the recurrent laryngeal, marginal mandibular, and superior laryngeal nerves.
 - d. **Recurrent carotid stenosis** has been reported to be as high as 5–10% of cases, although symptoms occur in fewer than 3%. Two types of lesions have been characterized. A myofibroblastic lesion that occurs early (within 3 years) results from uncontrolled proliferation of the medial smooth-muscle cells and extracellular matrix. Recurrent atherosclerosis can also cause restenosis. The presence of symptoms is an indication for treatment of a recurrent lesion. Frequently, these lesions do not lend themselves to endarterectomy and are best treated by vein patching or excision and saphenous vein bypass grafting.

Renovascular Disease

Stenosis or occlusion of the renal arteries may result in hypertension or ischemic nephropathy, or both. Renovascular hypertension is the most common form of surgically correctable secondary hypertension. However, because of the predominance of primary hypertension and difficulties in clinical diagnosis of renovascular

hypertension, its exact prevalence is difficult to assess.

I. Clinical presentation

- A. A high index of suspicion is necessary to identify patients with potentially correctable renovascular hypertension from the majority of patients with primary hypertension. There are several **clinical features** that may be used to distinguish patients with potential renovascular hypertension.
1. **The presence of severe hypertension in a child or young adult or an adult older than age 50 years**
 2. **The sudden development of or worsening of hypertension at any age**
 3. **Hypertension and unexplained impairment of renal function**
 4. **Hypertension that is refractory to appropriate three-drug therapy**
 5. **Hypertension in a patient with extensive coronary disease, cerebral disease, or PVD**
- B. On **physical examination**, these patients may have an epigastric, subcostal, or flank bruit. The finding of a unilateral small kidney by any clinical study is a possible indicator.
- C. **Deterioration of renal function in the elderly** may also be a clinical clue, as up to 15% of elderly patients hospitalized for the treatment of renal failure are ultimately diagnosed with ischemic nephropathy.

II. Pathophysiology

- A. **Renal arterial stenosis** is perceived by the ipsilateral kidney as a hypovolemic state and as such activates the renin-angiotensin-aldosterone system. Typical renovascular hypertension results from **unilateral renal artery stenosis (renin-dependent hypertension)** because renin release by the affected kidney remains high since the resulting increase in intravascular volume is eliminated by the contralateral kidney. **Bilateral renal artery stenosis (volume-dependent hypertension)** results in an initial rise in renin; once the intravascular volume increases, however, feedback mechanisms return renin levels to near normal.
- B. Even in the absence of hypertension, a **significant renal artery stenosis** may be present and may lead to renal failure.
1. In **acute renal failure**, a renal arterial stenosis should be considered in the differential diagnosis if the urinary sediment is unremarkable and there are no signs of acute tubular necrosis, glomerulonephritis, or interstitial nephritis. Acute ischemic nephropathy may occur within 2 weeks of starting an angiotensin-converting enzyme (ACE) inhibitor or other antihypertensive or diuretic.
 2. Renal artery stenoses may account for up to 24% of unexplained **chronic renal failure** in patients older than 50 years of age. The diagnosis is more likely in those with generalized atherosclerosis, recurrent pulmonary edema, or uncontrolled hypertension.
 3. **Isolated unilateral renal artery stenosis** generally does not cause an increase in the serum creatinine concentration to greater than 2.0 mg/dL.
- C. **Atherosclerosis** causes approximately two-thirds of all renovascular lesions in adults and usually affects the ostia and proximal 2 cm of the renal artery. Associated extrarenal atherosclerosis occurs in 15–20% of patients.
- D. The second most common renovascular lesion is **fibromuscular dysplasia**, most commonly medial fibroplasia. These lesions are multifocal, have a characteristic **string-of-beads** appearance on angiography, and typically occur in young or middle-aged women.

III. Diagnosis

- A. **Testing** for clinically significant renal artery disease must evaluate the anatomic and physiologic changes. Anatomic lesions of the renal arteries by themselves correlate poorly with physiologic effect. More important, they correlate poorly with treatment response.
- B. **Arteriography** remains the best test for making the diagnosis of anatomic renal artery stenosis. However, the usual caveats regarding the risks of arteriography, especially the nephrotoxic effects of the contrast agent, are important considerations.
- C. In patients with a relative contraindication to arteriography, **duplex scanning** is useful for screening. However, this procedure is highly technician dependent.
- D. **MR angiography** with gadolinium-based i.v. contrast is an excellent test to evaluate kidney and main renal artery morphology without nephrotoxins. It may also be useful for evaluation of the functional significance of renal artery stenoses. Limitations include imaging of distal renal artery stenoses and patients with implanted devices, morbid obesity, or claustrophobia.
- E. The two most commonly used tests to determine the functional significance of a renal artery lesion are **captopril renal scintigraphy** and **selective renal vein renin measurement**. These tests can be complicated to perform and interpret, especially in patients with bilateral disease or in those taking ACE inhibitors or beta-blockers.

IV. Management of fibromuscular disease

- A. **Renal artery stenoses** resulting from fibromuscular dysplasia rarely cause renal failure, and endovascular therapy of the lesions is frequently successful in treating the hypertension.
- B. **Failure of endovascular treatment** can be due to recurrent or branch disease. Surgical therapy may be employed, including *ex vivo* repair for complex disease as described in [section V.C.2.d](#).

V. Management of atherosclerotic disease

- A. **Therapy for renal artery stenosis due to atherosclerotic disease** has a dual purpose: to control target organ damage from hypertension and to avoid progressive ischemic renal failure. However, even with functional studies, response to therapy is difficult to predict because the hypertension may be primarily essential and the renal failure due to hypertensive glomerulosclerosis. No careful studies have been done comparing best medical therapy to surgical or endovascular intervention.
- B. **Medical therapy** with antihypertensive drugs is often successful in the management of patients with renovascular hypertension. A combination of beta-blockers with a calcium channel blocker or an ACE inhibitor or an angiotensin II receptor inhibitor is commonly used as first-line therapy. ACE inhibitors are often effective therapy, but they should be used cautiously when the entire renal parenchymal mass is at risk, as occurs in renovascular hypertensive patients who have bilateral disease or a solitary kidney. Diuretics should be reserved for second-tier therapy.
- C. **Surgical therapy**
1. The **indications** for intervention are beginning to change. Classically, surgery usually has been reserved for patients with uncontrollable hypertension that is refractory to maximal medical therapy. Because of the effectiveness of modern antihypertensive medical therapy, renal revascularization for hypertension is more rarely performed. Evidence is accumulating that revascularization may be more important as a means of maintaining renal mass. Consideration of renal revascularization should also be given in patients undergoing aortic bypass for aneurysmal or occlusive disease with concomitant renal stenoses.
 2. **Procedures**
 - a. **Aortorenal bypass** is the classic open treatment for renal revascularization. The stenotic renal artery is isolated with a segment of infrarenal aorta, and bypass is accomplished using saphenous vein, autologous hypogastric artery, or prosthetic graft.
 - b. **Renal endarterectomy** is also an excellent choice for renal revascularization and is the treatment of choice for bilateral orificial lesions. Most commonly, a transverse arteriotomy is made over the orifices of both renal arteries. Distal end-points and adequacy of endarterectomy can be assessed intraoperatively using duplex scanning.
 - c. **Alternative bypass procedures** are available for patients who are not good candidates for aortorenal bypass due to prior aortic surgery, the presence of severe aortic disease, or favorable anatomy. Grafts can be taken from the supraceliac aorta or the superior mesenteric, common hepatic, gastroduodenal, splenic, or iliac arteries. Results for these procedures are comparable to those for direct aortic reconstruction but with significantly less morbidity and mortality.
 - d. ***Ex vivo* renal artery reconstruction with renal autotransplantation** is useful in patients with complex lesions that require microvascular techniques and in which exposure *in situ* is inadequate. These procedures are time consuming, and adequate hypothermic protection from ischemic renal injury may be difficult with the kidney *in situ*. In general, operations requiring more than two branch artery reconstructions or anastomoses should be considered for *ex vivo* repair. Once repair is accomplished, the kidney can be transplanted orthotopically or heterotopically to the ipsilateral iliac fossa.
 - e. **Percutaneous transluminal angioplasty** works best for fibromuscular lesions and select nonostial atherosclerotic lesions. Durability may be inferior to surgical revascularization, but recurrences are frequent after dilation of ostial lesions. Primary stenting for ostial lesions may improve patency rates.
 - f. **Nephrectomy** may be required in patients who have renal infarction, severe nephrosclerosis, severe renal atrophy, noncorrectable renal vascular lesions, failed revascularizations, or a normal contralateral kidney and who are high-risk surgical candidates.
 3. **Postoperative care**
 - a. **Immediately** after operation, patients should be kept well hydrated to maintain adequate urine output. Concern about the patency of the reconstruction may be addressed by a renal scan or duplex scan.
 - b. **Patient follow-up** should consist of routine BP monitoring, a renal scan, and creatinine determination at 3 months, 12 months, and then yearly. Any

recurrence of hypertension or deterioration in renal function should prompt arteriography. Duplex scanning is also a useful test for following these reconstructions.

4. **Complications** of renal revascularization include persistent hypertension, acute renal failure, renal artery restenosis, thrombosis, aneurysm formation, and distal embolization.

Mesenteric Ischemia

Diagnosing mesenteric vascular disease can be difficult. Most patients are asymptomatic until late in the disease process. Although considerable advances have been made in perioperative care as well as in the diagnosis and treatment of intestinal ischemia, mortality remains as high as 70–80% due to delays in diagnosis.

I. Acute occlusion

A. Clinical presentation

1. Patients with **emboli to the gastrointestinal tract** usually have acute abdominal pain in the presence of significant cardiac disease, frequently atrial fibrillation. Patients who experience arterial thrombosis usually have associated severe atherosclerotic disease and may have a history consistent with chronic intestinal ischemia. Patients with mesenteric thrombosis usually are older and may have a recent history of a low–cardiac-output event.
2. Because of previous underlying vascular disease, the time until frank bowel infarction may be delayed by collateral circulation. In either case, the pain usually is sudden in onset and intermittent at first, progressing to continuous severe pain, often described as **pain out of proportion to the degree of abdominal tenderness**. These patients may also have bloody diarrhea before or after the onset of pain.
3. **Patients develop abdominal tenderness** when the bowel perforates due to transmural necrosis.
4. **Mesenteric venous thrombosis** presents with varying manifestations, ranging from an asymptomatic state to catastrophic illness. Patients usually complain of prolonged, generalized abdominal pain that develops somewhat less rapidly than with acute mesenteric arterial occlusion. These patients may have occult gastrointestinal bleeding but no frank hemorrhage.

B. **Pathophysiology.** The cause may be either an embolus to or thrombosis of the superior mesenteric artery. Bowel ischemia also can result from portomesenteric venous thrombosis.

C. Diagnosis

1. The mainstay of diagnosis is **angiography** of the mesenteric circulation, including lateral views of the celiac axis and superior mesenteric artery.
2. **Other laboratory findings** can include an elevated white blood cell count with a left shift, persistent metabolic acidosis, and an unexplained elevated serum potassium level.
3. After acute arterial occlusion, the **abdominal plain X-ray** appears relatively normal. After venous thrombosis, X-rays may show small-bowel wall thickening or air in the portal venous system. If mesenteric venous thrombosis is suspected, a CT scan may be useful in making the diagnosis.

D. Surgical therapy

1. If an embolus or thrombosis is demonstrated, an emergent **embolectomy** should be performed. If a proximal stenosis is present in the superior mesenteric artery, aortomesenteric bypass should be performed using autologous graft.
 - a. **Assessment of bowel viability** at laparotomy is made based on the gross characteristics of the bowel. The bowel is likely viable if it appears pink and if active peristalsis and arterial pulsations are present. A number of other techniques have been described, including the use of fluorescein dye, Doppler studies, and tissue oximetry, but these are not significantly better than experienced clinical judgment.
 - b. **Second-look procedures** are prudent when bowel viability is questionable. Whether to perform a second operation 24–48 hours after initial exploration is decided at the time of initial laparotomy, and that decision should not be changed even if the patient's condition improves. This approach is especially important in patients who have extensive bowel involvement in whom resection of all questionable areas would result in the loss of most of the bowel.
2. For **venous occlusion**, surgical intervention rarely is helpful, although anecdotal reports suggest that portomesenteric venous thrombectomy may be beneficial. Similarly, the role of lytic therapy in the treatment of this disorder is unclear. It is imperative to begin systemic anticoagulation as soon as the diagnosis is made to limit the thrombotic process. Frequently, the diagnosis is made at laparotomy. If the diagnosis is made before exploration, however, operation should be reserved until evidence of bowel infarction exists.

E. **Perioperative care** usually requires maximal medical support; these patients frequently are hemodynamically unstable and develop multiple organ system failure. Admission to the ICU, prolonged endotracheal intubation, parenteral nutrition, and broad-spectrum antibiotic therapy are typical.

II. Chronic intestinal ischemia

- A. **Clinical presentation.** Patients with chronic intestinal ischemia present with intestinal angina, which is pain related to eating, usually beginning within an hour after eating and abating within 4 hours. Such patients experience significant weight loss related to the decreased ability to absorb food and decreased intake secondary to recurrent pain. The diagnosis usually is made from history alone because the only physical finding may be the presence of an abdominal bruit. In patients with a low–cardiac-output state and chronic intestinal angina, persistent shock may represent acute thrombosis and intestinal necrosis. This can occur in the absence of major vessel thrombosis on angiography, and the mortality associated with this is as high as 90%.
- B. **Surgical therapy.** Elective revascularization of the superior mesenteric and celiac arteries via an autologous graft from the aorta or aortic endarterectomy is the treatment of choice. The orificial nature of most of these lesions has contributed to the poor success of balloon angioplasty.
- C. **Perioperative care.** These patients often are malnourished. Some advocate parenteral nutrition for 1–2 weeks before surgery, which is continued postoperatively. Some patients develop a revascularization syndrome consisting of abdominal pain, tachycardia, leukocytosis, and intestinal edema. Concern about the adequacy of revascularization should prompt angiography.

23 Aneurysmal Arterial Disease

Thelinh Q. Nguyen and Robert W. Thompson

Introduction

Central Arterial Aneurysms

I. Abdominal aortic aneurysms (AAAs)

II. Thoracic aortic aneurysms (TAAs)

Peripheral Arterial Aneurysms

I. Popliteal aneurysms

II. Femoral artery aneurysms

III. Subclavian artery aneurysms

IV. Ulnar artery aneurysm (hypothenar hammer syndrome)

Splanchnic Arterial Aneurysms

I. Splenic artery aneurysms

II. Hepatic artery aneurysms

Other Arterial Aneurysms

I. Renal artery aneurysms

II. Infected aneurysms

Introduction

An arterial aneurysm is a weakness of the arterial wall resulting in a permanent localized dilatation greater than 50% of the normal vessel diameter. A classification of arterial aneurysms is listed in [Table 23-1](#).

Table 23-1. Arterial aneurysm classification schemes

Central Arterial Aneurysms

I. Abdominal aortic aneurysms (AAAs)

- A. **Incidence.** AAAs are the most common type of arterial aneurysm, occurring in 3–10% of people older than 50 years in the United States. They are 5 times more common in men than in women and 3.5 times more common in Caucasians than in African-Americans.
- B. **Pathophysiology.** Ninety percent of AAAs are believed to be degenerative in origin; 5% are inflammatory. Matrix metalloproteinases have been documented to have increased activity in aneurysmal tissues. Infection and possible autoimmune processes may play a role in AAA formation. Chlamydia pneumonia, B-lymphocytes, plasma cells, and large amounts of immunoglobulin have been found in the walls of AAAs. Familial clustering of AAAs has been noted in 15–25% of patients undergoing surgery for AAAs. AAAs range from 3 to 15 cm in diameter. The risk of rupture correlates with wall tension in accordance with Laplace's law such that the risk of aneurysm rupture correlates directly with aneurysm diameter ([Fig. 23-1](#)). Ninety-five percent of AAAs are infrarenal, 25% involve the iliac arteries, and 2% involve the renal or other visceral arteries. Four percent are associated with peripheral (e.g., femoral or popliteal) aneurysms. The major complications of AAA are ruptures, of which 20% are intraperitoneal and immediately fatal.

Fig. 23-1. The 5-year risk of rupture is plotted against aneurysm size, showing the sharp increase in risk of rupture beyond a diameter of 6 cm. (From Hollier L, Rutherford RB. Infrarenal aortic aneurysms. In: Rutherford RB, ed. *Vascular surgery*, 3rd ed. Philadelphia: WB Saunders, 1989:912, with permission.)

C. Diagnosis

1. **Clinical manifestations.** Seventy-five percent of AAAs are asymptomatic and are found incidentally. Aneurysm expansion or rupture may cause severe back, flank, or abdominal pain and varying degrees of shock. Distal embolization, thrombosis, and duodenal or ureteral compression can produce symptoms.
2. **Physical examination.** Fifty percent of AAAs are identified on physical examination as a pulsatile mass at or above the umbilicus.
3. **Laboratory evaluation** is nonspecific for AAAs but may reveal important abnormalities that should be identified. Complete blood cell count, serum electrolyte and creatinine levels, blood urea nitrogen, coagulation studies, blood type and cross-matching, and urinalysis should be obtained. Erythrocyte sedimentation rate may be elevated in patients with inflammatory AAAs. In unstable patients with suspected AAA rupture, all laboratory studies except cross-matching of blood should be deferred until laparotomy.
4. **Radiologic evaluation**
 - a. **Abdominal cross-table lateral films.** In 75% of patients with an AAA, arterial wall calcification permits estimation of aneurysm diameter.
 - b. **Ultrasonography and computed tomographic (CT) scanning** demonstrate AAAs with an accuracy of 95% and 100%, respectively, and are useful for serial examinations of small aneurysms.
 - c. MR scan is comparable to CT but avoids radiation exposure and is useful in patients with intravenous contrast contraindications.
 - d. **Aortography** is not sensitive for the diagnosis of AAA because it may underestimate the aneurysm size or fail to reveal the aneurysm owing to the presence of mural thrombus. However, aortography is indicated to evaluate suspected renal or mesenteric artery stenosis and lower-extremity occlusive disease.
- D. **Differential diagnosis** includes a tortuous aorta or an abdominal mass lying adjacent to the normal aorta that might transmit aortic pulsations (e.g., lymphoma, pancreatic pseudocysts or carcinoma, mesenteric masses). AAA rupture may mimic renal colic, peritonitis, duodenal perforation, pancreatitis, degenerative spine disease, acute disk herniation, or myocardial infarction. Given its immediately life-threatening nature and potential for surgical repair, AAA must be considered first in symptomatic patients.
- E. **Elective management of AAA.** The risk of aneurysm rupture correlates best with aneurysm size ([Fig. 23-1](#)). However, even small aneurysms can rupture.
 1. **Medical management.** Patients with small aneurysms without risk factors for rupture can be followed using ultrasound or CT scan every 6 months, with larger ones followed more frequently. Smoking cessation and hypertension control are very important. There is increasing evidence suggesting that beta-blockade, particularly propranolol, may decrease the rate of AAA expansion.
 2. **Surgical management.** Indications for repair include symptomatic aneurysms of any size, aneurysms exceeding 5.0 cm, those increasing in diameter by more than 0.5 cm per year, and saccular aneurysms (usually infected). Relative indications for repair of smaller AAAs include poorly controlled hypertension (diastolic BP \geq 100 mm Hg) and significant chronic obstructive pulmonary disease (1-second forced expiratory volume $<$ 50% of predicted value). Relative contraindications to elective repair include recent myocardial infarction, intractable congestive heart failure, unreconstructible coronary artery disease, life expectancy of less than 2 years, and incapacitating neurologic deficits after a stroke.
 - a. **Assessment of risks.** Operative mortality ranges from less than 2% for uncomplicated AAA to more than 50% for ruptured AAA. Five-year survival after elective repair of AAA is no different from that for age-matched patients without AAA. Associated cardiovascular disease, hypertension, decreased renal function, chronic obstructive lung disease, and morbid obesity increase operative risk. [Table 23-2](#) outlines the cardiac evaluation of patients with an AAA.

Table 23-2. Schemes of cardiac evaluation in patients with abdominal aortic aneurysms (AAAs)

- b. **Preoperative management** includes optimizing cardiopulmonary function; a pulmonary artery catheter or a central venous line may help guide therapy in this regard. An arterial line permits continuous BP and blood gas monitoring. Two peripheral venous catheters are placed to ensure adequate access for hydration. Patients may donate autologous blood during the weeks before elective AAA repair.
- c. **Operative management.** In the standard repair, the aneurysm is approached through a midline abdominal incision and exposed by incising the retroperitoneum. Alternatively, a left retroperitoneal approach is advantageous in obese patients or those with previous intraabdominal surgery. In addition, proximal control of the aorta at or above the celiac axis is more easily achieved via this approach. Next, the duodenum and left renal vein are dissected off the aorta. After heparinization, the aorta is cross-clamped first distal and then proximal to the aneurysm. Aortotomy is then made and extended longitudinally to the aneurysm "neck," where the aorta is either transected or cut in a T fashion. The aneurysm is opened, thrombus is removed, and bleeding lumbar arteries are suture ligated. Using a tube or bifurcation graft, the proximal anastomosis is performed to nonaneurysmal aorta. The distal anastomosis is completed at the aortic bifurcation (tube graft) or at the iliac or femoral arteries (bifurcation graft), as the disease dictates. Cross-clamping can be safely performed for at least 60 minutes. After the clamps have been removed and hemostasis is ensured, the aneurysm wall is closed over the graft. Very-high-risk patients or those with infected aneurysms may undergo an extraanatomic bypass, consisting of axillofemoral bypass grafts with ligation of the aorta. Infected aneurysms should be débrided as well.
- d. **Postoperative management** involves intensive care for cardiopulmonary monitoring. The central venous pressure is measured every 2 hours and maintained near the patient's baseline. The urinary output is monitored hourly and maintained at 0.5 mL/kg per hour or greater. Hypertension should be controlled with short-acting agents, such as sodium nitroprusside or esmolol. Management is otherwise similar to that for other patients undergoing major vascular surgery.

F. Management of ruptured AAA

1. **Preoperative management.** Unstable patients are resuscitated with fluids (crystalloid, colloid, or blood) and transferred immediately to the operating room for laparotomy. Stable patients may undergo emergency ultrasonography or CT scanning to confirm the diagnosis.
2. **Operative management** is aimed at rapidly controlling the aorta. Anesthetic induction is delayed until the surgeon is ready to make the abdominal incision. Through a midline incision, the aorta is cross-clamped or compressed at the diaphragmatic hiatus. The retroperitoneal hematoma is opened, and the aneurysm is identified. After obtaining distal control of the iliac and collateral arteries, the proximal clamp is placed immediately above the aneurysm to allow perfusion of the visceral and renal arteries. Subsequent management is similar to repair of an elective AAA. Bifurcation grafts should be avoided in favor of the more expeditious tube graft techniques. In addition, heparin should be avoided.
3. **Postoperative management** is similar to that for elective AAA repair.

G. Endovascular management of AAA is discussed in [Chapter 24](#).

H. Complications

1. **Arrhythmia, myocardial ischemia, or infarction** may occur.
2. **Intraoperative hemorrhage** can be reduced by clamping the aorta proximal to the aneurysm and the iliac arteries distally. Once the aneurysm is opened, retrograde bleeding from lumbar arteries must be controlled rapidly with transfixing ligatures. Blood should be salvaged in the operating room and autotransfused to the patient.
3. **Aortic cross-clamping shock**, which may occur on release of the aortic cross-clamp, may be obviated by adequate hydration and slow release of the aortic cross-clamp.
4. **Renal insufficiency** may be related to intravenous contrast, inadequate hydration, hypotension, a period of aortic clamping above the renal arteries, or embolization of the renal arteries.
5. **Lower-extremity ischemia** may result from embolism or thrombosis, especially in emergency operations for which heparin might not be used. Embolism to the lower extremities can be prevented by perfusing the hypogastric arteries before perfusing the external iliac arteries or by using a Fogarty catheter to remove a clot from lower-extremity vessels.
6. **Microemboli** arising from atherosclerotic debris can cause cutaneous ischemia ("trash foot"), which should be treated expectantly. Amputation may be required if significant necrosis results.
7. **Gastrointestinal complications** may consist of prolonged paralytic ileus, anorexia, periodic constipation, or diarrhea. This problem is diminished by using the left retroperitoneal approach. A more serious complication, ischemic colitis of the sigmoid colon, is related to ligation of the inferior mesenteric artery in the absence of adequate collateral circulation. Symptoms include leukocytosis, large fluid requirement in the first 8–12 postoperative hours, fever, and peritoneal irritation. Diagnosis is made by sigmoidoscopy to 20 cm above the anal verge. Necrosis that is limited to the mucosa may be treated expectantly. Necrosis of the muscularis causes segmental strictures, which may require delayed segmental resection. Transmural necrosis requires immediate resection of necrotic colon and construction of an end colostomy. Contaminated grafts require removal and extraanatomic bypass.
8. **Paraplegia** may occur after repair of a ruptured AAA when the aorta is clamped near the diaphragm, owing to spinal cord ischemia. Paraplegia is rare following elective infrarenal AAA repair. Obliteration or embolization of important spinal artery collateral flow via the internal iliac arteries or an abnormally low origin of the accessory spinal artery (artery of Adamkiewicz) can result in paraplegia.
9. **Sexual dysfunction** and retrograde ejaculation result from damage to the sympathetic plexus during dissection near the aortic bifurcation, especially the proximal left common iliac artery.

II. Thoracic aortic aneurysms (TAAs)

- A. **Incidence.** Ascending and transverse arch aneurysms each comprise 25% of TAAs. The remaining 50% occur in the descending aorta (thoracic or thoracoabdominal).
- B. **Pathophysiology.** The most common causes of TAAs are degenerative (atherosclerosis), dissection (see [Chapter 36](#)), inherited degenerative disease (Marfan's syndrome), idiopathic aortic annular dilatation, and trauma. TAAs are divided into five main types: ascending, transverse, descending, thoracoabdominal, and traumatic. Ascending aortic aneurysms usually are caused by medial degeneration. Transverse, descending, and thoracoabdominal aortic aneurysms are related to atherosclerosis. Most descending TAAs begin just distal to the left subclavian artery. Traumatic aortic aneurysms (actually pseudoaneurysms) are caused by blunt chest trauma that produces partial aortic disruption.
- C. **Diagnosis**
 1. **Clinical manifestations** usually are absent; most nontraumatic TAAs are detected as incidental findings on chest films obtained for other purposes. However, patients may present with chest discomfort or pain that intensifies with aneurysm expansion or rupture, aortic valvular regurgitation, congestive heart failure, compression of adjacent structures (recurrent laryngeal nerve, left main-stem bronchus, esophagus, superior vena cava, porta hepatis), or erosion into adjacent structures (esophagus, lung, airway).
 2. **Physical examination** often is normal or reveals findings consistent with the presenting signs or symptoms.
 3. **Laboratory tests** are nonspecific but should include complete blood cell count, serum electrolyte and creatinine levels, blood urea nitrogen, coagulation studies, blood type and cross-matching, and urinalysis in addition to other appropriate studies.
 4. **Radiologic evaluation**
 - a. **Chest films** may reveal a widened mediastinum or an enlarged calcific aortic shadow. Traumatic aneurysms may be associated with skeletal fractures.
 - b. **MR or CT scanning** with intravenous contrast provides precise estimation of the size and extent of these aneurysms and facilitates the planning of surgical therapy.
 - c. **Echocardiography** is useful in evaluating aneurysms involving the aortic arch.
 - d. **Aortography** demonstrates the proximal and distal extent of the aneurysm and the vessels arising from it.
- D. **Management** depends on the type and location of the TAA. Repair of proximal arch aneurysms requires cardiopulmonary bypass and circulatory arrest. Preclotted woven polyethylene terephthalate (Dacron) is the graft of choice. The ascending and transverse arches are repaired through a median sternotomy incision. The descending and thoracoabdominal aortas are approached through a left posterolateral thoracotomy incision. Intraoperative management of patients undergoing thoracotomy is facilitated by selective ventilation of the right lung using a double-lumen endobronchial tube. Cerebrospinal fluid drainage during and after surgery can lower the incidence of postoperative paraplegia.
 1. **Ascending aortic arch aneurysms**
 - a. **Indications** for surgical repair include symptomatic or rapidly expanding aneurysms, aneurysms greater than 6 cm in diameter, ascending aortic dissections, mycotic aneurysms, and asymptomatic aneurysms greater than 5.5 cm in diameter in patients with Marfan's syndrome.
 - b. **Operative management.** An aneurysm arising distal to the coronary ostia is replaced with an interposition graft. A proximal aneurysm resulting in aortic valve incompetency is replaced with a composite valved conduit (Bentall procedure) or a supracoronary graft with separate aortic valve replacement.

Ascending arch aneurysms due to Marfan's syndrome or cystic medial necrosis are repaired with aortic valve replacement owing to the high incidence of valvular incompetence associated with aneurysmal dilation of the native aortic root. When a composite graft is used, the coronary arteries are anastomosed directly to the conduit.

2. **Transverse aortic arch aneurysms**
 - a. **Indications** for repair include aneurysms greater than 6 cm in diameter, aortic arch dissections, and ascending arch aneurysms that extend into the transverse arch.
 - b. **Operative management.** After opening the aorta under hypothermic circulatory arrest, the distal anastomosis is performed using a beveled graft, followed by anastomosis of an island of the brachiocephalic vessels to the superior aspect of the graft. The proximal anastomosis is constructed to the supracoronary aorta (if the aortic valve is not involved) or to a segment of composite valved conduit interposed to complete the arch reconstruction. Involvement of the transverse arch and its branch vessels requires interposition grafting to the involved vessels.
3. **Descending TAAs**
 - a. **Indications** for repair include symptomatic aneurysms and asymptomatic aneurysms greater than 6 cm in diameter.
 - b. **Operative management.** Before cross-clamping, the anesthesiologist pharmacologically controls proximal BP with sodium nitroprusside. After the distal clamp is applied, a proximal clamp is placed just distal to the left subclavian artery or between the left common carotid and left subclavian arteries. Selected intercostal branches are reattached to the aortic interposition graft.
 - c. **Endovascular technology** has also been applied to treatment of descending TAA using self-expanding stainless steel stents covered with polyethylene terephthalate. Aneurysms well suited to this approach are relatively straight, have 2 cm or more of normal aorta distal to the left subclavian artery, and have the same length of normal aorta proximal to the celiac axis. Some series report a perioperative mortality of approximately 9%, which compares well to the standard surgical repair. Patients who have both an AAA and a TAA of the descending aorta may be candidates for this approach. The treatment strategy in this group is to repair the AAA as an open operation and place a stent-graft across the TAA via a temporary side limb sewn to the abdominal aortic prosthesis.
4. **Thoracoabdominal aneurysms**
 - a. **Indications** for repair include symptomatic aneurysms and aneurysms greater than 6 cm in diameter.
 - b. **Operative management** consists of tube or patch (if small and noncircumferential) graft replacement, along with anastomosis of major branches to the graft. Aneurysms involving the thoracic and proximal abdominal aortic segments may be approached through a left posterolateral thoracotomy extended to the umbilicus. Sodium nitroprusside is given before cross-clamping to reduce proximal BP. Cerebrospinal fluid drainage may decrease the incidence of postoperative paraplegia. Heparin, shunting, and hypothermia generally are not used. The aneurysm is opened, and the orifices of all major aortic branches are occluded with balloon catheters or suture ligatures. The proximal anastomosis is performed, followed by end-to-side anastomosis of significant aortic branches to the graft. The distal anastomosis is made to uninvolved aorta or to the iliac or femoral arteries.
5. **Traumatic aortic aneurysms**
 - a. **Indications.** Urgent repair is indicated except when precluded by more compelling life-threatening injuries or major central nervous system trauma.
 - b. **Operative management.** Using proximal and distal control, these aneurysms may be repaired with primary aortorrhaphy, aneurysmectomy, and end-to-end reanastomosis, or interposition grafting.
- E. **Complications.** Possible complications of thoracic aortic surgery include arrhythmia, myocardial infarction, intraoperative hemorrhage, stroke, aortic cross-clamp shock, renal insufficiency, lower-extremity ischemia, microemboli, and disseminated intravascular coagulopathy. The incidence of paraplegia can be reduced by multimodal therapies implemented to prevent or minimize spinal cord ischemia: distal aortic perfusion, intercostal and lumbar artery reimplantation, preoperative or intraoperative localization of spinal blood supply, hypothermia, cerebrospinal fluid drainage, or pharmacotherapy.

Peripheral Arterial Aneurysms

Popliteal and femoral artery aneurysms account for more than 90% of all peripheral aneurysms and usually are associated with aneurysms in other locations. The male-female ratio is greater than 30:1. Nearly all are degenerative and associated with atherosclerosis. These aneurysms have a propensity to thrombose, but rupture, compression of adjacent structures, or distal embolization can occur.

I. Popliteal aneurysms

- A. **Incidence.** Popliteal aneurysms account for nearly 70% of peripheral aneurysms. Fifty percent to 70% are bilateral, 40–50% are associated with AAA, and nearly 40% have femoral artery aneurysm.
- B. **Pathophysiology.** In addition to atherosclerosis, trauma and infection (e.g., syphilis) can also cause these aneurysms.
- C. **Diagnosis**
 1. **Clinical manifestations** related to thromboembolism include claudication, rest pain, ulceration, and neuropathy. Mass effect may produce a pulsatile mass or venous obstruction. Approximately 45% are asymptomatic.
 2. **Physical examination** may reveal a pulsatile mass in the popliteal fossa if the aneurysm is patent. Conversely, a thrombosed popliteal aneurysm may feel firm but not pulsatile.
 3. **Laboratory tests** are nonspecific.
 4. **Radiologic evaluation**
 - a. **Ultrasonography, CT scanning** with intravenous contrast, or **MR scanning** confirms the diagnosis and is used to rule out bilateral aneurysms or an associated AAA.
 - b. **Lower-extremity plain films** may demonstrate calcified aneurysms.
 - c. **Arteriography** is useful in evaluating distal runoff and in planning reconstruction.
- D. **Operative management** is aimed at preventing thromboembolic complications and restoring adequate blood flow to the distal extremity. The procedure most often used is ligation of the artery proximal and distal to the aneurysm to exclude it from the circulation, followed by bypass, preferably using an autologous conduit. Alternatively, popliteal aneurysms may be repaired with aneurysmectomy and interposition of autologous vein or prosthetic graft. For thrombosed aneurysms, preoperative thrombolysis lyses thrombosed runoff vessels for potential bypass outflow and improves limb salvage. An intraoperative arteriogram after repair is recommended to confirm adequacy of outflow. Amputation is required in 10–20% of patients who present with acute thrombosis and limb-threatening ischemia.

II. Femoral artery aneurysms

- A. **Incidence.** Femoral artery aneurysms are the second most common peripheral aneurysm. Seventy percent are bilateral, 80% are associated with AAA, and 40% are associated with popliteal aneurysms.
- B. **Pathophysiology.** Fifty percent of these aneurysms occur proximal to the femoral bifurcation (type I); the other 50% involve the profunda femoris (type II). Degenerative disease (atherosclerosis) is the major etiology, although connective tissue disorders can also play a role.
- C. **Diagnosis**
 1. **Clinical manifestations** usually are limited to a pulsatile groin mass. Approximately 40% are asymptomatic at the time of diagnosis. Symptomatic patients may present with local pain, mass effect with nerve or vein compression, or lower-extremity ischemia.
 2. **Laboratory tests** are nonspecific.
 3. **Radiologic evaluation**
 - a. **Ultrasonography, CT, or MR scan** can confirm the diagnosis and is useful in evaluating the infrarenal aorta and popliteal regions.
 - b. **Angiography** is useful in demonstrating involvement of the profunda femoris ostium and in evaluating distal runoff.
- D. **Differential diagnosis** includes pseudoaneurysms due to percutaneous catheterization, anastomotic leaks, trauma, or intravenous drug abuse misadventures.
- E. **Operative management** consists of aneurysmectomy and end-to-end graft replacement using autologous vein or prosthetic graft repair. In type II aneurysms, a separate graft can be used to connect the profunda femoris to the main graft. Preservation of the profunda femoris is necessary for limb salvage.

III. Subclavian artery aneurysms

- A. **Proximal subclavian artery aneurysms** arise most commonly from atherosclerosis. The presence of such an aneurysm is associated with a 30–50% incidence of aortoiliac or peripheral artery aneurysms and should therefore provoke an evaluation to identify synchronous aneurysmal disease.
 1. **Presentation** of symptomatic subclavian artery aneurysms can be characterized into three categories.
 - a. **Acute expansion or rupture** may cause pain in the patient's neck, chest, or shoulder.
 - b. **Local compression** of adjacent structures, such as the brachial plexus, recurrent laryngeal nerve, or trachea, may cause corresponding symptoms of upper-extremity neuropathies, hoarseness, or respiratory distress, respectively.

- c. **Ischemia** can occur due to thromboembolism to the brain or the upper extremity.
 - 2. **Physical examination** may reveal a pulsatile supraclavicular mass or bruit, absent or diminished upper-extremity pulses, “blue finger syndrome,” sensory and motor deficits in the ipsilateral upper extremity, vocal cord paralysis, or Horner’s syndrome.
 - 3. **Diagnosis** is made using complete aortic arch and upper-extremity angiography. This imaging test defines the aneurysm, assesses for distal occlusive disease, and evaluates the contralateral vertebral system, especially when the ipsilateral vertebral artery arises from the aneurysm.
 - 4. **Treatment** of proximal subclavian artery aneurysms requires resection with either primary anastomosis or interposition grafting using prosthetic or autologous grafts. A lesion on the right is generally approached through a median sternotomy, whereas a left-sided aneurysm can be treated through a left thoracotomy.
- B. **Distal subclavian artery aneurysms** are also called *subclavian-axillary artery aneurysms*. They arise secondary to compression of the artery by a cervical rib or band of fibrous and muscular tissue as part of thoracic outlet syndrome. Poststenotic dilatation progresses to aneurysmal changes in the arterial wall over time. Once the arterial wall is aneurysmal, symptoms of ischemia or local compression develop as with proximal subclavian aneurysms.
- 1. **Diagnosis** is established with angiography as previously described (see [section III.A.3](#)).
 - 2. **Treatment** includes complete resection of the cervical rib and scalenus anterior. Aneurysm resection is indicated for asymptomatic patients with aneurysms larger than twice the normal vessel diameter or those with thromboembolic complications. Catheter embolectomy is mandatory for recently occluded distal arteries. Reconstruction of the subclavian artery is similar to that for proximal lesions (see [section III.A.4](#)).

IV. Ulnar artery aneurysm (hypothenar hammer syndrome)

- A. **Incidence.** Ulnar artery aneurysms are most commonly seen in men younger than 50 years of age and in people who use the palms of their hands for pushing, pounding, or twisting.
- B. **Pathophysiology.** Repetitive trauma to the ulnar artery is the most common cause.
- C. **Diagnosis**
 - 1. **Clinical manifestations.** Many patients have a severe lacerating pain over the hypothenar eminence at the time of injury followed by a chronic, dull aching pain. Ischemic symptoms, most commonly of the fourth and fifth fingers or any digit except the thumb, develop weeks or months later.
 - 2. **Physical examination** reveals the ischemic changes of the fingers, tenderness, and/or pulsatile mass over the hypothenar eminence. An abnormal Allen’s test is present in the majority of patients.
 - 3. **Radiologic evaluation**
 - a. **Digital plethysmography and duplex ultrasonography** are helpful in making the diagnosis.
 - b. **Angiography** is mandatory.
 - 4. **Treatments** include cervicodorsal sympathectomy, excision of the ulnar artery aneurysm with ligation of the ulnar artery, and aneurysmectomy with microsurgical reconstruction of the ulnar artery by reanastomosis or interposition vein graft. Preoperative thrombolytic therapy plays an important role in patients with ulnar aneurysm thrombosis. Medical therapies including calcium channel blockers, smoking cessation, and avoiding further hand trauma can also be helpful.

Splanchnic Arterial Aneurysms

Splanchnic artery aneurysms are uncommon. The arteries most often involved are the splenic (60%), hepatic (20%), superior mesenteric (5.5%), celiac (4%), gastric and gastropiploic (4%), and intestinal (3%) arteries. Most of these aneurysms are caused by atherosclerosis. The risk of rupture is greater than 50% in hepatic, celiac, and superior mesenteric aneurysms.

I. Splenic artery aneurysms

- A. **Incidence.** These aneurysms occur in 0.02–0.16% of the United States population, with a 4:1 female predominance in women of childbearing age. Incidence correlates with multiparity.
- B. **Pathophysiology** is related to systemic arterial fibrodysplasia, portal hypertension, and increased splenic blood flow during pregnancy. Most splenic artery aneurysms occur at the bifurcation of the distal splenic artery. Twenty percent are multiple. Rupture most often occurs into the lesser sac and can progress to free intraperitoneal hemorrhage via the foramen of Winslow. The overall risk of rupture is 5–10%; however, 95% of ruptures occur during pregnancy.
- C. **Diagnosis**
 - 1. **Clinical manifestations.** Most are asymptomatic. Vague left upper quadrant or epigastric pain may occur with acute aneurysm enlargement. Gastrointestinal bleeding occurs with rupture into the stomach or pancreatic duct.
 - 2. **Physical examination** may confirm the aforementioned signs and symptoms.
 - 3. **Laboratory tests** are nonspecific.
 - 4. **Radiologic evaluation**
 - a. **Abdominal plain films** reveal a signet ring–like calcification in the left upper quadrant in 70% of patients.
 - b. **CT or MR scan** often can visualize the aneurysm and confirm any leak from it.
 - c. **Aortography** confirms the diagnosis.
- D. **Differential diagnosis** for ruptured splenic aneurysms in pregnant women includes placental abruption, uterine rupture, and amniotic fluid embolization. A ruptured hepatic adenoma should also be ruled out. Splenic artery pseudoaneurysms secondary to pancreatitis can present in a manner similar to that of true splenic artery aneurysms.
- E. **Operative management** is indicated in symptomatic aneurysms, pregnant patients, and women who anticipate future pregnancy. Proximal aneurysms are approached through the lesser sac and are treated with aneurysmectomy or ligation without arterial reconstruction. Midsplenic artery aneurysms can be ligated from within the aneurysm sac. Distal aneurysms may be resected with a distal pancreatectomy. Splenic hilar aneurysms are treated with splenectomy, suture ligation, or aneurysmorrhaphy. Transcatheter embolization may be performed in high-risk patients. After aneurysm rupture, operative maternal and fetal mortality is 70% and 95%, respectively.

II. Hepatic artery aneurysms

- A. **Incidence.** These are the second most common splanchnic artery aneurysms, occurring most often in the elderly, with a male-female ratio of 2:1.
- B. **Pathophysiology** is related to atherosclerosis (32%), medial degeneration (24%), trauma (22%), and mycotic infection (10%). The vast majority are extrahepatic and involve the common hepatic (63%), right hepatic (29%), left hepatic (5%), or right and left hepatic (4%) arteries. Rupture and compression of the hepatobiliary tree are the most frequent complications. Fifty percent of ruptures are intraperitoneal; the other 50% are into the hepatobiliary tree.
- C. **Diagnosis** usually is made incidentally.
 - 1. **Clinical manifestations** usually are absent but may include persistent right upper quadrant or epigastric pain. Severe pain or radiation to the patient’s back suggests an expanding aneurysm. Rupture into the hepatobiliary tree produces hematemesis, biliary colic, and jaundice.
 - 2. **Physical examination** may reveal an abdominal bruit or a pulsatile right upper quadrant or an epigastric mass (uncommon).
 - 3. **Laboratory tests** may reveal elevated serum bilirubin, alkaline phosphatase, or liver enzymes in the presence of hepatobiliary obstruction.
 - 4. **Radiologic evaluation.** Arteriography is the diagnostic tool of choice and is recommended for planning surgical therapy. Many aneurysms are identified first on a CT scan.
- D. **Differential diagnosis** includes AAA, cholecystitis, pancreatitis, and perforated duodenal ulcer.
- E. **Operative management** usually is indicated owing to the high mortality (>35%) associated with rupture. Common hepatic artery aneurysms can be treated with aneurysmectomy, aneurysmorrhaphy, or aneurysm exclusion with or without arterial reconstruction, depending on the adequacy of collateral flow. Aneurysms of the proper hepatic artery are treated with aneurysmorrhaphy, aneurysmectomy with primary artery repair (small aneurysms), or bypass. Intrahepatic aneurysms can be treated with liver resection, proximal artery ligation, or percutaneous transcatheter embolization (in high-risk patients).

Other Arterial Aneurysms

I. Renal artery aneurysms

- A. **Incidence.** Renal artery aneurysms occur in 1–10% of the population and constitute approximately 1% of all aneurysms.
- B. **Pathophysiology.** These aneurysms may be either extrarenal (85%) or intrarenal (15%) in location. Extrarenal renal artery aneurysms are subdivided into

saccular (most common), fusiform, and dissecting. Saccular aneurysms classically occur near the bifurcation of the renal artery. Fusiform aneurysms are poststenotic dilatations associated with renal artery stenosis. Renal artery dissections are associated with renal artery fibroplasias. Intrarenal aneurysms may be congenital, traumatic, or related to collagen vascular disease.

C. **Diagnosis** usually is made incidentally.

1. **Clinical manifestations** usually are absent until complications arise. Rupture and dissection may produce flank pain or hematuria (intrarenal aneurysms).
2. **Physical examination** commonly reveals hypertension and an abdominal bruit. A palpable mass occurs in fewer than 10% of cases.
3. **Laboratory tests** may reveal anemia or hematuria.
4. **Radiologic evaluation**
 - a. **Abdominal films** may demonstrate ring-shaped calcifications in the renal hilum in patients with calcific saccular aneurysms.
 - b. **CT scan** may reveal an incidental renal artery aneurysm.
 - c. **Arteriography** confirms the diagnosis and details the anatomy of the renal branches.

D. **Operative management.** Treatment is indicated for aneurysms that rupture, are associated with dissection, or produce renal artery stenosis leading to hypertension. Saccular aneurysms are repaired in pregnant women owing to an increased risk of rupture. Small aneurysms at the bifurcation can be treated with aneurysmectomy and reconstruction of the bifurcation. Aneurysmectomy with aortorenal or splenorenal bypass is advised for large aneurysms or stenotic lesions. Polar renal artery aneurysms can be excised with end-to-end arterial reanastomosis. Ruptured renal artery aneurysms are treated with nephrectomy.

II. Infected aneurysms

A. **Incidence.** The incidence of infected arterial aneurysms has risen with the increased prevalence of immunocompromised patients, invasive transarterial procedures, and drug addiction.

B. **Pathophysiology.** Infected aneurysms can be divided into four types: mycotic aneurysm, microbial arteritis with aneurysm, infected preexisting aneurysm, and posttraumatic infected false aneurysm. The clinical characteristics of each type are summarized in [Table 23-3](#). *Staphylococcus aureus* is the most common pathogen. *Salmonella* species (arteritis), *Streptococcus* species, and *Staphylococcus epidermidis* (preexisting aneurysms) also predominate. The risk of rupture for gram-negative infections exceeds that for gram-positive infections.

Table 23-3. Clinical characteristics of infected aneurysm

C. **Diagnosis**

1. **Clinical manifestations** may be absent or include fever, tenderness, or sepsis.
2. **Physical examination** may demonstrate a tender, warm, palpable mass in an extremity aneurysm.
3. **Laboratory tests** may reveal leukocytosis. Aerobic and anaerobic blood cultures should be obtained, but only 50% of cultures are positive.
4. **Radiologic evaluation**
 - a. **Abdominal plain films.** Aneurysms associated with vertebral body erosion suggest infection.
 - b. **MR or CT scanning** can demonstrate an aneurysm and verify its rupture.
 - c. **Angiography** delineates the characteristics of the aneurysm. Aneurysms that are saccular, multilobed, or eccentric with a narrow neck are more likely a result of infection.

D. **Management**

1. **Preoperative management.** Broad-spectrum antibiotics should be administered intravenously after aerobic and anaerobic blood cultures have been obtained.
2. **Operative management** includes (1) controlling hemorrhage; (2) obtaining arterial specimens for Gram stain, aerobic and anaerobic cultures, and drug sensitivities; (3) resecting the aneurysm with wide débridement and drainage; and (4) reconstructing major arteries through uninfected tissue planes. Extraanatomic bypass may be necessary to avoid contamination of the graft.
3. **Postoperative management** requires adequate drainage of the aneurysm cavity and long-term antibiotic therapy.

24 Endovascular Surgery

Jason K. Lee and Luis A. Sanchez

Introduction

Techniques and Indications for Endovascular Surgery

I. Techniques and devices

II. Specific indications for endovascular surgery

Introduction

Endovascular approaches to the treatment of peripheral vascular disease allow avoidance of major vascular reconstructions with its associated morbidity and costs. The endovascular approach also provides a favorable treatment alternative to individuals at high risk for complications with traditional vascular reconstructions. Development of a variety of endovascular techniques and devices has allowed a broader application of endovascular surgery to peripheral vascular disease. Because of the ongoing evolution of devices and endovascular techniques, there are few long-term clinical outcomes data, and guidelines for the application of endovascular techniques are evolving.

Techniques and Indications for Endovascular Surgery

I. Techniques and devices

- A. **Angioscopy.** Satisfactory endovascular visualization requires a small, flexible fiberoptic scope and sufficient irrigation to avoid opacification by blood.
 1. **General indications.** Angioscopy can be used to assess various interventions, including thromboembolectomy, endarterectomy, venous valvulotomy, and venous bypass side branch occlusion. Angioscopy has been found to be most useful in infrainguinal bypass graft procedures (with *in situ* vein bypass and with revision of failed bypass grafts).
 2. **Complications.** Adequate visualization may require vigorous irrigation. This may lead to fluid overload or air embolization. Passage of the angioscope may also injure the vessel wall or venous conduit.
- B. **Intravascular ultrasound** allows evaluation of vessel wall morphology and intraluminal pathology.
 1. **General indications**
 - a. **Allows diagnosis of complex arterial pathology**
 - b. **Provides guidance in maneuvering intravascular instruments**
 - c. **Evaluates results of endovascular interventions**
 2. **Complications** include vessel wall injury with passage of the ultrasound probe.
- C. **Balloon angioplasty**
 1. **General indications.** Best results are obtained with short-segment (<10 cm) stenoses within large, high-flow vessels (e.g., iliac artery).
 2. **Results.** Patency rates for balloon angioplasty of occlusive lesions are generally inferior to those of open surgical bypass except at the aortoiliac level. Unsuccessful angioplasty usually occurs because of inability to cross the lesion, elastic recoil, severe arterial calcification, and vessel wall dissection. However, angioplasty failures and restenoses are often amenable to repeat endovascular intervention with variable results.
 3. **Complications.** Balloon angioplasty causes luminal dilation by fracturing the occlusive atherosclerotic plaque. This may expose the bloodstream to thrombogenic material, which may lead to vessel occlusion. Balloon dilation also may create an intimal flap, leading to medial dissection and luminal narrowing or occlusion.
- D. **Intravascular stenting.** Two types of intravascular stents are available: balloon expandable (e.g., Palmaz stent) and self-expanding (Wallstent, Nitinol stent). Important characteristics of balloon-expandable stents include significant rigidity, radial strength, and minimal stent shortening with deployment. By contrast, self-expanding stents are more flexible, have less radial strength, and often shorten with expansion, making precise stent placement more difficult.
 1. **General indications.** Intravascular stents have been used to address occlusive arterial lesions in a variety of locations. Stent placement has generally been reserved for cases of early and late angioplasty failure (i.e., unsuccessful angioplasty and restenosis after prior angioplasty). However, at this time, stents are often used primarily in lesions known to have decreased patency with angioplasty alone. Their role for infrainguinal disease is still very limited.
 2. **Results.** Restenosis rates after intravascular stent deployment have been shown to be similar to results with balloon angioplasty treatment alone in infrainguinal vessels but better in other territories (i.e., iliac, renal, aortic arch branches).
 3. **Complications.** In addition to complications associated with device introduction (embolization, dissection, vessel rupture, thrombosis, pseudoaneurysm formation at the arteriotomy site), stent migration or inappropriate stent deployment may occur. Pseudoaneurysms may also occur at the site of stent deployment.
- E. **Endovascular grafts**
 1. **General indications.** Intravascular stents covered with prosthetic graft material [polytetrafluoroethylene (PTFE) or Dacron] have been developed for treatment of aneurysmal, occlusive, and traumatic arterial lesions. Early designs included stent elements supporting the ends of the graft. However, most of the current designs include full-length stent support to prevent rotation or kinking of the unsupported graft.
 2. **Results.** Overall, there has been limited experience with these devices. The short-term (<4 years) results have been encouraging in the treatment of aneurysms, selected occlusive lesions, and traumatic arterial lesions.
 3. **Complications** are similar to those with intravascular stents.
- F. **Atherectomy devices.** Various devices have been developed to mechanically debulk arterial occlusive lesions.
 1. **General indications.** The indications for use of atherectomy devices are very narrow in patients with peripheral vascular disease: instances in which balloon angioplasty is unsuccessful or contraindicated (e.g., highly calcified lesions, emboligenic ulcerated lesions, dissections) and the lesions are focal.
 2. **Results and complications.** These devices are associated with significant complication rates (i.e., early thromboses). In addition, none of these devices prevents restenosis, and long-term results are no better than with balloon angioplasty alone.

II. Specific indications for endovascular surgery

A. Aneurysms

1. **Abdominal aortic aneurysm (AAA)**
 - a. **Indications.** The indications for endovascular treatment of AAA are no different than for traditional open operative repair. Generally, any symptomatic AAA or aneurysm with a diameter greater than 5.0 cm warrants surgical intervention.
 - b. **Selection criteria.** The most important selection criteria for endovascular treatment of abdominal AAAs is aortoiliac anatomy. Assessment of the proximal neck of the AAA includes the following factors: length and diameter of nondilated and healthy infrarenal aorta, the angle between the neck and aneurysm, the presence of intraluminal thrombus, and the shape of the aortic neck. Most devices require a 1.5-cm length proximal neck, and some devices allow suprarenal attachment with an open stent segment. Current endograft dimensions at the proximal end are up to 32 mm in diameter. Significant angulation between the neck and adjacent aneurysm (>60 degrees) makes proximal graft deployment technically difficult. Significant mural thrombus or atheroma in the proximal neck also would prevent adequate seating of an endograft and therefore represents a contraindication for endovascular treatment. The proximal neck segment geometry is also important in that a cone-shaped neck or reverse taper (i.e., widens more distally) precludes adequate apposition of the endograft to the aortic wall. The distal neck of the AAA is important only for placement of straight aortic endografts, which are rarely used currently. The vast majority of aortic endografts placed today are bifurcated and extend to the iliac arteries.
 - c. **The anatomy of the iliac arteries** also has a significant influence on whether an aortic endograft can be placed successfully. Iliac artery tortuosity, calcification, and luminal narrowing in combination with the profile of the delivery system are critical factors for successful endograft delivery and deployment without complication. Patent aortic branches may influence the decision as to whether to proceed with endograft placement. A renal artery or accessory renal artery arising from the proximal neck or aneurysm or the presence of a horseshoe kidney with multiple renal arteries is often a contraindication for endograft placement. Patent lumbar arteries arising from the aneurysm do not preclude endograft placement. Although some have suggested preoperative embolization of lumbar branches to prevent endoleaks, many have observed spontaneous thrombosis of these vessels after graft deployment. A patent inferior mesenteric artery (IMA) associated with large mesenteric collaterals (e.g., meandering mesenteric artery) or a large patent IMA suggests abnormal mesenteric blood supply and risk of large-bowel ischemia with endograft coverage of the IMA orifice. Therefore, these vascular

patterns are contraindications to endograft placement.

- d. **Imaging studies.** Spiral computed tomographic (CT) scans with three-dimensional reconstruction are obtained to assess anatomic feasibility of endovascular stent-graft deployment. Calibrated conventional angiography is also obtained to assess aneurysm length and luminal dimensions for selection of endovascular graft attachment zones.
 - e. **Technique.** All current aortic endovascular grafts require femoral artery exposure and arteriotomy for device introduction. Most endovascular grafts are bifurcated, and many devices are modular (consisting of 2 or more components). Endografts typically have a full-length stent skeleton that prevents graft kinking. The deployment is mostly by unsheathing of self-expanding stents. Oversized stent-grafts (by 10–15%) are selected based on preoperative assessment of arterial diameter to ensure adequate graft apposition to the aortic wall. If necessary, graft length can be tailored with the use of overlapping extension segments in modular devices. Positioning of the iliac limbs in bifurcated unibody aortic stent grafts is performed using guidewires and pullwires placed across the aortic bifurcation. Modular aortic stent-grafts include a bifurcated portion with short and long limbs. After deployment of the main graft and long limb, a separate wire is placed through the short graft limb in a retrograde fashion from the contralateral femoral artery to guide deployment of an appropriate limb extension. The hypogastric artery is identified by intraoperative angiography, and the distal attachment site of the stent-graft is positioned proximal to the hypogastric artery.
 - f. **Complications.** Complications of endograft placement include distal embolization, graft thrombosis, and arterial rupture (especially external iliac artery avulsion at the common iliac bifurcation in patients with tortuous diseased iliac arteries). These complications are addressed urgently when they are discovered and may require emergent conversion to open repair. Arterial dissection may occur with device introduction, but this may not require additional treatment if the endograft spans the segment of dissection. As with open surgical AAA repair, bowel ischemia may occur postoperatively secondary to embolization or hypoperfusion. Renal dysfunction may occur due to the nephrotoxicity of the contrast agent used for intraoperative angiography or direct injury due to embolization or renal artery occlusion. **Endoleak**, a unique complication of endovascular repair, refers to blood flow within the aneurysm sac but outside of the graft. Management strategies for endoleaks discovered on follow-up imaging studies are evolving. In general, endoleaks from the proximal or distal attachment sites (type I) warrant intervention, because these are associated with increasing aneurysm sac size. Endoleak from collateral flow or type II (IMA, lumbar arteries) are generally closely observed. For any endoleak that is associated with aneurysm sac enlargement, intervention is required. Treatment of endoleaks may be done with open surgery or endovascular techniques. Proximal endoleaks may be sealed with angioplasty or placement of a stent graft segment; collateral bleeding may be treated with embolization through collateral vessels (via the hypogastric artery for lumbar branch bleeding; via the superior mesenteric artery for IMA bleeding) or directly through the aneurysm sac via a translumbar approach.
 - g. **Results.** The short and midterm results (4 years) are encouraging, but long-term results are not yet available. Close follow-up with CT scanning every 3–6 months initially and yearly after the first year is essential to understand the behavior of these devices. Future fenestrated and branch devices may allow treatment of pararenal AAAs.
2. **Thoracic aortic aneurysm (TAA)**
 - a. **Indications and technique.** Because of the considerable morbidity and mortality associated with surgical repair of descending TAAs, the endovascular approach to aneurysm exclusion is especially attractive. The decision to proceed with endovascular graft placement is based on similar anatomic considerations as for AAA: adequate length (2 cm) and diameter (20–40 mm) of the proximal and distal aneurysm necks, cylindrical shape of aneurysm neck, absence of significant mural thrombus within the neck, and aortic and iliofemoral anatomy amenable to device introduction. In situations in which proximal neck length is too short, seating of the proximal graft end over the origin of the left subclavian artery has been performed successfully with or without an adjunctive left carotid–left subclavian transposition or bypass.
 - b. **Results and complications.** Early experience with TAA stent-grafting was associated with a significant incidence (~25%) of endoleaks when “homemade” devices were being used. The early results of commercial devices being tested are very encouraging. In addition to cardiovascular- and cerebrovascular-related complications, cases of paraplegia have been reported, but the rate seems to be lower than with open surgical repair despite occlusion of multiple intercostals during endovascular repair. Future fenestrated and branched devices may allow endovascular treatment of more complex arch and thoracoabdominal aneurysms.
 3. **Aortic dissection: indications and technique.** In descending aortic dissections (type B, Stanford classification), organ ischemia may occur under two situations: The dissection proceeds into branch vessels without a reentry lesion, causing narrowing of the true lumen, or the intimal flap of a dissection reentry point occludes the lumen of an aortic branch. In both of these instances, stent placement in the involved aortic branch vessels may restore adequate perfusion. Endovascular strategies to promote increased blood flow in the true aortic lumen include stent-graft coverage of the intimal tear (under evaluation) and intimal flap balloon fenestration to create a communication between the true and false lumens. These interventions increase true lumen blood flow and help to prevent true lumen collapse and end-organ ischemia.
 4. **Peripheral aneurysms**
 - a. **Iliac aneurysms.** Most iliac artery aneurysms occur in association with AAA, and isolated iliac artery aneurysms are rare. Unilateral common iliac aneurysms can be treated by stent-graft placement with preservation of hypogastric artery flow, if possible. When the unilateral common iliac aneurysm is located close to the iliac bifurcation, there are two options: (1) proximal embolization of the hypogastric artery to maintain pelvic collateral flow and stent-graft placement across the common iliac aneurysm, or (2) open bypass for preservation of hypogastric artery blood flow. Internal iliac (hypogastric) artery aneurysms can be treated with embolization of the aneurysm distally (and proximally if possible) with endovascular graft occlusion of the internal iliac artery orifice to assure exclusion.
 - b. **Popliteal aneurysms.** Experience with endovascular stent-graft placement across popliteal aneurysms is limited. With currently available devices, the incidence of popliteal aneurysm stent-graft thrombosis is unacceptably high. Based on the known inferior patency rates of prosthetic grafts in this position, open surgical bypass with vein graft and aneurysm ligation is to date the preferred treatment. Endovascular treatment, therefore, should be limited to patients in poor medical condition who are unable to tolerate open reconstruction. Patency of endovascular devices is more likely in patients with good runoff and large superficial femoral and distal popliteal arteries for graft attachment.
 - c. **Subclavian and axillary aneurysms.** These aneurysms can be readily excluded by endovascular means with very limited morbidity and mortality when no major branches (i.e., vertebral, carotid, internal mammary in patients with a coronary bypass graft) need to be sacrificed. The durability of these devices in the thoracic outlet has been in question due to the compression forces at the site. Nevertheless, endovascular grafts have been successfully used to exclude true and false aneurysms in this location with good short-term results.

B. Occlusive disease

1. Aortoiliac occlusive disease

- a. **Indications.** Balloon angioplasty and intravascular stent placement across aortoiliac occlusive lesions have been demonstrated to be technically feasible with excellent results. These procedures are indicated for symptomatic stenotic lesions (i.e., emboligenic lesions or lesions causing hypoperfusion) and in cases of distal bypass graft construction to improve graft inflow. Short-segment (<10 cm) concentric stenotic lesions are most favorable for angioplasty. Asymmetric occlusive lesions often cannot be effectively dilated, in which case use of intravascular stents is beneficial. **Angioplasty failure** (residual luminal diameter reduction of 30% or more, 5–10 mm Hg or greater mean blood pressure gradient across the lesion, intimal flap, and medial dissection) is an indication for arterial stenting. Other relative indications for iliac stenting include eccentric stenotic lesions, recanalized iliac occlusions, restenosis after previous angioplasty, and ulcerated emboligenic lesions.
- b. **Technique.** Intraarterial access for iliac artery angioplasty and stenting is generally through the ipsilateral femoral artery. In cases in which the occlusive lesion is too close to the access site or access is not possible, contralateral femoral artery access is also feasible. For balloon angioplasty, the angioplasty balloon is positioned across the stenosis over a guidewire under fluoroscopy. When the occlusive lesion is in the distal aorta or proximal common iliac artery close to the aortic bifurcation, angioplasty should be performed using two balloons, one in each iliac artery and both partially projecting into the distal aorta (**kissing balloons**). The rationale for this is that lesions in proximity to the aortic bifurcation typically involve the distal aorta and both common iliac arteries. Unilateral balloon dilatation may cause plaque fracture with dissection and narrowing of the contralateral vessel. Balloon inflation to 6–10 atmospheres of pressure is maintained for 30–90 seconds, after which a second inflation is performed to check for residual stenosis (as seen by a deformed balloon profile). In cases of residual stenosis, repeat dilation to 10–12 atmospheres of pressure may be attempted. If this is unsuccessful, stenting may produce a more favorable result. Finally, completion angiography is performed. Intraarterial pressures also may be recorded across the dilated region to assess for hemodynamically significant residual stenosis. Pullback pressures are also obtained after pharmacologic vasodilation (nitroglycerin or papaverine) of the distal vascular bed. A resting gradient of 5–10 mm Hg and vasodilated gradient of 10–20 mm Hg are considered hemodynamically significant. For stent placement, an introducer sheath (usually 6–9 French) is maneuvered across the stenotic lesion over a guidewire. After fluoroscopic positioning of the undeployed stent, the introducer sheath is withdrawn. Balloon-expandable stents are generally overdilated by 10–15% greater than the adjacent normal artery to ensure satisfactory stent apposition to the vessel wall. Balloon angioplasty is also commonly performed after deployment of self-expanding stents (e.g., Wallstent) to ensure good stent apposition.
- c. **Complications** include arterial wall dissection, vessel occlusion (either from thrombosis or dissection), arterial rupture, and distal embolization.
- d. **Results.** The two most common causes of early balloon angioplasty failure are elastic recoil of atherosclerotic plaque and arterial wall dissection. Cases of late failure are due to intimal hyperplasia or progressive atherosclerosis. Iliac artery balloon angioplasty 2-year patency rates of between 60% and 70% have been reported (*Surg Clin North Am* 79:575, 1999). Most reports on iliac artery stenting demonstrate between 80% and 90% 2-year patency rates (*Surg Clin North Am* 78:617, 1998). The results in general are better for common iliac artery lesions compared to external iliac artery lesions.

2. Infrainguinal occlusive disease

- a. **Indications.** Balloon angioplasty and stenting of infrainguinal occlusive lesions have inferior results compared to surgical bypass procedures to date. Nevertheless, endovascular treatment of these lesions may be appropriate in certain circumstances. In cases of limb-threatening ischemia in patients without sufficient autologous conduit or in patients at high operative risk, angioplasty and stenting may allow limb salvage despite poor long-term patency. There is no evidence to support primary stenting of femoral-popliteal stenotic lesions. Stenting may be useful in cases of unsuccessful angioplasty (residual stenosis, intimal flap, dissection) or recurrent stenosis after previous angioplasty. Balloon angioplasty has been proposed for treatment of focal (<1.5 cm) infrainguinal vein bypass graft stenoses. Although surgical revision is the treatment of choice, endovascular treatment is reasonable for certain high-risk patients in whom reoperation is deemed difficult, risky, or both.
 - b. **Results.** Results of femoral-popliteal artery angioplasty revealed 63%, 51%, and 36% patency rates at 1, 3, and 6 years, respectively (*Radiology* 183:767, 1992). The most important factor associated with early success was the type of lesion (stenosis better than occlusion); late success was most dependent on the runoff status. In addition, long-segment lesions (>10 cm) were associated with poor long-term results. Infrageniculate vessel angioplasty has been associated with particularly poor results (20% 3-year success rate). Intravascular stenting of superficial femoral artery lesions has been attempted, with reported 1-year primary patency rates of 22–81%. One-year restenosis rates of 10–43% have been reported. Two-year patency rates of 66% have been reported after balloon angioplasty of favorable vein graft stenotic lesions. Focal anastomotic stenoses with prosthetic grafts also can be balloon dilated with reasonable results. A new technique of subintimal angioplasty has been proposed, with early encouraging results, but long-term and multiple site data are lacking to date.
3. **Renal artery stenosis**
- a. **Indications.** Clinically significant renal artery stenosis typically manifests as hypertension or ischemic nephropathy. Indications for interventional therapy of clinically significant renal artery stenosis include poorly controlled hypertension or worsening renal function in medically treated patients. Balloon angioplasty is the treatment of choice for clinically significant fibromuscular dysplastic lesions of the renal artery. Angioplasty of renal artery atherosclerotic lesions has less favorable results but entails significantly less procedure-related morbidity compared to surgical bypass or endarterectomy. When indications exist for open aortic surgery (e.g., AAA repair), renal artery bypass or endarterectomy can be performed at the same time. Surgical therapy also may be preferable in cases of long-segment stenoses or occlusions or when atherosclerosis is severe and widespread (i.e., to avoid the risk of atheroembolic complications with intraarterial instrumentation). Therefore, indication for angioplasty of renal artery stenosis includes the failure of medical management of renovascular hypertension in the absence of any clear indications for open aortic surgery. Renal artery stents are placed selectively: after an unsuccessful angioplasty result (elastic recoil or occlusive dissection), for restenosis after previous angioplasty, and for the treatment of atherosclerotic ostial lesions.
 - b. **Technique.** Intravascular access to the renal artery may be obtained from the femoral, brachial, or axillary arteries (access from the upper extremity may be preferable in cases of caudally angled renal arteries). In patients at high risk for renal failure (e.g., type II diabetes, preexisting renal insufficiency), nephrotoxic contrast may be avoided and angiography can be performed using gadolinium or carbon dioxide. Technical success for renal artery angioplasty is defined as a less than 30% residual stenosis and a pressure gradient across the lesion of less than 5–10 mm Hg. In patients receiving stents, postprocedural antiplatelet agents (aspirin, ticlopidine, or clopidogrel) are routinely given.
 - c. **Results.** Primary patency rates of 87% at 10 years have been reported, with restenosis rates up to 15–20% at 1 year. Clinical improvement in renovascular hypertension after stent-angioplasty has been reported in 61% of patients (*J Vasc Interv Radiol* 10:689, 1999).
4. **Carotid occlusive disease**
- a. **Indications.** The indications for surgical treatment of carotid occlusive disease are based on several large prospective studies [e.g., Asymptomatic Carotid Atherosclerosis Study (ACAS), North American Symptomatic Carotid Endarterectomy Trial (NASCET)], and standard carotid endarterectomy is performed with minimal morbidity and mortality. Indications for endovascular treatment of these lesions, therefore, require a large-scale prospective study [e.g., Carotid Revascularization Endarterectomy versus Stent Trial (CREST), Endarterectomy versus Angioplasty in Patients with Severe Symptomatic Carotid Stenosis Study (EVA-3S)] to demonstrate that angioplasty and stenting can be performed with equivalent or less morbidity and mortality than traditional endarterectomy. The lesions most suitable for this approach may be those for which endarterectomy would be difficult and risky, for example, reoperative neck, cervical fibrosis from irradiation, and high internal carotid lesions, and in patients with severe comorbidities.
 - b. **Technique.** Vascular access to carotid occlusive lesions is usually from the femoral artery. As with endarterectomy, one of the biggest risks of endovascular treatment of these lesions is embolic stroke. Therefore, several different cerebral protection devices have been developed to minimize cerebral embolic events with intravascular instrumentation. Two types of protection devices are currently being evaluated: occlusive balloons and filter devices. With the balloon devices, a subset of patients with insufficient collateral flow do not tolerate internal carotid artery (ICA) balloon occlusion during stent deployment and require temporary shunt placement. The advantage to filter devices is the maintenance of antegrade carotid flow (and better angiographic visualization) during stent placement and deployment. Several small studies suggest that balloon- and filter-based cerebral protection devices lowered the incidence of neurologic complications with ICA stenting.
 - c. **Results.** Conversion to endarterectomy is occasionally required due to various technical problems: inability to cross the occlusive lesion, thrombus within the stent, and ICA kink. Several small studies have reported few (0–2%) periprocedural (30 days) neurologic complications after internal carotid artery angioplasty and stenting using cerebral protection devices. This compares to the 7–10% neurologic complication rates reported before the development of cerebral protection devices and lower-profile stents. A large prospective study is needed to better assess the role of carotid artery stenting.
5. **Supraaortic trunk occlusive disease**
- a. **Indications.** The indications for endovascular treatment of occlusive lesions of aortic arch branches are the same as those for open surgical treatment. For proximal subclavian artery lesions, indications are arm claudication, digital embolization, symptoms of vertebrobasilar insufficiency, and in preparation for coronary artery bypass using the internal mammary artery. For innominate and common carotid lesions, indications are stenosis of greater than 75% or referable symptoms such as transient ischemic attack, amaurosis fugax, or ipsilateral nondisabling stroke.
 - b. **Technique.** Access to the occlusive lesion is either from the femoral, brachial, or common carotid arteries. Early results with angioplasty alone were notable for poor short- and midterm patency rates. Therefore, current endovascular treatment of these lesions is with stent deployment. The majority of devices currently used are balloon-expandable stents for orificial lesions because of their increased radial force. Self-expanding, more flexible stents are used for more tortuous areas.
 - c. **Results.** Prospective results for angioplasty and stenting of supraaortic trunk occlusive lesions (innominate and subclavian arteries) were compiled in a study of 73 patients, demonstrating an 84% patency rate at 35 months (*J Vasc Surg* 28:1059, 1998). Long-term results of endovascular treatment of these lesions are not yet available. Nevertheless, endovascular treatment of these lesions is significantly less morbid than open operative approaches that require thoracotomy, sternotomy, or claviclectomy.
- C. **Vascular trauma: indications.** Experience with endovascular treatment of traumatic arterial injuries has largely been limited to arteriovenous fistulas and pseudoaneurysms in the hemodynamically stable patient. Successful stent-graft treatment of arteriovenous fistulas and pseudoaneurysms at various locations has been reported (*Ann Vasc Surg* 13:121, 1999). However, the overall experience has been limited, and besides demonstrating technical feasibility, no guidelines have been determined regarding appropriate indications for endovascular treatment. In patients with multiple injuries, endovascular treatment of severe arterial lesions is very attractive despite the limited long-term results available.

25 Venous Disease, Thromboembolism, and Lymphedema

Louis L. Nguyen and Brian G. Rubin

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Venous Anatomy

Venous anatomy is divided into the superficial, deep, and perforator components. In the lower extremity, the major superficial veins are the greater saphenous vein (located anterior to the medial malleolus and traveling medially to the fossa ovalis in the groin), the lesser saphenous vein (posterior to the lateral malleolus, coursing posterolaterally to the popliteal fossa), and the posterior arch vein, also called *Leonardo's vein* (beginning in the medial ankle and joining the greater saphenous vein below the knee). The deep veins in the leg are named according to their paired arteries. The superficial and deep systems are connected by perforating veins (direct and indirect). Most blood flows from the superficial to the deep system via the direct perforators, but indirect perforators (small superficial veins draining into muscles that connect to the deep system via intramuscular veins) are also important. Blood is propelled toward the heart by compression of the deep veins by calf muscle contractions during walking, and flow is unidirectional due to a series of one-way valves. Of surgical interest are five groups of direct medial calf perforators joining either the greater saphenous vein or the posterior arch vein to the posterior tibial vein (*J Vasc Surg* 24:800, 1996). They are named **Cockett** or **paratibial perforators**, depending on their anatomic location, which can be variable. The saphenous nerve travels close to the saphenous vein near the ankle, making it susceptible to injury during stripping of the lower saphenous vein.

Chronic Venous Insufficiency

Chronic venous disease spans a broad clinical spectrum, which includes cosmetically undesirable telangiectases, varicose veins, and venous ulceration. Advances in noninvasive imaging techniques (duplex scanning) and minimally invasive surgical techniques are being used to tailor medical and surgical therapies, resulting in marked improvement in clinical outcomes and patient satisfaction.

I. Pathophysiology

- A. **Reflux disease** from venous valvular incompetence accounts for most (>80%) chronic venous disease. Valve malfunction can be inherited or acquired through sclerosis or elongation of valve cusps; it also may result from dilation of the valve annulus despite normal valve cusps. Varicose veins may represent superficial venous insufficiency in the presence of competent deep and perforator systems, or they may be a manifestation of perforator or deep disease. Valvular disease below the knee appears to be more critical in the pathophysiology of severe disease than does deep valvular disease above the knee. The perforator veins are frequently implicated when venous ulcers exist, but any component of the venous system, either alone or in combination, may be incompetent. Therefore, all components need evaluation in the workup of chronic venous insufficiency (CVI) (*Am J Surg* 169:572, 1995).
- B. **Obstructive physiology** is a less common cause of venous pathology, with reflux often being present simultaneously.

II. Etiology. Venous disease can be congenital (although it may present later in life), primary (cause is undetermined), or secondary (postthrombotic, posttraumatic, or other). Deep vein thrombosis (DVT) accounts for most secondary causes and may be responsible for a significant number of other cases because many deep vein thrombi are asymptomatic. Other contributing factors include pregnancy, hormone therapy, and obstruction in a proximal segment (e.g., from adenopathy, arterial compression, or pregnancy).

III. Diagnosis is made by history, physical examination, and noninvasive studies.

- A. **History.** A history of any DVT or trauma should be sought, as well as any family history of varicose veins or CVI. Patients may complain of lower-extremity edema, aching, skin irritation, or varicose veins. Leg pain is described as a dull ache, worsening at the end of the day, and often relieved with exercise or elevation. In severe cases, individuals can experience acute, bursting pain with ambulation (**venous claudication**). Prolonged rest and leg elevation (20 minutes) are needed to obtain relief.
- B. **Physical examination** can reveal ankle edema, subcutaneous fibrosis, hyperpigmentation (brownish discoloration secondary to hemosiderin deposition), lipodermatosclerosis, venous eczema, and dilatation of subcutaneous veins, including telangiectases (0.1–1.0 mm), reticular veins (1–4 mm), and varicose veins (>4 mm). Ultimately, ulcers develop, typically proximal to the medial malleolus. Any signs of infection should be noted. Arterial pulses should be examined and are usually adequate.
- C. **Noninvasive studies**
 1. **Duplex scanning** (B-mode ultrasound imaging combined with Doppler frequency shift display) has become invaluable in assessing valvular incompetence and obstruction. For evaluation of patients with valvular incompetence, a modified duplex evaluation is performed. With the individual standing, cuffs are placed on the thigh, calf, and foot and inflated; then the cuffs are rapidly deflated in an attempt to create retrograde venous blood flow in segments of valvular incompetence. Competent valves generally take no more than 0.5–1.0 seconds to close. Detailed mapping of valve competence of each segment of the venous system is possible, including the common femoral, superficial femoral, greater saphenous, lesser saphenous, popliteal, posterior tibial, and perforator veins. In one report, this method was found to have a predictive value of 77% for diagnosing reflux leading to severe symptoms compared to a predictive value of 44% for descending phlebography, previously considered the gold standard (*J Vasc Surg* 16:687, 1992). Descending phlebography is limited by its inability to study valves distal to a competent proximal valve.
 2. **Continuous-wave Doppler** examination is easily performed in the office using a handheld probe. The study is helpful for screening for reflux at the saphenofemoral and saphenopopliteal junctions. Its use is limited owing to its inability to quantitate reflux and to provide precise anatomic information.
 3. **Air plethysmography** measures changes in lower-extremity volume owing to changes in the venous blood volume. Limb volume changes are measured in relation to gravity and exercise to calculate a residual volume fraction and ejection fraction, which have been shown to correlate with reflux and ulceration. The test is limited by lack of anatomic information.
 4. **Trendelenburg's test** is easy to perform but has been largely replaced by the much more accurate duplex imaging studies. First, the patient's leg is elevated to drain venous blood. An elastic tourniquet is applied at the saphenofemoral junction, and the patient then stands. Rapid filling (<30 seconds) of the saphenous system from the deep system indicates perforator valve incompetence. When the tourniquet is released, additional filling of the saphenous system occurs if the saphenofemoral valve is also incompetent.

IV. Differential diagnosis. Lower-extremity venous disease must be differentiated from arterial occlusive disease, chronic lymphedema, squamous cell carcinoma,

trauma, arteriovenous malformations, and orthostatic edema. Ischemic ulcers from arterial disease are more likely to be on the patient's foot, with discrete edges and pale bases; they are more painful than venous ulcers. Other signs or symptoms of arterial disease may be present, such as poor pulses, dependent rubor, pallor with elevation, and claudication. Lymphedema typically causes pitting edema without pigmentation and ulceration. Lymphedema is less responsive to elevation, usually requiring several days to improve.

V. Nomenclature. Based on the conclusions of an international consensus committee, a standardized nomenclature of chronic venous disease has been established (*J Vasc Surg* 21:635, 1995). Disease is classified according to a **CEAP** system: **c**linical signs, **e**tiology, **a**natomic distribution, and **p**athophysiology ([Table 25-1](#) and [Table 25-2](#)).

Table 25-1. Classification of chronic lower-extremity venous disease

Table 25-2. Clinical classification of chronic lower-extremity venous disease

VI. Nonsurgical treatment

- A. **Infected ulcers** necessitate treatment of the infection first. *Staphylococcus aureus*, *Streptococcus pyogenes*, and *Pseudomonas* species are responsible for most infections and can usually be treated with local wound care, wet-to-dry dressings, and oral antibiotics. Topical antiseptics should be avoided. Severe infections require intravenous antibiotics.
- B. **Leg elevation** can temporarily decrease edema and should be instituted when swelling occurs. This should be done before a patient is fitted for stockings or boots.
- C. **Compression therapy** is the primary treatment for CVI.
 1. **Elastic compression stockings** are fitted to provide a 30– to 40–mm Hg compression gradient, with the greatest compression at the ankle. They should be donned on arising from bed and removed at bedtime. Stockings are effective in healing ulcers but can take months to obtain good results. In a study of 113 patients treated with initial bedrest, local wound care, and elastic compression stockings, there was a 93% ulcer healing rate in a mean of 5.3 months (*Surgery* 109:575, 1991). Because stockings do not correct the abnormal venous hemodynamics, they must be worn after the ulcer has healed to prevent recurrence. Their principal drawback is patient compliance. Recurrence for compliant patients in the same study was 16% at a mean follow-up of 30 months.
 2. **Unna boots** are paste gauze compression dressings that contain zinc oxide, calamine, and glycerin and are used to help prevent further skin breakdown. They essentially provide nonelastic compression therapy. Typically, medical personnel apply the dressing, changing it once or twice a week. Healing time of ulcers is less than that of elastic compression alone, with 70% of ulcers healed by 7 versus 11 weeks (*J Am Acad Dermatol* 12:90, 1985).
 3. **Pneumatic compression devices** provide dynamic sequential compression. They are used primarily in the prevention of deep vein thrombi in hospitalized patients, but they have also been used successfully to treat venous insufficiency. In a prospective study comparing local wound care and gradient compression stockings with or without sequential pneumatic compression for 4 hours a day, there was an ulcer healing rate of 2.1% of the ulcer area per week without dynamic compression compared to 19.8% with dynamic compression (*Surgery* 108:871, 1990).
- D. **Topical medications** for venous stasis ulcers have largely been ineffective. Topical therapy is directed at absorption of wound drainage and avoiding desiccation of the wound. Antiseptics can be counterproductive. Hydrogen peroxide, povidone-iodine, acetic acid, and sodium hypochlorite are toxic to cultured fibroblasts and should be used for the shortest duration necessary to control ulcer infection.
- E. **Sclerotherapy** can be effective in treating telangiectases, reticular varicosities, and small varicose veins. If saphenous reflux is present, it should be corrected first. Contraindications include arterial occlusive disease, immobility, acute thrombophlebitis, and hypersensitivity to the drug. Sclerosing agents include 1% or 3% sodium tetradecyl sulfate, sodium morrhuate (rarely used because of anaphylactic reactions), hypertonic saline, or polidocanol (not currently approved in the United States). Varices are marked while the patient is standing. A 25-gauge needle is used to inject 0.25–0.50 mL sclerosant slowly into the lumen of larger veins. A 30-gauge needle is used for sclerosing reticular veins and telangiectases in supine patients. Compression stockings are applied at the end of the procedure and are worn for several days to 6 weeks. Patients should walk for 30 minutes after the procedure. Complications include cutaneous necrosis, hyperpigmentation, telangiectatic matting (new, fine, red telangiectases), thrombophlebitis, anaphylaxis, and allergic reaction (*Dermatol Surg* 21:19, 1995).

VII. Surgical therapy is indicated for severe disease refractory to medical treatment and for patients who cannot comply with the lifelong regimen of compression therapy. Surgical therapy includes skin grafting, vein stripping, stab avulsion, subfascial endoscopic perforator ligation, and valvuloplasty.

- A. **Preoperative evaluation** consists primarily of duplex imaging in a reliable vascular laboratory. All components of the venous system should be studied and the precise location of pathology noted. Surgical therapy is directed at the component or components found to have pathology. For valve reconstruction, ascending or descending phlebography may be useful.
- B. **Skin grafting** is occasionally used to speed healing of large ulcers. The ulcer bed should be dry and free of infection. A fenestrated split-thickness skin graft is preferred to allow for serous drainage. Bedrest is recommended until the ulcer has healed completely. Recurrence is high unless either the underlying venous pathology is corrected or conservative support (elastic compression) is initiated and maintained after skin grafting.
- C. **Stripping the greater saphenous vein** has been one of the classic treatments of varicose veins. The vein should be stripped only in the regions where pathology occurs. If the entire vein is involved, one incision is made anterior to the medial malleolus and another just below the inguinal crease. A stripper is inserted into the vein lumen at one site and run through the lumen to the other site. High ligation of the vein is done at the saphenofemoral junction, including all tributaries. Reconnection of the saphenous to the femoral system via multiple tributaries near the saphenofemoral junction is thought to be the major cause of recurrent varices. The vein is then stripped from one site to the other. Compressive bandages are applied to reduce hematoma formation, and compression stockings are worn for several weeks. Complications include ecchymosis, DVT, and saphenous nerve injury. Stripping only the thigh portion is probably the most important aspect of the procedure (*Lancet* 348:210, 1996), and it eliminates much of the risk to the saphenous nerve because it is closely associated with the saphenous vein from the knee to the ankle. This also allows the portion of the vein below the knee to be used for arterial bypass if needed in the future. The same technique can be applied to the lesser saphenous vein.
- D. The **stab avulsion technique** allows a cosmetically acceptable surgical approach to varicose veins. Preoperatively, the patient's varicose veins are carefully marked with indelible ink while the patient is standing. Some authors consider this the most important technical step in the procedure (*Am J Surg* 172:278, 1996). An incision (2–3 mm) is made next to the markings. The vein is pulled out of the incision with a small vein hook, and the two arms of the vein are pulled taut and avulsed. This can be repeated many times to remove large clusters of veins. The small incisions can be closed with Steri-Strips. The patient's leg is covered with compression stockings for several days to weeks. This technique is often used in conjunction with saphenous stripping to provide optimal results. Alternatively, the greater saphenous vein can be removed by sequential avulsion instead of stripping. This has been shown prospectively to decrease pain and bruising (*Br J Surg* 83:1559, 1996).
- E. For most severe CVI, **more extensive surgery** is required, usually directed at correction of saphenous reflux and ligation of incompetent medial calf perforators. Even in the presence of combined superficial and deep incompetence, treatment of only the superficial and perforator incompetence can significantly improve clinical symptoms and hemodynamics (*J Vasc Surg* 24:711, 1996) by disrupting continued transmission of high deep venous pressure to the skin. Traditionally, treatment of perforator reflux has been accomplished via an **open surgical subfascial ligation** (the Linton procedure), which requires an incision through diseased skin and extensive subcutaneous dissection. Wound complications have limited the acceptance of this procedure. **Subfascial endoscopic perforator surgery** is becoming increasingly popular because of its decreased morbidity. This procedure is performed by making small port incisions in unaffected skin in the calf and fascia of the posterior superficial compartment. Various types of endoscopes (laparoscopic, arthroplastic, or bronchoscopic) may be used for visualization. Carbon dioxide insufflation in the subfascial space may or may not be used. A balloon expander can expand the subfascial space to improve visualization. Typically, two to six perforators are identified and ligated. Most patients are discharged within 24 hours of surgery. Preliminary results indicate an ulcer-healing rate of 88% for a mean follow-up of 5 months (*J Vasc Surg* 25:94, 1997). Pain, edema, pigmentation and lipodermatosclerosis have all been

shown to improve. Complications include wound infection, superficial thrombophlebitis, cellulitis, and saphenous neuralgia, but the overall morbidity is improved over the open technique. A newer technique of “mini incision” ligation of perforators has had good results with fewer wound complications. In this method, perforators are first identified in the vascular laboratory and their location marked on the skin. Small purse-string sutures are then placed at these marks to ligate the vessels (*J Vasc Surg* 25:437, 1997).

- F. **Direct venous valve reconstruction** can be performed to restore competence to floppy or redundant valves. Generally, this excludes patients with venous reflux secondary to deep venous thrombosis because the inflammatory thrombotic process tends to fibrose and shorten the valve cusps. Valvuloplasty can be done using an open or closed technique (*J Vasc Surg* 16:694, 1992). For the open procedure, a venotomy is made above the valve, and the cusps are sutured until competence is achieved. Alternatively, an angioscope can be inserted through a tributary, and repair of the valve can be viewed directly without a venotomy. To correct an incompetent superficial femoral vein, transfer of a valved vein segment is performed by end-to-end anastomosis either to the proximal saphenous vein or to the deep femoral vein, thus providing a competent proximal valve. Autotransplantation of a segment of axillary or brachial vein into the proximal popliteal vein has also been used with variable success.

Venous Thromboembolism

I. Epidemiology. Venous thromboembolism, which includes DVT and pulmonary embolism (PE), is a common cause of death. The true incidence of DVT is difficult to obtain, because its clinical diagnosis can be inaccurate and DVT often occurs in the setting of other critical illnesses. One population-based study in Worcester, Massachusetts, reported 48 cases of DVT per 100,000 (*Arch Intern Med* 151:5, 1991). These figures were based on hospital discharge diagnoses and are likely to be underrepresentative of the true incidence. Extrapolation of the Worcester data results in approximately 260,000 cases of DVT diagnosed in acute-care hospitals annually.

II. Pathophysiology. DVT starts as a platelet nidus, usually in the venous valves of the calf. The thrombogenic nature of the nidus activates the clotting cascade, leading to a thrombus that grows by accumulating more platelets and fibrin. The fibrinolytic system is also activated, and thrombogenesis and thrombolysis compete for dominance. The thrombus grows if thrombogenesis predominates. A thrombus can detach from the endothelium and migrate into the pulmonary system, becoming a PE. A thrombus can also organize and grow into the endothelium, resulting in venous valve incompetency and phlebitis. Thrombi localized to the calf have a decreased tendency to embolize as compared to thrombi that extend to the thigh veins (*Am Rev Respir Dis* 141:1, 1990). Approximately 20% of cases of calf DVT propagate to the thigh, and 50% of thigh or proximal DVT embolize. Significant PE results in increased right ventricular afterload, which can further lead to right ventricular dysfunction, decreased cardiac output, or arrhythmias. Remodeling or fibrinolytic dissolution of the embolus also occurs, although few patients have complete normalization of pulmonary arterial pressures. Most patients survive the first hour after a PE, although many of these occurrences are not detected. If not diagnosed, recurrent episodes of PE pose a greater risk of mortality. Among patients correctly diagnosed and treated, the recurrence rate of clinically detected PE is 8.3%, and the early fatality rate is approximately 2.5% (*N Engl J Med* 326:19, 1992). PE usually recurs within 1 week, and the early fatalities occur within 2 weeks of the initial episode. One year after PE, approximately 24% of patients die from underlying cardiovascular disease, pulmonary disease, or malignancy.

III. Risk factors for venous thromboembolism

- A. **Malignancy.** Trousseau was the first to suggest an association between malignancy and a hypercoagulable state when he observed episodic migratory thrombophlebitis in his cancer patients. Several pathogenic mechanisms have been described for this association. Tumor cell activation of the clotting cascade can occur directly through interactions with factors VIIa, X, and tissue factor (TF). Indirect clotting activation can occur through stimulation of mononuclear cells to produce TF or factor X activators and macrophages to produce TF activators.
- B. **Endothelial injury.** Another mechanism for the association of malignancy and thrombus formation is endothelial injury. Adhesion of tumor cells to endothelium can lead to disruption of endothelial intracellular junctions and expose the highly thrombogenic subendothelial surface. Chemotherapeutic drugs, such as bleomycin, carmustine, vincristine, and doxorubicin (Adriamycin), can also cause vascular endothelial cell damage.
- C. **Reactive thrombocytosis** can occur in patients with malignancy, especially those with advanced disease of the lung, colon, stomach, or breast. Thrombocytosis may be caused by spontaneous clumping of platelets or increased levels of thrombopoietin, a glycoprotein that regulates the maturation of megakaryocytes.
- D. **Venous stasis** can be caused by immobility, venous obstruction, increased venous pressure, and increased blood viscosity. Venous stasis promotes thrombus formation by reducing clearance of activated coagulation factors and by causing endothelial hypoxia, leading to reduced levels of surface-bound thrombomodulin and increased expression of TF. Two very common causes of immobility leading to DVT formation are surgery and critical illness. Major chest surgery, abdominal/pelvic surgery, and lower-extremity surgery have all been associated with increased risk of DVT development. Similarly, a prolonged nonambulatory state, such as fracture of the hip, pelvis, or leg; multisystem trauma; neurologic injury; or other critical injury requiring bedrest can increase DVT risk.
- E. The use of **oral contraceptives (OCPs)** and **estrogen hormone replacement therapy** has been linked to increased risk of venous thrombus formation. Many studies have found an odds ratio of 3.0–5.0 for risk of DVT in patients taking OCPs compared to non-OCP–using patients. This increased risk is still found with patients using third-generation OCPs containing new progestins. In a matched-control study from Oxford, England, the authors found a 3.7-fold increase in the risk of venous thrombus in older women undergoing hormone replacement therapy. The mechanism causing the increased risk is still unknown, especially because the estrogens used in hormone replacement therapy are different from those used in OCPs.
- F. **Hypercoagulable states** can also lead to DVT formation. Primary hypercoagulable states are inherited conditions that can lead to abnormal endothelial cell thromboregulation (e.g., decreased thrombomodulin-dependent activation of protein C, impaired heparin binding of antithrombin III, or downregulation of membrane-associated plasmin production) or decreased thrombogenic inhibitors (e.g., antithrombin III, protein C, and protein S). Secondary hypercoagulable states are conditions in which endothelial activation by cytokines leads to an inflammatory thrombogenic vessel wall. Antiphospholipid syndrome, heparin-induced thrombopathy, myeloproliferative syndromes, and cancer are among the more common syndromes leading to secondary hypercoagulable states. Some chemotherapy agents, such as cyclophosphamide, methotrexate, and 5-fluorouracil, can cause a decrease in plasma levels of proteins C and S.

IV. Diagnosis of venous thromboembolism. Approximately 75% of patients who present with suspected DVT or PE turn out not to have these conditions. Nevertheless, objective studies are indicated when either is high, because clinical diagnosis alone is inaccurate and undetected/untreated DVTs or PEs can have fatal outcomes.

- A. The **initial evaluation** for suspected venous thromboembolism in a patient begins with an assessment of the risk factors mentioned earlier in this chapter. The clinical presentation of a patient with DVT includes pain of the extremity, increased circumference of that extremity with respect to the contralateral extremity, dilation of superficial veins of the suspected extremity only, and calf pain on dorsiflexion of the ankle. A more severe presentation of DVT is phlegmasia cerulea dolens, in which limb pain and swelling are accompanied by cyanosis, a sign of arterial ischemia.
- B. **PE** can have many clinical presentations, most of which are nonspecific. Tachycardia, fever, hypoxia, cardiac arrhythmia, myocardial ischemia, and cardiogenic shock are the more common presentations. Many presentations of venous thromboembolism occur without clear clinical signs.
- C. For **suspected DVT**, compression ultrasonography of the femoral, popliteal, and calf trifurcation veins is highly sensitive (>90%) in detecting thrombosis of the proximal veins (femoral and popliteal) but less sensitive (50%) in detecting calf vein thrombosis. Ultrasonography is the preferred diagnostic modality because it is less invasive than the reference standard of venography and more sensitive than impedance plethysmography. Approximately 2% of patients with initial normal ultrasound results have positive results on repeat tests performed 7 days later. This delayed detection rate is attributed to extension of calf vein thrombi or small, nonocclusive proximal vein thrombi.
- D. For **suspected PE**, radionuclide lung imaging is the first step. If the results are “low probability” of PE, the diagnosis of PE is excluded, whereas a “high probability” result strongly suggests the presence of PE. However, more than 50% of patients have “intermediate probability” results. Because approximately 25% of these patients have PE, further evaluation or initiation of empiric treatment must be considered.
- E. Further evaluation can include **ultrasonography or impedance plethysmography**, although negative results from these tests do not truly rule out PE.
- F. **Pulmonary angiography**, the reference test, is reserved for patients whose diagnosis is still uncertain.
- G. **Contrast-enhanced spiral computed tomography (CT)** is a new technique that has comparable sensitivity (70–90%) for detecting PE when compared to angiography. Spiral CT is gradually being favored over angiography because it is less invasive and less expensive.

V. Prevention and treatment of venous thromboembolism. The prevention of venous thromboembolism depends on knowledge of risk factors in individual patients. As detailed previously in this chapter, prolonged immobility, cancer, estrogen use, and hypercoagulable states are well-known risk factors. Other risk factors include age greater than 40, prior DVT, obesity, varicose veins, congestive heart failure, myocardial infarction, and stroke. The rationale for prophylaxis of venous thromboembolism is based on the silent nature of the disease. DVT and PE manifest few clinical symptoms, and the diagnosis is sometimes uncertain. Although treatment of detected venous thromboembolism is effective, many patients who die from PE do so in the first 30 minutes of the event, too soon for anticoagulation to have full effect. In the absence of prophylaxis, the frequency of fatal postoperative PE ranges from 0.1% to 0.8% in patients who are undergoing elective general surgery, 0.3% to 1.7% in patients undergoing elective hip surgery, and 4% to 7% in patients undergoing emergency hip surgery (*Haemostasis and thrombosis: basic*

principles and clinical practice, 3rd ed. Philadelphia: JB Lippincott Co., 1994). Prophylaxis is achieved by either altering the blood coagulation state, preventing venous stasis, or preventing embolization.

- A. **Low-dose unfractionated heparin** is given subcutaneously at 5,000 units 2 hours before surgery and every 8 or 12 hours postoperatively. Low-dose heparin reduces the risk of venous thromboembolism by 50–70% (*N Engl J Med* 318:18, 1988) and does not require laboratory monitoring. Because of the potential for minor bleeding, it should not be used for patients undergoing cerebral, ocular, or spinal surgery. **Graduated compression stockings** are effective in preventing DVT formation by reducing venous stasis. However, this efficacy has been shown for calf DVTs, whereas insufficient data exist to conclude the same for prevention of proximal DVTs or PEs. In surgery patients, the combination of graduated compression stockings and low-dose heparin is significantly more effective than low-dose heparin alone (*Br J Surg* 72:7, 1985). Graduated compression stockings are relatively inexpensive and should be considered for all high-risk patients, even when other forms of prophylaxis are used.
- B. **Intermittent pneumatic compression of the extremities** enhances blood flow in the deep veins and increases blood fibrinolytic activity. For patients with significant bleeding risk with anticoagulation, pneumatic compression is an effective alternative. Compression devices should not be placed on an extremity with a known DVT. In the case of known bilateral lower-extremity DVT, the compression device can be placed on the upper extremity. Pedal compression devices are also effective in patients whose body habitus does not allow conventionally sized devices to fit around their thighs or calves.
- C. **Low-molecular-weight heparins (LMWHs)** have several advantages over unfractionated heparin. They have longer half-lives, the dose response is more predictable, and in laboratory animals, they cause fewer bleeding complications with equivalent anticoagulation effects. In large randomized trials of patients with DVT, outpatient treatment with LMWH was as safe and effective as inpatient treatment with intravenous unfractionated heparin. LMWH seems to be only slightly better than low-dose heparin for prophylaxis against DVT formation because LMWH causes fewer wound hematomas and is more convenient to dose.
- D. **Caval interruption with intracaval filters** has been available since the 1960s as a method of preventing PE. However, unfractionated heparin followed by warfarin therapy for 3 months is 95% effective in preventing PE for patients with known DVT. Thus, for these patients, filter placement was restricted for those who had contraindications to anticoagulation or had failure of anticoagulation therapy. With the development of percutaneous methods of filter placement (and low complication rates), indications for filter use have widened. Recent estimates from industry manufacturers show that 30,000–40,000 filters are inserted in the United States annually.
- E. The **choice of prophylaxis method** depends on the risk of venous thromboembolism compared to the risk of anticoagulation. For low-risk patients, such as those younger than 40 years old undergoing uncomplicated surgery, resulting in minimal immobility, no specific prophylaxis other than early ambulation is necessary. In moderate-risk patients, such as those who are older than 40, are undergoing major operations, and do not otherwise have venous thromboembolism risk factors, elastic stocking and low-dose unfractionated heparin can be used. Intermittent pneumatic compression is a good alternative when bleeding risk is high. In high-risk patients, such as those with prior DVT, a combination of pharmacologic and mechanical methods can be used. In very-high-risk patients, such as those with current DVT, perioperative anticoagulation with warfarin, intravenous unfractionated heparin, or subcutaneous LMWH can be used with modulation for bleeding risk.

Lymphedema

I. Pathophysiology

- A. **Primary lymphedema** is the result of congenital aplasia, hypoplasia, or hyperplasia of lymphatic vessels and nodes that causes the accumulation of a protein-rich fluid in the interstitial space. Swelling of the patient's leg initially produces pitting edema, which progresses to a nonpitting form and may lead to dermal fibrosis and disfigurement. Primary lymphedema is classified according to age at presentation, as congenital (present at birth), praecox (early in life), or tarda (late in life). Congenital lymphedema (10–15% of cases) can be hereditary (Milroy's disease) or nonhereditary. Praecox lymphedema (70–80% of cases) is seen in female patients 80–90% of the time and presents during adolescence. Lymphedema tarda (10–15% of cases) is seen equally in men and women and presents after the third or fourth decade.
- B. **Secondary lymphedema** results from impaired lymphatic drainage secondary to a known cause. Surgical or traumatic interruption of lymphatic vessels (often from an axillary or groin lymph node dissection), carcinoma, infection, venous thrombosis, and radiation are causes of secondary lymphedema.

II. Lymphedema is diagnosed by history, physical examination, and exclusion of other causes of a swollen extremity. Imaging studies, especially lymphoscintigraphy, can aid in the diagnosis. Typically, however, imaging studies are directed at ruling out venous thrombosis or extrinsic venous compression as the cause of leg swelling.

A. Clinical presentation

1. **Symptoms.** Early lymphedema is characterized by unilateral or bilateral arm or pedal swelling that resolves overnight. With disease progression, the swelling increases and extends up the extremity, producing discomfort and thickened skin. With more advanced disease, swelling is not relieved overnight. Significant pain is unusual. With secondary lymphedema, symptoms related to the principal disease are present. Patients commonly present with repeated episodes of cellulitis secondary to high interstitial protein content.
2. **Physical examination** reveals edema of the affected extremity. When the lower extremity is involved, the toes are often spared. With advanced disease, the extremity becomes tense with nonpitting edema. Dermal fibrosis results in skin thickening, hair loss, and generalized keratosis.

B. Imaging studies

1. **Lymphoscintigraphy** is the injection of radiolabeled (technetium-99m) colloid into the web space between the patient's second and third toes or fingers. The patient's limb is exercised periodically, and images are taken of the involved extremity and the whole body. Lymphedema is seen as an abnormal accumulation of tracer or as slow tracer clearance along with the presence of collaterals. For the diagnosis of lymphedema, the study has a sensitivity and specificity of 92% and 100%, respectively (*J Vasc Surg* 9:683, 1989).
2. **CT and MR scan** are able to exclude any mass obstructing the lymphatic system. MR scan has been able to differentiate lymphedema from chronic venous edema and lipedema (excessive subcutaneous fat and fluid).
3. **Lymphangiography** involves catheter placement and injection of radiopaque dye directly into lymphatic channels; it has largely been replaced by lymphoscintigraphy and CT. A decreased total number of lymphatic channels and structural abnormalities can be seen. Lymphangiography can demonstrate the site of a lymphatic leak in postsurgical or traumatic situations. Complications include lymphangitis and hypersensitivity reaction to the dye.

III. Differential diagnosis includes all other causes for a swollen extremity, including trauma, infection, arterial disease, venous disease, lipedema, neoplasm, radiation effects, and other systemic diseases, such as right ventricular failure, myxedema, nephrosis, nephritis, and protein deficiency. These causes must be excluded before invasive study.

IV. Treatment

- A. **Medical management** is limited by the physiologic and anatomic nature of the disease. The use of diuretics to remove fluid is not effective because of the high interstitial protein concentration. Development of fibrosis and irreversible changes in the subcutaneous tissue further limit options. Objectives of conservative treatment are to control edema, maintain healthy skin, and avoid cellulitis and lymphangitis.
 1. **Combination of physical therapies (CPT)** is the primary approach recommended in a consensus document by the International Society of Lymphology Executive Committee (*Lymphology* 28:113, 1995). CPT involves gentle manual manipulation of tissues to direct lymph flow, physical therapy exercises, and compression bandages. This is followed by wearing custom-made compression garments. In a study of 119 patients with 3-year follow-up, CPT reduced lymphedema by 63% (*Oncology* 11:99, 1997).
 2. **Sequential pneumatic compression** has been shown to improve lymphedema. Several designs have been used with various degrees of success. Elastic stockings or sleeves should be fitted and worn afterward to maintain results. Extremity elevation may also help.
 3. **Skin care and good hygiene** are important. Topical hydrocortisone cream may be needed for eczema.
 4. **Benzopyrones** (such as warfarin) have been effective in reducing lymphedema due to filariasis. Their action is believed to be from enhanced macrophage activity and extralymphatic absorption of interstitial proteins.
 5. **Cellulitis and lymphangitis** should be suspected with the sudden onset of pain, swelling, or erythema of the leg. Intravenous antibiotics should be initiated to cover staphylococci and beta-hemolytic streptococci. Broad-spectrum penicillins, cephalosporins, or vancomycin usually are adequate. Limb elevation and immobilization should be initiated, and warm compresses can be used for symptomatic relief. Topical antifungal cream may be needed for chronic infections.
- B. **Surgical options** include excision with skin grafting, closure of disrupted lymphatic channels, omental transposition, lymphatic transposition, and microsurgical lymphovenous anastomosis. Only 10% of patients with lymphedema are surgical candidates, and surgery is directed at reducing limb size. Indications for operation are related to function because cosmetic deformities persist postoperatively. Results are best when surgery is performed for severely impaired movement and recurrent cellulitis.

1. **Total subcutaneous excision** is performed for extensive swelling and skin changes. Circumferential excision of the skin and subcutaneous tissue from the tibial tuberosity to the malleoli is performed. The defect is closed with a split- or full-thickness skin graft from the resected specimen or a split-thickness skin graft from an uninvolved site. Good functional results can be expected in 60–100% of patients (*Plast Reconstr Surg* 60:589, 1977). Recurrent lymphedema and hyperpigmentation occur more frequently when split-thickness skin grafts are used. Lymphatic reconstruction includes direct (lymphovenous bypass, lymphatic grafting) and indirect (mesenteric bridge, omental flap) procedures.
2. **Lymphovenous anastomoses** bypass the obstructed lymphatic system in patients with chronic lymphedema. With improved microvascular techniques, patency rates of 50–70% can be expected months after surgery (*J Vasc Surg* 4:148, 1986). Lymphatic grafting is performed for upper-extremity or unilateral lower-extremity lymphedema. Good results have been reported in 80% of patients (*Plast Reconstr Surg* 85:64, 1990). A mesenteric bridge is formed by suturing a segment of mucosa-stripped ileum with intact blood supply to transected distal iliac or inguinal nodes. An omental flap placed in a swollen limb is believed to improve lymphatic drainage through spontaneous lympholymphatic anastomoses. Due to their complexity and associated complications, indirect procedures are not widely used.

Tumors and Genetics**I. Breast cancer****II. Colorectal cancer****III. Endocrine tumors****IV. Sarcomas****V. Melanoma****VI. Genetic testing and counseling**

Tumors and Genetics

I. Breast cancer. Inherited breast cancer accounts for approximately 5–10% of all breast cancers, amounting to 9,000–18,000 cases per year in the United States.

- A. **BRCA1 and BRCA2 genes** account for 45% and 35% of inherited breast cancer, respectively. The BRCA1 gene was cloned from the long arm of chromosome 17 and spans approximately 100,000 nucleic acids, with more than 1,600 different mutations reported. Over 100 different mutations have been reported for BRCA2, an even larger gene. Based on high-risk families, a patient has an 87% lifetime risk of breast cancer with a mutated BRCA1 gene and an 80% risk with a mutated BRCA2 gene. In the Ashkenazi Jewish population, BRCA1 and BRCA2 mutations confer a risk of only 56% by age 70. Different mutations may have varying risks of breast cancer associated with them. Ovarian cancer in these patients appears to have a better prognosis than sporadic ovarian cancer. Men who are BRCA2 mutation carriers have a higher risk of breast cancer, although not as high as women.
1. **Diagnosis and screening.** A family history should be taken in any individual with breast cancer and should include paternal as well as maternal information. Commercial tests to detect mutations in the BRCA1 and BRCA2 genes are available for suspected individuals at a cost of approximately \$2,000. Genetic testing may identify a mutation of unknown significance. Genetic counseling is essential before any test is administered.
 2. **Treatment** for BRCA mutation carriers includes close surveillance, chemoprevention, and prophylactic surgery. Treatment options should be tailored to individuals based on their desires and the perceived risk. Tamoxifen has been shown to reduce the risk of cancer in the contralateral breast after an initial diagnosis of cancer. A review of high-risk women found a 90% reduction in breast cancer after bilateral prophylactic mastectomies (*N Engl J Med* 340:77, 1999). A prospective study found a significant reduction in breast cancer in women with BRCA1 or BRCA2 mutations who elected to have prophylactic mastectomies (*N Engl J Med* 345:159, 2001). If mastectomies are performed, the patient needs to continue to have close follow-up because breast tissue remains.
- B. **Li-Fraumeni syndrome (LFS)** is a rare autosomal dominant disorder, characterized by an inherited predisposition to breast cancer and at least five childhood cancers: soft-tissue sarcoma, osteosarcoma, leukemia, brain tumor, and adrenocortical carcinoma. Other tumors have been reported as well. Breast cancer is most common, with almost 90% of carriers developing it by age 50. Approximately 50% of carriers develop some type of invasive cancer by age 30. Patients also have an increased sensitivity to radiation. Approximately 50% of LFS families have been found to have germline mutations in the p53 tumor-suppressor gene (*Am J Hum Genet* 56:608, 1995). Because a significant number of LFS kindreds do not have a p53 germline mutation, genetic heterogeneity is likely. The protein product of the gene functions as a transcription factor, preventing DNA synthesis when damage has occurred to the cell's DNA.
- C. **Cowden's disease (multiple hamartoma syndrome)** is an autosomal dominant syndrome characterized by cutaneous facial lesions, seen in almost all patients. The spectrum of disease includes multiple facial trichilemmomas, oral papillomas, "cobblestoning" of the tongue, acral keratoses, bilateral breast cancer, gastrointestinal polyposis, and thyroid tumors. Lipomas, hemangiomas, macrocephaly, and brain tumors have also been reported. Breast cancer develops in 30–50% of patients by age 50. Germline mutations in the PTEN gene are responsible for the syndrome (*Nat Genet* 15:307, 1997). The protein product is a tyrosine phosphatase.
- D. **Ataxia-telangiectasia** is an autosomal recessive disease with a varied phenotype, including progressive cerebellar ataxia, oculocutaneous telangiectasias, progeric skin changes, immune dysfunction, and increased cancer susceptibility. The hallmark of ataxia-telangiectasia is cerebellar ataxia, often seen at a young age. Most patients are unable to walk by age 10. The responsible gene (ATM) is required for transcription of p53 in response to radiation damage. These individuals are extremely sensitive to ionizing radiation. Carriers are prone to a variety of cancers, particularly leukemia and lymphoma, but also breast, pancreatic, stomach, bladder, and ovarian cancer. Heterozygote carriers also are reported to have an increased risk of breast cancer, but this is controversial.

II. Colorectal cancer. Familial cancers may account for up to 15% of the 150,000 new cases of colorectal cancer diagnosed annually in the United States.

- A. **Familial adenomatous polyposis (FAP)** is an inherited autosomal dominant syndrome characterized by florid colonic polyposis and caused by mutations in the tumor-suppressor gene APC. Virtually 100% of affected individuals develop colorectal cancer if not treated. Carriers typically develop adenomatous polyps that progress to carpet the large intestine during the second or third decades of life. The polyps are indistinguishable from sporadic polyps, but because of their enormous numbers and the early age at which they develop, the likelihood of one of them progressing to malignancy is close to 100%. The median age of cancer diagnosis for untreated individuals is 39 years, almost 30 years earlier than for sporadic colorectal cancer. Affected individuals are also prone to development of duodenal, gastric, and ileal polyps and are at risk for developing duodenal, stomach, periampullary, thyroid, and other cancers as well.
1. The **APC gene** was positionally cloned in 1991 on the long arm of chromosome 5. The gene encodes a 300-kd protein, which can bind microtubules and beta-catenin. The location of a mutation within the gene can produce different phenotypes. Congenital hypertrophy of the retinal pigment epithelium, once thought to be a signature of the disease, occurs when the mutation resides between codons 463 and 1387. There are several phenotypic variations of FAP that were previously thought to be distinct syndromes. **Gardner's syndrome**, associated with mutations in codons 1403–1578, is characterized by desmoid tumors, osteomas of the mandible or skull, and sebaceous cysts. **Hereditary flat adenoma syndrome** is characterized by ordinary adenomas as well as flat adenomas. **Turcot's syndrome** is associated with malignant central nervous system tumors, gastrointestinal polyposis, and colorectal cancer. Somatic mutations in the APC gene also occur in more than 80% of sporadic cases of colorectal cancer as well as in early adenomas.
 2. **Screening.** Any suspected individuals whose families have never been tested should have their entire APC gene sequenced to detect a mutation. The protein truncation test detects more than 80% of mutations but should only be performed as a screening test once a mutation that causes truncation has been established in the family. Screening for affected children should begin in their preteen age years via lower endoscopy and should continue annually. Affected individuals should undergo lifelong surveillance of their upper gastrointestinal tract for a second malignancy.
 3. **Surgery** is performed when polyposis is detected, usually in the late teen years or early twenties. Two options exist to remove the colon: either a total proctocolectomy with an ileal pouch–anal anastomosis or a colectomy with an ileorectal anastomosis. This anastomosis is usually better tolerated, but by leaving rectal mucosa, a significant risk for developing rectal cancer exists. Close surveillance of this mucosa is necessary. The rate of rectal cancer after ileorectal anastomosis has been found to range from 12% to 20% at 20 years. Multiple factors should be considered when deciding on the appropriate surgical procedure, including extent of rectal disease, location of any cancer, sphincter function, extracolonic disease, surgeon experience, and patient follow-up.
 4. **Upper gastrointestinal cancers**, usually duodenal tumors, pose the greatest risk of mortality once the risk of colorectal cancer is eliminated. Upper endoscopic surveillance should be performed every 3 years. Polypectomy, if possible, is thought to be adequate treatment for most polyps, followed by annual screening. Currently, prophylactic resection is not recommended.
 5. **Desmoids** appear as a phenotypic characteristic in 10–20% of FAP patients. Although not malignant, they can be locally aggressive and a significant cause of mortality. Desmoids often arise in surgical scars or small-bowel mesentery after abdominal surgery. Diffuse fibrosis may lead to ureteral, vascular, or gastrointestinal obstruction. Further surgical resection may not be possible, and, when initially successful, desmoids often recur.
- B. **Hereditary nonpolyposis colorectal cancer syndrome (HNPCC)** is an autosomal dominant disease caused by one of at least five DNA mismatch repair genes and accounts for about 5% of all colorectal cancers. The lifetime risk of colorectal cancer is approximately 70–80%, with a median age of cancer development of 44 years. These patients do not develop florid polyposis, but the polyps that do develop have an accelerated rate of tumor progression. The cancers tend to be right sided (70% proximal to the splenic flexure). In addition, synchronous and metachronous colorectal cancers are common. These patients do better, stage for stage, than their sporadic counterparts. Extracolonic malignancies are also seen, especially endometrial cancer, as well as ovarian, small-bowel, stomach, pancreas, urologic tract, bladder, and, possibly, breast cancer.
1. **The Amsterdam criteria clinically define HNPCC.** These consist of (1) a family with at least three relatives having proven colorectal cancer, in which one individual is a primary relative of the other two; (2) at least two generations are affected; and (3) one individual was diagnosed before age 50.
 2. **MSH2, MLH1, MSH6, PMS1, and PMS2** are the DNA mismatch repair genes responsible for most cases of HNPCC. MSH2 and MLH1 account for two-thirds of HNPCC. If these genes are inactivated, the cell cannot repair mistakes made by DNA polymerase during DNA replication, which leads to genome wide

mutations. Microsatellite instability (additions or deletions in the number of dinucleotide DNA repeats) characterizes the DNA of these individuals.

3. **Genetic testing** for MSH2 and MLH1 is done at a few centers, largely within research protocols. High-risk individuals should be screened with annual colonoscopy beginning at age 21. Women should undergo endometrial aspiration curettage annually beginning at age 30. Surveillance for ovarian cancer is limited, but transvaginal ultrasound and CA-125 should be considered.
 4. **Treatment** for HNPCC kindreds has not been well defined. If a germline mutation is detected, prophylactic surgery should be considered. The same general principles of treatment exist as for FAP, but a colectomy with an ileorectal anastomosis may be a more reasonable alternative, because these tumors have a proximal predilection. Alternatively, nonoperative management with screening colonoscopy every 1–2 years in known gene carriers is a reasonable option. Prophylactic total abdominal hysterectomy and bilateral salpingo-oophorectomy is an option for women who have completed their families.
- C. **Muir-Torre syndrome** is an autosomal dominant hereditary cancer syndrome characterized by the presence of at least one sebaceous gland tumor and a visceral cancer. The sebaceous gland tumors often appear as yellow facial papules and are a hallmark of this disease. The most common internal malignancy is colorectal cancer, but other tumors occur, especially genitourinary. Clinical similarities with HNPCC include a tendency for proximal colon cancers and for better survival than for sporadic cancers. Microsatellite instability is found in tumors in about half of affected individuals. Germline mutations have been found in the MSH2 and the MLH1 genes in different kindreds.
- D. **Peutz-Jeghers syndrome** is a rare autosomal dominant disease, characterized by gastrointestinal hamartomatous polyposis and mucocutaneous pigmentation. The melanin pigmentations occur on the lips, oral mucosa, hands, feet, and perianal and umbilical areas. The polyps have an uncertain malignant potential but can cause obstruction, intussusception, and bleeding. Malignancies of the gastrointestinal tract, including pancreatic cancer, occur with increased frequency. There also appears to be increased risk of extra gastrointestinal malignancies. These patients should have a baseline upper endoscopy and colonoscopy at approximately the age of 20, with frequent surveillance thereafter. Some have advocated a subtotal colectomy if the polyps are too numerous to follow. The STK11 gene has recently been linked to the syndrome.
- E. **Familial juvenile polyposis** is another rare autosomal dominant disease characterized by hamartomatous polyposis. The polyps, usually numbering 50–200, primarily occur in the colon and rectum. The lifetime risk for developing colorectal cancer is estimated to be 25–50%, mostly at an early age. There is also an increased risk for upper gastrointestinal tumors. Associated congenital abnormalities include cerebral and pulmonary arteriovenous malformations, cardiac anomalies, polydactyly, malrotation, and cranial malformations. Colonoscopic surveillance should begin by age 12. The SMAD4 gene has recently been linked to some of the kindreds with this syndrome (*Science* 15:1086,1998).

III. Endocrine tumors

- A. **Multiple endocrine neoplasia type 1 (MEN-1)** is an autosomal dominant syndrome characterized by tumors of the parathyroid glands, pancreatic islet cells, and pituitary gland. Hyperparathyroidism occurs in virtually all patients. Clinical evidence of pancreatic islet cell and pituitary tumors develops in 50% and 25% of patients, respectively. Lipomas, thymic or bronchial carcinoid tumors, and tumors of the thyroid, adrenal cortex, and central nervous system (CNS) may also develop. The gene responsible for MEN-1, MENIN, is located on chromosome 11q13 and appears to act through transcription factors (*Science* 276:404, 1997). If genetic testing is not available, screening of family members should begin in their early teens, including yearly determinations of plasma calcium, glucose, gastrin, fasting insulin, vasoactive intestinal polypeptide, pancreatic polypeptide, prolactin, growth hormone, and beta-human gonadotropin hormone.
1. **Hyperparathyroidism** consists of generalized (4-gland) parathyroid enlargement. Surgery should consist of 3 1/2-gland parathyroidectomy or a total parathyroidectomy with autotransplantation of parathyroid tissue to the forearm.
 2. **Pituitary tumors** most commonly are benign prolactin-producing adenomas, but growth-hormone, adrenocorticotrophic hormone-producing, and nonfunctioning tumors are also seen. Patients may present with headache, diplopia, or symptoms referable to hormone overproduction. Bromocriptine inhibits prolactin production and may reduce tumor bulk and obviate the need for surgical intervention. Transsphenoidal hypophysectomy may be necessary if medical treatment fails.
 3. **Pancreatic islet cell tumors** pose the most difficult clinical challenge and account for most of the morbidity and mortality of the syndrome. Gastrinomas are most common, but vasoactive intestinal polypeptide-secreting tumors, insulinomas, glucagonomas, and somatostatinomas are also encountered. The pancreas is usually diffusely involved with islet cell hyperplasia and multifocal tumors. Tumors may be found in the proximal duodenum and peripancreatic areas (gastrinoma triangle) and are virtually always malignant. The treatment goal is relief of symptoms related to excessive hormone production and cure or palliation of the malignant process. Patients frequently require medical and surgical therapy. Before surgical exploration the patient should be evaluated for an adrenal tumor by measuring urinary excretion rates of glucocorticoids, mineralocorticoids, and sex hormones.
- B. **MEN-2 syndromes** are characterized by medullary thyroid carcinoma (MTC) and include MEN-2A, MEN-2B, and familial, non-MEN MTC (FMTC). These autosomal dominant syndromes are caused by mutations in the RET proto-oncogene. RET encodes a transmembrane tyrosine kinase receptor, in which mutations cause constitutive activation (tyrosine phosphorylation) of the RET protein, which drives tumorigenesis. Genetic testing should be performed on all suspected individuals.
1. **MTC develops in all MEN-2A patients**, whereas pheochromocytomas arise in approximately 50% and hyperplasia of the parathyroid glands in approximately 25%.
 - a. **MTC** develops at an early age on a background of C-cell hyperplasia and is multifocal. Treatment is total thyroidectomy with central lymph node neck dissection, which entails removal of all nodes and fatty tissue between the carotid sheaths and the innominate artery. Parathyroidectomy with autotransplantation of parathyroid tissue into the forearm (MEN-2A) or sternocleidomastoid (MEN-2B, FMTC) can be performed. Patients should be screened for pheochromocytomas before undergoing surgery. MTC cells make calcitonin, which can be measured in screening and follow-up examinations, especially for residual or recurrent disease. Infusing calcium gluconate and pentagastrin before measuring calcitonin provides more reliable results than following basal calcitonin levels. Patients who are found to have a known RET mutation based on genetic testing should undergo preventative thyroidectomy before age 7 for MEN-2A and in infancy for MEN-2B.
 - b. **Pheochromocytomas** in MEN-2A and MEN-2B are generally not metastatic or extraadrenal. Screening should be done at the same time as screening for MTC and consists of 24-hour urinary measurements of catecholamines, vanillyl mandelic acid (VMA), and metanephrine. Operatively, most tumors can be approached laparoscopically.
 2. **MEN-2B** is a variant of MEN-2A, in which patients develop MTC and pheochromocytomas but not hyperparathyroidism. Patients also develop ganglioneuromatosis and a characteristic physical appearance, with hypergnathism of the midface, marfanoid body habitus, and multiple mucosal neuromas. MTC is particularly aggressive in these patients.
 3. **FMTC** is characterized only by development of MTC and no other tumors. MTC is generally more indolent in these patients.

IV. **Sarcomas** are associated with the LFS (described previously), hereditary retinoblastoma, and neurofibromatosis types 1 and 2.

- A. **Neurofibromatosis type 1 (NF1)** is a common autosomal dominant disorder, affecting more than 80,000 people in the United States and caused by mutations in the NF1 gene. The three hallmarks of the disease are multiple neurofibromas, café au lait spots, and Lisch nodules (benign iris hamartomas). Neurofibromas may be removed for pain, functional impairment, or cosmetic reasons. In the gastrointestinal tract, neurofibromas may result in bleeding, obstruction, or intussusception. NF1 patients have a much greater likelihood of developing neurofibrosarcomas and are also at risk for pheochromocytomas, CNS tumors, and leukemias. An enlarging mass, localized pain, or neuropathy should be considered a neurofibrosarcoma until proved otherwise. These tumors are generally resistant to chemoradiation.
- B. **Neurofibromatosis type 2 (NF2)** is a much rarer disorder, characterized by the defining feature of bilateral vestibular schwannomas and caused by the NF2 gene. The syndrome is also characterized by skin neurofibromas, café au lait spots, cataracts, and other CNS tumors, including gliomas, meningiomas, schwannomas, and neurofibromas.

V. **Melanoma** is familial in approximately 10% of cases, often associated with multiple atypical moles. **Familial atypical multiple-mole melanoma syndrome (FAMMM)** has also been called *dysplastic nevus syndrome*, *B-K syndrome*, and *large atypical nevus syndrome*. A National Institutes of Health Consensus Conference defined FAMMM as having (1) the occurrence of malignant melanoma in one or more first- or second-degree relatives; (2) a large number of melanocytic nevi, usually more than 50, some of which are atypical and variable in size; and (3) melanocytic nevi that have certain histopathologic features, including an architectural disorder with asymmetry, subepidermal fibroplasia, and lentiginous melanocytic hyperplasia with spindle or epithelial melanocyte nests. The lesions predominantly occur on the trunk but are also found on the buttocks, scalp, and lower extremities. The relative risk for developing melanoma when multiple atypical moles are present ranges from 5 to 11 based on multiple studies. The median age for melanoma diagnosis is 34.

- A. **CDKN2**, a cell cycle protein gene, has been found to contain germline mutations in some kindreds with familial melanoma (*Nat Genet* 8:15, 1994). Other malignancies have been related to mutations in the CDKN2 gene, especially pancreatic cancer. There may be other genes contributing to FAMMM.
- B. **Screening** begins at around puberty and consists of yearly physical examinations, including a total body skin examination. For patients who have a large number of moles, baseline photographs or computerized scanning are helpful. Patients should examine their own skin regularly. Suspicious lesions should undergo biopsy. Sun exposure should be avoided. Regular ophthalmologic examinations should be performed due to the increased risk of ocular nevi and

ocular melanoma.

VI. Genetic testing and counseling. A family history should be taken on all patients with cancer. An inherited cancer syndrome should be suspected if the same cancer affects several close relatives, if the cancer occurs at an earlier age than sporadic cases, or if the cancer is bilateral. A family pedigree helps identify potential gene carriers and guides counseling and testing. Genetic counselors are an important complement to surgeons to discuss test results, limitations, confidentiality, and therapeutic options in a nonbiased manner. In an evaluation of the use of the commercial APC gene test, more than 30% of physicians misinterpreted the test results (*N Engl J Med* 336:823, 1997). Genetic counselors should be consulted for any patient in whom genetic testing is done.

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The Washington Manual of Surgery

27 Endocrine Surgery

Jennifer Killeen Lowney and Terry C. Lairmore

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Thyroid

I. Evaluation of thyroid disorders

- A. **Clinical manifestations** of hyperthyroidism reflect increased catabolism and excessive sympathetic activity caused by excess circulating thyroid hormones. Symptoms of hyperthyroidism include weight loss despite normal or increased appetite, heat intolerance, excessive perspiration, anxiety, irritability, palpitations, fatigue, muscle weakness, and oligomenorrhea. Signs of hyperthyroidism include goiter, sinus tachycardia or atrial fibrillation, tremor, hyperreflexia, fine or thinning hair, thyroid bruit, and muscle wasting. The presentation of hyperthyroidism can vary considerably with age: Young patients usually present with hypermetabolism, whereas older patients may present primarily with tachyarrhythmias or cardiac failure. Rarely, elderly patients experience only wasting, apathy, confusion, or depression (apathetic hyperthyroidism). **Clinical features of hypothyroidism** include cold intolerance, weight gain, constipation, edema (especially of the eyelids, hands, and feet), dry skin, weakness, somnolence, and menorrhagia.
- B. **Biochemical thyroid function testing** confirms clinically suspected abnormalities in thyroid function; however, test results must be interpreted in the context of clinical findings. The introduction of sensitive thyrotropin assays has transformed thyroid function testing from strategies based on thyroxine (T_4) to strategies based on thyroid-stimulating hormone (TSH) (*Mayo Clin Proc* 69:469, 1994). Currently, measurement of serum TSH level and free T_4 (FT_4) is the best and most efficient combination of blood tests for diagnosis of most patients with thyroid disorders.
1. Measurement of **TSH** (0.3–5.0 mIU/L) by a second-generation sensitive TSH (sTSH) test is the single most useful biochemical test in the diagnosis of thyroid illness. In most ambulatory and hospitalized patients without pituitary disease, increased sTSH signifies hypothyroidism, suppressed sTSH suggests hyperthyroidism, and **normal sTSH reflects a euthyroid state**. Significantly, critically ill, hospitalized patients, especially those receiving drugs such as dopamine or glucocorticoids, may have transient changes in sTSH (usually elevated) without true abnormalities in thyroid function.
 2. Assessment of T_4 concentration corroborates identified abnormalities in TSH and provides an index of severity of thyroid dysfunction. **Total T_4** (3–12 $\mu\text{g/dL}$) measurements quantify bound and unbound hormone and do not reflect directly the small “free” or active T_4 fractions. Factors that increase the thyroxine-binding globulin (TBG) concentration (estrogens, pregnancy, liver disease) may elevate the total T_4 or triiodothyronine (T_3) despite a normal free hormone concentration and a euthyroid state. Androgens, severe hypoproteinemia, chronic liver disease, and acromegaly result in decreased TBG.
 3. **The resin T_3 uptake (RT_3U)** (20–40%) test measures unoccupied thyroid hormone-binding sites on TBG by allowing radiolabeled T_3 to compete for binding between TBG and a resin. This assay provides an indirect measure of FT_4 . In patients with hyperthyroidism, the resin uptake is elevated because most of the sites on TBG are occupied by T_4 so that more radioactive T_3 binds to the resin. The RT_3U is related directly to the FT_4 fraction and inversely related to the TBG binding sites.
 4. **The FT_4 index** ($FT_4I = \text{total } T_4 \times RT_3U$) (0.85–3.50) correlates more closely with the level of FT_4 , eliminates ambiguity introduced by altered thyroglobulin levels, and is the preferred test to estimate FT_4 .
 5. **Measurement of T_3** (80–200 ng/dL) is an unreliable test in hypothyroidism. This test is useful in the occasional patient with suspected hyperthyroidism, suppressed sTSH, and normal FT_4I (T_3 thyrotoxicosis).
 6. **Antithyroid microsomal antibodies** are found in the serum of patients with autoimmune thyroiditis (Hashimoto's thyroiditis), and measurement of these antibodies is helpful to diagnose this common cause of hypothyroidism. **Anti-TSH receptor antibodies**, which stimulate the TSH receptor, are detectable in more than 90% of patients with autoimmune hyperthyroidism (Graves' disease); however, their measurement is not often needed in the diagnosis of this disease.
 7. A useful **thyroid function test algorithm** (*Clin Lab Med* 13:673, 1993) includes sTSH assay as the initial test. If this is normal, no further tests are needed. If sTSH is elevated, FT_4I and microsomal antibodies are measured to confirm hypothyroidism, which is often autoimmune. If sTSH is suppressed, FT_4I is measured to confirm primary hyperthyroidism. If TSH is low and FT_4I is normal, T_3 is measured to diagnose T_3 thyrotoxicosis.
- C. **Thyroid imaging** is most often accomplished with radionuclide scanning or ultrasound; other imaging modes, including computed tomographic (CT) scanning and MR scan, are useful in special circumstances.
1. **Technetium thyroid scanning** 20 minutes after the intravenous injection of technetium-99m (^{99m}Tc) is useful in determining the size of the thyroid and in differentiating solitary functioning nodules from multinodular goiter or Graves' disease. Hypofunctioning areas (cyst, neoplasm, or suppressed tissue adjacent to autonomous nodules) are “cold,” whereas areas of increased synthesis are “hot.” Thyroid scans cannot differentiate benign from malignant thyroid nodules. “Cold” nodules have a 15–20% risk of malignancy; hence, most should be removed. “Hot” nodules are almost never malignant. ^{99m}Tc thyroid scans are most useful as adjunctive tests to assess risk of malignancy in patients with indeterminate thyroid nodule cytology or in hyperthyroid patients suspected of having a hyperfunctioning thyroid adenoma. Thyroid scanning 4–24 hours after oral iodine-131 (^{131}I) is useful to identify metastatic differentiated thyroid tumors and to both confirm a diagnosis of Graves' disease and predict a response to ^{131}I radioablation.
 2. **Thyroid ultrasonography** with high-frequency (7.5–10.0 MHz) transducers accurately determines gland volume as well as the number and character of thyroid nodules (*Am J Med* 99:642, 1995). Although not completely reliable, features suggestive of malignancy on ultrasound include hypoechoic pattern, incomplete peripheral halo, irregular margins, and microcalcifications. Ultrasound is useful to guide fine-needle aspiration (FNA) biopsy and cyst aspiration. Cysts seen on ultrasound, especially those larger than 3 cm, are malignant in up to 14% of cases.
 3. **CT scanning and MR scan** of the thyroid are costly and generally are reserved for assessing substernal or retrosternal masses suspected to be goiters.

II. Specific thyroid disorders

- A. Autoimmune diffuse toxic goiter (**Graves' disease**) is the most common cause of hyperthyroidism and may be caused by stimulating immunoglobulins directed against the TSH receptor. Graves' disease may be treated with antithyroid drugs, ablation with radioactive iodine (RAI), or surgery, depending on the clinical situation.
1. Thionamide drugs, such as **propylthiouracil (PTU) or methimazole**, are the initial therapy in most cases. PTU (100–300 mg p.o. t.i.d.) is given for 4–6 weeks until the patient becomes euthyroid; then the dosage usually is decreased (PTU, 100 mg p.o. t.i.d.). The patient is then treated empirically for 6–18 months, at which time the drugs are withdrawn. Clinical features that favor remission include small gland size and mild hyperthyroidism of recent onset, although overall, long-term remission is achieved in less than 20–30% of patients. Antithyroid drugs also are used to prepare thyrotoxic patients for surgery or ablative therapy. PTU may be given during pregnancy at reduced doses, especially if thyroidectomy is necessary in the second trimester. Minor adverse reactions occur infrequently and include rash, hepatitis, arthralgias, and a lupuslike syndrome. Agranulocytosis is a rare (0.5%) but serious side effect of thionamide therapy.
 2. **Ablation with RAI is the treatment of choice for most patients with Graves' disease.** A dose of 5–10 mCi of ¹³¹I is given orally and is 75% effective after 4–12 weeks. In the 25% of patients with persistent thyrotoxicosis after 12 weeks, double the initial dose is repeated. After treatment, there is a high incidence (70%) of eventual permanent hypothyroidism, which is managed easily by replacement therapy. There are virtually no other long-term side effects of RAI (i.e., no significantly increased risk of thyroid cancer, leukemia, or teratogenicity). Contraindications to radiotherapy include pregnant women, newborns, patients who refuse, or patients with low RAI uptake (<20%) in the thyroid. Treatment of children or young adults (younger than 30 years) with RAI is controversial because of presumed long-term oncogenic risks.
 3. **Thyroidectomy for Graves' disease** may be indicated for children or adolescents, pregnant women (late second or early third trimester), patients unresponsive to or noncompliant with medical therapy, or patients who refuse RAI. A bilateral subtotal thyroidectomy should be performed with the goal of leaving a 1- to 2-g vascularized cuff of thyroid on each side. However, some centers advocate total thyroidectomy as primary treatment to decrease recurrence rate of goitrous disease. Risks of surgery are extremely small (<1%) in experienced hands but include hypoparathyroidism and injury to the recurrent laryngeal nerve. Surgery results in hypothyroidism less frequently than does RAI (*N Engl J Med* 311:426, 1984). The long-term incidence of recurrent hyperthyroidism after surgery is approximately 10%. Patients with recurrent hyperthyroidism after thyroidectomy should be treated with RAI.
- B. In **multinodular goiter**, a large goiter or retrosternal extension can compress the trachea. Subtotal or total thyroidectomy is the treatment of choice if there are symptoms of compression, suspicion of malignancy, or questionable nodules, or if the gland is cosmetically bothersome.
- C. **Toxic adenoma** is an autonomously functioning thyroid nodule that produces hyperthyroidism and is treated by surgical excision (lobectomy).
- D. **Rare causes** of hyperthyroidism include self-administration of excessive thyroid hormone (factitious hyperthyroidism), iodine-induced hyperthyroidism, pituitary TSH-secreting adenoma, trophoblastic tumors secreting chorionic gonadotropin (which has TSH-like activity), struma ovarii, and thyroiditis.

III. Hypothyroidism is almost always caused by primary hypofunction of the thyroid gland. Clinically, hypothyroid patients should be separated into those without goiter (primary atrophy), those with goitrous hypothyroidism (i.e., Hashimoto's thyroiditis, drug-induced hypothyroidism, iodine deficiency, and congenital causes of dysmorphogenesis), and those with postablative hypothyroidism (after thyroidectomy or treatment with RAI). Postablative hypothyroidism and Hashimoto's thyroiditis are the most important causes of hypothyroidism encountered by the surgeon. Diagnosis rests on the characteristic clinical features and laboratory findings of an elevated TSH (usually >15 units/mL) and a decreased FT₄ level. A low TSH in association with low T₄ suggests pituitary or hypothalamic failure.

IV. Thyroiditis represents a diverse group of autoimmune and inflammatory disorders characterized by infiltration of the thyroid with inflammatory cells and subsequent fibrosis of the gland.

- A. **Hashimoto's thyroiditis** is a chronic autoimmune disorder characterized by destructive lymphocytic infiltration of the thyroid. The disease is 15 times more common in women, and more than 90% of patients have circulating antibodies directed against thyroid microsomes and thyroglobulin. Although patients initially are euthyroid, hypothyroidism may occur later. A firm symmetric or asymmetric goiter is palpable and usually (but not always) nontender. Cervical lymphadenopathy is uncommon. Euthyroid patients may not require treatment. Thyroid hormone is given to hypothyroid patients both as replacement therapy and to suppress TSH. Thyroidectomy is indicated for compressive symptoms or for a dominant nodule suspicious for malignancy.
- B. **Acute suppurative thyroiditis** is rare and is caused by infection with *Streptococcus* or *Staphylococcus* species. Treatment consists of appropriate antibiotic therapy and surgical drainage of abscesses.
- C. **Subacute (de Quervain's) thyroiditis** is a rare condition that occurs in young women, often after a viral upper respiratory tract infection. Symptoms of fatigue, weakness, and painful thyroid enlargement radiating to the patient's jaw or ear are treated with nonsteroidal antiinflammatory drugs or with steroids. The condition almost always remits spontaneously within a few weeks. Thyroidectomy may be indicated in rare cases of persistent thyroiditis after months of unsuccessful steroid treatment.
- D. **Riedel's thyroiditis** is a rare, progressive, inflammatory condition of the entire thyroid gland, strap muscles, and other neck structures. Its cause is unknown, and it can be associated with other fibrotic processes, including retroperitoneal fibrosis, sclerosing cholangitis, and fibrosing mediastinitis. The lymphocytic infiltrate and dense fibrous tissue reaction in the thyroid result in a firm, nontender goiter with a woody texture. Riedel's thyroiditis may require surgical excision to exclude malignancy or relieve compressive symptoms.

V. A solitary thyroid nodule occurs commonly (4–7% of adults) and is usually a benign lesion. Such nodules may be associated with a multinodular goiter or with an otherwise normal thyroid. The malignant potential of the newly discovered thyroid nodule is of justifiable concern to both physician and patient, and the goal of diagnostic testing is to separate the relatively few patients with thyroid malignancy from the larger group of patients with benign thyroid nodules. Surgical intervention is appropriate for all malignant or suspicious thyroid nodules. The frequency of cancer in surgically excised nodules is 8–17% (*N Engl J Med* 328:553, 1993).

- A. **History and physical examination** are invaluable in the management of the thyroid nodule. Nodules in the very young and very old (especially men) are more likely to be malignant. Exposure to ionizing radiation increases the incidence of both benign and malignant thyroid nodules and is a well-recognized risk factor for the development of thyroid carcinoma. A family history of thyroid malignancy, familial polyposis, or other endocrine disease also increases risk of cancer. Rapid nodule growth, pain, compressive symptoms, or hoarseness of voice increase the likelihood of malignancy but are nonspecific. Physical findings of a solitary nodule with firm or irregular texture or with fixation to surrounding structures suggest malignancy. Similarly, the presence of enlarged cervical lymph nodes is extremely important because this finding is highly indicative of thyroid cancer. Malignancy is uncommon in hyperfunctioning nodules, and all patients should have a serum TSH measured to exclude clinical or subclinical thyrotoxicosis.
- B. **FNA** is the initial diagnostic test of choice for the euthyroid patient with a solitary thyroid nodule. This procedure is safe, inexpensive, and easy to perform, and it allows better selection of patients for operation than does any other technique (*Ann Intern Med* 118:282, 1993).
 1. **The technique of FNA** (*World J Surg* 5:61, 1981) involves palpation of the nodule, aspiration of sufficient material, and adequate preparation and staining of a smear of the aspirated cells. The equipment required includes a 25-gauge hypodermic needle, a 10-mL syringe, and a syringe holder (e.g., Cameco Ltd., London), which allows operation with a single hand. The thyroid gland is palpated with the patient supine and his or her neck slightly extended. The operator stands on the side opposite the lobe to be aspirated and immobilizes the nodule between the index and middle fingers of his or her nondominant hand. The overlying skin is cleansed with povidone-iodine or alcohol. No local anesthetic is required. The needle is introduced into the nodule, then the piston of the syringe can be retracted to create suction, or material can be allowed to pass into the syringe by passive action, to minimize the blood in the specimen. The needle tip can be gently angled in multiple directions within the nodule, again trying to avoid excess trauma and bloody specimen. Before removing the needle, the suction is released to prevent aspiration of the needle contents into the syringe. Then the needle is detached, and air is introduced into the syringe before aspirated cells are expressed onto a slide. The smear is air-dried and stained, using the May-Grünwald-Giemsa method, or it is alcohol-fixed and stained, using the Papanicolaou method. Usually, two or three passes are made to limit sampling error.
 2. **Cytologic results of FNA** can be benign, malignant, or indeterminate, provided that an adequate specimen is obtained (an adequate specimen has at least 10 groups of well-preserved follicular cells, with 10 cells/group, obtained from two separate needle passes). The accuracy of these results ranges from 70% to 90% and is highly dependent on the skill and experience of the cytopathologist. The false-negative rate of FNA is low (<6%), and patients with negative (i.e., not cancerous or indeterminate) cytology can be followed safely (*Surgery* 106:980, 1989). Indeterminate aspirations pose difficult management decisions because carcinoma occurs in 10–30% of these cases. Follicular and Hurthle cell neoplasms and the follicular variant of papillary cancer account for many indeterminate results, whereas papillary, medullary, and undifferentiated thyroid carcinomas usually have distinctive cytopathologic features. Diagnostic thyroid lobectomy is indicated for all FNA-indeterminate and FNA-positive thyroid nodules and for all nodules, regardless of FNA result, in patients with a history of radiation exposure. An inadequate FNA biopsy is obtained in up to 20% of cases; half of these can be diagnosed with repeated aspiration. The management of nodules with persistently inadequate FNA should be guided by clinical criteria.
- C. **Radionuclide thyroid scans** detect areas of active or decreased thyroid hormone synthesis but do not provide information that allows clear separation of benign and malignant nodules. Approximately 20% of nodules that are hypofunctioning ("cold") on thyroid scintiscan are malignant, but the majority are benign. Hyperfunctioning ("hot") nodules carry a low risk of malignancy, but exceptions occur. Thyroid scanning may be useful in patients with indeterminate FNA

cytology because, in such patients, hyperfunctioning nodules almost always are benign.

- D. **Ultrasonography** is a sensitive method for determining whether a lesion is solid or cystic, but it cannot distinguish reliably between benign and malignant nodules. Although thyroid cysts have a lower likelihood of being malignant, larger carcinomas can undergo cystic degeneration. Cysts may disappear after FNA, but those that persist, recur, or yield insufficient material for interpretation should be excised.
- E. **Thyroid lobectomy** is indicated for (1) nodules with malignant or indeterminate aspiration cytology, (2) nodules in children, (3) nodules in patients with either a history of neck irradiation or a family history of thyroid cancer, and (4) symptomatic or cosmetically bothersome nodules.

VI. Thyroid neoplasms

A. **Differentiated (papillary and follicular) thyroid cancer** is among the most curable of human cancers (*N Engl J Med* 338:297, 1998). These cancers are rare in children and increase in frequency with age; the female to male ratio is approximately 2.5:1.0. The cause of these cancers is unknown, but childhood exposure to radiation (10–1,500 cGy) is the best known etiologic factor. Approximately 30% of exposed children develop thyroid nodules, and of these, an estimated 30% are malignant. The appropriate initial procedure for a solitary thyroid nodule suspected of being malignant is a total lobectomy and isthmectomy. Controversy exists about the extent of surgery (lobectomy versus total thyroidectomy) that should be performed for patients with proven differentiated thyroid cancer, principally because these patients have a good prognosis irrespective of the surgical treatment. Prognosis depends mostly on the patient's age as well as extent and histologic subtype of disease. Several prognostic index systems have been developed, including the MACIS (metastasis, age, completeness of resection, invasion, and size) and the tumor, node, metastasis classification adjusted for patient age. In all, 85–90% of patients fall into a low-risk category with favorable prognosis.

1. **Papillary thyroid carcinoma (PTC)** represents 85% of thyroid carcinomas and occurs in any age group, especially children and women younger than 40 years. PTC is often multifocal and frequently metastasizes to cervical lymph nodes. Occult, clinically insignificant foci of microscopic PTC are found in 4–28% of autopsies or in thyroidectomy specimens for benign diseases. A young age at diagnosis is a favorable prognostic factor, and even involvement of cervical nodes does not affect the prognosis adversely. Total thyroidectomy is appropriate for patients with gross evidence of bilateral disease, multifocal PTC, or a history of neck irradiation. Total thyroidectomy is arguably the treatment of choice for unilateral tumors larger than 1.5 cm. Advantages of total thyroidectomy include the ability to treat extrathyroidal metastases or recurrences with RAI and to use serum thyroglobulin to monitor therapy. Retrospective studies also show a decreased risk of recurrence or death. Complications of total thyroidectomy include hypoparathyroidism and recurrent laryngeal nerve (RLN) injury, which occur in less than 1% of cases. Prophylactic neck dissection is not indicated for PTC, but a modified ipsilateral, central neck dissection is indicated for patients with palpable metastases in cervical nodes. Radioablation of residual thyroid tissue or metastatic or residual cancer is performed with 75–100 mCi of ¹³¹I 4 weeks after total thyroidectomy while the patient is hypothyroid (i.e., **TSH >30** on no replacement of T₄). Thyroid replacement may be resumed for 3–6 months and then discontinued for 1 month before obtaining a second ¹³¹I scintiscan to test for remaining functional thyroid tissue. Ablation may be repeated at higher doses if the scan is positive. Long-term suppression therapy with T₄ decreases recurrences and may improve survival.
 2. **Follicular thyroid carcinoma** (10% of thyroid carcinomas) is rare before age 30 years and has a slightly worse prognosis than does PTC. Unlike PTC, follicular thyroid cancer spreads hematogenously to bone, lung, or liver, either at the time of diagnosis or years after resection. Small, unilateral follicular carcinomas with limited invasion of the tumor capsule or spread along blood vessels may be treated with thyroid lobectomy, whereas multicentric tumors and tumors with more extensive invasion or distant metastases are treated with total thyroidectomy. Radioablation is indicated after total thyroidectomy, followed by lifelong thyroid hormone suppression.
- B. **Medullary thyroid carcinoma (MTC)** arises from the thyroid C cells that derive from the neural crest and secrete calcitonin. MTC may occur sporadically or may be inherited either alone or as a component of multiple endocrine neoplasia (MEN) types 2A or 2B. Sporadic MTC usually is detected as a firm, palpable, unilateral nodule with or without involved cervical lymph nodes. Patients with hereditary MTC develop bilateral, multifocal tumors and often are diagnosed on the basis of family screening. MTC should be suspected if tumor calcification is noted on plain X-rays or if the patient has profuse diarrhea and episodic flushing (caused by excess calcitonin release). MTC spreads early to cervical lymph nodes and may metastasize to liver, lungs, or bone. All patients with suspected or known MTC should have a careful family history taken, be tested biochemically for pheochromocytoma before thyroidectomy, and be genetically tested for DNA mutations in the RET proto-oncogene (*Hum Mol Genet* 2:851, 1993).
1. **The biochemical diagnosis** of MTC is made by the demonstration of an elevated plasma calcitonin level after the intravenous administration of calcium and pentagastrin. The patient should fast overnight. A peripheral 21-gauge “butterfly” intravenous line connected to a three-way stopcock is started to provide a simple and rapid way to administer medicines and draw blood samples. Blood is drawn before and at 1, 2, 3, and 5 minutes after the intravenous administration of calcium gluconate (2 mg/kg over 1 minute) and pentagastrin (0.5 µg/kg over 5 seconds). Plasma calcitonin is measured by radioimmunoassay. Stimulated calcitonin testing is also a sensitive way to detect residual or recurrent MTC postoperatively.
 2. **Treatment** of MTC is total thyroidectomy with removal of the lymph nodes in the central zone of the patient's neck (from the sternal notch to hyoid bone and laterally to the carotid sheaths). A modified neck dissection is indicated for clinically involved ipsilateral cervical lymph nodes. Of patients with persistently elevated calcitonin levels after initial thyroidectomy, 28% had normalized early postoperative calcitonin levels after a subsequent meticulous bilateral microdissection of cervical lymph nodes (*Surgery* 114:1090, 1993).
- C. **Undifferentiated or anaplastic thyroid carcinoma** (1–2% of thyroid carcinomas) carries an extremely poor prognosis, usually presents as a fixed, sometimes painful goiter, and usually occurs in patients older than the age of 50 years. Invasion of local structures, with resultant dysphagia, respiratory compromise, or hoarseness due to recurrent laryngeal nerve involvement can preclude curative resection. External irradiation or chemotherapy may provide limited palliation.
- D. **Primary malignant lymphoma of the thyroid** often is associated with Hashimoto's thyroiditis. Radiotherapy is the treatment of choice for disease confined to the patient's neck, and radical surgical resection is not indicated once a diagnosis is made. Compressive symptoms usually respond rapidly to radiotherapy.

VII. Postoperative **thyroid hormone replacement** is necessary after total or near total thyroidectomy or ablation with radioiodine. Oral levothyroxine is begun before discharge at an average dose of 100 µg per day (0.8 µg/lb per day). Adequacy of thyroid hormone replacement is assessed by measuring T₄ and TSH at 6–12 weeks after surgery. Adjustments to the dose should not be more frequent than monthly in the absence of symptoms and should be cautious (12.5- to 25.0-µg increments).

VIII. Management of complications after thyroidectomy

- A. **Hemorrhage** is a rare but serious complication of thyroidectomy that usually occurs within 6 hours of surgery. Management can require control of the airway by endotracheal intubation and can rarely mandate urgent opening of incision and evacuation of hematoma before returning to the operating room for wound irrigation and ligation of the bleeding point.
- B. **Transient hypocalcemia** commonly occurs 24–48 hours after thyroidectomy but infrequently requires treatment. Patients who are markedly symptomatic or who have serum calcium below 7 mg/dL are given 1–2 ampules (10–20 mL) of 10% calcium gluconate i.v. over 1–2 minutes followed by temporary oral calcium carbonate (500 mg p.o. t.i.d.). More prolonged intravenous replacement is achieved by mixing 6 ampules of 10% calcium gluconate (540 mg elemental calcium) in D5W, 500 mL, for infusion at 1 mL/kg per hour. Permanent hypoparathyroidism is uncommon after total thyroidectomy. Normal parathyroid tissue removed or devascularized at the time of total thyroidectomy may be autotransplanted into sternocleidomastoid muscle to prevent postoperative hypocalcemia (*Ann Surg* 223:472, 1996).
- C. **Recurrent laryngeal nerve (RLN) injury** is a devastating complication of thyroidectomy that should occur rarely (<1%). Unilateral RLN injury causes hoarseness, and bilateral injury compromises the airway, necessitating tracheostomy. Repeat neck exploration, thyroidectomy for extensive goiter or Graves' disease, and thyroidectomy for fixed, locally invasive cancers are procedures particularly prone to RLN injury. Intentional (as with locally invasive cancer) or inadvertent transection of the RLN can be repaired primarily or with a nerve graft, although the efficacy of these repairs is not known. Temporary RLN palsies can occur during thyroidectomy, and these usually resolve over a period of 4–6 weeks. The **external branch of the superior laryngeal nerve** may be injured if not identified during ligation of the superior thyroid pole vascular bundle. This injury results in weakness of the patient's voice at high pitch. The best prevention of these injuries is a thorough understanding of the anatomy of these nerves.

Parathyroid

I. **Hyperparathyroidism (HPT)** refers to hypercalcemia caused by inappropriate parathyroid hormone (PTH) release from the parathyroid glands. Primary HPT results from autonomous release of PTH from parathyroid adenoma or hyperplastic parathyroid glands. Secondary HPT results from a defect in mineral homeostasis (e.g., renal failure), with a compensatory increase in parathyroid function. *Tertiary HPT* refers to the development of autonomous, calcium-insensitive parathyroids after prolonged secondary stimulation (e.g., prolonged renal failure).

A. Primary HPT

1. **Incidence.** Primary HPT has an incidence of 0.25–1.00/1,000 in the United States and is especially common in postmenopausal women. It most often occurs sporadically, but it can be inherited alone or as a component of familial endocrinopathies, including MEN types 1 and 2A.
2. The more common **manifestations of HPT** include nephrolithiasis, osteoporosis, hypertension, and emotional disturbances. The widespread use of the

multichannel autoanalyzer has led to more patients being diagnosed with “asymptomatic” hypercalcemia or with earlier symptoms, such as muscle weakness, polyuria, anorexia, and nausea. When carefully questioned, many patients with HPT are indeed symptomatic.

3. **Differential diagnosis of hypercalcemia** includes HPT, malignancy, granulomatous disease (e.g., sarcoidosis), immobility, hyperthyroidism, milk-alkali syndrome, and familial hypocalciuric hypercalcemia (FHH). Patients with hypercalcemia and suspected HPT should minimally have serum calcium, phosphate, creatinine and PTH measured. The diagnosis of HPT is biochemical and requires demonstration of hypercalcemia (serum calcium of >10.5 mg/dL) and an elevated PTH level. Currently, the assay of choice for PTH is the highly sensitive and specific intact PTH level by radioimmunoassay. Free calcium levels (ionized calcium) is a more sensitive test of physiologically active calcium and is the test of choice for hypercalcemia. Hypercalcemia without an elevated PTH can be due to a variety of causes (especially malignancy, Paget's disease, sarcoidosis, milk-alkali syndrome) that must be excluded. Serum alkaline phosphatase is elevated (10–40% of patients), and serum phosphate is decreased in 50% of patients with HPT if renal function is normal. Elevated PTH promotes bicarbonate excretion, causing a hyperchloremic metabolic acidosis; the chloride-phosphate ratio exceeds 33 in 96% of patients with HPT (*Ann Intern Med* 80:200, 1974). Radiographic features of HPT are seen in advanced cases and include decreased bone density, osteitis fibrosa cystica, and the pathognomonic sign of subperiosteal bone resorption on the radial aspect of the phalanges of the second or third digits of the hand.
 4. **Preoperative localization** of parathyroid adenomas is generally not necessary before a careful neck exploration by an experienced endocrine surgeon and as stated by the 1991 National Institutes of Health Consensus conference. However, current practices use several recent techniques to facilitate limited neck exploration to ensure success rates with optimal cosmesis in the outpatient setting. These techniques include radio- and/or image-guided exploration (sestamibi-guided or ultrasound-guided), or videoscopic exploration, and intraoperative intact PTH level monitoring (*Surgery* 122:1107, 1997). The most frequently applied approach is preoperative sestamibi scanning, followed by direct excision of the scan-identified gland and confirmation of cure by intraoperative PTH measurement. This intraoperative test requires the availability of a rapid assay of intact PTH that confirms the success of the surgery immediately if the PTH level falls greater than 50% 10 minutes after the apparent source of PTH has been removed. If the preoperative localization scan is not informative, then the standard full neck exploration is appropriate.
 5. **Parathyroidectomy is indicated** for all patients with symptomatic HPT. Nephrolithiasis, bone disease, and neuromuscular symptoms are improved more often than are renal insufficiency, hypertension, and psychiatric symptoms. Parathyroidectomy for asymptomatic HPT is somewhat controversial: A recent large prospective study of patients with asymptomatic HPT found that 27% developed symptoms of hypercalcemia with 10-year follow-up (*N Engl J Med* 341:1249, 1999). Accepted indications include markedly elevated serum calcium, hypercalcemic crisis, reduced creatinine clearance, asymptomatic kidney stones, markedly elevated urinary calcium excretion, and advanced osteoporosis. Close observation is required for patients not treated surgically.
 6. **Neck exploration and parathyroidectomy** for HPT result in normocalcemia in more than 95% of patients when performed by an experienced surgeon without any preoperative or intraoperative localization studies. The neck is exposed through a transverse cervical incision of minimal length (2.5–4.0 cm, on average). This is performed under general anesthesia, although the current techniques more and more use local or regional anesthesia. A thorough, orderly search and identification of all four parathyroid glands are the cornerstone of surgical management of HPT. Parathyroid glands are red-brown to yellow and flat or oval, with a characteristic vascular architecture; however, it may be difficult to distinguish them from fat or lymphoid tissue. Superior parathyroid glands develop from the fourth pharyngeal pouch and most commonly are located dorsally on the middle or upper thyroid lobe, near the intersection of the inferior thyroid artery and recurrent laryngeal nerve. Ectopic superior glands usually are found posteriorly in the tracheoesophageal groove or cranial to the superior thyroid pole. Inferior parathyroid glands develop in conjunction with the thymus from the third pharyngeal pouch, are more variable in position than are the superior glands, and usually are located at the inferior pole of the thyroid lobe within the thyro-thymic ligament. Ectopic inferior glands most likely are found in the mediastinum embedded in the thymus. The normal combined weight of the parathyroid glands is 90–200 mg. Determination that a parathyroid gland is abnormally enlarged is best made by the surgeon at the time of operation and not by hypercellularity or other features on histologic examination. Most often, a single adenomatous gland is found; the other, normal parathyroid glands should be left in place. Routine biopsy of normal parathyroid glands is expensive, risks devascularization of the gland, and is not routinely necessary. Occasionally, multiple parathyroid adenomas are found, which should be removed, leaving at least one normal parathyroid behind. Management of four-gland parathyroid hyperplasia is controversial and may include total parathyroidectomy and parathyroid autotransplantation or 3.5-gland parathyroidectomy. The dictation of a clear, factual operative note detailing the identification and position of each parathyroid gland is essential. This information is invaluable in the unlikely event of reoperation for persistent or recurrent HPT.
 7. **Familial primary HPT** may occur alone or as a component of MEN type 1 and type 2A. HPT in these syndromes is primarily chief cell hyperplasia and variable enlargement of all four glands. These patients have a higher incidence of recurrent or persistent hypercalcemia after parathyroidectomy. Familial benign hypercalcemia, or FHH, is an autosomal dominant form of HPT in which affected family members have mild hypercalcemia, hypocalciuria (urinary excretion of <100 mg of calcium daily), hypomagnesemia, and rarely develop renal or bone complications (*J Pediatr* 17:1060, 1972). Parathyroidectomy is ineffective for FHH.
- B. Hypercalcemia from **secondary and tertiary HPT** is treated initially with dietary phosphate restriction, phosphate binders, and vitamin D supplementation. Patients with medically unresponsive, symptomatic HPT (e.g., bone pain and osteopenia, ectopic calcification, or pruritus) may be surgically treated with total parathyroidectomy and heterotopic autotransplantation.

II. Reoperative parathyroid surgery may be necessary for persistent or recurrent HPT. In all cases, HPT must be reconfirmed biochemically, and 24-hour urinary calcium should be obtained to exclude FHH. Preoperative localization is mandatory and includes careful review of the operative note from the initial surgery and concordant noninvasive studies. Approximately 70–80% of patients undergoing reexploration after an initial failed operation have a missed gland that is accessible through a cervical incision. Reoperative parathyroid surgery carries a substantially higher risk of injury to the recurrent laryngeal nerve and of hypocalcemia due to postoperative scarring and disruption of normal tissue planes.

A. Localizing studies

1. **Preoperative localization** of the hyperfunctioning parathyroid should be attempted in nearly all reoperative cases by ^{99m}Tc-sestamibi scintigraphy and ultrasound or CT scanning. These noninvasive studies are successful in localizing the missed gland in 25–75% of cases (*Radiology* 162:133, 1987). With combined use of CT scan, ultrasonography, and scintigraphy, at least one imaging study identifies the tumor in more than 75% of patients. Noninvasive localizing studies must be interpreted in the context of the operative note. Invasive imaging tests, including angiography, are associated with greater morbidity and measurable mortality; they should be reserved for complex cases with negative or equivocal noninvasive studies. The success of these tests in localizing the concealed tumor is highly dependent on the skill and experience of the radiologist (*Radiology* 162:138, 1987). Invasive studies are most helpful to identify rare glands located outside the patient's neck (e.g., mediastinum).
- B. **Operative strategy.** The goal of reexploration is an orderly search based on the information gained from the initial operation and from localization studies.
1. **Missed parathyroid glands** are found either in the usual position or in ectopic sites, as determined by the embryology of parathyroid development. Rarely, a parathyroid gland is intrathyroidal (especially in patients with multinodular goiter), and intraoperative thyroid ultrasound followed by thyroid lobectomy can be performed if an exhaustive search fails to identify the parathyroid adenoma. If four normal glands have been located, the adenoma is likely to represent a supernumerary (fifth) gland. Intraoperative ultrasound is an effective tool for localizing parathyroid glands.
 2. **Mediastinal adenomas** within the thymus are managed by resecting the cranial portion of the thymus by gentle traction on the thyro-thymic ligament or by a complete transcervical thymectomy using a specialized substernal retractor (*Ann Surg* 214:555, 1991). Median sternotomy carries a higher morbidity and increased postoperative pain, and the possibility of this should be discussed with the patient preoperatively.

III. Parathyroid autotransplantation

- A. Indications for **total parathyroidectomy and heterotopic parathyroid autotransplantation** include HPT in patients with renal failure, in patients with four-gland parathyroid hyperplasia, and in patients undergoing neck reexploration in which the adenoma is the only remaining parathyroid tissue. Parathyroid autotransplantation may be performed into either the sternocleidomastoid muscle or the brachioradialis muscle of the patient's nondominant forearm. Parathyroid grafting into the patient's forearm is advantageous if recurrent HPT is possible (e.g., MEN type 1 or 2A). If HPT recurs, the hyperplastic parathyroid tissue may be partially excised from the patient's forearm under local anesthesia.
- B. **Technique.** A freshly removed parathyroid gland is cut into 40–50 pieces approximately 1 mm by 1 mm by 2 mm and placed in sterile iced saline. An incision is made in the patient's nondominant forearm, and separate intramuscular beds are created by spreading the fibers of the brachioradialis with a fine forceps. Approximately four to five pieces are placed in each site, and a total of 15–20 pieces of parathyroid tissue are transplanted. The beds are closed with a silk suture to mark the site of the transplanted tissue. Transplanted parathyroid tissue begins to function within 14–21 days of surgery.
- C. **Cryopreservation of parathyroid glands** is performed in MEN patients and all patients who may become aparathyroid after repeat exploration. Autotransplantation may be performed by freezing the tissue in vials containing 10% dimethyl sulfoxide, 10% autologous serum, and 80% Waymouth's medium. Cryopreserved parathyroid tissue can be used for autotransplantation in patients who become aparathyroid or in patients with failure of the initial grafted parathyroid tissue.

IV. Postoperative hypocalcemia

- A. **Transient hypocalcemia** commonly occurs after total thyroidectomy or parathyroidectomy and requires treatment if severe (total serum calcium <7.0 mg/dL) or the patient is symptomatic. Chvostek's sign (twitching of the facial muscles when the examiner percusses over the facial nerve anterior to the patient's ear) is a

sign of relative hypocalcemia but is present in up to 15% of the normal population. This sign is not necessarily an indication for calcium replacement.

- B. Patients with **persistent hypocalcemia** after total thyroidectomy or after parathyroid autotransplantation can require continued supplementation for 6–8 weeks postoperatively. Usually, patients are given calcium carbonate, 250–500 mg p.o. q.i.d., and 1,25-dihydroxyvitamin D₃, 0.25–2.00 µg orally per day.
- C. **Hypocalcemic tetany** is a medical emergency that is treated with rapid intravenous administration of 10% calcium gluconate or calcium chloride until the patient recovers. Specifically, 1–2 ampules (10–20 mL) of 10% calcium gluconate are given intravenously over 10 minutes, which may be repeated every 15–20 minutes, as required. Subsequently, a continuous infusion of 10% calcium gluconate (90 mg elemental calcium/10 mL), 60 mL in 500 mL D5W (1 mg/mL), is initiated at 0.5–2.0 mg/kg per hour to maintain the serum calcium at 8–9 mg/dL. Patients with severe hypocalcemia also must have correction of hypomagnesemia.

V. Parathyroid carcinoma is rare and accounts for less than 1% of patients with HPT. Approximately 50% of these patients have a palpable neck mass, and serum calcium levels may exceed 15 mg/dL.

- A. **Diagnosis** is made by the histologic finding of vascular or capsular invasion, lymph node or distant metastases, or gross invasion of local structures.
- B. **Surgical treatment** is radical local excision of the tumor, surrounding soft tissue, lymph nodes, and ipsilateral thyroid lobe when the disease is recognized preoperatively or intraoperatively. Reoperation is indicated for local recurrence in an attempt to control malignant hypercalcemia.
- C. Patients with parathyroid carcinoma and some patients with benign HPT may develop **hyperparathyroid crisis**. Symptoms of this acute, sometimes fatal, illness include profound muscular weakness, nausea and vomiting, drowsiness, and confusion. Hypercalcemia (16–20 mg/dL) and azotemia are usually present. Ultimate treatment of “parathyroid crisis” is parathyroidectomy; however, hypercalcemia and volume and electrolyte abnormalities should be addressed first. Treatment is warranted for symptoms or a serum calcium level greater than 12 mg/dL. First-line therapy is infusion of 300–500 mL per hour of 0.9% sodium chloride (5–10 L per day i.v.) to restore intravascular volume and to promote renal excretion of calcium. After urinary output exceeds 100 mL per hour, furosemide (80–100 mg i.v. every 2–6 hours) may be given to promote further renal sodium and calcium excretion. Thiazide diuretics impair calcium excretion and should be avoided. Hypokalemia and hypomagnesemia are complications of forced saline diuresis and should be corrected. If diuresis alone is unsuccessful in lowering the serum calcium, other calcium-lowering agents may be used. These include the **bisphosphonates**, pamidronate (60–90 mg in 1 L 0.9% saline infused over 24 hours), or etidronate (7.5 mg/kg i.v. over 2–4 hours daily for 3 days); **mithramycin**, 25 µg/kg i.v. over 4–6 hours daily for 3–4 days (malignant hypercalcemia only); and salmon **calcitonin** (initial dose, 4 IU/kg s.c. or i.m. every 12 hours, increasing as necessary to maximum dose of 8 IU/kg s.c. or i.m. every 6 hours). Orthophosphate, gallium nitrate, and glucocorticoids also have calcium-lowering effects.

Endocrine Pancreas

Pancreatic islet cell tumors are rare tumors that produce clinical syndromes related to the specific hormone secreted. Insulinomas are the most common of these tumors, followed by gastrinoma, then the rarer VIPoma (vasoactive intestinal polypeptide-secreting tumor), glucagonoma, and somatostatinoma. Recognition of **characteristic syndromes** is key to the diagnosis, which **must be confirmed biochemically**. Islet cell tumors are often occult and their localization may be difficult, especially for small, multifocal, or extrapancreatic tumors. Islet cell tumors may occur sporadically or as a component of MEN type 1 or von Hippel-Lindau disease (nearly always multifocal). Islet cell tumors may be benign or malignant, although prediction may be based on hormone produced rather than tumor size.

I. Insulinoma

- A. **Clinical features.** Patients with insulinoma develop profound hypoglycemia during fasting or after exercise. The clinical picture includes the signs and symptoms of neuroglycopenia (anxiety, tremor, confusion, and obtundation) and the sympathetic response to hypoglycemia (hunger, sweating, and tachycardia). These bizarre complaints initially may be attributed to malingering or a psychosomatic etiology unless the association with fasting is recognized. Many patients eat excessively to avoid symptoms, causing significant weight gain. **Whipple's triad** refers to the clinical criteria for the diagnosis of insulinoma: (1) hypoglycemic symptoms during monitored fasting, (2) blood glucose levels less than 50 mg/dL, and (3) relief of symptoms after administration of intravenous glucose. Factitious hypoglycemia (excess exogenous insulin administration) and postprandial reactive hypoglycemia must be excluded.
- B. A supervised, in-hospital **72-hour fast** is required to diagnose insulinoma. Patients are observed for hypoglycemic episodes and have 6-hour measurement of plasma glucose, insulin, proinsulin, and C peptide. During the fast, the patient is allowed noncaloric beverages, and the fast is terminated when symptoms of neuroglycopenia develop. Nearly all patients with insulinoma develop neuroglycopenic symptoms and have inappropriately elevated plasma insulin (>5 µU/mL) associated with hypoglycemia (glucose <50 mg/dL). Elevated levels of C peptide and proinsulin usually are present as well (*Curr Probl Surg* 31:79, 1994).
- C. **Localization.** Insulinomas typically are small (<2 cm), solitary, benign tumors that may occur anywhere in the pancreas. Rarely, an insulinoma may develop in extrapancreatic rests of pancreatic tissue. Dynamic CT scanning at 5-mm intervals with oral and intravenous contrast is the initial localizing test for insulinoma, with success in 35–85% of cases. Endoscopic ultrasound is also effective but is operator dependent (*N Engl J Med* 326:1721, 1992). Indium-111 (¹¹¹In)-octreotide scintigraphy is less effective (approximately 50%) for localization of insulinoma than other islet cell tumors because insulinomas typically have few somatostatin receptors. Selective arteriography with observation of a tumor “blush” is the single best diagnostic study for the primary tumor and hepatic metastases. If a tumor is still not identified, regional localization to the head, body, or tail of the pancreas can be accomplished by portal venous sampling for insulin or by calcium angiography. Calcium angiography involves injection of calcium into selectively catheterized pancreatic arteries and measurement of plasma insulin through a catheter positioned in a hepatic vein.
- D. **Treatment of insulinoma is surgical in nearly all cases.** Surgical management of insulinomas consists of localization of the tumor by careful inspection and palpation of the gland after mobilization of the duodenum and the inferior border of the pancreas. Use of intraoperative ultrasonography greatly facilitates identification of small tumors, especially those located in the pancreatic head or uncinate process. Most insulinomas can be enucleated from surrounding pancreas, although those in the body or tail may require resection. In general, blind pancreatectomy should not be performed when the tumor cannot be identified. Approximately 5% of insulinomas are malignant, and 10% are multiple (usually in association with MEN type 1). Medical treatment for insulinoma with diazoxide, verapamil, or octreotide has limited effectiveness but may be used in preparation for surgery or for patients unfit for surgery.

II. Gastrinoma

- A. Patients with **gastrinoma and the Zollinger-Ellison syndrome (ZES)** have severe peptic ulcer disease (PUD) due to gastrin-mediated gastric acid hypersecretion. Most patients present with epigastric pain, and 80% have active duodenal ulceration at the time of diagnosis. Diarrhea and weight loss are common (40% of patients). ZES is uncommon (0.1–1.0% of PUD), and most patients present with typical duodenal ulcer. Gastrinoma and ZES should be considered in any patient with (1) PUD refractory to treatment for *Helicobacter pylori* and conventional doses of H₂ blockers or omeprazole; (2) recurrent, multiple, or atypically located (e.g., distal duodenum or jejunum) peptic ulcers; (3) complications of PUD (i.e., bleeding, perforation, or obstruction); (4) PUD with significant diarrhea; and (5) PUD with HPT, nephrolithiasis, or familial endocrinopathy. All patients considered for elective surgery for PUD should have ZES excluded preoperatively.
- B. **Diagnosis of ZES requires demonstration of fasting hypergastrinemia and basal gastric acid hypersecretion.** A fasting serum gastrin level of 100 pg/mL or greater with basal gastric acid output (BAO) of 15 mEq per hour or more (>5 mEq per hour in patients with previous ulcer surgery) secures the diagnosis of ZES in nearly all cases. Fasting hypergastrinemia without elevated basal gastric acid output is seen in atrophic gastritis, renal failure, and in patients taking H₂-receptor antagonists or omeprazole. Fasting hypergastrinemia with elevated basal gastric acid output is seen in retained gastric antrum syndrome, gastric outlet obstruction, and in antral G-cell hyperplasia. A **secretin stimulation test** is used to distinguish ZES from these conditions. This test is performed by measuring fasting serum gastrin levels before and 2, 5, 10, and 15 minutes after the intravenous administration of secretin (2 units/kg). Eighty-five percent of patients with ZES have an increase in gastrin levels (>200 pg/mL over baseline) in response to a secretin stimulation test, whereas patients with other conditions do not. This test is most useful when ZES is suspected in patients who have had prior gastric surgery and in patients with moderately increased (100–1,000 pg/mL) fasting gastrin levels.
- C. **Localization of gastrinoma** should be performed in all patients considered for surgery. Approximately 80% of gastrinomas are located within the “gastrinoma triangle” that includes the duodenum and head of the pancreas. Gastrinomas are often malignant, with spread to lymph nodes or liver occurring in up to 60% of cases. Approximately 20% of patients with ZES have familial MEN type 1; these patients often have multiple, concurrent islet cell tumors. Dynamic CT scanning, ¹¹¹In-octreotide scintigraphy, endoscopic ultrasound, and MR scan are useful noninvasive tests to localize gastrinoma; however, preoperative localization is unsuccessful up to 50% of the time. Selective angiography with or without secretin injection of the gastroduodenal, superior mesenteric, and splenic arteries and measurement of hepatic vein gastrin can localize occult gastrinoma in up to 70–90% of cases.
- D. **Medical treatment** of gastric hyperacidity with H₂ histamine receptor antagonists and omeprazole is highly effective in ZES. These medications are indicated preoperatively in patients undergoing operation for cure and in patients with unresectable or metastatic gastrinoma. Increased doses of oral histamine receptor antagonists can be required and often are given at 6-hour intervals. Starting doses (cimetidine, 300 mg p.o. every 6 hours; ranitidine, 150 mg p.o. every 6 hours; famotidine, 20 mg p.o. every 6 hours) are increased until the gastric acid secretion is less than 10 mEq per hour before the next dose. Alternatively, a continuous intravenous infusion can be initiated (e.g., ranitidine, 0.5 mg/kg per hour) and increased until control of gastric acid secretion is achieved.

Omeprazole is given by mouth or nasogastric tube at 20 mg per day or b.i.d.

- E. **Surgical management of ZES** is indicated in all fit patients with nonmetastatic, sporadic gastrinoma. Goals of surgery include precise localization and curative resection of the tumor. Resection of primary gastrinoma alters the malignant progression of tumor and decreases hepatic metastases in patients with ZES. Intraoperative localization of gastrinomas is facilitated by extended duodenotomy and palpation, intraoperative ultrasonography, or endoscopic duodenal transillumination. Gastrinomas within the duodenum, pancreatic head, or uncinate process are treated by enucleation, whereas tumors in the body or tail of the pancreas can be removed by distal or subtotal pancreatectomy. Immediate cure rates are 40–90% in experienced hands; however, half of patients initially cured biochemically experience recurrence within 5 years. **Gastric acid hypersecretion is controllable with H₂ blockers or omeprazole in nearly all patients with ZES, rendering gastrectomy unnecessary.** If a gastrinoma cannot be localized intraoperatively, a parietal cell vagotomy may be performed. Surgical debulking of metastatic or unresectable primary gastrinoma facilitates medical treatment and prolongs life expectancy in select patients. Patients with ZES and MEN 1 most often cannot be cured surgically and usually are treated medically.

III. Unusual islet cell tumors

- A. **VIPomas** secrete vasoactive intestinal peptide and cause profuse secretory diarrhea (fasting stool output of >1 L per day), hypokalemia, and either achlorhydria or hypochlorhydria (watery diarrhea, hypokalemia, and achlorhydria or Verner-Morrison syndrome). Hyperglycemia, hypercalcemia, and cutaneous flushing may be seen. Other, more common causes of diarrhea and malabsorption must be excluded. A diagnosis of VIPoma is established by the finding of elevated fasting serum vasoactive intestinal peptide levels (>190 pg/mL) and secretory diarrhea in association with an islet cell tumor. Octreotide (150 µg s.c. every 8 hours) is highly effective to control the diarrhea and correct electrolyte abnormalities before resection. Most VIPomas occur in the distal pancreas and are amenable to distal pancreatectomy. Metastatic disease is commonly encountered (50%); nevertheless, surgical debulking is indicated to alleviate symptoms.
- B. **Glucagonomas** secrete excess glucagon and result in type II diabetes, hypoglycemia, anemia, weight loss, and a characteristic skin rash, necrolytic migratory erythema. Diagnosis is suggested by symptoms and by biopsy of the skin rash but is confirmed by elevated plasma glucagon levels (usually >1,000 pg/mL). Tumors are large and are readily seen on CT scan. Resection is indicated in fit patients after nutritional support, even if metastases are present.
- C. **Somatostatinomas** are the rarest of the islet cell tumors and cause a syndrome of diabetes, steatorrhea, and cholelithiasis. These tumors are frequently located in the head of the pancreas and are often metastatic at the time of presentation.
- D. **Other rare islet cell tumors** include pancreatic polypeptide-, neurotensin-, and adrenocorticotrophic hormone (ACTH)-secreting tumors, as well as nonfunctioning islet cell tumors. These tumors usually are large and often malignant. Treatment is surgical resection.

Carcinoid Tumors

Carcinoid tumors are classified according to their embryologic origin: foregut (bronchial, thymic, gastroduodenal, and pancreatic), midgut (jejunal, ileal, appendiceal, right colic), and hindgut (distal colic, rectal). Depending on the site of origin, carcinoids secrete hormones differently and have different clinical features. Carcinoid tumors most frequently occur in the gastrointestinal tract. Bronchial and thymic carcinoids occur less commonly. In general, the diagnosis of carcinoid rests on the finding of elevated circulating serotonin or urinary metabolites [5-hydroxyindoleacetic acid (5-HIAA)] and localizing studies. The single best biochemical test is an elevated urinary 5-HIAA (normal, 2–8 mg per 24 hours). Rectal or jejunoileal tumors may be visualized by contrast studies, whereas bronchial carcinoids can be identified on chest X-rays, CT scans, or bronchoscopy. Abdominal or hepatic metastases are best identified by CT scanning, ultrasonography, or angiography. As with other neuroendocrine tumors, some carcinoids can be detected with metaiodobenzylguanidine (¹³¹I-MIBG) scanning, and most are detectable by ¹¹¹In-octreotide scintigraphy.

I. **Carcinoid of the appendix** is by far the most common carcinoid tumor, occurring in up to one in 300 appendectomies. The risk of lymph node metastases and the prognosis of appendiceal carcinoids depends on the size: Tumors less than 1 cm never metastasize, tumors 1–2 cm have a 1% risk of metastasis, and tumors larger than 2 cm have a 30% risk of metastasis (*World J Surg* 20:183, 1996). Extent of surgery for appendiceal carcinoid is based on size: simple appendectomy for tumors less than 1 cm, right hemicolectomy for tumors larger than 2 cm, and selective right hemicolectomy for tumors 1–2 cm. Prognosis for completely resected appendiceal carcinoid is favorable, with 5-year survival of 90–100%.

II. **Small-intestinal carcinoid tumors** usually present with vague abdominal symptoms that uncommonly lead to preoperative diagnosis. Most patients are operated on for intestinal obstruction, which is caused by a desmoplastic reaction in the mesentery around the tumor rather than by the tumor itself. Extended resection, including the mesentery and lymph nodes, is required, even for small tumors. Meticulous examination of the remaining bowel is mandatory because tumors are multicentric in 20–40% of cases, and synchronous adenocarcinomas are found up to 10% of the time. An almost linear relationship exists between size of tumor and risk of nodal metastases, with up to 85% for tumors larger than 2 cm. Prognosis depends on size and extent of disease; overall survival is 50–60%, which is substantially decreased if liver metastases are present. Small-bowel carcinoids have the highest propensity to metastasize to liver and produce the carcinoid syndrome.

III. **Rectal carcinoids** are typically small, submucosal nodules that are often asymptomatic or produce nonspecific symptoms of bleeding, constipation, or tenesmus. These tumors are hormonally inactive and almost never produce the carcinoid syndrome, even when spread to the liver occurs. Treatment of small (<1 cm) rectal carcinoids is endoscopic removal. Transmural excision of tumors 1–2 cm can be done locally. Treatment of 2-cm and larger tumors or invasive tumors is controversial but may include anterior or abdominoperineal resection for fit patients without metastases.

IV. **Foregut carcinoids** include gastroduodenal, bronchial, and thymic carcinoids. These are a heterogeneous group of tumors with variable prognosis. They do not release serotonin and may produce atypical symptoms (e.g., violaceous flushing of the skin) related to release of histamine. Gastroduodenal carcinoids may produce gastrin and cause ZES. Resection is advocated for localized disease.

V. The **carcinoid syndrome** occurs in less than 10% of patients with carcinoid and develops when venous drainage from the tumor gains access to the systemic circulation, as with hepatic metastases. The classic syndrome consists of flushing, diarrhea, bronchospasm, and right-sided cardiac valvular fibrosis. Symptoms are paroxysmal and may be provoked by alcohol, cheese, chocolate, or red wine. Diagnosis is made by 24-hour measurement of urinary 5-HIAA or of whole blood 5-hydroxytryptamine. Surgical cure usually is not possible with extensive abdominal or hepatic metastases; however, debulking of the tumor may alleviate symptoms and improve survival, when it can be performed safely. Hepatic metastases also have been treated with chemoembolization using doxorubicin, 5-fluorouracil, and cisplatin. **Carcinoid crisis** with severe bronchospasm and hemodynamic collapse may occur perioperatively in patients with undiagnosed carcinoid. Prompt recognition is crucial, as administration of octreotide (100 µg i.v.) can be lifesaving.

Adrenal-Pituitary Axis

I. Adrenal cortex

- A. **Cushing's syndrome** results from exogenous steroid administration or excess endogenous cortisol secretion. The clinical manifestations of Cushing's syndrome include hypertension, edema, muscle weakness, glucose intolerance, osteoporosis, easy bruising, cutaneous striae, and truncal obesity (buffalo hump, moon facies). Women may develop acne, hirsutism, and amenorrhea as a result of adrenal androgen excess.
1. **Pathophysiology**
 - a. The most common cause of Cushing's syndrome is **iatrogenic**, resulting from administration of exogenous glucocorticoids or ACTH.
 - b. Hypersecretion of ACTH from the anterior pituitary gland (**Cushing's disease**) is the most common pathologic cause (65–70% of cases) of endogenous hypercortisolism. The adrenal glands respond normally to the elevated ACTH, resulting in bilateral adrenal hyperplasia. Excessive release of corticotropin-releasing factor by the hypothalamus is a rare cause of hypercortisolism.
 - c. Abnormal secretion of cortisol from a primary adrenal adenoma or carcinoma is the cause of hypercortisolism in 10–20% of cases. **Primary adrenal neoplasms** secrete corticosteroids independently of ACTH and usually result in suppressed plasma ACTH levels and atrophy of the adjacent and contralateral adrenocortical tissue.
 - d. In approximately 15% of cases, Cushing's syndrome is caused by **ectopic secretion of ACTH** or an ACTH-like substance from a small-cell bronchogenic carcinoma, carcinoid tumor, pancreatic carcinoma, thymic carcinoma, medullary thyroid cancer, or other neuroendocrine neoplasm. Patients with ectopic ACTH-secreting neoplasms can present primarily with hypokalemia, glucose intolerance, and hyperpigmentation but with few other chronic signs of Cushing's syndrome.
 2. **Diagnosis** of Cushing's syndrome is biochemical. Goals are to first establish hypercortisolism and then identify the source. However, all of the available tests are complicated by a lack of specificity and an overlap in the biochemical responses of patients with the different disease states.

- a. **Establishing presence of hypercortisolism**
 1. The best **screening test for hypercortisolism** is measurement of the urinary excretion of free cortisol. Urinary excretion of more than 100 µg per day of free cortisol in two independent collections is virtually diagnostic of Cushing's syndrome. Measurement of plasma cortisol level alone is not reliable to diagnose Cushing's syndrome due to overlap of the levels in normal and abnormal patients.
 2. An **overnight dexamethasone suppression test** (dexamethasone, 1 mg p.o. at 11 p.m., and measurement of plasma cortisol at 8 a.m.) is used to confirm Cushing's syndrome, especially in obese or depressed patients who may have marginally elevated urinary cortisol. Patients with true hypercortisolism have lost normal adrenal-pituitary feedback and usually fail to suppress the morning plasma cortisol level to less than 5 µg/dL.
 - b. **Localization of the cause of hypercortisolism**
 1. **Determination of basal ACTH by immunoradiometric assay** is the best test to determine the cause of hypercortisolism. Suppression of the absolute level of ACTH below 5 pg/mL is nearly diagnostic of adrenocortical neoplasms. ACTH levels in Cushing's disease may range from the upper limits of normal (15 pg/mL) to 500 pg/mL. Highest plasma levels of ACTH (>1,000 pg/mL) have been observed in patients with ectopic ACTH syndrome.
 2. Standard **high-dose dexamethasone suppression testing** is used to discern a pituitary from an ectopic source of ACTH. Normal individuals and most patients with a pituitary ACTH-producing neoplasm respond to a high-dose dexamethasone suppression test (2 mg p.o. every 6 hours for 48 hours) with a reduction of urinary free cortisol and urinary 17-hydroxysteroids to less than 50% of basal values. Most patients with a primary adrenal tumor or an ectopic source of ACTH production fail to suppress to this level. However, this test does not separate clearly pituitary and ectopic ACTH hypersecretion because 25% of patients with the ectopic ACTH syndrome also have suppressible tumors.
 3. Additional tests that may be useful include the **metyrapone test** (an inhibitor of the final step of cortisol synthesis) and the **corticotropin-releasing factor infusion test**. Patients with pituitary hypersecretion of ACTH respond to these tests with a compensatory rise in ACTH and urinary 17-hydroxysteroids, whereas patients with a suppressed hypothalamic-pituitary axis (primary adrenal tumor, ectopic ACTH syndrome) usually do not have a compensatory rise.
 3. **Imaging tests** are useful to identify lesions suspected on the basis of biochemical testing. Reliance on radiologic studies to diagnose the cause of Cushing's syndrome should be discouraged.
 - a. **Gadolinium-enhanced MR scan** of the sella turcica is the best imaging test for pituitary adenomas suspected of causing ACTH-dependent hypercortisolism. Patients with ACTH-independent hypercortisolism require thin-section **CT scan or MR scan of the adrenal gland**, both of which identify adrenal abnormalities with more than 95% sensitivity. Patients with ACTH-dependent hypercortisolism and either markedly elevated ACTH or a negative pituitary MR scan should have **CT scan of the chest** to identify a tumor-producing ectopic ACTH.
 - b. Bilateral **inferior petrosal sinus sampling** can delineate unclear cases of Cushing's disease from other causes of hypercortisolism. Simultaneous bilateral petrosal sinus and peripheral blood samples are obtained before and after peripheral intravenous injection of 1 µg/kg corticotropin-releasing hormone. A ratio of inferior petrosal sinus to peripheral plasma ACTH of 2.0 at basal or of 3.0 after corticotropin-releasing hormone administration is 100% sensitive and specific for pituitary adenoma.
 4. **Surgical treatment of Cushing's syndrome** involves removing the cause of cortisol excess (a primary adrenal lesion or pituitary or ectopic tumors secreting excessive ACTH). Transsphenoidal resection of an ACTH-producing pituitary tumor is successful in 80% or more of cases of Cushing's disease. Treatment of ectopic ACTH syndrome involves resection of the primary lesion, if possible. Primary adrenal causes of Cushing's syndrome are treated by removal of the adrenal gland containing the tumor. All patients who undergo adrenalectomy for primary adrenal causes of Cushing's syndrome require perioperative and postoperative glucocorticoid replacement because the pituitary-adrenal axis is suppressed.
- B. Primary aldosteronism** (Conn's syndrome) is a syndrome of hypertension and hypokalemia caused by hypersecretion of the mineralocorticoid aldosterone. This uncommon syndrome accounts for less than 1% of unselected patients with hypertension. An aldosterone-producing adrenal adenoma (APA) is the cause of primary aldosteronism in two-thirds of cases and is one of the few surgically correctable causes of hypertension. Idiopathic bilateral adrenal hyperplasia (IHA) causes 30–40% of cases of primary aldosteronism. Adrenocortical carcinoma and autosomal dominant glucocorticoid-suppressible aldosteronism are rare causes of primary aldosteronism. **Secondary aldosteronism** is a physiologic response of the renin-angiotensin system to renal artery stenosis, cirrhosis, congestive heart failure, and normal pregnancy. In these conditions, the adrenal gland functions normally.
1. **Diagnosis.** Aldosterone-mediated retention of sodium and excretion of potassium and hydrogen ion by the kidney causes **hypokalemia and moderate diastolic hypertension**. Edema is characteristically absent. Laboratory diagnosis of primary aldosteronism requires demonstration of hypokalemia (<3.5 mEq/L), inappropriate kaliuresis (>30 mEq per day), and elevated aldosterone (>15 ng/dL) with normal cortisol. Upright plasma renin activity (PRA) of less than 3 ng/mL per hour corroborates the diagnosis. A ratio of plasma aldosterone (ng/dL) to PRA (ng/mL per hour) of greater than 20–25 further suggests primary hyperaldosteronism. Confirmation of primary aldosteronism involves determination of serum potassium; PRA; and a 24-hour urine collection for sodium, cortisol, and aldosterone after 5 days of a high-sodium diet. Patients with primary hyperaldosteronism do not demonstrate aldosterone suppressibility (>14 µg per 24 hours) after salt loading. Alternatively, plasma aldosterone and PRA can be measured before and 2 hours after oral administration of 25 mg of captopril. Failure to suppress plasma aldosterone to less than 15 ng/dL is a positive test. Before biochemical studies, all diuretics and antihypertensives are discontinued for 2–4 weeks, and a daily sodium intake of at least 100 mEq is provided. **Differentiation between adrenal adenoma and IHA is important** because unilateral adenomas are treated by surgical excision, whereas bilateral hyperplasia is treated medically. Because suppression of the renin-angiotensin system is more complete in APA than in IHA, an imperfect separation (approximately 85% accuracy) of these two disorders is provided by measuring plasma aldosterone and PRA after overnight recumbency and then after 4 hours of upright posture. Patients with IHA usually have an increase in PRA and aldosterone in response to upright posture, whereas in patients with adenoma, PRA usually remains suppressed, and aldosterone does not change or falls paradoxically. In practice, this test usually is not necessary because, after a biochemical diagnosis of primary hyperaldosteronism, sensitive imaging tests are used to localize the lesion or lesions.
 2. **Localization.** High-resolution adrenal CT scan should be the initial step in localization of an adrenal tumor. CT scanning localizes an adrenal adenoma in 90% of cases overall, and the presence of a unilateral adenoma larger than 1 cm on CT scan and supportive biochemical evidence of an aldosteronoma are generally all that is needed to make the diagnosis of Conn's syndrome. Uncertainty regarding APA versus IHA after biochemical testing and noninvasive localization may be definitively settled by bilateral adrenal venous sampling for aldosterone and cortisol. Simultaneous adrenal vein blood samples for aldosterone and cortisol are taken: A ratio of aldosterone to cortisol is greater than 4:1 for a diagnosis of aldosteronoma or less than 4:1 for IHA.
 3. **Treatment.** Surgical removal of an APA through a posterior or laparoscopic approach results in immediate cure or substantial improvement of hypertension and hyperkalemia in more than 90% of patients with Conn's syndrome. The patient should be treated with spironolactone (200–400 mg per day) preoperatively for 2–3 weeks to control blood pressure and to correct hypokalemia. Patients with IHA should be treated medically with spironolactone (200–400 mg per day). A potassium-sparing diuretic, such as amiloride (5–20 mg per day), and calcium channel blockers have also been used. Surgical excision rarely cures bilateral hyperplasia.
- C. Acute adrenal insufficiency** is an emergency and should be suspected in stressed patients with a history of either adrenal insufficiency or exogenous steroid use. Adrenocortical insufficiency is most often caused by acute withdrawal of chronic corticosteroid therapy but can result from autoimmune destruction of the adrenal cortex, from adrenal hemorrhage (Waterhouse-Friderichsen syndrome), or rarely from infiltration with metastatic carcinoma.
1. **Signs and symptoms** include fever, nausea, vomiting, severe hypotension, and lethargy. Characteristic laboratory findings of adrenal insufficiency include hyponatremia, hyperkalemia, azotemia, and fasting or reactive hypoglycemia.
 2. **Treatment of adrenal crisis must be immediate, based on clinical suspicion**, before laboratory confirmation is available. Intravenous volume replacement with normal or hypertonic saline and dextrose is essential, as is immediate intravenous steroid replacement therapy with **4 mg dexamethasone**. Then, a rapid ACTH stimulation test is used to test for adrenal insufficiency. Synthetic ACTH (250 µg) is administered intravenously and plasma cortisol levels are measured at 0, 30, and 60 minutes later. Normal peak cortisol response should exceed 20 µg/dL. Thereafter, 100 mg of hydrocortisone is administered intravenously every 6–8 hours and is tapered to standard replacement doses as the patient's condition stabilizes. Subsequent recognition and treatment of the underlying cause, particularly if it is infectious, usually resolve the crisis. Mineralocorticoid replacement is not required until intravenous fluids are discontinued and oral intake resumes.
 3. **Prevention.** Patients who have known adrenal insufficiency or have received suprathreshold doses of steroid for at least 1 week in the year preceding surgery should receive 100 mg of hydrocortisone the evening before and the morning of major surgery followed by 100 mg of hydrocortisone every 8 hours during the perioperative 24 hours.

II. Adrenal medulla: pheochromocytoma

- A. **Pathophysiology.** Pheochromocytomas are neoplasms derived from the chromaffin cells of the sympathoadrenal system that result in unregulated, episodic oversecretion of catecholamines. Most pheochromocytomas secrete predominantly norepinephrine and smaller amounts of epinephrine. Rarely, epinephrine is secreted predominantly or exclusively.
- B. **Clinical features.** Approximately 80–85% of pheochromocytomas in adults arise in the adrenal medulla, whereas 10–15% arise in the extraadrenal chromaffin tissue, including the paravertebral ganglia, posterior mediastinum, organ of Zuckerkandl, or urinary bladder. Symptoms of pheochromocytoma are related to excess sympathetic stimulation from catecholamines and include paroxysms of pounding frontal headache, diaphoresis, palpitations, flushing, or anxiety. The most common sign is episodic or sustained hypertension, but pheochromocytoma accounts for only 0.1–0.2% of patients with sustained diastolic hypertension. Uncommonly, patients present with complications of prolonged uncontrolled hypertension (e.g., myocardial infarction, cerebrovascular accident, or renal disease). Pheochromocytomas can occur in association with several hereditary syndromes that include MEN types 2A and 2B and von Hippel-Lindau syndrome.

Tumors that arise in familial settings frequently are bilateral.

- C. The **biochemical diagnosis** of pheochromocytoma is made by demonstrating elevated 24-hour urinary excretion of catecholamines and their metabolites (metanephrines, vanillylmandelic acid). If possible, antihypertensive medications (especially monoamine oxidase inhibitors) should be discontinued before the 24-hour urine collection, and creatinine excretion should be measured simultaneously to assess the adequacy of the sample. Plasma catecholamines also are elevated during the paroxysms of hypertension, but they are more difficult to measure and interpret and therefore have limited clinical application.
- D. **Radiographic tests** are used to demonstrate the presence of an adrenal mass.
1. **CT scanning** is the imaging test of choice and identifies 90–95% of pheochromocytomas larger than 1 cm. MR scan can also be useful because T2-weighted images have a characteristic high intensity in patients with pheochromocytoma and metastatic tumor compared with adenomas.
 2. **Scintigraphic scanning** after the administration of ¹³¹I-MIBG provides a functional and anatomic test of hyperfunctioning chromaffin tissue. MIBG scanning is very specific for both intra- and extraadrenal pheochromocytomas.
- E. **The treatment of benign and malignant pheochromocytomas is surgical excision.**
1. **Preoperative preparation** includes administration of an alpha-adrenergic blocker to control hypertension and to permit reexpansion of intravascular volume. Phenoxybenzamine, 10 mg p.o. b.i.d., is initiated and increased to 20–40 mg p.o. b.i.d. until the desired effect or prohibitive side effects are encountered. Postural hypertension is expected and is the desired end-point. Beta-adrenergic blockade (e.g., propranolol) may be added if tachycardia or arrhythmias develop but only **after complete alpha-adrenergic blockade**. Patients with cardiopulmonary dysfunction may require a pulmonary artery (Swan-Ganz) catheter perioperatively, and all patients should be monitored in the surgical intensive care unit in the immediate postoperative period.
 2. **The classic operative approach** for familial pheochromocytomas is exploration of both adrenal glands, the preaortic and paravertebral areas, and the organ of Zuckerkandl through a midline or bilateral subcostal incision. In patients with MEN type 2A or 2B and a unilateral pheochromocytoma, it is acceptable policy to remove only the involved gland (*Ann Surg* 217:595, 1993). In patients with a sporadic, unilateral pheochromocytoma localized by preoperative imaging studies, adrenalectomy may be performed by an anterior or posterior approach or (increasingly) by laparoscopic adrenalectomy. Intraoperative labile hypertension can occur during resection of pheochromocytoma. This can be prevented by minimal manipulation of the tumor but can be controlled most effectively with intravenous sodium nitroprusside (0.5–10.0 µg/kg per minute) or phentolamine (5 mg).

III. Adrenocortical carcinoma is a rare but aggressive malignancy; most patients with this cancer present with locally advanced disease. Syndromes of adrenal hormone overproduction may include rapidly progressive hypercortisolism, hyperaldosteronism, or virilization. Large (>6 cm) adrenal masses that extend to nearby structures on CT scanning likely represent carcinoma. **Complete surgical resection of locally confined tumor is the only chance for cure of adrenocortical carcinoma.** Definitive diagnosis of adrenocortical carcinoma requires operative and pathologic demonstration of nodal or distant metastases. Any adrenal neoplasm weighing more than 50 g should be considered malignant. Often, patients with adrenocortical carcinoma present with metastatic disease, most often involving the lung, lymph nodes, liver, or bone. Palliative surgical debulking of locally advanced or metastatic adrenocortical carcinoma may provide these patients with symptomatic relief from some slow-growing, hormone-producing cancers. Chemotherapy with mitotane may be somewhat effective. Overall, the prognosis for patients with adrenocortical carcinoma is poor.

IV. Incidental adrenal masses are detected in 0.6–1.5% of abdominal CT scans obtained for other reasons. Most incidentally discovered adrenal masses are benign, nonfunctioning cortical adenomas of no clinical significance. Because of the high morbidity and relative frequency of pheochromocytoma and hypercortisolism, biochemical testing in asymptomatic, normotensive patients should be limited to 24-hour urine collection for cortisol, catecholamines, vanillylmandelic acid, and metanephrines. Lesions that are solid, homogeneous, and nonfunctioning on endocrine testing and are smaller than 6 cm may be followed conservatively with a repeat CT scan in 3–6 months (*Ann Intern Med* 98:940, 1983). Functioning lesions or nonfunctional tumors larger than 6 cm should be resected. Some authors advocate removal of any adrenal mass, functional or nonfunctional, that is larger than 3.5 cm to avoid late diagnosis of adrenocortical carcinoma (*Surg Gynecol Obstet* 163:203, 1986).

V. Endocrine emergencies are discussed in [Chapter 7](#).

Introduction

Trauma Care

I. Prehospital care

II. Initial hospital care

III. Definitive hospital care

IV. Damage control surgery

V. Miscellaneous aspects of general trauma care

VI. Conclusion

Introduction

Injury remains a leading cause of death and disability around the world. This chapter outlines an overall approach to trauma care, provides a framework for therapy, highlights critical aspects of decision making, and focuses on selected key diagnoses and interventions. These points illustrate the four main features of effective trauma care: (1) comprehensive therapy extending from the initial field evaluation through the completion of rehabilitation; (2) multidisciplinary therapy involving the coordination of a dedicated team of health professionals; (3) systematic therapy providing a framework for the timely and accurate identification of all injuries and comorbidities; and (4) rapid therapy resulting in the proper prioritization of injuries and the interventions required to treat them. The most successful outcomes occur when a knowledgeable and skilled surgeon leads a cohesive team according to these principles. Each member of the team has specific responsibilities, and the collective effort results in the timely identification and treatment of life threats, an accurate and complete injury inventory, and an appropriately prioritized plan of care.

Trauma Care

I. Prehospital care of the trauma patient is provided by a wide range of emergency medical service (EMS) personnel with varying levels of EMS skills training (first responders, emergency medical technicians, and paramedics). These field professionals are responsible for performing the three major functions of prehospital care: (1) assessment of the injury scene; (2) stabilization and monitoring of the injured patient; and (3) safe and rapid transportation of critically ill patients to the appropriate trauma center. The observations and interventions of EMS personnel provide important data that guide the resuscitation of an injured patient. On the arrival of the patient to the trauma center, these data must be communicated to the trauma team in an efficient, organized manner. The **MVIT system** (mechanism, vital signs, injury inventory, treatment) of reporting is one frequently used method that takes between 30 to 45 seconds to complete.

- A. The **mechanism** of a trauma provides important information about the pattern and severity of injuries sustained in the event.
 1. The **pattern of injury** can often be predicted based on the type of trauma. Front-end car collisions can cause direct contact between the driver's knees and the dashboard. Such trauma can result in combination patellar fracture, posterior knee dislocation (with popliteal artery injury), femoral shaft fracture, and posterior rim fracture of the acetabulum. Feet-first falls from significant heights cause axial loading with a possible combination of calcaneal fracture, lower extremity long bone fracture, acetabular injury, and lumbar spine compression fracture. Adult pedestrians struck by motor vehicles often have a pattern of three distinct injuries: (1) injury to the tibia and fibula from striking the bumper; (2) injury to the head from striking the windshield or hood after being swept from the ground; and (3) injury to the upper extremity from falling to the pavement. In young children struck by a motor vehicle, the constellation of injuries is known as Waddell's triad: (1) femur fracture from striking the bumper; (2) abdominal solid organ injury (liver or spleen) from striking the fender; and (3) opposite side head injury from landing on the pavement.
 2. **Injury severity** can be intuited from the mechanism of trauma through estimating the **amount of energy transfer**, which is proportional to the calculation $m(v_i^2 - v_f^2)$, where m is mass, v_i is the initial velocity, and v_f is the final velocity. A pedestrian struck by a motor vehicle at high speed therefore will likely sustain more severe injuries than an athlete hit by a baseball. Even though the differences between the squares of the final and initial velocities of the vehicle and the baseball may be similar, their masses (and hence the energy transfer) are of different orders of magnitude.
- B. **Vital signs**, including level of consciousness and voluntary movement, give insight into the clinical trajectory of the patient. EMS providers typically measure and report these values, often in less-than-ideal conditions. Deterioration of vital signs en route to the trauma center suggests the existence of life threats requiring immediate intervention. Improvement in vital signs en route may reflect transient compensatory responses by the patient. They may, however, only reflect human error in obtaining data in difficult circumstances.
- C. The **injury inventory** consists of the description of injuries as observed by the EMS personnel. The inventory should be considered a minimal list and every aspect of the report must be confirmed or excluded by the trauma team. Important prehospital observations include whether the patient was trapped in a vehicle, crushed under a heavy object, or suffered significant exposure secondary to prolonged extrication. Such findings alert the trauma team to critical secondary injuries, including rhabdomyolysis, traumatic asphyxia, and hypothermia, that can have a profound impact on outcome.
- D. **Treatment.** Prehospital treatment is aimed at stabilization of the injured patient and involves securing an airway, providing adequate ventilation, assessing and supporting circulation, and stabilizing the spine. EMS caregivers fulfill these goals through various therapies that include (but are not limited to) administration of oxygen and intravenous fluids, prevention of heat loss, and immobilization of the spine with a backboard and properly fitting hard cervical collar. Any patient suspected of having injuries to the cervical spine must be placed in a rigid collar. The effects of such prehospital interventions must be taken into account during the initial evaluation of a seemingly stable patient.

II. Initial hospital care. Trauma deaths have a trimodal distribution: (1) immediate death occurring at the time of injury due to devastating wounds; (2) early death occurring within the first few hours of injury due to major intracranial, thoracic, abdominal, pelvic, and extremity injuries; and (3) late death occurring days to weeks after the initial injury due to secondary complications (sepsis, acute respiratory distress syndrome, systemic inflammatory response syndrome, or multiple organ dysfunction and failure). Initial hospital care usually takes place in the emergency department and has two main components: the primary and secondary surveys. The **goal of the primary survey** is to identify and treat those injuries that can result in early death within the first few minutes of injury. The **goal of the secondary survey** is to initiate and maintain the resuscitation of physiologic functions, catalogue all injuries sustained, and institute appropriate therapy or supportive measures.

- A. The **primary survey** is a systematic, rapid evaluation of the injured patient following the mnemonic **ABCDE** (airway, breathing, circulation, disability, exposure). On completion of the survey, the patient should have an established airway with cervical spine control, adequate ventilation and oxygenation, proper intravenous access and control of hemorrhage, an inventory of neurologic status and disability, and complete exposure with environmental control. During the survey, a rudimentary history is obtained (if possible). This history follows the acronym **AMPLE** (allergies, medications, past medical history, past oral intake, and events surrounding the injury).
 1. **Airway.** Establishing a patent airway is the highest priority in the care of a trauma patient, because without one irreversible brain damage can occur within minutes. Because the status of the cervical spine in an injured patient is often unknown, the airway should always be secured under cervical spine control. Engaging the patient in conversation on arrival to the emergency department allows for immediate evaluation of the airway. A patient who is able to respond verbally has a patent airway. A patient who cannot respond verbally must be assumed to have an obstructed airway until proved otherwise. The causes of airway obstruction in the trauma patient can be myriad, and the means of establishing an airway vary from simple maneuvers to emergent surgical procedures. The trauma team must be familiar with every method and its indications. Every trauma patient initially should have oxygen administered (via nasal cannula or bag valve facemask) and an oxygen saturation monitor (i.e., pulse oximeter) placed. An oximeter device is helpful, but it is important to remember that its output readings can be misleading in certain clinical situations (i.e., patients with severe anemia, insufficient pulse pressure, or burn trauma with inhalation injury).
 - a. **Basic maneuvers.** A frequent cause of airway loss in the trauma patient is mechanical obstruction caused by vomitus, phlegm, or other debris in the oropharynx. Simple suctioning can remove such blockage. In the semiconscious or unconscious patient, the tongue itself can occlude the airway. The jaw-thrust maneuver can successfully displace it anteriorly from the pharyngeal inlet, relieving the obstruction. In the unconscious patient, the placement of an oropharyngeal airway (or, in the absence of head trauma, a nasopharyngeal airway) can mechanically displace the tongue anteriorly, securing patency. In the semiconscious patient, the nasopharyngeal airway can be used in certain circumstances to ensure an open airway. Both devices, however, can cause significant irritation of the upper aerodigestive tract with resultant vomiting, and, as a result, they should not be used on fully conscious patients. Even though the above basic maneuvers can be lifesaving by providing temporary airway function, tracheal intubation is the definitive means of securing an airway in the trauma patient.

- b. **Tracheal intubation** is indicated in any patient in whom concern for airway integrity exists (unconscious or semiconscious patients, patients with mechanical obstruction secondary to facial trauma or debris, combative and hypoxic patients). The emergent tracheal intubation of an uncooperative trauma patient is a high-risk undertaking. The most skilled operator available, therefore, should secure the airway by the most expeditious means possible. The preferred method of intubation is via the orotracheal route using rapid sequence induction. Nasotracheal intubation should be discouraged unless the operator is proficient in the technique and the patient does not have any major head or midface injuries. Rapid sequence intubation follows a systematic protocol to ensure successful provision of an airway. Before the arrival of the patient, all the appropriate airway equipment should be tested and placed in an easily accessible location. Induction medications should also be readily available. If intubation is indicated, the patient is first **spontaneously ventilated with 100% oxygen**. During this time, a team member provides in-line cervical spine stabilization to prevent unintentional manipulation as the hard cervical collar is removed anteriorly. Another team member provides anterior pressure on the cricoid cartilage to occlude the esophagus (Sellick maneuver). This pressure prevents aspiration during intubation. Following preoxygenation, a short-acting **sedative or hypnotic medication** is administered via a functioning intravenous line with a stopcock. The choice of medication depends on the clinical situation. In general, etomidate, 0.3 mg/kg i.v., or a short-acting benzodiazepine, such as midazolam, 1.0–2.5 mg i.v., is used because they tend to have minimal effects on the cardiovascular status of the patient. In addition, midazolam provides anterograde amnesia. Opiates, such as fentanyl citrate, 2 µg/kg i.v., should be used only in patients who are adequately perfused because their mild cardiac depressant activity can cause unexpected cardiovascular decompensation in hypoxic, hypoperfused patients. Sodium thiopental, 2–5 mg/kg i.v., is exclusively reserved for the well-perfused patient with a seemingly isolated head injury because it diminishes the transient elevation in intracranial pressure (ICP) associated with tracheal intubation. **A paralytic agent is administered immediately after the sedative**. Succinylcholine, 1.00–1.25 mg/kg i.v., is the paralytic of choice because, as a depolarizing muscle relaxant, it has a rapid onset (fasciculations within seconds) and a short half-life (recovery within 1–2 minutes). Contraindications in the acute trauma setting are limited to patients with known pseudocholinesterase deficiency or previous spinal injury. **Succinylcholine can be used safely in patients with acute burns or spinal trauma**. Rocuronium, 0.60–0.85 mg/kg i.v., is an alternative paralytic, but as a nondepolarizing relaxant, it has a slower onset (up to 90 seconds) and a longer half-life (recovery after 40 minutes) than succinylcholine. After onset of paralysis, the endotracheal tube (7.5 mm for women and 8.0 mm for men) is inserted through the vocal cords under direct vision with the assistance of a laryngoscope. The occluding balloon is inflated after positioning the tip of the tube to allow bilateral ventilation (21 cm from the incisors in women and 23 cm from the incisors in men). Proper positioning of the tube in the trachea should be confirmed by exhalation of carbon dioxide over several breaths (using a litmus paper device, capnometer, or their equivalent). **Adequacy of ventilation** should be verified by bilateral auscultation in each axilla. A **chest X-ray** should be taken within the next few minutes and checked to ensure proper endotracheal tube position. Tracheal intubation should secure an airway within 90–120 seconds (about three attempts). If it is unsuccessful, an airway placed directly through the cricoid membrane is often necessary.
- c. **Direct cricoid membrane airways**. In the majority of injured patients, orotracheal intubation provides a secure airway. In certain situations (unsuccessful orotracheal attempts, massive facial trauma), however, a more direct route is required. The two main means of providing such an airway are via cricothyrotomy or percutaneous transtracheal ventilation.
- Cricothyrotomy** is the method of choice for establishing a surgical airway in adults. The cricoid membrane is easily palpated between the cricoid cartilage and the larynx. Being both superficial and relatively avascular, it provides rapid, easy access to the trachea. After identification of the membrane, a 1.5-cm transverse skin incision is made over it (no skin preparation is necessary). A scalpel is used to poke a hole through the membrane. Care is taken to avoid exiting through the trachea posteriorly, thereby injuring the esophagus. Next, the scalpel handle, a tracheal spreader, or some similar surgical instrument is used to expand the hole. Finally, a 6.0-mm endotracheal or tracheostomy tube is inserted into the trachea through the cricothyrotomy, the balloon is inflated, and the tube sutured into place. Historically, a cricothyrotomy would eventually require revision to a tracheostomy to decrease the risk of tracheal stenosis but this has been challenged and many institutions will now use the cricothyrotomy site as a tracheostomy site. Cricothyrotomy is contraindicated in children younger than 12 years of age because of the anatomic difficulty in performing the procedure. In this situation, percutaneous transtracheal ventilation is an alternative.
 - Percutaneous transtracheal ventilation** can provide a temporary airway until a formal surgical airway can be supplied, especially in young children in whom cricothyrotomy is not possible. A small cannula (usually a 14-gauge intravenous catheter) is placed through the cricoid membrane. The cannula is connected to oxygen tubing containing a precut side hole. Temporary occlusion of the side hole provides passage of oxygen into the lungs via the cannula. Exhalation occurs passively through the vocal cords. Through this means, alveolar oxygen concentrations are transiently maintained for approximately 30–45 minutes.
2. **Breathing**. Once an airway is established, attention is directed at assessing the patient's breathing (i.e., the oxygenation and ventilation of the lungs). A patent airway does not ensure adequate breathing because the trachea can be ventilated without successfully ventilating the alveoli. Through the secured airway, 100% oxygen is administered. The chest is then examined. **The axillae are auscultated** to assess gas delivery to the peripheral lung. Any abnormal sounds suggest potentially life-threatening conditions. The **chest wall motion** is observed. The position of the trachea is noted. The use of accessory muscles of respiration is sought (this is often a sign of severe respiratory compromise and impending cardiovascular collapse). In this manner, important life-threatening abnormalities involving the thorax can be identified and treated.
- Life threats**. While assessing breathing during the primary survey, the trauma team must be vigilant for the following potentially fatal conditions that require immediate attention:
 - Tension pneumothorax**. Signs of a tension pneumothorax include the absence of breath sounds and hyperresonance in a lung field with tracheal deviation away from the side of the abnormality. Immediate decompressive therapy is indicated (a chest X-ray should not delay treatment). Decompression involves placement of a 14-gauge intravenous catheter in the second intercostal space in the midclavicular line. A tube thoracostomy should promptly follow decompression. A chest X-ray should be obtained only after the chest tube placement is complete.
 - Pneumothorax/hemothorax**. Absent or decreased breath sounds in a lung field without tracheal deviation usually indicate a simple pneumothorax or hemothorax on the affected side. A chest X-ray can usually confirm these conditions. Treatment consists of tube thoracostomy (28 Fr. or larger). The chest tube should be connected to an underwater, seal-suction device adjusted to 20-cm water suction.
 - Flail chest**. Paradoxical chest wall motion with spontaneous respirations indicates a flail chest (three or more ribs with two or more fractures per rib). Pulmonary contusion often accompanies such an injury. Chest X-ray often reveals the extent of fractures and underlying lung injury. Treatment involves adequate pain control, aggressive pulmonary toilet, and respiratory support. This injury is mentioned at this point because many of these patients will require early mechanical ventilatory support. Intensive care monitoring is recommended for elderly or debilitated patients.
 - Open pneumothorax**. Any chest wound communicating with the pleural space that is greater than two-thirds the diameter of the trachea will preferentially draw air into the thorax. Open chest wounds therefore must be covered with a partially occlusive bandage secured on three sides (securing all four sides can result in a tension pneumothorax and should be avoided). In this manner, air is prevented from entering the thorax through the wound but can exit via the wound if necessary. Prompt tube thoracostomy should follow placement of the partially occlusive dressing.
 - Tracheobronchial disruption**. Severe subcutaneous emphysema with respiratory compromise can suggest tracheobronchial disruption. The tube thoracostomy placed on the affected side will reveal a large air leak, and the collapsed lung may fail to reexpand. Bronchoscopy is diagnostic. Treatment involves intubation of the unaffected bronchus followed by operative repair (see [section III.D.2](#)).
 - Technique of tube thoracostomy** (see Chapter 43, [section II.B](#)).
 - Mechanical ventilation**. After an endotracheal tube is in place, the patient requires some form of ventilatory support. In the hemodynamically unstable or arresting patient, bag-valve ventilation using 100% oxygen is the method of choice. Once a patient has been stabilized, a mechanical ventilator can be used. Appropriate settings depend on the clinical situation, but initial settings would include an intermittent mandatory ventilation mode at a rate of 10 breaths per minute, a tidal volume of 12 mL/kg, an inspired oxygen concentration of 100%, and a positive end-expiratory pressure of 5 mm Hg. The goal of mechanical ventilation is to maintain an adequate oxygenation (i.e., a saturation of 95% or greater measured by pulse oximetry) with adequate ventilation (i.e., a PCO₂ of about 40 mm Hg corresponding to an end-tidal carbon dioxide value of approximately 32 mm Hg). Hyperventilation is no longer recommended in the initial management of the brain-injured patient. Additionally, more advanced forms of ventilatory support, such as permissive hypercapnia, pressure-controlled inverse-ratio ventilation, or high-frequency oscillatory ventilation, play little role in the initial ventilatory management of the trauma patient. They should be reserved for those patients with acute lung injury or acute respiratory distress syndrome in the intensive care unit.
3. **Circulation**. After evaluation of both the airway and breathing, the circulation is assessed. The goal of this portion of the primary survey is to identify and treat the presence of shock in the patient. Initially, all active external hemorrhage is controlled with direct pressure, and obvious fractures are stabilized. The pulse is characterized, and a blood pressure (BP) is obtained. The skin perfusion is determined by noting skin temperature and evaluating capillary refill. Over time, end-organ perfusion during a trauma resuscitation is estimated using mental status and urine flow as markers. **Shock** is defined as the inadequate delivery of oxygen and nutrients to tissue. The etiologies of shock can be divided into three broad categories: hypovolemic, cardiogenic, and distributive. The trauma team must be familiar with the manifestations and therapy of each category of shock because any of the three may be encountered in the injured patient.
- Hypovolemic shock**. The most common type of shock encountered in the trauma patient is a form of hypovolemic shock known as **hemorrhagic shock**. It occurs as a result of decreased intravascular volume secondary to blood loss and can be divided into four classes. In its severe form, it can manifest as a rapid pulse, decreased pulse pressure, diminished capillary refill, and cool, clammy skin. Therapy involves restoring the intravascular volume. As a result, the patient should have two large-bore intravenous lines placed (14- or 16-gauge). The antecubital veins are the preferred sites. If a peripheral intravenous catheter cannot be placed secondary to venous collapse, an 8.5 Fr. cannula (Cordis catheter) should be placed via the Seldinger technique into the femoral vein. The subclavian and internal jugular veins should be reserved for those patients in whom major venous intraabdominal injury would

prevent intravascular repletion. Short, fat intravenous catheters are used to maximize the flow of resuscitation fluids into the circulation (the rate of fluid flow is proportional to the cross-sectional area of a conduit and inversely proportional to the fourth power of its radius). A blood specimen should be simultaneously obtained for cross-matching. Resuscitation should consist of an initial bolus of 2 L of a balanced salt solution, typically Ringer's solution (children should receive an initial bolus of 20 mL/kg). All fluids administered should be warmed to prevent hypothermia. Administration of crystalloid fluid should not exceed 50 mL/kg. Whole blood should be administered if further resuscitation is required. If it has not been cross-matched (as is frequently the case), type O blood should be used. Premenopausal women should receive Rh⁻ blood. Men and menopausal or postmenopausal women can receive either Rh⁻ or Rh⁺ blood. In the setting of penetrating torso injury involving a large blood vessel, less aggressive resuscitation (keeping BP around 90 mm Hg) until formal surgical control of the bleeding site is obtained has been shown to have some benefit in diminishing blood loss (*N Engl J Med* 331:1105, 1994). Emergency department thoracotomy (EDT) is sometimes indicated for severe cardiopulmonary collapse (see [section V.A.1](#)).

- b. **Cardiogenic shock** occurs when the heart is unable to provide adequate cardiac output to perfuse the peripheral tissues. In the trauma setting, such shock can occur in one of two ways: (1) extrinsic compression of the heart leading to decreased venous return and cardiac output or (2) myocardial injury causing inadequate myocardial contraction and decreased cardiac output. Patients in cardiogenic shock secondary to extrinsic compression of the heart usually present with cool, pale skin, decreased BP, and distended jugular veins. They often respond transiently to an initial fluid bolus, but more definite therapy is always needed. Tension pneumothorax is the most common etiology. Therapy involves needle decompression and tube thoracostomy [see [section II.A.2.a.\(1\)](#)]. Cardiac tamponade is a less common cause. It usually occurs in the setting of a penetrating injury near the heart. Therapy consists of pericardial drainage and repair of the injury, usually a proximal great vessel or cardiac wound (see [section III.D.5.a](#) and [section III.D.6.a](#)). EDT may be required (see [section V.A](#)). Patients in cardiogenic shock secondary to myocardial injury can also present with cool skin, decreased BP, and distended jugular veins. Acute myocardial infarction can manifest in this way. Often, it is responsible for the traumatic event, but it can also occur as a result of the stress following an injury. Diagnosis is via electrocardiogram (ECG) and troponin levels. Therapy should follow advanced cardiac life support guidelines, keeping in mind that anticoagulants may need to be avoided early on until active bleeding related to the trauma has been excluded. Severe blunt cardiac injury is another manifestation. It usually occurs in the setting of high-speed motor vehicle crashes. An ECG and possibly an echocardiogram are essential. Therapy ranges from close monitoring with pharmacologic support in an intensive care unit to operative repair (see [section III.D.6.b](#)).
 - c. **Distributive shock** occurs as a result of an increase in venous capacitance leading to decreased venous return. Neurogenic shock secondary to acute quadriplegia or paraplegia is one such form. Loss of peripheral sympathetic tone is responsible for the increased venous capacitance and decreased venous return. These patients present with warm skin, absent rectal tone, and inappropriate bradycardia. They often respond to an initial fluid bolus but will eventually require pharmacologic support to help restore sympathetic tone. Phenylephrine is the drug of choice. It is administered via a central venous catheter at an initial rate of 1–2 µg/kg per minute and titrated to effect (using BP as a guide).
4. **Disability.** Assessment of the neurologic status of the injured patient follows the evaluation of the circulation. The goal of this phase of the primary survey is to identify and treat life-threatening neurologic injuries, and priority is given to evaluating level of consciousness and looking for lateralizing neurologic signs. The level of consciousness is quickly assessed using the **APVU system** (ascertaining whether the patient is awake, opens eyes to painful stimulus, opens eyes to voice, or is unarousable). The pupils are examined, noting size, symmetry, and responsiveness to light. Focal neurologic deficits are noted. Signs of significant neurologic impairment include inability to follow simple commands, asymmetry of pupils or their response to light, and gross asymmetry of limb movement to painful stimuli. Severe neurologic injuries require urgent evaluation and are either intracranial or spinal in origin.
- a. **Intracranial injuries.** Head injury remains a leading cause of trauma fatality in the United States. Herniation (either uncal or cerebellar) is often the final common pathway leading to death. Vigilance on the part of the trauma team can sometimes trigger interventions before such an event becomes irreversible. For example, uncal herniation is often associated with a “blown” (fixed, dilated) pupil on the side of herniation secondary to compression of either the constrictor (Edinger-Westphal) nucleus of the pupil or its outgoing fibers in the third cranial nerve. As this compression begins, the pupil assumes an ovoid or football shape. Such a finding can alert the trauma team to impending herniation and the need for immediate intervention. Acute therapy of severe intracranial injuries focuses on maximizing cerebral perfusion pressure (CPP) to provide an adequate supply of glucose and oxygen to the injured tissue. *CPF* is defined as the difference between mean arterial pressure (MAP) and ICP: $CPP = MAP - ICP$. Maximization of CPP therefore involves manipulating both MAP and ICP, and it is achieved when the BP is adequate (MAP >70–80 mm Hg) and the ICP is normal (<10–15 mm Hg in adults). A CPP of more than 60–70 mm Hg is the goal.
 1. **MAP.** Maintaining an adequate MAP is very important in the patient with head trauma because hypotension is a major risk factor for poor outcome. The patient should be kept euvolemic or hypervolemic. In addition, pharmacologic support should be used as necessary to maintain an adequate BP. Extreme hypertension should be avoided. Hypoxia is also a deleterious influence for traumatic head injuries, and all efforts to maintain adequate oxygenation should be maintained during trauma resuscitations.
 2. **ICP** is defined according to the modified Monro-Kellie hypothesis. It states that the intracranial contents are contained in a rigid sphere (skull) in which the total volume occupied by the three major constituents—brain, blood, and cerebrospinal fluid (CSF)—is constant and in which the pressure is evenly distributed. An increase in the volume occupied by one constituent therefore must be accompanied by a decrease in the volume occupied by one of the remaining constituents, or there will be a rise in the pressure. In the trauma setting, early and rapid delineation of intracranial injuries by computed tomographic (CT) scan is important so that decisions regarding the need for ICP monitoring can be made early. Although usually reserved for the intensive care unit or operating room setting, ICP monitoring is usually accomplished via the placement of a subarachnoid pressure monitor (“bolt”). An intraventricular catheter placed in the nondominant lateral ventricle can also be used. It has the advantage of providing a means of draining CSF when necessary. General measures used to prevent an increase in the ICP include elevating the head of the bed to 30–45 degrees and maintaining the patient's head in the midline position to prevent obstruction of jugular venous outflow. If ICP remains elevated, pharmacologic diuretic therapy to reduce the volume of both the CSF and the brain can be used. Mannitol, 0.25–1.00 g/kg, is the preferred agent, but furosemide can also be used. Sedation and therapeutic paralysis can acutely lower ICP, but they have the disadvantage of obscuring ongoing clinical neurologic examination. Although once advocated as an initial means of lowering ICP, hyperventilation is no longer recommended as a first-line therapy because of its adverse ischemic effects (it decreases ICP by causing intracranial vasoconstriction secondary to inducing a hypocarbic alkalosis). Currently, it is reserved as a second-line modality when other therapies have failed to lower ICP. When it is used, the PCO_2 should be closely monitored and kept at a level of 30–35 mm Hg. Monitoring of cerebral oxygen extraction should be initiated using a jugular bulb catheter, especially if prolonged hyperventilation is anticipated. The effective reduction in ICP secondary to hyperventilation lasts no more than 24–48 hours because of renal compensation of the respiratory alkalosis. Another second-line modality to decrease ICP is barbiturate-induced coma (pentobarbital or thiopental), which decreases cerebral blood flow by reducing the metabolic needs of the central nervous system (CNS). It should not be used in the acute setting due to severe vasomotor instability that may be associated with barbiturate administration.
 - b. **Spinal cord injuries.** Acute injury to the spinal cord can result in neurogenic shock which should be treated appropriately (see [section II.A.3.c](#)). In addition, spinal cord trauma produces debilitating neurologic loss of function. The appropriate acute management of such deficits remains somewhat controversial. A landmark multicenter trial showed that high-dose infusion of methylprednisolone immediately after blunt injuries to the spinal cord (complete or incomplete) resulted in modest but statistically significant functional preservation (*N Engl J Med* 322:1405, 1990). This study has been followed by several other prospective trials. Currently, most trauma centers administer an intravenous bolus dose of methylprednisolone at 30 mg/kg over 1 hour followed by a 23-hour infusion of the same drug at a rate of 5.4 mg/kg per hour in all patients presenting with blunt spinal trauma within 8 hours of injury. Patients presenting with penetrating spinal trauma or greater than 8 hours after blunt spinal trauma should not receive any regimen.
 - c. **Neurosurgical consultation.** A neurosurgeon should be consulted immediately in all patients with severe neurologic injuries who will likely need more advanced therapy (ICP monitoring, ventriculostomy, CSF drainage, evacuation of hematomas). Early radiologic evaluation of the CNS to exclude evacuable intracranial mass lesions is also critical (see [section II.B.4.d](#)).
5. **Exposure.** The last component of the primary survey is exposure with environmental control. Its purpose is to allow for complete visual inspection of the injured patient while preventing excessive heat loss. The patient is first completely disrobed, cutting away clothing so as not to disturb occult injuries. The patient then undergoes the visual inspection, including logrolling to examine the back, splaying of the legs to examine the perineum, and elevation of the arms to inspect the axillae. The nude patient loses heat rapidly to the environment unless specific countermeasures are undertaken. The resuscitation room should be kept as warm as possible. Any cold metal backboard should be removed as quickly as possible, and all soggy clothing or bedclothes should be taken off expeditiously. All resuscitation fluid should be warmed. Finally, the patient should be covered with warm blankets or a “hot air” heating blanket.
- B. **Completion of the primary survey.** The completion of the primary survey should be followed by a brief assessment of the adequacy of the initial resuscitation efforts.
1. **Monitoring.** Appropriate monitoring is essential to determine the clinical trajectory of the injured patient. If not already in place, ECG leads and a pulse oximeter should be applied. Provision for serial BP measurements should be made, preferably by an automatic cuff. Finally, an indwelling urinary catheter should be placed. Before insertion of the catheter, however, the urethral meatus should be inspected and found free of blood (the labia and scrotum should not harbor a hematoma). In addition, all male patients require palpation of the prostate to ensure that it is in the normal position, not displaced superiorly (“high riding”). If any genitourinary structures are abnormal, a retrograde urethrogram is necessary. If it is normal, the catheter may be passed. If urethral injuries are present, immediate consultation with a trained urologist is required before attempting to pass the catheter.
 2. **Laboratory values.** After placement of two intravenous catheters (see [section II.A.3.a](#)), laboratory values should be obtained. The most important test to obtain is the cross-match. Other investigations include blood chemistries, hematologic analysis, coagulation profile, toxicologic analysis (with ethanol level), urine analysis, and beta-human chorionic gonadotropin level if the patient is a woman of child-bearing age. The hematocrit is the most commonly misinterpreted test because it is not altered immediately with acute hemorrhage. It should not, therefore, be considered an indicator of circulating blood

volume in the trauma patient. (Serial hematocrit values, however, may give an indication of ongoing blood loss.) Finally, an arterial blood gas analysis should be drawn.

3. **Adequacy of resuscitation.** The adequacy of resuscitation can best be determined using urine output and arterial pH as indices because they are excellent global indicators of adequate end-organ perfusion. Resuscitation, therefore, should strive for a physiologic pH of 7.4 and a urinary output of 0.5–1.0 mL/kg per hour (50–75 mL per hour) in adults (1–2 mL/kg per hour in children). Base deficit and lactic acid levels are also used as markers of adequate resuscitation and have been shown to have prognostic value.
4. **Radiographic investigations.** Essential radiographic investigations are ordered during this period. These tests can provide critical data regarding injuries sustained in a trauma, but their performance should be worked around ongoing physical examinations and interventions. They should not interrupt or interfere with essential care.
 - a. **Blunt trauma.** Patients who have sustained blunt trauma with major energy transfer require a lateral cervical spine radiograph (which must include a clear view of the C-7 to T-1 interface), supine chest X-ray, and an anteroposterior pelvic radiograph. If time permits and the patient is stable, a formal three-view cervical spine series (lateral, anteroposterior, and odontoid) should be obtained. If there is not any evidence of spinal or pelvic injury, an upright chest X-ray should be obtained because it provides crucial information with regard to hemothorax, pneumothorax, mediastinal widening, and subdiaphragmatic gas that sometimes cannot be gleaned from a supine film. Finally, plane radiographs should be obtained of any area of localized blunt trauma, especially if fractures are suspected on the basis of physical exam.
 - b. **Penetrating trauma.** Patients who have sustained penetrating injuries require regional plane radiographs to localize foreign bodies and exclude perforation of gas-filled organs (e.g., intestines, lungs). When obtaining these films, all entrance and exit sites should be identified with a radiopaque marker. This technique gives insight into the trajectory of the penetrating object and the potential organs injured.
 - c. **Trauma ultrasonography.** Many trauma centers in North America now employ focused abdominal sonography for trauma (FAST) following the primary survey as an initial radiographic screening evaluation for all trauma. As the name implies, it is a focused examination designed to identify free intraperitoneal fluid and/or pericardial fluid. An ultrasound machine is used to take multiple views of six standard areas on the torso: (1) right paracolic gutter, (2) Morison's pouch, (3) pericardium, (4) perisplenic region, (5) left paracolic gutter, and (6) suprapubic region. Free fluid in the abdomen and within the pericardium appears anechoic. FAST has many advantages; it is portable, rapid, inexpensive, accurate, noninvasive, and repeatable. Its disadvantages include operator dependence as well as difficulty of use in morbidly obese patients or those with large amounts of subcutaneous air. It is most useful in evaluating patients with blunt abdominal trauma, especially those who are hypotensive. It may not be as useful in evaluating children or patients with penetrating trauma. Finally, some trauma centers are using sonography to evaluate the thorax for traumatic effusions.
 - d. **CT.** Occasionally, a severely injured patient may require immediate CT evaluation before completion of the secondary survey. For example, patients with severe head injury in whom a compressive intracranial lesion is suspected should undergo emergent CT evaluation to rule out an operable (and potentially lifesaving) condition (i.e., epidural or subdural hematoma). In general, however, the use of CT scan for evaluation of the abdomen/pelvis has become a standard of care and the availability of more rapid scanners has allowed for the development of protocols in some centers that incorporate the scanning of trauma patients with a complete body scan (i.e., head/cervical spine/chest/abdomen/pelvis). Radiology investigation protocols for the trauma setting continue to evolve but usually remain dependent on the availability of equipment and skilled technicians or radiologists in any particular institution.
- C. **Secondary survey.** The secondary survey follows the primary survey. It is a complete head-to-toe examination of the patient designed to inventory all injuries sustained in the trauma. Thoroughness is the key to avoid missing injuries, and a systematic approach is required. Only limited diagnostic evaluation is necessary to make a decision about subsequent interventions or evaluations. A review of important aspects of the secondary survey according to anatomic region follows. This review emphasizes only highlights and is not to be considered exhaustive.
 1. **Head.** The patient should be evaluated for best motor and verbal responses to graded stimuli to compile a Glasgow coma score (GCS). The GCS is highly reproducible, with little interobserver variability. Severity of head trauma can be stratified according to the score obtained. **Any patient with a GCS of 8 or below is considered to have severe neurologic depression and should be intubated to protect the airway** (see [Chapter 39](#) and [Table 39-1](#)). Inspection and palpation of the head are used to identify obvious lacerations and bony irregularities. All wounds require specific evaluation for evidence of depressed skull fractures or devitalized bone. Signs suggestive of basal skull fractures should be sought. These include periorbital hematomas ("raccoon eyes"), mastoid hematomas ("battle sign"), hemotympanum, otorrhea, and CSF rhinorrhea.
 2. **The face should be inspected** for lacerations, hematomas, asymmetry, and deformities. The cranial nerves should be evaluated. The bones should be palpated in a systematic fashion to search for evidence of tenderness, crepitus, or bony discontinuity. In particular, the presence of a midfacial fracture should be sought by grasping the maxilla and attempting to move it. If present, it should be classified according to the LeFort criteria. The nares should be examined for evidence of a septal hematoma. The oral cavity should be illuminated and inspected for evidence of mucosal violation (commonly seen in mandibular fractures). All dentures and/or displaced teeth should be removed to prevent airway occlusion. The conscious patient should be asked to bite down to determine if abnormal dental occlusion is present (highly suggestive of a maxillary or mandibular fracture). The eyes should be examined for signs of orbital entrapment, and the pupils reexamined. Finally, a nasogastric tube (contraindicated if there is a question of trauma to the midface) or orogastric tube (in patients with midfacial fractures or who are comatose) should be placed to decompress the stomach.
 3. **The neck should be inspected and palpated** to exclude cervical spine, vascular, or aerodigestive tract injury.
 - a. **Cervical spine evaluation.** Assessing the status of the cervical spine is an important aspect of the secondary survey. Signs of cervical spine injury include midline cervical spine tenderness or vertebral step-off on palpation. Excluding the presence of a cervical injury can often be challenging. The proper algorithm is often dictated by the overall condition of the patient.
 1. **Awake, unimpaired patient.** In the awake, unimpaired, neurologically intact patient, the cervical spine should be palpated for signs of injury (e.g., midline cervical spine tenderness, vertebral step-off). If positive findings are present, the stabilizing cervical collar should remain in place, and a formal three-view cervical spine X-ray series should be obtained (see [section II.B.4.a](#)). If the physical examination is normal, the patient may be allowed under supervision to move the neck through the full range of motion. If there is not any cervical spine pain during this movement, the likelihood of a cervical spine injury is very low, and the stabilizing cervical collar can be removed. If any cervical spine pain is elicited during this movement, the stabilizing cervical collar should remain in place, and a formal cervical spine X-ray series should be obtained. If these X-rays are interpreted as normal, then the possibility of ligamentous injury should be entertained, and the patient should undergo supervised flexion-extension radiographs of the neck. If these films are interpreted as normal, the likelihood of cervical spine injury is low, and the stabilizing cervical collar can be removed (a soft collar may be placed for comfort).
 2. **Unconscious or impaired patient.** In the unconscious or impaired (e.g., acutely intoxicated) patient, the cervical spine should be considered unstable until a reliable clinical examination can be performed because significant ligamentous instability can exist with a normal three-view cervical spine X-ray series. The stabilizing cervical collar, therefore, should remain in place until the patient is fully awake and unimpaired. The patient then can be evaluated as previously described [see [section II.C.3.a.\(1\)](#)]. A CT or MR scan of the cervical spine is a useful adjunct in patients who are unlikely to regain consciousness for extended periods of time.
 - b. **Vascular/aerodigestive evaluation.** In addition to evaluating the cervical spine, the neck should be inspected for active hemorrhage and palpated for local tenderness, hematomas, and evidence of subcutaneous air. Wounds should be classified according to their depth and their location. A wound is considered superficial if it does not penetrate the platysma; it is considered deep if the platysma is penetrated. The neck is divided anatomically into three zones: Zone I covers the thoracic inlet (manubrium to cricoid cartilage); zone II encompasses the midneck (cricoid cartilage to angle of the mandible); and zone III spans the upper neck (angle of mandible to base of skull).
 4. **Thorax.** Significant pulmonary, cardiac, or great vessel injury may result from both penetrating and blunt trauma. In all cases, examination of the thorax includes inspection, palpation, percussion, and auscultation. Particular attention should be directed at observing the position of the trachea, checking for symmetric excursion of the chest, palpating for fractures and subcutaneous emphysema, and auscultating the quality and location of breath sounds. Two points bear further comment. First, thoracic extraanatomic air (subcutaneous air, pneumomediastinum, or pneumopericardium) is frequently noted on physical examination or chest radiography in trauma patients (*Surg Clin North Am* 76:725, 1996). Such a finding should alert the trauma team to four potential etiologies: (1) pulmonary parenchymal injury with occult pneumothorax (most common cause), (2) tracheobronchial injury, (3) esophageal perforation, and (4) cervicofacial trauma (usually self-limiting). Second, symmetric breath sounds are not a guarantee of adequate ventilation and oxygenation. End-tidal carbon dioxide, oxygen saturation, and arterial blood gas values must be monitored to ensure that breathing is intact.
 5. The **abdomen** extends from the diaphragm to the pelvic floor, corresponding to the space between the nipples and the inguinal creases on the anterior aspect of the torso. When examining the abdomen during the secondary survey, the primary goal is to determine the presence of an intraabdominal injury rather than to characterize its exact nature. Detecting those patients with occult injuries of the abdomen requiring operative intervention remains a diagnostic challenge. The mechanism of injury, however, often provides important clues.
 - a. **Penetrating trauma.** Stab wounds to the abdomen can be divided into thirds: One-third do not penetrate the peritoneal cavity; one-third penetrate the peritoneal cavity but do not cause any significant intraabdominal injury; and one-third penetrate the peritoneal cavity and do cause significant intraabdominal damage. As a result, the ability to exclude penetration of the peritoneal cavity in the patient with a stab wound to the abdomen has important therapeutic implications. In the stable patient without obvious signs of intraabdominal injury (e.g., peritonitis), local wound exploration remains a viable screening option. It is a well-defined procedure that entails preparing and draping the area of the wound, infiltrating the wound with local anesthetic, and extending the wound as necessary to follow its track. If the track terminates without entering the peritoneum (occurring in approximately half of the patients who undergo the procedure), the injury can be managed as a deep laceration. Otherwise, penetration of the peritoneum is assumed, and significant injury must be excluded by further diagnostic evaluation. Options include laparoscopy or celiotomy, diagnostic peritoneal lavage (DPL with

a lowered red blood cell count of >10,000 as the positive cutoff value), FAST, or admission and observation. Gunshot wounds within the surface markings of the abdomen have a 95% probability of causing a significant intraabdominal injury and therefore require immediate celiotomy, but this has recently been challenged for those patients with stable hemodynamics and no peritoneal signs on physical examination. In this latter group of patients, it occasionally may be warranted to proceed with CT scan investigations to delineate the trajectory of the bullet missile and to observe the patient if no significant injuries are noted on CT scan.

- b. **Blunt trauma.** In the patient sustaining blunt abdominal trauma, physical signs of significant organ involvement are often lacking. As a result, a number of algorithms have been proposed to exclude the presence of serious intraabdominal injury.
 1. In the **awake, unimpaired patient** without abdominal complaints, hospital admission with serial abdominal examination is a cost-effective strategy to exclude serious abdominal injury as long as the patient is not scheduled to undergo an anesthetic that would interfere with observation. However, such patients are rare in the trauma setting.
 2. **Unstable patient with abdominal injury.** The unstable patient with injuries confined to the abdomen requires immediate celiotomy.
 3. **Unstable patient with multiple injuries.** The unstable patient with multiple injuries in whom there is uncertainty regarding the abdomen as the source of shock requires DPL (see [section V.B](#)). FAST can also be used in this situation (see [section II.B.4.c](#)).
 4. **Stable patient with multiple injuries.** The stable patient with multiple injuries in whom the abdomen may harbor occult organ involvement that is not immediately life-threatening requires CT evaluation (see [section V.C](#)). In addition to identifying the presence of intraabdominal injury, CT scanning can also provide useful information regarding the probability that a celiotomy will be therapeutic. Laparoscopy has also been proposed as an adjunct in this situation.
6. **The pelvis** should be assessed for stability by palpating the iliac wings. Signs of fracture include scrotal hematoma, unequal leg length, and iliac wing hematomas. Careful inspection for lacerations (and possible open fracture) is undertaken.
7. **The back** should be inspected for wounds and hematomas, and the spine should be palpated for vertebral step-off or tenderness. If there are positive signs of spinal injury, anteroposterior and lateral radiographs of the appropriate region (i.e., thoracic, lumbar, sacral) should be obtained.
8. **The genitalia and perineum** should be inspected closely for blood, hematoma, and lacerations. In particular, signs of urethral injury should be sought (see [section II.B.1](#)). A rectal examination is mandatory to assess rectal tone and to look for the presence of gross blood in the rectum.
9. **The extremities** should be inspected and palpated to exclude the presence of soft tissue, orthopedic, vascular, or neurologic injury. Inspection should look for gross deformity of the limb, active bleeding, open wounds, expanding hematomas, and evidence of ischemia. Obvious dislocations or displaced fractures should be reduced as soon as possible. All wounds should be examined for continuity with joint spaces or bone fractures. The limb should be palpated for subcutaneous air, hematomas, and the presence and character of peripheral pulses. A thorough neurologic examination should be undertaken to determine the presence of peripheral nerve deficits. Radiographs of suspected fracture sites should be obtained, and ankle arm indices (AAls) measured in the setting of possible vascular injury.
10. **General.** During the secondary survey (and throughout the initial evaluation of the injured patient), any rapid decompensation by the patient should initiate a return to the primary survey in an attempt to identify the cause. Finally, in any penetrating trauma, all entrance and exit wounds must be accounted for during the secondary survey to avoid missing injuries.

III. Definitive hospital care. With the completion of the primary and secondary surveys, definitive hospital care is undertaken, ranging from emergent celiotomy to admission and further assessment. During this phase of care for the trauma patient, extensive diagnostic evaluations are completed and therapeutic interventions performed. In this section, important therapeutic principles are discussed according to the anatomic location of the injury.

A. Head injuries

1. **Lacerations.** Active bleeding from scalp wounds can result in significant blood loss. Initial therapy involves application of direct pressure and inspection of the wound to exclude bone involvement (i.e., depressed skull fracture). If significant bone injury has been excluded, the wound may be irrigated and débrided. A snug mass closure incorporating all the layers of the scalp will effectively control any hemorrhage and should be done as soon as possible (i.e., before CT evaluations).
2. **Intracranial lesions.** Traumatic intracranial lesions are myriad. They include extraparenchymal lesions, such as epidural hematomas, subdural hematomas, and subarachnoid hemorrhages, as well as intraparenchymal injuries such as contusions and hematomas. CT examination is the diagnostic modality of choice. Acute therapy to control ICP and maximize CPP has been discussed previously [see [section II.A.4.a.\(2\)](#)]. A neurosurgeon should be consulted early because emergent surgical intervention can be required.

B. Maxillofacial injuries

1. **Lacerations.** All lacerations of the face should be meticulously irrigated, débrided, and closed primarily with fine nonabsorbable sutures. Alignment of anatomic landmarks is essential. Wounds involving the eyes, lips, ears, nose, and oral cavity may require consultation with a qualified specialist. Given the highly vascular nature of the face, primary closure can be performed up to 24 hours after an injury as long as it is accompanied with adequate irrigation and débridement. Any deep lacerations in the region of the parotid or lacrimal ducts should be examined for ductal involvement. If either parotid or lacrimal ductal injury is present, consultation with the appropriate specialist should be undertaken. Any patients with mucosal violation require appropriate antibiotic bacterial coverage of oral flora (i.e., penicillin or clindamycin).
2. **Fractures.** Patients with significant craniofacial soft-tissue injury or clinical signs of facial fractures require radiographic evaluation to determine bony integrity. Plane radiographs of the midface and mandible are helpful in the awake, cooperative patient. Commonly ordered films include the Caldwell view (for the orbital walls and frontal sinus), Waters' view (for orbit, nose, zygoma, and lateral maxilla), submentovertex view (for zygomatic arch), modified Towne's view (for mandible), and Panorex (for mandible). Facial CT (with both axial and coronal 3-mm views) is often required in complex midface fractures to define fracture fragments in detail. Therapy is predicated on the type of fracture present.
 - a. **Frontal sinus fractures.** Therapy of frontal sinus fractures varies depending on the severity of injury. Nondisplaced anterior table fractures are treated with broad-spectrum antibiotics and observation. Displaced anterior table fractures and posterior table fractures require operative intervention by a specialist.
 - b. **Nasal fractures** are one of the most common maxillofacial fractures encountered in the injured patient. Displaced fractures can usually be reduced nonoperatively, with subsequent packing of the nasal cavity for stability. The presence of a septal hematoma requires immediate incision and drainage to prevent avascular necrosis and resultant saddle-nose deformity.
 - c. **Maxillary fractures** are classified according to the LeFort system. These fractures often require complex open reduction and fixation. A surgical specialist experienced in these complicated repairs is essential.
 - d. **Mandibular fractures.** Fractures of the mandible typically occur at areas of relative weakness, including the parasymphiseal region, angle, and condyle. These injuries can often be treated with maxillomandibular fixation, but such therapy requires a 4- to 6-week interval. Rigid fixation using plates is another option.

C. Neck injuries

1. Penetrating neck wounds

- a. **The diagnostic evaluation** of penetrating neck trauma depends on both the depth and location of the wound. Lacerations that are superficial to the platysma should be irrigated, débrided, and closed primarily. Wounds deep to the platysma require evaluation based on the anatomic zone of the injury.
 1. **Zone I injuries.** Thoracic inlet injuries commonly involve the great vessels. Routine four-vessel arteriography followed by immediate exploration (if positive) has been advocated by many surgeons, owing to difficulty in the clinical evaluation and operative exposure of this region. Selective arteriography, however, has recently been shown to be feasible in asymptomatic patients with normal chest radiographs. In two prospective studies (*Br J Surg* 80:1534, 1993; *World J Surg* 21:41, 1997), only 5% of zone I injuries required operation for vascular trauma. Furthermore, routine arteriography did not identify any clinically significant vascular injuries that did not already possess "hard" (severe active hemorrhage, shock unresponsive to volume expansion, absent ipsilateral upper extremity pulse, neurologic deficit) or "soft" (bruit, widened mediastinum, hematoma, decreased upper extremity pulse, shock responsive to volume expansion) evidence of vascular trauma. Additionally, patients without clinical evidence of vascular trauma who were managed conservatively did not have any morbidity or mortality as a result of missed vascular injuries. A recent retrospective analysis supported such observations (*J Trauma* 48:208, 2000). Evaluation of the aerodigestive tract can also be approached selectively. Patients with clinical evidence of aerodigestive tract injury (hemoptysis, hoarseness, odynophagia, subcutaneous emphysema, or hematemesis) should undergo dual evaluation with bronchoscopy and meglumine diatrizoate (Gastrografin) or thin barium swallow. Esophagoscopy may be substituted for obtunded patients or patients otherwise unable to participate in the swallow study. CT scan of the chest and to delineate the trajectory of the bullet missile has also been used to determine the need for ancillary studies or surgical interventions.
 2. **Zone II injuries.** The proper diagnostic algorithm for midneck injuries is somewhat controversial. Little disagreement exists in the need for immediate operative exploration in the patient with evidence of obvious vascular or aerodigestive tract injury. In those stable patients without such signs, multiple selective approaches have been advocated. Some authors have recommended observation alone based on prospective data (*J Vasc Surg* 32:483, 2000). Other authors have recommended routine arteriography with panendoscopy, whereas there are those who claim that color flow Doppler ultrasonography is comparable to four-vessel arteriography in identifying clinically significant vascular injuries (*Arch Surg* 130:971, 1995). Contrast-enhanced CT scanning of the neck to identify significant aerodigestive injury has been advocated, and one prospective study has shown the equivalency of CT angiography to arteriography (*Radiology* 216:356, 2000). Finally, some authors continue to recommend the traditional ipsilateral

cervical exploration. None of these algorithms for management of penetrating zone II injuries has shown superiority over the other, and management is probably best dictated by the manpower and imaging services available to the surgeon.

3. **Zone III injuries.** Upper neck injuries with clinical evidence of vascular involvement require prompt arteriography because of the difficulty in gaining exposure and control of vessels in this region. Embolotherapy can be used for temporary or definitive management, except for the internal carotid artery. Injuries without clinical evidence of vascular trauma may be managed selectively (i.e., arteriography, color flow Doppler ultrasonography, CT). Direct pharyngoscopy suffices to exclude aerodigestive trauma.

b. **Operative therapy.** Regardless of the location of the cervical injury, **common operative principles** apply once surgical exploration is undertaken. Adequate exposure, including proximal and distal control of vascular structures, is essential. The most common approach is an incision along the anterior border of the sternocleidomastoid muscle. A collar incision is reserved for repair of isolated aerodigestive injuries or for bilateral explorations (e.g., transcervical injuries). The track of the wound must be followed to its termination. Arterial injuries are repaired primarily if possible. Otherwise, prosthetic vascular grafts can be used. Veins can be ligated, except in the case of bilateral internal jugular injury. Tracheal and esophageal injuries should be repaired primarily using synthetic absorbable sutures. If these injuries occur in tandem, a well-vascularized flap of muscle or fascia should be interposed between the repairs to decrease the incidence of posttraumatic tracheoesophageal fistula. Unexpected laryngeal injuries should be evaluated with endoscopy. A drain should be placed if there is any suspicion of aerodigestive tract violation. This maneuver will allow for controlled cutaneous drainage of any leak, thereby preventing lethal mediastinitis. In the case of combined aerodigestive and vascular injuries, the aerodigestive repair should be drained to the contralateral neck to prevent breakdown of the vascular repair from gastrointestinal (GI) secretions should the aerodigestive repair leak.

2. **Blunt neck trauma.** Severe blunt neck trauma can result in significant laryngeal and vascular injuries. In the patient with a stable airway, CT is the best modality for evaluation of a suspected laryngeal injury because it can help determine the need for operative intervention. Minor laryngeal injuries can be treated expectantly with airway protection, head-of-bed elevation, and corticosteroids. Major laryngeal injuries require operative exploration and repair. Blunt vascular trauma usually involves the internal or common carotid artery, but there may also be injury to the vertebral vessels without symptomatology. These injuries can be devastating because they often are not diagnosed until the new onset of neurologic deficits. Four-vessel arteriography is the preferred diagnostic modality, but color flow Doppler has also been suggested. Because the severity of the deficit and the time to diagnosis are strongly associated with outcome, a high index of suspicion is needed. Some authors have even advocated selective arteriography in asymptomatic patients with injury patterns or mechanisms suggestive of a blunt carotid or vertebral artery injury (i.e., severe hyperextension or flexion with rotation of the neck; direct blow to the neck; significant anterior neck soft-tissue injury; cervical spine fracture; displaced midface fractures or mandibular fractures; and basilar skull fracture involving the sphenoid, mastoid, petrous, or foramen lacerum) (*Ann Surg* 228:462, 1998). Current recommendation is for operative repair of surgically accessible lesions. Systemic anticoagulation (unless contraindicated) with heparin appears to improve neurologic outcome, and it is therefore recommended for surgically inaccessible lesions.

D. **Thoracic injuries.** Rapid diagnosis and treatment of thoracic injuries are often necessary to prevent devastating complications.

1. **Chest wall injuries.** Lacerations of the chest without pleural space involvement require simple irrigation, débridement, and closure. A chest wound communicating with the pleural space constitutes an open pneumothorax and should be treated accordingly [see [section II.A.2.a.\(4\)](#)]. Significant soft-tissue loss may occasionally be encountered and can be initially repaired with polytetrafluoroethylene mesh. Complex myocutaneous flap or prosthetic closure, however, is often required for definitive treatment. **Rib fractures are common**, especially in blunt trauma. They are readily identified on chest X-ray. Any rib fracture can trigger a progression of pain, splinting, atelectasis, and hypoxemia. Preventing this cascade through the use of adequate analgesia and pulmonary toilet is essential. Parenteral narcotics are often required. In the case of multiple rib fractures, intercostal regional blockade using local anesthetics or epidural analgesia using either narcotics or local anesthetics may be useful. Flail chest often results in significant respiratory embarrassment and must be treated aggressively [see [section II.A.2.a.\(3\)](#)]. Finally, the location of rib fractures can provide insight into possible associated injuries (i.e., thoracic aortic injuries with first and second rib fractures and hepatic or splenic injuries with lower rib fractures).

2. **Tracheobronchial injuries** often present with massive subcutaneous emphysema. Prompt diagnosis and initial stabilization are essential [see [section II.A.2.a.\(5\)](#)]. The operative approach is dictated by the location of the injury. Upper tracheal injuries require a median sternotomy. Distal tracheal or right bronchial injuries are repaired via a right thoracotomy. Left bronchial injuries mandate a left thoracotomy. Penetrating injuries can be débrided and repaired primarily using synthetic absorbable suture. Transections resulting from blunt injuries usually require débridement of the tracheobronchial segment with reanastomosis. Tracheal defects involving up to two rings can usually be repaired primarily through adequate mobilization. Complex bronchoplastic procedures or pulmonary resections are rarely required.

3. **Esophageal injuries** are most commonly encountered after penetrating trauma, and they can pose difficult diagnostic and therapeutic challenges. These injuries require prompt recognition because delay in diagnosis is often lethal (mortality can increase threefold when repair is delayed by 24 hours). Esophagoscopy combined with meglumine diatrizoate (Gastrografin) swallow can detect virtually all injuries, but either modality is probably adequate to evaluate the thoracic esophagus. Such evaluations must be performed expeditiously, however, because their use can cause delays that impact the morbidity of an esophageal injury repair (*J Trauma* 50:289, 2001). Much like tracheobronchial injuries, the operative approach is determined by the location of the injury. A right thoracotomy provides excellent exposure for most thoracic esophageal injuries, particularly those in the midesophagus. A left thoracotomy is recommended for distal esophageal injuries. Primary repair should be undertaken whenever possible and consists of closure using an absorbable synthetic suture. The repair can be buttressed with a vascularized flap (i.e., pleural or pericardial) or fundoplication (for distal injuries). Drain placement near (but not adjacent to) the repair is recommended. Treatment options in late recognized esophageal injuries include esophageal repair and wide pleural drainage, diversion with injury exclusion, complex flap closure, and esophageal resection (reserved for the esophagus with underlying pathology). Morbidity and mortality are high in this situation.

4. **Pulmonary injuries.** All pulmonary injuries can potentially have an associated pneumothorax (simple or tension). Prompt diagnosis and treatment can be lifesaving (see [section II.A.2.a](#)).

a. **Pulmonary contusion** can be associated with both blunt and penetrating thoracic trauma. These lesions often have adequate perfusion but decreased ventilation. The consequent ventilation-perfusion mismatch results in severe hypoxemia. Diagnosis is often made by chest X-ray. Therapy consists of aggressive pulmonary toilet and respiratory support. Occasionally, positioning the patient with the uninjured side in the dependent position will promote perfusion to this better-ventilated lung. Severe contusions often require intubation and mechanical ventilatory support. The management of such patients is extremely challenging because unusual modes of ventilation (e.g., pressure-controlled inverse-ratio ventilation) may be needed. Consultation with a critical care specialist is often essential.

b. **Hemothorax** is typically diagnosed as opacification on chest X-ray, and it commonly arises from penetrating chest injuries. In the majority of cases, tube thoracostomy is sufficient therapy. A chest X-ray obtained after placement of the tube should be inspected for both tube placement and adequacy of drainage of the hemothorax. A persistent hemothorax with a properly placed thoracostomy tube should raise the possibility of persistent hemorrhage within the hemithorax. Operative intervention is often based on the amount of initial sanguinous drainage and ongoing hemorrhage from the tube. Guidelines vary according to institution and should be individualized to the clinical situation. In general, patients who drain more than 1.5 L of blood at tube insertion or who have an ongoing blood loss greater than 100 mL per hour over 6 hours should undergo operative thoracotomy for control of hemorrhage. Significant intrathoracic bleeding can result from pulmonary hilar or great vessel injury (see [section III.D.5](#)). Pulmonary parenchymal hemorrhage can often be controlled with pulmonary tractotomy and oversewing of bleeding intrapulmonary vasculature. Pulmonary resection (lobectomy or pneumonectomy) may be considered for intractable pulmonary hemorrhage (usually from a hilar injury). Morbidity and mortality after pulmonary resection in the trauma setting, however, are significant, and it should therefore be considered a last resort. Air embolism can develop in the setting of significant pulmonary parenchymal injury, especially in the patient on positive-pressure mechanical ventilation. It usually presents as sudden cardiovascular collapse, and therapy consists of placing the patient in steep Trendelenburg, aspirating air from the right ventricle, and providing cardiovascular support. Chest wall intrathoracic hemorrhage usually originates from an intercostal or internal mammary artery and is best treated by ligation.

5. **Great vessel injury**

a. **Penetrating trauma.** Thoracic great vessel injury most commonly occurs secondary to penetrating trauma. These patients often present in profound shock with associated hemothorax. Occasionally, they may present with pericardial tamponade due to a proximal aortic or vena caval injury. Often, diagnostic investigations are not performed because immediate operative intervention is indicated (e.g., massive hemothorax, pericardial tamponade). In certain circumstances, however, diagnostic evaluation is possible and can be rather extensive. For example, the stable patient suffering from a transmediastinal gunshot injury requires evaluation of the thoracic great vessels, esophagus, trachea, and heart unless the trajectory of the missile clearly avoids these structures. Aortography, endoscopy with radiographic swallow study, echocardiography, and CT may be needed, therefore, to exclude significant injury. A CT scan of the chest to initially delineate the trajectory of the bullet missile has been used in some centers to determine the need for ancillary studies or surgical interventions. This strategy may provide a more directed approach and avoid potential complications related to the use of diagnostic or operative procedures. The operative approach depends on the vessel involved. Median sternotomy is ideal for access to the proximal aorta, superior vena cava, right subclavian artery, and carotid artery. A left infraclavicular extension ("trapdoor") to the median sternotomy provides exposure to the left subclavian artery, but a high left anterolateral thoracotomy is probably a better approach. Finally, rapid median sternotomy with either right or left infraclavicular extensions is most appropriate in the patient who has undergone EDT before arrival in the operating room. Whenever possible, primary repair should be performed for arterial and vena caval injuries. Prosthetic grafting may be necessary, however, for complex reconstructions. Brachiocephalic and innominate venous injuries can be ligated.

b. **Blunt trauma** associated with rapid deceleration (e.g., motor vehicle crashes, falls) can result in thoracic great vessel injury. The descending thoracic

aorta just below the origin of the left subclavian artery is particularly prone to rupture from rapid deceleration because of the presence of the ligamentum arteriosum in this region. Often, such a trauma results in complete transection of the aorta and immediate death from exsanguination. In some patients, however, only partial disruption of the aorta occurs, and there is tamponade of the hemorrhage. These patients can arrive to the trauma center alive. If their injury goes unrecognized, however, mortality is near universal. All patients presenting with blunt trauma associated with rapid deceleration therefore must be screened with a chest X-ray, preferably upright. Those patients with positive findings on chest X-ray (widened mediastinum, obscured aortic knob, deviation of the left main-stem bronchus or nasogastric tube, and opacification of the aortopulmonary window) require further evaluation. Helical (spiral) CT is then undertaken, and if it is interpreted as normal, the likelihood of a blunt aortic injury is near zero. If the helical CT is interpreted as indeterminate for aortic injury, immediate four-vessel arteriography is necessary. Arteriographic evidence of an aortic injury mandates prompt operative intervention. In some institutions, direct helical CT evidence of an aortic injury is sufficient to mandate operative repair, whereas in other institutions, it is followed by arteriography. Transesophageal echocardiography is an appropriate diagnostic modality in patients who are unable to undergo helical CT or arteriography. When a blunt aortic injury is present, operative repair should be undertaken as quickly as possible. A left anterolateral thoracotomy is the preferred approach to this portion of the aorta. Often, a prosthetic interposition graft is inserted at the level of the injury, but primary repair can also be performed. The issue of the use of partial cardiopulmonary bypass as a circulatory adjunct versus the "clamp-and-sew" technique remains controversial. Currently, definitive studies demonstrating the superiority of either method in terms of morbidity and mortality do not exist. Sufficient prospective data do exist, however, to recommend delaying operative repair in patients requiring other emergent interventions (i.e., laparotomy or craniotomy) for more immediately life-threatening injuries or in patients who are poor operative candidates due to age or comorbidities (*J Trauma* 48:1128, 2000). These patients require close pharmacologic control of their BP until surgical repair can be accomplished. The use of endovascular stents in this setting remains experimental at this time but offers another potential management strategy.

6. Cardiac injury

- a. **Penetrating trauma.** Cardiac injury is usually associated with penetrating anterior chest trauma between the midclavicular lines, but it can occur in the setting of penetrating trauma outside these anatomic landmarks. The presentation of a penetrating cardiac injury can range from hemodynamic stability to complete cardiovascular collapse. Pericardial tamponade should be suspected in the patient presenting in shock with distended neck veins and diminished heart sounds (Beck's triad). Tension pneumothorax must be excluded, however, by auscultating the lung fields. Additionally, patients with pericardial tamponade can present with jugular venous distention on inspiration (Kussmaul's sign). In the hemodynamically stable patient with suspicion for an occult penetrating cardiac injury, echocardiography is the diagnostic modality of choice. Transesophageal examination is preferred. The presence of pericardial fluid warrants emergent operative exploration. Another diagnostic modality is immediate subxiphoid pericardial exploration, especially in the setting of multiple injuries requiring emergent interventions. This procedure is performed in the operating room under general anesthesia. The pericardium is exposed via a subxiphoid approach, and a 1-cm longitudinal incision is made along it. The presence of straw-colored fluid within the pericardium constitutes a negative examination. Blood within the pericardium mandates definitive exploration and cardiorrhaphy. In the hemodynamically unstable patient, EDT is often the means of diagnosis. The preferred operative approach to repair penetrating cardiac injuries is via median sternotomy. Atrial and ventricular cardiac wounds are repaired primarily using interrupted or running monofilament sutures. Skin staples may also be used (especially in the setting of EDT). Care must be taken to avoid injury to coronary arteries during the repair. Wounds adjacent to major branches of the coronary circulation therefore require horizontal mattress sutures placed beneath the artery. Distal coronary artery branches may be ligated. Early consultation with a cardiothoracic surgeon is essential, especially in cases involving complex repairs or cardiopulmonary bypass.
- b. **Blunt trauma.** Blunt cardiac injury should be suspected in all patients presenting with the appropriate mechanism of injury (e.g., motor vehicle crash with chest trauma) or in those manifesting an inappropriate cardiovascular response to the injury sustained (*J Trauma* 44:941, 1998). Presentations range from unexplained sinus tachycardia to cardiogenic shock with cardiovascular collapse. ECG is the screening modality of choice (cardiac enzymes are not necessary). A normal ECG excludes blunt cardiac injury. The presence of an ECG abnormality (i.e., arrhythmia, ST changes, ischemia, heart block, or unexplained sinus tachycardia) in the stable patient warrants admission for 24-hour continuous cardiac monitoring. In the unstable patient, a transthoracic echocardiogram should be performed to identify any dyskinetic/akinetic myocardium or valvular damage. If the transthoracic evaluation is suboptimal, a transesophageal study is mandatory. Therapy is dictated by the echocardiogram findings. Patients with frank myocardial or valvular rupture require emergent operative repair. Otherwise, supportive therapy with continuous monitoring and appropriate pharmacologic support (i.e., inotropes and vasopressors) in an intensive care setting is warranted. Any arrhythmias are managed according to standard advanced cardiac life support protocols. Rarely, invasive mechanical cardiac support is necessary. Aneurysmal degeneration can be a long-term complication of blunt cardiac injury.

E. Abdominal injuries. The management of abdominal injuries must often be individualized to meet the needs of each patient, but certain guidelines do apply. All patients undergoing laparotomy for trauma should be prepared and draped from the sternal notch to the symphysis pubis anteriorly and from each posterior axillary line laterally to have access to the thorax if needed. Additionally, a lower extremity should be prepared and draped from the groin to the knee in case harvesting of the saphenous vein is required for vascular reconstruction.

1. **Diaphragmatic injuries** occur most commonly as a result of penetrating thoracic or abdominal trauma. Blunt trauma, however, can produce rupture secondary to rapid elevation of intraabdominal pressure. Frequently, diagnosis is made during celiotomy, but injury can occasionally be recognized on radiographic studies (e.g., chest X-ray). Therapy entails primary repair using monofilament synthetic sutures in a horizontal mattress stitch. In certain circumstances, polytetrafluoroethylene (PTFE) mesh closure may be required to cover large defects. Immediate repair prevents the long-term complications associated with diaphragmatic hernias.
2. **Abdominal esophageal injuries** are managed much like thoracic esophageal wounds (see [section III.D.3](#)). In addition to primary repair and drain placement, the fundus of the stomach can be used to buttress the site via a 360-degree (Nissen) wrap. Exposure of this portion of the esophagus can be difficult. Often, the left lobe of the liver must be mobilized and the crus of the diaphragm partially divided. Finally, placement of a feeding jejunostomy should be considered to allow for enteral nutrition in the postoperative period.
3. **Gastric injuries.** Injuries to the stomach occur most often in the setting of penetrating trauma. Sanguinous drainage from a nasogastric (or orogastric) tube should raise the possibility of gastric injury. Diagnosis is usually made at laparotomy. Simple lacerations can be repaired in one layer using synthetic absorbable suture. Alternatively, a full-thickness closure can be reinforced with Lembert stitches. Massive devitalization may require formal resection with restoration of GI continuity via gastroenterostomy. In such cases, vagotomy is helpful in reducing the risk of marginal ulcer.
4. **Hepatic injuries.** The liver is the most commonly injured solid organ in abdominal trauma.
 - a. **Penetrating trauma.** The diagnosis of penetrating hepatic injury is usually made at exploratory laparotomy, although CT has been used to identify injuries. Hemorrhage in the setting of hepatic trauma can be massive, and familiarity with maneuvers to gain temporary and definitive control of such bleeding is essential.
 1. **Initial hemostasis.** Rapid mobilization of the injured lobe with bimanual compression can often provide initial hemostasis. Perihepatic packing with laparotomy pads placed over the bleeding site and on the anterior and superior aspects of the liver to compress the wound is an extremely effective alternative. Temporary occlusion of the contents of the hepatoduodenal ligament (Pringle maneuver) with a vascular clamp decreases hepatic vascular inflow and is successful in controlling most intraparenchymal bleeding. It is often employed to allow for further mobilization of the liver and exposure and repair of injuries. Occlusion times should not exceed 30–60 minutes because longer intervals of warm ischemia are poorly tolerated by the liver. Failure of the Pringle maneuver to significantly decrease bleeding suggests major hepatic venous involvement, including juxtahepatic and retrohepatic inferior vena caval injuries. Prompt recognition and temporary vascular control of such injuries via the placement of an atriocaval shunt (Schrock shunt) can be lifesaving. Another option in this setting is total hepatic vascular isolation by placing vascular clamps on the hepatoduodenal ligament (if not already done), descending aorta at the level of the diaphragm, and suprahepatic and suprarenal vena cava. Finally, bleeding from deep penetrating injuries (e.g., transhepatic gun shot wounds) can sometimes be temporarily controlled through placement of an occluding intrahepatic balloon catheter.
 2. **Definitive hemostasis** is attained via multiple techniques. Raw surface oozing can be controlled by electrocautery, argon beam coagulation, or parenchymal sutures [horizontal mattress stitches placed in a plane parallel to the injury using large absorbable (No. 2 chromic) sutures on a wide-sweep, blunt-tip needle]. Topical hemostatic agents are also useful (i.e., microcrystalline collagen, thrombin, oxidized cellulose). Deeper wounds are usually managed by hepatotomy and with selective ligation of bleeding vessels. A finger-fracture technique is employed to separate overlying liver parenchyma within a wound until the injured vessel is identified, isolated, and controlled. Major venous injuries should be repaired primarily. Omental packing of open injuries can provide buttressing. Resectional débridement is limited to frankly devitalized tissue. Hepatic artery ligation is reserved for deep lobar arterial injuries where hepatotomy may result in significant blood loss. Formal anatomic resection should be avoided because of its high associated morbidity and mortality. Finally, closed suction drains should be placed near the wound to help identify and control biliary leaks.
 3. **Damage control principles** are frequently applied to complex hepatic injuries. Perihepatic packing with intensive care admission and resuscitation followed by return to the operating room in 24–48 hours is common place. On occasion, an intrahepatic balloon catheter is used. Liberal use of this algorithm can be lifesaving.
 - b. **Blunt trauma.** The management of blunt hepatic trauma has undergone a dramatic change over the last decade, largely due to improvement in CT imaging. Currently, CT is the recommended diagnostic modality for evaluation of the stable patient suspected of having blunt hepatic trauma because it can reliably identify and characterize the degree of an injury. Oral and intravenous contrast is necessary to help exclude concomitant hollow viscus injury. In the presence of hepatic trauma, therapy should be predicated on the hemodynamic status of the patient. The unstable patient requires operative exploration and control of hemorrhage as described (see [section III.E.4.a](#)). The stable patient without an alternate indication for celiotomy should be admitted for close hemodynamic monitoring (preferably in an intensive care setting) and serial hematocrit determinations. Operative intervention should

be promptly undertaken for hemodynamic instability. Evidence of ongoing blood loss in the hemodynamically stable patient warrants angiographic embolization of the bleeding source. Transfusions are administered as indicated. The frequency of follow-up CT evaluation of the lesion should be dictated by the clinical status of the patient. Resumption of normal activity should be based on evidence of healing of the injury. Stable patients therefore do not require strict bedrest. Complications of blunt hepatic trauma include biliary leak and abscess formation, both of which are readily amenable to endoscopic and percutaneous therapy. Delayed hemorrhage is rare, but pseudoaneurysm formation can occur. Nonoperative management is successful in the vast majority of blunt hepatic injuries and has even been reported in certain cases of penetrating hepatic wounds.

5. **Gallbladder injuries.** Injury to the gallbladder frequently coexists with hepatic, portal triad, and pancreaticoduodenal trauma. Treatment consists of cholecystectomy. The gallbladder also provides an effective means of assessing biliary tree integrity via cholangiography.
6. **Common bile duct injuries.** Penetrating trauma is most often responsible for common bile duct injuries. Like gallbladder injuries, they often occur in association with other right upper quadrant organ trauma. Most often, diagnosis is apparent at the time of laparotomy, but occult injuries can occur. Intraoperative cholangiography, therefore, is warranted when biliary involvement is suspected. Primary repair of the injured duct over a T tube is the preferred management, but Roux-en-Y choledochojejunostomy is sometimes required (i.e., when significant segmental loss of the duct is present). Choledochooduodenostomy and cholecystojejunostomy are poor options and should be avoided.
7. **Duodenal injuries** frequently coexist with devastating GI and abdominal vascular trauma and, as a result, can represent a diagnostic and therapeutic challenge. The type and severity of duodenal injury determine management.
 - a. **Duodenal hematoma.** Intramural duodenal hematomas usually occur after blunt trauma to the upper abdomen. Patients present with abdominal pain, nausea, and vomiting. Diagnosis is made with CT or upper GI fluoroscopy using meglumine diatrizoate (Gastrografin). Therapy consists of long-term nasogastric decompression and nutritional support (parenteral or enteral distal to the level of injury). The majority of duodenal hematomas are effectively treated in this manner, but operative evacuation may be indicated if obstruction persists for more than 14 days and CT reimaging confirms persistent hematoma.
 - b. **Duodenal perforation** can be difficult to diagnosis. Patients often complain only of vague back or flank pain, and symptoms can evolve slowly. Plane radiographic signs suggestive of perforation include evidence of retroperitoneal gas, blurring of the right psoas muscle, and leftward scoliosis. Upper GI fluoroscopy using water-soluble contrast (meglumine diatrizoate) may also show evidence of a leak. The diagnostic modality of choice, however, is CT using oral and intravenous contrast. Operative therapy depends on the degree of injury, but complete mobilization of the duodenum (Kocher maneuver) is essential for proper visualization and repair. Most defects (approximately 80%) can be repaired primarily in two layers with a transverse closure to avoid luminal narrowing. Closed suction drainage placed around the repair is strongly recommended to control any anastomotic leak. Nasoduodenal decompression should be instigated. Alternatively, antegrade or retrograde (preferred) tube duodenostomy can be performed in conjunction with tube gastrostomy and feeding jejunostomy, the so-called triple tube drainage (*J Trauma* 19:334, 1979).
 - c. **Complex duodenal injuries** are an operative challenge, and management remains controversial, especially in the presence of tissue devitalization. Whenever possible, débridement with primary repair should be performed. The repair should be protected via triple-tube drainage or pyloric exclusion with diverting gastrojejunostomy. Duodenal diverticularization can also be employed on rare occasions. For large defects not amenable to primary closure, a retrocolic Roux-en-Y duodenojejunostomy is an option. Finally, pancreaticoduodenectomy (Whipple procedure) should be reserved only for the most complex injuries, including duodenal devascularization or severe combined injuries involving the pancreatic head and bile duct. This procedure has a very high morbidity and mortality in the trauma setting.
8. **Pancreatic injuries.** Injury to the pancreas often occurs as a result of penetrating trauma, although a significant number of cases do involve blunt mechanisms. Associated morbidity and mortality remain significant and increase with the number of associated injuries. However, isolated pancreatic trauma is rare. Typically, the liver or stomach is also involved, but concomitant duodenal/pancreatic or biliary/pancreatic injuries do happen. Currently, CT is the best diagnostic imaging modality available, but, occasionally, endoscopic retrograde cholangiopancreatography or MR cholangiopancreatography studies should be used to help clarify the presence or absence of pancreatic injury. Pancreatic enzymes are not helpful in diagnosis. Treatment focuses on determining the presence and location of major ductal involvement. Commonly, such information is obtained during operative inspection of the gland, but, occasionally, intraoperative pancreatography (endoscopic or transduodenal) may be necessary. Adequate exploration entails performing a Kocher maneuver (to visualize the head of the pancreas) as well as transecting the gastrohepatic and gastrocolic ligaments (to inspect the body and tail of the pancreas). If necessary, the retroperitoneal attachments along the inferior border are divided (to view the posterior aspect of the pancreas) (*Curr Probl Surg* 36:325, 1999). When the pancreatic duct is intact, injuries (i.e., contusions, lacerations) are often treated with débridement and closed drainage. Pancreatorrhaphy is employed when indicated. Transection of the pancreatic duct requires more extensive procedures. For ductal injuries occurring to the right of the superior mesenteric vessels, treatment consists of closing the proximal end of the duct with a stapler or suture and draining the distal end via a Roux-en-Y pancreaticojejunostomy. Distal pancreatectomy (with or without splenectomy) should be used for transections occurring to the left of the superior mesenteric vessels. Additionally, it is an option for more proximal injuries in which resection would preserve greater than 10% of the pancreas. Whatever the procedure, the proximal end of the duct should be closed, and the pancreatic bed should be extensively drained. The liberal use of closed suction drainage helps decrease morbidity by controlling pancreatic leaks. Finally, severe injury to the head of the pancreas, especially in conjunction with duodenal and biliary trauma, may require pancreaticoduodenectomy.
9. **Splenic injuries.** The spleen is the second most common solid organ injured in abdominal trauma. Like hepatic trauma, the management of splenic injuries has undergone an evolution over the past decade.
 - a. **Penetrating trauma.** In general, penetrating splenic injuries are diagnosed at laparotomy, although they are sometimes identified on CT imaging. Management depends on complete mobilization of the spleen. Initial hemostasis is possible through manual compression. Minor injuries contained within the splenic capsule do not require any intervention. Bleeding from small capsular lacerations can be controlled with direct pressure or topical hemostatic agents. More complex injuries are treated according to the hemodynamic status of the patient. In the stable patient, splenorrhaphy can be employed in an attempt to preserve immune function (requiring salvage of 40% of the splenic mass). Devitalized tissue should be débrided and the wound closed with absorbable horizontal mattress sutures (usually 2-0 chromic). Alternatively, the spleen can be wrapped in absorbable mesh. Partial resection is indicated for isolated superior or inferior pole injuries. In the unstable patient or in those patients in whom splenic salvage fails, splenectomy should be performed in an expeditious manner. Drainage of the splenic bed is not necessary. All patients who undergo emergent splenectomy are at risk for overwhelming postsplenectomy sepsis infection. Although this complication is rare (maximum risk is 0.5% in prepubertal children), the mortality is up to 50%. All patients undergoing emergent splenectomy therefore require postoperative immunization against *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Neisseria meningitidis*. Some authors even recommend penicillin prophylaxis for children because they are at the highest risk. Yearly viral influenza vaccines are also recommended for postsplenectomy patients.
 - b. **Blunt trauma.** Today, most blunt splenic injuries are initially treated with nonoperative observation. CT remains the diagnostic modality of choice. All hemodynamically stable patients without an alternate indication for laparotomy should undergo close observation with continuous monitoring of vital signs, initial bed rest, nasogastric decompression (unless contraindicated), and serial hematocrit determinations. Patients with evidence of continuing blood loss who remain stable should undergo transfusion and selective angiographic embolization. Hemodynamically unstable patients or those failing nonoperative management (i.e., continuing transfusion requirement) should undergo operative exploration and therapy as described (see [section III.E.9.a](#)). Most often, splenectomy is performed. CT reimaging should be performed as clinical status indicates. Evidence of an intravenous contrast "blush" on CT can be a predictor of failure of nonoperative management. Patients should resume activity as tolerated and based on healing of injury. Routine postinjury CT imaging is not necessary.
10. **Small-bowel injuries.** Given its large volume and anatomy (tethering at the duodenojejunal flexure), the small bowel is prone to both penetrating (e.g., gunshot) and blunt (e.g., lap belt) trauma. Diagnosis is made at laparotomy or via radiographic imaging (plane radiograph or CT). Treatment consists of primary repair or segmental resection with anastomosis. Mesenteric defects should be closed.
11. **Large-bowel injuries.** The management of large-bowel injuries is currently undergoing a transition.
 - a. **Colonic injuries** typically occur secondary to penetrating trauma and are diagnosed at the time of laparotomy. Traditional management emphasized débridement and two-layer primary closure for stab wounds or low-velocity gunshot wounds. Excision, diverting colostomy creation, and Hartmann's pouch formation was advocated in patients with multiple injuries, prolonged shock, or large wounds requiring resection, and in cases of significant fecal contamination. Recently, however, a prospective multicenter study demonstrated that the surgical management (primary repair versus diversion) of penetrating colonic injuries did not affect the incidence of abdominal complications regardless of associated risk factors (*J Trauma* 50:765, 2001). The only independent risk factors for such complications were severe fecal contamination, large transfusion requirement (greater than four units) in the first 24 hours, and single-agent antibiotic prophylaxis. Primary repair, therefore, should be considered in all penetrating colonic injuries.
 - b. **Rectal injuries.** Penetrating trauma is also responsible for most rectal injuries. They often occur in association with genitourinary or pelvic vascular trauma, and they can be diagnosed via proctoscopy or CT or at laparotomy. Traditional management advocated rectal washout (débridement), diverting sigmoid colostomy creation, and presacral drain placement (the so-called three Ds). A prospective randomized trial, however, demonstrated that omission of presacral drains in the management of low-velocity penetrating rectal injuries did not increase infectious complications (*J Trauma* 45:656, 1998). Débridement (with primary repair of rectal wounds when possible) and diverting colostomy formation therefore seem sufficient management. Distal stump mucous fistula construction can simplify subsequent reconstruction because it obviates the need to search for a Hartmann's pouch in the pelvis. Reversal can be undertaken after 2 weeks if barium enema reveals healing of the rectum and the patient is medically stable.

F. Retroperitoneal injuries

1. **Retroperitoneal vascular injuries.** Injuries to the major retroperitoneal vessels or their abdominal branches can be life-threatening. These wounds usually

present with frank intraabdominal hemorrhage or retroperitoneal hematoma formation. Management is based on both mechanism of trauma and location of injury.

- a. **Penetrating trauma.** The majority of retroperitoneal vascular injuries are the result of penetrating trauma. By definition, any hematoma formed by a penetrating mechanism is uncontained and requires prompt exploration.
 1. **Initial access and hemostasis.** At times, vascular injuries present with massive intraabdominal bleeding, and familiarity with techniques to control such hemorrhage expeditiously and to obtain access to vessels efficiently can be lifesaving. Packing the site of injury with laparotomy pads is always a reliable temporizing option. Often, initial control requires occluding the supraceliac aorta at the level of the diaphragmatic hiatus using a vascular clamp, T bar, or direct pressure. Division of the gastrohepatic ligament with mobilization of the stomach and esophagus can provide access to this section of the aorta. Occasionally, division of the diaphragmatic crus is necessary for more proximal control. Once the proximal aorta has been occluded, definitive identification and repair of a vascular injury require adequate exposure of the involved vessels. A left medial visceral rotation (Mattox maneuver) provides excellent access to the aorta, celiac axis, superior mesenteric artery (SMA), left renal artery, and iliac arteries. A right medial visceral rotation (Catell maneuver) readily exposes the vena cava (with a combined Kocher maneuver), right renal vessels, and iliac veins. The infrarenal aorta may also be approached via a transperitoneal incision at the base of the mesocolon.
 2. **Repair of vascular injuries.** Most aortic and iliac arterial injuries can be repaired directly by lateral arteriorrhaphy. On occasion, reconstruction with graft prosthesis or autologous venous graft is necessary for significant circumferential or segmental defects. If enteric contamination is extensive, extraanatomic bypass with oversewing of the proximal stump is mandatory. Injuries to the celiac root or certain of its branches (left gastric or splenic arteries) can often be ligated without adverse outcome, especially in young patients. Splenectomy must follow splenic artery ligation. Common hepatic artery injuries should be repaired when possible (via lateral arteriorrhaphy, resection and reanastomosis, or graft), but ligation can be tolerated at times. SMA defects must be repaired. Vena caval and iliac venous injuries are repaired by lateral venorrhaphy. Injuries to the superior mesenteric vein (SMV) and portal vein should undergo repair, but cases of successful outcome after ligation have been reported. Because of the risk of postoperative thrombosis leading to portal hypertension or superior mesenteric infarction, SMV and portal venous reconstructions must be closely followed, and anticoagulation is often administered. Finally, major renal arterial and venous injuries require primary repair, whereas partial nephrectomy is recommended for segmental vessel involvement.
- b. **Blunt trauma** can cause retroperitoneal vascular injury with resultant hematoma formation. Often, these hematomas are discovered at operative exploration, but they are sometimes seen on preoperative imaging. The character and location of the hematoma determine management.
 1. **Central abdominal hematomas (zone I).** All central abdominal hematomas caused by blunt trauma require operative exploration. Supramesocolic hematomas are usually due to injuries to the suprarenal aorta, celiac axis, proximal SMA, or proximal renal artery. They should be approached via a left medial visceral rotation. Inframesocolic hematomas are secondary to infrarenal aortic or inferior vena caval injuries and are best exposed by a transperitoneal incision at the base of the mesocolon. As with any vascular repair, proximal and distal control of the involved vessel should be obtained prior to exploration if possible.
 2. **Flank hematomas (zone II).** Flank hematomas are suggestive of renal artery, renal vein, or kidney parenchymal injury. Unless they are rapidly expanding, pulsatile, or ruptured, they should not be explored if discovered at the time of celiotomy. Radiographic evaluation of the ipsilateral kidney is necessary in this situation to assess its function, usually by means of CT imaging. Evidence of nonfunction should prompt arteriography of the renal artery because blunt abdominal trauma can often cause intimal tears with resulting thrombosis of the artery. If it is discovered within 12 hours of the injury, revascularization is performed. Otherwise, nonoperative management is preferred. Nephrectomy is indicated when laparotomy is performed for associated injuries. In this setting, removal of the nonfunctioning kidney will decrease long-term renal complications (urinoma, hypertension, and delayed bleeding). When operative exploration of a flank hematoma is required, vascular control should be obtained outside Gerota's fascia. Total or partial (to preserve renal mass) nephrectomy may be necessary for a shattered kidney.
 3. **Pelvic hematomas.** Central pelvic hematomas in the setting of blunt trauma are usually due to pelvic fractures. If discovered at celiotomy, they should not be explored unless iliac vessel injury is suspected (loss of ipsilateral groin pulse, rapidly expanding hematoma, pulsatile hematoma) or rupture has occurred. Bleeding from pelvic fractures can be massive, and management should focus on nonoperative control. Unstable pelvic fractures in association with hypotension should undergo some form of external stabilization. In extreme circumstances, temporary control of hemorrhage can be achieved by wrapping a sheet tightly around the pelvis. Formal external fixation should follow as soon as possible. It should also be considered in those patients with unstable pelvic fractures who require celiotomy or who are hemodynamically stable but have a need for continued resuscitation. Pelvic angiography with selective embolization is the preferred intervention for patients in whom major pelvic fractures are the suspected source of ongoing bleeding. It should also be considered in patients with major pelvic fractures when CT imaging reveals evidence of arterial extravasation in the pelvis or when bleeding in the pelvis cannot be controlled at laparotomy.
2. **Genitourinary injuries.** Injuries to the genitourinary tract are discussed in detail in [Chapter 41](#). Three points, however, warrant discussion in the context of trauma.
 - a. **Urethral injuries** complicate the placement of an indwelling catheter. Their diagnosis and management have been discussed (see [section II.B.1](#)).
 - b. **Hematuria** is suggestive of an injury to the urinary tract. In the setting of blunt abdominal trauma, its presence (gross or microscopic) in association with hypotension mandates urologic evaluation. The absence of hematuria, however, does not always exclude an injury to the urinary tract, especially in the setting of penetrating torso trauma.
 - c. **Radiographic imaging.** CT is the best imaging modality to demonstrate urologic injury in the trauma patient who does not require laparotomy for other reasons. It allows visualization of the kidneys, ureters, and bladder (when the indwelling catheter is clamped). In addition, it provides information regarding kidney perfusion. Although once commonly used, excretory urography in the trauma patient is often unsatisfactory and is now rarely used.
- G. **Orthopedic injuries** are discussed in detail in [Chapter 40](#), but three important considerations bear mentioning.
 1. **Blood loss.** Fractures can produce large blood losses. A broken rib is associated with a 125-mL blood loss, a forearm fracture with a 250-mL blood loss, a broken humerus with a 500-mL blood loss, a femur fracture with a 1,000-mL blood loss, and a complex pelvic fracture with a 2,000-mL or more blood loss. Stabilization of fractures can minimize the amount of bleeding. Although the MAST trouser (pneumatic antishock device) has been largely discredited as a device for raising BP, it may afford transient pneumatic stabilization to lower extremity and pelvis fractures, thereby attenuating further blood loss while the patient is being prepared for more specific interventions (e.g., traction, fixation, or arteriography and embolization).
 2. **Spinal fractures.** Fractures of the spine are multiple in 10% of cases. Complete radiographic evaluation of the spine is necessary, therefore, when a single fracture is discovered.
 3. **Joint involvement.** Two joints overlie single-access arteries: **the elbow and the knee**. Fractures or dislocations of either of these joints increase the risk of ischemic complications of the involved distal limb. The integrity of the underlying artery therefore must be confirmed by serial evaluation, color flow duplex examination, or, in many cases, arteriography.
- H. **Extremity injuries.** Extremity trauma can result in devastating injuries requiring the coordination of multiple specialists to perform complex reconstructions. The goal of management is limb preservation and restoration of function, and it should focus on ensuring vascular continuity, maintaining skeletal integrity, and providing adequate soft-tissue coverage.
 1. **Penetrating trauma.** Penetrating extremity trauma typically occurs in males younger than 40 years old. Multiple injuries can occur in association with such trauma, and a high index of suspicion is necessary to diagnose and repair them expeditiously.
 - a. **Vascular injuries.** A wounded extremity can tolerate approximately 6 hours of ischemia before the onset of irreversible loss of function. Quickly identifying and repairing vascular injuries, therefore, are essential in any extremity trauma. Immediate operative exploration is indicated for obvious (hard) signs of vascular involvement (pulse deficit, pulsatile bleeding, bruit, thrill, or expanding hematoma) in gunshot or stab wounds without associated skeletal injury. Arteriography should be employed for those patients with hard vascular signs in the setting of associated skeletal injury (fracture, dislocation) or shotgun trauma. Patients with probable (soft) signs of vascular injury (nerve deficit, nonexpanding hematoma, associated fracture, significant soft-tissue injury, or history of bleeding/hypotension) require evaluation of vascular integrity. A useful algorithm is to check the ankle-arm index (AAI) initially. If the AAI on the affected limb is greater than 0.9, no further radiographic evaluation is necessary. If it is less than 0.9, noninvasive Doppler ultrasonography should follow to exclude vascular injury. If ultrasonography is equivocal, arteriography is indicated; if it is positive, either operative exploration or arteriography can follow (depending on the institution). Patients without hard or soft signs do not require arteriography to exclude vascular involvement. Occult vascular injuries can be managed nonoperatively with subsequent repair as indicated without an increase in morbidity. Arterial injuries should be repaired within 6 hours to maximize limb salvage rates. **Operative approach** is similar to elective vascular procedures. Proximal and distal control of the involved vessel is essential. Primary repair using monofilament suture should be performed for limited arterial lacerations. For complex injuries (large segmental or circumferential defects), resection with reanastomosis, patch angioplasty, or interposition grafting is preferred. Whenever possible, autologous vein should be used instead of PTFE for patching or grafting because of its higher patency rates. Ligation of single-artery forearm and calf injuries is possible in the presence of normal counterparts. Restoration of blood flow (via temporary shunt or formal repair) should precede any skeletal reconstruction in cases of combined injuries. Completion arteriography should be performed after any arterial repair. Venous injuries should undergo lateral venorrhaphy or resection with end-to-end reanastomosis if the patient is hemodynamically stable. Ligation with postoperative leg elevation and compression stocking placement (to reduce edema) is indicated in all other cases. Multiple compartment fasciotomies should be liberally used, especially after prolonged ischemia or in the presence of associated injuries.
 - b. **Skeletal injuries** are diagnosed with plane radiography. Restoration of skeletal integrity is attained by means of either internal or external fixation. Temporary vascular shunting should be performed before stabilization of an unstable fracture in the setting of combined injuries. External fixation is

preferred in the presence of gross contamination or tissue loss (see [Chapter 40](#) for further details).

- c. **Soft-tissue injuries.** Definitive closure of large soft-tissue defects rarely occurs at the initial operation for extremity trauma. Complex wounds are often thoroughly irrigated and débrided, dressed, and reviewed daily in the operating room. Delayed closure is then undertaken and may require advanced soft-tissue flaps (pedicle or free). On rare occasions with a so-called mangled extremity, primary amputation may be necessary in the setting of severe soft-tissue defects, major bone injury, or unreconstructable peripheral nerve injury with loss of limb function.
2. **Blunt trauma.** Blunt extremity trauma can result in debilitating crush or near-avulsion injuries. Diagnosis and management are the same as in penetrating trauma (see [section III.H.1](#)), but limb salvage and preservation of function tend to be worse due to the extent of injury. These wounds often require the coordinated involvement of multiple specialists.
3. **Extremity compartment syndromes.** Compartment syndromes are common in distal (forearm and leg) extremity trauma. They typically occur in association with prolonged limb ischemia or external pressure, fractures, crush or vascular injuries (especially combined arterial and venous injury), and burns. Increased tissue pressure (>30 mm Hg) within the inelastic fascial compartment leads to occlusion of capillary flow and ischemia. Signs and symptoms of compartment syndrome include pain (especially on passive motion), pressure, paralysis, paresthesia, pulselessness, and pallor (the so-called 6 Ps). A high index of suspicion is necessary for early diagnosis because signs often occur late in the process. Serial compartment pressure measurements therefore should be undertaken in any patient with risk factors. Fasciotomy of all involved compartments is necessary when pressures are 30–40 mm Hg (or lower, if evidence of ischemia exists). Additionally, fasciotomy should be performed if pressures cannot be obtained.

IV. Damage control surgery. Over the last decade, the concept of damage control has gained increasing support among trauma surgeons as a valuable adjunct in the surgical care of severely injured patients (*Surg Clin North Am* 77:753, 1997). Evolving in response to changing patterns of injury in urban American settings, the damage control philosophy centers on coordinating staged operative interventions with periods of aggressive resuscitation to salvage trauma patients sustaining major injuries. These patients are often at the limits of their physiologic reserve when they present to the operating room, and persistent operative effort results in exacerbation of their underlying hypothermia, coagulopathy, and acidosis (the “triad of death”), initiating a vicious cycle that culminates in death. In these situations, abrupt termination of the procedure after control of surgical hemorrhage and contamination followed by intensive care resuscitation and staged reconstruction can be lifesaving. Although often discussed in the context of abdominal trauma, the practice of damage control can be applied to all organ systems. It is divided into three phases: initial exploration, secondary resuscitation, and definitive operation.

- A. **Phase I (initial exploration).** The first phase in the damage control algorithm consists of performing an initial operative exploration to attain rapid control of active hemorrhage and contamination. The decision to revert to a damage control approach should occur early in the course of such an exploration. In the setting of abdominal trauma, the patient is prepared and draped as previously described (see [section III.E](#)), and the abdomen is entered via a midline incision. Any clot or debris present on entering the abdomen is promptly removed. If exsanguinating hemorrhage is encountered, four-quadrant packing should be performed. The packing is then removed sequentially, and all surgical hemorrhage within a particular quadrant is controlled before proceeding. Following control of bleeding, attention is directed at containment of any enteric spillage. Any violations of the GI tract should be treated with suture closure or segmental stapled resection. Anastomosis and stoma formation should be deferred until later definitive reconstruction, and any stapled ends of the bowel should be returned to the abdomen. External drains are placed to control any major pancreatic or biliary injuries. Laparotomy packs are then reinserted (especially in the presence of coagulopathic bleeding), with care being taken to reapproximate violated tissue planes. Often, primary abdominal fascial closure is not possible secondary to edematous bowel or hemodynamic instability. Alternative methods of closing the abdomen include skin closure via towel clips or running suture, Bogota bag placement, prosthetic mesh insertion, or abdominal wall zipper creation. Of all these techniques, the Bogota bag is the most commonly used. It is fashioned by opening the seams of a 3-L sterile urologic irrigation bag and suturing it to the skin edges. Throughout the initial operative exploration, communication between the surgeons, nursing staff, and the anesthesia team is essential for optimal outcome.
- B. **Phase II (secondary resuscitation).** The second phase in the damage control approach focuses on secondary resuscitation to correct hypothermia, coagulopathy, and acidosis. Following completion of the initial exploration, the critically ill patient is rapidly transferred to the intensive care unit. Invasive monitoring (systemic and pulmonary arterial catheter) and complete ventilatory support are often needed. Rewarming is initiated by elevating the room temperature, placing warming blankets, and heating ventilator circuits. All intravenous fluids, blood, or blood products are prewarmed. As the body temperature normalizes, coagulopathy improves, but rapid infusion of clotting factors (fresh frozen plasma, cryoprecipitate, and platelets) is usually still required. Circulating blood volume is restored with aggressive fluid, blood, and blood product resuscitation, improving end-organ perfusion and correcting acidosis. With these interventions, hemodynamic stability returns, urinary output increases, invasive monitoring parameters improve, and serum lactate levels and arterial pH analysis improve. In the setting of abdominal trauma, a potentially lethal complication that can occur during this phase is abdominal compartment syndrome. It is a form of intraabdominal vascular insufficiency secondary to increased intraabdominal pressure. Presentation includes abdominal distention, low urinary output, ventilatory insufficiency in association with high peak inspiratory pressures, and low cardiac output secondary to decreased venous return (preload). Diagnosis is made via measurement of urinary bladder pressure (>25 cm H₂O). When present, prompt operative reexploration is mandated to relieve the increased pressure. If surgical bleeding is found to be the cause of the intraabdominal hypertension, it should be controlled and the abdomen closed. If severe edema of the intraabdominal contents is the source of the compartment syndrome, the abdomen should be closed using a Bogota bag to reduce intraabdominal pressure. Following correction of the problem, phase II resuscitation is continued.
- C. **Phase III (definitive operation).** The third phase of damage control consists of planned reexploration and definitive repair of injuries. This phase typically occurs 48–72 hours following the initial operation and after successful secondary resuscitation. In the setting of abdominal trauma, all complex injuries are repaired with precedence going to those involving the vasculature. Conservative principles should be applied. Risky GI anastomoses or complex GI reconstructions should be avoided. The abdomen should be closed primarily if possible. Otherwise, mesh or simple skin closure with staged repair of the resulting ventral hernia should be performed. Even though the damage control approach allows for salvage of many severely injured patients, it is still associated with substantial morbidity and mortality. Outcome is often determined by providing excellent supportive care (ventilation, nutrition, appropriate antibiotics, and physical therapy with rehabilitation services).

V. Miscellaneous aspects of general trauma care

- A. **Emergency department thoracotomy (EDT)** is performed as a final attempt to salvage a certain subset of patients presenting in extremis to the emergency department. The goals are to control intrathoracic hemorrhage, relieve cardiac tamponade, cross-clamp the thoracic aorta, and restore cardiac output.
 1. **Indications.** The indications for EDT have been refined over time. Currently, it should be used in the management of penetrating chest trauma associated with significant hemodynamic deterioration (systolic BP of <60 mm Hg) or cardiopulmonary arrest occurring within the emergency department or shortly before arrival (<10–12 minutes). Additionally, it can be used in certain cases of penetrating abdominal trauma fulfilling the same criteria. Current thinking is that it is contraindicated in blunt chest or abdominal trauma resulting in cardiopulmonary arrest because survival after such an intervention almost never occurs.
 2. **Technique.** EDT is performed via an anterolateral left thoracotomy in the fifth or sixth intercostal space. The skin, subcutaneous tissues, and intercostal musculature are opened sharply. A Finochietto retractor is placed to spread the ribs and aid in exposure. First, the pericardium is identified and incised anterior to the phrenic nerve. Any clot or debris is removed from around the heart. Specific cardiac injury is then sought, and repair is undertaken as previously described (see [section III.D.6.a](#)). After cardiorrhaphy, air is evacuated from the heart by needle aspiration, and the adequacy of cardiac filling is assessed to determine intravascular volume status. In the absence of associated pulmonary vascular or great vessel injury, vigorous volume resuscitation is undertaken. If peripheral vascular access is insufficient, direct infusion into the right atrial appendage can be performed. In severely hypovolemic patients, the descending thoracic aorta may be exposed and cross-clamped to maintain coronary and cerebral perfusion. The aorta should also be clamped if any intraabdominal hemorrhage is suspected. During volume resuscitation, open cardiac massage is employed to provide adequate circulation. After restoration of adequate circulatory volume, the underlying cardiac rhythm is assessed, and internal cardioversion is used when appropriate. The patient should be transported to the operating room for definitive injury management and wound closure after a successful resuscitation.
 3. **Complications** of EDT are manifold. They include lung injury in gaining access to the heart, transection of the phrenic nerve while performing pericardotomy, injury to the coronary vessels during cardiorrhaphy, and esophageal trauma in clamping the descending thoracic aorta. Care must be taken during each step of the procedure, therefore, to avoid causing additional injuries.
- B. **Diagnostic peritoneal lavage (DPL).** Since the advent of FAST and rapid helical CT imaging, DPL is being used less frequently in the evaluation of patients with suspected intraabdominal injuries. It remains, however, still a useful diagnostic modality in certain situations. Percutaneous DPL is technically controversial and is not discussed here.
 1. **Indications.** DPL is useful in excluding the presence of significant intraabdominal organ injury in the presence of blunt trauma or stab wound to the abdomen. It should be employed when less invasive techniques (e.g., serial abdominal examinations, CT, or FAST) are either not feasible due to the condition of the patient (e.g., severe hypotension) or unavailable. The only absolute contraindication to DPL is a planned celiotomy. Pelvic fracture, pregnancy, and prior abdominal surgery often mandate a change in indication and technique. All patients undergoing DPL require prior evacuation of the stomach via a gastric tube as well as drainage of the bladder by indwelling catheter.
 2. **The technique** of DPL is discussed in Chapter 43, [section III.B](#). Aspiration of 10 mL of gross blood or any enteric contents is considered a positive DPL. Additionally, the microscopic presence of 100,000 red blood cells/μL or 500 white blood cells/μL in the setting of blunt abdominal trauma and 10,000 red blood cells/μL or 50 white blood cells/μL in the setting of penetrating abdominal trauma is considered a positive finding on DPL.

3. **Complications.** DPL can produce false-positive results due to bleeding near the incision or from pelvic fractures hemorrhaging into the anterior peritoneal space of Retzius. False-negative findings can occur from improper placement of the catheter and infusion of fluid into the space of Retzius. Puncture of viscera is also possible, especially in setting of pregnancy or adhesions from prior abdominal operations. An open technique is essential in such circumstances. Although it is associated with certain complications, DPL is a safe, simple, reliable procedure for detecting intraabdominal injuries with excellent sensitivity (>95%).
- C. **CT imaging in trauma care.** CT imaging has emerged as a powerful tool in the evaluation of the trauma patient. Since its inception, its role has expanded from assessing intracranial injuries (1970s) to whole body imaging. Although it is an extremely useful modality, three points must be kept in mind when deciding to perform a CT in the trauma setting.
1. **Patient status.** The overall status of the trauma patient is crucial in determining whether CT is the proper modality to use. Even though a CT facility may be located adjacent to a resuscitation area, a patient is often inaccessible while centered in the CT torus for an examination. Unstable patients therefore should not undergo CT imaging. Instead, they should have specific interventions performed to stabilize vital functions.
 2. **Appropriateness of CT evaluation.** CT imaging may be an inappropriate modality early in the course of a trauma patient. For example, a patient with evidence of free air on plane radiography requires emergent celiotomy for treatment of a ruptured hollow viscus, not an immediate CT evaluation of the abdomen. Furthermore, a high-resolution CT of a facial injury should not delay treatment of more immediate life threats. CT would be an appropriate modality, however, to determine major organ system injuries in a patient without significant external evidence of trauma. It is also particularly effective in recreating the trajectory of a penetrating missile and its relationship to major vascular or aerodigestive structures. Soft-tissue hemorrhage and edema, bone and missile fragments, passenger foreign bodies, and soft-tissue gas can be followed through successive CT panels to determine the course of the missile through the body and its relationship to important structures.
 3. **Order and timing of CT evaluations.** Often, trauma patients will require multiple CT examinations as part of their diagnostic evaluation. Determining the proper order of such imaging is important. Head CT should take precedent over all other CT studies because emergent craniotomy may be necessary to drain a mass lesion. Chest CT should follow the head CT (unless craniotomy is needed) when suspicion of a mediastinal hematoma (i.e., great vessel injury) exists. Abdominal CT usually follows the head and chest CTs. The patient should receive enteral contrast before arriving to the CT scanner, especially if duodenal hematoma is suspected. It is not, however, absolutely required. Intravenous contrast should be administered for chest and abdominal CTs unless contraindicated. Use of dynamic and spiral protocols improves the resolution and information of abdominal imaging. CT evaluation of bony structures (e.g., face, spine, and pelvis) often requires multiple, tightly spaced images that take time to perform. Such studies, therefore, should not be obtained immediately unless emergent operative repair is planned.
- D. **Antibiotics in trauma care.** Many injuries violate natural barriers to infection. Contamination of wounds with microorganisms therefore is common, and antibiotic therapy is used to help reduce bacterial counts to allow effective immune system response. The selection of antibiotics is based on the nature of the barrier violation and the organisms most likely to be involved.
1. **Skin injuries.** Traumatic violation of the skin is not an indication for antibiotic therapy in and of itself. Débridement and irrigation of the wound usually reduce bacterial counts to levels at which healing can occur without infection. Antibiotic therapy is indicated when the patient is at high risk for infection due to age or intercurrent disease (e.g., immunosuppression). It should also be used when the potential contaminating organisms are particularly virulent (e.g., those found in pond water, human saliva, or soil). Rarely, antibiotics are given because local wound care is inadequate. A first-generation cephalosporin, such as cefazolin, should be used to cover endogenous skin flora. Clindamycin is a safe alternative when hypersensitivity to beta-lactams exists. When covering virulent organisms, specific coverage should be tailored to those most likely to contaminate the wound. Finally, with any skin wound, tetanus prophylaxis should be given when indicated.
 2. **GI injuries.** Traumatic violation of the GI tract releases anaerobes and gram-negative organisms. These bacteria usually can be treated with a second-generation cephalosporin or cephamycin, such as cefotetan or cefoxitin. Use of combination therapy, such as clindamycin with gentamicin or aztreonam, is usually reserved for patients with documented beta-lactam hypersensitivity. All patients undergoing celiotomy for abdominal trauma should receive a single preoperative dose of a broad-spectrum antibiotic with aerobic and anaerobic coverage as prophylaxis. In the setting of penetrating trauma with hollow viscus injury, the antibiotic need only be continued for 24 hours after the operation. Adjusted doses are needed for hemodynamically unstable patients.
 3. **Orthopedic injuries.** Open fractures are prone to infection. Although débridement and irrigation are the mainstay of therapy, adjunctive antibiotics are indicated. Because the most common contaminants are skin flora, a first-generation cephalosporin is often selected. An aminoglycoside is sometimes added for complex wounds.
 4. **CNS injuries.** A traumatic violation of the dura exposes the meninges, CSF, and neural tissue, predisposing them to infection. Although controversy exists regarding the necessity of antibiotic therapy, most neurosurgeons routinely prescribe a beta-lactam drug (e.g., oxacillin) that easily crosses the blood-brain barrier. Planned placement of an ICP monitoring catheter into a trauma placement does not require antibiotic therapy.
- E. **Deep venous thrombosis** with pulmonary embolus is the leading cause of preventable morbidity and mortality in trauma patients. Some form of prophylaxis is required in all such patients. When neural injuries (i.e., CNS or spinal cord) are absent, subcutaneous heparin should be administered. When lower extremity injuries do not preclude their use, sequential pneumatic compression devices are beneficial. Combination therapy of compression devices with subcutaneous heparin is thought to be synergistic. Low-molecular-weight heparin solutions are also being used in many centers as the primary form of deep venous thrombosis prophylaxis. When the preceding techniques are contraindicated, serious consideration should be given to early placement of a vena caval filter. Although such a filter does not prevent thrombosis, it substantially decreases the risk of pulmonary embolus.
- F. **Nutrition in trauma care.** Nutritional support remains a cornerstone in the care of trauma patients. In general, the passage of 5 days without protein-calorie support renders most injured patients deeply catabolic, potentially compromising their immune system. Nutrition should be started quickly, therefore, especially if a prolonged hospital course is anticipated. The GI tract is the preferred route of administration, and placement of a feeding enterostomy should be considered at the end of every celiotomy for trauma. Enteral nutrition helps maintain the immune function and integrity of the GI tract, thereby preventing bacterial translocation. Glutamine is the fuel of the enterocyte and functions as an essential amino acid in the setting of trauma. In patients without a functional GI tract, parenteral nutrition should be used. It requires central venous access for its administration, and therapeutic complications (e.g., line sepsis, vein thrombosis) tend to be greater.
- G. **Gastroduodenal ulceration.** Injured patients remain at risk for stress gastroduodenal ulceration with concomitant hemorrhage. Prophylaxis therefore is recommended. Enteral feeding remains the most effective method. When such feeding is not possible, a topical agent such as sucralfate is a safe, inexpensive alternative. Parenteral histamine-receptor blockers also prevent posttraumatic GI bleeding. Finally, newer intravenous proton pump inhibitors may find a role in prophylaxis, but their expense may prove prohibitive.
- H. **Analgesia in trauma care.** Providing adequate analgesia for a trauma patient continues to remain a therapeutic challenge. When properly administered, analgesia can control sympathetic hyperactivity and allow the patient to maintain ventilation and mobility. In general, the best techniques are those that address the injured parts directly (e.g., neuraxis and locoregional analgesia). They often, however, require time, normal coagulation parameters, and patient cooperation. When these criteria are not met, patient-controlled intravenous opiate administration is the best alternative. Most commonly, morphine sulfate is given by means of a patient-controlled analgesic device. Intermittent intravenous opiate therapy is a less optimal option. Intramuscular opiate injection is an ineffective and inefficient means of analgesic control and should be avoided.
- I. **Neuropsychiatric disorders in trauma care.** Neuropsychiatric disorders are common in trauma patients. They can include substance withdrawal, anxiety, depression, or underlying psychosis. The onset of posttraumatic delirium should always trigger a search for a toxic, metabolic, or structural cause. Failure to identify such an etiology should suggest the possibility of a treatable psychiatric disturbance. A mental health history from the patient or family obtained at admission can provide important insight into the cause of the delirium. Additionally, a high index of suspicion for substance withdrawal is needed because certain types can be fatal (e.g., alcohol). Patients undergoing withdrawal from a drug typically present with signs and symptoms in opposition to the typical effects of the substance abused. Treatment is tailored according to the withdrawal syndrome encountered.
- J. **Rehabilitation in trauma care.** Rehabilitation is a crucial aspect of trauma care, and its planning should begin at the time of admission. Contractures and pressure sores can begin within hours of injury, and as a result, standardized prevention must be initiated promptly on the arrival of the patient to the ward. Regular turning of the patient, placement of air mattresses, and elevation of distal extremities (especially the heel) off the bed can decrease the formation of debilitating pressure sores. Specially designed orthotic splints, braces, and stockings prevent joint and scar contractures that can inhibit return of function. Finally, early physical and occupational therapy initiates the recovery process and prepares the patient for the often difficult rehabilitation to daily activity.

VI. Conclusion. A chapter of this nature is brief by necessity, but a comprehensive strategy for the care of patients who have been involved with traumatic events will clearly improve outcomes. This care should be coordinated by dedicated general surgeons with an interest or special training in trauma and should ideally use surgical specialty services staffed by individuals with trauma expertise. Although all institutions will not be able to have dedicated trauma-oriented surgeons on staff, the development of statewide trauma systems facilitates the care of patients by directing care of these patients toward those institutions with appropriate resources. For these statewide trauma systems to survive and for the hospitals within the systems to remain financially intact, there needs to be ongoing governmental financial support for the care of trauma victims. Trauma is a serious public health concern that requires serious public health and governmental strategies to control and manage the complicated issues surrounding the problem.

29 Transplantation

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Transplant Organ Procurement

I. Donor selection. The greatest obstacle to organ transplantation today is the lack of suitable donor organs. The use of organs from living donors has become widely accepted for kidney transplantations and is occasionally appropriate for lung or liver transplantations. Most organs continue to be obtained from cadaveric donors. The number of patients waiting for organs still outnumbers the available organs, and the waiting list for organ transplants grows each year. In the United States, more than 75,000 people await a solid-organ transplant (<http://www.unos.org>).

II. Cadaveric donors. The causes of death in cadaveric donors are most frequently intracerebral hemorrhage or trauma. Approximately 1% is the result of primary brain cancer. Strict criteria for establishing brain death include the presence of irreversible coma and apnea and the absence of brainstem reflexes (i.e., pupillary, corneal, vestibulo-ocular, or gag reflexes). Other useful diagnostic tests include blood flow scans or arteriography and an apnea test. **Consent** is requested from the family, and inquiries into the donor's medical history should be made. Ideally, the donor should have stable hemodynamics, although vasopressors are common. The fact that a potential donor required cardiopulmonary resuscitation may not preclude donation, particularly in a witnessed arrest with prompt institution of resuscitation and recovery of vital signs. The criteria for donor organ acceptance and use are not absolute; therefore, all brain-dead patients should be considered as potential donors. Contraindications for organ donation include a history of intravenous drug abuse or the presence of a malignancy (with the exception of a primary brain tumor). No strict age criteria apply to organ donation. Organs from donors older than age 70 years are not commonly used for transplants but have been used occasionally. **Exclusion criteria** for specific organs also exist. Potential kidney donors ideally have normal renal function before brain death. Acute tubular necrosis (ATN) in the donor, underlying medical disease, or prolonged cold ischemia time may preclude the use of a donor kidney. The use of kidneys from donors with a history of hepatitis C may be used in recipients previously exposed to hepatitis C. Selection of a donor liver for a given recipient takes into account donor size, ABO blood type, age, liver function studies, hospital course, hemodynamics, and prior medical and social history. A history of diabetes or pancreatitis in the donor is a contraindication.

III. Living donors. With a growing list of patients waiting for cadaveric organs, the use of living organ donors is increasing. Most living donors are relatives (not necessarily genetically related) of the recipient. The specific aspects of living kidney or liver donors are discussed later in this chapter.

IV. Cadaver donor organ recovery. The initial dissection identifies hepatic hilar structures, including the common bile duct, portal vein, hepatic artery, and any aberrant arterial blood supply, such as a left hepatic artery branch arising from the left gastric artery or a right hepatic artery from the superior mesenteric artery (SMA). After this dissection, the liver is flushed and cooled with University of Wisconsin (UW) preservation solution (a cold-storage solution containing a high concentration of potassium, lactobionate, hydroxyethyl starch, and other antioxidants) via cannulae placed in the portal vein and the aorta proximal to its bifurcation. The donor liver is removed with its diaphragmatic attachments, a cuff of aorta surrounding the celiac axis and the SMA, and a portion of the supra- and infrahepatic vena cava. The liver is packaged in UW solution and surrounded by iced saline during transportation. The remainder of the liver dissection is performed in the recipient's operating room under cold-storage conditions. With the advent of UW solution, donor livers can be preserved for up to 12 hours before revascularization with a low incidence of allograft dysfunction. Ideally, cold ischemia time is minimized to less than 8 hours. The donor kidneys are removed *en bloc*. The ureters are dissected widely to minimize devascularization and are divided near the bladder. The kidneys are removed *en bloc* and then separated. This technique minimizes risk of injury to the arteries and allows identification of multiple renal arteries, if present.

V. Histocompatibility. Antibodies to human leukocyte antigen (HLA) do not occur naturally but are produced in response to exposure to foreign histocompatibility antigens that may occur after pregnancy, blood transfusions, or previous transplants. The traditional test used for detecting specific sensitization against donor histocompatibility antigens is termed a **cross-match** or **complement-dependent lymphocytotoxicity assay**. Several methods are available for performing the cross-match, each involving the addition of recipient serum, donor cells (T cells, B cells, or monocytes), and complement. If specific antidonor antibodies are present, antibody binding results in complement fixation and lysis of the donor lymphocytes. Flow cytometry also can be used for cross-matching. This method permits the detection of noncytotoxic antibodies and allows the definition of cell specificity or antibody binding. Polymerase chain reaction (PCR) and enzyme-linked

immunosorbent assay technology are being used increasingly for HLA typing, particularly for the major histocompatibility (MHC) class II HLA antigens. Enzyme-linked immunosorbent assay technology is being used increasingly for pretransplant screening of MHC class I and class II antibodies. HLA matching is of practical significance only in renal and pancreatic transplantation.

Immunosuppression

A variety of immunosuppressive medications are used in transplant patients to prevent rejection. The protocols outlined in the following sections are the ones currently in use at Washington University and are intended to serve as a guideline. Variations on these protocols are used at other transplantation centers. Most protocols rely on the use of several drugs due to the different mechanisms and synergies of these medications.

I. Immunosuppressive medications

- A. **Prednisone or methylprednisolone sodium succinate (Solu-Medrol).** Steroids are part of most multiple-drug immunosuppressive regimens and are the first-line drug in the treatment of rejection. Steroids modify antigen processing and presentation, inhibit lymphocyte proliferation, and inhibit cytokine and prostaglandin production. After surgery, patients are placed on a steroid taper and maintained on a low dose of prednisone. The acute and chronic side effects of steroid therapy may include diabetes mellitus, infections, cataracts, hypertension, weight gain, and bone disease.
- B. **Cyclosporine (Sandimmune, Neoral, Gengraf).** Cyclosporine's development and use has been one of the most significant advancements in transplantation. It is a small, fungal cyclic peptide that blocks T-cell activation, inhibiting T-lymphocyte proliferation, interleukin-2 (IL-2) production, IL-2 receptor expression, and interferon-gamma release. Two-hour peaks, 12-hour troughs, or both, are monitored and increased until the serum trough level is between 100 and 300 ng/mL, depending on time from transplant, HLA match, and so forth. Side effects include nephrotoxicity, hypertension, tremors, seizures, hyperkalemia, hyperuricemia, hypercholesterolemia, gingival hyperplasia, and hirsutism. Cyclosporine is metabolized by the liver. Cyclosporine levels may be increased or decreased with the administration of other medications.
- C. **Tacrolimus (Prograf, FK506)** is a macrolide that has a mechanism of action similar to that of cyclosporine but is approximately 100 times more potent. Tacrolimus doses are adjusted to maintain 12-hour trough levels between 5 and 10 ng/mL. Current indications include prophylaxis of organ rejection in patients receiving allogeneic kidney or liver transplants. The side effect profile is similar to that of cyclosporine but does not include hirsutism and gingival hyperplasia.
- D. **Azathioprine (Imuran)** is an antimetabolite that is a thioguanine derivative of mercaptopurine. This purine analogue alters the function or synthesis of DNA and RNA, inhibiting T- and B-lymphocyte proliferation. One of the major side effects of this drug is bone marrow suppression, manifested as leukopenia and thrombocytopenia. An important drug interaction occurs with allopurinol, which blocks the metabolism of azathioprine and increases the degree of bone marrow suppression.
- E. **Mycophenolate mofetil (CellCept)** is a relatively selective inhibitor of T- and B-cell proliferation, cytotoxic T-cell generation, and antibody formation. It is used in transplant patients who are considered at high immunologic risk for rejection [i.e., those with second transplants, high panel-reactive antibodies (PRAs), or a positive B-cell cross-match before transplantation]. Mycophenolate mofetil is also used as a substitute for azathioprine in transplant patients with gout that requires treatment with allopurinol (which precludes the use of azathioprine). Major toxicities include gastrointestinal (GI) disturbances and increased cytomegalovirus (CMV) infection.
- F. **Polyclonal antithymocyte antibodies.** Polyclonal antibodies are immunologic products with antibodies to a wide variety of T-cell antigens, adhesion molecules, costimulatory molecules, cytokines, the T-cell receptor, and classes I and II MHC molecules. The two preparations available in the United States are thymoglobulin and ATGAM. **Thymoglobulin**, a rabbit-derived product, is more potent than ATGAM, a horse-derived product. Common side effects of both include fever, leukopenia, and thrombocytopenia.
- G. **Monoclonal antibodies. OKT3** is a murine monoclonal antibody that recognizes the T-cell receptor and blocks antigen recognition, hindering T-cell effector functions and potentiating T-cell lysis. OKT3 usually is administered to patients with steroid-resistant, severe rejection. It can also be given to patients who have allergies or preformed antibodies to ATGAM or thymoglobulin. OKT3 is administered at a dosage of 5 mg i.v. slowly over 5 minutes for 7–10 days. Immediate side effects can include fever, chills, hypotension, respiratory distress, and pulmonary edema, all of which are secondary to the **cytokine release syndrome**. Daclizumab (Zenapax) and basiliximab (Simulect) are IL-2 receptor–specific monoclonal antibodies indicated for the prophylaxis of renal rejection. Both have been shown in trials to be more potent than placebo with few side effects.
- H. **Other antibodies** affecting the activation or costimulation of T cells are being explored as immunosuppressants. Leflunomide (Arava) is a pyrimidine antagonist that is approved for use in rheumatoid arthritis but has been used in some transplant recipients. The usual dose is 20 mg per day. It may help prevent or reverse chronic rejection experimentally and has antiviral activity for CMV and herpes simplex virus (HSV). Patients on rapamycin (Sirolimus) showed a delay in the time to first acute rejection episode and decreased frequency of moderate and severe rejection in one multicenter trial (*Lancet* 356:194, 2000). Antibodies against costimulatory molecules, including leukocyte function–associated antigen 1, intercellular adhesion molecule 1, CD4, and CD52, are currently being tested for their potential efficacy in the treatment of rejection.

II. Complications of immunosuppression

- A. **Bacterial infections.** Infectious complications after transplantation characteristically are caused by opportunistic organisms. Routine bacterial infections, such as pneumonia or urinary tract infections, can occur.
- B. **Viral infections.** The most common viral infections after transplantation include **CMV**, **Epstein-Barr virus (EBV)**, and **HSV**. See [Table 29-1](#) for a summary of prophylaxis and treatment for common viral and fungal infections.

Table 29-1. Prophylaxis and treatment of infections in immunosuppressed patients

1. **CMV** infection can occur at any time after transplantation but is most commonly seen 4–6 weeks after increases in immunosuppression for rejection. CMV infrequently infects the recipient's liver, lungs, or gastrointestinal tract. Signs and symptoms of CMV infection include fever, chills, malaise, anorexia, nausea, vomiting, cough, abdominal pain, hypoxia, leukopenia, and elevation in liver transaminases. CMV antibody titers [immunoglobulin (Ig) G, IgM] and peripheral blood PCR are the most common tools for diagnosing CMV infection. CMV can be associated with significant morbidity and even mortality, but it typically responds well to early diagnosis and treatment.

Prophylactic ganciclovir administration (1,000 mg p.o. t.i.d. for 3–6 months) may be useful in any patient who receives a CMV-positive allograft because many of these patients develop a significant CMV infection if left untreated. Treatment consists of decreasing immunosuppression and administering ganciclovir (5 mg/kg i.v. every 12 hours for 3 weeks), which inhibits DNA synthesis. Ganciclovir dosing must be adjusted for renal dysfunction. The most common side effects of ganciclovir are anemia, neutropenia, and thrombocytopenia.

2. **EBV** can infect B cells at any time after transplantation and may be associated with the development of a lymphoproliferative disorder (lymphoma), usually of B-cell origin. Infiltration of the hematopoietic system, CNS, lungs, or other solid organs may occur. The patient usually presents with fever, chills, sweats, enlarged lymph nodes, and an elevated uric acid. Diagnosis is made by physical examination; EBV serology; CT scan of the head, chest, and abdomen looking for lymph nodes or masses; and biopsy of potential sites or lesions. Treatment consists of reducing or withdrawing immunosuppression. Intravenous ganciclovir inhibits EBV-associated DNA polymerase and may be added but is not of proven benefit. Acyclovir prophylaxis for life (200 mg b.i.d.) may be considered in EBV donor +/recipient – patients. Additionally, standard chemotherapy should be considered for advanced disease or polyclonal tumors that have not responded to other measures.
3. **HSV** causes characteristic ulcers on the oral mucosa, in the genital region, and in the esophagus. Renal transplant patients are given prophylactic acyclovir at a dosage of 200 mg p.o. b.i.d. for 3 months. Active HSV infections are treated by decreasing the patient's immunosuppression and instituting acyclovir therapy (5–10 mg/kg i.v. every 8 hours for 7–10 days). Side effects of acyclovir are rare but include nephrotoxicity, phlebitis, bone marrow suppression, and CNS toxicity.
- C. **Fungal infections** can range from mild asymptomatic colonization to lethal invasive infections. Oral **candidiasis** can be prevented and treated with oral nystatin (Mycostatin, 500,000 units p.o. q.i.d. for 3 months). Esophageal candidiasis can be treated with a short course of intravenous amphotericin B or fluconazole (100 mg p.o. b.i.d.). Serious fungal infections are treated with intravenous amphotericin B.
- D. **Other opportunistic infections. *Pneumocystis carinii* pneumonia** is a potentially lethal pneumonia that occurs in 5–10% of renal transplant patients receiving

no prophylactic treatment. Patients typically present with fever, dyspnea, nonproductive cough, hypoxia, and pulmonary infiltrates. The diagnosis is made by bronchoalveolar lavage or a lung biopsy. It can be prevented by low-dose trimethoprim-sulfamethoxazole or inhaled pentamidine. Treatment of pneumonia involves much higher doses of these agents, with a concomitant decrease in immunosuppression.

- E. **Malignancies.** Some of the cancers that occur at a higher frequency in transplant recipients than in the general population are squamous cell carcinoma, basal cell carcinoma, Kaposi's sarcoma, lymphomas, hepatobiliary carcinoma, and cervical carcinoma. Other common cancers do not have a higher incidence among transplant recipients.

Dialysis

Acute renal failure (ARF) is defined by a rise in the serum creatinine of more than 0.5 mg/dL when the baseline serum creatinine is less than 3.0 mg/dL, or more than a 1.0-mg/dL rise when the serum creatinine baseline is 3.0 mg/dL or above. **End-stage renal disease (ESRD)** results when the functioning renal mass deteriorates to less than 10–20% of normal. ESRD may affect multiple organ systems, resulting in altered fluid and electrolyte homeostasis, accumulation of metabolic waste products, anemia, hypertension, and metabolic bone disease. Currently, there are more than 400,000 patients receiving therapy for ESRD in the United States, with 75% being maintained on dialysis.

I. Treatment of renal failure

- A. **Conservative** treatment of renal failure begins with dietary restrictions. Fluid intake is limited to urine output plus insensible losses, usually 1.5–2.0 L per day. Protein is restricted to 0.7–1.2 g/kg per day to minimize the rise in blood urea nitrogen (BUN). Sodium chloride is restricted to 2 g per day (sodium, 35 mEq per day), potassium chloride is restricted to 2 g per day (potassium, 25 mEq per day), and phosphorus, magnesium, and aluminum are avoided as much as possible. Control of serum phosphorus can be accomplished through the use of calcium carbonate (500–2,500 mg p.o. within 30 minutes of meals and at bedtime). Sodium bicarbonate (650 mg p.o. b.i.d.–q.i.d.) is used to control acidosis when the serum bicarbonate is less than 20 mg/dL.

Loop diuretics in combination with thiazide or thiazidelike diuretics are useful adjuncts to maintain fluid homeostasis. Typically, chlorthalidone (500 mg i.v. b.i.d.) or metolazone (5 mg p.o. b.i.d.) followed by furosemide (80–200 mg i.v. b.i.d.) is given. The thiazide prevents distal tubule adaptation and salt reclamation, which occurs after administration of furosemide. When conservative therapy is inadequate, death ensues without either dialysis or transplantation.

- B. **Dialysis** removes fluids and wastes and adjusts acid-base and electrolyte disturbances by diffusion and osmosis across a semipermeable membrane. This is accomplished in hemodialysis (HD) by the semipermeable membrane of an artificial kidney or in peritoneal dialysis (PD) by the semipermeable peritoneal membrane. Both methods may be used either acutely or chronically.
- C. **Transplantation** is the treatment of choice for many patients with ESRD.
- D. **Mortality** for ARF is as high as 50% for patients admitted to the intensive care unit with ARF, despite the availability of dialysis. This high mortality is generally due to the patient's underlying disease processes and associated comorbidity.

II. Indications for dialysis can be remembered by the mnemonic AEIOU-PP.

- A. **Acidosis.** Dialysis for acidosis should be considered when the serum bicarbonate is less than 10 mg/dL and administration of further alkali is contraindicated.
- B. **Electrolyte disturbances.** A serum potassium acutely greater than 6 mEq/dL is the most common indication for dialysis. Sodium, calcium, and magnesium also can be corrected with dialysis.
- C. **Intoxicants.** Common dialyzable intoxicants include lithium, ethanol, methanol, salicylates, and theophylline.
- D. Fluid **overload** that is unresponsive to diuretics is another common indication for dialysis.
- E. **Uremia** is a syndrome of vomiting, anorexia, nausea, itching, listlessness, and asterixis associated with renal failure. A patient's symptoms may not correlate directly with the degree of azotemia because many uremic toxins are not yet identified. Furthermore, in ARF, no survival advantage is conferred by early dialysis for patients with azotemic symptoms.
- F. **Pericarditis** and **polyneuropathy** are two absolute indications for dialysis caused by uremia. Pericarditis due to uremia may not necessarily present with elevated ST segments on electrocardiography. Pericardial friction rubs often are ephemeral and may require repeated examinations to detect. Polyneuropathy most commonly manifests as a wrist or foot drop. Without early dialysis, these symptoms may become irreversible.

III. HD

- A. **HD access.** The types of vascular access that can be placed for HD include primary arteriovenous fistulas, polytetrafluoroethylene grafts, and percutaneous intravenous catheters. Primary arteriovenous (Brescia-Cimino) fistulas are generally formed between the radial artery and cephalic vein at the wrist. They have the longest complication-free patency, but not all patients are candidates for this type of fistula. When these vessels are not adequate, a polytetrafluoroethylene-loop arteriovenous graft is placed between the brachial artery and vein, at the elbow. Shorter-term venous access for HD may be obtained by placing a large (11.0–13.5 French) double-lumen dialysis catheter into one of the large central veins (preferably the jugular but alternatively the subclavian or femoral veins).

B. Types of HD

1. **Chronic intermittent maintenance HD** usually requires dialysis three times a week for 3–4 hours per treatment. Determination of dialysis adequacy uses clearance of BUN as a marker for treatment of uremia. For HD, a urea reduction ratio (predialysis BUN – postdialysis BUN/predialysis BUN) of more than 70% or a KT/V of more than 1.3 [where K is the clearance of the dialyzer (in mL per minute), T is the duration of dialysis in minutes, and V is the volume of distribution of urea (in mL)] confers a survival advantage in chronic renal failure.
2. **Continuous arteriovenous hemofiltration (CAVH).** This method of HD and **continuous venovenous hemofiltration (CVVH)**, also known as **slow continuous ultrafiltration**, are the methods of HD used most frequently in critically ill patients with hemodynamic instability and volume overload. Because the patient's arterial BP provides the ultrafiltration pressure, a systolic pressure of 80 mm Hg is required to support CAVH. The access for CAVH is obtained by placing a 7 French single-lumen catheter into a femoral artery and a large central vein. CVVH is accomplished with a double-lumen venous cannula. The ultrafiltrate is essentially plasma, and its rate of collection may exceed 1,000 mL per hour.

C. Complications of HD

1. **Access.** Difficulty with access sites continues to be one of the major problems complicating HD. Arterial and venous stenosis, clotting, and infection are the most common problems encountered. As previously mentioned, the primary Brescia-Cimino fistulas have the lowest rate of complications. However, patients with small or scarred veins may not be candidates for these fistulas. The polytetrafluoroethylene graft can establish HD access in most individuals. However, more than 50% thrombose at least once in the first year, and 70% thrombose within 2 years of placement. These prosthetic grafts also are more prone to infection. The steal syndrome is another complication of either type of arteriovenous fistula. Blood flows from the artery anastomosed to the low-resistance vein and “steals” additional blood flow in a retrograde fashion from the patient's hand and forearm, resulting in ischemia. The steal syndrome occurs more frequently in end-to-side anastomoses, and “banding” of the graft or ligation of the distal artery can correct this problem, assuming that the palmar arch is intact.

The use of temporary catheters may be associated with inadequate flow rates, high recirculation between the proximal and distal side holes of the dialysis catheter, frequent thrombosis, venous stenosis, and infection (up to 20% within 2 weeks). Subclavian vein stenosis related to the prior placement of a percutaneous temporary catheter in the subclavian vein can cause subsequent problems with clotting or arm swelling for fistulas subsequently placed in that arm. Therefore, the subclavian vein should be avoided as a site for venous access if at all possible. Interventional radiologists can be extremely valuable in diagnosing and, for some lesions, treating venous stenosis with angioplasty or stents.

2. **Hypotension** commonly occurs with HD and may occur even without ultrafiltration. Measures to prevent it include the use of bicarbonate (rather than acetate) dialysis fluid; low-temperature (35°C) dialysate; infusion of a saline, blood, or albumin prime at the beginning of dialysis or during the dialysis run; high-sodium dialysate; or ultrafiltration without HD during the first hour.
3. **Dyspnea.** Activation of complement and adhesion molecules on circulating leukocytes and endothelium results in leukocyte pooling in the pulmonary circulation and dyspnea. Use of biocompatible cellulose acetate or synthetic membranes helps to prevent dyspnea.
4. **Bleeding** occurs secondary to dysfunctional platelets associated with uremia or the use of heparin. Bleeding can be minimized by performing dialysis with low-dose or no heparin or by preventing clotting with the use of citrate or frequent flushes with saline.
5. **Disequilibrium syndrome** is characterized by mental status deterioration associated with dialysis and is due to the rapid removal of metabolic waste

products or fluid and electrolyte shifts. It may be prevented by limiting the percent reduction in urea to 25% with the first dialysis session.

IV. PD

- A. **Technique.** Access is gained through an intraperitoneal soft silicone (Tenckhoff) catheter. Continuous ambulatory PD requires 4–5 exchanges of 2–3 L of dialysis fluid daily. The dialysis fluid is composed of dextrose at various concentrations (1.5%, 2.5%, or 4.0%) and electrolytes. Each exchange dwells in the peritoneum for 2–4 hours, with the last exchange of the day remaining overnight. A typical exchange takes 20–40 minutes. Some patients are able to perform continuous cycler-assisted PD. This form of PD uses a machine to warm, infuse, and drain 10–16 L of dialysis fluid overnight while the patient sleeps. For PD, a weekly total KT/V of 2.0 per week and total creatine clearance of 60 L per week per 1.73 m^2 is considered minimal adequate dialysis. Acute PD is used when acute HD cannot be performed, most often owing to hemodynamic instability.
- B. **Complications of PD**
1. **Peritonitis.** Despite improvements in equipment and techniques (i.e., Y-system, bagless system, and ultraviolet light box disinfectant bag spiker), one-third of patients on PD develop peritonitis each year. The diagnosis is suggested by a fever, abdominal pain, cloudy dialysate fluid, more than 100 polymorphonuclear cells/mL of PD fluid, bacteremia, or a positive peritoneal fluid Gram stain or culture. The infection is usually caused by gram-positive organisms. Less frequent causes include gram-negative bacteria, yeast, and mycobacteria or atypical mycobacteria. Treatment usually is empiric, with antibiotics added to the dialysate. Fungal and pseudomonal peritonitis are difficult to eradicate and often require removal of the PD catheter.
 2. **Access.** Catheter malposition, obstruction, leakage, and (rarely) bowel perforation are complications associated with PD catheters. Exit-site infections occur commonly and can often be treated locally. A tunnel infection is more difficult to eradicate and often leads to peritonitis.
 3. **Obesity.** Dextrose is the osmotic agent used in PD and provides 200–500 calories per exchange. With normal serum glucose, 80% of the dextrose is absorbed. This caloric intake must be considered relative to the patient's needs.
 4. **Membrane failure** occurs eventually and more frequently after episodes of peritonitis. After 1 year, only one-half of the patients who start PD are able to remain on it.
 5. **Other.** Abdominal wall hernias, low back pain, and protein loss are also potential complications of PD.

Kidney Transplantation

I. **Indications** for renal transplantation include the presence of ESRD with an irreversible glomerular filtration rate of less than 20 mL per minute. Excellent short-term and long-term results can be achieved regardless of the cause of renal failure ([Table 29-2](#)). Renal failure secondary to diabetes mellitus, once thought to be a contraindication to transplantation, is now the single most common disease process in the United States requiring renal transplantation, comprising as many as 25% of all cases.

Table 29-2. Causes of renal failure requiring renal transplantation

II. **Contraindications.** Although the indications for transplantation are broad, some conditions must be considered contraindications.

- A. **Recent or metastatic malignancy.** There are two main reasons for excluding patients with malignant disease: (1) Immunosuppressive drugs may unfavorably influence the natural history of the malignancy, and (2) it is not reasonable to offer transplantation to someone whose life expectancy is shortened by malignant disease. In general, most transplantation centers require a 2-year disease-free interval after the treatment of a malignant tumor.
- B. **Chronic infection.** The presence of any chronic infection precludes transplantation and the use of immunosuppressive therapy. If the infection can be treated either medically or surgically, the patient should be reconsidered for transplantation after therapy. Infection with the human immunodeficiency virus (HIV) is a strong contraindication to renal transplantation at most centers.
- C. **Severe extrarenal disease** may preclude transplantation in certain circumstances, either because the patient is not an operative candidate or because the transplantation and associated immunosuppression may accelerate disease progression (i.e., chronic liver disease, chronic lung disease, and advanced uncorrectable heart disease). Severe peripheral vascular disease may also be a contraindication.
- D. **Noncompliance.** Any patient with a history of repeated noncompliance with medical therapy should be considered high risk. A period of compliance before being placed on the waiting list is generally advised. This is especially true of adolescent patients.
- E. **Psychiatric illness.** Organic mental syndromes, psychosis, and mental retardation that impairs the patient's capacity to understand the transplantation procedure and its complications are contraindications to transplantation. Patients with alcohol or other drug addiction must enter and prove completion of a rehabilitation program before being offered transplantation.

III. **Preoperative workup and evaluation.** Patients referred to a transplantation center are seen by the transplantation surgeon, nephrologist, social worker, and transplantation coordinator. Evaluation of a potential recipient is outlined in [Table 29-3](#). The evaluation identifies coexisting problems or disease entities that must be addressed to improve the outcome of the transplantation. Family history is important because it may provide information about the patient's kidney disease and allows a discussion about potential living donors (i.e., polycystic kidney disease). When the evaluation is complete, the patient is presented at an evaluation committee meeting, where a decision is made as to whether to accept the patient as a potential recipient. Allocation of a given organ to a specific patient is determined from a computer-generated algorithm run by the United Network for Organ Sharing and is based on specific criteria, including blood type, HLA matching, waiting time, prior sensitization (i.e., high PRA), and medical urgency. Once a patient is active on the waiting list, blood is sent monthly to the tissue-typing laboratory for cross-matching and to determine the PRA.

Table 29-3. Pretransplantation evaluation of renal transplant recipients

The **PRA** helps predict the likelihood that a patient will have a positive cross-match. It is determined by testing the potential recipient's serum against a panel of cells of various HLA specificities in a manner similar to the cross-match. The percentage of specificities in the panel that the patient's sera react with is the PRA. Most normal individuals do not have preformed anti-HLA antibodies and thus have low PRAs (0–5%). Patients who have been exposed to other HLAs through blood transfusions, previous transplantations, or pregnancies or who have autoimmune diseases with antibodies recognizing HLAs may have high PRAs. These patients are more likely to have a positive cross-match.

- A. **Special considerations.** The lower urinary tract should be sterile, continent, and compliant before transplantation. In patients with a history of bladder dysfunction, diabetes mellitus, and recurrent urinary tract infections, a voiding cystourethrogram should be obtained before transplantation. Transplant ureter implantation into the native bladder is preferred and usually can be achieved, even in small bladders and those that have been diverted previously.
- B. **Pretransplantation native nephrectomy** has been avoided secondary to anemia that develops on dialysis and is only performed in patients with chronic renal parenchymal infection, infected renal calculi, heavy proteinuria, intractable hypertension, massive polycystic kidney disease with pain or bleeding, renal cystic disease that is suspicious for carcinoma, and infected reflux nephropathy. Erythropoietin renders pretransplantation nephrectomy more acceptable, especially in patients with intractable hypertension whose posttransplantation management can be difficult without nephrectomy.
- C. **Living donors.** With the referral of an increasing number of patients with ESRD for transplantation, efforts to increase the number of available donor organs have emphasized the use of living donors. Most living donors are close relatives of the recipient. Parent-child or sibling combinations are the most common. Advantages of living-donor transplantation include improved short- and long-term graft survival (1-year survival >95%); immediate allograft function, planned

operative timing to allow for optimization of the recipient's medical condition (and, in many cases, avoidance of dialysis), fewer rejection and infection episodes, and shorter hospital stays. Although expanded-criteria cadaveric donors (e.g., older kidneys) have increased the donor pool, a living donor is preferred to a cadaveric donor, if available. The use of unrelated, emotionally committed donors (e.g., husband and wife) also is acceptable and provides results equivalent to those of transplants between genetically related pairs (*N Engl J Med* 333:333, 1995).

The primary goal in evaluating a potential living donor is to ensure the donor's well-being and safety. The donor must be in excellent health and must not have any illnesses, such as hypertension or diabetes, that may threaten his or her own renal function in the future. The donor anatomy is evaluated preoperatively with arteriography, or MR angiography at some centers. Experience with laparoscopic donor nephrectomy continues to increase with graft survival and morbidity results favoring minimally invasive approaches (*Ann Surg* 232:392, 2000).

IV. Preoperative considerations. When a kidney becomes available, the recipient is admitted to the hospital, and the surgeon, nephrologist, and anesthesiologist perform a final preoperative evaluation. Routine laboratory studies and a final cross-match are performed. Additionally, preoperative immunosuppression is begun with azathioprine. The need for preoperative dialysis depends on the patient's volume status and serum potassium. Generally, a patient with evidence of volume overload or a serum potassium greater than 5.6 mEq/L requires preoperative dialysis. However, perioperative dialysis negatively affects short- and long-term outcome.

V. Operative considerations

- A. **Technique.** In the operating room, a Foley catheter is inserted, and the patient's bladder is irrigated with antibiotic-containing solution. A central venous pressure (CVP) line is inserted, and a first-generation cephalosporin is administered. Antilymphocyte therapy is administered at the beginning of the operation to limit T-lymphocyte exposure to donor antigen. The renal vein and artery typically are anastomosed to the external iliac vein and artery, respectively. A heparin bolus of 3,000 units is administered before venous clamping. Before reperfusion of the kidney, mannitol (25 g i.v.) and furosemide (100 mg i.v.) are administered, and the patient's systolic BP is maintained at 120–140 mm Hg, with a CVP of at least 10 mm Hg to ensure optimal perfusion of the transplanted kidney. The ureter can be anastomosed to either the recipient bladder or the ipsilateral ureter, although the bladder is the preferred location. Establishing an antireflux mechanism is essential to prevent posttransplantation reflux pyelonephritis. Indwelling ureteral stents usually are not required.
- B. **Intraoperative fluid management.** The newly transplanted kidney is sensitive to volume contraction, and adequate perfusion is essential for immediate postoperative diuresis and ATN prevention. Volume contraction must not occur, and volume status is constantly monitored by the patient's cardiac function, CVP, and BP. The initial posttransplantation urine outputs can vary dramatically based on many factors. It is imperative to know the patient's native urine volume to assess the contribution of the native and the transplanted kidney to posttransplantation urine output. Dopamine may be administered at a level of 2–5 µg/kg per minute i.v. to promote renal blood flow.

VI. Postoperative considerations

- A. **General care.** Many aspects of postoperative care are the same as those for any other general surgical patient. Early ambulation is encouraged, and the need for good pulmonary toilet and wound care remains the same. Due to immunosuppression, sutures and skin staples are left in place for 2–3 weeks to allow for slower wound healing. The bladder catheter is left in place for 3–7 days. Meperidine is avoided because its metabolites are excreted renally and can rise to toxic levels in the patient whose allograft is not functioning immediately after transplantation.
- B. **Intravenous fluid replacement.** In general, the patient should be kept euvolemic or mildly hypervolemic in the early posttransplantation period. Hourly urine output is replaced with one-half normal saline on a milliliter-for-milliliter basis because the sodium concentration of the urine from a newly transplanted kidney is 60–80 mEq/L. Insensible fluid losses during this period typically are 30–60 mL per hour and essentially are water that can be replaced by a 5% dextrose solution at 30 mL per hour. Therefore, during the early posttransplantation period, the patient's intravenous fluid consists of 5% dextrose with one-half normal saline at a rate equal to the previous hour's urine and nasogastric output plus 30 mL. This formula necessitates that the patient's volume status be assessed repeatedly. If the posttransplantation urine output is low and, after clinical and hemodynamic evaluation, the patient is thought to be hypovolemic, isotonic saline boluses are given. Potassium chloride replacement usually is not required unless the urine output is very high and even then should be given with great care. Potassium chloride especially should be avoided in the oliguric posttransplantation patient.
- C. **GI tract.** Gastritis and peptic ulcer disease occur secondary to steroid therapy in the transplantation patient. Therefore, patients are prophylactically treated with famotidine (20 mg per day p.o. or i.v.). If severe gastroesophageal reflux, leukopenia, or mental status changes occur while the patient is receiving famotidine, omeprazole may be given (20 mg per day p.o.).
- D. **Renal allograft function or nonfunction.** If the patient's urine output is low in the early postoperative period (<50 mL per hour), volume status must be addressed first. If the patient is hypovolemic, 250–500 mL isotonic saline should be given in bolus fashion and repeated once, if needed. If the patient is euvolemic, the bladder catheter should be irrigated to ensure patency. If clots are encountered, a larger catheter may be needed. If the catheter is patent and the patient is euvolemic or hypervolemic, furosemide (100–200 mg i.v. for recipients of cadaveric transplants, 20–40 mg i.v. for those with living-donor transplants) should be given. If diuresis follows these maneuvers, urine output is again replaced milliliter for milliliter with one-half normal saline.

Early nonfunction of a transplanted kidney is most commonly due to reversible ATN. Ischemia of the kidney is the most frequent cause, due to hypotension in the donor, warm ischemia during procurement, prolonged cold ischemia, or excessive warm ischemia during the transplantation procedure. Immunologic injury and reperfusion injury also may play some role in the mechanism of injury leading to ATN. Before the diagnosis of ATN can be made, however, noninvasive studies (renal Doppler ultrasonography or technetium-99m renal scan) demonstrating vascular patency and good renal blood flow in the absence of hydronephrosis or urinary leak (renal ultrasonography) must be obtained. If flow is confirmed, dialysis can be continued until the transplanted kidney recovers.

- E. **Immunosuppression.** A variety of immunosuppressive protocols exist. The protocol currently in use at Washington University is outlined here. On posttransplantation days 0–30, patients receive cyclosporine (Neoral, 4 mg/kg p.o. b.i.d.) to maintain a trough level of 300 ng/mL; azathioprine (5.0 mg/kg p.o. for days 0, 1, and 2 followed by 2.5 mg/kg p.o. at bedtime); and methylprednisolone (1 mg/kg p.o. or i.v. every morning for days 0–13 and 0.5 mg/kg p.o. every morning for days 14–30). Additionally, as induction therapy, 1.5 mg/kg thymoglobulin is given during the first 3 posttransplantation days.

VII. Rejection. An allografted kidney is a foreign body that is capable of eliciting an immune response and thus can be rejected. There are several different types of rejection; some are preventable, whereas others can be treated with varying degrees of success.

- A. **Hyperacute** rejection occurs when preformed anti-HLA antibodies bind the endothelium of the allograft and initiate a cascade of events culminating in vascular thrombosis and ischemic necrosis of the graft. Hyperacute rejection usually can be prevented by cross-matching donor lymphocytes with recipient serum. Hyperacute rejection usually occurs within minutes of cross-clamp release and is irreversible. Viability of the allograft can be assessed by intraoperative biopsy. The only therapeutic option is to remove the allograft immediately.
- B. **Accelerated** rejection also appears to be antibody-mediated and usually occurs 12–72 hours after transplantation. The patient usually is anuric or oliguric and has fever and graft tenderness. Although treatment for this form of rejection is not well defined, administration of an antilymphocyte preparation may salvage the graft. Accelerated rejection can lead to an immunologically mediated ATN from which good renal function recovery can occur.
- C. **Acute** rejection is cell-mediated and involves T lymphocytes and soluble mediators called *lymphokines*. It happens in 20–40% of patients and typically occurs 1–6 weeks after transplantation. The development of a rising creatinine should prompt the consideration of rejection. Technetium-99m renal scan demonstrates decreased but persistent perfusion. Diagnosis is confirmed by percutaneous needle biopsy. There are two basic treatment modalities ([Table 29-4](#)): high-dose methylprednisolone or an antilymphocyte preparation. The latter generally is used for steroid-resistant rejection, although antilymphocyte therapy may be used as first-line therapy for moderate or severe rejections with arteritis. More than 90% of acute rejection episodes can be treated successfully.

Table 29-4. Treatment of rejection

- D. **Chronic** rejection is a poorly understood phenomenon that can occur months to years after transplantation. Leflunomide and rapamycin have shown promise in preclinical studies, but, to date, there is no effective treatment, and eventually the kidney function deteriorates to the point that either retransplantation or dialysis is required.

VIII. Surgical complications of renal transplantation. Wound seromas, hematomas, and infections are treated according to the usual surgical principles. Other complications require special consideration.

- A. **Lymphoceles** are collections of lymph that occur because of lymphatic leaks in the retroperitoneum. They present from one to several weeks after transplantation and are best diagnosed by ultrasonography. They may produce ureteral obstruction, deep venous thrombosis, leg swelling, or incontinence secondary to bladder compression. Most lymphoceles arise from leakage of lymph from the donor kidney. Treatment consists of open or laparoscopic internal drainage by marsupialization into the peritoneal cavity. Repeated percutaneous drainage is not advised and seldom leads to resolution of the lymphocele.
- B. **Renal artery and vein thrombosis.** Arterial and venous thrombosis most often occur in the first 2–3 days after transplantation. If the kidney had been functioning, graft thrombosis should be suspected if a sudden cessation of urine output occurs. A rapid rise in serum creatinine, graft swelling, and local pain ensue. If the allograft had not been functioning or if the native kidneys make a large amount of urine, there may be no signs of graft thrombosis. The transplanted kidney has no collateral circulation and has a low tolerance for warm ischemia. The diagnosis is made by technetium-99m renal scan or Doppler ultrasonography. Unless the problem is diagnosed quickly and repair is done immediately, the graft will be lost, and transplantation nephrectomy will be required.
- C. **Urine leak.** The etiology is usually anastomotic leak or ureteral sloughing secondary to ureteral blood supply disruption. Urine leaks present with pain, rising creatinine, and possibly urine draining from the wound. Diagnosis is made by locating the fluid collection with ultrasonography and then aspirating the fluid and comparing its creatinine level to the serum creatinine level. A renal scan demonstrates radioisotope outside the urinary tract, and a cystogram shows leakage of contrast outside the bladder. Urine leaks are treated by placing a bladder catheter to reduce intravesical pressure and by early surgical exploration. The type of repair depends on the level of the leak. Bladder fistulas should be repaired primarily. If an anastomotic leak is found, the distal ureter can be resected and reimplanted. If the transplantation ureter is nonviable or of inadequate length, ureteroureterostomy over a double-J stent using the ipsilateral native ureter can be performed. The stent can be removed via cystoscopy several weeks later.

IX. Long-term follow-up. Immunosuppression (Table 29-5) and infection prophylaxis (see section VII) should be tapered with time. After the initial 3-month period, when acute rejection becomes less of a risk, cyclosporine and steroid dosages are tapered. Chronic long-term immunosuppression can be maintained at lower levels than those required for induction. However, immunosuppression can almost never be discontinued completely. Specific metabolic consequences of cyclosporine include hypertension, nephrotoxicity, hypercholesterolemia, and hyperuricemia. Gradual dose reduction can be helpful, but often, specific therapy is needed to correct these side effects. Weight gain is the predominant side effect of steroid therapy. Dietary manipulation and gradual dosage reduction are important. Long-term complications of steroids include joint deterioration with avascular necrosis, osteoporosis, cataract formation, and diabetes mellitus (10% of patients). The incidence of these problems can be minimized by using as low a dose of prednisone as possible. Antibiotic prophylaxis should be used before any surgical or dental procedure in transplantation recipients.

Table 29-5. Long-term maintenance immunosuppression for cadaveric and living-related renal transplantation

Pancreas Transplantation

I. Indications. Insulin-dependent diabetes mellitus (IDDM) affects 5% of Americans and is the fourth leading cause of death. It is the leading cause of renal failure and blindness in adults. Other long-term complications caused by diabetes include amputations, myocardial infarctions, strokes, and neuropathy. Methods to maintain euglycemia and prevent the long-term complications of IDDM include autoregulating insulin pumps, pancreatic islet cell transplants, and whole organ pancreatic transplantation.

Although isolated pancreas transplantations have been performed, the majority of pancreas transplantations have been combined pancreas and kidney transplantations in patients with IDDM and ESRD. The type 1 IDDM patient accepted for combined kidney-pancreas transplantation is similar to candidates for renal transplantation alone.

II. Contraindications to pancreas transplantation are the same as those for kidney transplantation, including disabilities secondary to IDDM, such as peripheral gangrene, intractable cardiac decompensation, and incapacitating peripheral neuropathy. Continued tobacco use also is considered a relatively strong contraindication to pancreas transplantation.

III. Preoperative workup and evaluation. The potential pancreas transplantation patient workup is similar to that of the kidney recipient and identifies coexisting diseases, as outlined in Table 29-3. To allow the identification of beneficial effects secondary to pancreas transplantation on the complications of IDDM, a careful preoperative evaluation of the patient's neurologic and ophthalmologic status should be performed.

IV. Donor pancreas procurement occurs as part of a multiorgan retrieval. Contraindications to pancreas donation include the presence of diabetes, pancreatitis, trauma to the pancreas, or a history of chronic alcohol abuse. Although the liver and pancreas may share blood supply, combined retrieval can be performed safely without compromising either organ. During the organ retrieval procedure, it is important to identify accessory or replaced hepatic arteries that may arise from the left gastric artery or the SMA. The abdominal viscera are flushed with UW solution, and the liver, pancreas, duodenum, and spleen are removed *en bloc* and separated under cold-storage conditions. The splenic artery is divided from the celiac artery, and the SMA is divided at its origin or distal to a replaced right hepatic artery, if present. The mesentery of the small intestine is either oversewn or stapled.

V. Cadaveric pancreas transplantation operation

A. Forms of pancreatic transplantation

1. **Isolated pancreas transplantation.** The most widely accepted technique of pancreatic transplantation in the United States uses whole organ pancreas with venous drainage into the systemic circulation and exocrine drainage into the urinary bladder. An increasing number of centers are advocating portal venous drainage and enteric exocrine drainage of the pancreas allograft.

Under cold-storage conditions, the portal vein is isolated. If it is too short to allow for a tension-free anastomosis, an extension autograft is placed using donor iliac vein. The SMA and splenic artery then are reconstructed with a donor iliac artery Y-bifurcation autograft. Only the second portion of the duodenum is retained with the pancreas. Then the portal vein is anastomosed to the iliac vein or the superior mesenteric vein, and the donor common iliac artery is anastomosed to the recipient's external iliac artery. The duodenal segment of the transplant is then opened, and a duodenocystostomy is created. Alternatively, the duodenal segment can be anastomosed to a Roux-en-Y limb for enteric drainage. The pancreas transplant is left in the right paracolic gutter, and if kidney transplantation is to be performed, it is done on the left side.

2. **Simultaneous kidney-pancreas transplantation** may be considered in insulin-dependent diabetic patients who are dialysis-dependent or dialysis-imminent with a creatinine clearance of less than 30 mL per minute. Some of the advantages of combined transplantation include the ability to monitor rejection of the pancreas by monitoring renal rejection and the fact that the patient is exposed to only one set of donor antigens.
3. **Pancreatic islet cell transplantation** is still in the investigational stage. Pancreatic islet cells are isolated and can be implanted at various sites, including under the renal capsule, in the splenic parenchyma, or injected into the portal vein for engraftment in the liver. The major problems encountered have been obtaining enough islet cells to attain glucose homeostasis, the difficulty of diagnosing rejection, and the failure of long-term graft survival. Investigation with fetal pancreas, which contains a higher ratio of endocrine to exocrine pancreas, is also being explored. Transplantation of adult and fetal islet cells remains investigative at this point. New immunosuppressive protocols and improved isolation techniques, however, have shown promise to what have historically been low success rates (*N Engl J Med* 343:230, 2000).

B. Exocrine drainage

1. **Bladder drainage.** Advantages of this technique include the ability to measure urinary amylase, which can facilitate the early diagnosis of rejection.

Cystoscopic transduodenal needle biopsy can also be performed in the diagnosis of rejection. The major disadvantages of bladder drainage are fluid and electrolyte disturbances and urologic complications (including hematuria, urinary tract infections, urethral strictures, or reflux pancreatitis) caused by the drainage of fluid, bicarbonate, and enzymes into the bladder.

2. **Enteric drainage.** Many programs are now using enteric drainage, which avoids the acidosis, volume depletion, and urologic complications associated with bladder drainage. Enteric drainage can be performed by anastomosing the duodenal segment to small bowel in a side-to-side fashion or via a Roux-en-Y limb. Disadvantages of enteric drainage include the inability to monitor exocrine secretions.

VI. Postoperative management and monitoring

- A. **Immunosuppression** consists of quadruple therapy (10–14 days of induction antilymphocyte therapy with OKT3, thymoglobulin, or ATGAM). Cyclosporine, prednisone, and azathioprine are started immediately after operation. A marked reduction in rejection rates in pancreas transplantation has occurred with the potent immunosuppressive regimen of tacrolimus, mycophenolate mofetil, and prednisone after induction therapy.
- B. **Serum glucose** is followed during and after the transplantation. Intravenous insulin infusions are stopped within the first few hours after pancreas transplantation.
- C. **Rejection** of the pancreas transplant is suggested by a rise in serum amylase or a fall in urinary amylase. Rejection of pancreas and kidney transplants usually occurs in parallel but at times may be discordant. The diagnosis of kidney rejection is suggested by a rise in creatinine, which is then confirmed by biopsy. Biopsy of the pancreas transplant is performed percutaneously or via cystoscopy. Rejection is treated with corticosteroids or antilymphocyte preparations.
- D. **Graft-related complications.** Besides rejection, complications of pancreas transplantation include metabolic acidosis and dehydration. These are due to the loss of sodium and bicarbonate into the urine from the transplanted duodenum, and they are avoided with enteric drainage. Other common complications include pancreatitis, urinary tract infections, urethritis, and anastomotic leak from the duodenocystostomy. Infections with CMV also may occur.

VII. Effect on secondary complications of diabetes. The full effect of pancreatic transplantation on secondary complications of diabetes is still unknown. Pancreatic transplantation may prevent the development of diabetic nephropathy in the transplanted kidney. It also may stabilize diabetic retinopathy and improve diabetic neuropathy.

Intestinal Transplantation

Intestinal failure occurs when the functioning GI tract mucosal surface area has been reduced below the minimal amount necessary for adequate digestion and absorption of food. This may be caused by intestinal loss or intestinal disease ([Table 29-6](#)). The development of total parenteral nutrition (TPN) has led to the possibility of long-term survival of infants and adults with intestinal failure. However, TPN has limitations and its own associated morbidity.

Table 29-6. Causes of intestinal failure

I. Indications. Adults and children who have documented intestinal failure without the potential for long-term survival on TPN are candidates for intestinal transplantation. **Intestinal failure** is said to occur when any child younger than 1 year requires more than 50% of his or her caloric needs from TPN after neonatal small-bowel resection, or when a child older than 4 years requires more than 30% of calories from TPN. Older children and adults receiving more than 50% of their nutritional requirements from TPN for more than 1 year also should be considered for intestinal transplantation. Other considerations include venous access, recurrent infectious complications related to central venous catheters, prolonged hospitalizations, growth retardation, and hepatobiliary dysfunction secondary to prolonged TPN.

II. Donor intestinal procurement uses multiorgan recovery techniques. The liver, duodenum, pancreas, and small intestine are removed *en bloc* and separated under cold-storage conditions.

III. Intestinal transplantation operation. Patients who receive isolated intestinal allografts have vascular anastomoses created between the donor superior mesenteric vein and recipient portal vein and between the donor SMA and recipient aorta. Vascular reconstruction of patients who receive combined liver-intestinal grafts parallels that of a standard orthotopic liver transplantation (OLT). Supra- and infrahepatic vena caval anastomoses are completed, and arterial inflow is accomplished after the portal vein anastomosis by using a patch of aorta that contains the SMA and celiac artery.

IV. Postoperative management

- A. **Immunosuppression and infectious prophylaxis.** Posttransplantation immunosuppression uses tacrolimus and prednisone. Monitoring for graft rejection is accomplished with the use of frequent endoscopic biopsies. Watery diarrhea may be a sign of either rejection or suprainfection. With the return of intestinal function, feedings are begun with an elemental diet and then advanced as tolerated. Viral and fungal infection prophylaxis includes ganciclovir, gamma globulin, oral antibiotic bowel preparation, and low-dose amphotericin B.
- B. **Potential complications.** Inherent risks with intestinal transplantation include up to 40% graft failure (rejection) at 3 years. Patient survival is 80%. Combined liver-intestine transplantation carries all the additional risks inherent to liver transplantation. Risks that are increased in intestinal transplant recipients include the development of graft-versus-host disease and posttransplantation lymphoproliferative disease. Complications related to tacrolimus-based immunosuppression include diabetes mellitus, headaches, CNS neurotoxicity, peripheral neurotoxicity, and nephrotoxicity. As with any effective immunosuppressant, there is an increased risk of infection and malignancy.

Liver Transplantation

I. Indications for hepatic transplantation are complications attributable to end-stage liver disease (ESLD). In the absence of other medical contraindications, virtually any disease resulting in ESLD is amenable to transplantation. The most common diseases for which OLT is performed are listed in [Table 29-7](#). Common indications for OLT in patients with ESLD include variceal hemorrhage, intractable ascites, intractable encephalopathy, spontaneous bacterial peritonitis, and poor synthetic function.

Table 29-7. Indications for orthotopic liver transplantation

II. Contraindications. There are a few absolute contraindications to liver transplantation, including insufficient cardiac or pulmonary reserve, recent intracranial hemorrhage or irreversible neurologic impairment, active substance abuse, intractable hypotension, evidence of infection with HIV, ongoing bacterial infection outside the biliary tract, the expected inability to comply with postoperative medications and follow-up, and extrahepatic malignancy.

Portal vein thrombosis no longer is considered a contraindication to liver transplantation because techniques are available to bypass the obstructed venous segment. Renal insufficiency, either chronic or acute, increases the morbidity of hepatic transplantation but is not a contraindication. Renal transplantation can be performed at

the time of liver transplantation for patients with ESRD. Some degree of preoperative renal insufficiency often is reversible after successful liver transplantation. Older patients and those with comorbid conditions can be considered for OLT if no absolute contraindications are present.

III. Preoperative evaluation. Referrals to transplantation centers are made on an urgent or an elective basis. The evaluation determines the need and urgency for as well as the technical feasibility of OLT.

- A. **Elective transplantation.** Under elective conditions, the potential candidate is presented to a multidisciplinary committee for evaluation. The patient's evaluation is based on history, physical examination, laboratory evaluation, results of endoscopic procedures, cardiac and pulmonary evaluation, and radiologic examination (Table 29-8). Active infection should be treated promptly, and transplantation should be postponed until the infection resolves. Patients with spontaneous bacterial peritonitis require antibiotic treatment for 48 hours before OLT, but in the emergent setting, OLT can be performed when the polymorphonuclear count of the ascitic fluid is less than 250/mL.

Table 29-8. Pretransplantation evaluation of liver transplant recipients

- B. **Urgent transplantation.** Acceptable results with OLT also can be achieved in selected patients with acute liver failure. The pretransplantation evaluation is performed in a manner similar to that outlined for the elective patient; however, timing, neurologic status, and hemodynamic stability may limit the number of tests obtained.

A careful neurologic examination must be done in this setting, and the grade of coma should be determined. Patients in grade IV (unresponsive) coma have been shown in some studies to benefit from continuous perioperative monitoring of intracranial pressure (ICP) because untreated severe elevations in ICP can result in permanent brain injury and death. An attempt is made to keep cerebral perfusion pressure (mean arterial BP minus ICP) above 60 mm Hg. Low mean arterial BP is treated with vasopressors after volume resuscitation. Elevation in ICP is treated with hyperventilation, mannitol, and elevation of the head of the bed more than 45 degrees.

Patients with acute hepatic failure may develop ARF as well, which can require hemofiltration or HD. Sepsis also is seen in acute hepatic failure and requires broad-spectrum antibiotics and antifungals. Pulmonary insufficiency is a common accompaniment of acute liver failure and may require intubation, high-concentration oxygen, and positive end-expiratory pressure.

IV. Donor selection. Selection of an appropriate donor liver takes into account donor size, ABO blood type, age, presence of infection, history of malignancy, liver function studies, hospital course, hemodynamic stability, and prior alcohol or drug use. Absolute contraindications to the use of a donor liver include the presence of extracerebral malignancy, HIV status, and hepatitis B surface or core antibody status.

V. Hepatic transplantation procedure

- A. **Whole-organ liver transplantation.** Conceptually, transplantation of the liver can be thought of as comprising three distinct sequential phases. The **first phase** involves the dissection of the recipient's diseased liver. The **second phase**, known as the **anhepatic phase**, refers to the period starting with devascularization of the recipient's liver and ending with revascularization of the newly implanted liver. During the anhepatic phase, venovenous bypass (VVB) may be used. VVB shunts blood from the portal vein and infrahepatic inferior vena cava (IVC) to the axillary, subclavian, or jugular veins. Maintenance of venous return from the kidneys and lower extremities during the anhepatic phase results in a smoother hemodynamic course, allows time for a more deliberate approach to hemostasis, reduces visceral edema and splanchnic venous pooling, and lowers the incidence of postoperative renal dysfunction. The liver allograft is implanted by anastomosing first the suprahepatic vena cava and then the infrahepatic IVC. The portal vein anastomosis is performed, and blood flow to the liver is reestablished. The patient is taken off VVB, and the hepatic arterial anastomosis is performed. If the recipient hepatic artery is not suitable for anastomosis, a donor iliac arterial graft can be used as a conduit from the infra- or suprarenal aorta. The **third phase** includes biliary reconstruction and abdominal closure. Biliary continuity is established via a duct-to-duct anastomosis over a T tube or a choledochojejunostomy. A duct-to-duct anastomosis is preferable, but it may not be possible when there is a donor-recipient bile duct size discrepancy or a diseased recipient bile duct (e.g., with primary sclerosing cholangitis, biliary atresia, and secondary biliary cirrhosis).

A modification of this technique has been used in our center for several years. The recipient's retrohepatic IVC is preserved, and the donor suprahepatic IVC is anastomosed to the confluence of the recipient's right, middle, and left hepatic veins. The donor infrahepatic IVC is then oversewn. A temporary end-to-side portacaval shunt is also created at the beginning of the hepatectomy. This technique has all the advantages of VVB without its associated risks and costs.

- B. **Reduced and split-liver transplantation** have been developed to support the needs of pediatric patients awaiting appropriately sized transplants and have been used in some adults as well. The liver has a remarkable capacity for regeneration. It can be divided based on the anatomic segments of Couinaud into a left lateral segment (segments 2 and 3), a left lobe graft (segments 2–4), or a right lobe graft (segments 5–8). The left lateral segment is most commonly used in children. Comparison of the size of the donor and the recipient is used to determine the appropriate-sized graft. The mortality of pediatric recipients on the waiting list for a liver transplant has been reduced by the use of reduced liver transplantation. In split-liver transplants, one liver is divided between two recipients.
- C. **Living-donor liver transplantation** has been developed as a result of the success of reduced liver transplantation. Most living-donor liver transplantations occur from parent to child. The left lateral segment or left lobe of the liver is usually used as the donor graft. Advantages similar to those observed with living-related kidney donors have also been observed, such as reduced ischemic time and the elective nature of the operation. Adult-to-adult living-donor liver transplantation necessitates the use of the larger right hepatic lobe. Approximately 0.1% of liver volume per patient weight (700 g for a 70-kg recipient) is required.

VI. Postoperative care

- A. **Hemodynamic.** Intravascular volume resuscitation usually is required in the immediate postoperative period secondary to third-space losses, increasing body temperature, and vasodilatation. Adequate perfusion is assessed by left and right heart filling pressures, cardiac output, urine output, and the absence of metabolic acidosis.
- B. **Pulmonary.** Ventilatory support is required postoperatively until the patient is awake and alert, is able to follow commands and protect the airway, is able to maintain adequate oxygenation and ventilation, and demonstrates adequate hepatic function.
- C. **Hepatic allograft function.** Monitoring of hepatic allograft function begins intraoperatively after revascularization. Signs of satisfactory graft function include hemodynamic stability and normalization of acid-base status, body temperature, coagulation studies, maintenance of glucose metabolism, and bile production. Reassessment of hepatic allograft function continues postoperatively, initially every 6 hours. Satisfactory hepatic allograft function is indicated by an improving coagulation profile, decreasing transaminase levels, normal blood glucose, hemodynamic stability, adequate urine output, bile production, and clearance of anesthesia. Early elevations of bilirubin and transaminase levels may be indicators of preservation injury. The peak levels of serum glutamic-oxaloacetic transaminase and serum glutamate pyruvate transaminase usually are less than 2,000 units/L and should decrease rapidly over the first 24–48 hours postoperatively. After the patient leaves the intensive care unit, liver function tests are obtained daily. Bile is inspected daily; a cholangiogram may be obtained to ensure adequate biliary drainage and to rule out extravasation. If hepatic dysfunction becomes evident at any time, prompt evaluation must be undertaken and treatment must be initiated. It is important to make the correct diagnosis about the cause of liver dysfunction, because each has its own unique treatment.
1. **Primary nonfunction and initial poor function.** The use of UW solution for organ preservation has decreased the incidence of primary nonfunction. For poorly understood reasons, however, 1% of transplanted livers fail immediately after the surgery. Primary nonfunction is characterized by hemodynamic instability, poor quantity and quality of bile, renal dysfunction, failure to regain consciousness, increasing coagulopathy, persistent hypothermia, and lactic acidosis in the face of patent vascular anastomosis (as demonstrated by Doppler ultrasonography). Without retransplantation, death ensues.
 2. **Rejection.** Rejection is relatively common after liver transplantation, with 60% of recipients experiencing at least one cell-mediated or acute rejection episode. However, rejection is an extremely uncommon cause of graft loss. The most common causes of early graft loss include primary nonfunction or hepatic artery thrombosis.
 3. **Technical complications.** A variety of technical problems can lead to liver allograft dysfunction, including hepatic artery stenosis or thrombosis, portal vein

stenosis or thrombosis, biliary tract obstruction, bile duct leak, and hepatic vein or vena caval thrombosis. **Hepatic artery thrombosis** that occurs in the early posttransplantation period may lead to fever, hemodynamic instability, and rapid deterioration of the patient, with a marked elevation of the transaminases. An associated bile leak may be noted soon after liver transplantation due to the loss of the bile ducts' main vascular supply. Acute hepatic artery thrombosis may be treated by attempted thrombectomy. If this is unsuccessful, retransplantation is needed. Hepatic artery thrombosis that occurs long after liver transplantation may produce intra- and extrahepatic bile duct strictures and may be an indication for elective retransplantation. Occasionally, hepatic artery thrombosis is completely asymptomatic.

Portal vein stenosis or thrombosis is very rare. When it occurs, the patient's condition may deteriorate rapidly, with profound hepatic dysfunction, massive ascites, renal failure, and hemodynamic instability. Urgent retransplantation is almost always necessary. Late portal vein thrombosis may allow normal liver function but usually results in variceal bleeding and ascites.

Bile duct obstruction is diagnosed by cholangiography. A single short bile duct stricture may be treated by either percutaneous or retrograde balloon dilation. A long stricture, ampullary dysfunction, or failed dilation necessitates revision of the biliary tract anastomosis. Fever and abdominal pain in the early posttransplantation period should raise the question of biliary anastomotic disruption, which requires urgent surgical revision.

4. **Recurrent infection and neoplasm.** CMV can cause hepatic allograft dysfunction. It usually occurs at the end of the first posttransplantation month and can mimic rejection. Diagnosis is made by liver biopsy, with CMV inclusion bodies being found with light microscopy or by PCR in peripheral blood. Treatment consists of decreasing baseline immunosuppression and administering ganciclovir (5 mg/kg every 12 hours via central venous access for 3 weeks).

Viral hepatitis B and C and malignancy (e.g., hepatoma, cholangiocarcinoma, neuroendocrine tumors) can recur in the hepatic allograft but are uncommon in the early posttransplantation period. The clinical presentation includes elevation of liver function tests. The diagnosis is made with liver biopsy. Imaging studies (e.g., CT scan and liver ultrasonography) are important for following patients transplanted for neoplasms. Patients transplanted for hepatocellular carcinoma also should have surveillance with tumor markers at regular intervals.

- D. **Electrolytes and glucose.** The use of diuretics may result in hypokalemia, whereas cyclosporine or tacrolimus toxicity may cause hyperkalemia. Magnesium levels are maintained above 2 mg/dL because the seizure threshold is lowered by the combination of hypomagnesemia and cyclosporine or tacrolimus. Calcium should be measured as free ionized calcium and kept above 1.1 mmol/L. Phosphorus levels should be maintained at greater than 2.5 mg/dL to avoid respiratory muscle weakness and altered oxygen hemoglobin dissociation. Glucose homeostasis is necessary because steroid administration may result in hyperglycemia, which is best managed with intravenous insulin because it is short acting and easily absorbed. Cyclosporine and tacrolimus are diabetogenic immunosuppressants and may alter glucose homeostasis. Hypoglycemia is a complication of liver failure, and in the presence of liver dysfunction, glucose administration may be necessary.
- E. **GI tract.** H₂ blockade, proton pump inhibition, and/or antacids are used to prevent stress ulcers. Endoscopy is performed liberally for any GI bleeding to determine the etiology. Nystatin and GI tract decontamination solution, containing gentamicin and polymyxin B, are used in the perioperative period to prevent esophageal candidiasis and translocation of bacterial pathogens.
- F. **Nutrition.** Patients who are severely malnourished should be placed on nutritional supplementation as soon as a stable fluid and electrolyte status and adequate graft function have been reached. Patients with adequate preoperative nutrition can be maintained on routine intravenous fluids until GI tract function returns (usually 3–5 days). Enteral nutrition is used as soon as the postoperative ileus resolves. TPN is indicated when the GI tract is nonfunctional.
- G. **Infection surveillance.** The most common causes of bacterial infection after liver transplantation include line sepsis, urinary tract infection, infected ascites, cholangitis, pneumonia, biliary anastomotic leak, and intraabdominal abscess. Prophylactic antibiotics covering biliary pathogens are administered for the first 48 hours after liver transplantation. If a fever develops in the liver transplant recipient, a thorough examination should be performed. A chest X-ray and cultures of blood, urine, indwelling lines, and bile also are necessary. A T-tube cholangiogram and Doppler ultrasonography of the liver can be performed to rule out perihepatic fluid collection and to evaluate hepatic vasculature.

Hepatitis B or C recurs in the liver allograft after transplantation. Therefore, protocols are currently under investigation using different combinations of hepatitis B Ig, hepatitis B vaccinations, lamivudine, interferon, retroviral agents, and monoclonal antibodies. The diagnosis is suspected due to a rise in liver transaminases and is confirmed by biopsy. Recurrent disease may be severe enough to lead to life-threatening hepatitis and cirrhosis. Strategies to prevent hepatitis B recurrence include the use of lamivudine (3-TC) before transplant to arrest hepatitis B virus replications and high-dose hepatitis B Ig and lamivudine after transplant. Hepatitis C recurrence after transplant, although it is ubiquitous, most commonly does not lead to significant problems for many years and is associated with a mild transaminitis.

- H. **Posttransplantation immunosuppression.** Currently, the immunosuppressive agents used to prevent rejection include corticosteroids, azathioprine, cyclosporine, polyclonal and monoclonal antibodies, and FK506. (See also the section [Immunosuppression](#).)

VII. Rejection. Many liver transplant recipients experience at least one acute rejection episode, and it commonly occurs between the fourth and twenty-first day postoperatively. Rejection is characterized by fever, increased ascites, decreased bile quality and quantity, and elevation of total white blood cell and eosinophil count, bilirubin, and transaminase levels. Liver transplant rejection is diagnosed by percutaneous liver biopsy. In the early posttransplantation period, technical causes of hepatic dysfunction are ruled out by Doppler ultrasonography to ensure vascular patency, and T-tube cholangiography is obtained to rule out a bile duct obstruction or leak. Typical biopsy findings consistent with acute rejection include a triad of portal lymphocytosis, endothelialitis (subendothelial deposits of mononuclear cells), and bile duct infiltration and damage. The first-line treatment for acute rejection is a bolus of corticosteroids (methylprednisolone, 1 g i.v.). If the rejection responds appropriately, the patient undergoes steroid recycling (methylprednisolone, 50 mg i.v. q.i.d. for 4 doses; 40 mg i.v. q.i.d. for 4 doses; 30 mg i.v. q.i.d. for 4 doses; 20 mg i.v. q.i.d. for 4 doses; 20 mg i.v. b.i.d. for 2 doses; and finally prednisone, 20 mg per day p.o.) and optimizing of the immunosuppressive regimen. If no response is seen after steroid therapy, treatment with the monoclonal antibody OKT3 or the polyclonal antibody thymoglobulin is begun. If the rejection response is recalcitrant, the patient can be converted to immunosuppression with tacrolimus.

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Introduction

Burns are tissue injuries resulting from direct contact with hot liquids, gases, or surfaces; caustic chemicals; electricity; or radiation. Most commonly, the skin is injured, which compromises its function as a barrier to injury and infection and as a regulator of body temperature, fluid loss, and sensation. More than 2 million individuals in the United States sustain burns each year, of whom more than 50,000 are hospitalized. Seventy-five percent of those hospitalized have less than 10% body-surface area (BSA) burns (*N Engl J Med* 335:1581, 1996). Advances in burn care management continue to reduce mortality and improve the quality of life for burn victims.

Assessment and Management of Burn Injuries

I. Assessment

- A. **Mechanism of injury** identified by the patient or witnesses helps to direct the assessment. Burns sustained in a closed environment, such as a structure fire, often produce inhalation injury in addition to thermal trauma. Explosions can cause barometric injury to the lungs and may cause blunt trauma. Burn source, duration of exposure, time of injury, and environment are documented carefully.
- B. **Associated injuries** may be present in the burn patient and can result from explosions, falls, or jumping in escape attempts. Fractures, abdominal organ injury, pulmonary contusion, and pneumothorax sometimes occur.
- C. **Patient age** has a major effect on outcome, with infants and elderly patients being at highest risk. Inpatient, outpatient, or burn unit management decisions also are influenced by patient age. Burns are a common form of child abuse and should be suspected when the mechanism of injury appears to be incongruent with the injury pattern. Elderly patients often have diminished organ system reserve and comorbid medical problems that place them at increased risk.
- D. **State of health.** Preexisting medical problems affecting management should be noted, including allergies, medications, hypertension, and diabetes mellitus. A careful review of systems should be obtained, with particular attention paid to cardiac, pulmonary, renal, and gastrointestinal systems.
- E. **Prehospital treatment** is ascertained and recorded, including care provided by the patient and that provided by the emergency response team. Administered fluids are documented carefully and subtracted from estimated fluid requirements for the first 24 hours of injury.
- F. **Physical examination**
 1. **Airway** assessment and support have the highest priority. Supraglottic tissue edema progresses over the first 12 hours and can obstruct the airway rapidly. The larynx protects subglottic tissue from direct thermal injury but not from injury due to inhaled toxic gases. Inhalation injury should be suspected if the patient was burned in an enclosed structure or explosion. Physical signs include hoarseness, stridor, facial burns, singed facial hair, expectoration of carbonaceous sputum, or presence of carbon in the oropharynx. Direct laryngoscopy is useful in equivocal cases but should not delay expeditious endotracheal intubation with a large-bore tube based on clinical indications. Bronchoscopy is particularly helpful in diagnosing inhalation injury in patients with clinically silent airway injuries, facilitating difficult intubations, and in predicting the onset of adult respiratory distress syndrome (*J Trauma* 36:59, 1994).
 2. **Breathing** is evaluated for effort, depth of respiration, and auscultation of breath sounds. Wheezing or rales suggest either inhalation injury or aspiration of gastric contents. Circumferential deep burn of the thorax can restrict inspiration, necessitating escharotomies in the anterior axillary lines bilaterally. Carboxyhemoglobin levels of greater than 10% indicate inhalation injury (in nonsmokers). Levels greater than 30% are associated with mental status changes, and those greater than 60% are not compatible with survival.
 3. **Circulation** is assessed for the presence of shock (rapid, weak, or absent pulse) and tissue perfusion. Signs of impairment in central perfusion include cyanosis, agitation, and reduced mentation. Intravascular volume shifts to the interstitial compartment, coupled with exudative and evaporative water loss from the burn injury, can reduce circulating blood volume rapidly. Full-thickness circumferential extremity or neck burns require escharotomy if circulation distal to the injury is impaired. Escharotomies are rarely needed within the first 6 hours of injury.
 4. **Remove all clothing** to halt continued burn from melted synthetic compounds or chemicals and to assess the full extent of body-surface involvement in the initial examination. Irrigate injuries with water or saline to remove harmful residues. **Remove jewelry** (particularly rings) to prevent injury resulting from increasing tissue edema.
- G. **Depth of burn** ([Table 30-1](#))

Table 30-1. Treatment algorithm for the three clinically important burn depths^a

1. **First-degree burns** are limited to the epidermis. The skin is painful and red. There are no blisters. These burns should heal spontaneously in 3–4 days.
2. **Second-degree burns**, which are subdivided into **superficial or deep partial-thickness**, are limited to the dermal layers of the skin. The **superficial** partial-thickness burns involve the papillary dermis. They appear red, warm, edematous, and blistered, often with denuded, moist, mottled red or pink epithelium. The injured tissue is very painful, especially when exposed to air. Such burns frequently arise from brief contact with hot surfaces, liquids, flames, or chemicals. **Deep** second-degree burns involve the reticular dermis and thus can damage some dermal appendages (e.g., nerves, sweat glands, or hair follicles). Hence, such burns can be less sensitive, or hairs may be easily plucked out of areas with deep partial-thickness burns. Still, the only definitive method to differentiate superficial and deep partial-thickness burns is by length of time to heal. Superficial burns heal in less than 2 weeks; deep ones require at least 3 weeks. Further, any partial-thickness burn can convert to full-thickness injury over time, especially if early fluid resuscitation is inadequate.
3. **Full-thickness** (third- or fourth-degree) burns involve all layers of the skin and some subcutaneous tissue. In **third-degree** burns, all the skin appendages, including hair follicles, sweat and sebaceous glands, and sensory fibers for touch, pain, temperature, and pressure are destroyed. This results in an initially painless, insensate dry surface that may appear either white and leathery or charred and cracked, with exposure of underlying fat. **Fourth-degree** burns also involve fascia, muscle, and bone. They often result from prolonged contact with thermal sources or high electrical current. All full-thickness burns are managed surgically, and immediate burn expertise should be sought.

H. Percentage of BSA estimation

1. **Small areas: palm of patient's hand = 1% of BSA.**
2. **Large areas: rule of nines.** Regions of the body approximating 9% BSA or multiples thereof are shown in [Table 30-2](#). Note that infants and babies have a proportionally greater percentage of BSA in the head and neck region and less in the lower extremities compared to adults.

Table 30-2. Rule of nines estimation of percent of body surface area

II. Management

A. Emergency room

1. **Resuscitation.** A surgical consultation is initiated for all patients with major injury (*N Engl J Med* 335:1581, 1996).
 - a. **Oxygen** should be provided to patients with all but the most minor injuries. A 100% oxygen high-humidity face mask for those with possible inhalation injury assists the patient's expectoration from dry airways and treats carbon monoxide poisoning. Others can benefit from 2–6 L oxygen via nasal cannula.
 - b. **Intravenous access.** All patients with more than 20% BSA burns require intravenous fluids. A 16-gauge or larger peripheral venous access should be started immediately to provide circulatory volume support. Peripheral access in the upper extremities is preferred over central venous access because of the risk of catheter-related infection. An intravenous catheter may be placed through the burn if other suitable sites are unavailable. Avoid lower-extremity catheters, if possible, to prevent phlebotic complications.
 - c. **Fluid** is administered intravenously to all patients having a 20% or greater BSA burn. Increased capillary permeability in injured tissue results in edema and evaporative losses. Evaporative cooling results in heat loss, and hypothermia may result. Acute metabolic acidosis usually is secondary to inadequate fluid resuscitation. Persistent metabolic acidosis also can result from anaerobic metabolism, secondary to carbon monoxide binding to cellular cytochrome.
 1. **Modified Parkland formula.** The estimated crystalloid requirement for the first 24 hours after injury is calculated based on patient weight and percentage BSA burn. **Lactated Ringer's solution volume in the first 24 hours = 4 mL × % BSA (second-, third-, or fourth-degree burns only) × body weight (kg).** One-half of the calculated volume is given in the first 8 hours after injury, and the remaining volume is infused over the next 16 hours. Fluid resuscitation calculations are based on the time of injury, not the time when the patient is evaluated. Prehospital intravenous hydration is subtracted from the total volume estimate. It should be emphasized that formulas are only estimates, and more or less fluid may be required to maintain adequate tissue perfusion as measured by rate of urine output. Patients with inhalational injury, associated mechanical trauma, electrical injury, escharotomies, or delayed resuscitation require more fluid than that based on the formula alone. Further, for children weighing less than 30 kg, 5% dextrose (D5) in ¼ normal saline maintenance fluids should supplement the Parkland resuscitation to compensate for ongoing evaporative losses. Patient **body weight** is determined early after the burn as a baseline measurement for fluid calculations and as a daily reference for fluid management.
 2. **Colloid-containing** solutions are best held for intravenous therapy until after the first 24 hours postburn. The role of albumin therapy in resuscitation has been reviewed recently, indicating that it should be used with great caution (*BMJ* 317:235, 1998; *Evidence Based Medicine* 4:19, 1999; *ACP J Club* 130:6, 1999). If given to patients with inhalation injury early in resuscitation, albumin may move into the interstitium and may increase pulmonary complications. By 24 hours, capillary leak diminishes. For patients with greater than 30% BSA burn, infuse a one-time bolus of 5% albumin solution (0.3–0.5 mL/kg/% BSA) to restore plasma oncotic pressure. Otherwise, replace insensible losses with D5W in adults or D5 in ¼ normal saline in children weighing less than 30 kg. Such fluids replace evaporative losses and mitigate the hypernatremia after resuscitation (*Shock* 5:4, 1996).
 - d. **A Foley catheter** is used to monitor hourly urine production as an index of adequate tissue perfusion. In the absence of underlying renal disease, a minimum urine production rate of 1 mL/kg per hour in children (weighing ≥30 kg) and 0.5 mL/kg per hour in adults is the guideline for adequate intravenous infusion. To minimize edema, consider reducing intravenous hydration if urine output exceeds 1.5 mL/kg per hour in adult patients.
 - e. **Nasogastric tube** insertion with low intermittent suction is performed if patients are intubated or develop nausea, vomiting, and abdominal distention consistent with adynamic ileus. Virtually all patients with greater than 25% BSA burns have an adynamic ileus.
 - f. **Escharotomy** may be necessary in full-thickness circumferential burns of the neck, torso, or extremities when increasing tissue edema impairs peripheral circulation (digital Doppler signals) or when chest involvement restricts respiratory efforts. Full-thickness incisions through (but no deeper than) the insensate burn eschar provide immediate relief. Longitudinal escharotomies are performed on the lateral or medial aspect of the extremities and the anterior axillary lines of the chest where indicated ([Fig. 30-1](#)). Usually, they are done at the bedside and require no anesthesia. However, if the digits were burned so severely that desiccation results, midlateral escharotomies have minimal benefit. Escharotomies are rarely required within the first 6 hours after injury.

Fig. 30-1. Placement of escharotomies. Midaxial escharotomies should be performed if vascular compromise occurs. Incisions should be performed through the dermis and subcutaneous tissue to allow maximal expansion of the underlying fascia. (From Eichelberger M. *Pediatric Trauma*. St. Louis: Mosby, 1993, with permission.)

2. **Monitors.** Continuous-pulse oximetry to measure oxygen saturation is useful. A caveat is that falsely elevated levels can be observed in carbon monoxide poisoning.
3. **Laboratory examination** includes a baseline complete blood cell count, type and cross-match, electrolytes and renal indices, beta-human chorionic gonadotropin (in women), arterial carboxyhemoglobin, arterial blood gas, and urinalysis. A toxicology screen and an alcohol level are obtained when suggested by history or mental status examination. A chest X-ray is obtained with the understanding that it rarely reflects early inhalation injury. Additional chest films are obtained should endotracheal intubation or central line placement become necessary. An electrocardiogram is useful initially, particularly in elderly patients or those with electrical burns. Fluid and electrolyte fluxes during resuscitation and later mobilization of third-space edema can result in arrhythmias and interval electrocardiographic changes.
4. **Moist dressings** applied to partial-thickness burns provide pain relief from air exposure. Cool water applied to small partial-thickness burns can provide relief but must be avoided in patients with major burns (>25% BSA) and especially in infants—groups that are at high risk for hypothermia. Cold water also can cause vasoconstriction and can extend the depth and surface area of injury.
5. **Analgesia** is given intravenously every 1–2 hours to manage pain but in small doses to guard against hypotension, oversedation, and respiratory depression.
6. **Photographs or diagrams** of the BSA involvement and thickness of burns are useful in documenting the injury. They also can facilitate communication between the various members of the team caring for the patient and serve medicolegal purposes in the case of assault or child abuse.
7. **Early irrigation and débridement** are performed using normal saline and sterile instruments to remove all loose epidermal skin layers, followed by the application of topical antimicrobial agents and sterile dressings. In general, it is safe to leave blisters intact because they permit healing in a sterile environment and offer some protection to the underlying dermis. Once they are ruptured, or if the bullae are large (>2 cm) and thin-walled, débridement is indicated to prevent infection. If burns resulted from liquid chemical exposure, they are irrigated continuously for 20–30 minutes. Dry chemicals are removed from the skin before irrigation to prevent them from dissolving into solution and causing further injury. Corneal burns of the eye require continuous irrigation for several hours and immediate ophthalmologic consultation.
8. **Topical antimicrobial agents** are the mainstay of burn management. The most common organisms complicating the burn injury are *Staphylococcus aureus*, *Pseudomonas aeruginosa*, *Enterococcus* species, *Enterobacteriaceae*, group A streptococci, and *Candida albicans*. Systemic antibiotics are not administered prophylactically but are reserved for documented infection.
 - a. **Silver sulfadiazine** (i.e., Silvadene and others) is the most commonly used agent because it is not irritating and has the fewest adverse side effects, the worst being a transient leukopenia in the first 1–3 days. It is formulated as a cream, which helps to minimize evaporative water and heat loss and thus diminishes caloric requirements. It is **contraindicated** in patients with glucose 6-phosphatase deficiency.
 - b. **Mafenide acetate** (Sulfamylon) has better gram-negative (particularly against *P. aeruginosa*) and anaerobic coverage as well as deeper eschar penetration. Further, burns over avascular cartilage, such as the ear, are ideal for mafenide acetate therapy. However, it is painful and readily absorbed systemically; it can also lead to metabolic acidosis by inhibiting carbonic anhydrase.
 - c. **Polymyxin B sulfate (Polysporin)** is tolerated well on facial burns and does not discolor skin as silver sulfadiazine sometimes can.
 - d. **Silver nitrate** has lost favor due to the severe electrolyte abnormalities resulting from Na⁺, K⁺, and Cl⁻ leaching from the wound and because it readily stains skin and clothing. However, for patients with a sulfa allergy, it is a reasonable choice, provided that electrolytes are closely monitored.
 - e. **Acticoat** (Westaim Biomedical, Exeter, NH) has recently gained acceptance. This dressing comes as an easy-to-apply sheet, slowly releases silver ions, has good antimicrobial activity, and can be left in place for 3 days. However, its expense may prove prohibitive for some types of injury.
9. **Tetanus prophylaxis** should be administered as tetanus toxoid, 0.5 mL i.m., if the last booster dose was more than 5 years before the injury. If immunization status is unknown, also administer human tetanus immunoglobulin (Hyper-Tet), 250–500 units i.m., using a syringe and injection site different from those used for tetanus toxoid administration.
10. **Stress ulcer prophylaxis** (e.g., H₂ blockers, antacids, or omeprazole) should be provided for patients who have major burns and can receive nothing by mouth, especially those with coagulopathy.

- B. Outpatient.** Only minor first-degree or partial-thickness injuries should be considered for outpatient management. This decision depends on many factors, including patient reliability, opportunity for follow-up, and accessibility to health professionals. Surgical consultation is recommended at the time of initial evaluation in all but the most minor injuries.
- Dressings** are often managed by the patient when the injury is easily accessible. Home health nursing is a useful adjunct when self-application is suboptimal or wounds are in early healing stages, requiring close follow-up. Silver sulfadiazine is often applied as a light coating, followed by sterile dressings once or twice daily.
 - Antibiotics** are not prescribed prophylactically because they allow resistant organisms to multiply. Their use is limited to documented infection of the wounds.
 - Follow-up** usually is once or twice a week during the initial healing of partial-thickness burns and split-thickness skin grafts until epithelialization is complete. Thereafter, patients are followed at 1- to 3-month intervals to evaluate and treat scar hypertrophy (application of foam tape or Jobst garments), hyperpigmentation (avoiding direct sunlight, using sunscreen), dry skin (unscented lotion massage), pruritus (antihistamines), and rehabilitation potential and therapy (physical, occupational, social, and psychological).

C. Inpatient

- Transfer to a burn center** should follow the guidelines of the American Burn Association. These criteria reflect multiple studies showing that age and % BSA burns remain the two most important prognostic factors.
 - Patients younger than 10 years old or older than 50 years** sustaining partial- or full-thickness burns to more than 10% BSA
 - Partial- or full-thickness burns to more than 20% BSA** in other age groups
 - Specialized regions** involving joints, hands, feet, perineum, genitalia, face, eyes, or ears
 - Full-thickness burns to more than 5% BSA**
 - Significant inhalation, chemical, or electrical injury**
 - Burns in combination with significant associated mechanical trauma or preexisting medical problems**
 - Patients requiring specialized rehabilitation, psychological support, or social services (including suspected neglect or child abuse)**
- Nutrition.** The daily estimated metabolic requirement (EMR) can be calculated from the Curreri formula: $EMR = [25 \text{ kcal} \times \text{body weight (kg)}] + (40 \text{ kcal} \times \% \text{ BSA})$. Protein losses from metabolism and burn wound extravasation are replaced by supplying 1.5–2.0 g/kg per day.
 - Enteral** feedings are the preferred route when tolerated and can be administered through an enteral feeding tube positioned in the duodenum. For severe burns, early feeding within the first 24 hours has been shown to improve a number of outcome measures, including length of hospital stay [*Burns* 23(Suppl 1):519, 1997].
 - Total parenteral nutrition** should be initiated after fluid resuscitation only if the patient is unable to tolerate enteral feeding.
 - Daily vitamin** supplementation in adults should include 1.5 g ascorbic acid, 500 mg nicotinamide, 50 mg riboflavin, 50 mg thiamine, and 220 mg zinc. Patients with large burns may remain hypermetabolic for weeks to months after the burn wound is closed; early tapering of nutritional intake in these patients should be avoided.
- Wound care**
 - Analgesia and sedation** for dressing changes are necessary for major burns. Valium (0.1 mg/kg i.m.) plus ketamine (0.5 mg/kg i.m.) is one sedative regimen that has been used. Alternatively, in patients with a secure airway (typically intubated), intravenous propofol has the desired effects of ease of titration and quick onset and offset of action. Either of these sedative regimens in concert with narcotic analgesia is well tolerated.
 - Daily dressing changes.** While the wounds are exposed, the surgeon can properly assess the continued demarcation and healing of the injury. Physical therapy with **active range of motion** is performed at this time before reapplying splints and dressings.
 - Débridement** of all nonviable tissue should take place using sterile technique and instruments when demarcation occurs. Soft eschar can be abraded lightly, using wet gauze. Enzymatic treatments (i.e., Travase, sutilains ointment) can be useful in dissolving eschar to develop granulation tissue for tissue grafting.
 - Temporary dressings for massive burns with limited donor sites.**
 - Biologic.** Fresh or cryopreserved cadaver allografts have been the gold standard. Recently, however, our center has had success using porcine xenografts. This alternative provides the advantages of ease of acquisition and application, while providing barrier protection and a biologic bed under which dermis can granulate.

After several days, allograft can be removed and meshed autograft may be replaced for definitive coverage. The use of cultured autologous epithelium, cultured allogeneic epidermis, and allograft dermis has had encouraging results in some burn centers but remains largely investigational. (*Surg Clin North Am* 73:363, 1993).

- Synthetic** membranes (e.g., Integra artificial skin, Dermagraft-TC) are an attractive alternative. Integra consists of an epidermal analogue, Silastic film, and a dermal analogue, a collagen matrix with chondroitin 6-sulfate. The patient's dermal fibroblasts can grow into this matrix. Once adequate vascularization is seen through the Silastic film, the film is removed, and an ultrathin autograft is placed onto the artificial dermis. The autograft is thin so that donor sites can be reharvested more quickly. This technique has been reported to give results similar to those of treatment with allografts (*J Trauma* 50:358, 2001). Dermagraft-TC has had similar success. This bilaminar skin substitute consists of a dermal matrix impregnated with human neonatal fibroblasts and a silicone epidermal analogue. After a few days, the bilaminar artificial skin comes off easily and can be replaced with autograft (*J Burn Care Rehabil* 18:52, 1997).

D. Operative

- Early tangential excision** of burn eschar to the level of bleeding capillaries should follow the resuscitation phase. Debate persists as to the optimal timing of burn wound excision (range is 1–10 days). Excision can be performed using a Goulian or Humbly knife for small surfaces and a power- or gas-driven dermatome for larger surfaces. For each trip to the operating theater, consider limiting burn excision to less than 20% BSA or 2 hours operating time. Even within such limits, aggressive débridement frequently produces profound blood loss and hypothermia.
- Split-thickness skin grafts** are harvested at a thickness of 12–15 thousandths of an inch with a meshed expansion ratio from 1.5:1 to 3.0:1. The graft is immobilized with absorbable sutures or staples. For very large wounds, 4:1 autograft can be overlaid with meshed allograft skin ([Fig. 30-2](#)). However, cosmesis is poor, and graft take may be less; thus, this technique is used on the back, flanks, or other less visible areas. Nonadherent dressings and bolsters are applied to minimize shear forces on the fresh grafts. **Splints or pins** may be required to improve graft survival at joints and to prevent contracture. **Ideal point positions** are extension in the neck, knee, elbow, wrist, and interphalangeal joints; **15-degree flexion** at metacarpophalangeal joints; and **abduction** at the shoulder.

Fig. 30-2. Combined skin graft to cover burn wounds too extensive for other methods. The widely meshed autograft would allow continued fluid fluxes during the more extended time required for epithelialization. A more narrowly meshed allograft placed superficial to the autograft can accelerate the process by providing temporary coverage while the autograft fills in. (From Eichelberger M. *Pediatric Trauma*. St. Louis: Mosby, 1993, with permission.)

- For large burns that cannot be completely covered with available autograft**, allograft or xenograft can be used to cover the remaining wound temporarily. In 3–5 days, the temporary grafts can be removed, and autograft should take well onto the granulating dermis.

E. Follow-up

- Wound healing**
 - Infection** is minimized by using topical antimicrobial agents. When identity is established, antibiotic therapy is targeted to the specific organism. It may be useful on occasion to diagnose invasive infection. The technique requires a 500-mg biopsy of suspicious eschar and underlying unburned tissue. The presence of microorganisms in viable tissue confirms the diagnosis. The number of microorganisms in viable tissue correlates with mortality. Treatment requires infected eschar excision and appropriate topical/systemic antibiotic therapy (*World J Surg* 22:135, 1998).
 - Granulation** tissue that fails to epithelialize at skin graft sites can be cauterized using an applicator stick tipped with silver nitrate.
 - Hyperpigmentation** is best prevented by avoiding direct sunlight exposure for up to 1 year. When exposure to sunlight is unavoidable, a topical sunblock agent should be used.
 - Scar hypertrophy** is minimized by local tissue compression, tailored Jobst garments, foam sponge, foam tape, or silicone gel sheets.
 - Contractures** are best prevented by using active range of motion. When present, release (Z-plasty) or excision and skin grafting may be necessary.
 - Pruritus** can be palliated with antihistamines.
- Rehabilitation** is provided with ongoing evaluation by occupational and physical therapists.

III. Complications include burn wound infection, pneumonia sepsis, ileus, Curling's ulcer (gastroduodenal), acalculous cholecystitis, and superior mesenteric artery syndrome.

IV. Burn mechanisms: special considerations

A. Inhalational. Thermal injury to the airway generally is limited to the oropharynx or glottis. The glottis generally protects the subglottic airway from heat, unless the patient has been exposed to superheated steam. Edema formation can compromise the patency of the upper airway, mandating early assessment and constant reevaluation of the airway (*J Trauma* 36:59,1994). **Gases** containing substances that have undergone incomplete combustion (particularly aldehydes), toxic fumes (hydrogen cyanide), and carbon monoxide can cause tracheobronchitis, pneumonitis, and edema. Further, the mortality may be increased by as much as 20%. **Carbon monoxide** exposure is suggested by a history of exposure in a confined space with symptoms of nausea, vomiting, headache, mental status changes, and cherry-red lips. Carbon monoxide binds to hemoglobin with an affinity 249 times greater than that of oxygen, resulting in extremely slow dissociation (250-minute half-life with room air) unless the patient is administered supplemental oxygen (40-minute half-life with 100% oxygen via nonrebreathing mask). The arterial **carboxyhemoglobin** level is obtained as a baseline and, if it is elevated (>5% in nonsmokers or >10% in smokers), oxygen therapy should continue until normal levels are achieved. Inhaled chemical products of combustion may include acids, phosgene, and cyanide derived burning polyvinylchloride and polyurethane. The increased ventilation-perfusion gradient and reduction in peak airway flow in distal airways and alveoli can be evaluated using a xenon-133 ventilation-perfusion lung scan. **Management** of minor inhalation injury is by delivery of humidified oxygen. Major injuries require endotracheal intubation with a large-bore tube (7.5–8.0 mm) to facilitate pulmonary toilet of viscous secretions and mechanical ventilation with positive pressure. Hypoxic intubated patients may benefit from oscillation-type mechanical ventilation (*Crit Care Med* 25:937, 1997).

B. Electrical

- 1. Factors influencing severity** include the voltage (high >1,000 V), current, duration of contact, resistance, and the grounding efficiency. Electrical current passes in a straight line between points of body contact with source and the ground. When current passes through the heart or brain, cardiopulmonary arrest can result. In most cases, these injuries respond to resuscitation and usually do not cause permanent damage. **Severity of injury frequently is underestimated** when only the entrance and exit wounds are considered.
 - a. Tissue resistance.** Heat and subsequent injury from thermal necrosis is directly proportional to resistance to current flow. Tissues are listed from lowest to highest resistance: nerve, blood vessels, muscle, skin, tendon, fat, and bone. In addition to direct tissue injury, thrombosis can occur with distal soft-tissue ischemia. Peripheral perfusion should be monitored closely because fasciotomy may become necessary to treat compartment syndrome. Fluid resuscitation requirements often are higher than calculated by published formulas.
 - b. Current**
 - 1. Alternating** current (household, power lines) produces severe muscle contraction with each cycle and can result in fractures in addition to the thermal injury at skin entrance and exit points. High-voltage injury, which is commonly seen in workers operating near power lines, can present with full-thickness, charred skin at the entrance and exit wounds, with full arrest, and with fractures sustained while current passed through the body or during a fall.
 - 2. Direct** current emanates from batteries and lightning. With a voltage of at least 100 million volts and a current of 200,000 A, lightning kills 150–300 people in the United States every year. Injury can result from direct strikes or side flashes. Current can travel on the surface of the body rather than through it, producing a “splashed-on” pattern of skin burn.
 - 2. Complications** include **cardiopulmonary arrest** (more common with alternating current), **thrombosis**, **associated fractures** related to fall or severe muscle contraction, **spinal cord injury**, and **cataracts**. **Rhabdomyolysis** may occur and result in myoglobin release from injured cells. Precipitation of protein in the renal tubules can cause acute renal failure. Dark urine is the first clinical indication of myoglobinuria, and intravenous lactated Ringer's solution should be administered to maintain a urine output of greater than 100 mL per hour. Alternatively, 3 ampules of sodium bicarbonate (~135 mEq sodium) in 1 L D5W provides an isotonic solution to alkalinize the urine more effectively and minimize nephrotoxicity from the myoglobinuria.
- C. Chemical injury** may result from contact with alkali, acid, or petroleum compounds. Removal of the offending agent is the cornerstone of treatment. Dry chemicals should be brushed off or aspirated into a closed suction container before irrigating with copious amounts of water for at least 20–30 minutes. Alkali burns, which penetrate more deeply than acid burns, require longer periods of irrigation. Neutralizing the chemicals is no longer recommended because the resulting reaction generates more heat to exacerbate the injury. Eye injury from alkali burns mandates 8 hours of continuous irrigation after injury. **Tar** can cause ongoing burn if not removed. Cool the tar with cold water. Then, use an adhesive remover to remove any remaining tar.

D. Cold injury

- 1. Classification of hypothermia** is based on core temperature (usually approximated with a rectal or esophageal thermometer): mild, 32–35 °C; moderate, 30–32 °C; and severe, less than 30 °C. The elderly and children are particularly susceptible, and contributing factors include drug or alcohol ingestion, hypothyroidism, immobilization, moisture, sepsis, diabetes mellitus, and cerebral ischemia. Signs include reduced levels of consciousness, and patients may appear cold, gray, cyanotic, or asystolic. Rewarming is accomplished by a warm-water bath, endotracheal intubation, ventilation with warm gases, and/or central venous infusion of warm lactated Ringer's solution. More invasive methods include peritoneal lavage, thoracic-pleural lavage, hemodialysis, and cardiopulmonary bypass. Serial arterial blood gases are obtained during rewarming to monitor the development of systemic acidosis as tissue beds reperfuse. Care is taken to avoid overstimulation, which can lead to ventricular dysrhythmias. The heart becomes increasingly irritable at core temperatures below 34 °C. Asystole may occur below 28 °C, and cardiopulmonary resuscitation should be started and maintained until the patient is at 36 °C.
- 2. Frostbite** results from the formation of intracellular ice crystals and microvascular occlusion. Factors affecting severity are temperature, duration of exposure, and environmental conditions promoting rapid heat loss, such as wind velocity, moisture, immobilization, and open wounds. The fingers, toes, and ears are most commonly injured, particularly when reduced tissue perfusion has resulted from other causes, such as shock.
 - a. Classification**
 - 1. First-degree:** hyperemia and edema, without skin necrosis
 - 2. Second-degree:** superficial vesicle formation containing clear or milky fluid surrounded by hyperemia, edema, and partial-thickness necrosis
 - 3. Third-degree:** hemorrhagic bullae and full-thickness necrosis
 - 4. Fourth-degree:** gangrene with full-thickness involvement of skin, muscle, and bone
 - b. Treatment** is rapid rewarming using 40 °C water until the tissue perfusion returns. Because mechanical pressure or friction can injure the tissue further, massage and weightbearing are discouraged. Rewarming can be painful, and therefore intravenous analgesia should be provided. Dry dressings are applied, and the injured area is kept warm and elevated to reduce edema formation. Tetanus prophylaxis is administered, and follow-up over several weeks is recommended to allow for demarcation of full-thickness injury. Escharotomy may be required for severe injury. Early amputation is not recommended because improvement in tissue viability can occur weeks after injury.

V. Future burn care management. Burn mortality has dramatically improved. At the U.S. Army Institute of Surgical Research, the LA50, the size of a burn that kills half of patients, is 75.6% in adults and 90% in children younger than 15 years of age (*J Burn Care Rehabil* 18:S2, 1997). With aggressive early tangential excision, early coverage, alternative dressings, and better understanding of aggressive wound infection, clinicians have an augmented arsenal to combat morbidity and mortality. Ongoing immunologic and clinical studies on burn-induced immunosuppression, cytokines, and artificial bilaminar skin may provide the next advances in burn care. The biggest challenge, however, may still be prevention education.

31 Soft-Tissue and Skin Tumors

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Diagnosis of Skin Lesions and Soft-Tissue Masses

Most patients present with a visible or palpable lesion or mass. Physical examination is a key to diagnosis. Biopsy is often performed to confirm or establish the diagnosis. For large or deep soft-tissue tumors, radiologic evaluation often precedes biopsy.

I. Skin lesions

- A. **History.** Pigmented lesions with a change in size, borders, and coloration are important. The presence of itching, bleeding, or ulceration should be noted.
- B. **Physical examination.** Color, size, shape, borders, elevation, location, firmness, and surface characteristics should be recorded for each skin lesion. In general, uniformly colored, small, round, circumscribed lesions are benign. Irregularly colored, larger, asymmetric lesions with indistinct borders and ulceration are more likely to be malignant.
- C. **Biopsy.** Lesions that change over a period of observation should have a tissue diagnosis. Any lesion that needs a full thickness of tissue, particularly suspected melanomas, should receive a punch biopsy or excisional biopsy. In general, shave biopsies should be avoided. A punch biopsy involves using a cylindrical blade to remove a small core of skin. Excisional biopsy is the same as for soft-tissue masses, discussed in [section II.D.](#)

II. Soft-tissue masses

- A. **History.** An enlarging, painless mass is the most common presentation. There is a frequent perceived association with antecedent trauma. Pain is a late symptom. Any symptom or enlargement is important. Lesions often are misdiagnosed as hematomas or pulled muscles.
- B. **Physical examination.** The key factors to note on physical examination are size, extent of anatomic relationships with surrounding structures, borders, and mobility. A neurovascular examination of the affected area should be performed.
- C. **Radiologic evaluation**
 1. **MR scan** is the best choice for imaging soft-tissue masses. However, it can be difficult to distinguish tissue edema surrounding the tumor from tumor itself. The combination of T2-weighted image and gadolinium enhancement is very good at distinguishing tumor from surrounding normal tissue.
 2. **Computed tomography (CT)** is used for larger, deeper tumors to assess the character and extent of the tumor and involvement of other structures, as well as to help determine surgical access to the tumor. CT-guided core-needle biopsy can be attempted for deep tumors with difficult surgical access. A CT of the chest should be performed on all patients with soft-tissue sarcomas (STS), and CT may be helpful in evaluating the pelvis and retroperitoneum.
- D. **Biopsy.** Ideally, the oncologic surgeon who will perform the definitive resection should perform the biopsy.
 1. **Incisional biopsy** is the gold standard. It is usually performed in the operating room under appropriate anesthesia. A small incision should be made so that it can be excised for a possible subsequent operation. The incision should be oriented along the long axis of the lesion. Incisional biopsy rather than excisional biopsy should be performed for a tumor greater than 3 cm (or >5 cm if it is consistent with a lipoma) because if the tumor is a sarcoma, the subsequent total excision will be complicated by the potential contamination of a broad area by the initial excision. Drains should be avoided; meticulous hemostasis to prevent hemorrhage from spreading tumor is critical. If drains are needed, drain sites should be in line with the incision, to be excised in any subsequent operation.
 2. **Core-needle biopsy** provides a section of intact tissue for histologic analysis and can be performed in the clinic; it has the potential to provide the same information as that rendered by incisional biopsy if a good core of tissue is obtained. A very small incision is made to allow easy entrance of the needle through the skin. Most indeterminate or negative results should be confirmed with either incisional or excisional biopsy.
 3. **Excisional biopsy** is performed for tumors that are probably benign or less than 3 cm in diameter. The usual procedure is an elliptical incision around the tumor along the skin lines of minimal tension, with anticipation of primary closure. The tumor should be excised completely with a thin envelope of normal tissue.
 4. **Fine-needle aspiration** is the least invasive but also the least informative form of tissue diagnosis. As the needle enters the mass, multiple passes are made through the mass in various directions; the plunger is released before removing the needle from the mass. The specimen is then fixed and sent for cytopathologic evaluation. Fine-needle aspiration usually cannot give the grade but often can determine the presence of malignancy and the histologic type of tumor. Indeterminate results should be followed by further evaluation.

Benign Lesions

I. Seborrheic keratoses are benign skin growths that originate in the epidermis. They appear in older people as multiple, raised, irregularly rounded lesions with a verrucous, friable, waxy surface and variable pigmentation from yellowish to brownish black. They are often found on the face, neck, and trunk. Treatment may consist of curettage followed by electrodesiccation, as well as topical agents, such as trichloroacetic acid, or cryotherapy with liquid nitrogen.

II. Actinic keratoses are caused by sun exposure and are found predominantly in elderly, fair-skinned patients. They are small, usually multiple, flat to slightly elevated lesions with a rough or scaly surface ranging from red to yellowish brown to black and are found in areas of chronic sun exposure. Unlike seborrheic keratoses, these lesions have malignant potential: 15–20% of lesions become squamous cell carcinoma. Metastases are rare under these circumstances. Standard treatment consists of topical application of 5-fluorouracil twice a day for 2–6 weeks.

III. Nevi. Junctional nevus cells actually are located in the epidermis and at the dermal-epidermal junction. These nevi are small (<6 mm), well-circumscribed, light

brown or black macules found on any area of the body. They rarely develop in people older than 40 years, and any new lesion in someone older than 40 should be considered a possible early melanoma.

IV. Epidermal inclusion cysts are lined by epidermal cells containing lipid and keratinous material. Asymptomatic cysts may be removed for diagnosis, prevention of infection, or cosmesis. Excision of the cyst should include the entire cyst lining, preferably without interruption of the lining, to prevent recurrence and should include any skin tract or drainage site.

V. Neurofibromas are benign tumors that arise from Schwann cells and are seen most frequently in the setting of neurofibromatosis (von Recklinghausen's disease). Neurofibromas are soft, pendulous, sometimes lobulated subcutaneous masses that vary widely in size. The overwhelming majority of these tumors do not require excision unless increase in size is observed or for cosmetic reasons.

VI. Ganglion cysts are subcutaneous cysts attached to the joint capsule or tendon sheath of the hands and wrists, most commonly seen in young and middle-aged women. They present as firm, round masses often seen on the dorsum of the wrist but also can be found on the radial volar wrist, along the flexor tendon sheaths of the hand, or in the dorsum of the distal interphalangeal joint. Surgical excision has an extremely low recurrence rate; in addition to the cyst, the capsular attachment and a small portion of the joint capsule should be removed to prevent recurrence.

VII. Lipomas are benign tumors consisting of lobules of fat and are perhaps the most common human neoplasms. There is very little potential for malignancy; sarcomatous elements occur in fewer than 1% of cases. They are soft, fatty, subcutaneous masses and vary widely in size. Asymptomatic small tumors can be followed clinically, but rapidly growing tumors should be removed. Large tumors (>5 cm) should be evaluated by incisional biopsy to be certain they are not malignant. Lipomas are enucleated easily from the surrounding normal fat.

VIII. Dermatofibrosarcoma protuberans is a locally aggressive tumor that does not metastasize. Margins of 2–5 cm should be achieved if possible. Mohs' micrographic surgery involves serial excisions of the tumor, with microscopic examination for areas of positive margins that have been mapped and are then reexcised, one section at a time, until a negative margin is reached. Although time consuming and expensive, this surgery has been advocated in the management of dermatofibrosarcoma protuberans for improved tissue conservation, cosmetic advantages, and low recurrence rates.

IX. Desmoid tumors are nonmetastasizing but locally aggressive tumors that arise from connective tissue. Wide excision with a margin of normal tissue should be performed if possible, but limb function should be spared. Local recurrences are common, and reexcision is often required. Tamoxifen, nonsteroidal antiinflammatory drugs (e.g., sulindac), or a combination have been used with only anecdotal success and may be attempted as a conservative alternative to surgery. These drug regimens have been advocated in recurrent or unresectable cases as well. Recommendations as to future pregnancies are conflicting and unclear. Patients with a desmoid tumor should undergo colonoscopy to exclude the diagnosis of familial adenomatous polyposis.

Melanoma

The incidence of malignant melanoma is increasing at a rate faster than any other cancer. The annual incidence in the United States is approximately 40,300, with approximately 7,700 deaths expected in the year 2000.

I. Lesions. Most pigmented lesions are benign, but approximately one-third of all melanomas arise from pigmented nevi. It is essential to differentiate between benign, premalignant, and malignant lesions.

A. Premalignant lesions

1. **Dysplastic nevi** have variegated color (tan to brown on a pink base), are large (5–12 mm), appear indistinct with irregular edges, and have macular and papular components. There is a familial association between dysplastic nevi and a high incidence of melanoma. Melanomas may develop *de novo* or from preexisting dysplastic nevi.
2. **Congenital nevi** are notable for their presence since birth and are commonly referred to as "birthmarks." They can be premalignant; there is an increased risk of melanoma developing from these lesions, particularly if the nevus is larger than 20 cm.

B. Malignant lesions

1. **Superficial spreading melanoma** is the most common form of melanoma (80%), with approximately one-half arising from a preexisting mole. The lesions usually are slow growing and brown, with small discrete nodules of various differing colors. They tend to spread laterally but usually are slightly elevated. They are found most commonly on the back in men and in women and on the lower extremities of women.
2. **Nodular melanoma** is the most aggressive form, rapidly becoming a palpable, elevated, firm nodule that may be dense black or reddish blue-black. A distinct convex nodular development indicates deep dermal invasion. They arise from the epidermal-dermal junction and invade deeply into the dermis and subcutaneous tissue. Approximately 5% are amelanotic (flesh colored).
3. **Lentigo maligna melanoma** usually is found on older patients as a large melanotic freckle on the temple or malar region known as **Hutchinson's freckle**. It usually is slow growing but becomes large, often reaching 5–6 cm in diameter. Initially, it is flat, but it becomes raised and thicker, with discrete brown to black nodules and irregular edges.
4. **Acral lentiginous melanoma** occurs on the palms, soles, and nail beds; occurs primarily in darker-skinned people; and metastasizes more frequently than do other melanomas.
5. **In-transit metastases** and **satellites** both signify a poor prognosis with a high risk of local recurrence and distant metastasis. In-transit metastases are lesions in the skin more than 2 cm from the primary lesion; they arise from tumor cells in intradermal lymphatics. Satellites are metastatic lesions in the skin within 2 cm of the primary tumor.

II. History and risk factors. A history of melanoma should include an assessment of risk factors and family history for melanoma.

A. **Risk factors.** Each of the risk factors listed below are considered to carry a more than threefold increase in risk for melanoma; the presence of three or more risk factors carries approximately 20 times the risk.

1. **Family or personal history of melanoma**
2. **Blond or red hair**
3. **Freckling of the upper back**
4. **Three or more blistering sunburns before age 20 years**
5. **Presence of actinic keratosis**
6. **Blue, green, or gray eyes**

B. **Clinical features.** Early melanoma and dysplastic lesions can be recognized by the features highlighted in the mnemonic **ABCD: asymmetry, border** irregularity, **color** variegation, and **diameter** greater than 6 mm. Advanced lesions are more readily apparent and may be nodular or ulcerated.

III. Staging and prognosis. Tumor thickness is the most important factor in staging the tumor. Tumors less than 1 mm thick have a cure rate greater than 95%, whereas 10-year survival for lesions more than 4 mm thick is 46%. Thickness also has been correlated with the risk of regional node and distant metastasis. The Breslow scale for thickness is helpful in classifying melanoma depth ([Table 31-1](#)). The anatomic level of invasion, known as *Clark's level* ([Table 31-2](#)), is also still occasionally used, but the American Joint Committee on Cancer (AJCC) system of TNM (tumor, node, metastasis) classification ([Table 31-3](#) and [Table 31-4](#)) is preferred over both these systems. Older age, male gender, satellitosis, ulceration, and location on the back, posterolateral arm, neck, or scalp (the BANS region) all carry a worse prognosis. The presence of regional node metastasis severely worsens prognosis (10-year survival, 25–30%). Distant metastases have a dismal prognosis (median survival, 2–11 months).

Table 31-1. Breslow's classification (thickness) of melanoma

Table 31-2. Clark's classification (level of invasion) of melanoma

Table 31-3. TNM (tumor, node, metastasis) definitions of melanoma

Table 31-4. American Joint Committee on Cancer groupings of melanoma

IV. Treatment

A. Surgery

1. **Wide local excision** of the lesion is the primary definitive treatment for most melanomas and premalignant lesions. The size of the surgical margins depends on the tumor thickness: Thin melanomas (<1 mm) should have a margin of 1 cm; lesions between 1 and 2 mm and all scalp lesions should have a margin of 2 cm. Lesions greater than 2 mm should have margins of 2–3 cm (*Ann Surg Oncol* 7:87, 2000). Melanoma of fingers and toes requires digital amputation. In general, the wounds should be closed primarily, with flaps or skin grafts reserved for large defects if needed. Mohs' micrographic surgery has been advocated by some groups for areas where wide and deep excisions are difficult. Further trials using this technique for melanoma are ongoing.
2. **Elective lymph node dissection** is still a controversial issue: Several large trials, such as the World Health Organization trial number 1, World Health Organization trial number 14, the Mayo Clinical Surgical Trial, and the Intergroup Melanoma Surgical Trial, have attempted to address this topic. The Intergroup Trial showed that elective lymph node dissection in patients with intermediate-thickness tumors (1–4 mm) improves survival, especially in those under the age of 60. Elective lymph node dissection has not been shown to benefit other subgroups (*Ann Surg* 224:255, 1996).
3. **Sentinel lymph node (SLN) biopsy.** The efficacy of intraoperative lymphatic mapping and selective lymphadenectomy has been established for melanoma. This technique is based on the documented pattern of lymphatic drainage of melanomas to a specific lymph node, termed the *sentinel lymph node*, before further spread. The histology of SLN should reflect that of the rest of the nodal basin. If the SLN is negative for metastases, a more radical and morbid lymph node dissection can be avoided. This procedure requires expertise and a multidisciplinary approach, including the participation of nuclear medicine, pathology, and radiology. The SLN can be accurately identified 96% of the time using dyes and radiolymphoscintigraphy. Currently, it appears that SLN is most beneficial for intermediate-thickness melanomas (1–4 mm) (*Ann Surg* 233:250, 2001).

The presence of disease in the SLN identifies patients who may benefit from a complete lymph node dissection or adjuvant therapy. Recently, diagnosis of submicroscopic disease in the SLN based on genetic changes detectable by the polymerase chain reaction has been used. Initial studies indicate improved prediction of disease recurrence and improved survival over histologic evaluation alone.

4. **Therapeutic lymph node dissection** should be performed for involved axillary and superficial inguinal lymph nodes unless unresectable distant metastases are present. Deep inguinal node dissection should be reserved for patients whose survival is thought to justify the potential morbidity of the procedure.
 5. **Resection of metastases.** Satellites can be treated by including them in the excision of the primary tumor. Distant metastases that are few and accessible may be resected for palliation and (in rare cases) for cure.
- B. **Adjuvant therapy.** Chemotherapeutic approaches to treating melanoma have been disappointing. No single agent has a greater than 25% response rate, and dacarbazine is the only U.S. Food and Drug Administration–approved agent for treatment of melanoma. Interferon-alpha 2b is the only adjuvant therapy that has been shown to improve disease-free survival and overall survival (in patients with stage IIB–III disease). Other biologic response modifiers, such as interferon-gamma and interleukin-2, are being investigated. Extensive research into immune-based therapies is under way. Several melanoma tumor antigens have recently been identified, and vaccines using these antigens with dendritic cells have shown initial success. Larger trials and newer treatment approaches are currently being planned.

Other Malignant Skin Tumors

I. Basal cell carcinoma is the most common malignant neoplasm of the skin; it derives from the basal cells of the epidermis and adnexal structures. They are slow growing and very rarely metastasize (<0.1%) but can be locally aggressive. Sun exposure is the most significant epidemiologic factor; consequently, this neoplasm is found most commonly on the skin of the head and neck (85%) in fair-skinned patients older than 40 years.

A. **Lesions.** It is particularly important to identify the morpheaform carcinoma because it is more aggressive, with a tendency toward deep infiltration and local recurrence. These carcinomas are flat, indurated lesions with a smooth, whitish, waxy surface and indistinct borders. The noduloulcerative form is the most common form and is characterized by shiny, translucent nodules with a central umbilication that often becomes ulcerated, with pearly, rolled, telangiectatic edges.

B. Treatment

1. **Excisional biopsy** is adequate treatment for small tumors, with intraoperative frozen-section analysis (to confirm negative margins) and primary closure. Larger tumors may be diagnosed by incisional or punch biopsy followed by removal, with closure using a flap or skin graft if necessary. A margin of 2–4 mm on all sides of visible tumor should be obtained, and positive margins on frozen-section analysis should be reexcised. Margins of dysplasia or actinic changes need not be reexcised because local recurrence generally does not occur in these cases.
2. **Mohs' micrographic surgery** may be useful for recurrent tumors or in situations in which tissue conservation is important.
3. **Curettage with electrodesiccation** can be performed for small superficial tumors, with little risk of recurrence.
4. **Liquid nitrogen** can be used for tumors of less than 1 cm in diameter.
5. **Radiation therapy** can be used in certain situations for structures difficult to reconstruct, such as the eyelids. It also can be used for palliation in patients who have large tumors and who might refuse an extensive operation, especially the elderly. Although reexcision is indicated for recurrences or positive margins, radiation therapy can be used in individual and unique circumstances.

II. Squamous cell carcinoma is the second most common skin cancer in fair-skinned people and is the most common cancer of darkly pigmented people. As for the other skin malignancies, sunlight is the major etiology, with the greatest risk in elderly men who have a history of chronic sun exposure. The mean age of presentation is 68 years, and men predominate in a 2:1 ratio. Squamous cell carcinoma can be found on any sun-exposed area, including mucous membranes. It also is known to develop from draining sinuses, radiation, chronic ulcers, and scars (particularly burn scars, in which case it is called a **Marjolin's ulcer**).

A. **Lesions.** Squamous cell carcinoma presents as small, firm, erythematous plaques with a smooth or verrucous surface and indistinct margins. As they grow, the lesions become raised, fixed, and ulcerated. Ulceration tends to occur earlier in aggressive lesions. Most lesions are preceded by actinic keratosis that results in a slow-growing, locally invasive lesion without metastases. If not preceded by actinic keratosis, the cancer tends to be more aggressive, with more rapid growth, invasion, and metastatic spread. Perineural invasion has a poorer prognosis and higher recurrence rate.

- B. **Treatment** is similar to that for basal cell carcinoma. Tumor-free margins of 5 mm for tumors of less than 1 cm and 1–2 cm for tumors of more than 2 cm in diameter should be obtained. Curettage with electrodesiccation and laser vaporization have been used for small, superficial squamous carcinomas, but there is no way to assess margins of treatment. Solitary metastases should be resected if possible, because there is a relatively high cure rate compared to other cancers.

Soft-Tissue Sarcomas

STS derive from mesodermal tissues and are rare, constituting approximately 1% of adult malignant neoplasms and causing 3,100 deaths annually. Most of these tumors arise *de novo*, rarely from premalignant tumors. In a minority of cases, STS are associated with cancer predisposition syndromes such as von Recklinghausen's disease, Werner's syndrome, or Li-Fraumeni syndrome. Lymphedema and radiation have been shown to be etiologic factors in certain rare sarcomas.

I. Lesions. Sarcomas are classified by histologic cell type of origin and grade. The most common subtype is malignant fibrous histiocytoma (40%), followed by liposarcoma (25%). Patients typically present with an asymptomatic lump or mass that has grown to be visible or palpable. Retroperitoneal tumor can reach massive proportions before increased abdominal girth and vague symptoms bring it to the physician's attention. Tumors also may grow unnoticed to large sizes in the thigh or trunk.

II. Diagnosis. Biopsy (usually core or incisional) is adequate for diagnosis. Care is needed to orient incisions to aid in the definitive operation. Even small, apparently benign lesions should be biopsied or excised.

III. Staging and prognosis (Table 31-5). The AJCC staging system is based on tumor size, nodal status, histologic grade, and metastasis. Of these, size and grade are the most important.

Table 31-5. Soft-tissue sarcoma staging

- A. **Grade.** The grade of the tumor is the major prognostic factor. Grade is obtained from histopathologic analysis of biopsy tissue and is generally based on the number mitotic index, nuclear morphology, and degree of anaplasia. However, many centers have varying criteria, and discordancy rates of up to 40% have been observed between even expert pathologists.
- B. **Staging** for STS includes physical examination and computed tomographic scan or MR scan to assess the size and extent of tumor. Because metastases most commonly are found in the lungs, computed tomographic scan of the lungs is a required study for grade II and III lesions. Abdominal computed tomographic scan is required for evaluation of retroperitoneal sarcomas, not only to evaluate the tumor but also to assess for hepatic metastases, which are more common for this site. In general, retroperitoneal and truncal STS have worse prognoses than extremity STS.
- C. **Prognosis.** Almost 80% of metastases are to the lungs and occur within 2–3 years of diagnosis. If the pulmonary disease is resectable, 30% survival at 3 years can be expected. In addition, tumor size, grade, margins after resection, and anatomic location all have an impact on various outcome measures such as local recurrence, overall survival, or tumor-free survival. Local recurrence should be resected aggressively, and long-term follow-up is required, as late recurrences may occur.

IV. Surgical treatment

- A. **Resection.** Smaller, grade I tumors can be excised with a minimum 1-cm margin, usually without adjuvant radiation. Larger tumors may benefit from a larger margin or radiation to prevent recurrence. Grade II and III tumors, in general, require radiation therapy in addition to excision to avoid more radical surgery. Depending on the size and grade of tumor, compartment resection may be indicated.
- B. **Limb-sparing resection** combined with radiation therapy offers equivalent survival to amputation (*Ann Surg* 196:305, 1982). Limb-sparing procedures have a distinct psychological as well as functional advantage and are the procedures of choice for most tumors. The tumor should be removed with an envelope of normal tissue surrounding it, if possible. The resection should include the area of previous incision and biopsy and any drain sites. The resection field should be marked with clips to guide radiation therapy.
- C. **Retroperitoneal sarcomas** are considerably more difficult to treat because the tumors often involve vital structures, but general therapeutic principles remain the same (i.e., resection of as much tumor as possible with as wide a margin as possible). Radiation therapy may be used in some cases but is associated with relatively high morbidity. Wide margins as a rule are not achievable in the retroperitoneum and limit the effectiveness of therapy.

V. Other adjuvant therapy

- A. **Interstitial perioperative radiation therapy (brachytherapy)** involves the use of catheters or implants placed at the time of surgery to provide radiation directly to the tumor bed. Afterloading involves loading of the radiation source through catheters postoperatively to deliver localized high-dose radiation to the tumor bed. Brachytherapy has at least two advantages: It requires a short course of in-hospital treatment rather than 5–6 weeks of outpatient external-beam radiation therapy, and it may provide dose control near sensitive areas, such as joints and blood vessels. There is evidence that it is effective at decreasing local recurrence for high-grade tumors when combined with surgery.
- B. **Chemotherapy.** Several randomized prospective trials failed to show any improvement in survival with adjuvant chemotherapy for adult grade II or III sarcomas. The two drugs with the greatest efficacy are doxorubicin and ifosfamide; however, even these have at best a 40–60% response rate. The use of various chemotherapeutic agents, patterns of delivery, and their combination with cytokine administration continue to be evaluated and are currently being studied in randomized trials.
- C. **Isolated limb perfusion** provides increased delivery of therapy (e.g., hyperthermic therapy and chemotherapy) to an extremity sarcoma while reducing systemic toxicity. There is some suggestion of decreased local recurrence with definite downstaging of the tumor, but there is no improvement in survival. Although systemic toxicity may be reduced, local toxicity can be severe. This approach would benefit from a randomized prospective trial comparing it to wide excision (compartment) and external-beam radiation therapy.

- I. Inguinal hernias
- II. Femoral hernias
- III. Internal hernias
- IV. Abdominal wall hernias

I. Inguinal hernias

A. Incidence. The true incidence and prevalence of inguinal hernia remain unknown. In the United States, 756,000 inguinal hernia repairs were performed in 1996, of which 176,000 were bilateral. Laparoscopic studies have reported rates of contralateral defects as high as 22%, with 28% going on to become symptomatic during short-term follow-up. The male-female ratio is greater than 10:1. Lifetime prevalence is 25% in men and 2% in women. Two-thirds of inguinal hernias are indirect. Nearly two-thirds of recurrent hernias are direct. Inguinal hernias have an approximate incidence of incarceration of 10%, and a portion of these may become strangulated. Recurrence rates are less than 1% in children and vary in adults according to the method of hernia repair.

B. Terminology

1. **Direct hernias** are those in which viscera protrude through a weakness in the posterior inguinal wall. The base of the hernia sac is medial to the inferior epigastric vessels through Hesselbach's triangle, which is limited by the inferior epigastric artery, the lateral edge of the rectus sheath, and the inguinal ligament.
2. **Indirect hernia sacs** pass through the internal inguinal ring lateral to the inferior epigastric vessels and lie within the spermatic cord. The sac is covered by cremaster muscle fibers.
3. **In combined (pantaloon) hernias**, direct and indirect hernias coexist.
4. A **sliding hernia** (usually indirect inguinal in location) is a hernia in which a part of the wall of the hernia sac is formed by an intraabdominal viscus (usually colon, sometimes bladder). In a Richter's hernia, part (rather than the entire circumference) of the bowel wall is trapped. A Littre's hernia is one that contains a Meckel's diverticulum.
5. **Incarcerated hernias** cannot be reduced into the abdominal cavity, whereas strangulated hernias have incarcerated contents with vascular compromise; frequently, intense pain owing to ischemia of the incarcerated segment occurs.

C. Diagnosis

1. Clinical presentation

- a. **Most inguinal hernias** present as an intermittent bulge that appears in the groin, usually related to exertion or long periods of standing. The patient may complain of unilateral discomfort without noting a mass. Often, a purposeful Valsalva maneuver can reproduce the symptoms. In infants and children, a groin bulge often is noted by caregivers during episodes of crying or defecation. Rarely, patients present with bowel obstruction without noting a groin abnormality. All patients with a small-bowel obstruction must be questioned carefully and examined for hernias.
- b. **Physical examination.** The main diagnostic maneuver for inguinal hernias is palpation of the inguinal region. The patient is best examined while standing and straining (cough or Valsalva). Hernias manifest themselves as bulges with smooth, rounded surfaces that become more evident with straining. The hernia sac can also be examined more clearly by invaginating the hemiscrotum to introduce an index finger through the external inguinal ring. This may become uncomfortable for the patient and is unnecessary if an obvious bulge is present. Incarcerated inguinal hernias present with abdominal distention, nausea, and vomiting due to intestinal obstruction.

2. **Radiographic evaluation.** X-ray studies are rarely indicated. Ultrasonography or CT scanning may occasionally be used to diagnose an occult groin hernia, particularly in the obese patient. Plain abdominal X-rays may verify intestinal obstruction in cases of incarceration.

D. Differential diagnosis. Inguinal hernias should be distinguished from femoral hernias, which originate below the inguinal ligament. Inguinal adenopathy, lipomas, dilatation of the saphenous vein, epididymitis, testicular torsion, and groin abscess all should be considered in appropriate situations.

E. Treatment

1. **Preoperative evaluation and preparation.** Most patients with hernias should be treated surgically, although "watchful waiting" may be appropriate for elderly individuals with small asymptomatic hernias. Associated abnormalities that can increase intraabdominal pressure (such as chronic cough, constipation, or bladder outlet obstruction) should be evaluated and remedied to the extent possible before elective herniorrhaphy. In cases of intestinal obstruction and possible strangulation, broad-spectrum antibiotics and nasogastric suction may be indicated. Correction of volume status and electrolyte abnormalities is important when there is an associated small-bowel obstruction.
2. **Reduction.** Temporary management includes manual reduction. In uncomplicated cases, the hernia reduces with palpation over the inguinal canal with the patient supine. If this does not occur, the physician applies gentle pressure over the hernia with the concavity of the palm of his or her hand and fingers. The palm of the physician's hand exerts a steady but gentle pressure and also maintains the direction to be followed: craniad and lateral for direct hernias, craniad and posterior for femoral hernias. If the herniated viscera do not reduce, gentle traction over the mass with compression may allow bowel gas to leave the herniated segment, making the mass reducible. Sedation and the Trendelenburg position may be required for reduction of an incarcerated hernia, but it can be difficult to distinguish an acutely incarcerated hernia from a strangulated one because the inguinal canal can become quite tender without ischemic contents. When an incarcerated hernia is reduced nonsurgically, the patient should be observed for the potential development of peritonitis caused by perforation of a loop of strangulated bowel. If there is strong suspicion of strangulation, no attempt should be made to reduce the hernia because of the potential for *en masse* reduction of a gangrenous segment of bowel.

3. Surgical treatment

- a. **Choice of anesthetic.** Local anesthesia, which has several advantages over general or regional (spinal or epidural) anesthesia, is the preferred anesthetic for elective open repair. Local anesthesia results in better postoperative analgesia, a short recovery room stay, and a negligible rate of postoperative urinary retention; it is the lowest-risk anesthetic for patients with underlying cardiopulmonary disorders. Commonly, a mixture of short-acting (lidocaine 1%) and longer-acting (bupivacaine 0.25–0.50%) agents is used. The dose limits for local anesthesia are 300 mg plain lidocaine or 500 mg lidocaine with epinephrine and 175 mg plain bupivacaine or 225 mg with epinephrine. Use of local anesthesia for herniorrhaphy in our hospital is routinely supplemented by monitored anesthesia care and administration of intravenous midazolam and propofol. Virtually all patients who undergo hernia repair under local anesthesia can be managed as outpatients unless associated medical conditions or extenuating social circumstances necessitate overnight observation in the hospital. Laparoscopic hernia repair has been carried out under local or regional anesthesia but is more commonly done under general anesthesia.
- b. **Treatment of the hernia sac.** In indirect hernias, the sac is dissected free from the cord structures and cremasteric fibers. The sac should be opened away from any herniated contents. The contents are then reduced, and the sac is ligated deep to the internal ring with an absorbable suture. Large, indirect sacs that extend into the scrotum should not be dissected beyond the pubic tubercle because of an increased risk of ischemic orchitis. Similarly, one should avoid translocating the testicle into the inguinal canal during hernia repair owing to the risk of ischemia. Cord lipomas are frequently encountered during repair and should be excised to avoid future confusion with a recurrent hernia. Sliding hernia sacs can usually be managed by reducing the sac and attached viscera. Direct sacs are usually too broadly based for ligation and should not be opened but instead are simply freed from attenuated transversalis fibers and inverted. In preperitoneal repairs, the sac is usually reduced but not ligated because the repair is insinuated between the peritoneum and abdominal wall.
- c. **Inguinal floor reconstruction.** Some method of reconstruction of the inguinal floor is necessary in all adult hernia repairs to prevent recurrence. Various techniques for inguinal floor repair are available, and factors that influence the choice of repair include the type of hernia as well as the surgeon's preference and expertise. Three broad categories of repairs are available: primary tissue repairs, anterior tension-free mesh repairs, and preperitoneal repairs, including the laparoscopic approach.

1. **Primary tissue repairs.** Primary repairs without mesh have been the mainstay of hernia surgery for decades. The advantages of this approach are simplicity of the repair and the absence of any foreign body in the groin. Disadvantages include higher recurrence rates (5–10% for primary repairs and 15–30% for repair of recurrent hernias) due to tension on the repair and a slower return to unrestricted physical activity. Consequently, these repairs have become less popular in North America and have been supplanted by mesh repairs in many centers. The principal features of the more commonly performed tissue repairs are the following:

- a. **Bassini repair.** The inferior arch of the transversalis fascia or conjoint tendon is approximated to the shelving portion of the inguinal ligament with interrupted, nonabsorbable sutures ([Fig. 32-1](#)). The Bassini repair is appropriate for simple, indirect hernias, including most inguinal hernias in women ([Fig. 32-2](#)).

Fig. 32-1. Exposure of the right inguinal region for repair of an indirect hernia.

Fig. 32-2. Stitch placement for high ligation of the hernia sac, once separated from the cord structures, and the Bassini (iliopubic tract) repair.

- b. **McVay repair.** The transversalis fascia is sutured to Cooper's ligament medial to the femoral vein and the inguinal ligament at the level of, and lateral to, the femoral vein. This operation usually requires placement of a relaxing incision medially to avoid undue tension on the repair. The McVay repair closes the femoral space and, therefore, unlike the Bassini repair, is effective for femoral hernias.
 - c. **Shouldice repair.** In this repair, the transversalis fascia is incised (and partially excised if weakened) and reapproximated. The overlying tissues (the conjoint tendon, iliopubic tract, and inguinal ligament) are approximated in multiple, imbricated layers of running nonabsorbable suture. The experience of the Shouldice Clinic with this repair has been excellent, with recurrence rates of less than 1%, but higher recurrence rates have been reported in nonspecialized centers.
2. **Open tension-free repairs.** The most common mesh repairs are the tension-free mesh hernioplasty (Lichtenstein repair) and the patch-and-plug technique. In the Lichtenstein repair, an approximately 5-in. × 3-in. piece of polypropylene mesh is used to reconstruct the inguinal floor. The mesh is sutured to the fascia overlying the pubic tubercle inferiorly, the transversalis fascia and conjoint tendon medially, and the inguinal ligament laterally (Fig. 32-3). The mesh is slit at the level of the internal ring, and the two limbs are crossed around the spermatic cord and then tacked to the inguinal ligament, effectively creating a new internal ring of mesh. This repair avoids the approximation of attenuated tissues under tension, and recurrence rates with this technique have been consistently 1% or less. Moreover, because the repair is without tension, patients are allowed to return to unrestricted physical activity in 2 weeks or less. The mesh plug technique entails placement of a preformed plug of mesh in the hernia defect (e.g., internal ring) that is sutured to the rings of the fascial opening. An onlay piece of mesh is then placed over the inguinal floor, which may or may not be sutured to the fascia. Mesh plugs may be ideally suited for the repair of small, tight defects, such as femoral hernias.

Fig. 32-3. Placement of mesh to effect a tension-free conjoint tendon–to–iliopubic tract repair.

3. **Laparoscopic and preperitoneal repairs.** Approximately 15% of hernia repairs in the United States are now carried out laparoscopically, using a preperitoneal approach. The laparoscopic hernia repair is based on the technique of Stoppa, who used an open preperitoneal approach to reduce the hernia and placed a large piece of mesh to cover the entire inguinal floor and myopectineal orifice. Preperitoneal hernia repairs may also be performed without mesh, an approach that is rarely used today for routine hernia repair but that can be a good option in patients with strangulated hernias. Advantages of the preperitoneal approach in this setting are that it may facilitate reduction of the incarcerated or strangulated hernia contents, and, if gangrenous bowel is found, resection can be carried out through the preperitoneal incision, whereas this is difficult to accomplish through a standard groin incision.

In **laparoscopic hernia repair**, the preperitoneal space is reached by either transabdominal laparoscopy [the transabdominal preperitoneal (TAPP) procedure] or by a totally extraperitoneal repair (TEP). With the TAPP repair, the peritoneal space is entered by conventional laparoscopy at the umbilicus and the peritoneum overlying the inguinal floor is dissected away as a flap. With the TEP repair, the preperitoneal space is developed with a balloon inserted between the posterior rectus sheath and the peritoneum. The balloon is then inflated to dissect the peritoneal flap away from the posterior abdominal wall and the direct and indirect spaces, and the other ports are inserted into this preperitoneal space without ever entering the peritoneal cavity. The advantages of the TAPP approach are that there is a large working space, familiar anatomic landmarks are visible, and the contralateral groin can be examined for an occult hernia. The advantages of the TEP repair are that the abdominal cavity is not violated, the peritoneum is not opened, much of the dissection is done by balloon, and the procedure can be performed under regional or even local anesthesia (with sedation).

After laparoscopic dissection and reduction of the hernia sac, a large piece of mesh (6 in. × 4 in.) is placed over the inguinal floor. This is stapled superiorly to the posterior abdominal wall fascia on either side of the inferior epigastric vessels, medially to Cooper's ligament and the midline, and laterally to the iliopubic tract above the internal ring. Staples must not be placed in or posterior to the iliopubic tract or lateral to the iliac crest because of the risk of neurovascular injuries to the ilioinguinal, genitofemoral, lateral femoral cutaneous, and femoral nerves as well as the external iliac vessels. Comparative studies of laparoscopic and open hernia repair have shown that laparoscopic repairs are associated with less postoperative pain and faster recovery than open repairs, but hospital costs have been higher for the laparoscopic technique. Operative times, complications, and recurrence rates (<3% for laparoscopic repair) have been similar. Laparoscopic hernia repair is at this time still controversial for patients with a unilateral inguinal hernia. Special circumstances in which laparoscopic repair may be favored include (1) recurrent hernias to avoid the scar tissue in the inguinal canal; (2) bilateral hernias, because both sides of the groin can be repaired with the same three small incisions used for the unilateral repair; (3) individuals with a unilateral hernia for whom a rapid recovery is critical (e.g., athletes and laborers); and (4) obese patients. Laparoscopic hernia repair is contraindicated in patients who have large scrotal hernias or who have undergone prior extensive lower abdominal surgery.

- d. **Complications.** Surgical complications include wound hematoma, infection, nerve injury (ilioinguinal, iliohypogastric, genital branch of the genitofemoral, lateral femoral cutaneous, femoral), vascular injury (femoral vessels, testicular artery, pampiniform venous plexus), vas deferens injury, ischemic orchitis, and testicular atrophy. Recurrence rates after tension-free mesh repairs for primary hernias are 1–2% or less.
- e. It is more difficult to repair **recurrent inguinal hernias** adequately because the scar makes dissection difficult and the disease process has continued. Recurrence within 1 year of initial repair suggests an inadequate initial attempt, such as overlooking an indirect hernia sac. Recurrence after 2 or more years suggests progression of the disease process that caused the initial hernia (increased intraabdominal pressure, degeneration of tissues). Recurrences should be repaired because the defect usually is small with fixed edges that are prone to complications, such as incarceration or strangulation. Repair can be done by an anterior approach through the old operative field or by a posterior (open preperitoneal or laparoscopic) approach. Prosthetic mesh frequently is used for a tension-free repair and to reinforce attenuated tissues.

II. Femoral hernias

- A. **Incidence.** Femoral hernias constitute 2–4% of all groin hernias; 70% of femoral hernias occur in women. Of femoral hernias, 25% become incarcerated or strangulated, and a similar number are missed or diagnosed late.
- B. **Anatomy.** The abdominal viscera and peritoneum protrude through the femoral canal into the upper thigh. The boundaries of the femoral canal are the lacunar ligament medially, the femoral vein laterally, the iliopubic tract anteriorly, and Cooper's ligament posteriorly.
- C. **Diagnosis**
 1. **Clinical presentation**
 - a. **Symptoms.** Patients with femoral hernias may complain of an intermittent groin bulge or a groin mass that may be tender. Because femoral hernias have a high incidence of incarceration, small-bowel obstruction may be the presenting feature in some patients. Elderly patients, in whom femoral hernias occur most commonly, may not complain of groin pain, even in the setting of incarceration. Therefore, an occult femoral hernia should be considered in the differential diagnosis of any patient with small-bowel obstruction, especially if there is no history of previous abdominal surgery.
 - b. **Physical examination.** The characteristic finding is a small, rounded bulge that appears in the upper thigh just below the inguinal ligament. An incarcerated femoral hernia usually presents as a firm, tender mass. The differential diagnosis is the same as for inguinal hernia.

2. **Radiographic evaluation.** Radiographic studies are rarely indicated. Occasionally, a femoral hernia is found on a CT scan or gastrointestinal contrast study performed to evaluate a small-bowel obstruction.
- D. **Treatment.** The surgical approach can be inguinal, preperitoneal, or femoral.
1. **Inguinal approach.** A Cooper's ligament repair (McVay) through the inguinal canal approach allows reduction of the hernia sac with visualization from above the inguinal ligament and closure of the femoral space. Occasionally, it may be necessary to divide the inguinal ligament to reduce the hernia. The repair can be performed with or without mesh. This is the most familiar and preferred approach for uncomplicated femoral hernias.
 2. **Preperitoneal approach.** A transverse suprainguinal incision permits access to the extraperitoneal spaces of Bogros and Retzius. The hernia is reduced from inside the femoral space, and the hernia defect is repaired preperitoneally, usually with mesh, but can be repaired primarily. This approach is especially useful for incarcerated or strangulated femoral hernias. Uncomplicated femoral hernias can also be repaired laparoscopically.
 3. **Femoral approach.** A horizontal incision is made over the hernia, inferior and parallel to the inguinal ligament. After the hernia sac is dissected free, it can be resected or invaginated. The femoral canal is closed by placing interrupted stitches to approximate Cooper's ligament to the inguinal ligament or by using a plug of prosthetic material.
 4. **Complications.** Complications are similar to those for inguinal hernia repair. The femoral vein may be especially susceptible to injury because it forms the lateral border of the femoral canal.

III. Internal hernias

- A. **Incidence.** Of patients who present with acute intestinal obstruction, fewer than 5% have an internal hernia. When internal hernias are complicated by intestinal volvulus, there is an 80% incidence of strangulation or gangrene.
- B. **Etiology.** Internal hernias occur within the abdominal cavity owing to congenital or acquired causes. Congenital causes include abnormal intestinal rotation (paraduodenal hernias) and openings in the ileocecal mesentery (transmesenteric hernias). Other, less frequent types are pericecal hernias, hernias through the sigmoid mesocolon, and hernias through defects in the transverse mesocolon, gastrocolic ligament, gastrohepatic ligament, or greater omentum. Acquired causes include hernias through mesenteric defects created by bowel resections or ostomy formation. The small bowel may also herniate beneath an adhesion from previous surgery.
- C. **Diagnosis**
1. **Clinical presentation.** These hernias usually are diagnosed because an intestinal segment becomes incarcerated within the internal defect, resulting in small-bowel obstruction. Patients with congenital causes usually have not had prior abdominal surgery. The reported mortality in acute intestinal obstruction secondary to internal hernias is 10–16%.
 - a. **Symptoms** usually are of intestinal obstruction (see Chapter 14, [section I](#)) without evidence of external hernias. When there is intestinal obstruction or intestinal strangulation, the diagnosis is based on clinical rather than on laboratory findings.
 - b. **Physical examination** (see Chapter 14, [section I.C.2](#)).
 2. **Radiographic studies.** Plain abdominal films may show small-bowel obstruction. An abdominal CT scan can sometimes establish the diagnosis of an internal hernia preoperatively. Contrast studies may also sometimes be useful.
- D. **Differential diagnosis** includes other causes of intestinal obstruction, such as adhesions, external hernia, malignancy, gallstone ileus, and intussusception, among others (see Chapter 14, [section I.D](#)).
- E. **Surgical treatment.** The diagnosis of internal hernia is often made at laparotomy for small-bowel obstruction. Intestinal loops proximal to the obstruction are dilated, friable, and edematous above the obstruction and collapsed distal to it. Once the hernia is reduced, intestinal viability is assessed, and nonviable intestine is removed. If a large percentage of bowel is of questionable viability, a limited bowel resection followed by a laparotomy in 24–48 hours may preserve small-bowel length. The hernia defect should be closed primarily with nonabsorbable suture.

IV. Abdominal wall hernias

- A. **Incidence and etiology**
1. **Incisional hernias** occur at sites of previous incisions at which there has been dehiscence of the abdominal wall. The causes are multiple and include wound infections, obesity, malnutrition, and technical wound closure factors. Hernias occur in up to 14% of patients undergoing abdominal operations and are most commonly seen with midline incisions.
 2. **Umbilical hernias** are congenital defects. They are more frequent in African-Americans than in Caucasians. Most newborn umbilical hernias close spontaneously by the second year of life. However, umbilical hernias are also common in adults. Patients with ascites have a high incidence of umbilical hernias. When large, the hernias may cause gastrointestinal tract symptoms. When small, they rarely cause symptoms and may go unnoticed. Umbilical hernias have a fairly high rate of incarceration, usually with preperitoneal fat or omentum.
 3. **Epigastric hernias** are hernias of the linea alba above the umbilicus. They occur more frequently in athletically active young men. When small or in obese individuals, epigastric hernias may be hard to palpate, making the diagnosis difficult as well. Usually, they produce epigastric pain that may be falsely attributed to other abdominal diagnoses. The diagnosis is made by palpation of a subcutaneous epigastric mass; most such hernias occur within a few centimeters of the umbilicus and are associated with a small (1–2 cm) fascial defect.
 4. **Spigelian hernias** protrude through the Spigelian fascia, near the termination of the transversus abdominis muscle along the lateral edge of the rectus abdominis near the junction of the linea semilunaris and linea semicircularis. Because the herniated visceral contents are intraparietal (between the abdominal wall muscles), these hernias can be difficult to diagnose and therefore are included in the differential diagnosis of obscure abdominal pain. Ultrasonography, CT scan, or laparoscopy can be useful confirmatory tools in patients with focal symptoms in the appropriate region.
 5. The **most common type of lumbar hernia** is an incisional hernia from a previous retroperitoneal or flank incision. Lumbar hernias may also occur in two different triangles: Petit's triangle and Grynfeltt's triangle. Lower lumbar hernias of Petit's triangle are located in a weak area limited posteriorly by the latissimus dorsi, anteriorly by the external oblique muscle, and inferiorly by the iliac crest. Grynfeltt's hernias are upper lumbar in location, below the lowest rib.
 6. **Obturator hernias** are very rare hernias that occur predominantly in thin older women and are difficult to diagnose. Patients classically present with bowel obstruction and focal tenderness on rectal examination. Pain along the medial aspect of the thigh, known as the *Howship-Romberg sign*, results from obturator nerve compression and, when present, may aid in the clinical diagnosis of an obturator hernia.
- B. **Treatment and operative management.** Small epigastric, umbilical, obturator, and Spigelian hernias may be repaired primarily. Most incisional hernias and lumbar and obturator hernias require the use of prosthetic mesh because of their size, the often poor quality of surrounding tissue, and high recurrence rates after primary repair.
1. **Open repairs.** The principles for ventral hernia repair include dissection and identification of all defects and repair with nonabsorbable sutures placed in healthy tissue. Most sizable incisional hernias are now repaired with some type of mesh prosthesis that should be anchored by nonabsorbable sutures placed in healthy fascial tissue several centimeters beyond the margins of the defect. The mesh should be durable and well tolerated by the patient, with a low risk for infection. A variety of mesh products are available for repair, including polypropylene, polytetrafluoroethylene (PTFE, Gore-Tex), and composite mesh of polypropylene and PTFE. One should try to avoid placing polypropylene mesh in direct contact with the intestine because of the risk of adhesion formation and fistulization. Rarely, in patients with massive incisional defects and loss of domain of intestinal contents, preoperative pneumoperitoneum can be used to stretch the abdominal wall to provide sufficient autogenous tissue for repair. Peritoneal insufflation of air (500–1,000 mL per day for 5–10 days) may allow primary closure when not otherwise possible and may obviate the need for a prosthetic graft.
 2. **Laparoscopic repairs.** The laparoscopic approach is an increasingly used alternative method for repair of incisional hernias. The repair is generally performed intraabdominally with placement of an intraperitoneal mesh prosthesis to cover the hernia defect. The contents of the hernia should be reduced, but the sac itself is not removed. There should be a 3- to 5-cm margin of mesh lateral to the hernia defect. The mesh should then be anchored in place with sutures or staples. Early results show that the techniques are safe, simple, and effective, with equivalent, if not better, results than with open repairs. Early recurrence rates are reported at 1–10% and complication rates at 10–25%. Length of hospital stay and pain medication requirements are less than with open repairs. Contraindications to laparoscopic ventral hernia repair include inability to establish pneumoperitoneum safely, an acute abdomen with strangulated or infarcted bowel, or the presence of peritonitis.

33 Breast Surgery

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Diagnosis and Evaluation

I. Patients seek medical attention most commonly for an abnormal mammogram, a new breast mass, pain and tenderness without a mass, nipple discharge, or skin changes. **Patient history** should include questions regarding the duration of the symptoms or mass, change in size, associated pain or skin changes, relationship to pregnancy or the menstrual cycle, and previous trauma. Nipple discharge should be characterized according to its color and whether it is spontaneous, unilateral, or emanating from a single duct. Any skin changes of the nipple or areola should be noted. The hormonal history includes age of menarche, date of last menstrual period, regularity of menstrual cycle, number of pregnancies, age at first-term pregnancy, lactational history, and age at menopause or surgical menopause (note if oophorectomy performed). A history of previous breast biopsies, breast cancer, or cyst aspiration should be ascertained, including any known pathology results and treatment regimens. A history of previous oral contraceptive and hormonal replacement therapy should be elicited. The patient should provide dates of previous mammograms and location of the films. A detailed family history of breast and gynecologic cancer should be recorded, including the age at diagnosis and the location. This history should include at least two generations as well as any associated cancers, such as ovary, colon, or prostate (in men).

II. Physical examination

- A. **Inspect the breasts** with the patient in the upright position initially with the arms and pectoral muscles relaxed, then with the pectoral muscles contracted, and, finally, with the arms raised. Look for symmetry; deformity; skin changes, such as erythema or edema; and prior biopsy scars. Skin retraction may be more obvious with the patient's arms raised. The nipples are inspected for retraction, discoloration, inversion, ulceration, and eczematous changes.
- B. The **regional nodes should be palpated** with the patient in the upright position, pectoral muscles relaxed. Axillary and supraclavicular nodal regions are evaluated. Size, number, and fixation of nodes should be noted. The patient's breasts should be palpated in the upright and supine positions. In the supine position, the patient's breast is examined with the ipsilateral arm raised above and behind the head. The flat surface of the examiner's fingers should be used to palpate the entire breast systematically. The examination should extend to the clavicle, sternum, lower ribcage, and midaxillary line. If a dominant mass is palpated, its size, shape, texture, tenderness, fixation to skin or deep tissues, location, and relationship to the areola should be noted. A diagram in the chart noting these features is helpful. Examine for nipple discharge by palpating around the areola and noting where pressure elicits discharge. Note is made as to whether the discharge is from a single duct, its appearance (serous, sanguineous, etc.), and whether it is associated with a mass. A Hemocult test should be performed to evaluate for the presence of blood.

III. Breast imaging

A. Mammography

1. A **screening mammogram** is performed in the asymptomatic patient and consists of two standard views, mediolateral and craniocaudal. Patients are not examined by a mammographer, and the films are batch-read. Patients receive notification of any abnormalities. Studies have shown that screening mammography reduces mortality by 24% to 44%, depending on the age group. The current recommendation from the National Cancer Institute and American College of Surgeons is annual screening mammography for women aged 40 years and older. In the presence of hereditary breast cancer with known BRCA mutations, annual mammograms should begin at age 25–30 with semiannual physical examinations. In patients with a strong family history of undocumented genetic mutation, annual mammograms and semiannual physical examinations should begin 10 years earlier than the age of the youngest affected relative and no later than age 40 years.

The American College of Radiology has described categories for grading breast lesions ranging from 1 to 5, where 1 = negative; 2 = benign-appearing lesion; 3 = probably benign lesion, 6-month follow-up recommended; 4 = findings suspicious for breast cancer, biopsy recommended; and 5 = highly suspicious for breast cancer. Any suspicious lesion should undergo further evaluation.

2. **Diagnostic mammograms** are performed in the symptomatic patient or to follow up an abnormality noted on a screening mammogram. Patients are usually examined by a mammographer, and the films are interpreted immediately. Additional views, such as spot-compression views or magnification views, are performed to further characterize any lesions noted. The false-negative and false-positive rates are approximately 10%. A normal mammogram in the presence of a palpable mass does not exclude malignancy, and either further workup with a different imaging modality (ultrasound) or a biopsy should be performed. Mammography is not generally performed in lactating women or patients younger than age 30 years unless the degree of clinical suspicion is high. In the augmented breast, displacement views should be ordered to maximize the amount of parenchyma that can be visualized.
3. **Benign mammographic findings**
 - a. **Radial scar.** The finding of a radial scar on mammography is an indication for biopsy. This variant of fibrocystic breast condition (FBC) is associated with proliferative epithelium in the center of the fibrotic area, in approximately one-third of cases.
 - b. **Fat necrosis** can occur after local trauma to the breast, such as a seatbelt injury. The patient may not recall any history of trauma. Fat necrosis may resemble carcinoma on palpation and on mammography. Tissue diagnosis should be obtained to exclude carcinoma. The fat may liquefy instead of scarring, which results in a characteristic oil cyst.
 - c. **Milk of calcium** is associated with FBC and is caused by calcified debris in the base of the acini. Characteristic microcalcifications appear discoid on craniocaudal view and sickle-shaped on mediolateral view. These changes are benign and do not require biopsy.

- d. **Cysts** may feel like smooth, mobile, well-defined masses on palpation. If a cyst is tense with fluid, its texture may be firm, resembling a malignant mass. Solid masses cannot be distinguished from cysts by mammography. Aspiration can quickly determine the nature of the mass. Cyst fluid may vary in color from clear to straw-colored to dark green. Cytology is not routinely necessary. If the aspirate is bloody or a mass remains after drainage, excisional biopsy is indicated. If no palpable mass is present after drainage, the patient should be evaluated in 3–4 weeks. If the cyst recurs, it can be reaspirated; however, repeated recurrence is an indication for excisional biopsy to exclude intracystic tumor (*Arch Surg* 124:253, 1989).
4. **Mammographic findings** suggestive of malignancy are new or spiculated masses, clustered microcalcifications in linear or branching arrays, and architectural distortion.
- B. **Ultrasonography** is used to further characterize a lesion identified by either physical examination or mammography. Ultrasound can be used to determine whether a lesion is solid or cystic or to better define its size, contour, or internal texture. Although not a useful screening modality by itself due to significant false-positive and false-negative rates, when used as an adjunct with mammography, ultrasonography may improve diagnostic sensitivity of benign findings to greater than 90%, especially among younger patients for whom mammographic sensitivity is lower.
- C. **Other imaging modalities.** MR scan of the breast is still considered experimental. It has been shown to be useful as an adjunct to mammography in detecting multicentric disease and in evaluating the dense distorted breast. It is also the most sensitive modality for evaluating the integrity of implants. Limitations of MR include an inability to identify microcalcifications or differentiate between inflammatory breast cancer and abscesses.

IV. Breast biopsy

A. Palpable masses

1. **Fine-needle aspiration biopsy (FNAB)** is a reliable and accurate office technique with sensitivity greater than 90%. A 22- to 25-gauge needle on a 10-mL syringe is advanced into the mass, and suction is applied. The needle is moved back and forth within the tumor with quick short strokes in nearly the same line as the original puncture. Cells are collected in the hub of the needle. The suction is released and the needle withdrawn. The contents of the needle are expelled onto a glass slide. A second glass slide is inverted over the first, and the two are pulled apart. One slide is fixed immediately, and the second is allowed to air dry. Two to three passes are performed for a total of four to six slides. False-negative findings are caused by inadequate sampling or improper specimen processing. FNAB results should be concordant with clinical impression and mammographic findings of the lesion (triple diagnostic test). Fine-needle aspiration diagnoses the presence of malignant cells; however, it does not give information on tumor grade or the presence of invasion. Fewer than 5% of malignant masses are comprised of ductal carcinoma *in situ* (DCIS). Nondiagnostic or indeterminate aspirates do not exclude malignancy and require a surgical biopsy (*Am J Surg* 174:372, 1997). Estrogen and progesterone receptors can be determined by immunohistochemistry on malignant FNAB specimens.
2. **Core biopsy**, with either a Tru-Cut or spring-loaded Monopty device, can be used to obtain more tissue. The skin is infiltrated with lidocaine and a nick made in the skin. The needle is inserted into the mass and fired. Three to five cores are taken and placed in formalin. Invasion, grade, and receptor status can be determined. For indeterminate specimens, an open surgical biopsy is necessary.
3. **Excisional biopsy** is performed in the operating room using local anesthesia and intravenous sedation. Incisions should be oriented along Langer's lines for optimal cosmesis (curvilinear, parallel to the areola). All incisions should be planned such that they can be incorporated into a mastectomy incision. Masses should be excised as a single specimen, oriented such as placing a short suture superiorly and long suture laterally, and margins inked. Improper specimen handling may obscure margin status.
4. **Incisional biopsy** removes a wedge of tissue from a palpable breast mass. It is indicated for the evaluation of a large breast mass that is suspicious for malignancy for which a definitive diagnosis cannot be made by FNAB or core biopsy.

B. Nonpalpable lesions

1. **Stereotactic core biopsy** is a minimally invasive method of obtaining core samples of impalpable, mammographically suspicious lesions under radiographic control. This **technique** is ideally suited to establish tissue diagnoses of several foci in disparate quadrants of the breast. Using a computer-driven stereotactic unit, two mammographic images, each at a 15-degree angle from the center, are taken to triangulate the position of the site to be biopsied in three-dimensional space. A computer determines the depth of the lesion and alignment of the needle, which can be positioned within 1 mm of the intended target. Biopsies are taken, and postfire images are obtained of the breast and specimen. **Contraindications** include lesions close to the chest wall or in the axillary tail and thin or ptotic breasts that would allow needle strike-through. Radial scars should undergo needle-localized biopsy (NLB) because the entire lesion must be evaluated for definitive diagnosis. A diagnosis of atypical ductal hyperplasia (ADH) mandates NLB to exclude the presence of coexistent DCIS or invasive cancer, which can be missed by core needle biopsy. Core needle biopsy may underestimate the degree of pathology in lesions that contain ADH and DCIS. As many as 50% of women with ADH and 20% of women with DCIS on core biopsy were determined to have DCIS or invasive cancer, respectively, at the time of surgical excision. For indeterminate specimens, an open surgical biopsy is necessary. Nondiagnostic and insufficient specimens should also undergo NLB.
2. **Vacuum-assisted biopsy**, with either the Mammotome (Ethicon Endo-Surgery, Cincinnati, OH) or the Minimally Invasive Breast Biopsy device (US Surgical, Norwalk, CT), has arisen as a result of the difficulties that FNAB and core biopsy have with evaluating microcalcifications and DCIS. The Mammotome uses an 11-gauge biopsy probe to contiguously acquire tissue, which is pulled into the probe by vacuum suction. The benefit of this tool is that it can pull back several, larger volume samples of tissue into the probe while the device remains in the breast. This technique allows removal of all of the tissue around a cluster of calcifications in a single insertion of the probe. This device also has the ability to place a marking clip through the probe to allow for future identification of the biopsy site. These modalities have fewer underestimates of ADH and DCIS pathology than does FNAB or core biopsy and have become the most commonly used tissue acquisition instruments for the percutaneous biopsy of DCIS lesions (*JACS* 192:197, 2001).
3. **NLB** is performed by placing a needle and hookwire into the patient's breast adjacent to the lesion under mammographic guidance. The patient is then brought to the operating room. With the localization mammograms as a map, an excisional biopsy is performed, encompassing the tissue around the wire and lesion. The specimen is oriented and a radiograph obtained to confirm the presence of the lesion in the specimen. It is not necessary to remove skin around the needle insertion site.
4. **Emerging techniques.** Iodine-125 seed localization biopsy is a new technique that avoids needle placement for localization and allows for greater flexibility in operative planning. A titanium seed containing 0.05–0.3 mCi ¹²⁵I is inserted into the breast lesion or area of microcalcifications by the nuclear medicine radiologist under radiographic guidance, and a skin marker is placed. The titanium seed is localized by dissection with the help of a hand-held gamma detector, and the tissue around the seed is excised. The remaining cavity can be probed for residual activity to ensure adequate circumferential dissection, and the biopsy specimen is examined radiographically to confirm removal of the seed and the lesion. Seeds can be inserted the day before a scheduled biopsy to allow for more flexibility in operative planning.

V. Assessment of breast cancer risk

- A. Breast cancer is the most common malignancy among women worldwide (*Int J Cancer* 80:827, 1999). **Genetics, hormonal exposure, or pathologic factors** may be correlated to a risk for breast cancer. A family history of breast cancer in a first-degree relative is associated with an approximate doubling of risk. If two first-degree relatives have a history of breast cancer (i.e., a mother and a sister have had breast cancer), the risk is even higher. These familial effects are enhanced if the relative had either early-onset cancer or bilateral disease. Factors that increase a patient's risk by 1.5- to 4.0-fold include increased exposure to estrogen or progesterone by early menarche (before age 12 years) and late menopause (age >55 years), high body-mass index after menopause, presence of hyperplastic breast tissue with atypia, and exposure to ionizing radiation. A late age at first full-term pregnancy is an important determinant of breast cancer risk. Women with a first birth after age 30 years were shown to have twice the risk of those with a first birth before age 18 years. Breast-feeding may exert a protective effect from the development of breast cancer. Lifetime and 5-year breast cancer risk can be estimated using the Gail model, which is based on age, onset of menses, onset of menopause, age at first birth, and prior breast biopsies. This model is used for entering women into chemopreventive trials.
- B. **Certain pathologic features observed on breast biopsy** can be associated with increased breast cancer risk. No increased risk is associated with adenosis, cysts, duct ectasia, or apocrine metaplasia. There is a slightly increased risk with moderate or florid hyperplasia, papillomatosis, and complex fibroadenomas. Atypical ductal or lobular hyperplasia carries a four- to fivefold increased risk of developing cancer; risk increases to 10-fold if there is a positive family history. Patients with increased risk should be counseled appropriately. Those with atypia or lobular carcinoma *in situ* (LCIS) should be followed with semiannual physical examinations and yearly mammograms.
- C. **BRCA.** BRCA1 and BRCA2 are breast cancer susceptibility genes associated with 80% of hereditary breast cancers and account for approximately 5–10% of all breast cancers. Women with BRCA1 mutations have an estimated risk of 85% for breast cancer by age 70 years, a 50% chance of developing a second primary breast cancer, and a 20% chance of developing ovarian cancer. BRCA2 mutations carry a lower risk for breast cancer and account for 4–6% of all male breast cancers. Surveillance should include a monthly breast self-examination, semiannual clinical examination, and annual mammography beginning at age 25–35 years. Screening for BRCA1 and BRCA2 gene mutations should be reserved for women with a strong family history who have undergone a multidisciplinary evaluation including genetic counseling. Prophylactic bilateral mastectomy provides a 90–100% cancer risk reduction and is an option for some patients. Preliminary studies show a significantly reduced risk of breast cancer after prophylactic oophorectomy in patients with BRCA mutations; however, no large randomized prospective trials have been completed to date.
- D. **ErbB2 (Her2/neu) oncogene overexpression** is seen in approximately 30% of breast adenocarcinomas, and its presence in a tumor specimen is a negative prognostic factor. Current research is investigating methods of targeting this oncogene for future therapies.

E. Chemoprevention. The first large chemopreventive trial was conducted by the National Surgical Breast and Bowel Project (NSABP) P-1. This was a large, randomized prospective trial begun in 1992 to evaluate the use of tamoxifen, an estrogen antagonist, as a cancer prevention drug in women at risk for developing breast cancer. This study entered 13,175 women with a Gail model score of 1.7 and an average follow-up of 47.7 months. Women taking tamoxifen achieved an overall risk reduction of developing invasive breast carcinoma of 49% and a 50% risk reduction of developing noninvasive breast cancer. In the subgroup of women with a history of LCIS or atypical hyperplasia, tamoxifen reduced the risk of developing invasive breast cancer by 65% and 86%, respectively. Tamoxifen also provided a significant reduction in hip fractures in women over 50 years of age. There was no difference noted in the incidence of ischemic heart disease for women taking tamoxifen. The NSABP B-24 trial looked at the benefit of tamoxifen in women with DCIS as adjuvant therapy after lumpectomy and radiation. After a median follow-up of 74 months, tamoxifen provided a 37% overall risk reduction in all breast cancers (invasive and noninvasive). Toxicities of this drug include an increased risk of endometrial cancer and thrombotic vascular events. Women on tamoxifen also reported increases in vasomotor symptoms (hot flashes) and vaginal discharge. The U.S. Food and Drug Administration–approved uses of tamoxifen are for (1) the treatment of metastatic breast cancer, (2) adjuvant treatment of breast cancer, and (3) chemoprevention of invasive or contralateral breast cancer in high-risk women. The dosage approved for chemoprevention is 20 mg per day for 5 years. It is estimated that chemoprevention could prevent as many as 500,000 invasive and 200,000 noninvasive breast cancers over 5 years in the United States alone. Current clinical trials are evaluating newer selective estrogen-receptor modulators as well as retinoids, peroxisome proliferator-activated receptor-gamma ligands, and cyclooxygenase-2 inhibitors.

Nonmalignant Breast Conditions

I. Fibrocystic breast changes is a descriptive term encompassing several of the following pathologic features: stromal fibrosis, macro- and microcysts, apocrine metaplasia, hyperplasia, and adenosis, which may be sclerosing, blunt-duct, or florid. FBC is common and may present as breast pain, mass, nipple discharge, or abnormalities on mammography. The patient presenting with a breast mass or thickening and suspected FBC should be reexamined on day 10 of the menstrual cycle, when the hormonal influence is at a nadir. Often, the mass will have diminished in size. A persistent dominant mass must undergo further evaluation, biopsy, or both to exclude carcinoma.

II. Breast cysts frequently present as a tender mass or as a smooth, well-defined mass on palpation. Symptomatic cysts should be aspirated. Cysts discovered by mammography are confirmed as simple cysts by ultrasound and, if asymptomatic, are usually observed. Cysts associated with a solid mass that recur after aspiration or have a bloody aspirate should be excised.

III. Fibroadenoma is the most common discrete mass in women younger than 30 years. Fibroadenomas enlarge during pregnancy and involute after menopause. They present as smooth, firm mobile masses. On mammography and ultrasound they have well-circumscribed borders. These masses may be managed conservatively if clinical and radiographic appearance is consistent with a fibroadenoma. If, however, the mass enlarges or is greater than 2 cm, it should be excised.

IV. Breast pain. Most women experience some form of breast pain or discomfort during their lifetime. The pain may be cyclic or not, focal, or diffuse. Benign disease is the etiology in the majority of cases. However, pain may be associated with cancer in up to 10% of patients. Features that raise the suspicion of cancer are noncyclic pain in a focal area, pain associated with a mass, or bloody nipple discharge. Once cancer has been excluded, most patients can be managed successfully with reassurance. In 15% of patients, however, the pain may be so disabling that it interferes with activities of daily living.

A. Cyclic breast pain is often described as a heaviness or tenderness. It may be maximal in the upper outer quadrant and radiate to the inner surface of the upper arm. Many patients experience symptomatic relief by reducing the caffeine content of their diet or by ingesting vitamin E (400–800 units per day), although there is no scientific proof that these methods are valuable.

- 1. Evening primrose oil** (1,000 mg t.i.d.) is the first-line treatment. It contains the essential fatty acids linoleic and linolenic acid. Evening primrose oil is thought to act by increasing the synthesis of prostaglandin E₁, which inhibits the action of prolactin peripherally. After 2–4 months of ingestion, more than 50% of women experience improvement with few adverse reactions. Evening primrose oil can be purchased over the counter at health food stores.
- 2. Danazol** (a derivative of testosterone) is reserved for severe breast pain that fails to respond to other measures. The initial dosage is 100 mg per day for 2 months to induce remission; this is decreased to 100 mg per day from days 14 to 28 of the menstrual cycle. The dosage is reduced progressively thereafter. Adverse reactions include hirsutism, voice changes, acne, amenorrhea, and abnormal liver function. Treatment should start on the first day of the menstrual cycle to exclude pregnancy.

B. Noncyclic breast pain occurs in premenopausal and in postmenopausal women. It is described as burning, stabbing, or drawing and frequently occurs in the subareolar area or medial aspect of the breast. Evening primrose oil is the first-line treatment, followed by danazol for severe pain. An injection of lidocaine and prednisolone into the tender spot is helpful in some patients. Excision of the trigger spot via breast biopsy results in a 50% response rate.

C. Tietze's syndrome or costochondritis may be confused with breast pain. It may be unilateral or bilateral and involve the second to fourth costal cartilages. Patients are locally tender in the parasternal area. Treatment is with nonsteroidal antiinflammatory agents.

D. Superficial thrombophlebitis of the veins overlying the breast and occasionally the upper abdomen is referred to as *Mondor's disease*. It may present with breast pain. A cord can be palpated corresponding to the thrombosed vein. It is a self-limiting condition and usually resolves over several weeks. Nonsteroidal antiinflammatory agents may be helpful. Hot compresses may provide symptomatic relief.

E. Breast pain in pregnancy and lactation can occur from several other sources, including engorgement, clogged ducts, trauma to the areola and nipple from pumping or nursing, or any of the above-mentioned sources. Clogged ducts are usually treated with warm compresses, soaks, and massage.

F. Cervical radiculopathy can also cause referred pain to the breast.

V. Nipple discharge

A. Lactation is the most common physiologic cause of nipple discharge and may continue for up to 2 years after cessation of breast-feeding. In parous nonlactating women, a small amount of milk may be expressed from multiple ducts bilaterally. This requires no treatment.

B. Galactorrhea is milky discharge unrelated to breast-feeding. Physiologic galactorrhea is the continued production of milk after lactation has ceased and menses resumed. Often, it is caused by continued mechanical stimulation of the nipples.

- 1. Drug-related galactorrhea** is caused by medications that affect the hypothalamic-pituitary axis by depleting dopamine (tricyclic antidepressants, reserpine, methylidopa, cimetidine, or benzodiazepines), blocking the dopamine receptor (phenothiazine, metoclopramide, or haloperidol), or having an estrogenic effect (digitalis). Discharge is generally bilateral and nonbloody.
- 2. Spontaneous galactorrhea in a nonlactating patient** may be due to a pituitary adenoma or to a microadenoma producing prolactin. Amenorrhea may be an associated feature. The diagnosis is established with a serum prolactin level and computed tomographic (CT) scanning or MR scan of the pituitary gland. Treatment is with bromocriptine or surgical removal.

C. Pathologic nipple discharge is spontaneous and unilateral, originates from a single duct, and is either serous, serosanguineous, bloody, or watery. The presence of blood can be confirmed with a Hemoccult test. Cytologic evaluation of the discharge is not generally useful. In the absence of detectable abnormality by either physical examination or mammography, the most likely etiologies are benign intraductal papilloma, duct ectasia, or fibrocystic changes; however, malignancy is the underlying cause in 10% of patients. In lactating women, serosanguineous or bloody discharge can also be associated with duct trauma, infection, or epithelial proliferation associated with breast enlargement. A solitary papilloma with a fibrovascular core places the patient at marginally increased risk for the development of breast cancer. Patients with persistent spontaneous discharge from a single duct require a surgical microdochectomy or major duct excision.

- 1. Microdochectomy** involves excision of the involved duct and associated lobule. This procedure is performed with local anesthesia and often with intravenous sedation. Immediately before surgery, the involved duct is cannulated, and radiopaque or methylene blue contrast is injected. The ductogram identifies the location of the lesions as filling defects and serves as a guide to excision. After the ductogram is obtained, the patient is taken to the operating room. Through a circumareolar incision, the duct is identified and excised, along with the associated lobule.
- 2. Major duct excision** is performed through a circumareolar incision. An areolar flap is raised to reach the major ducts. All the retroareolar ducts are transected and excised, along with a cone of tissue extending up to several centimeters posterior to the patient's nipple. A preoperative ductogram is obtained to identify the location of the lesion. Major duct excisions are rarely indicated because the lesions should be identified on ductogram. Major duct excision may be used for women with bloody nipple discharge from multiple ducts or in postmenopausal women with bloody nipple discharge.

VI. Breast infections

A. Lactational mastitis may occur either sporadically or in epidemics. The most common causative organism is *Staphylococcus aureus*. The patient's breast is swollen, erythematous, and tender. Purulent discharge from the nipple is uncommon. In the cellulitic phase, it is treated with oral or intravenous antibiotics. If in the early stages of infection, the frequency of nursing or pumping should be increased. Approximately 25% of cases progress to abscess formation. Breast

abscesses often are not fluctuant, and therefore the diagnosis is made by failure to improve on antibiotics, abscess cavity seen on ultrasound, or aspiration of pus. Treatment is cessation of nursing, surgical drainage, and wound packing.

- B. **Nonpuerperal abscess** occurs as a result of duct ectasia with periductal mastitis, infected cysts, infected hematoma, or hematogenous spread from another source. If the abscess is subareolar, anaerobes are the most common causative agent. Treatment is with the appropriate antibiotic or surgical drainage for an abscess. These abscesses have a high recurrence rate. For women who experience recurrence, treatment is a central duct excision. Recurrent or unresolved infection requires biopsy of the abscess cavity to exclude cancer.

VII. Duct ectasia, periductal mastitis, or mammary fistula. The causes of duct ectasia and periductal mastitis are unclear. These conditions may present as subareolar abscess, periareolar cellulitis, and thick discharge from the nipple, or a fistula. Patients often have a chronic relapsing course with multiple infections requiring surgical drainage. Both anaerobic and aerobic organisms can be cultured from the ectatic ducts. Antibiotic treatment should cover both types of organisms. Repeated infections can result in a chronically draining periareolar lesion or a mammary fistula, which is lined with squamous epithelium. These are treated by duct excision along with excision of the fistula once the acute infection resolves. The patient should be advised that the condition can recur, even after surgery.

VIII. Gynecomastia is defined as hypertrophy of breast tissue in men. Pubertal hypertrophy occurs in young adolescent boys, is usually bilateral, and resolves spontaneously in 6–12 months. Senescent gynecomastia is commonly seen after age 70 years, as testosterone levels decrease. Drugs or excessive hormone production may cause gynecomastia in adults. Drugs associated with gynecomastia are similar to those that cause galactorrhea in women and include digoxin, spironolactone, methyl dopa, cimetidine, tricyclic antidepressants, phenothiazine, reserpine, and marijuana. Excess hormonal secretion of estrogens may be due to such tumors as testicular teratomas and seminomas, bronchogenic carcinomas, and adrenal tumors. Tumors of the pituitary and hypothalamus may also cause breast enlargement. Gynecomastia may be a manifestation of such systemic diseases as hepatic cirrhosis, renal failure, and malnutrition. Carcinoma should be excluded by mammography and biopsy. Excision of breast tissue via a periareolar incision is performed if workup fails to reveal a medically treatable cause or if the enlargement fails to regress or is cosmetically unacceptable.

IX. Breast conditions during pregnancy. Bloody nipple discharge may occur in the second or third trimester. It is the result of epithelial proliferation under hormonal influences and usually resolves by 2 months postpartum. Breast masses occurring during pregnancy include galactoceles, lactating adenoma, simple cysts, breast infarcts, and carcinoma. Fibroadenomas may grow during pregnancy due to hormonal stimulation. Masses should be carefully evaluated by ultrasound, and a biopsy should be performed for any suspicious lesion. Ultrasound distinguishes between a solid mass and cysts. FNAB, vacuum-assisted biopsy, and core biopsy can be safely performed. Mammography can be performed with uterine shielding but is rarely helpful due to the increased density of the patient's breast. If a breast lesion is diagnosed as malignant, the patient should be given the same treatment options stage for stage as a nonpregnant woman, and the treatment should not be delayed because of the pregnancy.

Malignancy of the Breast

Management of breast cancer is multidisciplinary, involving cooperation between the surgeon, radiation oncologist, and medical oncologist. Current staging of breast cancer is based on the TNM (tumor, node, metastasis) staging system ([Table 33-1](#) and [Table 33-2](#)). Patients should be assigned a clinical stage based on physical examination, which may be modified when the final pathology report is available.

Table 33-1. American Joint Committee on Cancer TNM (tumor, node, metastasis) staging for breast cancer

Table 33-2. American Joint Committee on Cancer classification for breast cancer based on TNM (tumor, node, metastasis) criteria

I. Preoperative staging in breast cancer (stages I and II). Before definitive surgery is undertaken, preoperative workup should include complete blood cell count, complete metabolic panel, and chest radiograph. A bone scan should be obtained if alkaline phosphatase or calcium level is elevated. Alterations in liver function tests are indications for CT scan of the liver. If metastatic disease is detected, local treatment is no longer a priority, and the patient should undergo chemotherapy, hormonal therapy, or both.

II. Noninvasive breast pathology is confined to the mammary ducts or lobules and is classified as either DCIS or LCIS, respectively.

- A. **DCIS**, or intraductal carcinoma, is treated as a malignancy. These lesions comprise malignant cells that have not penetrated the basement membrane. Mammographic screening has significantly increased the diagnosis of DCIS; because of this, DCIS is currently the subtype of breast cancer most rapidly increasing in incidence, with 39,000+ new cases in the United States in 1999 (18% of all new breast cancers). The most common mammographic findings are clustered pleomorphic calcifications. The physical examination is normal in the majority of patients. Impalpable, mammographically detected lesions require either NLB, stereotactic, or vacuum-assisted biopsy to obtain material for histology.
1. Several **classification systems** have been proposed for DCIS. From a DCIS pathology consensus conference in 1997, no unified classification system was agreed on; however, factors such as margin status, tumor size, nuclear grade, cell polarization, and architecture should be recorded on specimens. Although there are five architectural subtypes (papillary, micropapillary, solid, cribriform, and comedo), specimens are mainly grouped as comedo versus noncomedo. The comedo or high-grade subtype is more often associated with microinvasion, a higher proliferation rate, aneuploidy, Her2/neu gene amplification, and a higher local recurrence rate. DCIS may advance in a segmental manner with gaps between disease areas. Lesions can be multifocal (2 or more lesions greater than 5 mm apart within the same index quadrant) or multicentric (in different quadrants).
 2. Although complete **excision** should be curative, excision alone has a reported local recurrence rate of up to 40% at 5 years, with one-half the recurrences presenting as invasive ductal carcinoma. Margin status is an important factor in predicting risk of local recurrence, as patients with free margins greater than 10 mm have a 10–15% likelihood of recurrent disease (after additional radiation therapy). Traditionally, these patients were treated with mastectomy, which carries a 0–2% recurrence rate; however, based on recent clinical trials, breast conservation surgery is as effective as mastectomy in overall survival. Therefore, therapeutic options range from simple excision to total mastectomy or skin-sparing mastectomy with reconstruction, depending on the size, grade, margin status, multicentricity of disease, and age of the patient. Although the NSABP B-17 trial demonstrated that adjuvant radiation therapy was effective in decreasing the rate of recurrence in patients with DCIS, it may not be necessary for all patients with DCIS. Current clinical trials are evaluating which patients may benefit the most from radiation therapy. The addition of adjuvant radiation has not been shown to affect breast cancer mortality. However, it should be given to patients with DCIS who wish breast conservation. This is especially true for younger women with close margins or large tumors. Adjuvant tamoxifen may further reduce the risk of recurrence in these patients.
 3. Axillary dissection is not performed for pure DCIS. For patients with extensive DCIS lesions that are treated with mastectomy, a **sentinel lymph node biopsy (SLNB)** can be performed at the time of mastectomy to evaluate the axilla. The sentinel lymph node should be evaluated by hematoxylin and eosin staining followed by immunohistochemistry for cytokeratin if negative. A positive sentinel node indicates invasive breast cancer, changing the stage of the disease and possibly the treatment as well. For pure DCIS, there is no added benefit to chemotherapy, because this disease is confined to the ducts of the breast; however, adjuvant tamoxifen has been shown to reduce the risk of breast cancer recurrence by 37% over 5 years as well as to decrease the risk of developing cancer in the contralateral breast (NSABP B-24 trial). The Van Nuys Prognostic Index ([Table 33-3](#)) is a numerical algorithm used by many surgeons to stratify patients into three groups to determine who is at greatest risk of developing recurrent disease and would therefore benefit the most from a more aggressive treatment approach. This index uses several measurable factors (lesion size, margin width, grade, and presence of necrosis) to stratify patients into three groups. The low-scoring group is treated with excision alone, as no difference in recurrence rate is demonstrated with the addition of radiation. The intermediate-scoring group has been shown to benefit from adjuvant radiation therapy, and the high-scoring group should undergo

mastectomy, as the risk of recurrence with conservative excision is high (*Adv Surg* 34:29, 2000).

Table 33-3. Van Nuys scoring system*

B. **LCIS** is an incidental pathologic finding in a breast biopsy specimen. It may be multifocal or bilateral. It is not considered a preinvasive lesion but rather an indicator for increased breast cancer risk of approximately 1% per year or 35% lifetime risk. The cancer may be either invasive ductal or lobular and may occur in either breast. Two treatment options are currently accepted: either lifelong surveillance or possible prophylaxis with tamoxifen in the setting of a clinical trial. Bilateral total mastectomies with immediate reconstruction are reserved for selected women with a strong family history of breast cancer and LCIS, after appropriate counseling. Mastectomy for LCIS is much less frequently performed. If surveillance is chosen, patients should perform monthly self-examination and have annual mammograms and semiannual clinical examinations.

III. Invasive breast cancers are often histologically heterogeneous. Adenocarcinoma of the breast can be divided into five different histologic subtypes: infiltrating ductal (75–80%), infiltrating lobular (5–10%), medullary (5–7%), mucinous (3%), and tubular (1–2%). Surgical options for early-stage (I and II) invasive breast cancer include modified radical mastectomy (total mastectomy and axillary dissection) with or without reconstruction or breast conservation therapy (BCT), consisting of lumpectomy and axillary dissection (or SLNB) followed by breast irradiation. Skin-sparing mastectomy with immediate autologous reconstruction is an excellent alternative to mastectomy alone.

A. **Axillary lymph node dissection (ALND)** or SLNB should be performed in all patients with stage I and II breast cancer for staging purposes, control of the axilla, or both. Axillary staging is a component of modified radical mastectomy and BCT. ALND involves removal of level I (lateral to the pectoralis minor muscle) and level II lymph nodes (posterior to the pectoralis minor muscle) and, if grossly involved, possibly level III nodes. An adequate dissection should remove at least eight lymph nodes. Intraoperative complications of axillary dissection include damage to the long thoracic, medial pectoral, thoracodorsal, and intercostobrachial nerves. Postoperatively, the most frequent complications include wound infections and seromas. Several prospective studies have demonstrated a significant decrease in wound infection and seroma rates with the use of one preoperative dose of a cephalosporin and the placement of at least one closed suction drain. Persistent seroma may be treated with repeated aspirations or reinsertion of a drain. One long-term complication of ALND is the increased risk of upper-extremity lymph edema.

For surgeons with adequate experience, **SLNB** has been established as a useful minimally invasive technique for predicting axillary involvement in patients with T1 or T2 tumors. It involves intraoperative lymphatic mapping using **lymphazurin** blue dye or technetium-labeled sulfur colloid (or both) to identify the primary draining lymph node(s) in the nodal basin. Twenty percent to 30% of the time, more than one SLN is identified. The histology of the SLN predicts the involvement of the remaining axillary nodes. If the SLN(s) is negative, a more extensive lymph node dissection can be avoided. If the SLN is positive, a standard axillary dissection is performed or radiation therapy is given to the axilla. The procedure requires a multidisciplinary approach, including nuclear medicine, pathology, and radiology. In experienced hands (performing at least 30 SLNBs with ALND for confirmation), surgeons can identify the SLN in greater than 90% of patients, accurately predicting the patient's remaining lymph node status in 97–99% of cases. Serial sectioning and immunohistochemical staining of SLNB specimens may improve accuracy in detecting micrometastatic disease. Large current trials [NSABP B-32, American College of Surgeons Oncology Group (ACOSOG) z-0010 and z-0011] are under way to compare long-term regional control of disease and overall survival in women who undergo SLNB alone versus those who undergo SLNB followed by standard axillary dissection. These trials will help establish what role this modality and the predictive value of immunohistochemistry detection of micrometastasis will serve in the care of these breast cancer patients.

B. **BCT** is comprised of complete surgical excision of the cancer followed by radiation therapy and may be offered to patients with reasonable tumor-breast ratios. For patients with large tumors who desire BCT, preoperative chemotherapy or hormonal therapy may be offered to reduce the size of the tumor. Adjuvant radiotherapy has been demonstrated to decrease the breast cancer recurrence rate from 30% to less than 7% at 5 years. Radiotherapy is administered daily on an outpatient basis 5 days per week for approximately 6 weeks. After surgery, patients receive 4,500–5,000 cGy radiation to the breast, usually with a boost to the tumor bed.

1. **Contraindications to lumpectomy/quadrantectomy with adjuvant radiation** include two or more primary tumors in separate quadrants of the breast, persistently positive margins after multiple attempts at complete resection, pregnancy (especially in the first or second trimesters), prior radiation to the breast region, collagen vascular disease (e.g., scleroderma), diffuse disease throughout the breast precluding excision with negative operative margins, and the unavailability of radiation therapy. Extensive intraductal component (i.e., 25% of the primary tumor is intraductal) is not considered a contraindication to BCT, provided that microscopically negative margins can be obtained.
2. **Technique of lumpectomy.** Incisions should be curvilinear and parallel to the nipple-areolar complex. A gross margin of 1 cm should be attempted and the specimen oriented as already described. A small ellipse of skin is often removed to help orient the specimen, and meticulous hemostasis is achieved. The tumor bed can be marked with radiopaque clips. Use of drains impairs cosmesis. Internal flaps of breast tissue may be used to obliterate the surgical defect, especially if the pectoralis fascia is exposed. The subcutaneous tissue is closed, and the skin is reapproximated with a subcuticular suture. Incisions for lumpectomy and axillary dissection should be separate. Reconstruction is recommended any time that excision or reexcision of a lesion significantly affects the final shape and size of the breast.
3. **Complications of BCT** include infection and bleeding as well as the complications of axillary dissection. Other complications include specific side effects of radiotherapy to the breast, which include early skin changes such as breast edema, erythema, and moist desquamation. Late skin changes include edema, pigmentation changes, and telangiectasias. Radiation to the chest wall may result in interstitial pneumonitis, spontaneous rib fracture, breast fibrosis, pericarditis, pleural effusion, and chest wall myositis (rare). Patients with positive lymph nodes undergo radiation therapy to the axilla, which increases the risk of arm lymph edema to 15%.
4. **Follow-up for BCT** is similar to that for mastectomy. A posttreatment mammogram of the treated side is performed to establish a new baseline mammogram. Mammograms are then performed every 6–12 months after the completion of radiotherapy until the surgical changes stabilize, and then annually.

C. **Modified radical mastectomy** is the combination of a total mastectomy and axillary node dissection. It differs from the traditional Halstead radical mastectomy in that the pectoralis major muscle is preserved to enhance cosmesis of the chest wall. Complications include flap necrosis, bleeding, and infection, in addition to the complications of axillary dissection. Follow-up after mastectomy involves physical examination every 3–4 months for 2–3 years and every 6 months for the next 2–3 years. The chest wall should be examined for evidence of recurrence. Mammography of the contralateral breast should continue annually.

D. **Immediate reconstruction** at the time of mastectomy should be offered to eligible patients. Options include latissimus dorsi myocutaneous flaps, transverse rectus abdominis myocutaneous flaps, or inflatable tissue expanders followed by saline implants. A skin-sparing mastectomy may be performed, resulting in improved cosmesis. For this procedure, the nipple-areolar complex, a rim of periareolar breast skin, and the biopsy site are excised. Immediate reconstruction has been shown not to affect patient outcome adversely. The detection of recurrence is not delayed, and the onset of chemotherapy is not changed. Patients who undergo an immediate reconstruction after skin-sparing mastectomy often need additional outpatient procedures for nipple reconstruction or other contour adjustments.

IV. Locally advanced breast cancer (LABC) comprises T3 or T4, N1 or greater, and M0 patients (stages IIIA and IIIB).

A. **Patients with noninflammatory stage IIIB** (chest wall or skin involvement, skin satellites, ulceration, fixed axillary nodes) should receive induction chemotherapy (cyclophosphamide, 5-fluorouracil, and either doxorubicin or methotrexate) as the initial step in treatment, followed by surgery and radiation. The high response rates seen with this approach allow modified radical mastectomy to be carried out with primary skin closure and possible immediate reconstruction. Treatment comprises 3–4 cycles of neoadjuvant chemotherapy, followed by modified radical mastectomy with or without reconstruction, radiotherapy to the chest wall axilla and supraclavicular nodes, and further chemotherapy to complete a total of 6–12 cycles. Patients with stage IIIA disease receiving neoadjuvant chemotherapy can be converted to BCT candidates with no difference in overall outcome. Approximately 20% of patients with stage III disease present with distant metastases after appropriate staging has been performed.

B. **Inflammatory LABC (T4d).** Inflammatory breast represents 1–6% of all breast cancers. It is characterized by erythema, warmth, tenderness, and edema (*peau d'orange*). An underlying mass is present in 70% of cases. Associated axillary adenopathy occurs in 50% of cases. Delayed diagnosis is common owing to its similarity to mastitis. A breast biopsy that includes a portion of skin confirms the diagnosis. In two-thirds of cases, tumor emboli are seen in dermal lymphatics; 30% of patients have distant metastasis at the time of diagnosis. Inflammatory breast cancer requires aggressive multimodal therapy, as median survival is approximately 2 years, with a 5-year survival of only 5%.

C. **Staging in LABC.** Because of the frequent presence of distant metastasis at the time of presentation, all patients should undergo staging with complete blood

cell count, complete metabolic panel, bone scan, and CT scan of chest and abdomen before neoadjuvant chemotherapy.

D. **Follow-up.** Patients with LABC are at higher risk of local and distant recurrence and should be examined every 3 months by all specialists involved in their care.

V. Treatment of locoregional recurrence. All patients who present with locoregional recurrence should have a metastatic workup to exclude visceral or bony disease and should be evaluated for systemic chemotherapy or hormonal therapy.

A. **Recurrence in the breast after BCT** requires salvage mastectomy.

B. **Recurrence in the axilla.** Optimal control is obtained with surgical resection followed by radiation to the axilla and consideration of systemic therapy.

C. **Recurrence in the chest wall after mastectomy.** One-third of these patients have distant metastatic disease at the time of recurrence, and more than 50% will have distant disease within 2 years. Excision of the recurrence alone results in poor local control. Therefore, multimodal approaches are necessary. For an isolated local recurrence, excision followed by radiotherapy results in excellent local control. Rarely, patients require radical chest resection with myocutaneous flap closure.

VI. Adjuvant systemic therapy treatment is given when all gross tumor has been removed and no measurable tumor remains ([Table 33-4](#)).

Table 33-4. Adjuvant systemic treatment based on the St. Gallen Consensus Conference^a

A. All **node-positive patients** should receive adjuvant chemotherapy. This treatment is frequently followed with tamoxifen if the tumor is positive for estrogen receptor (ER). Chemotherapeutic regimens are comprised of four to eight cycles of a combination of cyclophosphamide/5-fluorouracil and methotrexate or doxorubicin (Adriamycin)/cyclophosphamide (Cytoxan) followed by a taxane. In patients with Her2/neu-positive tumors, a doxorubicin-based regimen is usually chosen. In postmenopausal women, chemotherapy is frequently used up to age 70. In older patients, chemotherapy is performed less frequently. In ER-positive tumors in postmenopausal women, tamoxifen is the drug of choice.

B. **Node-negative patients** may also benefit from adjuvant therapy with increased disease-free survival. Up to 30% of node-negative women die of breast cancer within 10 years if treated with surgery alone. Node-negative patients at high risk who benefit the most from adjuvant chemotherapy include those with larger tumors (>1 cm), higher nuclear grade, ER-negative tumors, and lymphovascular invasion. The NSABP B-20 trial and the International Breast Cancer Study Group trial IX have been performed to evaluate the effects of tamoxifen alone versus tamoxifen in combination with polychemotherapy in patients with ER-positive and ER-negative tumors. They demonstrate that polychemotherapy in combination with tamoxifen was superior to tamoxifen alone in improving disease-free and overall survival, especially in ER-negative patients regardless of tumor size. The NSABP B-23 trial looked at two different chemotherapy regimens with or without tamoxifen. Preliminary data showed no significant difference in overall survival between either chemotherapy regimen regardless of whether or not tamoxifen was given. The St. Gallen Consensus Panel (1998) recommends that patients with node-negative disease whose tumors are small (³¹1 cm) and ER-positive may be spared adjuvant chemotherapy but still may benefit from tamoxifen. Ultimately, the decision to use adjuvant therapy should involve an individualized discussion with the patient regarding the risks of recurrence without adjuvant therapy, the cost and toxicities of adjuvant therapy, and the expected benefit in risk reduction and survival from this therapy.

VII. Indications for postmastectomy radiation to the chest wall and regional node-bearing areas include T3 and T4 tumors, attachment to the pectoral fascia, positive surgical margins, involved internal mammary nodes, inadequate or no axillary dissection, more than eight positive lymph nodes, or residual tumor on the axillary vein. Randomized, prospective trials have shown a significantly decreased recurrence and improved survival in premenopausal women with these indications treated with chemotherapy and radiation therapy (*N Engl J Med* 337:949, 956, 1997).

VIII. Lymphedema occurs in approximately 10% of women undergoing axillary dissection, which was the rationale behind the development of SLNB. However, if axillary dissection occurs, the patient should avoid violations of the skin and be advised to avoid blood draws, blood pressure cuffs, and intravenous lines in her affected arm. Infections of the patient's hand or arm should be treated promptly and aggressively because infection can damage lymphatics further. Lymphedema presenting with simultaneous cellulitis should be treated aggressively with antibiotics and arm elevation. Lymphedema can become irreversible after repeated episodes of infection. If lymphedema is treated promptly while it is still reversible, the patient's arm may return to normal size. For persistent lymphedema, the most effective therapy is intense physiotherapy treatment. A graded pneumatic compression device has also been used to reduce arm swelling, followed by a professionally fitted compression sleeve. Good results have also been reported with professional massage therapy. With the increasing use of SLNB, this disabling complication may become a rarer event.

IX. Paget's disease of the nipple is characterized by eczematoid changes of the nipple, which may involve the surrounding areola. Paget's disease is almost always accompanied by an underlying malignancy, either invasive ductal carcinoma or DCIS. Burning, pruritus, and hypersensitivity may be prominent symptoms. Palpable masses are present in approximately 60% of patients. Mammography should be performed to identify other areas of involvement. If clinical suspicion is high, a pathologic diagnosis should be obtained by wedge biopsy of the nipple and underlying breast tissue. Treatment is mastectomy or BCT with excision of the nipple-areolar complex, followed by radiation therapy. The prognosis is related to tumor stage.

X. Breast cancer during pregnancy may be difficult to diagnose due to the low level of suspicion and breast nodularity and density. It carries an incidence of approximately 1 in 5,000 gestations and accounts for almost 3% of all breast cancers. Mammography and ultrasound may be helpful in characterizing masses. All dominant masses should undergo biopsy. Excisional biopsy can be safely performed under local anesthesia. Therapeutic decisions are influenced by the clinical cancer stage and the trimester of pregnancy and must be individualized. The standard preoperative staging workup is performed. Laboratory values such as alkaline phosphatase may be elevated during pregnancy. For advanced-stage disease, MR scan or ultrasound may be used in lieu of CT scans for staging. Although modified radical mastectomy is the standard treatment of a breast cancer diagnosed during pregnancy, BCT, radiation therapy, and adjuvant chemotherapy may also be applicable as long as the patient understands the risks and teratogenicity of these modalities (*Adv Surg* 34:275, 282, 2000). Chemotherapy may be given by the mid-second trimester, and radiation therapy may be given in the latter part of the third trimester.

XI. Breast cancer in men comprises fewer than 1% of male cancers and fewer than 1% of all breast cancers. BRCA2 mutations are associated with approximately 4–6% of these cancers. Patients generally present with a nontender hard mass. This contrasts with unilateral gynecomastia, which is usually firm, central, and tender. Mammography can be helpful in distinguishing gynecomastia from malignancy. Malignant lesions are more likely to be eccentric, with irregular margins, and are often associated with nipple retraction and microcalcification. Biopsy of suspicious lesions is essential. Modified radical mastectomy is the surgical procedure of choice. Eighty-five percent of malignancies are infiltrating ductal carcinoma and are positive for ERs. Adjuvant hormonal and chemotherapy treatment parallels that used in women. Overall survival per stage is comparable to that observed in women.

XII. Cystosarcoma phyllodes tumors account for 0.5–1.0% of breast cancers. Phyllodes tumor presents as a large, smooth, lobulated mass, and on physical examination, it may be difficult to distinguish from fibroadenoma. These tumors can occur in women of any age but most frequently present between ages 35 and 55. Skin ulcerations may occur secondary to pressure of the underlying mass. FNAB cannot reliably diagnose these tumors. Histologically, stromal overgrowth is the essential characteristic for differentiating phyllodes tumors from fibroadenomas. The biologic behavior of malignant tumors is similar to that of sarcomas. Treatment is wide local excision to tumor-free margins or total mastectomy. Axillary dissection is not indicated unless nodes are clinically positive (which is rare). Currently, there is no role for adjuvant radiation; however, tumors greater than 5 cm in diameter with evidence of stromal overgrowth may benefit from adjuvant chemotherapy with doxorubicin and ifosfamide (*Cancer* 89:1510, 2000). Patients should be followed with semiannual physical examinations and annual mammograms and chest radiographs if the tumor was malignant.

34 Otolaryngology: Head and Neck Surgery

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Otolaryngologic Disorders

I. Ear

A. Anatomy and physiology

1. The **external structures of the ear** include the auricle and external auditory canal. The auricle is composed of cartilaginous framework covered with skin. The lobule of the auricle is composed of skin and underlying fat. The lateral one-third of the external auditory canal consists of a cartilaginous framework covered with skin containing sebaceous and ceruminous glands. The medial two-thirds of the ear canal is composed of a thin layer of a glandular skin overlying bone.
2. The **tympanic membrane** is a thin, three-layered structure composed of a fibrous middle layer, an external epithelial layer, and an inner mucosal layer. The majority of the eardrum that is visible is rigid and is called the *pars tensa*. The small area superior to the head of the malleus is more flaccid and is called the *pars flaccida*.
3. The **middle ear**, or mesotympanum, is defined as the air space medial to the tympanic membrane and lateral to the otic capsule or inner ear. The meatus of the eustachian tube can be found in the anterior aspect of the middle ear or protympanum. The eustachian tube extends in an inferior-medial direction, opening into the nasopharynx. The function of the eustachian tube is threefold: protection of the middle ear from nasopharyngeal pathogens, aeration of the middle ear, and drainage of fluid from the middle ear space. The eustachian tube opens during swallowing by the action of the palatal muscles, mainly the tensor veli palatini. The posterior superior aspect of the middle ear space communicates with the mastoid cavity. Sound energy travels through the external auditory canal and is converted into mechanical energy, which is subsequently conducted via the tympanic membrane and ossicular chain to the oval window of the inner ear. The relative difference in the surface area of the tympanic membrane and oval window, and the lever action of the ossicular chain leads to an approximately 22-fold amplification of the sound energy that is conducted to the inner ear. From lateral to medial, the ossicular chain is composed of the malleus, incus, and stapes. The manubrium of the malleus is tightly attached to the tympanic membrane. The head of the malleus articulates with the body of the incus, which in turn articulates with the capitulum of the stapes via the long process of the incus. The footplate of the stapes then transmits this energy to the inner ear via the oval window.
4. The **otic capsule**, or inner ear, is a bony encasing that surrounds the sensory end organs of hearing and balance. The otic capsule can be divided into the pars superior and the pars inferior. The pars superior is composed of three semicircular canals and the utricle. The pars inferior is composed of the cochlea and sacculle. The cochlea is a snail-shaped structure containing $2\frac{1}{2}$ turns and contains the end organ of hearing, the organ of Corti. The three semicircular canals contain the end organs of balance and are responsible for spatial orientation. The utricle and sacculle are responsible for sensing motion or acceleration. The sensory end organs are bathed in endolymph and are surrounded by a membranous labyrinth. The membranous labyrinth is bathed with perilymph, which in turn is surrounded by the otic capsule. Mechanical energy transmitted to the inner ear is converted to bioelectrical energy by the sensory end organs within the endolymphatic compartment. This bioelectric energy is then transmitted via cranial nerve VIII, the vestibulocochlear nerve, to the central nervous system.
5. **Cranial nerve VII**, the facial nerve, is intimately related to the ear in its anatomic course through the temporal bone. The facial nerve exits the brainstem and enters the mesotympanum superiorly. It then runs in a posterior direction just superior to the stapes superstructure. The nerve then turns inferiorly, traversing the mastoid cavity, finally exiting the temporal bone via the stylomastoid foramen. Before exiting the temporal bone, the facial nerve gives off a motor branch to the stapedius muscle and the chorda tympani nerve, which supplies taste sensation to the anterior two-thirds of the tongue.

B. Hearing loss is generally classified as neurosensory, conductive, or mixed.

1. **Neurosensory hearing loss** is caused by lesions arising in the cochlea, cranial nerve VIII, or the central nervous system. Therefore, sound energy is transmitted to the inner ear, but bioelectrical impulses either are not generated or are not transmitted and processed. Presbycusis is a type of neurosensory hearing loss that occurs as a natural part of aging and is usually limited to hearing loss in the higher frequencies. Other causes of neurosensory hearing loss include excessive noise exposure, use of ototoxic medications, temporal bone trauma, otitis media, and tumors of cranial nerve VIII such as acoustic neuromas. Congenital deafness should be suspected with histories of congenital anomalies, maternal infections, or multiple family members with hearing loss. Neurosensory hearing loss is generally treated with sound amplification devices. Cochlear implants may be considered for the profoundly deaf.
2. **Conductive hearing loss** is caused by a malfunction in the transmittal of sound energy to the inner ear. This malfunction can arise from a defect involving the external ear canal, tympanic membrane, or middle ear ossicles. Common causes include impacted cerumen, otitis media, tympanic membrane perforation, destruction of the ossicular chain by infection, or fixation of the ossicular chain. Treatment of conductive hearing loss usually involves reestablishing the sound conduction pathway.
3. **Tinnitus** is a subjective sound perceived by the patient. It is usually described as a hissing, cricket chirp, or ringing noise. Tinnitus usually accompanies high-frequency nerve hearing loss and is an expected associated symptom of presbycusis. It can occur with other ear disorders.
4. **Bedside diagnostic or qualitative hearing tests** include the Weber's and Rinne tests. Weber's test consists of placing a vibrating 512-Hz fork on the patient's forehead. A normal test is to perceive the sound midline. In an abnormal test, the sound lateralizes to one ear or the other. The sound lateralizes to an ear with a conductive hearing loss or contralateral to an ear with a neurosensory hearing loss. The Rinne test is performed by determining which is louder, the sound from a vibrating 512-Hz fork held 6–8 inches from the ear (air conduction) or from the fork held on the mastoid process behind the ear (bone or cochlea nerve conduction). Air conduction is louder than bone conduction with normal hearing and in neurosensory hearing loss. On the other hand, with conductive hearing loss, bone conduction is louder than air conduction.
5. **Audiometric hearing tests** are quantitative measures of hearing. These tests measure the ability of a person to hear pure tones ranging from 250 to 8,000 Hz presented at increasing intensities, measured in decibels. Both air conduction and bone conduction (cochlea nerve) are tested. A gap between the air and bone conduction levels indicates a conductive hearing loss. Speech and word recognition, part of the audiometric testing, addresses the ability of the patient to hear and process pure tones. Auditory brainstem response measures action potentials generated in the neural circuitry connecting the inner ear to the central nervous system in response to a sound presented to the external ear. This test does not require patient cooperation and is therefore useful in testing infants.
6. **Tympanometry**, also a part of audiometric testing, measures the mobility of the tympanic membrane and ossicular chain. Middle ear effusions, ossicular chain discontinuity or fixation, tympanic membrane perforations, and eustachian tube dysfunction can affect the mobility of the tympanic membrane and ossicular chain.
7. **Vestibular testing**, or evaluation of balance function, is complex. The test most commonly used to evaluate the integrity of the semicircular canal is called **electronystagmography**. This test consists of applying cold or warm water to the external ear canal, which in turn stimulates the horizontal semicircular canal and produces nystagmus. The rate of nystagmus can be measured and used to judge the functioning of this semicircular canal. Other vestibular tests include the rotary chair and platform testing.

C. Inflammatory ear disease

1. **External otitis** is an inflammation of the external auditory canal. It is usually produced by chronic moisture in the canal. This leads to changes in local pH and bacterial overgrowth and invasion, with resultant injection and swelling of the canal. The bacterium most commonly associated with otitis externa is *Pseudomonas aeruginosa*. The canal may be obstructed and sensitive to manipulation. It occurs frequently in swimmers, therefore being commonly called "swimmer's ear." Treatment consists of antibiotic-steroid eardrops, avoiding moisture in the ear canal, and placement of a canal wick if the ear canal is obstructed by soft-tissue edema. The wick helps spread the administered eardrops medially throughout the ear canal. Chronic external otitis is usually produced by excessive abuse of the ear canal, frequently with cotton-tipped applicators. This produces itching of the ear canal, leading to more ear canal abuse, thus prolonging the problem. Treatment consists of steroid drops and avoidance of abusive ear habits. Fungal external otitis or otomycosis can present as an acute or chronic form of otitis externa. Otomycosis is commonly found in patients who live in humid climates or who have underlying chronic disease processes, such as diabetes. Treatment consists of vinegar-steroid eardrops. *Malignant external otitis* refers to an aggressive infection of the

external ear canal, usually occurring in diabetic or immunocompromised patients. This can be a fatal disease and requires aggressive therapy, including systemic antibiotics and surgical débridement.

2. **Eustachian tube dysfunction** is most frequent in young children and occurs when the tube does not consistently ventilate the middle ear space. The resulting negative pressure in the space produces a feeling of fullness in the ear. The negative pressure may pull the eardrum medially and produce retraction pockets. Prolonged tube dysfunction may result in weakening and thinning of the eardrum, which interferes with vibratory quality, thereby diminishing hearing ability.
3. **Chronic tubal dysfunction** may lead to serous otitis media, which is fluid in the middle ear space. Serous otitis media appears on physical examination as an amber fluid behind the eardrum. The drum is often retracted and air bubbles can be seen in the serous fluid behind the tympanic membrane. Tympanometry reveals decreased eardrum mobility. Eustachian tube dysfunction is treated with antibiotics (if infection is present), insufflation exercises, and myringotomy with tube placement if medical therapy fails. Tube placement through the eardrum creates a perforation of the eardrum, allowing ventilation of the middle ear space. The tube remains for 6–12 months, at which point it is extruded and the eardrum spontaneously heals. Eustachian tube dysfunction in young children frequently resolves with time. Placement of myringotomy tubes decreases the frequency of infections and maintains middle ear ventilation until time and nature resolve the eustachian tube problem.
4. **Acute otitis media** is predominantly a disease of young children (younger than 5 years old). The major etiologic factor is eustachian tube dysfunction. The most common bacterial pathogens are *Streptococcus pneumoniae*, *Homophiles influenzae*, and *Branhamella catarrhalis*. Symptoms range from none to fever with severe otalgia, decreased appetite, and irritability. Diagnosis is made by physical examination. The tympanic membrane appears erythematous, dull, and bulging, with a loss of normal anatomic landmarks. White, purulent material may be noted behind the tympanic membrane. Pneumotoscopy reveals decreased mobility of the tympanic membrane.
 - a. **Treatment** consists of an appropriate oral antibiotic. If the patient has had another recent bout of otitis media, then a beta-lactamase–stable antibiotic should be given. The usual course of treatment is 10 days. Symptom resolution should be seen in 24–48 hours. If symptoms progress, it is critical that the patient be reevaluated, complications of otitis media ruled out, and a different antibiotic initiated. Treatment failures are usually secondary to presence of antibiotic-resistant strains of bacteria. In cases of recurrent otitis media, it is usually necessary to perform a myringotomy with tube placement to overcome the eustachian tube dysfunction. Antibiotic prophylaxis has not been shown to be efficacious.
 - b. **Complications** of otitis media include eardrum perforation, meningitis, brain abscess, mastoiditis, labyrinthitis, sigmoid sinus thrombophlebitis, and facial nerve paralysis.
5. **Chronic suppurative otitis media** describes infection of the middle ear space associated with a persistent eardrum perforation. Therefore, one of the main symptoms is a chronic draining ear. The infections may recur intermittently or be persistent. Some cases may be managed medically, but most require surgery to remove the infection and close the perforation.
6. A **cholesteatoma** consists of skin debris entrapped medial to the eardrum. There are three types of cholesteatomas. Primary cholesteatomas occur superiorly in the pars flaccida of the eardrum. Secondary cholesteatomas occur in the pars tensa of the eardrum, through a perforation in the tympanic membrane. A congenital cholesteatoma is caused by a rest of keratinocytes anywhere medial to the tympanic membrane. The skin debris medial to the tympanic membrane can expand and erode adjacent cartilaginous and bony structures. This could lead to destruction of the ossicular chain (causing hearing loss), otic capsule (leading to a perilymphatic fistula, hearing loss, and/or vertigo), Fallopian canal (leading to facial nerve dehiscence and even paralysis), and the bone separating the middle ear from the brain (leading to meningitis). Treatment consists of complete and meticulous removal of the skin debris from the middle ear and mastoid space.
- D. **Ear trauma** may occur from a blunt blow to the external ear resulting in a hematoma of the auricle. Auricular hematomas should be drained immediately to prevent cartilage destruction and excessive scarring (cauliflower ear). Blunt or sharp blows may result in perforation of the eardrum. Usually, these heal with conservative management. If the perforation persists, surgical repair maybe necessary. With severe blunt trauma to the head, fractures of the temporal bone can arise, leading to hearing loss and even facial nerve paralysis.
- E. **Foreign bodies** of the external canal are sometimes found in children and adults. Care should be used to avoid trauma to the eardrum and ear canal during removal. Dried beans in the canal absorb moisture, enlarge, and become tightly wedged, making removal difficult.
- F. **Vertigo** is a sensation of turning or spinning in which the patient experiences a distinct sense of motion in relation to the environment. This should be differentiated from dizziness, which is a feeling of faintness. True vertigo usually indicates a disorder of the inner ear or the part of the central nervous system involved in processing the signals from the inner ear. Attacks of vertigo are usually severe and paroxysmal, lasting from minutes to days. In 80% of patients with vertigo, the cause is peripheral or related to the inner ear. Central vertigo is mild and more like a sensation of unsteadiness. It is vague, with no specific onset or termination.
 1. **Ménière's disease** presents a triad of symptoms of episodic vertigo, fluctuating neurosensorial hearing loss, and tinnitus. The cause is either overproduction or underabsorption of endolymph of the inner ear. Treatment consists of sodium restriction, diuretics, and vestibular suppressants. In refractory cases, surgical intervention is necessary.
 2. **Benign positional vertigo** is a common disorder characterized by transient vertigo precipitated by specific head movements. This may occur posttraumatically, after an episode of viral labyrinthitis, or spontaneously. This disease is thought to be caused by loose debris, which stimulates the sensory nerve endings within the semicircular canal. In specific cases the loose debris can be repositioned, using the Epley maneuver, rendering the particles innocuous. Refractory cases require surgical intervention.
- G. **Facial nerve paralysis.** Central paralysis from a supranuclear lesion (lesions proximal to the facial nucleus) spares the ipsilateral forehead because this area receives bilateral cortical innervation. Peripheral lesions (lesions of the facial nucleus or nerve) produce paralysis of the whole face or selected branches, depending on the site of the lesion. Bell's palsy is an idiopathic facial nerve paralysis. Suggested etiologies are viral infections, autoimmune disease, or vascular compromise, although none of these causes have been clearly demonstrated. Bell's palsy accounts for 80% of acute facial nerve paralysis. Treatment is systemic steroids. Most cases recover within 3 months. Other causes for facial paralysis include tumors of the ear, temporal bone or parotid gland, trauma, and inflammatory disorders.

II. Nose and sinus disorders

A. Anatomy and physiology

1. The anterior entrance to the nose is the anterior choana. The skin-lined nasal vestibule leads from the entrance into the nasal cavity. The nose exits posteriorly through the posterior choana into the nasopharynx. The medial wall of the nasal cavity is the nasal septum, which is composed of cartilage anteriorly and bone posteriorly. A small plexus of blood vessels (Kiesselbach's) in the mucosa of the anterior septum bilaterally is a common site for epistaxis. Along the lateral nasal wall are three scroll-like structures called *turbinates*: inferior, medial, and superior. The inferior turbinate has blood-filled spaces within the mucosal lining, which may become engorged, producing nasal airway obstruction. Just lateral to each turbinate is an air passage, or meatus. The olfactory nerve terminates as it penetrates the cribriform plate and is distributed in a small area in the most superior aspect of the nose. The nose is lined with a ciliated epithelium with a submucosa rich in mucous glands; nerves; blood vessels; and inflammatory cells, such as mast cells, basophils, and eosinophils. The submucosa is very responsive to a variety of stimuli, which can result in congestion, sneezing, increased mucus production, and inflammatory changes.
 2. **Sinuses**
 - a. The **paranasal sinuses** are mucosa-lined, paired, bony cavities in the forehead, cheeks, nose, and central skull (the frontal, maxillary, ethmoid, and sphenoid sinuses, respectively). The mucus produced in the sinuses circulates toward the ostia of each sinus by mucociliary flow.
 - b. The **maxillary, frontal, and anterior ethmoid sinuses** drain into the nose via the middle meatus, the air space just lateral to the middle turbinate.
 - c. The **posterior ethmoid sinuses** drain into the superior meatus, the air space just lateral to the superior turbinate.
 - d. The **sphenoid sinus** drains into the sphenoethmoidal recess, an area posterosuperior in the nasal cavity. The mucus in the nose is directed posteriorly along the lateral nasal wall, over the eustachian tube orifice, and finally into the nasopharynx.
 3. The **nose and sinus areas** have multiple functions. The nose provides a conduit for the passage, warming, and humidification of air before it enters the lower respiratory tract. The mucus-covered nasal walls clean the incoming air by trapping particles on the mucosal surface. The nose provides olfactory function via the olfactory nerve. The sinuses provide resonance to the voice, decrease the weight of the skull, and allow cushioning to the cranial vault in blunt head trauma. The **normal physiology** of the nose involves the nasal cycle, an alternate pattern of congestion and decongestion. This normal pattern of congestion occurs every 2–6 hours, in which one side is congested and the opposite side is decongested, followed in the next 2–6 hours by the opposite pattern. The nose also normally congests on the dependent side when lying supine. **Common symptoms of nasal-sinus disorders** are nasal congestion, nasal obstruction, excessive nasal secretions, postnasal drip, facial pain and pressure, eustachian tube dysfunction, and epistaxis.
- B. **Diagnostic tests.** Most nasal-sinus disorders are diagnosed by a thorough history and physical examination, which includes endoscopic intranasal visualization. Sinus X-rays and computed tomographic (CT) scans are helpful for acute and chronic sinusitis to delineate the sinuses involved and the extent of disease. Nasal mucus smears may be used to help distinguish an allergic inflammation (characterized by eosinophils) from a bacterial or viral infection (characterized by neutrophils and lymphocytes). Skin or radioallergosorbent tests confirm the diagnosis of allergy in allergic rhinitis.
- C. **Congenital disorders**
1. **Midline nasal masses** in young patients may be an encephalocele, a glioma, or a dermoid cyst. These may present as an external or intranasal mass. Manipulation of these masses should be avoided until the question of intracranial extension is ascertained.

2. **Posterior nasal choanal atresia** may be unilateral, bilateral, membranous, or bony. Bilateral choanal atresia is an airway emergency in newborns because they are obligate nasal breathers. Inability to pass a catheter through the nose confirms the diagnosis. A nipple taped to the mouth secures an oral airway.
- D. **Inflammatory disorders.** Disorders of the nose and sinus area are divided into nasal disorders and sinus disorders for purposes of discussion, understanding of symptoms, diagnosis, and treatment. In many cases, the whole nasal-sinus area is involved with the same disease process, and a more accurate, descriptive term is *rhinosinusitis*. In the following discussion, this term can apply to many disease states.
1. **Allergic rhinitis** is an inflammation produced by an allergic reaction, usually to an inhalant, such as ragweed pollen. In susceptible patients, inhaled pollen (allergen, antigen) stimulates a sensitive mucosa via immunoglobulin E to release mediators (histamine), which produce vasodilation and glandular secretion. This process results in nasal itching, sneezing, nasal congestion, and excessive nasal secretion. Seasonal allergic rhinitis usually occurs in the spring and fall seasons. In the spring, the common stimulants are trees and grasses. Weeds are the usual stimulants in the fall. Perennial allergic rhinitis is produced by nonseasonal inhalants, such as mold, house dust, and animal dander. **Diagnosis** is made by history and confirmed by allergy skin tests or radioallergosorbent tests. **Treatment** consists of environmental control, medications, and allergy immunotherapy. Environmental control involves avoidance measures and air filters. Medications include systemic and intranasal antihistamines, nasal steroids, cromolyn nasal spray, decongestants, and systemic steroids. Immunotherapy consists of injection of allergens to decrease the intensity of the allergy reaction (desensitization).
 2. **Nonallergic rhinitis.** This category includes other diseases that produce some of the same symptoms of congestion, excessive nasal secretions, and postnasal drip. In contrast however, the etiologic agent is not an allergen.
 - a. **Viral rhinitis** is the typical cold and the etiologic agent is one of many viruses. Viral rhinitis typically occurs in the winter and lasts 5–10 days. Treatment is supportive, including humidification, rest, and ingestion of liquids. Antibiotics are not indicated unless a secondary bacterial infection of the upper respiratory tract occurs.
 - b. **Rhinitis medicamentosa** is produced by prolonged use of decongestant nasal sprays. As the decongestant effect dissipates, rebound congestion occurs, which encourages use of more decongestant spray. Treatment consists of discontinuing usage of the spray.
 - c. **Drug-induced rhinitis** is produced by systemic drugs, including alcohol, antithyroid drugs, aspirin, estrogen, iodides, and reserpine. Topical drugs that may irritate the nose include cocaine, tobacco, and marijuana.
 - d. **Vasomotor rhinitis** is characterized by nasal congestion and watery secretions. The exact cause is unknown but may be secondary to autonomic dysfunction in the nasal membranes. The nasal mucosa remains in a chronically stimulated state and may be further stimulated by nonspecific stimulants, such as weather changes, stress, and chemical irritants. Treatment consists of antihistamines, decongestants, and nasal steroid sprays. Surgery to reduce the bulk of the turbinates may be indicated in refractory cases.
 - e. **Atrophic rhinitis** is characterized by atrophy of nasal membranes. It usually occurs in elderly patients but may occur in younger patients for unknown reasons. This disease results in malodorous crusts in the nasal cavity. Therapy is removal of the crusts and frequent cleaning of the affected area with saline solutions.
 - f. **Metabolic-endocrine rhinitis** most commonly occurs in pregnancy, parts of the menstrual cycle, hypothyroidism, and diabetes mellitus. Findings are congested, edematous mucosa. In the case of pregnancy, the nasal congestion usually dissipates after giving birth.
 - g. **Wegener's granulomatosis**, an autoimmune vasculitis, produces necrotizing lesions of the upper respiratory tract. In the nose, this disease causes an erythematous, thickened, and ulcerated mucosa, which may bleed frequently. Additionally, there may be ulceration and perforation of the nasal septum.
 3. **Nasal foreign body** is usually found in children or a mentally disturbed patient and is characterized by a unilateral foul-smelling nasal discharge. Topical decongestants and anesthetics should be used before removal. General anesthesia may be necessary in some cases.
- E. **Structural problems.** A **deviated nasal septum** may occur naturally as the nose grows because of variations in septal growth centers or may arise posttraumatically from a nasal fracture or a blunt nasal injury, which stimulates uneven septal growth. A significantly deviated septum can produce nasal obstruction or interfere with drainage from the sinuses, leading to sinusitis. Headaches may be produced by the deviated septum contacting the lateral nasal wall and causing a local inflammation. Treatment for a deviated nasal septum is surgical, involving the submucosal removal of the deviated septum.
- F. **Soft-tissue problems**
1. **Adenoid hypertrophy.** Although, strictly speaking, the adenoids are not in the nose but in the nasopharynx, they produce symptoms of nasal obstruction if they are enlarged and should be considered in the differential diagnosis of nasal obstruction, especially in children. When they obstruct the posterior choanae of the nose, they may produce mouth breathing, snoring, and recurrent upper respiratory tract infections. When the adenoids obstruct the eustachian tube, recurrent ear infections are common. If the resultant problems and hypertrophy are significant, removal is accomplished by an adenoidectomy. The adenoid pad usually atrophies with age; therefore, it is unusual to find enlarged adenoids in adults.
 2. **Nasal polyps** are pendulous-appearing collections of edematous, hyperplastic mucosa, found most commonly in the area of the middle turbinate bilaterally. The etiology of nasal polyps is unknown but is thought to be caused by a chronic allergic reaction. Therefore, they are most commonly present in patients with allergic rhinitis and recurrent sinusitis. Patients are often found who have a triad of nasal polyps, asthma, and aspirin sensitivity. Nasal polyps are also commonly found in patients with cystic fibrosis. In addition to causing nasal obstruction, nasal polyps may obstruct the ostia of the sinuses, leading to recurrent sinusitis. Treatment involves topical steroid sprays, treatment of associated infections, systemic steroids, and in persistent symptomatic cases, surgical removal of the nasal polyps via endoscopic surgery. An antral-choanal polyp is unilateral and originates in the maxillary sinus, protrudes into the nasal airway, and presents as a bulky nasal mass within the nose or nasopharynx. Treatment is removal of the polyp from the nose and the maxillary sinus. Because nasal polyp disease is commonly bilateral, unilateral presentations should therefore prompt a workup for nasopharyngeal tumors.
- G. **Neoplasms** of the nose and sinus area are uncommon. Papillomas are benign wartlike growths on the septum or lateral nasal wall. Inverting papillomas occur in the middle turbinate area and are locally invasive; wide resection is necessary to prevent recurrence. Malignancy is present in 10–20% of these cases. Juvenile nasopharyngeal angiofibroma occurs in adolescent boys and usually present as recurrent epistaxis. Treatment is embolization of the vascular supply followed by resection. Malignant lesions of the nasal-sinus area include squamous cell carcinoma, adenocystic carcinoma, melanoma, and sarcoma.
- H. **Sinus disease**
1. **Acute sinusitis** is an infection of the sinus cavity. It is usually preceded by an upper respiratory tract infection, which produces obstruction of the sinus ostium, providing a rich culture media in the sinus and leading to a bacterial superinfection. Other causes of sinus ostia blockage include allergic rhinitis, polyps, and structural problems. Immune deficiencies, contamination from swimming water, ciliary dysfunction, dental infections, and atmospheric pressure trauma may also be contributing factors.
 - a. **Symptoms** include facial pain and pressure sensation over the involved sinus. The nose may be congested and there may be a mucopurulent nasal discharge or postnasal drip. Systemic symptoms include malaise and fever.
 - b. The **diagnosis** is based on clinical suspicion and may be confirmed by sinus X-rays, which may reveal mucosal thickening and opacification in the involved sinus. Sinus X-rays are not necessary for the diagnosis. The most common bacteria are *S. pneumoniae*, *Branhamella catarrhalis*, and *Haemophilus influenzae*.
 - c. **Treatment** consists of antibiotics, decongestants, mucolytic agents, and humidification. Most cases of acute sinusitis resolve with treatment within 2–3 weeks. Recurrent acute sinusitis is recurrent infections that occur after complete resolution of a previous bout of sinusitis. Some patients may have up to two to four episodes of sinusitis per year and may require more protracted courses of treatment.
 2. **Chronic sinusitis** is an infection of the sinus cavity that recurs frequently (i.e., more than four episodes per year) or an infection that persists with permanent mucosal changes, frequently demonstrated by CT scan. Some acute episodes resolve, but mucosal changes persist, leading to a predilection for additional infections. Contributing factors may be an initial infection that failed to completely resolve or polyps, inhalant allergies, ciliary dysfunction, immune deficiencies, or structural blockage in the ostial area. **Treatment** consists of longer courses of antibiotics, decongestants, nasal and systemic steroids, mucolytic agents, and nasal saline lavages. CT scan frequently reveals mucosal swelling with blockage of the sinus ostia. Endoscopic sinus surgery may be indicated to reestablish the patency of the sinus ostia and remove blocking mucosa or bony structures.
 3. **Fungal sinusitis** may occur as a secondary infection in chronic sinusitis. The fungus usually resolves as the chronic infection resolves and the sinus is re-aerated. Active fungal infection is most commonly found in debilitated patients, such as those with uncontrolled diabetes mellitus and immunocompromised patients. Débridement, sinus aeration, and antifungal therapy may be necessary.
 4. **Complications of sinusitis.** Osteomyelitis of the involved bone, such as the frontal bone in frontal sinusitis, rarely occurs. Treatment is drainage of the involved sinus and high doses of antibiotics. Orbital cellulitis and abscess may result from ethmoiditis as the infection penetrates the small holes of the medial orbital wall. This requires drainage of the ethmoid sinuses and antibiotics. Central nervous system complications are meningitis, epidural or brain abscess, and cavernous sinus thrombosis. Rare systemic complications may involve infection or abscesses in other parts of the body.
- I. **Nasal-sinus trauma**
1. **Nasal fracture.** Blunt trauma may result in fractured nasal bones. Nondisplaced fractures may produce swelling but no cosmetic change. No therapeutic intervention is required in this situation. Displaced nasal fractures produce cosmetic changes and obstructive functional problems, requiring reduction of the nasal fractures. This can usually be accomplished by closed internal and external nasal manipulation combined with nasal packing. Severe displacement with multiple fragments may require an open reduction.
 2. **Septal hematoma** may occur with any nasal trauma. It is a collection of blood between the mucoperichondrium and cartilage of the nasal septum, usually producing severe pain. The hematoma may destroy the blood supply to the septal cartilage, resulting in a septal perforation. Septal hematomas mandate intranasal drainage.
 3. **Maxillary and midface fractures** are usually classified according to the Le Fort system ([Fig. 34-1](#)). Le Fort I fracture involves the lower midface: The fracture line runs through the lower maxilla and into the nasal cavity. The resulting loose segment involves teeth, the lower part of the maxillary sinus, and the hard

palate. Le Fort II fractures pass through the nasal and lacrimal bones, the floor of the orbit, and the inferior orbital rim and across the upper part of the maxillary sinus. Fractures of the cribriform plate may occur. Le Fort III (craniofacial dissociation) severs the facial bones from their cranial attachments. The fracture line extends across the orbits, the lateral wall of the orbit, and the fronto- and zygomaticomaxillary suture lines. Associated intracranial injuries frequently occur. In all these fractures, treatment involves realigning bony fragments, reestablishing normal occlusion, open reduction, and stabilization of the fractures. All three of the Le Fort fractures can lead to an acute compromise of the upper airway; therefore, in these patients it is important to immediately assess the airway and obtain control if necessary. Airway control is obtained by either intubation or tracheostomy.

Fig. 34-1. The Le Fort maxillary fracture classification. A Le Fort I fracture is a horizontal fracture separating the maxillary alveolus from the upper midface skeleton. The Le Fort II or pyramidal fracture separates a central, pyramid-shaped nasomaxillary segment from the upper midface skeleton. The Le Fort III fracture is a craniofacial dysjunction in which the midface is fractured through the upper portion of the orbits. Generally, Le Fort II and III fractures do not exist as single segments but display comminution. (From P Mason. Facial fractures. In: JW Smith, SJ Aston, eds. *Plastic surgery*, 4th ed. Boston: Little, Brown, 1991, with permission.)

III. Salivary gland

A. **Anatomy and physiology.** Major salivary glands include the parotid, submandibular, and sublingual glands.

1. The **parotid gland**, the largest salivary gland, lies just anterior to the external ear and mastoid process. It rests on the surface of the masseter muscle and ascending ramus of the mandible. The tail of the parotid lies just inferior to the angle of the mandible, and a deep lobe extends medial and posterior to the mandible. The parotid is intimately related to the facial nerve. The facial nerve exits the stylomastoid foramen and courses anteriorly, entering the substance of the parotid, where it splits into two major trunks that then further subdivide into five secondary branches. As the nerve passes through the parotid it divides the gland, for clinical purposes, into a superficial lobe and a deep lobe. The parotid duct (Stensen's duct), formed by the confluence of smaller ducts, exits the anterior portion of the gland. It crosses the masseter muscle, turning deep over the anterior border of the muscle, to pierce the buccinator. It enters the oral cavity through a small papilla opposite the second upper molar.
2. The **submandibular (submaxillary) gland** lies just inferior to the mandible in the digastric triangle of the upper neck. The posterior portion extends posteriorly toward the tail of the parotid gland. The anterior portion extends medially and superiorly in close contact with the floor of the mouth. The submandibular duct (Wharton's duct) exits the anterosuperior part of the gland to enter the floor of mouth just lateral to the frenulum of the tongue.
3. The **sublingual gland** lies in intimate contact with the submandibular duct. Its short, multiple ducts enter directly into the floor of the mouth along the course of the submandibular duct.
4. The **paired parotid, submandibular, and sublingual glands** supply most of the saliva, approximately 1,000–1,500 mL in 24 hours. The minor salivary glands are located submucosally in the palate, tongue, and oral mucosa. Saliva provides lubrication for the oral cavity and the esophagus, inhibits growth of bacteria, and contains the enzyme ptyalin, which initiates the digestion of starch. Saliva consists mainly of water but does contain calcium and phosphate salts, which may produce salivary calculi. The submandibular and sublingual glands are mixed glands in that they contain both mucous and serous glandular elements. The parotid gland consists mainly of serous glands.

B. **Diagnostic tests.** Radiographic studies may be used to confirm a diagnosis or to delineate the extent of disease. Contrast sialography (placing contrast material in the ductal system of the parotid or submandibular gland) outlines the ductal system and may locate a ductal stricture, calculus, or sialiectasia. It may be performed with conventional X-ray or CT scan. Needle biopsy may be performed on glandular masses. Incisional biopsies are rarely performed.

C. **Congenital disorders.** Ranulae are fluid-filled fluctuant masses in the floor of the mouth, usually just lateral to the frenulum of the tongue and in relationship to the submandibular duct. Ranulae appear to be retention cysts. They may require excision or marsupialization.

D. **Inflammation**

1. **Acute sialadenitis.** Acute inflammation of the salivary glands usually involves the parotid gland in adults. It usually affects adults and is of bacterial origin, with *Staphylococcus* being the most common etiologic agent. Historically, it occurred most commonly in postoperative patients who were chronically dehydrated. Currently, acute sialadenitis usually occurs in elderly or debilitated patients, such as those receiving immunosuppressive treatment (e.g., chemotherapy or radiation therapy). The involved gland is tender. Purulent exudate may be expressed from the duct by massage of the gland. Treatment includes hydration and high doses of appropriate antibiotics. Surgical therapy may be necessary to drain an abscess. Abscesses are usually not obvious because the multiple septa in the parotid gland prevent formation of a large abscess, and the diagnosis of an abscess is usually based on the lack of clinical response to medical treatment. To drain a parotid abscess, a skin flap is elevated over the gland; multiple perforations are created in the gland, taking care not to injure the facial nerve.
2. **Chronic sialadenitis** has decreased since the advent of antibiotics. It is characterized by recurrent or persistent symptoms over a long period. Stones or other obstructive problems in the ducts may lead to chronic inflammation and resultant fibrosis of the gland and the ducts. Symptoms may require repeated treatment with antibiotics. Excision of the gland may be required for repeated bouts that interfere with the patient's daily life.
3. **Mumps.** This viral disease usually occurs in young childhood and most commonly involves the parotid gland. It produces swelling and tenderness of the gland that develops acutely and resolves with supportive measures. The incubation period is 2–3 weeks, and one episode usually produces lifetime immunity.

E. **Obstructive salivary gland disease.** The most frequent cause of salivary gland obstruction is ductal calculi (sialolithiasis). Sudden swelling and pain of the involved gland, usually associated with eating, characterize obstructive disease. These stones occur most commonly in the submandibular duct. Most are radiopaque and many can be palpated in the floor of the mouth within the duct. The calculus may be diagnosed by probing the ductal orifice with a lacrimal probe. A stone closer to the gland may not be palpable. Treatment consists of incising the duct and removing the stone. Recurrent episodes may produce chronic inflammation of the gland, and excision of the gland may ultimately be required.

F. **Systemic disease.** Parotid enlargement may occur with starvation, bulimia, cirrhosis, hypothyroidism, menopause, and Cushing's disease. Enlargement may occur with ingestion of iodides, lead, copper, and phenothiazines.

G. **Trauma.** Injury to the parotid gland area potentially involves damage to the facial nerve, parotid duct, and glandular parenchyma. Facial nerve function should be assessed. Loss of nerve function requires exploration and repair of the nerve. Injury to the parotid duct requires repair of the duct over a stent. Injury to the glandular tissue usually heals spontaneously with pressure dressings. Persistent salivary fistulas may require parotidectomy.

H. **Tumors.** A discrete, unilateral salivary gland mass may be a primary tumor, but other diagnoses should be considered. Enlarged lymph nodes may be close to the submandibular gland, and parotid lymph nodes may be extraglandular or intraglandular. In the parotid area, enlargement of the masseter muscle and localized mandible hypertrophy may be confused with a parotid mass.

1. **Benign tumors.** Approximately 75% of parotid tumors, 50% of submandibular tumors, and 25% of minor salivary gland tumors are benign. Pleomorphic adenoma is the most common of all salivary gland tumors. These benign tumors grow slowly, are painless, and do not involve the facial nerve. Pseudopod extension of the tumor requires a parotidectomy to prevent recurrence. Lymphoepithelial cysts are frequently seen in the human immunodeficiency virus–positive patient and tend to present as bilateral parotid masses.
2. **Malignant tumors.** Approximately 25% of parotid tumors, 50% of submandibular tumors, and 75% of minor salivary gland tumors are malignant.
 - a. **Mucoepidermoid carcinoma** most commonly involves the parotid gland. They are classified as low-grade (well-differentiated) or high-grade (poorly differentiated). Low-grade tumors are usually treated with parotidectomy and facial nerve preservation, whereas high-grade tumors may be treated more aggressively (e.g., with facial nerve resection and removal of regional lymph nodes).
 - b. **Adenoid cystic carcinoma** has a propensity for distant metastases, frequently to the lungs. The tumor can also extend along nerves in the involved area, but it usually does not metastasize to the regional lymph nodes. Treatment is resection. This tumor is considered radiosensitive but not radiocurable.
 - c. **Other malignant tumors** are adenocarcinoma, malignant-mixed carcinoma, squamous cell carcinoma, and acinic cell carcinoma. Parotid lymph nodes may be involved with metastatic disease from other regional malignancies, such as malignant melanomas from the scalp. Lymphomas may also involve the parotid nodes.

IV. Larynx

A. **Anatomy and physiology**

1. The larynx is divided into the **supraglottis, glottis, and subglottis**. The epiglottis, arytenoids, and false vocal folds (cords) comprise the supraglottis. The ventricle is a space between the false and true vocal folds, dividing the supraglottis from the glottis. The subglottis is divided as the area 1 cm below the level of the vocal cords.

2. The **rigid support of the larynx** is from the thyroid, cricoid, and hyoid cartilages. The epiglottic cartilage helps prevent aspiration by covering the glottis. The arytenoid cartilages are essential for sound generation and airway protection. The cricoid is the only complete circular rigid support of the airway.
 3. The superior **laryngeal nerve** provides sensory innervation superior to the glottis and motor innervation to the cricothyroid muscle. The recurrent laryngeal nerve is sensory to the glottis and subglottis and innervates the intrinsic laryngeal musculature, including the vocalis muscle.
- B. Congenital**
1. **Laryngomalacia** is the most common congenital abnormality of the larynx. The exact cause of laryngomalacia is unknown; however, it is defined as inspiratory collapse of the supraglottis. This results in inspiratory stridor, feeding difficulties, and respiratory distress. Onset occurs at or near birth and usually resolves with time. Surgical treatment is reserved for severe cases.
 2. Newborns with **stridor** may have unilateral or bilateral vocal cord paralysis. Most are caused by central nervous system disorders, such as Arnold-Chiari malformation. The majority of patients with this disease present by 4 weeks of age. Airway management may include tracheotomy; however, treatment of the hydrocephalus from an Arnold-Chiari malformation usually leads to improvement of vocal fold function.
 3. **Other congenital laryngeal abnormalities** include subglottic stenosis, laryngeal saccular cysts, and laryngoceles. Faulty development of the larynx can lead to laryngeal clefts, webs, or atresia.
- C. Trauma**
1. **Laryngeal trauma** can occur in many ways. **Internal trauma** can be caused by swallowing a caustic agent or can be iatrogenic (e.g., intubation). **External trauma** can be blunt or penetrating, or both. The first step is always a careful assessment of the airway and its stability. Intubation may induce more damage and should be avoided if at all possible. A controlled tracheotomy is the best way to secure an airway if severe laryngotracheal injury is suspected.
 2. **Fiberoptic nasopharyngoscopy** is used in those patients with a stable airway to assess the degree of injury. High-resolution CT scan is often desirable to define any laryngeal framework injuries. Surgical intervention includes laryngoscopy, esophagoscopy, and exploration. Repair is directed at fractures and mucosal injuries and usually involves stenting. Long-term problems include hoarseness, aspiration, and glottic stenosis.
- D. Infection. Infectious laryngitis** is either acute or chronic. A virus typically causes acute laryngitis, usually by one of the etiologic agents of the “common cold” (rhinoviruses). Symptoms usually last no more than 5 days. Bacterial laryngitis is less common than viral laryngitis and is associated with pharyngotonsillitis. The most common pathogens are *S. pneumoniae*, *Streptococcus pyogenes*, *H. influenzae*, and *B. catarrhalis*. The cause of epiglottitis in children is most often *H. influenzae* type B, although the incidence is decreasing, probably as a direct result of the use of the *H. influenzae* type B vaccine. In adults, infection usually involves the entire supraglottis and is termed *supraglottitis*.
- E. Tumor.** Any growth on the larynx causes hoarseness. The type and extent of the tumor cause other symptoms, such as dysphagia, odynophagia, otalgia, and cough.
1. **Benign tumors** of the larynx are very rare; however, they can cause significant morbidity due to their location. They should be differentiated from malignant tumors so that conservative measures can be undertaken. Recurrent respiratory papillomatosis is the most common benign tumor of the larynx. Human papillomavirus types 6 and 11 are thought to be the causative agents. Extensive disease can develop, requiring tracheotomy. Long-term treatment usually involves repeated carbon dioxide laser excision. Other benign laryngeal tumors include oncocytic papillary cystadenoma, granular cell tumors, lymphangiomas, paragangliomas, and chondromas.
 2. **Laryngeal cancer** is the most common head and neck malignancy. Squamous cell carcinoma represents 85–95% of all laryngeal cancers. Precursor lesions include hyperplasia, hyperkeratosis, and various grades of dysplasia or atypia. There are approximately 12,500 new cases per year, and the major risk factors are tobacco use, alcohol abuse, and nutritional deficiencies. Grossly, they appear as papillary exophytic masses or as ulcerative, infiltrative lesions.
 3. The **diagnosis** is made by biopsy, usually during laryngoscopy, while the patient is under general anesthesia. Patients with large, bulky cancers may require a tracheotomy before laryngoscopy. A CT scan is valuable for supraglottic lesions because it can detect preepiglottic spread and spread to the base of the tongue. For glottic lesions, CT scans can detect cartilage invasion, indicating a T4 primary. For subglottic lesions, CT can determine the inferior extent of the lesion. The American Joint Committee on Cancer developed the staging for head and neck cancers ([Table 34-1](#)).

Table 34-1. American Joint Committee on Cancer generalized head and neck tumor staging system (except nasopharynx and thyroid)

4. The **treatment** of laryngeal cancers depends on the site, stage, and functional status of the patient. Small glottic cancers can be treated with radiotherapy, endoscopic excision with or without a laser, or via an open approach, such as laryngofissure. For larger cancers, the most definitive treatment is total laryngectomy. However, partial laryngectomy techniques can eliminate the need for a permanent tracheostomy and preserve glottic function. Small supraglottic cancers can be treated with radiation or surgery with equal effectiveness. In general, for stage II and IV cancers, combined modality therapy is preferred, combining resection postoperative external beam radiation. The extent of resection is often determined at the time of surgery; therefore, consent should be obtained from all patients for a possible total laryngectomy. Subglottic cancers are exceedingly rare. They often spread and involve cartilage or thyroid gland. Total laryngectomy and thyroidectomy (often total) followed by radiotherapy is the treatment of choice.
- F. Other laryngeal disorders**
1. **Neuromuscular disorders.** Several characteristic diseases affect the larynx. Patients with myasthenia gravis develop vocal incompetence with use of the voice, which resolves with rest. In addition, ocular, facial, and pharyngeal muscles can also be affected. Vocal cord paralysis due to dysfunction of the recurrent laryngeal nerve has a number of causes. In the case of unilateral paralysis, the most common causes are pulmonary neoplasm, iatrogenic nerve injury, and trauma. Bilateral vocal cord paralysis, although less common, is more dramatic because the airway is compromised. Treatment of unilateral vocal cord paralysis starts with observation and speech therapy. Recovery or accommodation may take place. Surgical treatments include injection laryngoplasty [e.g., with Gelfoam, fat, collagen, or polytetrafluoroethylene (Teflon)], medialization of the vocal cord with a soft silicone (Silastic) implant, and laryngeal reinnervation. Treatment of patients with bilateral vocal cord paralysis starts with airway management, often a tracheostomy. Arytenoidectomy is the procedure of choice and can be performed endoscopically or via an open procedure. Nerve-muscle pedicle transposition and reinnervation have also been used. Amyotrophic lateral sclerosis also affects the larynx via involvement of the pharyngeal and glossal muscles. Vocal cord dysfunction can also arise from several syndromes, including syringobulbia, brainstem lesions, and palatal myoclonus.
 2. **Spasmodic dysphonia** is caused by spasms (dystonias) of the larynx, which produces a strained voice with breaks and decreased loudness; this is the result of adductor hyperfunction. A less common form is abductor dystonia, which produces a breathy voice with breaks and decreased loudness. The pathophysiology of spasmodic dysphonia is unknown; however, this disease is more common in voice professionals. Treatment with baclofen can help in a small percentage of patients. Sectioning of the recurrent laryngeal nerve has also been tried with varied results. The treatment of choice is injection of botulinum toxin into the thyroarytenoid muscle. The results are good but last only several months, and the botulinum injections have to be repeated.

V. Neck

- A. Anatomy and physiology.** The neck is a complex amalgam of vital structures intimately associated with each other. There are three main compartments: the carotid sheath contents; the visceral compartment, containing the pharynx, larynx, and cervical esophagus; and the spinal cord surrounded by the vertebral column.
1. **Fascia of the neck.** The thin, superficial layer of fascia is located in the subcutaneous tissue and invests the platysma muscle. The deep fascia is divided into three layers. The superficial layer of the deep fascia (investing layer) is deep to the platysma and invests the strap, sternocleidomastoid, and trapezius muscles. This layer also invests the parotid and submandibular glands. The middle layer of the deep fascia surrounds the visceral compartment, and the deep layer encloses the paravertebral muscles and is called the prevertebral fascia. All three layers of the deep fascia contribute to the carotid sheath.
 2. The **posterior triangle** is posterior to the sternocleidomastoid and anterior to the trapezius. It contains cutaneous cervical branches and cranial nerve XI. The anterior triangle is anterior to the sternocleidomastoid, inferior to the mandible, and superior to the clavicle. There are many important structures in this triangle and its subdivisions. The carotid sheath contains the carotid artery and its branches, the internal jugular vein and its tributaries, the vagus nerve, and the ansa cervicalis. Superiorly, at the skull base, cranial nerves XI and XII become associated with the sheath contents. Cranial nerve XII hooks around the occipital artery. The ansa supplies the extrinsic laryngeal, or strap, muscles.
 3. There is an extensive network of **superficial and deep lymphatics and lymph nodes** throughout the head and neck ([Fig. 34-2](#)). Most lymphatics travel with neural and vascular structures—for example, the accessory chain of nodes that accompany cranial nerve XI and the facial nodes that lie adjacent to the facial artery. The parotid gland contains lymph nodes within it, unlike the submandibular gland. The superficial lymphatics drain into the deep chain, which runs adjacent to the carotid sheath. There is a large group of lymph nodes where the sheath crosses the digastric muscle called the *jugulodigastric nodes*. A similar group exists called the *juguloomohyoid nodes*. The entire upper aerodigestive tract drains into the deep lymphatics.

Fig. 34-2. Lymphatic drainage of the head and neck. (From RS Snell. *Clinical anatomy for medical students*, 3rd ed. Boston: Little, Brown, 1986, with permission.)

B. Congenital

- 1. Thyroglossal duct cyst.** The thyroid forms between the tuberculum impar and the hypobranchial eminence. The thyroid descends caudally and reaches its normal position by week 7 of gestation. As the thyroid gland descends, it leaves a trail or duct. The duct usually disappears by week 10. Persistence of the duct may give rise to cystic masses at any point along the path, from the floor of the pharynx (foramen cecum of the tongue) to the pyramidal lobe of the thyroid gland.
 - a. Diagnosis** is a cystic midline mass that moves with tongue protrusion and deglutition. Approximately 70% are located above the hyoid bone. It undergoes transient enlargement with upper respiratory tract infections.
 - b. Management.** The presence of a thyroid gland in the normal anatomic position should first be ascertained, either by physical examination or ultrasound. The Sistrunk procedure, which involves excision of the entire tract, cyst, central portion of the hyoid, and a small block of the base of the tongue, is the operation of choice rather than simple excision of just the cystic structure.
- 2. Branchial cleft anomalies.** Persistence of any portion of the branchial cleft results in a cyst (most common), a sinus, or a fistula. A cyst is a mucosal or epithelium-lined structure with no external or visceral connection. A sinus is a tract with or without a cyst that has an internal or external connection. A fistula is a connection from the upper digestive tract to the skin.
 - a. First branchial arch anomalies** usually present as painless swellings in the region of the parotid gland and ear. There are two types, differing in type of tissue, location and direction of the tract, and relationship to the facial nerve.
 - b. Second branchial arch abnormalities** are the most common, and the most common form is a cyst. Second branchial arch anomalies course from the tonsillar fossa, deep to the stylohyoid ligament, superficial to cranial nerves IX and XII, between the internal and external carotid arteries, to the anterior border of the sternocleidomastoid muscle.
 - c. Third branchial cleft fistulas** connect the pyriform sinus to the skin. The tract is deep to the carotid system, deep to cranial nerve IX, and superficial to cranial nerve XII. The tract pierces the thyrohyoid membrane above the internal branch of the superior laryngeal nerve to open into the pyriform sinus.
 - d. Fourth cleft fistulas** have not been documented with certainty. The tract would differ on the right and left side.
 - e. Management** involves complete excision of the cyst or the tract (or both), with careful dissection to avoid injuring important structures as described above. Control of infection should be done with antibiotics preoperatively.
- 3. Cystic hygromas** are benign lesions of the lymphatics of the head and neck. They usually present as slowly growing masses that are soft, compressible, and nontender. MR scanning has become the standard diagnostic imaging technique to delineate the extent of the lesion. Management is expectant, with surgery reserved for disfigurement, feeding or breathing difficulties, or recurrent infections. Complete removal is seldom achieved.
- 4. Teratomas and dermoid cysts.** Teratomas are composed of ectoderm, mesoderm, and endoderm; each tissue capable of achieving different degrees of differentiation. Dermoids are composed of only ectoderm and mesoderm and are more common. Both teratomas and dermoids can present as masses in the head and neck, and are sometimes found on preterm ultrasound.

C. Infection

- 1. Deep neck space abscess.** Several potential spaces in the head and neck can develop suppurative infections. In general, most are caused by dental sources, but they can be a result of trauma, tonsillitis, and suppuration of lymph nodes. **Ludwig's angina** is an abscess in the submental and submandibular spaces, which includes the sublingual space. This produces odynophagia, drooling, trismus, and an inability to talk. The patient has systemic signs of an infection, as well as extensive edema of the submental and submandibular area. The key finding is a firm and edematous floor of the mouth and ventral tongue with posterior displacement of the tongue. This eventually causes airway compromise. Therefore, treatment begins with airway control, either nasotracheal intubation or tracheotomy. Regardless of the method, airway control is best achieved in a controlled setting such as the operating room. Both surgical drainage with sump drains and intravenous antibiotics are required for effective treatment.
- 2. Infectious lymphadenopathy**
 - a. Viral** infections are a common cause of cervical lymphadenopathy, especially in children. Viral infections of the upper respiratory tract caused by adenoviruses, rhinoviruses, and enteroviruses are also the most common cause of acute infectious lymphadenopathy. Infectious mononucleosis is caused by the Epstein-Barr virus and often causes bilateral posterior cervical lymphadenopathy as well as the classic symptoms of malaise and myalgia. Cytomegalovirus, herpes viruses, and the human immunodeficiency virus can also cause cervical lymphadenopathy.
 - b. Bacterial.** Group A streptococci and *S. aureus* account for the majority of bacterial lymphadenitis and lymphadenopathy. Oral antibiotics usually suffice as treatment, and those patients who do not respond require incision and drainage. Lymphadenopathy can be caused by mycobacterial disease. In adults, *Mycobacterium tuberculosis* is the most common agent and usually responds to antituberculosis drugs. In children, nontuberculous organisms are primarily responsible, and treatment usually requires excision or curettage. Cat-scratch disease is caused by *Afipia felis* and *Rochalimaea henselae*. They usually cause a prolonged case of cervical lymphadenopathy; however, these infections usually resolve without specific treatment.
 - c. Fungal.** Fungi are a rare cause of lymphadenopathy. Histoplasmosis, blastomycosis, aspergillosis, and coccidioidomycosis can occur in immunocompetent individuals, whereas mucormycosis and cryptococcosis more often cause disease in immunocompromised patients.
- D. Tumor.** Any neck mass should elicit a comprehensive differential diagnosis. A complete history and thorough physical examination, including fiberoptic examination of the upper aerodigestive tract, should be performed.
 - 1. Benign neoplasms** include hemangioma, lymphangioma, fibroma, lipoma, schwannoma, and paraganglioma.
 - a. Paragangliomas** arise from the paraganglionic cells of the autonomic nervous system. The most common location is the neck, which includes the carotid body, the vagus nerve, and the jugular bulb. There are clusters of epithelial cells (Zellballen) that produce catecholamines. Although only a small number of paragangliomas produce enough catecholamine to cause symptoms, all patients should be screened by measuring serum catecholamine levels. Angiography is the most definitive diagnostic technique and may detect paragangliomas in other locations. Treatment is surgical excision, and the surgical technique is determined by the location of the tumor.
 - b. Schwannomas** are tumors derived from the sheath cells of peripheral nerves. They can develop in the neck, arising from cranial nerves, cervical sensory plexus, cervical sympathetic plexus, or the brachial plexus.
 - c. Neurofibromas** also arise from peripheral nerves, but unlike schwannomas, are associated with von Recklinghausen's disease. Cranial nerves are common locations, particularly cranial nerves II and VIII.
 - 2. Malignant.** In adults, a neck mass should be considered malignant cervical adenopathy until proved otherwise. Delay in the diagnosis adversely affects outcome; however, a premature open biopsy of a node containing squamous cell carcinoma can reduce overall survival. Cancer in the neck is most commonly caused by metastatic squamous cell carcinoma from the upper aerodigestive tract. Distant primaries account for less than 10% of cases. Less than 5% of the time, no primary is found. Lymph node metastasis to the neck reduces survival by approximately 50%. Primary neck cancers are less common and include lymphoma, sarcoma, thyroid carcinoma, and salivary gland carcinoma.
 - a. History.** A detailed list of symptoms and their duration is important, as well a history of weight loss. Tobacco use and alcohol consumption are the major risks for squamous cell carcinoma. Otalgia is associated with advanced hypopharyngeal cancers. Denial is a common reaction and leads to a delay in seeking treatment.
 - b. Physical examination.** The location, number, size, and fixation of the nodes should be assessed. Fixation indicates extracapsular spread. A thorough search for a primary is performed, which includes a fiber optic examination or a formal laryngoscopy. If a lesion is found, a biopsy is performed. This can be done in the clinic but is often performed while the patient is under general anesthesia. The neck mass is then considered spread of that primary.
 - c. Diagnosis.** If no lesion in the upper aerodigestive tract is discovered on initial examination, fine-needle aspiration of the neck mass is performed. This is a highly sensitive and specific test and usually performed before any other test. The initial radiographic test of choice is a CT scan with and without contrast, but an MR scan, a positron emission tomography scan, an angiogram, and a balloon-occlusion test of the carotid may be necessary as the workup progresses for presurgical planning. If the fine-needle aspiration shows squamous cell carcinoma, laryngoscopy, esophagoscopy, and bronchoscopy are performed. Directed biopsies are taken from the nasopharynx, base of tongue, pyriform sinus, and tonsils, as these are the most common sites of cancer origination.
 - d. Treatment** depends on many factors, including the pathologic diagnosis, extent of cancer, and the overall performance status of the patient. In general, surgery or radiation can be used to treat early squamous cell carcinoma of the head and neck. Stage III and IV disease should be treated with combined therapy, usually surgery followed by radiation. Chemotherapy is not as useful, but it is used for advanced cancers, typically combined with radiation, with surgery reserved for salvage.

VI. Oral cavity and pharynx

A. Anatomy and physiology

- 1. The oral cavity** extends from the lips posteriorly to the circumvallate papillae and the palatoglossal fold. It includes the oral tongue, floor of mouth, hard

palate, alveolar ridges, retromolar trigone, lips, and buccal mucosa. The intrinsic and extrinsic muscles of the tongue are innervated by cranial nerve XII. The anterior tongue sensation is from the lingual nerve, and taste is provided through the chorda tympani. The blood supply to the tongue is from the lingual artery and vein. The third division of cranial nerve V supplies motor innervation to the muscles of mastication.

2. The **oropharynx** starts at the soft palate and includes the soft palate, base of tongue, palatopharyngeal fold, tonsillar region, vallecula, and lateral pharyngeal walls. The muscles of the soft palate assist in swallowing by raising the tongue, narrowing the oropharyngeal opening, and closing the nasopharynx.
3. The **hypopharynx** is the area between the pharyngoepiglottic folds and the inferior margin of the cricoid. It contains the pyriform sinus, inferior pharyngeal walls, and the postcricoid area. The pharyngeal constrictors are innervated by cranial nerve X, except the stylopharyngeus, which is supplied by cranial nerve IX.

B. Congenital

1. **Pierre-Robin sequence** includes a U-shaped cleft palate, micrognathia, glossoptosis, and other associated defects. Airway obstruction and feeding difficulties can be treated with positioning and suturing of the tongue. Tracheotomy may be necessary, and surgical correction of the mandibular hypoplasia can be performed later in life.
2. **Tornwaldt's bursa** is a nasopharyngeal cyst that represents the persistence of a communication between the notochord and the roof of the pharynx. The cyst may become infected, causing postnasal discharge and pain. Antibiotic therapy, followed by marsupialization or excision, is the definitive treatment.
3. **Cleft lip and palate**
 - a. **Embryogenesis.** Clefting is a fusion abnormality of the midface skeleton and soft tissues. The incisive foramen separates the primary from the secondary palate. Failure of midline fusion results in clefts. Types of clefting include incomplete or complete, unilateral or bilateral, and cleft of the primary or secondary palate (or both). Cleft lip and palate may occur alone or together.
 - b. **Etiology.** The origin is multifactorial, involving genetic predisposition and environmental factors. The risk increases with the increasing number of affected family members.
 - c. **Treatment.** Patients with cleft lip and palate deformities should be treated by a multidisciplinary cleft team. Feeding, hearing, and speech difficulties must be addressed throughout the patient's life. Cleft lips are closed primarily in the first year of life. Cleft palate repair is undertaken with or separately from cleft lip repair. Cleft palates are usually repaired between the age 6 months and 2 years, in multiple stages, before the development of speech.

C. Trauma

1. Mandible fractures

- a. **Diagnosis.** The signs and symptoms of mandible fracture include malocclusion, trismus (inability to open the mouth), loose teeth, intraoral lacerations, hematomas, or sensation over the lower lip and chin. A Panorex usually shows the fracture well; however, full mandibular films, including a Towne view, provide clear images of the entire mandible. Occasionally, a CT scan is necessary. A cervical spine X-ray may be mandated, depending on the mechanism of injury, to rule out cervical spine fractures.
- b. **Treatment.** The principles of treatment for fractures are reduction, rigid fixation, and avoidance of infection. Maxillary-mandibular fixation involves wiring the maxillary teeth to the mandibular teeth, securing a normal occlusion and stabilizing bone fragments. Maxillary-mandibular fixation can be used in the treatment of nondisplaced fractures, as an adjunct to open reduction and fixation of more complicated fractures. Multiple types of plates are available for fixation of mandible fractures. Rarely external pin fixation is required. Although cumbersome, they are indicated in severely comminuted fractures, infected fractures, and fractures with bone loss.
- c. **Complications** of mandible fracture repair include infection, nonunion, malunion, temporomandibular joint ankylosis, and malocclusion.

D. Infection

1. **Pharyngotonsillitis** may be caused by virus or bacteria. Although viral causes are more common, it is important to screen for infection with beta-hemolytic streptococci. Antibiotics prescribed in a timely fashion decrease the risk of rheumatic fever. Fever, odynophagia, and sore throat are the most common symptoms of pharyngotonsillitis. Examination reveals erythema with an exudate on the tonsils. If the throat cultures are negative, only supportive care is required, although antibiotics are sometimes prescribed. Other diseases to consider are diphtheria, infectious mononucleosis, Vincent's angina, candidiasis, syphilis, gonococcal pharyngitis, and tuberculosis. All these diseases can present with similar symptoms.
2. **Peritonsillar abscess** (quinsy tonsillitis) is a collection of pus between the capsule of the tonsil and the fascia of the adjacent constrictor muscles. It is a complication of tonsillitis, is usually unilateral, and affects adolescents and young adults. Drooling, "hot potato voice," trismus, and continued fever are symptoms frequently associated with a peritonsillar abscess. Examination shows unilateral bulging of the tonsillar fossa and hemi-soft palate. Treatment is incision and drainage under local anesthesia. Smaller children may require general anesthesia. One dose of intravenous antibiotics is warranted, followed by oral administration. Occasionally, a patient requires admission because of dehydration. The risk of recurrence is 10–15%, and some physicians advocate tonsillectomy as definitive treatment.
3. **Retropharyngeal abscesses** occur primarily in children. The lymph nodes in the retropharyngeal space suppurate as a result of an upper respiratory tract infection. Symptoms include a sore and stiff neck, sore throat, odynophagia, dysphagia, and systemic signs of infection. Examination shows a posterior pharyngeal mass, but it can be missed if not specifically sought. Lateral soft-tissue films are helpful, but a CT scan better delineates the lesion. A chest radiograph is mandatory to look for evidence of mediastinitis. Incision and drainage in the operating room and intravenous antibiotics are the treatment of choice. Airway management should always be considered during the diagnostic and treatment plan.
4. **Parapharyngeal space abscess** occurs in the space lateral to the pharynx, between the skull base and the hyoid. Extension from infection in one or more other spaces in the head and neck serve as the most common source. These include the retropharyngeal, peritonsillar, masticator, parotid, submandibular and sublingual spaces. In addition to the signs and symptoms shared with other abscesses, otalgia and marked trismus stand out in this disease process. Timely intervention avoids complications such as cranial neuropathies and thrombosis of the internal jugular vein. Intravenous antibiotics and external drainage are curative. Again, the airway is at risk and should be monitored and managed carefully.

E. Tumor

1. Benign

- a. **Juvenile angiofibroma** is a highly vascular neoplasm that starts at the sphenopalatine foramen and grows slowly into the nasopharynx, with extension into adjacent areas. It affects adolescent boys, who present with unilateral nasal obstruction and epistaxis. Intranasal biopsy is not recommended because of the vascularity of the tumor. A CT scan with contrast, MR scan, or angiogram delineates the avascular nature of the tumor. Preoperative embolization and surgical excision is the treatment of choice.
- b. **Choanal polyps** actually start from the maxillary sinus and can grow to large sizes. They extend from the antrum posteriorly and often can be seen in the oropharynx. Surgical excision is typically curative.
- c. **Mucus retention cysts** can occur anywhere in the oral cavity and pharynx. They arise from the minor salivary glands and often are incidental findings during routine examination.
- d. **Squamous papillomas** are small tumors that resemble berries and are pink or tan. The etiology is thought to be viral, specifically the human papilloma virus. Because these lesions are benign, local excision is the treatment of choice.

2. Malignant

- a. **Nasopharynx.** Carcinoma of the nasopharynx is uncommon in the United States; it is very common in Hong Kong and parts of China. It is associated with the Epstein-Barr virus and certain environmental factors. There are three histologic categories: squamous cell carcinoma, nonkeratinizing carcinoma, and undifferentiated carcinoma. A high rate of cervical metastases occurs with this cancer. Treatment is radiation to the primary mass and the neck. Surgery is reserved for large tumor, significant neck disease, or persistent neck disease or local recurrence after radiation treatment.
- b. **The oral cavity is divided into several subsites.** These include the lips (vermillion), buccal mucosa, alveolar ridges, retromolar trigone, hard palate, anterior tongue, and floor of mouth.
 1. **Lip.** The vermillion border is the dominant feature of the lips and is the most anterior extent of the digestive tract. Lesions that arise from the skin or mucosa adjacent to the vermillion border are not considered lip cancers. Squamous cell carcinomas are the most common; others include verrucous, spindle cell, adenoid squamous cell, and basal cell carcinomas. Regional spread occurs in 5–20% of patients, usually to submental and submandibular nodes. A nonhealing ulcer is the most common presentation, which underscores the need for a high index of suspicion and routine biopsy of any lesion. Although radiation can cure early lip cancers, surgery is the most common modality of treatment. Typically, wide local excision techniques are used. A variety of reconstruction techniques exist to repair the residual defects. Mohs' micrographic excision is becoming a more popular modality of treatment, but it should be reserved for smaller tumors.
 2. **Tongue.** Cancers are more common on the lateral aspect of the tongue and spread easily within the tongue and to adjacent structures. A painful ulcer is common as the initial presentation. Surgery and radiation are the treatment modalities with proven success. In addition, the regional lymphatics should be treated.
 3. **Floor of mouth.** These insidious tumors are often underestimated and undertreated. Involvement of the mandible requires segmental mandibulectomy, postoperative radiation, and treatment of the regional lymphatics.
 4. **Hard palate.** This location is less common for cancers. Often, the tumor is a carcinoma of the minor salivary glands, such as mucoepidermoid carcinoma. Except for small lesions, surgical resection is the modality of choice. The bone is taken as a margin, in the form of a maxillectomy.
- c. **Oropharynx.** Cancers arising in this location tend to be aggressive, less differentiated, and more advanced than cancers at other sites. Most patients have one or more of the following symptoms: odynophagia, dysphagia, trismus, cervical adenopathy, and weight loss. Tongue base and tonsil cancers

have an 80% rate of regional spread. Diagnosis is made by biopsy, usually at the time of laryngoscopy. CT scan is imperative to assess the extent of the primary tumor and regional spread. MR scan is particularly useful to examine the base of the tongue and to look for submucosal spread. Multimodality therapy is the rule, such as surgery followed by postoperative radiation. The approaches to the oropharynx include transoral, mandibular swing with lip split, lateral pharyngotomy, and suprahyoid pharyngotomy. The size and site of resection dictate the planned reconstruction. Skin grafts, pedicled regional flaps, and free flaps are the main options for reconstruction.

- d. **Hypopharynx.** Cancers of the hypopharynx behave like oropharyngeal cancers, with a tendency to spread submucosally and have skip lesions. Because of the proximity to the larynx, resection of these tumors often involves a partial laryngectomy to achieve oncologic margins. Partial and total laryngopharyngectomies are reconstructed with gastric pull-up, free jejunal transfer, free tubed radial forearm free tissue transfer, or regional pedicled flaps. Each has its own advantages and disadvantages. In general, the survival statistics for these cancers are poor despite aggressive treatment.

F. Other conditions of the oral cavity and pharynx

1. **Pediatric obstructive sleep apnea.** Adenotonsillar hypertrophy in the pediatric patient can create airway obstruction during sleep. This causes obligate mouth breathing, snoring, and hyponasal speech. Secondary developmental problems of the palate can ensue. Diagnosis is made by history, examination, and a sleep study. Treatment is a tonsillectomy and adenoidectomy.
2. **Sleep apnea in the adult** is caused by collapse and obstruction of the upper airway during the deep stages of sleep. Snoring, restless sleep, daytime hypersomnolence, and impotence are common. Diagnosis is made by sleep study, but the location of the collapse is determined by examination. The Mueller test looks for the area of collapse while the patient inspires against a closed mouth and nose. Treatment options include continuous positive airway pressure ventilation when asleep or surgery. The most common surgical procedure, an uvulopalatopharyngoplasty, widens the oropharynx and is moderately successful in carefully selected patients. Other procedures are directed at preventing prolapse of the tongue when supine.

35 Plastic and Hand Surgery

Aaron G. Grand, Christopher J. Hussussian, and Susan E. Mackinnon

Introduction

Basic Techniques and Principles

I. The reconstructive ladder

II. Types of grafts

III. Types of flaps

IV. Tissue expansion

Specific Problems in Reconstructive Plastic Surgery

I. Peripheral neuropathy

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Hand Surgery

I. Assessment

II. Fractures

III. Dislocations and ligament injuries

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V. Amputation

VI. Infections

VII. Surgical emergencies

Introduction

Several topics germane to the broad discipline of reconstructive plastic surgery—including wound healing, thermal injury, head and neck surgery, and skin and soft-tissue tumors—are covered in other chapters and so are not duplicated here. Having no specific involvement with a particular organ system or region of the body, plastic surgeons generally concern themselves with the solution of difficult wound healing problems in a way that optimizes form and function. Although specific approaches to select individual problems are covered, most of this chapter describes basic principles that can be applied broadly to an unlimited number of surgical issues.

Basic Techniques and Principles

I. The reconstructive ladder. When considering surgical problems, the simplest approach is often the best approach. This is illustrated by the reconstructive ladder of soft-tissue coverage, in which planning begins with consideration of the least complex alternative (healing by secondary intention) and ends with the most complex (free tissue transfer). An advantage to this approach is that use of less complex procedures ensures alternatives in the case of initial failure.

- A. Allowing a wound to heal by **secondary intention** is the simplest approach but is not always feasible. Absolute contraindications include exposed vessels, nerves, tendons, viscera, or bone. Relative contraindications include a large or poorly vascularized wound with a prolonged (>3 weeks) anticipated period of healing and undesirable esthetic consequences.
- B. **Primary closure** usually provides the most esthetically pleasing result but can be contraindicated due to excessive tension on the skin, which may cause displacement of neighboring structures (e.g., lower eyelid) or necrosis of the skin flaps.
- C. **Skin grafting** is the most common method of closure for large wounds. Its use is contraindicated over exposed vessels, nerves, or viscera (except as temporary cover). A healthy bed is required for skin graft take; wound surfaces that do not support skin grafts include bare tendon, bare bone, radiation-damaged tissue, and any infected wound.
- D. **Local tissue transfers** of skin or muscle may be used in regions with convenient tissue nearby. Local flaps become less useful when the wound is in an area without available local tissue (e.g., the distal leg) or when coverage of the wound requires more soft-tissue bulk than is available nearby.
- E. **Distant tissue transfers** were the mainstays of difficult wound closure until free flaps were developed. This approach involves the transfer of tissue in staged procedures and has the inherent disadvantages of multiple operations, prolonged wound healing, immobilization for at least 3 weeks, and a limited choice of donor site.
- F. **Free tissue transfer** is the most technically demanding approach to wound closure but has several potential advantages. These include single-stage wound closure, a relatively wide variety of flaps to ensure closure specifically tailored to coverage needs, and, in many cases, a more acceptable esthetic outcome.

II. Types of grafts

A. Skin grafts

1. **Split-thickness grafts** consist of epidermis and a variable thickness of dermis. Thin grafts (<0.016 inches) have a higher percentage of engraftment, whereas thicker grafts are more durable and esthetically acceptable. Common donor sites are the thigh, buttock, and scalp.
 2. **Full-thickness grafts** include epidermis and a full layer of dermis. Common donor sites include groin, postauricular, and supraclavicular sites, but the hypothenar eminence and instep of foot can also be used. The donor site is usually closed primarily. These grafts are generally used in areas for which a high priority is placed on the esthetic result (e.g., face and hand) and where the area to be covered is limited.
 3. Grafts can be meshed in **expansion ratios** from 1.5:1.0 to 6:1. Meshing a graft allows it to cover a wider area and decreases the risk of seroma accumulating under the graft because fluid can escape through the fenestrations. The interstices are covered within 1 week by advancing keratinocytes. However, because the entire area is not covered by dermis, meshed grafts are less durable, and the meshing pattern remains after healing, making them inappropriate for esthetically important areas, such as the face.
 4. **Graft healing.** Initial metabolism is supported by imbibition or diffusion of nutrients from wound bed. Revascularization occurs between days 3 and 5 by ingrowth of recipient vessels into the graft (inosculation). Therefore, for a graft to take, the bed must be well vascularized and free of infection and the site immobilized for a minimum of 3–5 days. Prevention of shear forces is particularly important during this period of inosculation. Although bare bone and tendon do not engraft, periosteum and peritenon can support skin grafts, especially if they are first left to form a layer of granulation tissue.
- B. **Tendon grafts** are used to replace or augment tendons. Preferred donor sites are palmaris longus and plantaris tendons.
 - C. **Bone grafts** are used for repair of bony defects. Iliac bone is commonly used for donor cancellous bone, and ribs or outer table of cranium are commonly used for donor cortical bone.
 - D. **Cartilage grafts** are used to restore the contour of the ear, nose, and eyelid. Preferred donor sites include costal cartilage, concha of ear, and nasal septum.
 - E. **Nerve grafts** are used to repair damaged nerves when primary repair is not feasible. Preferred donor sites include the sural nerve and lateral or medial antebrachial cutaneous nerves. Use of the greater auricular nerve has been described for repair of the facial nerve. Allogeneic nerve grafting has been described using a short course of immunosuppression (*Plast Reconstr Surg* 90:695, 1992).
 - F. **Dermal or dermal-fat grafts** are used for contour restoration. Preferred donor sites include back, buttock, and groin. The long-term survival of grafted fat is variable but is generally unreliable.

III. Types of flaps. A flap is any tissue that is transferred to another site with an intact blood supply.

A. Classification based on blood supply

1. **Random cutaneous flaps** have a blood supply from the dermal and subdermal plexus without a single dominant artery. They generally have a limited length-to-width ratio (usually 3:1), although this varies by anatomic region (i.e., the face has a ratio of up to 5:1). These flaps are usually used locally to cover adjacent tissue defects but can be transferred to a distant site by use of a staged procedure. Depending on the size of the defect to be covered, moving a local tissue flap can create a donor defect, which may require skin grafting. All local flaps are comparatively easier to use with the loose skin of the elderly.
 - a. **Flaps that rotate** around a pivot point include rotation ([Fig. 35-1](#)) and transposition ([Fig. 35-2](#)) flaps. Planning for shortening of the effective length through the arc of rotation is important when designing these flaps. More complex rotation flaps include the bilobed ([Fig. 35-3](#)) and rhomboid ([Fig. 35-4](#))

flaps.

Fig. 35-1. Rotation flap. **A:** The edge of the flap is four to five times the length of the base of the defect triangle. **B,C:** A backcut or Bürow's triangle can be useful if the flap is under tension.

Fig. 35-2. **A:** Transposition flap. The secondary defect is typically covered with a skin graft. **B:** A backcut may be added to reduce tension at the pivot point.

Fig. 35-3. Bilobed flap. After the lesion is excised, the primary flap (P) is transposed into the initial defect, and the secondary flap (S) is moved to the site vacated by the primary flap. The bed of the secondary flap is then closed primarily. The primary flap is slightly narrower than the initial defect, whereas the secondary flap is half the width of the primary flap. To be effective, this must be planned in an area where loose skin surrounds the secondary flap site. Three choices for the secondary flap are shown (S₁, S₂, S₃).

Fig. 35-4. Rhomboid or Limberg flap. The rhomboid defect must have 60- and 120-degree angles so that the length of the short diagonal is the same as the length of the sides. The short diagonal is extended by its own length to point *E*. The line *EF* is parallel to *CD*, and they are equal in length. There are four possible Limberg flaps for any rhomboid defect; the flap should be planned in an area where loose skin is available to close the donor defect primarily.

b. **Advancement of skin** directly into a defect without rotation can be accomplished with a simple advancement ([Fig. 35-5](#)), V-Y advancement ([Fig. 35-6](#)), or bipedicle advancement flap.

Fig. 35-5. Single-pedicle advancement flap. **A:** Advancement using the skin's elasticity. **B:** Advancement using Bürow's triangle to equalize the length of the sides of the closure. **C:** Pantographic expansion.

Fig. 35-6. V-Y advancement. The skin to the sides of the V is advanced.

2. **Axial cutaneous flaps** contain a single dominant arteriovenous system. This results in greater potential length-to-width ratio.
 - a. **Peninsular flaps** are those in which the skin and vessels are moved together as a unit.
 - b. **Island flaps** are those in which the skin is divided from all surrounding tissue but maintained on an isolated, intact, vascular pedicle.
 - c. **Free flaps** are those in which the vascular pedicle is isolated and divided. The flap and its pedicle are then moved to a new location and microscurgically anastomosed to vessels at the recipient site, allowing for long-distance transfer of tissue.

B. Classification based on tissue type

1. **Cutaneous flaps** include the skin and subcutaneous fat. These are generally random flaps because the axial blood supply is deep to the fat.
2. **Fasciocutaneous flaps** are axial flaps with a single dominant blood supply contained in the deep fascia along with the overlying fat and skin. A wide variety of fasciocutaneous flaps have been described, but those commonly used include radial forearm, parascapular, and groin flaps.
3. **Muscle flaps** use the specific axial blood supply of a muscle to provide a well-vascularized soft-tissue bulk. These flaps can often be transferred with the overlying skin as a myocutaneous flap (see Basic Techniques and Principles, [section III.B.4](#)). Alternatively, they may be transferred without the overlying skin to fill a cavity or may be covered with a skin graft. Considerations in transfer of vascularized muscle include the pattern of circulation, arc of rotation, donor-site contour, and donor-site functional defects. Commonly used muscle flaps include the latissimus dorsi, pectoralis major, rectus abdominis, gastrocnemius, soleus, gracilis, tensor fascia lata, trapezius, and gluteus maximus, but any muscle can potentially be transferred as a flap.
4. **A musculocutaneous flap** involves transfer of a muscle with the overlying skin and subcutaneous tissue. The skin is vascularized via myocutaneous or septocutaneous perforating vessels.

C. Specialized flaps

1. **Fascial flaps** are used when thin, well-vascularized coverage is needed (e.g., for coverage of ear cartilage or the dorsum of the hand or foot). The temporoparietal fascia flap is a classic example, but other fasciocutaneous flaps (see basic Techniques and Principles, [section III.B.2](#)) can be transferred without the overlying skin.
2. **Vascularized bone flaps** are designed to meet specific reconstructive needs, as dictated by loss of bony structure. Because they must be transferred to a specific location, they are generally transferred as free flaps. They may or may not include muscle and/or overlying skin. Commonly used bone flaps include free fibula, scapular spine, iliac (with overlying internal oblique muscle), and rib (with pectoralis major or intercostal muscle).
3. **Functional muscle** may be transferred with its accompanying dominant nerve. Common functional muscle transfers involve gracilis for restoration of facial movement or latissimus for replacement of biceps function.
4. **Segmental muscle flaps** can be used when the blood supply to the muscle derives from more than one source. A portion of the muscle is used as a flap, leaving behind a vascularized, innervated, functional muscle, thus minimizing donor-site functional loss. Examples of muscles that can be transferred based on segmental blood supply include the serratus anterior and gluteus maximus.

IV. Tissue expansion is a reconstructive technique that uses inflatable silicone devices to provide donor tissue of similar color, texture, thickness, and sensation with minimal scar formation and donor-site morbidity. The technique takes advantage of the skin's ability to accommodate a slowly enlarging mass beneath it by increasing its surface area. The idea is to create and develop donor tissue, harvest it, and leave the original donor site preserved.

- A. **Advantages** include low donor-site morbidity and the provision of donor tissue of similar quality to the recipient tissue. It is a versatile technique that is simple and produces robust flaps.
- B. **Disadvantages** are that it is a staged technique, there is a visible deformity during the period of expansion, it requires frequent visits for expansion, and there is a relatively high complication rate.
- C. **Technique**
1. **Preoperative planning** involves assessing the defect size, locating matching tissue to be expanded, and deciding where final scars will be. In choosing the size of the expander, one rule of thumb is that the expanded convex circumference, not including base, minus the width of the base equals the length of expanded flap.
 2. **Expander placement** is usually performed through an incision at the junction of the lesion and the area of proposed expansion. The length of the incision is controversial, with some proposing one-third the length of the expander (it should be big enough to ensure full pocket creation). Placement of the filling port can be in a separate pocket or under the lesion to be removed. There may be advantages to partially filling the expander when placed in that it reduces the duration of the expansion phase and it may reduce fold flap failure.
 3. **The expansion phase** begins 2–3 weeks after expander placement. The expander is then inflated weekly with saline using sterile technique. The volume per instillation depends on patient comfort, skin tension, and blanching of overlying skin. A rough guide is 10% of expander volume per injection. The duration of the expansion phase can vary from 6 weeks to 3 months.
 4. **Removal of expander** is straightforward. However, premature removal may be required secondary to infection, exposure, or rupture.
 5. **Use of expanded tissue** is usually in the form of a random flap (rotation, advancement, or transposition). If more than one flap is created from expanded tissue, one needs to ensure that all have adequate blood supply. Excision of the capsule that forms around the expander may be necessary to maximize expansion.
- D. The **origin of the new tissue** is not completely understood. One potential source is new tissue that is created in response to the expansion process. Alternatively, tissue may derive from recruitment of adjacent tissues by stretching or creep and by stress relaxation. These possibilities are not mutually exclusive. Studies have shown an increase in the thickness of the epidermis with a decrease in the thickness of the dermis (*Clin Plast Surg* 14:435, 1987).
- E. **Tissue expanders are indicated** in patients in whom flap creation is otherwise not possible. In areas where little suitable tissue is available (e.g., scalp), it can be the esthetically superior option. The patient must be motivated and understand the process. Common indications include burn alopecia, congenital nevi, male pattern baldness, and postmastectomy breast reconstruction.
- F. **Relative contraindications** include malignancy or an open wound. Similarly, tissue expanders cannot be placed under burned tissue, scar, skin graft, or a prior incision. In addition, tissue expanders are less effective in areas that will be irradiated, as the skin in those areas thickens, scars, and contracts, minimizing the degree of expansion possible.
- G. **Complications** include pain, seroma, hematoma (rates widely variable), infection (1–5%), and exposure or extrusion (5–10%). Less common complications include striae, resorption of underlying bone, and neuropathia.

Specific Problems in Reconstructive Plastic Surgery

I. Peripheral neuropathy

- A. **Clinical assessment** of neuropathy requires evaluation of both motor and sensory function as well as electrodiagnostic evaluation of nerve conduction and muscle innervation.
1. **Standard classification schemes** are available for classification of motor and sensory nerve functions ([Table 35-1](#) and [Table 35-2](#)). In addition, specific testing of moving or static two-point discrimination, vibration and pressure thresholds, or grip strength may be appropriate.

Table 35-1. Classification of sensory (S) function

Table 35-2. Classification of motor function

2. **Diagnostic studies** for quantification of nerve dysfunction include nerve conduction studies (NCS) and electromyography (EMG). NCS characterizes the conduction of large-diameter, myelinated nerves, and normal values may be present in the face of partial nerve injury. NCS is useful in determining the degree of nerve dysfunction; the presence of segmental demyelination or axonal degeneration; the site of injury; and whether the injury is unifocal, multifocal, or diffuse. EMG samples the action potentials from muscle fibers and can detect individual motor unit potentials, which may indicate early reinnervation, and fibrillations, which represent denervation owing to axonal degeneration.
- B. **Acute nerve injury** results from transection, crush, or compression and represents the loss of nerve function distal to the area of injury. Axons are myelinated by Schwann cells and organized into fascicles surrounded by the perineurium. The fascicles are bundled into nerves by the epineurium. The prognosis of injury to a peripheral nerve is dictated by which structures are disrupted. The severity of nerve injury has been organized into a grading scheme ([Table 35-3](#)). Operative repair is indicated for fourth- through sixth-degree injury.

Table 35-3. Classification of nerve injuries

1. **The technique of nerve repair** affects the eventual degree of recovery. Several basic concepts are used to optimize outcome.
 - a. **Microsurgical technique** should be used, including magnification and microsurgical instruments and sutures.
 - b. When conditions allow, a **primary repair** should be performed. The repair should be tension free.
 - c. Positioning a limb or digit in extreme flexion or extension to facilitate an end-to-end repair is discouraged because of the joint and ligamentous problems that result. If a tension-free repair cannot be achieved in **neutral position**, an interposition nerve graft should be used.
 - d. An **epineural repair** is typically performed, but a grouped fascicular repair should be performed whenever the internal topography of the nerve is segregated into motor, sensory, or regional components.
 - e. **Postoperative motor and sensory reeducation** will help to optimize outcome.
2. **Indications** for peripheral nerve repair include partial or complete transection or in-continuity conduction block. These represent fourth- to sixth-degree nerve injuries and can be difficult to distinguish from lesser grades of injury based on clinical examination alone. This is true because all grades of injury can lead to complete loss of function. Some guidelines for surgical intervention are listed in the following sections.
 - a. **Nerves inadvertently divided** during operation are fifth-degree injuries and should be repaired immediately.
 - b. **Closed-nerve injuries that localize near an anatomically restrictive site** (e.g., the ulnar nerve at the elbow or the common peroneal nerve at the knee) can result in neurologic deficit secondary to conduction block from edema and compression. If no recovery occurs within 3 weeks, management includes surgical decompression at that site. Iatrogenic nerve deficit from positioning during long operative procedures is managed similarly.
 - c. **Closed-nerve injury from blunt trauma or traction** is usually a first-, second-, or third-degree injury, and full recovery can be expected in most cases. Patients are closely followed for signs of recovery, including an advancing Tinel's sign, indicating regenerating axons. Baseline NCS and EMG are obtained at 6 weeks. If there is no evidence of return of function at 3 months, repeat studies are obtained. If there is no improvement, the nerve is

explored and repaired.

- d. **Nerve deficit after sharp trauma** (e.g., stab wounds) usually represents partial or complete transection and should be explored and repaired urgently.
 - e. **Loss of nerve function after gunshot or open blunt trauma** is usually the result of first- or second-degree injury, and recovery can be expected in most cases. These cases are usually treated as for closed injuries. If the nerve is visible or the wound is explored for other reasons (i.e., vascular repair), the nerve is explored. If the nerve is in continuity, it is managed as for a closed injury. If the nerve is not in continuity, it is usually best to tag the ends of the nerve for ease of identification and delay definitive repair until the zone of injury to the nerve is clearer (generally by 3 weeks).
 - f. **Nerve deficit from compartment syndrome** is treated by emergent fasciotomy. If decompressed early (within 6 hours), there is usually a rapid return of function.
 - g. **Decompression of injured nerves** (e.g., ulnar nerve transposition or carpal tunnel release) at sites distal to trauma can be useful to avoid retardation of nerve regeneration across these areas. Multiple sites of injury or compression can have additive effects, and for first-through third-degree injuries, decompression can improve outcome.
 - h. **Division of a sensory nerve** can lead to a painful neuroma as the regenerating axons grow into the surrounding soft tissue. If the resulting neural deficit results in loss of function or protective sensation, these nerves can be repaired. If not, the neuroma is excised and the cut end of the nerve is transposed proximally well away from the wound, preferably into a nearby muscular environment.
- C. **Chronic entrapment neuropathy** due to compression or repetitive trauma represents a common clinical problem. Commonly involved nerves include the median nerve at the wrist, the ulnar nerve at the elbow or wrist, the anterior or posterior interosseous nerves in the forearm, the brachial plexus at the thoracic outlet, the common peroneal nerve at the knee, and the posterior tibial nerve at the ankle.
1. **Clinical assessment** of these conditions involves assessment of motor and sensory function as well as provocative testing (reproducibility of symptoms with extrinsic nerve compression) and presence of a Tinel's sign. EMG and NCS are appropriate if the clinical picture is unclear.
 2. **Initial management** is usually physical therapy, behavior modification, and splinting to avoid repetitive compression. At least 6 weeks of nonsurgical management without improvement is usually recommended before operation, although nerve compression at the cubital tunnel or thoracic outlet typically requires prolonged nonsurgical management.
 3. Operations generally involve **decompression** of the affected nerve or transposition to an unrestricted site.

II. Scalp, calvarial, and forehead reconstruction

A. Anatomy

1. The **scalp** consists of five layers: skin, subcutaneous tissue, galea aponeurotica, loose areolar tissue, and pericranium.
2. **Five major paired vessels** provide the scalp with an ample collateral blood supply: the supraorbital, supratrochlear, superficial temporal, posterior auricular, and occipital arteries.
3. The **scalp receives sensory innervation** from the supraorbital and supratrochlear branches of cranial nerve V1, the lesser occipital branch of C2 or C3, the greater auricular nerve, and the auriculotemporal branch of cranial nerve V2. The motor innervation to the frontalis derives from the frontal branch of the facial nerve.

B. **Congenital scalp defects** in the form of aplasia cutis congenita are rare but difficult problems. Initial management consists of prevention of desiccation that may result in rupture of the sagittal sinus. In most cases, definitive treatment requires free flap coverage (latissimus dorsi) once the infant is of sufficient size.

C. **Scalp lacerations** are common concomitant sequelae of blunt trauma to the head. As such, there may be associated skull, cervical spine, or intracranial injuries. The rich blood supply to the scalp can produce significant blood loss, and hemostasis is important to prevent subgaleal hematoma. Radical débridement is seldom indicated, and primary repair is usually feasible. Repair of the galea generally helps to prevent hematoma formation.

D. **Partial-thickness scalp loss from avulsion** usually occurs at the subaponeurotic layer. Large avulsions may be skin grafted. One can expect 20–40% contraction of the skin graft over the first 6–8 months. After this has leveled off, the grafted area can be removed by serial excisions.

E. **Full-thickness scalp loss** can occur from trauma or tumor extirpation. The optimal treatment varies depending on the size of the defect.

1. **Small defects** (<3 cm) can often be closed primarily after undermining of flaps. Local flaps, either random or based on blood supply, can be raised. Scoring the galea in a grid pattern of perpendicular lines spaced 1 cm apart can allow for expansion of the flap. Rotation flaps should involve a margin of at least five times the length of the defect. Bipedicled flaps are well-suited for coverage of the poles of the head (forehead, temporal areas, and nape of neck).
2. **Medium-sized defects** (3–10 cm) are usually covered with a scalp flap combined with skin grafting of the donor pericranium. Several specific flaps have been described for medium-sized defects, including the pinwheel flap, the three-flap technique, and the four-flap technique. All have been used with variable success.
3. **Large defects** (>10 cm) often require free tissue transfer. If the deficit is due to trauma, replant may be attempted. Because most of these injuries are from industrial accidents involving avulsion, however, the injury to the arterial intima can extend far into the scalp. Latissimus dorsi or omental free flaps with split-thickness skin grafts are described for complete scalp loss.

F. **Calvarial defects** in the parietal or occipital regions require cranioplasty for protection. Temporal defects are somewhat protected by the temporalis muscle.

1. **Alloplastic material** can be used to cover these defects, methylmethacrylate being the most commonly described. It is durable, is a poor heat conductor, and is easy to use, but infection rates are reported at approximately 5% and can be as high as 30% with infection nearby. Newer alloplastic materials are being developed to promote bony ingrowth and decrease the risk of infection. Some can be custom-made, based on three-dimensional reconstructions of computed tomographic scans.
2. **Autogenous tissue for cranioplasty** includes split-rib grafts, split-table calvarial bone grafts, and bone paste. These are somewhat more difficult to use but have the advantage of a lower complication rate.

III. Trunk

A. Breast

1. **Postmastectomy breast reconstruction** offers restoration of an important symbol of femininity and sexual intimacy. Reconstruction of breast symmetry can lead to a significant improvement in body image and is an important part of cancer rehabilitation for many women (*Scand J Plast Reconstr Surg* 18:221, 1984).
 - a. **Reconstruction aims** are to create symmetric breast mounds and, if desired, a new nipple-areola complex. The esthetic goal is defined by the patient and includes a symmetric appearance both clothed and unclothed. Extensive preoperative consultation is required to allow women to explore their options. It should be emphasized that each approach to breast reconstruction usually requires at least two procedures and that the reconstructed breast will never completely replicate the original. Reconstruction can be accomplished with or without the use of an implant, and most procedures can be performed either immediately at the time of the mastectomy or in a delayed fashion.
 - b. **Reconstruction of the breast mound** is accomplished with an implant in approximately two-thirds of cases (*Probl Gen Surg* 13:75, 1996). In most cases, enough skin is removed with the mastectomy that the desired size of the breast precludes closure of the wound without tension. When this is the case, a tissue expander is placed and serial expansions performed until the desired size is reached (usually after 6 weeks of expansion). At this time, the expander is replaced with a permanent implant filled with silicone gel or saline. The advantages of this approach to reconstruction are that minimal operative time is required, additional scars are minimized, and the recovery period is shorter. Disadvantages include the risks of permanent implants (rupture, infection) and the inability to reproduce certain natural contours.
 - c. **Autologous tissue** can be used to recreate a breast mound in the form of pedicled (rectus abdominis, latissimus dorsi) or free (rectus abdominis, gluteus maximus) myocutaneous flaps. The advantages include a more natural appearance for some patients, permanent reconstruction without the potential for future procedures to replace a ruptured implant, and fewer complications with subsequent radiation therapy. Disadvantages include a relatively long procedure, additional scars, and potential donor-site morbidity.
 - d. **Reconstruction of the nipple-areola complex** is chosen by approximately 50% of patients undergoing breast reconstruction. A variety of methods are used and include local flaps or nipple-sharing grafts to reconstruct a nipple-like prominence. Split-thickness skin grafting or tattooing can be used to recreate an areola.
 - e. **Procedures on the contralateral breast to improve symmetry** may be performed concomitantly or subsequently and include modification of an inframammary fold, removal of dog ears, liposuction of flaps, or reduction mammoplasty or mastopexy of the contralateral side. These procedures are almost always covered by insurance.
2. **Reduction mammoplasty** is performed for women with a variety of physical complaints and aberrations in body image.
 - a. **Common symptoms** are listed and are considered indications for reduction mammoplasty.
 1. **Personal embarrassment and psychosocial problems**
 2. **Shoulder and back pain**
 3. **Grooving of the soft tissue of the shoulders by bra straps**
 4. **Chronic inframammary skin breakdown, rash, or infection**
 5. **Inability to engage in vigorous exercise**
 6. **Symptoms of brachial plexus compression (rare)**

- b. A variety of procedures are designed to **reduce breast size**. All of them move the nipple-areola complex superiorly on the chest wall. The nipple-areola complex is maintained on a pedicled blood supply when possible, but in certain instances (e.g., pedicle length >15 cm or a patient who smokes), the nipple-areola complex is transferred as a full-thickness graft. There are always scars resulting from the movement of the nipple and resection of excess skin, and the configuration of these scars varies by the procedure chosen.

B. Chest wall reconstruction

1. **Before beginning chest wall reconstruction**, one must ensure complete resection of tumor and radiation-damaged or infected tissue.
2. **Dead space** in the chest allows for potential empyema and must be obliterated. This space is best filled with pedicled muscle (latissimus dorsi, pectoralis major, serratus anterior, or rectus abdominis) or omental flaps.
3. **Skeletal stabilization** is required if more than four rib segments or 5 cm of chest wall is missing. This can be achieved using autologous (rib, dermis, or fascial grafts or bulky muscle flaps) or prosthetic (Prolene mesh, Gore-Tex, Marlex/methylmethacrylate sandwich) material.
4. **Optimal soft-tissue coverage** usually requires pedicled myocutaneous flaps but can be achieved with pedicled muscle or omentum covered with split-thickness skin graft. Rarely, free tissue transfer is required.
5. **Median sternotomy dehiscence** owing to infection occurs in 1–2% of cardiac procedures. Predisposing factors include bilateral internal mammary artery harvest, diabetes mellitus, obesity, and multiple operations. Closure requires removal of wires and débridement of all infected tissue, including bone and cartilage. Closure of the resultant dead space is usually accomplished by advancing or rotating the pectoralis major and/or rectus abdominis muscles. The rectus abdominis muscle cannot be used as a rotational flap if the ipsilateral internal mammary artery has been harvested. Pedicled omental flaps are reserved as alternatives in case of initial failure.
6. **Poland's syndrome** is a congenital soft-tissue deficit of the chest wall that is sometimes associated with ipsilateral upper extremity deformities. Although the skin and nipple-areola complex are usually intact, the breast tissue, subcutaneous fat, and underlying pectoralis muscles are hypoplastic or absent. Reconstruction is usually performed by transposition of the latissimus dorsi muscle anteriorly and use of a breast implant.

C. Abdominal wall reconstruction

1. **Reconstruction of full-thickness abdominal wall defects** includes recreation of a fascial barrier and skin coverage. Restoration of a functional muscle layer is also helpful in maintaining abdominal wall functionality.
2. **Complete absence of all layers of the anterior abdominal wall** is usually the result of direct trauma or infection, with or without intraabdominal catastrophe. The open abdomen can be temporized by skin grafts placed directly on bowel serosa, omentum, or absorbable mesh through which granulation tissue has formed. This allows for resolution of intraabdominal edema and maturation of adhesions but usually results in a large ventral hernia.
3. **Primary closure of fascial defects** represents the best approach and can be assisted by sliding myofascial advancement flaps. This is accomplished by lateral release of the external oblique fascia. The anterior sheath of one or both rectus muscles can be divided and turned over to provide additional fascia for closure. Synthetic mesh is used when fascial defects cannot be primarily closed. These are made of various materials, each with different biomechanical properties.
4. **Muscle flaps** are required when the existing fascia is insufficient for closure after advancement. The most frequently used flaps are the tensor fascia lata, rectus femoris, and vastus lateralis. These flaps are usually not useful to close defects of the upper abdomen.
5. **Skin coverage** is accomplished with split-thickness skin grafts, the cutaneous portion of a myocutaneous flap or local tissue rearrangement (e.g., bipedicled flap or V-Y advancement flap). As skin grafts cannot survive directly on synthetic mesh, a muscle flap may be required to provide an adequate bed for skin grafting.

D. Pressure sores

1. The etiology and staging criteria have been described separately (see [Chapter 10](#)).
2. **Principles of nonoperative management** of pressure sores include (1) relief of pressure by positioning changes and appropriate cushioning; (2) bedside débridement of devitalized tissue; (3) optimization of the wound environment with aggressive wound care; (4) avoidance of maceration, trauma, friction, or shearing forces; and (5) reversal of underlying conditions that may predispose to ulcer development. This type of aggressive nonoperative management is often optimally coordinated by specially trained wound care nurses.
3. **Operative management** with soft-tissue flap closure is only indicated for large, deep, or complicated ulcers and then only in patients who are able to care for their wounds. A high degree of cooperation from the patient and caregivers is essential because the recurrence of pressure sores at the same site or new sores at other sites after operation is high. This is especially true for individuals who have spinal cord transection from firearm injuries, whose rate of recurrence is 91%, with a mean time to recurrence of 18 months (*Adv Wound Care* 7:40, 1994). This is most likely the result of breakdown in the postoperative support and care systems in this population. Most surgeons, therefore, require demonstration of the patient's ability to care for wounds before embarking on operative closure. Flaps commonly used for closure of pressure ulcers around the pelvic girdle include gluteus maximus, tensor fascia lata, hamstring, or gracilis-based rotation or advancement flaps.

IV. Lower extremity. Soft-tissue defects from trauma to the lower extremity are common. A multidisciplinary approach involving orthopedic, vascular, and plastic surgeons provides optimal care.

- A. Lower extremity injuries are first assessed according to **advanced trauma life support guidelines**. The general sequence of priorities is as follows:
 1. The first priority is assessment for **concomitant life-threatening injuries and control of active bleeding**. Blood loss from open wounds is often underestimated, and patients must be adequately resuscitated.
 2. The **neurovascular status** is determined. If a nerve deficit is progressive during observation in the emergency room, it is likely the result of ischemia from arterial injury or compartment syndrome.
 3. **Bony continuity** is assessed by radiographs of all areas of suspected injury.
 4. **Operative management** addresses bone stabilization followed by venous and arterial repair. Fasciotomies are indicated for compartment pressures greater than 30 mm Hg or clinical suspicion from preoperative neurovascular examination. Fasciotomy must be performed within 6 hours to avoid ischemic contracture. Nonviable tissue is débrided, and an assessment is made about delayed or immediate soft-tissue coverage.
- B. **Soft-tissue defects of the thigh** are usually closed by primary closure, skin grafts, or local flaps. The thick muscular layers ensure adequate local tissue for coverage of bone and vessels and adequate vascular supply to any fracture sites.
- C. **Open tibial fractures** frequently involve degloving of the thin layer of soft tissue covering the anterior tibial surface. The distal tibia is a watershed zone, and fracture with loss of periosteum or soft tissue leads to increased rates of infection and nonunion.
 1. Open tibial fractures are classified according to the scheme of **Gustilo** ([Table 35-4](#) and [Table 35-5](#)).

Table 35-4. Gustilo open fracture classification

Table 35-5. Gustilo type III subtypes

2. **Gustilo types IIb and c** frequently require flap coverage of exposed bone.
 - a. The **proximal third** of the tibia or knee can often be covered by a pedicled hemigastrocnemius flap.
 - b. The **middle third** of the tibia is often covered by a pedicled hemisoleus flap.
 - c. **Large defects of the distal third** of the tibia generally require coverage by free muscle transfer.
- D. **Limb salvage reconstruction for neoplasm** differs from that for trauma in that large segments of bone, nerve, or vessels may require replacement.
 1. **Skeletal replacement** can be accomplished using an endoprosthesis, allogeneic bone transplant, or vascularized free bone (fibula) transfer.
 2. **Rotationplasty** is an option when resection of the knee involves too much bone to maintain the knee joint. The operation involves replantation of the patient's distal leg, ankle, and foot onto his or her thigh, in a reversed orientation, such that the ankle joint functions as a new knee joint and allows fitting of a below-knee prosthesis. However, with advances in above-knee prostheses, the advantages of this procedure are limited.
- E. **The foot** is divided into regions for purposes of soft-tissue defects caused by trauma or ischemic, diabetic, or infectious ulceration. Optimal coverage of the plantar surface provides a durable, sensate platform.

1. **Small defects of the heel** can be covered using the non-weightbearing skin of the midsole. Larger defects require free muscle transfer and split-thickness skin grafting.
2. **Coverage over the metatarsal heads** is often successful with plantar V-Y advancement and fillet of toe flaps. Multiple fillet of toe flaps or free muscle transfer may be required for large defects.
3. **For fitting of proper footwear**, coverage of the dorsum of the foot must be thin. If paratenon is present, the dorsum can usually be covered with a skin graft. Small areas of exposed tendon may granulate, but larger areas require thin fascial free flaps (temporoparietal, parascapular, or radial forearm) covered by skin grafts.

Hand Surgery

I. Assessment must follow a systematic, efficient, and reproducible approach. Underestimating the extent of a hand injury or infection can lead to extended recovery or permanent loss of function.

- A. **History.** The mechanism and timing of the injury, hand position at the time of injury, hand dominance, and patient occupation are all important to diagnosis and management.
- B. **Examination**
 1. **Inspect the attitude of the patient's hand**, paying attention to the resting position of the digits, and recognize any deformity, swelling, or asymmetry.
 2. **Vascular assessment** requires observation of color, capillary refill, and temperature, as well as presence of pulses and an Allen's test to verify the integrity of the palmar arches. Bleeding is controlled by application of direct pressure, not by blindly clamping tissue, as this often results in serious injury to surrounding structures.
 3. **Motor examination**, both active and passive, involves testing for integrity of the tendons as well as for the ulnar, radial, and median nerves, which innervate the muscles in the hand and forearm ([Table 35-6](#)).

Table 35-6. Unambiguous tests of hand nerve function

- a. **Flexor digitorum profundus (FDP)** is tested by stabilizing the proximal interphalangeal (PIP) joint in extension and having the patient flex the distal interphalangeal (DIP) joint.
- b. **Flexor digitorum superficialis (FDS)** is tested by blocking all other fingers in full extension before asking the patient to flex at the PIP joint.
- c. **Extensor tendons** are tested by having the patient extend each finger individually. It should be noted that connections between neighboring tendons (juncturae tendineum) can mask a proximal laceration.
4. **Sensory testing** involves careful examination of two-point discrimination on the palmar aspect of both radial and ulnar sides of the digits and comparison to the uninjured hand. Normal two-point discrimination is 2–6 mm at the distal tip of the digit. Light touch is used to assess the sensation on the dorsum of the patient's hand and for his or her forearm. In children, observe for absence of sweating to suggest injury.
5. **Skeletal examination** involves palpating for any tenderness or deformity of the bones. Joint integrity is assessed by stressing the ligaments and noting any instability, crepitus, or pain. Any suspicion of fracture or dislocation requires radiographic examination.
- C. **Diagnostic radiology.** Plain radiographs of the injured area, including the joint above and below if the physical examination warrants it, are indicated for almost all hand trauma and should be considered in cases of hand infections, particularly in penetrating trauma. Images should include a true posteroanterior and lateral and, if the injury involves the digits, separate laterals of the involved digits as well. Oblique views are used to assess displacement of articular fractures. The description of the fracture pattern should include the following: simple versus comminuted, displaced versus nondisplaced, transverse versus oblique versus spiral, angulation of the distal fragment, and intraarticular versus extraarticular. Fractures in children involving the growth plate use the **Salter-Harris classification** ([Table 35-7](#)).

Table 35-7. The Salter-Harris classification of epiphyseal fractures

II. Fractures

- A. **Principles of management**
 1. **Reduction of displaced fractures** can most often be done in the emergency room using local or regional anesthesia. However, early referral to a hand surgeon is essential for all hand fractures.
 2. **Postreduction radiographs** should be done for all fractures while in the splint or cast.
 3. **Splinting the fracture** in a position that does not impair function during the healing phase is imperative. A splint made of plaster or fiberglass with appropriate padding and the hand in "intrinsic-plus" position may be used for almost all hand injuries. This places ligamentous structures in their longest position and minimizes stiffness should immobilization be required to treat the fracture. The interphalangeal (IP) joints are in full extension, metacarpal-phalangeal (MCP) joints at 60–90 degrees of flexion, and the wrist at 20–30 degrees of extension. Individual digits can be splinted without involving the remainder of the patient's hand and wrist. A thumb spica splint is used for fractures that involve the thumb and are proximal to the IP joint. The MCP joint is placed in extension, the thumb abducted, and the wrist placed in 20–30 degrees of extension. Even if operative management of the fracture is planned, a reduction with splinting in the emergency room is still appropriate for patient comfort and to prevent stiffness.
 4. **Early motion** is used whenever possible to minimize joint stiffness.
 5. **Operative intervention** is considered if the treatment does not obtain or maintain reduction of the fracture. Contaminated open fractures, associated soft-tissue injuries, malalignment (uncorrected rotated, angulated, or shortened deformities of the digit), and articular incongruity of greater than 1 mm are also indications for operative management.
- B. **Specific fractures**
 1. **Phalangeal fractures** require closed reduction and protective splinting for 4–6 weeks. Fractures of the distal phalanx may involve the insertion of either the flexor or extensor mechanisms. Disruption of the extensor mechanism at the distal phalanx results in a mallet finger deformity (see Hand Surgery, [section IV.B.1](#)) and can be treated by splinting the DIP joint in extension. Other fractures of the distal phalanx can generally be treated with a protective splint. Stable middle and proximal phalanx fractures can be adequately treated by taping the injured finger to its neighbor. Certain fracture patterns are considered unstable and require operative fixation. As always, the goal of early motion is desirable.
 2. **Boxer's fracture** describes a common transverse fracture at the neck of the ring or small finger metacarpal with volar angulation of the distal fragment. Any rotation or scissoring of the finger must be corrected by reduction. Volar angulation of the distal fragment of up to 40 degrees is acceptable in the fifth metacarpal because of its mobility, although this may cause prominence of the metacarpal head in the palm. Less angulation is accepted in the fourth metacarpal, and in the second and third metacarpals, angulation greater than 10 degrees is unacceptable. It is unnecessary to immobilize the MCP joint, and protection with a volar splint brought to the middle palmar crease is used until the patient sees a hand surgeon. Buddy taping of the ring and small fingers may be helpful in addition.
 3. **Transverse metacarpal shaft fractures** are caused by axial loading and follow the same guidelines as neck fractures in terms of angulation. Oblique and spiral fractures result from torsional forces and are often best treated with operative fixation, protective splinting, and early range-of-motion exercises.
 4. **Bennett's fracture** is an intraarticular fracture at the base of the first metacarpal resulting from an axial load to the thumb. The distal fragment displaces proximally through the pull of the abductor pollicis longus and angulates volarly through the adductor pollicis force. The ulnar fragment of the base is held fixed by the volar beak ligament. Closed reduction and splinting often yield a reduction that is anatomic; however, the deforming forces usually move the fragments out of reduction, and these fractures are best treated with reduction and fixation. Less common is the "baby Bennett's," or "reverse Bennett's," fracture of the fifth metacarpal base; it is similar to Bennett's, with the extensor carpi ulnaris representing the deforming force on the distal fragment.

5. **Epiphyseal fractures in children** can lead to alterations of growth of the involved bone. Treatment is similar to that for adults, although healing is often faster and immobilization is more acceptable because joint stiffness is less of a problem in children. Although reduction of the fracture is important, bone remodeling allows for angulation deformities of up to 20 or 30 degrees in the phalanges and metacarpals, provided it is in the anteroposterior plane. Rotatory deformity or deviation in the coronal plane should not be accepted because remodeling does not correct these deformities (*Clin Orthop* 188:12, 1984).
6. **Open fractures** require adequate irrigation, reduction, and fixation as necessary, with prophylactic antibiotic coverage.

III. Dislocations and ligament injuries

- A. **Principles of management** include pre- and postreduction films to confirm joint alignment and look for associated fractures. After reduction, the joint should be assessed for stability by stressing the periarticular structures and putting the joint through its range of motion. If either procedure demonstrates instability, operative management should be considered. A stable joint is managed with protective splinting and early range-of-motion exercise.
 1. **DIP joint and thumb IP joint dislocations** are uncommon injuries that are treated with closed reduction followed by splinting for 3 weeks with early protective range-of-motion exercise.
 2. **PIP joint injuries** require careful assessment and follow-up to prevent long-term stiffness.
 - a. **Dislocations usually occur dorsally or laterally**, although volar dislocations are also described. Closed reduction is accomplished by placing the joint in flexion to allow relaxation of the lateral bands. Stable injuries are treated with extension block splinting for 3 weeks to allow range of motion. If the joint cannot be reduced closed or instability does not allow for protective range of motion, operative management is indicated.
 - b. **Volar plate injuries** are common and result from hyperextension of the PIP joint. The ligament can be strained, ruptured, or avulsed from the base of the middle phalanx with or without a bone fragment. If the injury is to soft tissue only or the avulsion represents less than 20% of the articular surface with a stable joint, treatment involves buddy taping or extension block splinting with the joint in 30 degrees and immediate range of motion. If the bone fragment represents 20% or more of the articular surface with associated instability, open reduction and internal fixation are required.
 3. **MCP joint dislocations** are usually caused by hyperextension injuries and are most often seen in the index and small fingers. The dislocation is usually dorsal and is usually reducible in the emergency room. If the volar plate is interposed in the joint, however, open reduction may be required. If the joint is stable after reduction, it should be splinted for protection and early motion started. Occasionally, the metacarpal head can be held volarly by the flexor tendons on one side and the intrinsic muscles on the other side such that longitudinal traction tightens the “noose” around the head and prevents reduction. Open reduction is required in these situations.
 4. **The thumb MCP joint** is commonly injured but infrequently dislocated. The dislocation is usually dorsal and results from hyperextension forces. Closed reduction with a thumb spica splint and early range of motion is the usual treatment. More commonly, the ulnar collateral ligament (**skier's thumb**) is injured during a forced radial abduction. The ligament is partially or completely torn and may avulse with a bone fragment from the proximal phalanx. If there is joint stability and congruity, the MCP joint is splinted for 6–8 weeks, leaving the IP joint free. If the joint is unstable or the proximal portion of the torn ulnar collateral ligament is displaced superficial to the adductor pollicis (Stener's lesion), open reduction and internal fixation are required. Of note, a stable lesion may be converted to an unstable (Stener's) lesion by inexperienced examiners aggressively stressing the joint.
 5. **Carpometacarpal injuries** are usually dislocations with or without fractures. Ligamentous injuries are less common because the carpometacarpal articulation has less movement than do other joints. Dorsal dislocations with and without fractures result from a direct blow and are more common on the ulnar part of the hand. Closed reduction is frequently possible, but maintaining the reduction often requires percutaneous pinning of the joint.

IV. Tendon injuries

- A. **Flexor tendons** are frequently lacerated during everyday activities, and assessment and management of these injuries are critical to a satisfactory outcome.
 1. **The assessment** involves a careful history and examination, looking for a change in the resting tone of the digits and assessing the profundus and superficialis tendons independently. If flexion against resistance elicits pain, a partial laceration must be suspected. Careful neurovascular examination, including evaluation of two-point discrimination, often reveals a concomitant digital nerve injury.
 2. **Emergency room management** involves irrigation and closure of the wound, dorsal splinting with the patient's wrist in 20–30 degrees of flexion, MCP joint at 90 degrees of flexion, and the IP joints in extension. Operative exploration and repair are appropriate for all lacerations greater than 75% of the cross-sectional area of the tendon.
 3. **Anatomy**
 - a. **Zone I.** From the FDP insertion on the distal phalanx to the FDS insertion; in the thumb, from the flexor pollicis longus insertion to the IP joint
 - b. **Zone II.** From the FDS insertion to the A1 pulley; in the thumb, from the IP joint to the MCP joint
 - c. **Zone III.** From the proximal edge of the A1 pulley to the distal edge of the carpal tunnel
 - d. **Zone IV.** Within the carpal tunnel
 - e. **Zone V.** Proximal to the carpal tunnel
 4. **Technique of repair** involves a core, locking suture and an epitendinous repair. For tendon ruptures and lacerations within 1 cm of the FDP insertion, advancement and reinsertion of the tendon are used. A dorsal splint is applied as outlined, and a strict range-of-motion protocol directed by a hand therapist is started within 24–72 hours after repair and continues for 6–8 weeks.
- B. **Extensor tendon injuries** result from lacerations and closed, axial loading of the digits.
 1. **Zone I: over the DIP joint.** *Mallet finger* is the term used to describe this very common injury that frequently occurs from closed, forced flexion of the tip of the finger and rupture of the terminal tendon from the distal phalanx. These injuries can be treated with splinting of the DIP joint in extension for 6 weeks. Mallet finger can be associated with an avulsion fracture or joint subluxation. Operative management with reduction of the fracture and joint is only occasionally indicated. For open injuries, the tendon should be repaired and the joint pinned or splinted in extension for 6 weeks.
 2. **Zone II: over the middle phalanx.** Lacerations in this zone should be repaired using a figure-of-eight or mattress technique. The DIP joint may be transfixed with a pin or splinted for 4–6 weeks.
 3. **Zone 3: over the PIP joint.** If untreated, these injuries can result in a boutonnière deformity (PIP flexion and DIP hyperextension) of the digit. Closed injuries can have an associated avulsion fracture from the dorsal aspect of the base of the middle phalanx and should be initially treated with “intrinsic-plus” splinting. For open injuries, the tendon should be repaired and the joint transfixed with an oblique pin for 3–5 weeks. For tendon injuries associated with a fracture that is displaced, reduction and fixation of the fracture are advised. Protective splinting of the joint should be maintained for 6 weeks.
 4. **Zone 4: over the proximal phalanx.** The lacerations are often partial because of the width of the tendon at this level. Splinting of the PIP joint in extension for 3–4 weeks is often sufficient for these injuries. Repair of the tendon is required if there is any extension lag of the IP joints.
 5. **Zone 5: over the MCP joint.** These injuries often occur as a result of a fistfight, particularly with a blow to the mouth, or human bite. Contamination of the wound with oral flora can produce serious infection (see Hand Surgery, [section VI.B.5](#)). Aggressive wound exploration must be undertaken to rule out joint space involvement, as intraarticular infection can rapidly destroy the delicate cartilaginous surfaces. This often requires elongation of the laceration for adequate visualization and irrigation of the full extent of the wound. Only after the full extent of the wound has been evaluated and aggressively cleansed can the tendon or tendons be repaired and the joint splinted in 20–30 degrees of flexion. The wrist is splinted in 30 degrees of extension. Dynamic splinting is useful to avoid adhesions and improve early motion.
 6. **Zone 6: over the dorsum of the metacarpals and carpus.** Repair and splint as for zone 5 injuries.
 7. **Zone 7: at the level of the extensor retinaculum.** Repair and splint as for zone 5.
 8. **Zone 8: proximal to the extensor retinaculum.** Injury is often at the musculotendinous junction. Repair and splinting for 4–6 weeks are required.
 9. **Zone 1 and 2 thumb.** Closed injuries are rare, but splinting may be used as with the finger. Open injuries should be repaired with or without joint pinning and splinting.
 10. **Zone 5–7 thumb.** (Zones 3 and 4 are excluded for the thumb.) These zones involve the extensor pollicis longus and extensor pollicis brevis tendons, which should both be repaired when lacerated. The patient's wrist is splinted in 40 degrees of extension and the MP and IP joints in full extension for 3–4 weeks.
 11. **Zone 8 thumb.** Treat as for zone 8 of the fingers.

V. Amputation

- A. **Replantation or revascularization**
 1. **Indications** for replantation include amputation of the thumb; amputation of multiple digits; amputation at the metacarpal, wrist, or forearm level; or amputation at any level in a child. More controversial indications include amputation of the proximal arm and amputation of a single digit distal to the FDS insertion.
 2. **Contraindications** for replantation include multiple levels of amputation, severe crush or degloving injury to the part, prolonged ischemia time (12 hours for fingers and 6 hours for proximal limb amputations), and associated life-threatening injuries. Ring avulsion injury is a relative contraindication to replantation because of the extensive vascular and soft-tissue trauma.
 3. **Preparation for transfer** involves a moist dressing on the stump and splinting for comfort. The amputated part should be wrapped in saline-moistened gauze and placed in a clear plastic bag on a mixture of ice and water. The part should never be placed directly on ice or immersed in saline. Radiographs of the

stump and the amputated part are essential and can be done at the transferring facility, provided that this does not significantly delay transfer to the microsurgery center. Intravenous fluids, prophylactic antibiotics, and tetanus toxoid, when indicated, should be begun immediately to facilitate prompt transfer to the appropriate facility for replantation. The sequence of repair involves identification of neurovascular structures and tendons with preparation of the bone for fixation. After providing bony stability, the tendons are repaired, followed by repair of the arteries and then veins, nerves, and skin. The postoperative care involves careful monitoring of the splinted part (temperature, color, and turgor) and adequate intravenous hydration in a warm environment.

B. Amputations not suitable for replantation

1. As mentioned above (see Hand Surgery, [section V.A.2](#)), severely crushed or degloved tissues and tissues with prolonged ischemia times should not be replanted. Management of these injuries involves the following:
 - a. **Complete assessment, including radiographs**
 - b. **Antibiotics when bone is involved or soft tissues are crushed or contaminated**
 - c. **Preservation of length**
 - d. **Maintain sensation and motion**
 - e. **Esthetics**
 - f. **Early motion**
2. **Fingertip injuries** are optimally managed using primary closure without shortening. If this is not possible, lateral V-Y advancement or volar advancement flaps or skin grafts can be used to obtain closure. An alternative for small wounds is closure by secondary intention.
3. **Principles of more proximal amputations** involve shortening and contouring the bone, shortening the tendons, and identification of digital nerves and allowing them to be transposed away from the skin closure.
4. **A protective dressing** that allows joint motion is recommended, with early referral to a hand therapist for range-of-motion exercises and later desensitization of the tip of the stump.

VI. Infections

A. **Management.** Infections in the hand can progress rapidly via potential spaces and may risk the viability of tendons, bones, joints, and neurovascular structures by creating increased pressure from pus and edema in closed spaces.

1. **Surgical drainage** is required in most hand infections.
2. **Antibiotic coverage** should be directed against common skin flora such as *Staphylococcus aureus*, *Staphylococcus epidermidis*, or *Streptococcus* species.
3. **Gram stain and culture of wound.**
4. **Splinting and elevation of the hand.**
5. **Tetanus prophylaxis when appropriate.**

B. Local infections

1. **Paronychia** is a soft-tissue infection of the skin and soft tissue of the lateral nail fold; an eponychial infection may extend from this and involves the proximal nail fold. These localized infections often arise from self-inflicted trauma by nail biting or foreign body penetration, such as a needle-stick. Treatment requires incision and drainage with removal of the nail when the infection extends deep to the nail plate. Oral antibiotics are used if a cellulitis is present. **Chronic paronychia** can be associated with underlying osteomyelitis or fungal organisms.
2. **Felon** is a localized infection involving the pulp of the volar digit and usually originates with a puncture wound, although a paronychia infection may spread volarly. Purulent fluid is usually under pressure in the fibrous septa of the tip of the digit. Management involves incision and drainage of the abscess and systemic antibiotics if there is an associated cellulitis. In general, the incision is located where the felon is “pointing”; however, it should be carefully planned to avoid sensitive scars and destabilization of the pulp of the finger. As with a paronychia, aggressive cleansing with soap and water after incision and drainage promotes drainage and avoids premature closing of the wound.
3. **Cellulitis** in the hand usually occurs secondary to a laceration, abrasion, or other soft-tissue injury. Management involves draining an abscess if present. The fluid should be sent for culture and sensitivity, and oral or intravenous antibiotics should be administered, depending on the severity. When associated with swelling of the digits and hand, splinting in the “intrinsic-plus” position and elevation prevent stiffness.
4. **When cellulitis occurs after an animal bite**, the wound must be thoroughly irrigated to decrease the bacterial load and to remove any foreign body, such as a tooth. Bite wounds should be treated with oral antibiotics prophylactically and with intravenous antibiotics when an established infection is present. Although a greater percentage of cat bites become infected than do dog bites, the jaws of a dog are significantly more powerful and can inflict other injuries. The organisms most often involved from dog or cat bites include *Pasteurella multocida*, *S. aureus*, *Bacteroides* species, and *Streptococcus viridans*. Recommended oral antibiotics are amoxicillin-clavulanate or clindamycin with either ciprofloxacin or trimethoprim-sulfamethoxazole.
5. **Human bites** can involve particularly virulent organisms and frequently present in association with extensor tendon injuries or fractures sustained during physical altercations. An open wound, particularly if it overlies the dorsum of the hand with signs of infection or underlying soft-tissue or bony injuries, should prompt patient questioning about the source of the laceration. Typical organisms cultured from human bite wounds are *S. viridans*, *S. epidermidis*, and *S. aureus*, as well as anaerobic bacteria, such as *Eikenella corrodens* and *Bacteroides* species. Amoxicillin-clavulanate should be used prophylactically, and when signs of infection are present, treatment with ampicillin-sulbactam, cefoxitin, or clindamycin plus either ciprofloxacin or trimethoprim-sulfamethoxazole is recommended. The wound should be explored carefully and extensor tendon injuries should be repaired with a 3-0 or 4-0 nonabsorbable suture. During exploration, it is imperative to assess the wound with the patient's digits in extension and flexion to determine if the underlying joint has been violated. With the patient's hand in a fist position, the MCP joint is in flexion, and the sagittal bands are pulled distally over the metacarpal head. In extension (usual position for exploration), the sagittal bands move proximally and can obscure the tear in the joint capsule, through which the joint space was inoculated. Septic arthritis may ensue. If violated, the joint must be thoroughly irrigated with saline and the articular surface examined for chondral injuries. If a loose osteochondral fragment is present, it must be removed to avoid sequestrum formation; the bone should be curetted carefully to decrease the bacterial load. The joint and wound should be left open, the patient's hand should be splinted appropriately, and routine dressing changes and frequent assessments should be made to ensure resolution of the infection.
6. **Suppurative tenosynovitis and palmar space abscess** (see Hand Surgery, [section VII.B](#)).
7. **Necrotizing infections** (see Hand Surgery, [section VII.D](#)).

VII. Surgical emergencies

A. **Compartment syndrome** is seen in the hand and forearm as a group of signs and symptoms that result from increased pressure within an osseofascial space, causing compromise of the circulation and decreased perfusion pressure. If left untreated, the muscle and nerve ischemia progress to necrosis and fibrosis, causing Volkmann's ischemic contracture.

1. **Etiology.** Fractures that cause bleeding, crush and vascular injuries, circumferential burns, bleeding dyscrasias, reperfusion after ischemia, or tight dressings can lead to the syndrome.
2. **Diagnosis** is based on a high index of suspicion, clinical examination, and symptoms of pain that are exacerbated with passive stretch of the compartment musculature, paresthesias, paralysis, or paresis of ischemic muscles. Swelling, tenderness, and tightness are often present. Pulselessness may occur and indicates a late finding (usually indicating irreversible damage) or the presence of major arterial occlusion rather than compartment syndrome. A decrease in vibration threshold corresponds with compartment syndrome pressures. Measurement of a compartment pressure of greater than 30 mm Hg confirms diagnosis.
3. **Treatment** for incipient compartment syndrome involves close observation and frequent examinations and should include removal of tight casts and dressings. Elevation of the extremity to, or slightly above, the level of the heart is recommended. Acute or suspected compartment syndrome requires urgent fasciotomies of the involved areas. Decompression within 6 hours of established compartment pressures is necessary to prevent irreversible muscle ischemia. Forearm fasciotomies involve volar, carpal tunnel, and dorsal compartments. Hand fasciotomies include dorsal incisions for interossei and adductor pollicis, thenar, and hypothenar compartments, as well as midaxial incisions of the digits (ulnar for index, long and ring and radial for thumb and small).

B. **Suppurative tenosynovitis** involves infection of the flexor tendon sheath, which is usually caused by a puncture wound to the volar aspect of the digit or palm.

1. **Cardinal signs of Kanavel**
 - a. **Finger held in flexion**
 - b. **Fusiform swelling of the finger**
 - c. **Tenderness along the tendon sheath**
 - d. **Pain on passive extension**
2. **Management** involves urgent incision and drainage in the operating room, with placement of an irrigating catheter, such as a pediatric feeding tube, in the sheath for continuous irrigation with saline. Irrigation is maintained for 24–48 hours. Intravenous antibiotics are administered. Frequent reassessment to verify resolution is critical to avoiding ischemic injury to the tendon secondary to the contained infection.

C. **Palmar abscess** is usually associated with a puncture wound. The fascia divides the palm into thenar, midpalmar, and hypothenar spaces; each involved space

must be incised and drained. As with other infections, splinting, elevation, and intravenous antibiotics are required.

- D. **Necrotizing infections** threaten both limb and life. The incidence of invasive group A streptococcal infection is on the rise (*N Engl J Med* 335:547, 1996) and can occur after surgery or trauma. Aggressive surgical débridement, high-dose penicillin, and supportive management are the mainstays of treatment. Additional therapy with gentamycin or clindamycin provides antibacterial synergy and blocks production of bacterial toxins. Immune globulin and hyperbaric oxygen are adjuvant therapy.
- E. **High-pressure injection injuries** result from grease or paint injected at up to 10,000 pounds/square inch. Although the external wounds are often small and unassuming, deep tissue injury can be severe. Injury to the tissue is the result of both direct physical damage and chemical toxicity, and it leads to edema, thrombosis, and subsequent infection. Management involves urgent, thorough débridement, irrigation, decompression, systemic antibiotics, splinting, and frequent reassessments, with repeat débridement in 24 hours as required. When a digit has sustained significant injection, amputation may be required.

Cardiac Surgical Procedures

I. Coronary artery disease

II. Valvular heart disease

III. Perioperative care

IV. Cardiac transplantation

V. Diseases of the thoracic aorta

VI. Circulatory support

Cardiac Surgical Procedures

This chapter focuses on the perioperative care of the adult patient undergoing common cardiac surgical procedures, particularly coronary artery bypass grafting (CABG) and valve repair and replacement.

I. Coronary artery disease (CAD; atherosclerosis of the coronary arteries) is the leading cause of death in adults in North America. Risk factors for CAD include cigarette smoking, hypertension, diabetes mellitus, hyperlipidemia, male gender, obesity, advanced age, and a family history of CAD at a young age.

- A. **Coronary circulation.** The left main coronary artery branches to form the left anterior descending (LAD) and circumflex coronary arteries and their respective branches, the diagonal and obtuse marginal branches. The right coronary artery supplies the right ventricle. In 80–90% of patients, the posterior descending artery branch is a branch of the right coronary artery (a situation termed *right dominance*). In the balance of individuals, the posterior descending is the terminal branch of the circumflex (left dominance). Less commonly, there is no single posterior descending branch but, rather, multiple terminal branches of both the right and left circulations (balanced circulation). The coronary sinus, thebesian veins, and anterior cardiac veins drain the coronary circulation. Myocardial blood flow to the left ventricle occurs primarily during diastole, whereas that to the right ventricle occurs throughout the cardiac cycle and is accordingly more dependent on systolic arterial pressure. Oxygen extraction by the heart is maximal at rest, and increased oxygen demand must be met by increased delivery.
- B. The **clinical presentation** of CAD is determined by the distribution of the atherosclerotic lesions, the severity of stenosis, the level of myocardial oxygen demand, and the relative acuity or chronicity of the oxygen supply-demand mismatch. The three most common presentations for patients with CAD are angina pectoris, myocardial infarction (MI), and chronic ischemic cardiomyopathy.
 1. **Angina pectoris** is a symptom complex resulting from reversible myocardial ischemia without cellular necrosis. Patients typically complain of retrosternal chest pain or pressure that often radiates over the left shoulder and down the left arm or into the neck. Stable angina occurs with reproducible increases in myocardial oxygen demand (e.g., exercise) and resolves with rest or the administration of nitrates. *Unstable angina* refers to chest pain that occurs at rest or episodes of pain that are increasing in frequency, duration, or severity. Silent myocardial ischemia occurs when there is electrocardiographic (ECG) evidence of myocardial ischemia in the absence of any angina or angina-equivalent symptoms. The extent to which the patient's activity is limited can be graded according to the New York Heart Association classification: class I, no symptoms; class II, symptoms with heavy exertion; class III, symptoms with mild exertion; class IV, symptoms at rest.
 2. **Acute MI** results from interruption of myocardial oxygen supply with irreversible muscle injury and cell death. The patient typically presents with protracted and severe chest pain that may be associated with nausea, diaphoresis, or shortness of breath. There are increases in the troponin isoenzyme, creatine kinase-MB isoenzyme, or serum lactate dehydrogenase. ECG changes include ST-segment elevation, T-wave inversions, and the development of new Q waves. Early and late sequelae of acute MI can include atrial or ventricular arrhythmias, heart failure, rupture of the interventricular septum or ventricular free wall, dysfunction or rupture of the papillary muscle(s) with new mitral regurgitation (MR), or the development of a ventricular aneurysm.
 - a. **Arrhythmias** are common during the first 24 hours after acute MI. In addition to potentially fatal ventricular arrhythmias, patients can develop supraventricular tachycardia, atrial fibrillation, atrial flutter, heart block of any degree, or junctional rhythms. Specific therapies for these arrhythmias are outlined in [Chapter 3](#).
 - b. **Congestive heart failure** may result when a large portion (usually $\geq 25\%$) of the left ventricular myocardium is infarcted. Cardiogenic shock and death often occur with loss of more than 40% of the left ventricular myocardium.
 - c. **Rupture of the interventricular septum** occurs in approximately 2% of patients after MI (usually anterior wall in 60%, inferior wall in 40%) and leads to a ventricular septal defect (VSD). Septal perforation typically occurs when the myocardium is at its weakest, approximately 3–5 days after an acute MI, but it may develop 2 or more weeks later. Acute VSD is suggested by a new holosystolic murmur and an oxygen step-up from right atrium to pulmonary artery, as evaluated with a pulmonary artery catheter. This is determined by comparing the oxygen saturation of samples drawn simultaneously from the central venous port and the distal pulmonary artery port. A step-up of greater than 9% is generally held to be diagnostic of a left-to-right shunt. The diagnosis can be confirmed with echocardiography. More than 75% of patients survive the initial event and are candidates for urgent surgical repair of the VSD before the patients develop the sequelae of low output syndrome (i.e., multiorgan system failure), which greatly increases the operative risk. An intraaortic balloon pump (IABP) is indicated to support the failing circulation until surgical correction is possible. Ventricular free-wall rupture results in hemopericardium and cardiac tamponade that usually is fatal. For those patients who survive, emergent surgical repair is indicated.
 - d. **Acute MR** is caused by papillary muscle dysfunction or rupture after an infarction that has extended into the region of the papillary muscles (usually posteroinferior wall). The failing circulation should be supported with an IABP or percutaneous cardiopulmonary bypass (CPB), if necessary, until emergent operation can be performed.
 - e. **Ventricular aneurysm**, a well-defined fibrous scar that replaces the normal myocardium, develops in 5–10% of individuals after acute MI. The majority of aneurysms develop at the anteroseptal aspect of the left ventricle after infarction in the distribution of the LAD coronary artery. Large dyskinetic left ventricular aneurysms can reduce the left ventricular ejection fraction substantially, resulting in signs and symptoms of congestive heart failure. These scars can also serve as the substrate for ischemic reentrant ventricular arrhythmias. Additionally, the pooled blood that collects in the aneurysm can clot and shower emboli into the peripheral circulation.
 3. **Chronic ischemic cardiomyopathy** can develop after several MIs. Diffuse myocardial injury results in diminishing ventricular function and, eventually, signs and symptoms of heart failure. This presentation is most common in patients with diffuse small-vessel disease (e.g., in patients with diabetes mellitus).
- C. **Diagnostic tests**
 1. **ECG changes** can indicate the presence of CAD. The presence of ST-segment elevation or depression or T-wave inversion suggests ongoing ischemia or MI, whereas Q waves or loss of R-wave progression indicate previous MI.
 2. **Stress testing** is used to detect CAD or to assess the functional significance of coronary lesions. The exercise ECG is used to evaluate patients who have symptoms suggestive of angina but no symptoms at rest. A positive test is the development of typical signs or symptoms of angina pectoris associated with ECG changes (ST-segment changes or T-wave inversion). Accuracy is reduced in patients with an abnormal baseline ECG and may be improved if the test is combined with the administration of thallium. Thallium imaging is used to identify ischemic myocardium. The thallium in the blood is taken up in the normal myocytes in proportion to the regional blood flow. Decreased perfusion to a region of the myocardium during exertion with subsequent reperfusion suggests reversible myocardial ischemia, whereas the lack of reperfusion suggests irreversibly scarred, nonviable myocardium. In patients who cannot exercise, thallium imaging can be performed after administration of the coronary vasodilator dipyridamole.
 3. **Echocardiography** evaluates valve function, ventricular wall function, and ventricular ejection fraction.
 4. **Dobutamine stress echocardiography** evaluates regional ventricular function under resting and stress conditions, providing information about recruitable myocardium and myocardial ischemia.
 5. **Coronary arteriography** is used to document the presence and location of coronary artery stenoses. Separate injections are made of the right and left main coronary arteries. In general, the atherosclerotic process involves the proximal portions of the major coronary arteries, particularly at or just beyond branch points. Concomitant ventriculography provides an assessment of left ventricular function. A 75% decrease in cross-sectional area (50% decrease in luminal diameter) is considered a significant stenosis. Indications for coronary arteriography include suspected CAD (e.g., positive stress test), preparation for coronary revascularization, typical or atypical clinical presentations with normal or borderline stress testing when a definitive diagnosis of CAD is needed, and planned cardiac surgery (e.g., valve surgery) in patients with risk factors for CAD.
- D. **Coronary revascularization** may be accomplished via percutaneous transluminal coronary angioplasty (PTCA) or CABG. Indications depend on the patient but generally include intractable symptoms and proximal coronary stenoses that place a large portion of myocardium at risk.
 1. **PTCA** is often used for focal symmetric stenoses in proximal coronary vessels. It is generally contraindicated if there is significant left main coronary disease, multiple significant stenoses in the same vessel, or complex obstructive lesions. PTCA is associated with a high incidence of restenosis, which may be

reduced with the concomitant placement of an endoluminal stent.

2. **CABG** is indicated for patients with documented atherosclerotic CAD in several settings: (1) patients with unstable angina for whom maximal medical therapy has failed; (2) patients with severe chronic stable angina who have multivessel disease or left main or proximal LAD stenoses; (3) patients with severe, reversible left ventricular dysfunction (documented by stress thallium scan or dobutamine echocardiography); (4) patients who develop coronary occlusive complications during PTCA or other endovascular interventions; and (5) patients who develop life-threatening complications after acute MI, including VSD, ventricular free-wall rupture, or acute MR.

II. Valvular heart disease

A. Pathophysiology and clinical presentation

1. **Aortic stenosis (AS)**. Left ventricular outflow obstruction can occur at the subvalvular, the supra-annular, or (most commonly) the valvular level. Aortic valvular stenosis is usually the result of degeneration and calcification of a normal or congenitally bicuspid aortic valve. Less frequently, AS develops many years after an episode of acute rheumatic fever. AS places a pressure overload on the left ventricle. Adequate cardiac output is usually maintained until late in the course of AS but at the expense of left ventricular hypertrophy. Physical signs include a systolic ejection murmur, diminished carotid pulses, and a sustained, forceful, nondisplaced apical impulse. Symptoms often develop when the valve area decreases to 1.0 cm^2 or less.
 2. **Angina pectoris** develops in approximately 65% of patients with severe AS and results from ventricular hypertrophy (e.g., increased myocardial oxygen demand and reduced coronary perfusion) and the high incidence of concomitant CAD. Syncope (25% incidence) probably results from fixed cardiac output and decreased cerebral perfusion during systemic vasodilatation. Dyspnea usually represents the development of congestive heart failure. The development of symptoms is associated with a 50% 2-year mortality (*Am Heart J* 99:419, 1980).
 3. **Aortic insufficiency (AI)** is usually the result of valve leaflet pathology from rheumatic heart disease (often associated with mitral valve disease) or myxomatous degeneration. AI may also result from other causes of leaflet dysfunction or aortic root dilatation, including endocarditis, syphilis, connective tissue diseases (e.g., Marfan's syndrome), inflammatory disease (e.g., ankylosing spondylitis), hypertension, and aortic dissection. Chronic AI results in volume overload of the left ventricle, causing chamber enlargement and wall thickening (although a relatively normal ratio of wall thickness to volume is usually maintained). Gradual myocardial decompensation often progresses either without symptoms or with subtle symptoms (e.g., weakness, fatigue, or dyspnea on exertion). Physical signs include a hyperdynamic circulation with markedly increased systemic arterial pulse pressure, known as *Corrigan's water-hammer pulse*; forceful and laterally displaced apical impulse; and a decrescendo diastolic murmur. Acute AI is not well tolerated because of the lack of compensatory chamber enlargement and thus often results in fulminant pulmonary edema, myocardial ischemia, and cardiovascular collapse.
 4. **Mitral stenosis (MS)** is caused by rheumatic fever in almost all cases. Other less common causes include collagen vascular diseases, amyloidosis, and congenital stenosis. MS places a pressure overload on the left atrium with relative sparing of ventricular function. Left atrial dilatation to more than 45 mm is associated with a high incidence of atrial fibrillation and subsequent thromboembolism. A transvalvular pressure gradient is present with a valve area of less than 2 cm^2 , and critical MS occurs when the valve area is 1 cm^2 or less. Physical signs include an apical diastolic murmur, an opening snap, and a loud S_1 . Symptoms usually develop late and reflect pulmonary congestion (e.g., dyspnea), reduced left ventricular preload (e.g., low-cardiac-output syndrome), or atrial fibrillation (e.g., thromboembolism).
 5. **MR** results from abnormalities of the leaflets (e.g., rheumatic disease, myxomatous degeneration, endocarditis), annulus (e.g., calcification, dilatation, or destruction), chordae tendineae (e.g., rupture from endocarditis or MI, fusion, or elongation), or ischemic papillary muscle dysfunction or rupture. The most common cause of MR in the United States is myxomatous degeneration. MR places a volume overload on the left ventricle and atrium, causing chamber enlargement and wall thickening, although a relatively normal ratio of wall thickness to volume is usually maintained. Systolic unloading into the compliant left atrium allows enhanced emptying of the left ventricle during systole with only slight increases in oxygen consumption. Atrial fibrillation often develops because of left atrial dilatation. Physical signs include a hyperdynamic circulation with a brisk laterally displaced apical impulse, a holosystolic murmur, and a widely split S_2 . Gradual myocardial decompensation often progresses in the absence of symptoms (e.g., dyspnea on exertion, fatigue). In acute MR, adaptation is not possible, and fulminant cardiac decompensation often ensues.
 6. **Tricuspid insufficiency (TI)** most often results from a functional dilatation of the valve annulus caused by pulmonary hypertension, which, in turn, may be caused by intrinsic mitral or aortic valve disease. Causes of primary TI include rheumatic heart disease, bacterial endocarditis (usually in intravenous drug users), carcinoid tumors, and blunt trauma. Patients have a systolic murmur, a prominent jugular venous pulse, and a pulsatile liver. Mild to moderate TI usually is well tolerated.
- B. **Diagnostic tests.** Cardiac catheterization or echocardiography can be used to estimate valve areas, transvalvular pressure gradients, and the degree of regurgitant flow in patients with valvular heart disease.
- C. **Indications for operation**
1. **Aortic valve replacement** is usually indicated for AS in symptomatic patients and in asymptomatic patients with critical AS (e.g., a valve area $<1.0 \text{ cm}^2$ or a transvalvular pressure gradient $\geq 50 \text{ mm Hg}$). Timing of surgery for AI is critical because irreversible myocardial dysfunction often precedes the development of symptoms. Indications for operation include symptoms or objective evidence of ventricular decompensation (e.g., ejection fraction $<50\%$, increased left ventricular end-diastolic volume and end-systolic left ventricular diameter $>55 \text{ mm}$). If there is coexistent CAD documented by coronary arteriography, patients should undergo concomitant CABG.
 2. **Mitral valve replacement or repair** is indicated for patients with symptomatic MR, new-onset atrial fibrillation, or objective evidence of left ventricular dysfunction (same as for AI). Operation is indicated in symptomatic MS or asymptomatic patients with critical MS (valve orifice $<1 \text{ cm}^2$). With the advent of mitral valve repair, operation is often undertaken earlier if the valvular anatomy suggests that the valve can be repaired rather than replaced.
 3. **Tricuspid valve.** Significant tricuspid regurgitation may be repaired at the time of surgery for other cardiac anomalies. Intervention for isolated TI is uncommon. The majority of tricuspid valves can be repaired with simple annuloplasty techniques rather than replaced. Patients with endocarditis and TI can undergo simple valve excision.
 4. **Endocarditis.** The main indications for operation include hemodynamic instability, recurrent septic emboli, and persistent evidence of infection despite appropriate antibiotic therapy. Relative indications include severe acute mitral or aortic valvular insufficiency, heart block, and intracardiac fistulas.
- D. **Selection of a prosthetic valve** must be individualized for each patient. Despite years of research, there still is no ideal prosthetic valve for all patients. The general considerations for selecting an appropriate prosthetic valve are summarized in [Table 36-1](#).

Table 36-1. Selection of an appropriate prosthetic valve

1. **Bioprostheses** are made from animal tissues, usually the porcine aortic valve. Examples include the Carpentier-Edwards, Hancock, and Edwards stented valves and the St. Jude and Medtronic stentless valves. These prostheses are associated with a low rate of thromboembolism, even without long-term anticoagulation. However, they are less durable than mechanical valves. Their rate of deterioration depends on age, being relatively faster in younger patients and slower in the elderly. Overall, the mean time to failure is approximately 13 years. Bioprostheses are the preferred valves for older patients or patients with a contraindication to lifelong anticoagulation (e.g., young women who desire future pregnancies).
2. **Mechanical valves** are associated with excellent long-term durability, but the high rate of thromboembolic complications (0.5–3.0% per year) necessitates lifelong anticoagulation. Examples include the St. Jude, Medtronic-Hall, CarboMedics, Starr-Edwards, and Björk-Shiley valves. These valves usually are used in young patients who have a long life expectancy.
3. **Allograft and autograft valves** are useful for replacement of the aortic valve, particularly in the setting of endocarditis (*Circulation* 75:768, 1987). These prostheses have excellent durability and a low incidence of thromboembolism, but experience with them is limited by the supply of allografts and the relative difficulty of the autograft (Ross) procedure, in which a patient's pulmonic valve is used to replace the diseased aortic valve.

III. Perioperative care

- A. **Preoperative evaluation of the cardiac surgery patient.** The preoperative evaluation of patients undergoing cardiac surgery is similar to the evaluation of patients undergoing any major operation. All patients should have a complete history and physical examination. Laboratory studies usually include a complete blood cell count and determination of the serum electrolytes, creatinine, and glucose levels; prothrombin (PT) and partial thromboplastin times (PTT); and urinalysis. An arterial blood gas is indicated in patients with chronic obstructive pulmonary disease, heavy tobacco abuse, or other pulmonary pathology. In general, 2–4 units of packed red blood cells should be available for use during operation. For elective operations, this may be predated autologous blood. A chest radiograph (posteroanterior and lateral) should be obtained to evaluate calcification of the aortic arch and to assess the proximity of the cardiac silhouette to the sternum in patients undergoing repeat sternotomy. The chest radiograph is also examined for the presence of other intrathoracic pathology. The height

and weight of the patient should be measured, and the body-surface area (in square meters) should be calculated.

1. **Neurologic complications** can be a devastating complication after cardiac surgery. Perioperative cerebrovascular accidents (CVA) occur in 1–2% of low-risk patients but in up to 10% of the elderly. CVA may result from aortic atherosclerotic emboli that are loosened by cannulation, cross-clamping, or construction of the proximal anastomoses. Underlying cerebrovascular disease in conjunction with alterations in cerebral blood flow patterns during CPB may also play a role. Patients with a history of transient ischemic attack, amaurosis fugax, or CVA should undergo noninvasive evaluation of the carotid arteries with Doppler ultrasonography before operation. Because of the strong association between carotid artery and left main coronary stenoses, patients with left main disease should also undergo carotid Doppler examination. Evaluation of asymptomatic carotid bruits is more controversial. In general, only symptomatic carotid stenoses are addressed by carotid endarterectomy before or in combination with the planned cardiac surgical procedure. Correction of asymptomatic carotid stenoses is controversial.
 2. **Pulmonary disease**, particularly the obstructive form, occurs commonly in patients with cardiac disease because cigarette smoking is a risk factor for both disease processes. The preoperative chest X-ray may demonstrate suspicious pulmonary pathology and can be used in combination with a preoperative arterial blood gas to identify patients who are at high risk for difficulty in being weaned from the ventilator postoperatively. Smoking should be discontinued before operation.
 3. **Peripheral vascular examination.** The presence and quality of arterial pulses in the radial, brachial, femoral, popliteal, dorsalis pedis, and posterior tibial arteries should be documented preoperatively as a baseline for comparison should there be postoperative arterial complications. Blood pressure (BP) should be measured in both arms to evaluate for subclavian artery stenosis. Significant subclavian artery stenosis may preclude the use of an internal thoracic (mammary) artery as a conduit. A preoperative Allen's test should also be performed to assess the palmar arch and the feasibility of using the radial artery for bypass conduit. For obese patients and those with varicosities of the saphenous veins, preoperative vein mapping with ultrasonography can be used to assess the availability and quality of saphenous vein for conduit.
 4. **Infection.** Operation should be delayed, if possible, in patients with systemic infection or sepsis and in those with cellulitis or soft-tissue infection at the site of planned incisions. Specific infections should be identified preoperatively, if possible, and treated with appropriate antibiotic therapy. In patients with fever or leukocytosis in whom operation cannot be delayed, cultures should be obtained from all potential sources (including central venous catheters), and broad-spectrum intravenous antibiotics should be administered preoperatively.
 5. **Failed angioplasty.** Emergent operation is necessary for approximately 5% of patients with complications (vessel occlusion, dissection, or perforation) after PTCA or other endovascular interventions (*Ann Thorac Surg* 47:816, 1989). When patients are at high risk for such a complication, an operating room is held ready should emergent operation be required. There should be a detailed preoperative discussion with these patients on the possibility of emergent operation. For patients with hemodynamic instability or refractory angina after failed angioplasty, IABP support or percutaneous CPB may be helpful before emergent operation can be started.
 6. **Medications.** The cardiac surgery patient may be taking a variety of medications before operation. In general, nitrates and beta-adrenergic blocking agents should be continued throughout the entire perioperative period. If possible, antiplatelet agents (e.g., aspirin or ibuprofen) are stopped before surgery to prevent hemorrhagic complications. Digoxin and calcium channel blockers generally are discontinued at the time of operation and restarted only as needed in the postoperative period. For patients receiving heparin preoperatively for unstable angina, the heparin should not be discontinued before operation because this may precipitate an acute coronary syndrome. For patients receiving warfarin preoperatively (including patients with mechanical valves), the warfarin should be discontinued several days before operation; anticoagulation can be accomplished using intravenous heparin until several hours before elective operations.
- B. **Postoperative care** of the cardiac surgery patient is provided in three phases: the intensive care unit (ICU), the step-down unit, and after discharge from the hospital.
1. **ICU care** resources generally are required for 1–3 days after operations requiring CPB.
 - a. **Initial assessment.** Information on the patient's history, indications for operation, and technical details of operation (e.g., coronary arteries bypassed, conduits used, CPB time, and aortic cross-clamp time) should be related to the ICU staff by the surgeon. The anesthesiologist should relate information about the intraoperative course, including preoperative and intraoperative hemodynamic parameters (especially cardiac filling pressures) and current medications. A thorough physical examination should be performed with attention to the cardiovascular system. A baseline chest X-ray and ECG are usually obtained.
 - b. **Monitoring in the immediate postoperative period** is usually extensive. Continuous recordings are made of arterial, central venous, and pulmonary artery pressures; the ECG; and arterial oxygen saturation using pulse oximetry. The pulmonary artery wedge pressure is measured as indicated by the patient's status, and calculations are made of the cardiac output, cardiac index (cardiac output/unit of body-surface area), stroke volume, pulmonary vascular resistance, and systemic vascular resistance (by thermodilution technique). Normal values for these parameters are listed in [Table 36-2](#). Immediate attention is necessary to determine the etiology and to correct deviations from normal values of any of these parameters.

Table 36-2. Normal hemodynamic values

- c. **Body temperature** is monitored continuously using a pulmonary artery thermistor. Because early postoperative hypothermia may increase afterload, the systemic vascular resistance, and adversely affect blood clotting, hypothermia is treated aggressively by the application of air-warming blankets. Warming is discontinued when the core temperature reaches 36.0 °C. A cooling blanket is applied, and acetaminophen (650 mg p.o. every 4 hours) is administered if the body temperature is greater than 38.5 °C. For patients with persistent fever, an aggressive search should be made for evidence of infection.
- d. **Neurologic examination** of the patient is performed on admission to the ICU and periodically as needed. In general, the patient awakens from anesthesia several hours after operation. Changes in the neurologic examination warrant immediate investigation. Shivering increases oxygen consumption and should be treated in the early postoperative period by warming the patient or by the administration of meperidine (50–100 mg i.m. or i.v. every 3 hours) or, for the ventilated patient, pancuronium (0.04–0.10 mg/kg i.v.) or vecuronium (0.08–0.10 mg/kg slow i.v. bolus, then 1–5 mg per hour i.v. continuous infusion).
- e. **Cardiovascular**
 1. **Cardiac pump function** is assessed as described earlier. A cardiac index of 2.0 L per minute per m² is generally a minimum acceptable value. In general, patients with a palpable dorsalis pedis pulse have an acceptable cardiac index. A mixed venous oxygen saturation of less than 60% suggests inadequate peripheral tissue perfusion and increased peripheral oxygen extraction. Etiologies include reduced oxygen-carrying capacity (e.g., low hematocrit), reduced cardiac output, or increased oxygen consumption (e.g., shivering). Common causes of low cardiac output in the early postoperative period are hypovolemia and increased systemic vascular resistance due to persistent hypothermia or increased circulating catecholamines. Management of low cardiac output includes the following.
 - a. **Arrhythmias**, including bradycardia from resolving hypothermia, heart block secondary to persistent cardioplegia effect, and supraventricular tachyarrhythmias (e.g., atrial fibrillation or flutter) can be associated with reduced cardiac output and should be corrected. The heart can be paced using temporary epicardial pacing electrodes (placed at the time of operation) at 80–100 beats per minute to increase the cardiac output. Pacing can be performed using only the atrial leads (atrial pacing, or AAI mode), only the ventricular leads (ventricular pacing, or VVI mode), or with both sets of leads (atrioventricular sequential pacing or atrial tracking with ventricular pacing). If epicardial pacing is not necessary, the epicardial pacemaker generator is set in a backup mode to provide ventricular pacing only in the event of marked bradycardia. The pacemaker output threshold (in milliamperes) should be set to approximately twice the minimum threshold required to capture. The treatment of tachyarrhythmias is outlined in [Chapter 3](#).
 - b. **Preload** is increased by administering crystalloid solution (e.g., lactated Ringer's solution) or colloid solution (e.g., 6% hetastarch, 5% albumin) as needed to maintain the pulmonary arterial wedge pressure in the target range, as determined by the patient's diastolic compliance and systolic performance.
 - c. **Afterload reduction** in the volume-restored patient increases ejection fraction and cardiac output and decreases myocardial oxygen consumption. The body temperature should be returned to the normal range, and hypertension should be controlled. In general, the mean arterial BP should be maintained at approximately the preoperative level. For patients with valve replacement or aortic replacement, the systolic BP should be maintained 10–15 mm Hg below the preoperative level to prevent postoperative bleeding. Afterload is often initially titrated with parenteral infusions of sodium nitroprusside or nitroglycerin and then changed to longer-acting parenteral or enteral agents ([Table 36-3](#)).

Table 36-3. Afterload-reducing agents

- d. **Inotropic agents** are used only after ensuring an adequate preload and an appropriate afterload. Selection of a particular inotropic agent must be individualized based on the agent's specific effects on the heart rate, BP, cardiac output, systemic vascular resistance, and renal blood flow. All these agents increase the work of the heart and increase myocardial oxygen consumption and thus should be used judiciously. Dobutamine (5–15 µg/kg per minute) and dopamine (2–20 µg/kg per minute) are usually the first-line agents for left ventricular dysfunction. If cardiac output continues to be inadequate despite midrange dosages of a single inotropic agent, a second inotropic agent, usually epinephrine (0.01–0.20 µg/kg per minute), can be added. Agents such as amrinone and isoproterenol are less commonly used.
- e. **Mechanical support** in the form of an IABP or ventricular assist device (VAD) can be considered if other measures are ineffective in restoring adequate cardiac pump function (see [section VI](#)).
- f. **Cardiac tamponade** is a potentially lethal cause of low cardiac output early after operation. Clinical features include narrowed pulse pressure, rising cardiac filling pressures, pulsus paradoxus, widened mediastinal silhouette on chest radiograph, and decreased urine output. Definitive diagnosis is usually made by transthoracic or transesophageal echocardiography.
2. **Arrhythmias** occur in more than 50% of patients after cardiac surgical procedures and are more common in patients who receive inotropic support.
 - a. **Supraventricular arrhythmias** (atrial fibrillation, atrial flutter, or paroxysmal atrial tachycardia) are most common and are associated with an increased risk of transient or permanent neurologic deficit (*Ann Thorac Surg* 56:539, 1993). To reduce the incidence of postoperative arrhythmias, patients receiving beta-adrenergic blocking agents preoperatively should continue to be given these medications postoperatively. Patients with supraventricular arrhythmias and hemodynamic compromise should undergo immediate electrical cardioversion (with 50–100 J). Because the most frequent etiology is hypoxia or hypokalemia, the new onset of a supraventricular arrhythmia should be evaluated by measurement of the arterial blood gas and the serum potassium. In many patients with hemodynamically stable arrhythmias, prompt correction of the oxygen tension (to >70 mm Hg) and the serum potassium level (to >4.5 mg/dL) may terminate the arrhythmia. For patients with **atrial flutter**, overdrive pacing may be used to terminate the arrhythmia. The patient's atrial temporary epicardial pacing wires are connected to a pacemaker generator, and either burst (700–800 beats per minute for 3–4 seconds) or decremental (stepwise decrease from 300 to 180 beats per minute) pacing is used. For rapid atrial arrhythmias, rate control may be achieved using diltiazem (0.25-mg/kg i.v. bolus over 2 minutes if there is no effect, followed in 15 minutes by a 0.35-mg/kg i.v. bolus over 2 minutes and then 5–15 mg per hour i.v. continuous infusion). Other means of rate control are verapamil (2.5–10.0 mg i.v. slowly over 2 minutes every 10–20 minutes) or digoxin (1.0 mg i.v. divided over 4 hours, then 0.125–0.25 mg per day p.o. or i.v.). After rate control is obtained, an antiarrhythmic agent can be used to convert to and maintain normal sinus rhythm. Common agents include amiodarone (800–1,600 mg per day load for 1–3 weeks, then 200–400 mg per day), procainamide (17-mg/kg i.v. loading dose over 20–30 minutes and then 1–4 mg per minute i.v. infusion), quinidine gluconate (324–648 mg p.o. t.i.d.), quinidine sulfate (200–400 mg p.o. q.i.d.), and sotalol (initial dose 80 mg p.o. b.i.d.; maximum, 320 mg per day). The ECG should be monitored carefully in patients receiving type Ia agents, and therapy should be reduced or discontinued in patients with a prolongation of the QT interval of more than 50% over baseline. To reduce the incidence of thromboembolism, patients with intermittent or prolonged atrial arrhythmias should receive anticoagulants (e.g., heparin and then warfarin) for a period of at least 3 months. Frequent atrial premature contractions may precede the onset of other atrial arrhythmias.
 - b. **Ventricular arrhythmias** in the postoperative period are treated as they are in other patients (see [Chapter 3](#) and [Chapter 7](#)). Lidocaine (2- to 4-mg per minute infusion) may be used to suppress frequent (>6 per minute) or multifocal premature ventricular contractions. Prophylactic administration of lidocaine is not indicated except in patients presenting with acute MI. Ventricular arrhythmias other than premature ventricular contractions suggest significant underlying ischemic pathology.
3. **Perioperative MI** occurs in approximately 1–2% of patients and can be diagnosed by ECG changes, the onset of a new bundle branch block, biochemical criteria (e.g., elevated troponin or creatine kinase-MB), or echocardiography. Survival may be adversely affected if complications such as cardiogenic shock or ventricular arrhythmias develop.
4. **Postpericardiotomy syndrome** is a delayed pericardial inflammatory reaction characterized by fever, anterior chest pain, and pericardial friction rub, and it may lead to mediastinal fibrosis and premature graft occlusion. Treatment includes nonsteroidal antiinflammatory drugs for 2 weeks or corticosteroids for refractory cases.
- f. **Postoperative hemorrhage** is common after cardiac surgery and necessitates reexploration in up to 5% of patients. Hematologic parameters (complete blood count, PT, PTT) are measured on admission to the ICU and as needed. CPB requires heparinization, causes platelet dysfunction and destruction, and activates the fibrinolytic system. Initial focus is on adequate BP control, metabolic stability, maintenance of normothermia, and adequate reversal of heparin with protamine. Autotransfusion devices may be used to collect blood from the mediastinal and pleural chest tubes and return this blood to the patient for a period of 12–24 hours postoperatively. For patients with significant postoperative bleeding (>200 mL per hour), consideration should be given to platelet transfusion to maintain the platelet count at greater than 100,000/µL and transfusion of fresh frozen plasma if the PT is abnormal. Some surgeons advocate stripping the chest tubes every hour to prevent clotting. If clotting becomes apparent, sterile suction tubing can be used to evacuate blood clot. The formation of undrained clot in the mediastinum may result in cardiac tamponade. This diagnosis can be made by an unexplained drop in urine output or cardiac index with adequate filling pressures and can be confirmed by echocardiography. Indications for operative reexploration for bleeding include (1) prolonged bleeding (>200 mL per hour for 4–6 hours), (2) excessive bleeding (>1,500 mL), (3) a sudden increase in bleeding, and (4) cardiac tamponade. Pleural and mediastinal chest tubes generally are removed when the drainage is less than 200 mL in 16 hours. Several pharmacologic agents may be useful in managing postoperative hemorrhage.
 1. **Aprotinin** is a naturally occurring antifibrinolytic that has been shown to reduce postoperative blood loss and transfusion requirement if administered before the institution of CPB. The mechanism of action is believed to be via preventing alterations in platelet structure and function and by inhibiting fibrinolysis. Potential side effects of aprotinin include thromboembolic events, premature graft occlusion, and renal dysfunction. The main limitation in the use of aprotinin is that it is immunogenic; therefore, anaphylaxis may occur on reexposure. Thus, aprotinin is usually reserved for redo cardiac surgery and patients at high risk of bleeding complications during primary operations.
 2. **Epsilon-aminocaproic acid** (25 mg/kg i.v. over 1 hour, followed by 1–2 g per hour for 4 hours) is a synthetic antifibrinolytic that may be useful to control fibrinolysis (e.g., bleeding that occurs after initial clot formation) or bleeding refractory to other measures.
 3. **Desmopressin** (0.3 µg/kg i.v. over 30 minutes) increases expression of platelet membrane receptor GP Ib and increases serum levels of factor VIIIc and von Willebrand factor. The efficacy of desmopressin in reducing postoperative hemorrhage is controversial, but it may be useful in managing postoperative bleeding in the context of documented or presumed platelet dysfunction (e.g., recent antiplatelet agent use, prolonged bleeding time).
- g. **Mechanical ventilation** is used in the initial postoperative period with typical settings: intermittent mandatory ventilation, 10–16 breaths per minute; inspired oxygen concentration, 1.0; tidal volume, 10–15 mL/kg; and positive end-expiratory pressure, 5 cm H₂O. The patient can be extubated when (1) fully awake with a normal neurologic examination; (2) weaning parameters are satisfactory (e.g., respiratory rate <20 breaths per minute; minute ventilation <12 L per minute; negative inspiratory pressure >20 mm H₂O); (3) the arterial blood gas, with only continuous positive airway pressure, is satisfactory (pH approximately 7.40; CO₂ tension <45 mm Hg; oxygen tension >70 mm Hg); (4) there is little mediastinal bleeding (<100 mL per 8 hours); and (5) there is hemodynamic stability. Most patients can be extubated several hours after operation. After extubation, oxygen is administered by high-humidity face mask with an initial inspired oxygen concentration of 0.4. The oxygen can be weaned, as tolerated, to keep the arterial oxygen saturation above 93%.
- h. **Renal dysfunction** in the postoperative period can be due to decreased perfusion pressure during CPB or to inadequate perfusion of the kidneys in the postoperative period. Treatment of acute renal insufficiency in the postoperative period includes ensuring adequate hydration and avoiding nephrotoxic medications. Fluid and electrolyte balance is evaluated immediately after operation and then as needed. Early after operation, intravenous fluids are administered slowly (<30 mL per hour). A useful measure of a patient's intravascular volume status is the body weight, which is measured daily and compared to the preoperative weight. For patients with normal renal function, diuretics (e.g., furosemide, 20 mg i.v. every 12 hours) are administered beginning on the first postoperative day to return the patient to his or her preoperative weight. For patients with impaired renal function, other diuretics may be needed (bumetanide, 0.5–2.0 mg per day p.o. or 0.5–5.0 mg i.v. administered over 1–2 minutes every 3 hours with a maximum of 10 mg; chlorothiazide, 0.5–2.0 g per day p.o., or 250–500 mg per day i.v. administered slowly). Acetazolamide (125–250 mg i.v. every 12 hours) is used for patients with metabolic alkalosis. Serum potassium levels are maintained at greater than 4.5 mg/dL by intravenous (10 mEq per hour) or oral (40-mEq dose) administration to prevent atrial and ventricular arrhythmias. Metabolic acidosis can reflect a low-cardiac-output state.
- i. **Nutrition.** The patient is given nothing by mouth until after extubation. A clear liquid diet then is begun and is advanced to a regular diet as tolerated. Patients with prolonged ventilation should receive either enteral feedings or parenteral nutrition (see [Chapter 2](#)).
- j. **Infectious complications** are uncommon after cardiac surgical procedures but may lead to substantial morbidity and mortality.
 1. **Antibiotics** are administered (cefazolin, 1 g i.v. push bolus every 8 hours; in patients allergic to penicillin or cephalosporin antibiotics, vancomycin, 1 g i.v. push bolus every 12 hours) preoperatively and for 24–48 hours postoperatively for prophylaxis against wound infection.
 2. **Wound infection** occurs in 1–2% of sternotomy incisions and a higher proportion of saphenous vein harvest sites. Risk factors for deep sternal wound infection include diabetes mellitus, chronic obstructive pulmonary disease, and, possibly, the use of bilateral internal thoracic arteries during CABG

procedures in patients older than 65 years. Serous drainage from the skin incision is worrisome and should be treated by application of topical antibiotics [e.g., povidone-iodine (Betadine)], application of a sterile dressing twice daily, and the administration of intravenous antibiotics. Incisional pain that persists beyond the first few postoperative days, a sternal click, gross movement of the sternal edges, or substernal air on the chest X-ray may indicate a deep sternal infection. A computed tomographic (CT) scan of the chest can confirm this diagnosis. In general, deep sternal infections require operative débridement of devitalized sternal and substernal tissues with cultures of the tissue, administration of broad-spectrum intravenous antibiotics, and vascularized muscle flap closure of the soft-tissue defect several days later.

3. **Endocarditis** is suggested by the combination of fever and a new murmur. This diagnosis is confirmed by positive blood cultures and echocardiography. Appropriate intravenous antibiotics should be administered for 6 weeks. Prosthetic valve endocarditis may require replacement of the involved valve to eradicate the infection.

k. **Pain control** is accomplished using parenteral narcotics or nonsteroidal antiinflammatory agents, or both, during the early postoperative period (see [Chapter 6](#)).

l. **Gastrointestinal (GI) complications** are uncommon after cardiac surgical procedures. Stress gastritis is common after CPB and is thought to be secondary to subclinical ischemia of the gut mucosa. Although overt GI hemorrhage is uncommon, when it does occur, it is associated with a high mortality. Patients should receive sucralfate (1 g p.o. every 6 hours) or H₂-receptor antagonist therapy until a regular diet is begun. GI bleeding may arise from throughout the GI tract. The evaluation for patients with GI bleeding is summarized in [Chapter 13](#). Acute cholecystitis, usually acalculous, is associated with a high mortality. Acute pancreatitis, evidenced by hyperamylasemia, occurs in approximately 25% of patients after CPB, but only 5% of patients have abdominal pain.

2. **Step-down unit care** focuses on convalescence, management of fluid balance, activity level, and diet.

a. **Fluid status.** The patient is weighed daily. Patients receiving diuretics preoperatively resume the same regimen postoperatively. For patients who were not receiving diuretics preoperatively, oral diuretics are administered until the patient's weight falls to the preoperative value. For most patients, no restrictions are placed on the daily oral fluid intake.

b. **Activity.** The patient is encouraged to be out of bed to a chair and to ambulate as early as possible after operation. Patients are instructed not to perform any heavy lifting (of >5 lb) for a period of 6 weeks postoperatively.

c. **Diet.** A mild postoperative ileus may be present for several days after operation. A regular diet is begun as early as possible after operation. Some patients require a stool softener. Attention should be paid to maintaining a prudent diet that is low in salt and cholesterol.

3. **Care after hospital discharge** focuses on continued risk factor modification and surveillance for late complications. Common difficulties during the first 6–8 weeks after operation include decreased motivation, decreased appetite, depression, and insomnia. In general, these conditions are temporary, and the physician can provide reassurance.

a. **Physical rehabilitation** with a daily exercise program begins early after operation and continues after discharge from the hospital. Vigorous walking, with increasing distances and longer periods of activity, is the most useful form of exercise for most patients. Bicycling and swimming are acceptable alternatives after 6–8 weeks. Patients who were working before operation should return to work within 2 months after operation.

b. **Risk factor modification** may slow or possibly reverse the progression of atherosclerosis in bypass grafts after CABG.

1. **Smoking** should be discontinued. Referral to organizations with smoking cessation programs should be made before operation, if possible.

2. **Obesity.** Patients should reach an ideal body weight through planned exercise and dieting, if necessary.

3. **Hyperlipidemia** is a major risk factor for the development of graft atherosclerosis and should be treated aggressively, either with diet modification or by pharmacologic intervention. Current American Heart Association guidelines suggest the following target values: total serum cholesterol less than 200 mg/dL, serum triglycerides less than 150 mg/dL, and high-density lipoproteins greater than 35 mg/dL.

4. **Hypertension** must be controlled.

C. Long-term outcomes

1. **CABG** results in initial elimination of angina in more than 90% of patients. Perioperative mortality ranges from 1% to 2% in low-risk patients to more than 10% to 15% in high-risk patients. Graft patency after CABG is related to the bypass conduit used and the outflow vessel. Reverse saphenous vein grafts have 10-year patency rates of approximately 80% to the LAD and 50% to the circumflex or right. Internal thoracic artery grafts have patency rates of approximately 97% at 1 year and 90% at 10 years. Overall 10-year survival is approximately 80% with vein grafts and 89% if an internal thoracic graft was placed to the LAD artery. Antiplatelet therapy using aspirin (325 mg per day) beginning immediately after operation and continued indefinitely is recommended to increase the graft patency rate (*Circulation* 77:1324, 1988). There is no evidence to suggest that the addition of dipyridamole (50–75 mg p.o. t.i.d.) offers any advantage over aspirin alone. Surveillance ECG or stress testing is performed as needed after CABG. The need for reoperation is approximately 11% at 5 years and 38% at 10 years (*Ann Surg* 212:378, 1990).

2. **Valve repair or replacement** has a perioperative mortality of 5–10%, which is higher in double-valve operations or if concomitant CABG is performed. Valve-related complications are uncommon but may be serious when they occur.

a. **Valve degeneration** may occur for both bioprosthetic and mechanical valves. Structural failures associated with mechanical valves are rare and are specific for the particular type of valve. All bioprosthetic valves develop calcification over time that may lead to clinically significant regurgitation. This process is accelerated in younger patients and in those with renal failure or altered calcium metabolism. Bioprosthetic valves have a mean time to failure of 13 years.

b. **Paravalvular leak** occurs when the implanted valve dislodges from the valve annulus. This may lead to clinically significant valvular regurgitation that can be documented by echocardiography. Hemolytic anemia may be documented by an increased reticulocyte count, increased serum lactate dehydrogenase level, and increased urinary iron excretion. Replacement of the valve is indicated for paravalvular leak associated with moderate to severe valvular regurgitation.

c. **Thrombosis and thromboembolism.** Thrombus formation may occur on the surface of the artificial valve and lead to valve thrombosis or embolism. Embolic complications may include transient ischemic attack, stroke, or embolism to the kidneys or extremities. The use of appropriate anticoagulation [International Normalized Ratio (INR) = 2.5–3.5 times control] with mechanical valves may reduce the risk of thromboembolism to the level (approximately 0.5% per year) associated with bioprosthetic valves.

d. **Prosthetic valve endocarditis** may occur any time after valve implantation and occurs with equal frequency (approximately 2%) with bioprosthetic and mechanical valves. The mortality associated with this condition during the first 2 postoperative months approaches 75% but decreases to approximately 40% for late episodes. Antibiotic therapy alone may be sufficient for some late infections, but expeditious valve replacement usually is required. The American Heart Association guidelines for antibiotic prophylaxis to prevent this complication are summarized in [Chapter 7](#).

IV. Cardiac transplantation can provide relief from symptoms in patients with end-stage cardiomyopathy who are functionally incapacitated despite medical therapy and who are not candidates for any other cardiac operation. The 3-year survival for patients undergoing cardiac transplantation is now approximately 80% (*J Heart Transplant* 8:271, 1989).

A. **Indications** for cardiac transplantation include ischemic or idiopathic dilated cardiomyopathy (approximately 75% of patients), congenital heart disease, valvular heart disease, viral cardiomyopathies, and failure of a previous cardiac transplant, as well as familial and restrictive cardiomyopathies.

B. **Contraindications** to transplantation include age older than 65 years; systemic infection; untreated or incurable malignancy; irreversible renal, pulmonary, or hepatic disease; severe fixed pulmonary hypertension (>6 Wood units); active peptic ulcer disease; recent CVA; poorly controlled diabetes mellitus; psychological instability, unreliability, or history of poor compliance; and absence of adequate psychosocial support.

C. **Immunosuppressive therapy** generally includes cyclosporine, steroids, and azathioprine. Cyclosporine usually is started preoperatively. The dosage is adjusted to achieve circulating plasma levels of 250–350 ng/mL. Levels significantly above this range may cause nephrotoxicity. Steroid therapy is initiated in the operating room. When rejection episodes occur, 80% can be reversed with bolus doses of intravenous steroids. When rejection episodes are resistant to increased steroid doses, the monoclonal antibody OKT3 or rabbit antithymocyte serum can be added to the treatment regimen. Immunosuppression is covered in more detail in [Chapter 29](#).

D. **Acute allograft rejection** is diagnosed by **endomyocardial biopsy**. Biopsy forceps are passed into the right ventricle percutaneously, using fluoroscopic or echocardiographic guidance, usually via the right internal jugular vein, and several biopsies are taken to document the presence and degree of rejection. If there is evidence of isolated left ventricular dysfunction, biopsies of the left ventricle may be taken by introducing a biopsy instrument into the left ventricle via the femoral artery. During the early postoperative period, biopsies are performed several times each month. After the first 6 months, the frequency of biopsies is decreased to one to two times per year. Whenever a patient develops evidence of a rejection episode, a biopsy is performed. Complications of endomyocardial biopsy are rare but include ventricular perforation, pneumothorax, transient ventricular or supraventricular arrhythmias, hematoma, transient vocal cord paralysis, and infection. **Graft atherosclerosis**, thought to represent **chronic vascular rejection**, occurs in a significant percentage of cardiac transplant recipients and is a major limitation to the long-term success of cardiac transplantation. Graft atherosclerosis is usually not amenable to conventional revascularization and often requires retransplantation.

V. Diseases of the thoracic aorta include aortic dissection, traumatic disruption of the aorta, and thoracic aortic aneurysms (see [Chapter 23](#)).

A. **Dissection of the aorta** occurs when blood dissects within the media of the aorta, creating a false lumen. Dissections are classified according to their location

by one of two classification schemes. In the DeBakey classification, type I dissections involve the ascending aorta, the aortic arch, and the descending aorta; type II dissections involve the ascending aorta only; and type III dissections involve only the descending aorta. Type III dissections are subdivided into type IIIa, involving only the descending thoracic aorta, and type IIIb, also involving the abdominal aorta. The Stanford classification simplifies the above by labeling any dissection involving the ascending aorta type A and those involving only the descending aorta type B. Aortic dissection may be acute or chronic (arbitrarily >14 days).

1. **Risk factors.** Medial degeneration of the aorta (formerly cystic medial necrosis) is present in 20% of patients. Approximately 20–40% of patients with Marfan's syndrome develop acute aortic dissection. Other common associations include annuloaortic ectasia, bicuspid aortic valve, atherosclerosis, and coarctation of the aorta.
 2. **Pathophysiology.** The false lumen typically occupies one-half to two-thirds of the circumference of the aorta. There may be a small leak from the false lumen, producing a mediastinal hematoma. Rupture of the false lumen may occur into (1) the pericardium, producing pericardial tamponade; (2) the pleural space, producing hemothorax; or, rarely, (3) the abdomen. During the dissection process, the major branches of the aorta may be uninvolved, may derive their blood supply from the false lumen, or may occlude.
 3. **Clinical presentation.** Patients with rupture of the false lumen or occlusion of one or more coronary arteries may present in extremis. Severe hypovolemic shock may result from hemorrhage. Patients with acute dissection present most commonly with the sudden onset of excruciating back or chest pain. Other symptoms may be referable to occlusion of major arteries (e.g., splanchnic vessels). The differential diagnosis includes acute MI and pulmonary embolism. Unlike MI or pulmonary embolism, patients with acute aortic dissection often present with significant hypertension.
 4. **Diagnosis.** The chest X-ray may demonstrate widening of the mediastinum or cardiomegaly. **CT scan** is probably the most common way that acute aortic dissections are diagnosed and may be used to detect the presence of true and false lumens and to evaluate the extent of the dissection process. Although **MR scans** may be more sensitive and specific than CT, the time delay in obtaining the scans may prove prohibitive. **Transesophageal echocardiography** may demonstrate an intimal flap or wall motion abnormalities due to involvement of the coronary arteries and may be used to evaluate the presence of AI. Although transesophageal echocardiography is sensitive and specific, it is highly operator dependent. **Aortography** is the most sensitive diagnostic study. In some centers, it is performed routinely, whereas in others it is performed only if the diagnosis is not certain. **Selective coronary arteriography** is indicated in all stable patients with coronary risk factors or evidence of myocardial ischemia because CABG may be performed together with aortic repair (*Ann Thorac Surg* 59:585, 1995).
 5. **Treatment**
 - a. **DeBakey types I and II and Stanford type A dissections.** Any patient with dissection of the ascending aorta should undergo urgent operation for replacement of the ascending aorta. Relative contraindications include advanced age or other incurable diseases. Operation is performed through a median sternotomy. Options for replacement of the ascending aorta include tube grafts, composite grafts (aortic valve with conduit graft), and cryopreserved homografts. The aortic valve commissures can be resuspended if they have become detached from the proximal ascending aorta. When the dissection process involves the proximal coronary arteries, bypass grafts with saphenous vein may be required.
 - b. **DeBakey type III and Stanford type B dissections.** Treatment generally is medical, with the goal of maintaining a normal BP using sodium nitroprusside and a reduction in heart rate (to 60–70 beats per minute) and left ventricular work with beta-adrenergic receptor antagonists. With medical therapy, the 1-year survival rate is approximately 80%. Operation is indicated for patients with complications of the acute dissection process (e.g., hemothorax, persistent pain, limb or visceral ischemia, acute renal failure, or paraplegia) and chronic enlargement of the aneurysm to greater than 6 cm or enlargement of more than 1 cm per year.
 6. **Follow-up.** Good control of postoperative hypertension is associated with increased survival, decreased risk of false lumen aneurysm rupture (10–20% vs. 50% in patients with poorly controlled hypertension), and decreased risk of recurrent dissection. A chest X-ray and CT scan of the chest should be obtained every 3 months for 1 year and then every 6 months to evaluate for the presence of aneurysm formation. The risk of recurrent dissection is approximately 15%.
- B. Traumatic disruption of the aorta** usually occurs after severe blunt traumatic injuries, such as high-speed motor vehicle accidents. The disruption occurs most commonly just proximal to the ligamentum arteriosum in the upper descending aorta (70%) but may occur less commonly in the ascending aorta (10%) or other sites in the descending aorta (20%).
1. **Clinical presentation.** Patients with traumatic disruption of the aorta often have other severe injuries. Pain due solely to aortic disruption is uncommon. Decreased blood flow to the brain or extremities due to occlusion of the major arterial branches of the aorta also is uncommon.
 2. **Radiologic evaluation.** The chest X-ray may demonstrate widening of the mediastinum, hemothorax, tracheal shift to the right, or blurring of the aortic knob. The presence of associated injuries to the first and second ribs or the clavicles should raise the suspicion of injury to the aorta. **Aortography** is used to confirm the presence of aortic disruption and should be obtained in most patients before operation. **Transesophageal echocardiography** may be useful for documenting the presence of aortic disruption, as well as for evaluating the function of the aortic valve, and may obviate the need for aortography in selected patients (*N Engl J Med* 332:356, 1995). A chest CT scan may demonstrate mediastinal hematoma associated with aortic disruption but should not delay aortography or transesophageal echocardiography.
 3. **Natural history.** The risk of death is greatest in the period immediately after injury. This risk decreases over time, but there is a finite increased risk of death even 10 years after the injury. Late deaths are due typically to rupture of traumatic false aneurysms.
 4. **Operation** is indicated for any patient in whom aortic disruption is discovered within 5 days of injury and is strongly recommended for patients in whom the diagnosis is made later. For repair of aortic disruptions of the upper descending aorta, the patient is positioned in the right lateral decubitus position, and operation is performed through a left posterolateral thoracotomy. The aorta proximal and distal to the tear generally is replaced with a woven polyethylene terephthalate (Dacron) graft. The risk of in-hospital death is approximately 25%, with many deaths due to other injuries (*J Thorac Cardiovasc Surg* 100:652, 1990). The risk of paraplegia is approximately 10%.

VI. Circulatory support. A variety of temporary circulatory support devices is available to support the failing circulation during the perioperative period, including the IABP, VADs, and extracorporeal membrane oxygenation.

- A. IABP.** The IABP is used as the first-line device to provide circulatory support (*Ann Thorac Surg* 54:11, 1992).
1. **Physiologic benefits** of balloon pumping depend on proper inflation of the balloon during diastole and deflation of the balloon during systole. The resulting effects include a reduction in myocardial oxygen consumption, a reduction in left ventricular afterload and preload that results in increased cardiac output, and an increase in diastolic coronary artery blood flow.
 2. **Indications for the IABP** vary in relation to the timing of operation. In the preoperative period, the IABP is indicated for low-cardiac-output states and for unstable angina refractory to medical therapy (e.g., nitrates, heparin, beta-adrenergic blocking agents). Intraoperatively, the IABP is used to permit discontinuation of CPB when inotropic agents and cardiac pacing alone are not sufficient. In the postoperative period, the IABP is used primarily for low-cardiac-output states. The IABP may be used to support the circulation during periods of refractory arrhythmias and can also be used to provide support to the patient awaiting cardiac transplantation.
 3. **Insertion of the IABP** generally is accomplished percutaneously via the common femoral artery. Sheathless devices, because of a narrower diameter, may help decrease the incidence of lower-extremity ischemic complications. Correct placement should be confirmed by chest X-ray, with the radiopaque tip of the balloon positioned just below the aortic knob. At operation, the IABP may be placed directly into the transverse aortic arch with the balloon positioned antegrade in the descending aorta. Before **removal of the IABP**, the platelet count, PT, and PTT should be normal. Manual pressure should be applied for 30 minutes after removal to avoid the formation of a femoral artery pseudoaneurysm or arteriovenous fistula.
 4. **Management** of the device after placement focuses on ensuring proper diastolic augmentation. The ECG and the femoral (or aortic) pressure waveform are monitored continuously on a bedside console. The device may be triggered using either the ECG or the pressure tracing for every heartbeat (1:1) or less frequently (1:2, 1:3, 1:4, or 1:8). Anticoagulation during IABP support is optional. If the balloon must be repositioned or removed, the device should be turned off first. IABP support is withdrawn gradually by decreasing the augmentation frequency from 1:1 to 1:8 in steps of several hours each.
 5. **Complications of IABP** therapy include incorrect placement of the device, resulting in perforation of the aorta; injury to the femoral artery; or reduction in blood flow to the visceral or renal arteries. Ischemia of the lower extremity, evidenced by diminished peripheral pulses or other sequelae, may necessitate removal of the IABP or performance of an inflow arterial bypass procedure (e.g., femoral-to-femoral artery). Rupture of the balloon is an indication for immediate removal because blood may clot within the ruptured balloon, necessitating operative removal.
- B. VADs** may be used to support the left side of the circulation (**LVAD**) or the right side of the circulation (**RVAD**). When both an LVAD and an RVAD are used, the combination is termed a biventricular assist device.
1. **Physiologic effects** of the VAD include decompression of the left or right ventricles (or both), resulting in decreased myocardial oxygen consumption. The goal is to permit recovery of myocardium that is not irreversibly injured (e.g., "stunned" myocardium).
 2. **Indications for a VAD** include (1) inability to separate from CPB despite inotropic and IABP support, (2) postoperative cardiogenic shock, and (3) bridge to cardiac transplantation.
 3. **Centrifugal pumps (Biomedicus and 3M Sarns)** are used most frequently as bridges to recovery in patients with postcardiotomy cardiogenic shock and those who are unable to separate from CPB. If recovery does not occur and a suitable organ becomes available, the pumps may be used as a short-term bridge to transplantation. Advantages include widespread availability, low cost, and simplicity. Disadvantages include the need for systemic anticoagulation, limited duration of support, and the need for continuous supervision by specially trained personnel.

4. **External pulsatile devices (Aimed and Thoratec Laboratories)** have generally the same indications as centrifugal pumps, although the duration of support may be somewhat longer. Advantages are that the devices are designed to allow sternal closure (centrifugal pumps require an open chest) and that they may have a lower incidence of thromboembolism.
 5. **Long-term implantable devices (Novacor and HeartMate)** are used primarily as bridges to transplantation in patients with chronic heart failure. Emerging indications include long-term support of the failing heart in nontransplantation candidates.
 6. **Insertion** of VADs is accomplished operatively. In general, RVADs receive inflow from the right atrium and return outflow to the pulmonary artery using flexible cannulas. In general, LVADs receive inflow from the left atrium and return outflow to the ascending aorta or femoral artery.
 7. **Management** of the VAD after placement focuses on maintaining proper function and adequate anticoagulation. The activated clotting time should be monitored frequently and maintained at approximately 200 seconds. Patients should receive medications to ensure adequate sedation, analgesia, and muscle paralysis.
 8. **Complications** of VAD therapy include excessive bleeding, thrombus formation, embolization, and hemolysis. Associated complications not related to the device specifically include respiratory failure (due to infection or fluid overload) and renal failure.
- C. **Extracorporeal membrane oxygenation** is used rarely in adult patients with respiratory failure. A membrane oxygenator is connected in a serial fashion to a centrifugal pump system, typically an LVAD system.

37 General Thoracic Surgery

Scott I. Reznik and Richard J. Battafarano

Introduction

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Introduction

The scope of thoracic surgery encompasses benign and malignant diseases of the lung, pleura, and mediastinum. This chapter focuses on the presentation and surgical management of thoracic disorders limited to the lungs, pleurae, chest wall, and mediastinum.

Chest Wall and Pleural Disease

I. Pneumothorax is one of the most common problems encountered by surgeons. Chest tube insertion is an important skill for all surgical staff (see Chapter 43, [section II.B](#)).

- A. **Spontaneous pneumothorax** is nearly always caused by rupture of an apical bleb. Up to 80% of patients are tall young adults, and men outnumber women by 6 to 1; it is more common in smokers than in nonsmokers. The typical patient presents with acute onset of shortness of breath and chest pain on the side of the collapsed lung. Patients older than 40 years usually have significant parenchymal disease, such as emphysema. These patients present with a ruptured bulla and often have a more dramatic presentation, including tachypnea, cyanosis, and hypoxia. Other etiologies of spontaneous pneumothorax include cystic fibrosis and, rarely, lung cancer.
 1. **Physical examination** may demonstrate decreased breath sounds on the involved side if the lung is more than 25% collapsed. Careful examination for signs of tension pneumothorax (including deviation of the trachea to the opposite side, respiratory distress, and hypotension) must be carried out. Supplemental oxygen should be started. A prompt chest X-ray usually demonstrates the pneumothorax, with air present between the chest wall and visceral pleura.
 2. **Management options** include the following:
 - a. **Observation of a small spontaneous pneumothorax** in a healthy patient.
 - b. **Aspiration** using a small catheter, such as a 14-gauge thoracentesis catheter, with immediate and delayed chest X-ray.
 - c. **Insertion of small percutaneous catheters**, such as a Pneumocath, placed to a Heimlich valve or water seal is useful for small to moderate pneumothoraces.
 - d. **Tube thoracostomy** with a small- to medium-sized chest tube (16 French percutaneous or Thal tube to 24 French for adults) should be performed for larger pneumothoraces (>30%) or in patients with pulmonary or cardiac compromise. The tube should be placed in the anterior or midaxillary line through the fifth interspace with the tube positioned in the apical chest. Usually, the pneumothorax resolves quickly and the bronchopleural air leak disappears over the course of 1–2 days. Patients with severe chronic obstructive pulmonary disease often do not tolerate even small pneumothoraces and should have prompt placement of a chest tube.
 - e. **Indications for operation** for spontaneous pneumothorax include (1) recurrent ipsilateral pneumothoraces, (2) bilateral pneumothoraces, (3) persistent air leaks on chest tube suction (usually >5 days), and (4) first episodes occurring in patients with high-risk occupations (e.g., pilots, divers) or those who live a great distance from medical care facilities. The risk of ipsilateral recurrence of a spontaneous pneumothorax is 50%, 62%, and 80% after the first, second, and third episodes, respectively.
 - f. **Operative management** consists of stapled wedge resection of blebs or bullae, usually found in the apex of the upper lobe or superior segment of the lower lobe. Pleural abrasion (pleurodesis) should be done to promote formation of adhesions between visceral and parietal pleurae. Video-assisted thoracoscopic techniques (VATs) have allowed for a less morbid procedure in most cases. Using three small port incisions on the affected side, thoracoscopic stapling of the involved apical bulla and pleurodesis can be done. Alternatively, a transaxillary thoracotomy incision gives excellent exposure of the upper lung through a limited incision.
- B. **Iatrogenic pneumothoraces** usually are the result of inadvertent pleural injury during central venous access attempts, pacemaker placement, or transthoracic or transbronchial biopsy. A postprocedure chest X-ray is mandatory after these procedures. Physicians performing any of these procedures must be aware of the possible dangers and be prepared to act quickly to decompress pneumothoraces arising from errant access attempts. These pneumothoraces may progress to tension pneumothorax (a life-threatening problem) if not addressed promptly.
- C. **Traumatic pneumothoraces** may result from either blunt or penetrating thoracic trauma.
 1. **Evaluation and treatment** begin with the initial stabilization of airway and circulation. A chest X-ray should be obtained. Prompt chest tube insertion is performed to evacuate air and blood. In 80% of patients with penetrating trauma to the hemithorax, exploratory thoracotomy is unnecessary, and chest tube decompression with observation is sufficient. Indications for operation include immediate drainage of more than 1,500 mL blood after tube insertion or persistent bleeding greater than 200 mL per hour. Patients with multiple injuries who have proven pneumothoraces or significant chest injuries should have prophylactic chest tubes placed before general anesthesia because of the risk of tension pneumothorax with positive-pressure ventilation.
 2. **Tension pneumothorax** occurs secondary to the entry of air into the pleural space without egress of air. The expanding pneumothorax shifts and kinks the mediastinal structures, causing decreased venous return, hypotension, and cardiopulmonary compromise. Physical examination reveals decreased breath sounds on the affected side and tracheal deviation to the contralateral side. Emergent decompression with a large-bore angiocatheter inserted into the second interspace in the midclavicular line, followed by prompt insertion of a chest tube, can be lifesaving.
 3. The unusual circumstance known as a **sucking chest wound** consists of a full-thickness hole in the chest wall greater than two-thirds the diameter of the trachea. With inspiration, air preferentially flows through the wound because of the low resistance to flow. This requires immediate coverage of the hole with an occlusive dressing and chest tube insertion to reexpand the lung. If tube thoracostomy cannot be immediately performed, coverage with an occlusive dressing taped on three sides functions as a one-way valve to prevent the accumulation of air within the chest, although tube thoracostomy should be performed as soon as possible.
 4. **A traumatic bronchopleural fistula** can occur after penetrating or blunt chest trauma. If mechanical ventilation is ineffective secondary to the large air leak, emergent thoracotomy and repair are usually necessary. On occasion, selective intubation of the uninvolved bronchus can provide short-term stability in the minutes before definitive operative treatment.

II. Pleural effusion. More than 1 million pleural effusions are diagnosed annually in the United States. Increased density on chest X-ray can be secondary to parenchymal disease (infiltrates) or to effusions. Free-flowing effusion can be diagnosed by layering of the density on lateral decubitus X-ray. Ultrasound is often useful in identifying and localizing pleural effusions for diagnostic or therapeutic drainage. Chest computed tomography (CT) is the best radiographic examination for determining the extent and character of the effusion.

- A. **Etiology.** Causes of pleural effusion include (1) increased hydrostatic pressure (congestive heart failure), (2) increased capillary permeability secondary to pneumonia, (3) effusion associated with pancreatitis or subdiaphragmatic abscess, (4) decreased plasma colloid oncotic pressure (hypoalbuminemia), (5) increased negative intrapleural pressure (atelectasis), or (6) impaired drainage by lymphatics secondary to tumor (malignant effusion).
- B. **Symptoms** vary depending on the size of the effusion and the physiologic condition of the patient. A variable amount of dyspnea is the most common symptom. The first step in diagnosis and treatment is thoracentesis. A concave meniscus in the costophrenic angle on an upright chest X-ray suggests at least 250 mL pleural fluid. It should be noted that most postthoracotomy effusions are not clinically significant and do not need to be drained. Small effusions in patients with congestive heart failure are rarely the cause of dyspnea.
- C. **Thoracentesis**
1. **The technique** of thoracentesis is described in Chapter 43, [section II.A.2](#).
 2. **The fluid should be sent for culture and Gram stain, biochemical analyses [pH, glucose, amylase, lactate dehydrogenase (LDH), and protein levels], and a differential cell count and cytology to rule out malignancy.** Larger volumes (several hundred milliliters) can often aid the cytopathologists in making a diagnosis. Pleural effusions are broadly categorized as either transudative (protein-poor fluid not involving primary pulmonary pathology) or exudative (resulting from increased vascular permeability as a result of diseased pleura or pleural lymphatics). Protein and LDH levels measured simultaneously in the pleural fluid and serum provide the diagnosis in nearly all settings. Exudative pleural effusions satisfy at least one of the following criteria: (1) ratio of pleural fluid protein to serum protein greater than 0.5, (2) ratio of pleural fluid LDH to serum LDH greater than 0.6, or (3) pleural fluid LDH greater than two-thirds the upper normal limit for serum. The distinction is an important one because a transudative pleural effusion can usually be considered a secondary diagnosis; therefore, therapy should be directed at the underlying problem (congestive heart failure, cirrhosis, or nephrotic syndrome). Therapeutic drainage is rarely indicated because fluid rapidly reaccumulates unless the underlying cause improves. In general, thin, yellowish, clear fluid is common with transudative effusions; cloudy and foul-smelling fluid usually signals infection or early empyema; bloody effusions often denote malignancy; milky white fluid suggests chylothorax; and pH less than 7.2 suggests bacterial infection or connective tissue disease.
- D. For **recurrent or persistent effusions** that are resistant to medical correction or are caused by malignant disease or infection, the first line of treatment is insertion of a chest tube for drainage. The tube should be placed posteriorly in patients with free-flowing effusions. When draining a large pleural effusion, it is prudent to evacuate the fluid in stages. One method is to drain 1,500 mL initially, then to clamp the tube for a 2-hour period. This process is repeated, draining 1,500 mL at each 2-hour interval until drainage slows. The tube can then be connected to vacuum suction or to a water-seal system. This avoids the rare but catastrophic problem of reexpansion pulmonary edema. Malignancies of the breast, lung, and ovary as well as lymphomas account for 75% of malignant effusions. Given that the average survival time for patients who present with **malignant pleural effusion** is 4–6 months, the goal of management is successful palliation with minimal hospital stay. Chest-tube drainage alone is effective in only a small number of patients. The common therapy is chest-tube drainage with sclerosis (bedside pleurodesis) to obliterate the space between visceral and parietal pleurae.
1. **Bedside pleurodesis.** Chest-tube drainage is continued until the effusion is well drained and the lung is completely reexpanded. A sclerosing agent (doxycycline, bleomycin, or talc) is administered through the chest tube. This procedure can be quite uncomfortable for the patient, although, with adequate analgesia, it is usually well tolerated. Before administering any sclerosing agent, the patient should be given a patient-controlled analgesic pump, and immediately before sclerosis, bolus administration of narcotic or intravenous ketorolac (if tolerated), or both, can be helpful. Patients with benign lesions requiring pleurodesis tend to experience more pain than those with malignant pleural effusion. Talc is the most effective and least painful sclerosing agent. Talc, 5 g in 180 mL sterile saline, split into 360-mL catheter syringes, is administered via the chest tube and then flushed with an additional 60 mL saline. The tube is clamped, and the patient and the nursing staff are instructed to roll the patient from the supine to the right lateral decubitus to the left lateral decubitus position every 15 minutes for 2 hours. If patients can tolerate the prone, Trendelenburg, and reverse Trendelenburg positions, these positions should be included in the sequence. The nursing staff should be instructed to return the tube to suction if the patient becomes severely dyspneic or hypotensive. The tube is then unclamped and returned to continuous suction until the drainage is less than 100 mL in 24 hours. Doxycycline is an alternative agent; it is used as the agent of choice in patients with benign disease. It is administered as 500 mg in 100 mL normal saline. Doxycycline is extremely irritating to the pleural surfaces; therefore, 30 mL 1% lidocaine can be administered via the chest tube before the doxycycline is given and used to flush the drug again. The total dose of lidocaine should not exceed the toxic dose, which is usually 5 mg/kg. The patient is instructed to change positions as detailed above. Bedside pleurodesis often precipitates an inflammatory response in the lung, which can severely compromise an already tenuous patient.
 2. Bedside pleurodesis can also be performed for patients with **persistent air leaks from recent resection or bullous disease**. Usually, doxycycline is used as the sclerosing agent. If there is a danger of collapse of the lung, such as in a postoperative patient, the chest tube should not be clamped, but the drainage bottle or suction device should be placed above the patient's head. If the tube is clamped, the patient may develop a massive subcutaneous emphysema from trapped air escaping into the tissue or from a life-threatening tension pneumothorax.
 3. **Thoracoscopy, with pleural biopsy, and talc poudrage** involves general anesthesia, but it is almost 100% effective and usually involves only 2 days in the hospital. Patients with sterile exudative effusions with negative cytology and no identifiable cause should undergo open or video-assisted pleural biopsy and poudrage. Pleuroctomy or pleuroperitoneal shunts are occasionally used in patients with recurrent effusions in whom pleurodesis attempts have failed. Patients with severe liver disease complicated by pleural effusions sometimes respond to a Silastic catheter placed in the pleural space to drain the effusion over an extended time course. This procedure induces a slow fusion of the pleura.
- E. **Empyema** is a suppurative infection of the pleural space. Fifty percent of empyemas are complications of pneumonia; 25% are complications of esophageal, pulmonary, or mediastinal surgery; and 10% are extensions from subphrenic abscesses. Thoracentesis is diagnostic but is sufficient treatment in only the earliest cases.
1. The **clinical presentation** of empyema ranges from systemic sepsis requiring emergent care to the patient with a chronic loculated effusion who complains of fatigue. Other symptoms include pleuritic chest pain, fever, cough, and dyspnea.
 2. The **most common offending organisms** are gram-positive cocci (*Staphylococcus aureus* or streptococci) and gram-negative organisms (*Escherichia coli*, *Pseudomonas*, *Klebsiella*). *Bacteroides* species are also common.
 3. **Management** includes control of the infection by appropriate antibiotics, drainage of the pleural space, and obliteration of the empyema space. Once the diagnosis is made, treatment should not be delayed. Specific management depends on the phase of the empyema, which depends on the character of the fluid. Early or **exudative empyema** is usually adequately treated with simple tube drainage. **Fibropurulent empyema** may be amenable to tube drainage alone, but the fluid may be loculated. The loculations of empyema cavities are composed of fibrin. In advanced or **organizing empyema**, the fluid is thicker, and a fibrous peel encases the lung. Empyema does not exist in these separate phases but progresses on a continuum where portions may be free flowing, and other areas may be organized. Chest CT can be invaluable in determining if the fluid is loculated or whether drainage procedures are effective.
 - a. Patients with **fluid collections difficult to drain by bedside tube thoracostomy** can often be managed by image-guided catheters placed by interventional radiologists.
 - b. If a patient has a **persistent fluid collection with an adequately placed tube** as evidenced by chest X-ray CT, intrapleural fibrinolytic therapy may be indicated. Intrapleural streptokinase, 250,000 units, is divided into three doses, each in 60 mL normal saline. A dose is administered and flushed with 30 mL normal saline. The tube is clamped and the patient rolled as described for pleurodesis; then the tube is returned to suction. The procedure is repeated every 8 hours. Alternatively, 250,000 units can be administered daily for 3 days. The adequacy of treatment is determined by resolution of the fluid collection and complete reexpansion of the lung.
 - c. **Organized empyema is not amenable to tube drainage.** In this setting, a thoracotomy with decortication of the trapped lung is necessary to evacuate all infected material and to restore lung function. This procedure is successful in 80–100% of cases.
 4. A **postpneumonectomy empyema** is one of the most difficult complications in thoracic surgery to manage. Typically, there is a dehiscence of the bronchial stump and contamination of the pneumonectomy space with bronchial flora. The finding of air in the pneumonectomy space on chest X-ray is often diagnostic. The incidence of major bronchopleural fistula after pulmonary resection varies from 2% to 10% and has a high mortality (16–70%). Initial management includes thorough drainage (either open or closed) of the infected pleural space, antibiotics, and pulmonary toilet. Definitive surgical repair of the fistula may include primary closure of a long bronchial stump or closure of the fistula using vascularized muscle or omental flaps. The residual pleural cavity can be obliterated by a muscle transposition, thoracoplasty, or delayed Clagett procedure.
 - a. **Initially**, a chest tube is inserted to evacuate the empyema. Great caution should be taken in the insertion of chest tubes into postpneumonectomy empyemas. A communicating bronchial stump–pleural fistula can contaminate the contralateral lung rapidly when decompression of the empyema is attempted. The patient should be positioned with the affected side down so that the remaining lung is not contaminated with empyema fluid. This might best be handled in the operating room.
 - b. **After the patient is stabilized**, the next step usually is the creation of a **Clagett window** to provide a venue for daily packing and to maintain external drainage of the infected pleural space. This typically involves reopening the thoracotomy incision at its anterolateral end and resecting a short segment of two or three ribs to create generous access to the pleural space. The pleura then is treated with irrigation and débridement. After a suitable interval (weeks to months), the wound edges can be excised and the pneumonectomy space closed either primarily or with a muscle flap after it has been filled with 0.25% neomycin solution (*Surgical diseases of the pleura and chest wall*. Philadelphia: WB Saunders, 1986). Alternatively, the space can be filled with vascularized muscle flap.

III. **Mesothelioma** is a rare and deadly malignancy. The rising incidence is most likely because of the large number of people exposed to asbestos in the mid-twentieth century. Asbestos exposure is the primary cause for development of malignant mesothelioma. Other potential causes include materials with similar

physical properties to asbestos, radiation, viruses, and a variety of chemicals. Mesotheliomas arise from mesothelial cells but differentiate into a variety of histologic patterns. It is often difficult to differentiate mesothelioma from other tumors without the benefit of special stains or immunohistochemistry.

- A. **Mesothelioma has a long latency**, which can be 40–50 years. Patients may present variably with chest pain, malaise, cough, weakness, weight loss, and shortness of breath with pleural effusion. As the tumor progresses the pleural effusion may resolve because the pleural surfaces fuse.
- B. **CT scan** is useful in differentiating pleural from parenchymal disease. Malignant mesothelioma usually appears as a markedly thickened, irregular pleural-based mass or nodular pleura with a pleural effusion. Occasionally, only a pleural effusion is seen. MR scan has not been shown to have significant advantages over CT.
- C. The **diagnosis** can be made by thoracentesis in fewer than half of patients. Thoracoscopic or open pleural biopsy is usually necessary to confirm the diagnosis.
- D. There is no universally agreed upon **staging system**. The best staging system is the New International Staging System for Diffuse Malignant Pleural Mesothelioma (*J Thorac Cardiovasc Surg* 111:815, 1996). The extent of the tumor and its stage are determined at the time of surgery.
- E. **Median survival** in untreated patients with malignant mesothelioma is 4–12 months. Single-modality chemotherapy, radiotherapy, or surgery alone has had no impact on survival. Current aggressive multimodality therapy consists of pleurectomy and decortication or extrapleural pneumonectomy to decrease tumor mass, followed by chemotherapy and radiotherapy. Survival at 2 and 5 years is 38% and 15%, respectively, with a median survival of 19 months (*J Thorac Cardiovasc Surg* 117:54, 1999).
- F. **Intracavitary photodynamic therapy** is a new approach that has been combined with surgical resection. Talc pleurodesis can be performed for palliative treatment.

Parenchymal Lung Disease

I. Pulmonary contusion may follow either blunt or penetrating trauma. The contusion usually is evident on the initial chest X-ray (as opposed to aspiration, in which several hours may elapse before an infiltrative pattern appears on serial radiographs) and appears as a fluffy infiltrate that progresses in extent and density over 24–48 hours. The contusion may be associated with multiple rib fractures, leading to a **flail chest**. This occurs when several ribs are broken segmentally, allowing for a portion of the chest wall to be “floating” and to move paradoxically with breathing (inward on inspiration). The paradoxical movement and splinting secondary to pain and the associated pain lead to a reduction in vital capacity and ineffective ventilation. All patients with suspected contusions and rib fractures should have aggressive pain control measures, including patient-controlled analgesia pumps, epidural catheters, and intercostal nerve blocks. Intravenous fluid should be minimized as much as the patient’s clinical status tolerates because of associated increased capillary endothelial permeability. Serial arterial blood gas measurements are important for close monitoring of respiratory status. Close monitoring and a high index of suspicion for respiratory decompensation are necessary. Intubation, positive-pressure ventilation, and even tracheostomy are often necessary.

II. Hemoptysis can originate from a number of causes, including infectious, malignant, and cardiac disorders (e.g., bronchitis or tuberculosis, bronchogenic carcinoma, and mitral stenosis, respectively). **Massive hemoptysis** requires emergent thoracic surgical intervention, often with little time for formal studies before entering the operating room. The surgeon is called primarily for significant hemoptysis, which is defined as more than 600 mL of blood expelled over 48 hours or, more often, a volume of blood that is impairing gas exchange. Because the volume of the main airways is approximately 200 cc, even smaller amounts of blood can cause severe respiratory compromise. Prompt treatment is required to ensure survival. As baseline lung function decreases, a lower volume and rate of hemoptysis are necessary to severely compromise gas exchange.

- A. A **brief focused history** can often elucidate the etiology of the bleed, such as a history of tuberculosis or aspergillosis. A recent chest X-ray may reveal the diagnosis in up to half of cases. Chest CT is rarely helpful in the acute setting and is contraindicated in patients who are unstable. Trace amounts of hemoptysis can be evaluated by radiologic examinations in conjunction with bronchoscopy.
- B. **Bronchoscopy** is the mainstay of diagnosis and initial treatment. Although it may not eliminate later episodes of bleeding, it can allow for temporizing measures, such as placement of balloon-tipped catheters and topical or injected vasoconstrictors. In patients with massive hemoptysis, after a brief attempt at stabilizing the patient, he or she should be prepared for a rigid bronchoscopy, which is best performed in the operating room under general anesthesia. Asphyxiation is the primary cause of death in patients with massive hemoptysis. Rigid bronchoscopy allows for rapid and effective clearance of blood and clot from the airway, rapid identification of the bleeding side, and prompt protection of the remaining lung parenchyma (with cautery, by packing with epinephrine-soaked gauze, or placement of a balloon-tipped catheter in the lobar orifice).
- C. Even if the **etiology** and the precise source of the bleed is not identifiable by bronchoscopy, ongoing bleeding requires protection of the nonbleeding side. Selective ventilation, with either a double-lumen tube or by selectively intubating the contralateral main-stem bronchus, may be critical to avoid asphyxiation.
- D. **After isolation of the bleeding site, angiographic embolization** of a bronchial arterial source may allow for lung salvage without the need for resection. The bronchial circulation is almost always the source of hemoptysis. Bleeding from the pulmonary circulation is seen only in patients with pulmonary hypertension.
- E. **Definitive therapy** may require thoracotomy with lobar resection or, rarely, pneumonectomy. Infrequently, emergent surgical resection is necessary to control the hemoptysis. The etiology of the bleeding and the pulmonary reserve of the patient are important, because many patients are not candidates for surgical resection.

III. The solitary pulmonary nodule, or “coin lesion,” is a circumscribed lung mass in an asymptomatic individual. Certain settings favor either a benign or malignant diagnosis. In general, the chances of malignancy increase with the size of the lesion, advancing age, and more extensive smoking history.

- A. Lesions **larger than 3 cm** are almost always malignant. An increase in size over time (compared with old X-rays) also favors malignancy. The patient should be queried about any old radiographs, and those films should be reviewed.
- B. **CT scanning** can be used to provide information about homogeneity, feeding vessels, and calcification of the lesion. Specific types of calcification; central, onionskin, or lamellar layers; and popcornlike calcifications favor a benign diagnosis. Eccentric or stippled flecks of calcifications can often be seen with malignancy and warrant investigation. In areas endemic for histoplasmosis, as many as 25% of all nodules may be granulomas.
 - 1. The presence of **fatty densities** in the lesion suggests **benign hamartoma**.
 - 2. **Malignant lesions** more often demonstrate spiculated forms and variable intralobular densities on CT scan.
 - 3. **Benign-appearing nodules** that are stable by CT for 2 years are believed to be benign.
- C. **Noninvasive imaging** usually cannot rule out the presence of malignancy. However, fluorodeoxyglucose **positron emission tomography (PET) scanning** has demonstrated 95% sensitivity and 80% specificity in characterizing solitary pulmonary nodules. PET scan has a high negative predictive value for most lung cancers; however, bronchoalveolar carcinoma can be negative by PET scan. A positive PET scan requires follow-up with a CT-guided transthoracic needle biopsy because inflammatory or infectious processes can give false-positive results. Patients with central lung lesions should undergo bronchoscopy with biopsy. Patients with peripheral lesions can undergo wedge resection followed by lobectomy if the lesion is malignant. Patients with indeterminate lesions can undergo biopsy by VAT.

IV. Carcinoma of the lung is the leading cause of cancer death in the United States. The American Cancer Society estimates that in 2001, 169,500 new cases of lung cancer will be diagnosed, and 157,400 people will die of the disease. Fewer than 30% of patients in whom lung carcinoma is diagnosed are candidates for curative resection. Overall 5-year survival is approximately 14%.

- A. **Screening for lung cancer.** The major risk factor for lung cancer is cigarette smoking, and 90% of lung cancers occur in current or former smokers. Other factors include preexisting lung disease such as bullous emphysema, occupational exposure to asbestos, or materials related to the mining industries. Carcinoma occurs more frequently in the right lung than in the left (60% vs. 40%). Chest X-ray as screening modality has been extensively studied. These studies reveal earlier identification of lung cancers but no increase in survival. Screening with spiral CT has identified tumors at an earlier stage. However, the follow-up is still too short to demonstrate that CT screening offers a survival advantage.
- B. The **two main classes of lung tumors** are small cell (oat cell) carcinoma and non–small cell carcinoma.
 - 1. **Small cell carcinoma** accounts for approximately 20% of all lung cancers. It is highly malignant, usually occurs centrally near the hilum, occurs almost exclusively in smokers, and rarely is amenable to surgery because of wide dissemination by the time of diagnosis. These cancers initially respond to chemotherapy, but overall 5-year survival remains 10%.
 - 2. **Non–small cell carcinomas** account for 80% of all lung cancers and make up the vast majority of those treated by surgery. The three subtypes are **adenocarcinoma** (30–50% of cases), **squamous cell** (20–35%), and **large cell undifferentiated** (4–15%) (*General thoracic surgery*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2000).
- C. **Clinical presentation of lung cancer.** A significant number of resectable lung cancers are identified on routine chest X-ray. On occasion, the following clinical features may be present.
 - 1. **Bronchopulmonary features** include cough or a change in a previously stable smoker’s cough, increased sputum production, dyspnea, or new wheezing.

Minor hemoptysis causing blood-tinged sputum, even as an isolated episode, should be investigated with flexible bronchoscopy, especially in patients with a history of smoking who are 40 years of age or older. Lung cancer may also present with postobstructive pneumonia.

2. **Extrapulmonary thoracic symptoms** include chest wall pain secondary to local tumor invasion, hoarseness from invasion of the left recurrent laryngeal nerve near the aorta and left main pulmonary artery, shortness of breath secondary to malignant pleural effusion, and superior vena cava syndrome (usually secondary to tumor compression of the superior vena cava), causing facial, neck, and upper-extremity swelling. Pancoast's tumor (superior sulcus tumor) can lead to brachial plexus invasion, which causes arm numbness, weakness, and pain, as well as invasion of the cervical sympathetic ganglia, which causes an ipsilateral Horner's syndrome (ptosis, miosis, and anhidrosis). Rarely, lung cancer can present as dysphagia secondary to compression or invasion of the esophagus by mediastinal nodes or by the primary tumor.
 3. Common **sites of distant metastases** include the liver, skeleton, brain, and adrenal glands. Symptoms may include pathologic fractures and arthritis from bony involvement. Brain metastasis may cause headache, vision changes, or changes in mental status. Adrenal involvement may infrequently present with Addison's disease. Lung cancer is the most common tumor that causes adrenal dysfunction.
 4. Various **paraneoplastic syndromes** are common and occur secondary to the release of hormonelike substances by tumor cells. They include Cushing's syndrome (adrenocorticotropic hormone secretion in small cell carcinoma), syndrome of inappropriate antidiuretic hormone (SIADH), hypercalcemia (parathyroid hormone–related protein secreted by squamous cell carcinomas), hypertrophic pulmonary osteoarthropathy (clubbing of the fingers, stiffness of joints, and periosteal thickening on X-ray), and various myopathies.
- D. Preoperative evaluation and preparation**
1. After the diagnosis of non–small cell lung cancer, the **extent of disease and the appropriate therapy** are determined.
 2. A careful history and physical examination may elicit some of the symptoms noted earlier and may detail the presence of **comorbid conditions**.
 - a. The **most critical factor in planning lung resection** for cancer is the preoperative assessment of pulmonary function and estimation of postoperative pulmonary status. Pulmonary function tests and arterial blood gas analysis are the standard by which operability is usually determined. In general, pulmonary resection can be tolerated if the postresection FEV₁ (forced expiratory volume in 1 second) is 800 cc or greater. Preoperative hypercapnia (arterial carbon dioxide tension >50 mm Hg) is a relative contraindication for surgery.
 - b. Patients with an initial FEV₁ of less than 2 L should have a quantitative **ventilation-perfusion scan** to assess how much the area of planned pulmonary resection contributes to overall lung function.
 - c. **Evaluation of cardiac disease** is critical in minimizing postoperative complications. Patients with lung cancer are often at high risk of coronary disease because of extensive smoking histories. A detailed history and physical examination to elicit signs and symptoms of ischemia and a baseline ECG are the initial steps. Any abnormal findings should be aggressively pursued with stress tests, echocardiograms, or coronary catheterization.
 - d. **Preoperative exercise testing** has gained popularity. Information is obtained on respiratory and cardiac function, and this test can often unmask underlying cardiac disease. These tests usually measure maximal oxygen consumption; most studies identify a cutoff of maximal oxygen consumption at 1 L per minute or 15 mL/kg per minute, below which operation is not advised.
 - e. **Smoking cessation** preoperatively for as little as 2 weeks can aid in the regeneration of the mucociliary function and pulmonary toilet.
 3. **Clinical staging** of a diagnosed non–small cell lung cancer or a suspicious lesion is critical in determining operability. The single most important factor in determining prognosis of lung cancer is the status of the mediastinal lymph nodes (LNs).
 - a. After the initial **posteroanterior and lateral chest X-ray, CT scan of the chest and upper abdomen (to include liver and adrenal glands)** is the most useful noninvasive staging modality available. Information on location, size, and relation of the tumor to surrounding thoracic structures is critical to the planning of surgery. CT also provides information on the status of the mediastinal LNs and on the presence of liver and adrenal metastasis. CT scanning alone does not accurately determine the resectability of tumor adherent to vital structures. Patients with localized disease should undergo thoracotomy to determine resectability.
 - b. **CT evaluation of the mediastinal LNs** has a sensitivity of 65–80% and a specificity of 65% if the LNs are larger than 1 cm. In general, MR scan is not useful in evaluating mediastinal LNs. PET scan has been shown to accurately detect tumor in normal-sized LNs and to exclude tumor in enlarged LNs. PET scan is also useful in differentiating recurrent disease from treatment effects (fibrosis, scarring). The accuracy of PET scanning is limited by the presence of inflammation and ongoing infection.
 - c. **Mediastinoscopy** provides access to the pretracheal, subcarinal, and paratracheal node stations. It is the most accurate method for staging the mediastinal lymph nodes. Although invasive, it is safe, with less than a 1% complication rate [*J Thorac Cardiovasc Surg* 118(1):894–899, 1999]. The timing of mediastinoscopy, whether at the time of thoracotomy or before a planned resection, is controversial and depends on the surgeon's preference.
 - d. **Evaluation of the liver and adrenal glands** by CT scan may be revealing because up to 40% of patients with lung cancer have metastases to one or both of these organs.
 - e. **CT or MR imaging of the brain** to identify brain metastases is mandatory in the patient with neurologic symptoms but is controversial as a routine part of the workup.
 - f. **Bone scan** is obtained in all patients with specific symptoms of skeletal pain and selectively as part of the preoperative metastatic workup.
 - g. **Fiberoptic bronchoscopy** is important in diagnosing and assessing the extent of the endobronchial lesion. Although peripheral cancers rarely can be seen with bronchoscopy, preoperative bronchoscopy is important in ruling out other synchronous lung cancers (found in approximately 1% of patients) and in pulmonary toilet. Bronchial washings can be taken at the time of bronchoscopy.
 - h. **CT scan–guided fine-needle aspiration biopsy** for cytologic diagnosis is usually reserved for the patient in whom initial thoracoscopic or open wedge biopsy under anesthesia would be hazardous. A negative needle biopsy does not exclude malignancy.
 - i. In summary, **all patients should have a posteroanterior and lateral chest X-ray and a chest CT scan** to evaluate for metastatic disease and evaluate the mediastinum. This should be followed by either a PET scan or mediastinoscopy. A positive PET scan should be followed by mediastinoscopy. A negative PET scan or mediastinoscopy can be followed by surgery if there are no other contraindications to operative management. All patients should undergo a fiberoptic bronchoscopy before thoracotomy; this is usually done at the same setting as mediastinoscopy.
- E. An anatomic staging system** using the classification for tumor, nodal, and metastatic status was implemented internationally in 1986 (*Chest* 1997;111:1710) (Table 37-1 and Table 37-2). Surgery is the primary mode of therapy for all stage I and II patients and selected stage IIIa patients who have enough physiologic reserve to tolerate resection.

Table 37-1. American Joint Committee on Cancer staging system of lung cancer

Table 37-2. Five-year survival according to American Joint Committee on Cancer stage groups of TNM (tumor, node, metastasis) status

F. Operative treatment

1. The **operative mortality of lobectomy for lung cancer** is 2–3% and, for pneumonectomy, 6–8%. The extent of curative resection is important. In stage I lung cancers, limited resections (less than lobectomy) result in a 75% increase in local recurrence (*J Thorac Cardiovasc Surg* 106:1053, 1993). Limited resection also results in decreased overall and disease-free survival. Lobectomy using VAT is currently the subject of ongoing trials to determine 5-year survival and rate of tumor implantation port sites.
2. **Five-year survival rates** are 67–85% after resection for stage Ia disease. Stage Ib disease carries a survival of 43–78%. Stage IIa tumors, located completely within the lung and ipsilateral intrapulmonary LNs, have survival rates of approximately 57%. Patients with IIB disease, either T3, N0, M0 or T2, N1, M0, can expect a survival of 33–39%. The classification of stage III tumors was revised in an effort to identify patients with extrapulmonary extension of tumor who might benefit from surgery. Stage IIIa tumors involve the ipsilateral mediastinal and subcarinal LNs. These tumors are potentially resectable, and patients have a 22% overall 5-year survival. The best treatment for IIIa tumors is still evolving. Several trials comparing neoadjuvant chemoradiation followed by surgery versus chemoradiation alone are currently in progress. Stage IIIb tumors involve the contralateral mediastinal or hilar LNs, the ipsilateral scalene or supraclavicular LNs, extensive mediastinal invasion, or malignant pleural effusions. These tumors are considered unresectable. Stage IV tumors have distant metastases and are generally considered unresectable. If, however, a resectable lung cancer and solitary site of metastasis (e.g., brain) are present,

a combined resection can give 10–20% long-term cure.

Emphysema and Chronic Obstructive Pulmonary Disease

I. Patients with chronic smoking histories, in addition to being at increased risk for lung cancer, also may have significant pulmonary dysfunction secondary to emphysema or chronic obstructive pulmonary disease.

- A. These patients often have **elevated arterial carbon dioxide tension and low arterial oxygen tension levels**. Classically, many emphysema patients use their hypoxic drive to promote respiration; therefore, supplemental oxygen should be used judiciously.
- B. If surgery is necessary, **supplemental oxygen should be minimized postoperatively and pulmonary toilet efforts maximized** to avoid complications.

II. Emphysema patients may have bullous disease. This is defined as intraparenchymal air spaces that form secondary to the destruction of alveoli and lung distal to the terminal bronchioles.

- A. **Bullae** may present as multiple (or, on occasion, single) giant air sacs that can compress normal lung tissue.
- B. **Resection of giant bullae** can be performed via thoracotomy or VAT techniques. Resection allows the compressed normal lung to resume function and improve overall lung mechanics.

III. Many patients with **chronic obstructive pulmonary disease** have diffuse emphysematous destruction of both lungs rather than discrete bullae. Along with generalized alveolar destruction, these patients experience loss of elastic recoil of the lung and an ever-increasing size of the chest cavity with flattening of the diaphragm and widening of the intercostal spaces. These changes lead to ineffective ventilation. Volume-reduction surgery, via a median sternotomy or VAT, involves resection of diseased portions of each lung, thereby improving the mechanics of breathing. Lung volume reduction is a palliative procedure and can produce durable improvement in pulmonary function, gas exchange, and exercise tolerance in properly selected patients. It has been shown to improve quality of life in patients awaiting lung transplantation.

Lung Transplantation

Lung transplantation has become an option for many patients with end-stage pulmonary disease, although donor shortage remains a critical problem. Both single-lung and bilateral-sequential lung transplantation are performed with success. Living-donor lobar transplantation is currently being performed on a limited basis. As with any organ transplantation, there are strict recipient criteria, including limited life expectancy, adequate cardiac and nutritional status, and ability to understand and participate in postoperative care. Most patients undergo extensive screening and participate in a pretransplant exercise program to optimize their physical conditioning before transplantation.

I. The most frequent **indication** for lung transplantation is emphysema secondary to smoking or alpha₁-antitrypsin deficiency (accounting for one-third of all transplants). Both bilateral and single-lung transplantations are performed for emphysema. **Other common indications** are pulmonary vascular disease, pulmonary fibrosis, and cystic fibrosis. **The only absolute indication** for bilateral lung transplantation is cystic fibrosis, because a single-lung transplant would leave a chronically infected native lung in an immunosuppressed patient.

II. The most frequent causes of **perioperative morbidity and mortality** are sepsis and lung allograft dysfunction caused by reperfusion injury.

III. Chronic rejection in the form of **bronchiolitis obliterans** is the most frequent cause of late mortality after lung transplantation. Progressive fibrosis of the airways leads to decreased pulmonary function with air-flow limitation. In addition, the risk of late infections is always present in these chronically immunosuppressed patients.

Trachea

The **trachea** provides a passageway for inhaled gas to reach the gas-exchange regions of the lungs from the atmosphere. Its characteristic anatomy provides the surgeon with many challenges. The rigidity of the trachea is provided by cartilaginous rings that extend approximately two-thirds the circumference. The posterior wall is membranous, and the trachea is lined with a ciliated mucosa. The trachea has a segmental blood supply that is derived from the inferior thyroid artery in the proximal trachea and the bronchial arteries distally. Both benign and malignant processes affect the trachea, necessitating treatment.

I. Benign conditions tend to result in tracheal stenosis as the result of infection, trauma, burns, or compression.

- A. **Healed ulcers from tuberculosis** can result in long-segment stenosis of the distal trachea.
- B. **Blunt trauma** can be unrecognized at the time of injury and often results in stenosis.
- C. **Chemical or thermal burns** that cause airway obstruction are often limited only to the mucosa and usually are treated endotracheally with stenting or placement of a T tube.
- D. **Iatrogenic stenosis** may be the result of healed tracheal reconstruction or, more commonly, of intubation with a cuffed endotracheal tube. These lesions can vary from granulation tissue at previous suture lines to tracheal malacia. Careful observation of the pressure in cuffed endotracheal tubes is critical in avoiding these complications.
- E. **Other conditions** that can cause airway obstruction include extrinsic compression from the innominate artery, mediastinal or cervical masses, amyloidosis, and relapsing polychondritis.

II. Tracheal neoplasms are rare even though they are often histologically similar to more distal tumors. Malignant tumors are more common than benign tumors. Symptoms include dyspnea, exercise intolerance, hemoptysis, and change in voice.

- A. The **most common benign tumors** include chondroma, papilloma, fibroma, and hemangioma.
- B. **Squamous cell and adenoid cystic carcinoma** are the most common malignant tumor types, followed by carcinoid tumors.

III. The **initial evaluation** of patients with suspected tracheal pathology includes a thorough history and physical examination, standard chest X-ray, and neck X-ray. CT scan can determine local extension, degree of luminal compromise, and length of the trachea that is involved. Bronchoscopy is required in the evaluation and treatment of tracheal pathology. It should be noted that sedation may cause a patient with a previously compromised airway to completely obstruct the trachea. In addition, endoscopic biopsy may result in bleeding that may compromise the airway. Bronchoscopy should only be performed in the operating room by personnel who are skilled in rigid bronchoscopy. Treatment of tracheal pathology may include resection, endobronchial tumor resection, dilation of stenoses, or placement of endobronchial stents. The principles of tracheal surgery include preservation of the lateral tissue that provides the blood supply, cervical flexion, and tension-free repair. The distal trachea can be mobilized via many maneuvers, including dividing the inferior pulmonary ligament and transplanting the left main bronchus if necessary.

Mediastinum

I. Mediastinal tumors present in a vast array of sizes, locations, and origins. The location of the lesion can aid in preoperative identification and treatment planning. The mediastinum is divided into three zones for classification: anterior compartment, middle (visceral) compartment, and posterior compartment (paravertebral sulci). Mediastinal masses are rare when compared to lung masses. Most tumors have a predilection for a particular compartment of the mediastinum.

- A. The **signs and symptoms of mediastinal masses** depend on the location, nature, and biologic function of the tumor. Children tend to present earlier and with more symptoms than adults because of the smaller space in their mediastina. Children present with respiratory symptoms such as dyspnea, cough, stridor, and chest pain or fever and pneumonitis. Adults can report cough, dyspnea, or vague chest pain.
- B. **In adults, less than half of anterior mediastinal masses are malignant**; however, all lymphatic masses in the anterior mediastinum are malignant. In the

paravertebral sulci, most masses are benign.

C. **Diagnostic evaluation** can include radiologic, biochemical, and invasive procedures.

1. **Chest CT** is usually the first study performed. It can provide much anatomic information, although it cannot always differentiate between cystic and solid masses. CT can demonstrate invasion of the spinal canal and the relationship to the great vessels. Technetium-99m sestamibi scans can be invaluable in identifying parathyroid tissue. Other nuclear medicine studies, such as octreotide scanning, can be useful in identifying carcinoid tumors. PET scanning can be helpful in differentiating malignant from benign masses.
 2. **Biochemical assays** for alpha-fetoprotein and beta-human chorionic gonadotropin should be sent in young men with anterior mediastinal masses to identify germ cell tumors. Patients with paravertebral masses should have urinary catecholamine measured to rule out pheochromocytoma. When thymoma is a possibility, antiacetylcholine receptor antibodies should be measured.
 3. **Asymptomatic anterior lesions without elevated tumor markers** generally do not require tissue diagnosis before surgery. If tissue diagnosis is necessary, fine-needle aspiration under CT or ultrasound guidance is often successful. Extended cervical or anterior mediastinoscopy may be required in tumors not amenable to fine-needle aspiration.
- D. The **most common lesions** presenting in the anterior mediastinum are teratomas, thymomas, thyroid goiters, and lymphomas.
1. For most anterior tumors, excluding lymphoma, the preferred treatment is **surgical excision via a median sternotomy**. Wide exposure is important because malignant lesions may invade vital structures. In most instances (teratoma, benign thymoma), dissection is not difficult, and the tumor is easily excised.
 2. **Thymectomy for myasthenia gravis**
 - a. The role of the **thymus gland in myasthenia gravis** is poorly understood. Approximately 75% of patients demonstrate clinical improvement after thymectomy. Chances of improvement are increased if thymectomy is performed early in the course of disease (first signs of muscle weakness) and the myasthenia is not associated with a thymoma.
 - b. **Preoperative preparation** involves reduction of corticosteroid dosage, if appropriate, and the weaning of anticholinesterases. Plasmapheresis can be performed preoperatively to aid in discontinuation of anticholinesterase agents. Muscle relaxants and atropine should be avoided during anesthetic induction.
 - c. The **operative approach** for thymectomy for myasthenia in cases in which noninvasive imaging does not indicate the presence of thymoma or a mass lesion is controversial. The options range from median sternotomy to a transcervical thymectomy. The **transcervical approach** involves a low collar incision and is facilitated by using a table-mounted retractor to elevate the manubrium and expose the thymic tissue for resection.
 - d. **In instances of bulky thymic disease**, a median sternotomy approach is preferred to **provide maximal exposure for complete resection**.
 3. **Lymphomas presenting in the mediastinum** are not amenable to definitive surgical therapy. Most commonly, the LNs of the anterior compartment are involved, with occasional middle and posterior intercostal involvement.
 - a. The **radiographic presentation of anterior mediastinal lymphoma** is an irregular mass extending bilaterally with compression of surrounding structures.
 - b. Frequently, **cervical adenopathy** is palpable.
 - c. **Diagnosis** can usually be made by cervical LN biopsy or by CT-guided needle biopsy of the anterior mediastinal mass. Occasionally, anterior mediastinotomy and biopsy are necessary.
 - d. **Treatment of mediastinal lymphomas** is based on chemotherapy and radiotherapy. Surgical resection plays virtually no role.
- E. **Lesions that present in the middle mediastinum** include bronchogenic and esophageal cysts and lymphomas. A diagnosis of lymphoma in the middle mediastinum can be made by cervical mediastinoscopy. Resection of bronchogenic or esophageal cysts usually is performed through a posterolateral thoracotomy on the involved side. Most commonly, these cysts are associated intimately with the wall of the main-stem bronchi or esophagus. Dissection planes are well defined unless active infection is present.
- F. Paravertebral sulci (posterior) mediastinal tumors are most commonly neurogenic in origin. **Neurogenic tumors** are malignant in 50% of children and in fewer than 5% of adults. More than 90% of posterior mediastinal neurogenic tumors in adults are benign schwannomas or neurofibromas.
1. These tumors originate from neural crest cells and nearly always are found in association with the paravertebral sulcus along the sympathetic chain or in association with intercostal and spinal nerves.
 2. Most are asymptomatic and present on chest X-ray as smooth, rounded masses in the upper posterior mediastinum.
 3. **Surgical resection** is the treatment of choice via a posterolateral thoracotomy on the affected side. Preoperative CT scanning reveals intraspinal extension of the tumor in 10% of cases, and neurosurgical assistance is necessary for safe resection.
- G. **Postoperative care of the thoracic surgery patient** focuses on three factors: **control of incisional pain, maintenance of pulmonary function, and monitoring of cardiovascular status**. The **thoracotomy incision** is one of the most painful and debilitating in surgery. Inadequate pain control contributes heavily to nearly all postoperative complications. Chest wall splinting contributes to atelectasis and poor pulmonary toilet. Pain increases sympathetic tone and myocardial oxygen demand, provoking arrhythmias and cardiac ischemic episodes. The routine use of epidural catheter anesthesia perioperatively and during the early recovery period has improved pain management significantly. Close monitoring of the patient with **epidural anesthesia** is imperative because the two most significant complications (respiratory depression and hypotension) must be recognized and treated promptly. Stopping the epidural infusion is usually sufficient. Occasionally, naloxone, with or without intubation and mechanical ventilation, is required. Other effective analgesic maneuvers include intercostal blocks with long-acting local anesthetic before closure of the chest and intrapleural administration or local anesthetic via catheters placed at the time of thoracotomy.

II. Maintenance of good bronchial hygiene is often the most difficult challenge facing the postthoracotomy patient. A lengthy smoking history, decreased ciliary function, chronic bronchitis, and significant postoperative pain all contribute to the ineffective clearance of pulmonary secretions. Even aggressive pulmonary toilet with incentive spirometry and chest physiotherapy delivered by the respiratory therapist, along with adequate analgesia, are insufficient on occasion. **Diligent attention must be paid**, with frequent physical examination and daily chest X-ray and arterial blood gas evaluation to detect any changes in gas exchange.

- A. **Atelectasis and mucus plugging** can lead to ventilation-perfusion mismatch with ensuing respiratory failure. The clinician should make liberal use of nasotracheal suctioning, bedside flexible bronchoscopy, and mechanical ventilatory support if needed.
- B. Patients with copious secretions and an adequate cough may be candidates for **a percutaneously placed cricothyrotomy tube** (mini-trach), which allows for frequent suctioning. This also provides access for additional supplemental oxygen.

III. All physicians caring for the postthoracotomy patient should be familiar with **chest tube placement, maintenance, and removal**. The purpose of chest tube placement after thoracotomy and lung resection is to allow drainage of air and fluid from the pleural space and to ensure reexpansion of the remaining lung parenchyma.

- A. Chest tubes may be connected either to a **simple underwater-seal system** or to **vacuum suction**. The two most commonly used systems are the Pleurovac and Emerson systems. Both systems may be placed to a water seal (providing -3- to -5-cm H₂O suction) or to vacuum suction (typically -20 cm).
 1. **If the water-seal chamber bubbles with expiration or with coughing**, this is evidence that an air leak persists. The Pleurovac system suction is limited by the height of the water column in the suction control chamber, the maximum suction being nearly -25 cm H₂O.
 2. **If greater suction is required to control an air leak**, one must switch to the bottle system with Emerson suction, which allows for increased vacuum suction up to approximately -50 cm H₂O.
- B. Most commonly after **lobectomy**, two chest tubes are used. One is placed apically for evacuation of air and the other posteroinferiorly for drainage of fluid.
- C. **Chest-tube drainage** is not used routinely with pneumonectomy unless bleeding or infection is present. Some surgeons place a chest tube on the operative side and remove it on postoperative day 1. Balanced pneumonectomy Pleurovacs have been advocated to balance the mediastinum over the first 24-48 hours. A chest tube in the patient with a pneumonectomy space should not be placed to conventional suction because of the risk of cardiac herniation.
- D. **Chest tubes should never be clamped for an extended period** because a persistent air leak may lead to tension pneumothorax. If a slow air leak is suspected, the tubes may be clamped for a few hours before a chest X-ray is obtained. The patient should be carefully monitored during this interval.
- E. **Chest tubes are removed after the air leak has resolved and fluid drainage decreased** (usually <100 mL over 8 hours). Chest tubes usually are removed one at a time. The patient is instructed to take a large inspiratory breath and hold it while the tube is removed swiftly and the site is simultaneously covered with an occlusive dressing. The technique of chest tube removal is important to prevent air entry through the removal site.

IV. Cardiovascular complications in the postoperative period are second in frequency only to pulmonary complications, because the population that develops lung cancer is at high risk for heart disease. The three most common sources of cardiac morbidity are arrhythmias, myocardial infarctions, and congestive heart failure. A negative preoperative cardiac evaluation does not preclude the development of postoperative complications.

- A. **Cardiac arrhythmias** occur in up to 30% of patients undergoing pulmonary surgery. The highest incidence occurs in elderly patients undergoing pneumonectomy or intrapericardial pulmonary artery ligation. All patients should have cardiac rhythm monitoring after thoracotomy for at least 48 hours.
 1. **Treatment** of any rhythm disturbance begins with an assessment of the patient's hemodynamic status. Manifestations of these arrhythmias vary in acuity

from hemodynamic collapse to palpitations. If the patient is hemodynamically unstable, the advanced cardiac life support protocol should be followed (see [Chapter 3](#)). After the patient has been examined and hemodynamic stability confirmed, an electrocardiogram, arterial blood gas sample, and serum electrolyte panel should be obtained. Frequently, supplementary oxygen and aggressive potassium and magnesium replenishment are the only treatment necessary. Premature ventricular contractions often are signs of myocardial ischemia. They should be treated expediently with electrolyte correction and optimization of oxygenation and evaluation for ischemia.

2. **Supraventricular arrhythmias** (of which atrial fibrillation and flutter are by far the most common) are treated with initial correction of electrolytes and supplemental oxygen. If the rhythm does convert and the patient is not hypotensive, intravenous diltiazem often slows nodal conduction enough to allow conversion back to sinus rhythm. Beta-blockers are to be used with extreme caution because of their bronchoconstrictive side effects. Failure to convert to sinus rhythm may be an indication for antiarrhythmic drugs such as amiodarone. If hypotension occurs secondary to the atrial arrhythmia, electrical cardioversion is indicated.
- B. Chest pain associated with myocardial infarction** often goes unnoticed by caretakers and patients due to thoracotomy incisional pain and narcotic administration.
1. **Most patients experiencing perioperative ischemic events** have a history of coronary artery disease. When these events go unrecognized, patients have a significant perioperative mortality (>50% in some series) (*JAMA* 239:2566, 1978).
 2. When electrocardiographic evidence of ischemia or significant clinical suspicion of an impending cardiac event exists, the patient should be moved to an intensive care unit and treated expectantly with intravenous nitroglycerin and Swan-Ganz catheter monitoring. Every effort should be made to decrease myocardial oxygen demand.

V. The perioperative fluid management of thoracic surgery patients differs from that of patients after abdominal surgery. Pulmonary surgery does not induce large fluid shifts. In addition, collapse and reexpansion of lungs during surgery can lead to pulmonary edema. Pulmonary edema should be treated with aggressive diuresis. This is largely due to the limited pulmonary reserve, most graphically demonstrated in the pneumonectomy patient in whom 100% of the cardiac output perfuses the remaining lung. Judicious fluid management, including avoiding fluid overload and pulmonary edema, is critical in patients with limited pulmonary reserve. Discussions regarding the intraoperative fluid management should be held with the anesthesiologist before surgery. Physicians may need to accept transiently decreased urine output and increased serum creatinine. Mild hypotension may be treated with intravenous alpha-agonists such as phenylephrine. Cardiac dysfunction may also be the source of postoperative oliguria, pulmonary edema, and hypotension and should always be considered in patients who are not responding normally. Echocardiography or placement of a Swan-Ganz catheter may guide treatment.

Introduction**Care of the Pediatric Surgical Patient****I. Fluid, electrolytes, and nutrition****II. Preoperative preparation****III. Vascular access****IV. Common neonatal surgical problems****V. Problem-oriented approach to the treatment of surgical disease in children**

Introduction

Pediatric surgery involves the care of fetuses, neonates, children, and adolescents with surgical disorders. Although many diseases managed by the pediatric surgeon are unique to this age group, others, such as gastroesophageal reflux or inflammatory bowel disease, are managed similarly in adults. Aside from the obvious difference in the size of the pediatric surgeon's patient, the physiology of children, especially the neonate, provides many unique challenges to this diverse field. The focuses of this chapter are the diagnosis and treatment of selected problems common in pediatric surgical practice.

Care of the Pediatric Surgical Patient

I. Fluid, electrolytes, and nutrition are essential components in the overall care of the pediatric surgical patient.

A. Fluid requirements

1. **Normal daily fluid requirements for children**, especially premature infants, must account for insensible losses as a result of high body-surface area to volume ratio and immature kidneys in the neonate, which have a limited ability to concentrate urine. Fluid replacement can be calculated based on body weight, as shown:

Weight (kg)	24-Hour fluid requirements
<2 (premature)	140–150 mL/kg
2–10	100 mL/kg
11–20	1,000 mL + 50 mL/kg >10 kg
>20	1,500 mL + 20 mL/kg >20 kg

A simple method for calculating a child's hourly maintenance fluid requirements is to use 4 mL/kg per hour for each kilogram of the first 10 kg weight, plus 2 mL/kg per hour for each kilogram from 10 to 20 kg, plus 1 mL/kg per hour for each kilogram over 20 kg. For example, a 22-kg child would require (10 kg × 4 mL/kg) + (10 kg × 2 mL/kg) + (2 kg × 1 mL/kg), or 40 mL + 20 mL + 2 mL = 62 mL per hour as a maintenance rate.

2. **Postoperative fluid replacement** is determined from the normal daily requirements and losses incurred from any disease process and surgical procedures. Such third-space losses should be replaced with a balanced salt solution such as lactated Ringer's.
 - a. As a **first estimate**, an additional 25% of the calculated daily maintenance volume should be given for each quadrant of the peritoneal cavity involved or entered (*Ann Surg* 196:76, 1982).
 - b. **Output from tubes and drains** should be matched by replacement fluid with similar electrolyte composition.
 - c. The **total fluid administered** ultimately should be adjusted to support urine output between 1 and 2 mL/kg per hour.
 - d. **Central venous pressure monitoring** can be used to estimate intravascular volume more accurately.
 - e. In **full-term newborns**, total body water is 80% of body weight.
 - f. **Total blood volume in the newborn** is approximately 8% of body weight, which decreases to 5% in older infants.
- B. **Electrolyte requirements** generally are met in pediatric patients when the normal daily fluid requirements are given according to the following guidelines.
 1. **Children younger than 6 months** should be given 10% dextrose in 0.25% saline with potassium chloride, 20 mEq/dL, as a maintenance fluid.
 2. **Children older than 6 months** should be given 5% dextrose in 0.45% saline with potassium chloride, 20 mEq/dL, for maintenance fluid replacement.
 3. **Daily sodium requirements** are 2–3 mEq/kg, and daily potassium requirements are 1–2 mEq/kg.
 4. The **electrolyte composition** of gastric, pancreatic, and biliary secretions is similar in adults and children.
- C. **Nutritional requirements** are increased in infants and children compared to adults.
 1. The **minimum daily caloric needs for children** can be calculated using the table shown in [section I.A.1](#) (for daily fluid requirements), substituting kcal/kg for mL/kg.
 2. **Additional nutritional demands** are made on infants and children affected by stresses such as sepsis, burns, trauma, or fever. These excess caloric needs must be met to prevent the development of a catabolic state.
 3. **Carbohydrates** should supply 50%, **fats** 35%, and **protein** 15% of total calories in the diet.
 4. **Enteral nutrition** is the preferred method to deliver calories to a patient if the gastrointestinal (GI) tract is functioning. Otherwise, parenteral nutrition is indicated.
 5. Most **infant formulas** contain 20 kcal/oz; therefore, caloric needs can be calculated by the following formula:

Weight (kg) × 6 oz = volume of formula necessary to deliver 120 kcal/kg

6. A **newborn weight gain** of 15–30 g per day is expected.

II. Preoperative preparation

- A. A thorough and succinct **history and physical examination** are always required. Occasionally, patients have **signs and symptoms** of an upper respiratory infection, which may require postponement of an elective operation. The acuteness of the illness and the urgency of the procedure planned must be considered. Other signs, such as fever, leukocytosis, decreased appetite, or lethargy, may necessitate a delay of the procedure. Severe diaper rash with a planned groin surgery and signs of child abuse are other reasons to postpone surgery.
- B. **Laboratory tests** are not indicated routinely. The decision to obtain any preoperative laboratory tests should be made jointly by the surgical and anesthesiology teams on an individual basis. Typically, only a hematocrit is required for elective procedures.
- C. **Abstinence from food and drink** for a prolonged preoperative period is not required in children. Studies have indicated that clear liquids ingested 2–3 hours before induction of anesthesia do not increase the risk of aspiration (*Anesthesiology* 72:593, 1990).
 1. **Aspiration risks** can also be reduced by administering H₂-receptor antagonists, metoclopramide, and buffering agents, such as sodium citrate.
 2. **Gastric decompression preoperatively** may require a longer duration of n.p.o. status as well as intravenous hydration.
 3. **Standard n.p.o. guidelines** according to age are as follows (Preoperative evaluation. In: Rasch DK, Webster DE, eds. *Clinical manual of pediatric anesthesia*. New York: McGraw-Hill, 1994)
 - a. **Newborn to 1-year-old children** may receive regular formula feedings until 5 hours preoperatively. Clear liquids may be given until 2 hours before surgery.
 - b. **One- to 14-year-old children** may eat solid food until midnight the night before surgery. Clear liquids may be taken up to 3 hours before the procedure.
 - c. **Fourteen- to 19-year-old children** should have nothing to eat or drink after midnight the night before surgery.
- D. **Preoperative antibiotic prophylaxis** is required for patients with cardiac anomalies who are undergoing noncardiac surgery. Patients with ventriculoperitoneal shunts and those who have other prosthetic material should receive antibiotic prophylaxis. The most commonly recommended antibiotics are ampicillin (50 mg/kg), erythromycin (10 mg/kg), and clindamycin (10 mg/kg). Although specific recommendations may vary, usually one dose is given 1 hour before the

procedure, and the final dose is given 6 hours after the procedure.

III. Vascular access

- A. **Peripheral venous access** often is challenging in pediatric patients, especially neonates. However, cannulation of a vein can usually be accomplished in one of the following sites: dorsal vein of the hand, antecubital vein of the arm, saphenous vein at the ankle, dorsal vein in the foot, median tributary at the wrist, external jugular vein, or scalp veins.
- B. **Percutaneous central venous catheter insertion** is the method of choice when peripheral access is not available. The sites most often chosen are the subclavian vein, internal jugular vein, and femoral vein. Central venous catheter insertion often requires sedation before the procedure, but it can be done safely in newborns. Complications of central venous catheters include air embolus, pneumothorax, infection, and thrombosis. Strict adherence to sterile technique and regular dressing care is the best prevention against infection. If thrombosis of the catheter occurs, thrombolytic therapy may be effective at dissolving the clot. When occlusion is suspected, urokinase may be infused through the catheter. Urokinase, 2 mL (5,000 units/mL), is given for Hickman catheters and 0.8 mL for Broviac catheters. One hour after infusion of urokinase, the catheter should be aspirated and flushed with heparinized saline (*J Pediatr Surg* 24:257, 1989). This routine may be repeated twice more if necessary.
- C. **Saphenous vein cutdown** has been used for the pediatric patient in whom vascular access is not obtainable by a percutaneous approach. Even in experienced hands, however, a venous cutdown can be time consuming, and, therefore, during an emergency situation, alternative routes for the administration of fluids and medications must be sought.
- D. When performing a **venous cutdown in an elective situation**, it is helpful to use a peripherally inserted central catheter, or PCVC catheter. The saphenous vein at the saphenofemoral junction or the vena comitantes of the brachial artery are the usual sites chosen.
- E. **Intraosseous infusion** should be used in an emergency setting when other attempts at obtaining vascular access have failed. A location 1–3 cm distal to the tibial tuberosity is the recommended site. The needle should be directed inferiorly during insertion. Alternatively, the femur may be reached by inserting the needle in a cephalad direction 3 cm proximal to the condyles. Either a bone marrow aspiration needle or a 16- to 19-gauge butterfly needle is adequate.
- F. **Arterial catheters** are needed in some neonates and infants. The potential sites for placement of an arterial catheter include the umbilical, radial, femoral, posterior tibial, and temporal arteries; however, the radial artery is used most frequently. The typical catheters are 22- to 24-gauge and can be placed percutaneously or by cutdown. In a neonate who is within the first 2–4 days of life, the umbilical artery can often be cannulated through the umbilical stump.

IV. Common neonatal surgical problems

- A. **Congenital diaphragmatic hernia (CDH)** occurs when the diaphragm fails to form completely, allowing the abdominal organs to herniate into the chest. This anomaly occurs on the left in 90% of cases and on the right in 10%. The herniated viscera act as a space-occupying lesion, preventing normal lung development, primarily on the affected side. Thus, cardiorespiratory compromise is the hallmark of this disease.
 1. **Diagnosis of CDH** often is made prenatally by ultrasonography. In most cases, a child with CDH presents with respiratory distress, and bowel loops are seen in the chest on plain radiography.
 2. **Mortality** is directly related to the degree of pulmonary hypoplasia, pulmonary hypertension, and presence of associated congenital anomalies, including cardiac defects and chromosomal abnormalities.
 3. **Management**
 - a. **Immediate postnatal care** of an infant who has CDH includes several supportive measures.
 1. **Supplemental oxygen** is needed to maximize hemoglobin saturation.
 2. **Adequate ventilation** is critical to maintain gas exchange. If signs and symptoms of respiratory distress are present, endotracheal intubation is indicated. Avoid bag-mask ventilation, which exacerbates GI distention and further impedes lung ventilation.
 3. **Intravenous access** is necessary for the administration of fluids to maintain organ perfusion.
 4. **GI decompression** by orogastric or nasogastric intubation reduces distention of the stomach and intestines if they are encroaching on the lung.
 5. **Transport of the child** can be done safely once the preceding measures are complete. The patient should be positioned to avoid compression of his or her lung on the unaffected side. In addition, GI decompression should be continued throughout travel.
 - b. **Intensive care** delivered once the infant is at an appropriate referral center is aimed at controlling pulmonary hypertension and minimizing the right-to-left cardiac shunt.
 1. **Mechanical ventilation** is the primary means for respiratory support. Some centers have reported success by permitting a moderate hypercapnia with less aggressive ventilatory management. Some studies have shown the importance of ventilatory strategy in preventing lung injury.
 2. **Permissive hypercapnia** is a strategy designed to minimize barotrauma to the hypoplastic lungs. Ventilator settings are adjusted to accept a pH greater than 7.20, an arterial carbon dioxide tension of 60–70 mm Hg, and a preductal oxygen saturation greater than 90%. When peak airway pressures exceed 30 cm H₂O and mean airway pressures exceed 20 cm H₂O, the child is placed on extracorporeal membrane oxygenation.
 3. **If conventional ventilator techniques are inadequate**, the high-frequency oscillating ventilator and inhaled nitric oxide may be useful adjuncts.
 4. **Extracorporeal membrane oxygenation** to maintain adequate ventilation should be considered if the patient remains hemodynamically unstable or when peak ventilatory pressure exceeds 30 cm H₂O.
 - c. **Definitive surgical repair** usually is deferred until the patient's pulmonary status is stable. A subcostal incision on the affected side allows the herniated abdominal contents to be replaced in the peritoneal cavity. The defect is either repaired primarily or repaired with a synthetic patch, depending on the size of the defect. A 12 French chest tube can be placed to water-seal the patch.
- B. **Tracheoesophageal malformations** represent a spectrum of anomalies, including esophageal atresia (EA) and tracheoesophageal fistula (TEF), separately or in combination ([Fig. 38-1](#)). These are believed to arise from an interruption in the normal budding of the trachea from the foregut during embryonic development.

Fig. 38-1. Variants of tracheoesophageal fistula. **A:** Atresia without fistula (5–7% of cases). **B:** Proximal fistula and distal pouch (<1% occurrence). **C:** Proximal pouch with distal fistula (85–90% of cases). **D:** Atresia with proximal and distal fistulas (<1% of cases). **E:** Fistula without atresia (H-type) (2–6% occurrence).

1. The **diagnosis** of a tracheoesophageal malformation varies, depending on the type of anomaly present.
 - a. **EA**, with or without TEF, should be recognized in the immediate newborn period by the following symptoms and signs.
 1. **Excessive drooling**
 2. **Regurgitation** of feedings
 3. **Choking, coughing, or cyanosis** during feeding
 4. **Coiled orogastric tube** in the esophageal pouch on plain radiography
 5. **Gas in the GI tract** on plain radiograph, implying the presence of a distal TEF
 - b. **TEF usually is associated with EA.** Isolated TEF (“H-type” fistula; [Fig. 38-1E](#)) may present later in childhood and can be identified by the following signs and symptoms.
 1. **Choking or coughing** during ingestion of **liquids**
 2. **Recurrent pneumonia**
 3. **Abdominal distention**
 4. **Excessive flatulence**
 5. **Contrast in the trachea** on upper GI study
 6. A **fistula** visualized at bronchoscopy
2. **Management** of a suspected EA in a newborn must be carried out expeditiously.
 - a. Place the infant in the **prone position** with the **head elevated**.
 - b. Insert an **orogastric or nasogastric tube** into the proximal esophageal pouch for decompression and to prevent aspiration.
 - c. Administer **ampicillin (50 mg/kg)** and **gentamicin (2.5 mg/kg)** intravenously.
 - d. **Transport** the patient rapidly, accompanied by personnel experienced in neonatal airway management.
 - e. Once the child is at a tertiary care facility, an experienced surgeon should urgently gain **control of the TEF**.
 1. **Surgical ligation of the fistula** and **repair of the EA** at one anesthetic is the preferred management and is performed through a right extrapleural thoracotomy.
 2. In **neonates** too ill to withstand an operation, bronchoscopic placement of a **balloon-tipped catheter** to occlude the fistula is required.

- f. Integral to the **perioperative management** is an investigation for other anomalies that may occur in association with tracheoesophageal malformations. These anomalies are known as **VACTERL** (vertebral defects, imperforate anus, cardiac defects, tracheoesophageal malformations, renal dysplasia, and limb anomalies). Echocardiography and renal ultrasonography are necessary to screen for associated life-threatening anomalies.
- C. **Gastroschisis** is an abdominal wall defect that is believed to arise from an isolated vascular insult in the developing mesenchyme, which then impairs normal epidermal development. Typically, the midgut and stomach herniate through this defect. No membranous sac covering the abdominal organs exists. Unlike omphalocele, the incidence of associated anomalies is infrequent with gastroschisis.
1. **Diagnosis**
 - a. **Prenatal discovery** of gastroschisis is the most common timing of presentation.
 1. **Ultrasound examination** of a fetus after 13 weeks may reveal extraabdominal intestine with no echogenic covering. This intestine is located apart from the umbilical cord.
 2. **Maternal alpha-fetoprotein (AFP) concentrations** are markedly elevated.
 - b. A lack of **prenatal diagnosis** is uncommon if the mother receives appropriate prenatal care. When an unsuspected abdominal wall defect is recognized at birth, however, accurate pathologic description is key to the proper diagnosis.
 1. The **defect** usually is less than 4 cm in diameter.
 2. The **umbilical cord** is normal.
 3. The **intestines and stomach** typically are extruded from the abdomen to the right of the umbilical cord.
 4. The **bowel** may be matted and foreshortened.
 5. A thick, **fibrinous peel**, which highlights an inflammatory process, covers the intestine.
 2. **Management**
 - a. **Prenatally**, serial ultrasonography helps to monitor the course of the fetus with gastroschisis. If bowel dilatation and mural thickening of the eviscerated intestine are detected, delivery at the time of lung maturity may be indicated (*Surg Obstet Gynecol* 81:53, 1993).
 - b. **Postnatally**, resuscitation and surgical treatment of gastroschisis is **emergent**. The goals should be to hydrate the patient adequately and to minimize heat loss perioperatively.
 1. **Intravenous fluids** should be started with an initial bolus of normal saline at 20 mL/kg body weight. Subsequently, 5% dextrose in lactated Ringer's solution should be infused at a rate that is three to four times maintenance requirements until the urine output reaches 1.5–2.0 mL/kg per hour.
 2. **Exposed bowel** should be covered with moistened gauze and wrapped with dry gauze to hold the bowel in place and prevent vascular occlusion.
 3. **Heat and fluid losses** can be decreased by placing the infant in a plastic bag up to the neck.
 4. **Intestinal decompression** is advised, via a nasogastric or orogastric tube applied to low continuous suction.
 5. **Extension of the fascial defect** in the midline for 1–2 cm can be done if the bowel mesentery appears to be compressed by a narrow opening.
 6. **Broad-spectrum antibiotics** should be administered.
 7. **Expedient transport is crucial**. Delays should not occur owing to difficulty in obtaining intravenous access.
 8. **Reduction of the viscera and primary closure of the abdomen** are feasible in most patients. When ventilation or venous return to the heart is compromised by this technique, a synthetic silo can be constructed to facilitate a staged reduction.
 9. **The use of a premanufactured spring-loaded silo**, which can be placed in the delivery room or on arrival to the neonatal intensive care unit, has allowed definitive repair after the abdominal viscera are sequentially reduced (12–36 hours).
- D. **Omphalocele** is an abdominal wall defect in which the intestines protrude through the base of the umbilical cord and herniate into a sac. One theory holds that this anomaly is a persistent body stalk that is normally obliterated by the lateral mesoderm of the developing abdominal wall. Along with the abdominal wall defect is a high incidence of associated anomalies.
1. **Diagnosis** is made based on information similar to that needed to diagnose gastroschisis (see [section IV.C.1](#)).
 - a. **Prenatal studies** are the most common means by which to make the diagnosis.
 1. **Ultrasound examination** beyond 13 weeks of fetal development may reveal intestine herniating through the base of the umbilical cord. This intestine is covered by a smooth, echogenic sac. Because of the high incidence of associated anomalies, the presence of an omphalocele should direct a thorough search for other birth defects by ultrasonography and amniocentesis.
 2. **Maternal AFP levels**, as in gastroschisis, are markedly elevated.
 - b. **Postnatal diagnosis** is unusual, as in gastroschisis, and is made readily on physical examination.
 1. The defect is at the **base of the umbilical cord**.
 2. A **sac covering the intestine** is present and is composed of an external layer of amnion and an internal layer of peritoneum with mesenchyme (Wharton's jelly) in between.
 3. The **size of the defect** varies from a few centimeters to absence of most of the abdominal wall.
 4. The **contents of the sac** may include bowel alone, but frequently the liver or the entire GI tract is present.
 5. **Rupture of the sac** is infrequent, but it is distinguished from gastroschisis by the presence of residual sac in continuity with the umbilical cord.
 2. **Postnatal management**. The care of a patient with an omphalocele should be conducted according to the same principles as for gastroschisis; however, the timing is urgent rather than emergent.
 - a. **Intravenous fluid** usually is administered at a rate to achieve adequate urine output.
 - b. **The sac can be protected** by covering the defect with moistened gauze, followed by a bulky dressing.
 - c. **Heat and fluid conservation** is achieved [see [section IV.C.2.b.\(3\)](#)].
 - d. **GI decompression** via a nasogastric or orogastric tube is required [see [section IV.C.2.b.\(4\)](#)].
 - e. **Broad-spectrum antibiotics** are administered intravenously [see [section IV.C.2.b.\(6\)](#)].
 - f. **Transport of the child** should be immediate.
 - g. **Rupture through the sac** demands that the defect be treated as a gastroschisis.
 - h. **Repair of the abdominal wall defect** depends on its size. As for gastroschisis, primary closure of an omphalocele can be accomplished only if, when the bowel is reduced, the patient's ventilation and venous return to the heart are not compromised. Delayed primary closure with the use of a synthetic silo allows for gradual reduction of the intestines when the defect is large.
 - i. **The overall prognosis** for infants with omphalocele is significantly worse than that for gastroschisis owing to the anomalies associated with omphalocele.
- E. **Necrotizing enterocolitis (NEC)** is a syndrome characterized by discontinuous areas of bowel damage, which range from mucosal ulceration to gangrenous bowel to intestinal perforation. The inciting cause is unknown; however, most evidence supports the theory that perinatal stress produces selective intestinal ischemia. This disease primarily affects premature infants and accounts for 2% of all neonatal intensive care admissions, yet no definitive risk factors for NEC have been established.
1. **Diagnosis** of NEC is largely clinical. Radiographic studies and laboratory tests are used only to support a diagnosis based on a careful history and physical examination.
 - a. **Signs and symptoms** that characterize NEC include the following:
 1. **Abdominal distention**
 2. **High gastric residuals**
 3. **Vomiting** or bilious nasogastric tube drainage
 4. **Hematochezia**
 5. **Diarrhea**, which can be bloody
 6. **Temperature instability**
 7. **Apnea**
 8. **Lethargy**
 9. **Skin pallor or mottling**
 - b. **Physical examination findings** are not specific for NEC but can demonstrate the intraabdominal complications of this condition.
 1. The **abdomen** may be firm and distended, with bowel loops visible through the anterior wall.
 2. **Erythema or frank cellulitis** of the abdominal skin indicates either peritonitis or a local response to subjacent inflamed bowel.
 - c. **Plain radiographs** may reveal any of several findings that are consistent with NEC.
 1. **Pneumatosis intestinalis** is the most characteristic radiographic sign of NEC.
 2. **Dilated loops of intestine** often are present, particularly in an asymmetric pattern.
 3. **An intestinal loop that is static** on serial films may be recognized.
 4. **Ascites** may appear acutely on serial radiographs.
 5. **Intrahepatic portal vein gas** may be seen.
 6. **Pneumoperitoneum** may be apparent if intestinal perforation has occurred.
 7. **Gastric distention** may be pronounced as a result of a paralytic ileus.
 - d. **Laboratory tests** also are not specific for NEC, but the following suggest the onset of sepsis:
 1. **Metabolic acidosis**

2. **Thrombocytopenia**
3. **Leukocytosis or leukopenia**
2. The **management** of NEC varies, depending on the severity of the disease and the general condition of the infant.
 - a. **Nonoperative treatment** for NEC consists of bowel rest, nasogastric decompression, removal of umbilical catheters (if possible), and initiation of nutrition and broad-spectrum antibiotics parenterally. A good response to this therapy is indicated by decreased gastric residuals, diminished abdominal distention, and clearing of blood from the stool. Oral nutrition should be withheld for 7–14 days in these patients.
 - b. **Absolute indications for operative treatment** of NEC are as follows:
 1. **Intestinal perforation**
 2. **Intestinal obstruction**
 3. **Intraabdominal abscess**
 4. **Peritonitis**
 - c. **Relative indications for surgery** include the following:
 1. **Sepsis unresponsive to medical treatment.**
 2. **Inflammatory changes in the abdominal wall.**
 3. **Radiographic signs**, such as a static intestinal loop, asymmetrically dilated loops, or ascites, all of which suggest intestinal gangrene.
 4. **Laboratory tests** that reveal thrombocytopenia, coagulopathy, severe hyponatremia, or intractable acidosis, all of which suggest bowel necrosis.
 5. **Paracentesis fluid** that is brown and cloudy, contains bacteria (as revealed by Gram stain), or contains a high leukocyte count, with neutrophils predominating.
 6. **The operation performed depends on the pathology found.** Most often, resection of the involved intestine is done with the creation of stomas. When intestinal viability is questionable, reexploration within 24 hours is essential.
 - d. **Open peritoneal drainage** in critically ill neonates weighing less than 1,000 g can be a successful therapy. This procedure is performed at the Isolette by inserting a Penrose drain through a right lower quadrant abdominal incision. If not definitive treatment, this drainage technique may stabilize the infant's condition until he or she is better able to tolerate a laparotomy (*J Pediatr Surg* 25:1034, 1990).

V. Problem-oriented approach to the treatment of surgical disease in children

- A. **Alimentary tract obstruction** in a child may present with nausea, vomiting, GI bleeding, and abdominal pain and distention. Often, clues within this presentation exist that can direct an evaluation toward the site of obstruction or signal the presence of a life-threatening illness. For example, nonbilious emesis is the distinguishing feature of obstruction proximal to the ampulla of Vater, particularly pyloric stenosis. Another example is that the more distal an obstruction is, the more distended the abdomen can become. Alternatively, bilious emesis in an infant or child may signal intestinal malrotation with midgut volvulus, which can be lethal if not detected early in its course. The etiologies of intestinal obstruction include congenital and acquired diseases.
 1. **Congenital causes** of alimentary tract obstruction typically present in the newborn period.
 - a. **Intestinal malrotation** occurs when the intestine fails to undergo its normal rotation and fixation during embryologic development. Consequently, the small-bowel mesentery develops on a narrow base, and volvulus can occur.
 1. **Diagnosis** of malrotation is made from the clinical presentation and contrast radiographs. **Signs and symptoms** of midgut volvulus include bilious emesis, abdominal distention, pain, and hematochezia or hematemesis. The presence of these findings should immediately lead to an imaging study to evaluate this emergent situation. An upper GI contrast study is necessary to identify the ligament of Treitz, which normally is located at the level of the pylorus to the left of the midline. A corkscrew deformity of the proximal small intestine or a beak-shaped termination of the contrast column in the jejunum is diagnostic of midgut volvulus. Alternatively, a delayed or chronic manifestation of intestinal malrotation can present with long-standing bilious emesis, colic, diarrhea, vague abdominal pain, and failure to thrive. This history may represent intermittent midgut volvulus or partial intestinal obstruction without vascular compromise.
 2. **Management** of malrotation is surgical correction. If the intestine is not necrotic, the volvulus should be reduced by counterclockwise rotation and the peritoneal bands (Ladd's bands) divided. Infarcted bowel should be removed and a primary anastomosis performed. The bowel is placed in a state of nonrotation, in which the colon is positioned on the left side of the abdomen and the small bowel on the right to restore a long distance between the duodenojejunal junction and the ileocecal junction. To complete the procedure, an appendectomy is performed. If the possibility of the child developing short-gut syndrome exists, questionably viable intestine should not be resected. A repeat laparotomy 24 hours later is necessary to assess viability of the intestine.
 - b. **Intestinal atresia or stenosis** can occur anywhere from the duodenum to the colon. The distal ileum, proximal jejunum, and duodenum are the most common sites for atresias.
 1. **Diagnosis** of intestinal atresia may be suspected antenatally if polyhydramnios is present on ultrasonography. In the newborn period, babies typically present with bilious vomiting, abdominal distention, and failure to pass meconium. Infants with intestinal stenosis may **present** at a few weeks to months of age with vomiting, failure to thrive, and poor feeding. A **plain abdominal radiograph** should be obtained to delineate the level of obstruction. A "double-bubble" sign is diagnostic of duodenal obstruction. Contrast enema may be used to rule out a functional obstruction, such as meconium ileus or meconium plug syndrome.
 2. **Management** of intestinal atresia should begin with nasogastric decompression and intravenous fluid administration to maintain a urine output of 2 mL/kg per hour. Ampicillin (50 mg/kg) and gentamicin (2.5 mg/kg) should be started immediately. Surgical repair consists of resection of the dilated proximal segment and primary anastomosis. In contrast, for duodenal atresia, a duodenoduodenostomy is created to bypass the obstruction. Finally, during the operation, saline should be infused into the distal bowel to rule out synchronous intestinal atresias.
 - c. **Hirschsprung's disease** is a congenital disorder characterized by a variable length of intestinal aganglionosis of the hindgut.
 1. **Diagnosis** of Hirschsprung's disease is suggested by the history and is confirmed by histologic proof of absence of ganglion cells within the bowel wall. Neonates classically **present** with abdominal distention, bilious vomiting, and infrequent or delayed defecation. Rarely, the first manifestation of Hirschsprung's disease is enterocolitis with sepsis. Older infants and children present with chronic constipation or failure to thrive. Plain abdominal radiographs of patients with Hirschsprung's disease commonly show a pattern of distal obstruction. **Barium enema** usually demonstrates a transition zone between distal nondilated bowel and proximal dilated bowel. This transition zone is found most commonly in the rectosigmoid but may be seen anywhere in the colon. In neonates, a transition zone may not be identifiable, and patients with total colonic aganglionosis do not have a transition zone. **Rectal biopsy** is essential to make the diagnosis of Hirschsprung's disease. Full-thickness specimens are the ideal tissue samples to allow for identification of the absence of ganglion cells in Auerbach's myenteric and Meissner's submucosal plexus. In neonates, rectal suction biopsy often is sufficient to make the definitive diagnosis of Hirschsprung's disease.
 2. **Management** of patients with Hirschsprung's disease initially should include **colonic decompression** to prevent enterocolitis. Saline enemas may be used to evacuate impacted stool. A **nasogastric tube** should be placed if the child is vomiting. Operative treatment in patients who are unstable or who have massively dilated bowel involves placing a **diverting colostomy** proximal to the aganglionic segment until definitive reconstruction is performed. Daily rectal irrigations may obviate the need for a colostomy. **Reconstruction of intestinal continuity** involves bringing ganglionated bowel to within 1 cm of the anal verge. The three classic methods for this reconstruction are the procedures developed by Swenson, Duhamel, and Soave. Each of these operations has been modified to improve functional results and usually is reserved until the patient reaches 6–12 months of age. A primary pull-through procedure during infancy can be performed in many cases.
 - d. **Anorectal anomalies** can be classified as high, intermediate, or low, depending on whether the rectal atresia has descended below the puborectalis sling, is at the level of the puborectalis, or remains above that level. Eighty percent to 90% of infants with imperforate anus have an associated fistulous tract originating from the rectal segment.
 1. In **boys**, the fistulous tract is usually to the perineum (low) or to the urethra (intermediate or high). An infant with fistula to the urethra is best managed by initial diverting sigmoid colostomy as a neonate, with subsequent posterior sagittal anorectoplasty between 6 months and 1 year of age.
 2. **Girls** have a variety of anomalies, but intermediate or high rectal atresia with rectovaginal fistula also requires a colostomy in the neonatal period.
 3. **The higher the atresia, the worse the outcome** in terms of obtaining fecal continence. It is important that the puborectalis sling as well as the deep and superficial fibers of the external sphincter be preserved at the time of the pull-through procedure.
 4. Many children with anorectal anomalies have **sacral vertebral anomalies** or spinal dysraphic syndromes. Other anomalies in the VACTERL complex (see [section IV.B.2.f](#)) should be considered.
 - e. **Omphalomesenteric duct abnormalities** result from incomplete involution of the vitelline duct, which connects the primitive gut to the yolk sac. The omphalomesenteric duct remnant may persist as any of the following (which occur within 60 cm proximal to the ileocecal valve): (1) a simple ileal outpouching found on the antimesenteric side of the intestine, known as **Meckel's diverticulum** (a true diverticulum); (2) a **patent duct** between the ileum and the umbilicus; (3) an **umbilical cyst** lined by intestinal mucosa; or (4) a **fibrous cord** between an ileal diverticulum and the umbilicus or mesentery.
 1. **Diagnosis** of omphalomesenteric duct abnormalities depends on the type of remnant that persists. A **patent duct** is recognized by spillage of ileal contents through the child's umbilicus. **Umbilical cysts** of vitelline duct origin secrete a mucoid discharge at the umbilicus. A **fibrous band** remnant between the ileum and the abdominal wall usually presents as an internal hernia or intestinal volvulus around this cord of tissue, with the

accompanying signs of intestinal obstruction. A **Meckel's diverticulum** frequently contains ectopic gastric mucosa, resulting in peptic ulceration and hemorrhage in 22% of cases. This is recognized as painless rectal bleeding, which is often substantial and can be life threatening but usually is self-limited. Other complications of a Meckel's diverticulum include bowel obstruction caused by internal hernia around a vitelline duct band (13%), diverticulitis with abdominal pain (2%), and intussusception, with the Meckel's diverticulum acting as a lead point (<1%) (*Arch Surg* 122:542, 1987). More than one-half of the children with a symptomatic Meckel's diverticulum present before 2 years of age. Diagnostic evaluation of a patient suspected of having a Meckel's diverticulum should begin with a technetium-99m pertechnetate scintiscan. This isotope is taken up by the ectopic gastric mucosa of the Meckel's diverticulum.

2. **Management** of omphalomesenteric duct abnormalities is **surgical resection** of the remnant. When GI bleeding is the indication for the operation, a diverticulectomy plus either oversewing or resection of the bleeding site is recommended. Laparoscopic resection of the diverticulum using a stapling device can be performed. Occasionally, a large feeding vessel crosses onto the surface of the diverticulum from the mesentery, and this may require separate dissection and clipping or ligation. When radiographic reduction of an intussusception is unsuccessful, surgical reduction of the invaginated intestine and diverticulectomy should be attempted. If gangrenous bowel is present, bowel resection with primary anastomosis should be done. In infants or young children who have a Meckel's diverticulum discovered incidentally at laparotomy, diverticulectomy should be performed, particularly when a palpable mass is present within this enteric outpouching, because it may contain gastric mucosa with the potential for ileal mucosal ulceration.
- f. **Meconium ileus** is an intestinal obstruction that occurs in neonates with cystic fibrosis and is caused by inspissated meconium.
 1. **Diagnosis** of meconium ileus can be suspected prenatally by the detection of polyhydramnios. However, the typical **presentation** is abdominal distention, bilious vomiting, and failure to pass meconium within 24–48 hours of life. Complicated meconium ileus has an acute onset within 24 hours of birth and is characterized by progressive abdominal distention, pneumoperitoneum, peritonitis, and abdominal wall inflammation. Hypovolemia and sepsis may ensue. **Plain abdominal radiographic findings** are variable. Dilated loops of small bowel are common. A mixture of air bubbles and meconium produces a ground-glass, coarse, granular, or soap bubble–like appearance. Air-fluid levels are infrequent. Intraabdominal calcifications suggest prenatal perforation with subsequent meconium peritonitis. A defined mass containing calcium may signify a pseudocyst. Ascites or pneumoperitoneum suggests perforation after birth. Alternatively, up to 35% of infants with complicated meconium ileus show no plain radiographic abnormalities. A **water-soluble contrast enema** can confirm the diagnosis by demonstrating a microcolon and pellets of inspissated meconium in the ileum.
 2. **Management** of meconium ileus depends on the severity of the illness. **Nonsurgical treatment** for uncomplicated cases consists of the administration of a hyperosmolar, water-soluble, contrast enema, which is both diagnostic and therapeutic. This solution draws fluid into the bowel lumen and causes an osmotic diarrhea. The patient should be well hydrated and given broad-spectrum antibiotics before this therapy. **Operative intervention** is indicated for complicated meconium ileus or when enema therapy fails. Initially, an ileotomy is performed, and irrigation with an acetylcysteine solution is attempted. If gentle irrigation does not flush out the meconium, a 14 French ileostomy T tube may be placed, and routine irrigations are done beginning on postoperative day 1. This operation generally has good results. If intestinal volvulus, atresia, perforation, or gangrene complicates the illness, nonviable bowel must be resected, and an ileostomy with mucous fistula is made. Irrigation can then be accomplished through the stoma until primary anastomosis is performed 2–3 weeks later. When oral feedings are advanced postoperatively, pancreatic enzyme supplementation is started.
- g. **Meconium plug syndrome** has classically been described in patients without cystic fibrosis to distinguish it from meconium ileus. Altered colonic motility or a viscous meconium mass is believed to cause impaired transit of bowel contents and, eventually, obstruction of the intestinal lumen.
 1. **Diagnosis** [see [section V.A.1.f.\(1\)](#)].
 2. **Management** (see [section V.A.2.c](#)). Rarely, surgery is needed to relieve the obstruction. Further evaluation includes a suction biopsy to rule out Hirschsprung's disease and a sweat chloride test to ensure that cystic fibrosis is not the etiology of the disease.
- h. **Intestinal duplications** are cystic or tubular structures lined by various types of normal GI mucosa, which usually are located dorsal to the true alimentary tract. These lesions frequently share a common muscular wall and blood supply with the normal GI tract and may occur anywhere from the mouth to the anus. In 20% of cases, enteric duplications communicate with the true GI tract.
 1. **Diagnosis** of alimentary tract duplication can be made occasionally by prenatal ultrasonography. Postnatally, obstructive symptoms, such as vomiting, abdominal pain, and distention, may occur if the duplication compresses the lumen of the adjacent hollow viscus. Additionally, on physical examination, the neonate may have a palpable abdominal mass. **Ultrasonography or GI contrast studies** most commonly confirm the diagnosis by demonstrating external compression or displacement of the normal alimentary tract. In cysts that contain ectopic gastric mucosa, technetium radioisotope scans may aid in the diagnosis.
 2. **Management** of alimentary tract duplications is **surgical resection** or **internal drainage** of the cyst. When the cyst is within the bowel mesentery, resection of the adjacent bowel often is necessary because of a shared blood supply. Duodenal duplications usually are managed by marsupialization of the cyst into the lumen of the duodenum, which minimizes the risk of damage to the biliary system. If the duplication cyst is tubular, a common channel between the cyst and the adjacent hollow viscus can be made. When gastric mucosa is found in a cyst distant from the stomach, either the cyst is excised or the gastric mucosa is stripped and the cyst lumen joined to the adjacent intestine.
2. **Acquired causes of alimentary tract obstruction** in children can present at any time postnatally, but typically after the newborn period.
 - a. **Pyloric stenosis** is the most common surgical cause of nonbilious vomiting in infants, affecting boys four times more often than girls.
 1. **Diagnosis** of pyloric stenosis can be made clinically and confirmed by imaging studies. **Vomiting** is characteristically forceful and occurs 30–60 minutes after feeding in neonates who are 2–5 weeks of age. Undigested formula in the vomitus may lead to switching formulas in an attempt to improve feeding tolerance, but this maneuver typically is unsuccessful. Dehydration may occur and is manifested by lethargy, absence of tears, a sunken anterior fontanelle, dry mucous membranes, and a decreased urine output (measured by a reduction in the weight of diapers and the frequency of diaper changes). The physical examination is most reliable in an infant who is calm and whose abdomen is relaxed. This task may be accomplished with the infant suckling in the mother's arms. Palpation of the abdomen to the right and above the umbilicus may reveal the hypertrophied pylorus. This “olive” is approximately 2 cm in diameter, firm, and mobile. To relax a hungry, agitated child, sugar water may be given by mouth. This technique consists of placing a nasogastric tube to decompress the stomach and providing a means of removing the fluid. An upper GI contrast study is helpful when physical examination is nondiagnostic. An enlarged stomach, poor gastric emptying, and an elongated, narrow pyloric channel or “string sign” are consistent with pyloric stenosis. Abdominal ultrasonography may be useful when the history and physical examination are equivocal. Identification of a pyloric diameter greater than 14 mm, muscular thickness greater than 4 mm, and pyloric length exceeding 16 mm is diagnostic of pyloric stenosis with 91–100% sensitivity and 100% specificity (*J Pediatr Surg* 22:950, 1987).
 2. **Management** of a patient with pyloric stenosis should begin once evidence of dehydration is found. Preoperative fluid resuscitation and correction of the hypochloremic metabolic alkalosis that results from long-standing vomiting are paramount. Initially, a 20-mL/kg i.v. bolus of normal saline is given, followed by 5% dextrose in normal saline at a rate needed to achieve a urine output of 2 mL/kg per hour. When urine output is adequate, 20 mEq/L potassium chloride can be added to the solution. As the serum Na⁺ and Cl⁻ concentrations return toward normal, the intravenous solution can be switched to 0.45% saline. Surgery should be undertaken only after the patient has received adequate resuscitation, which is determined by the following: (1) normal skin turgor, (2) normal fontanelle pressure, (3) moist mucous membranes, (4) a urine output of 2 mL/kg per hour, and (5) electrolyte values within normal limits, especially a total carbon dioxide of less than 30 mEq/L. Operative treatment is a pyloromyotomy, which divides the hypertrophied pyloric muscle, leaving the mucosa intact. Postoperative feedings can begin 8 hours after pyloromyotomy, with small volumes of sugar water given frequently. Over the next 12 hours, formula or pumped breast milk can be started. The volume of feedings should increase, with a concomitant decrease in the feeding frequency. Full oral nutrition should be under way 24–36 hours after operation. Parents should be advised that vomiting may occur postoperatively as a result of swelling at the pyloromyotomy, but this problem is self-limited. If the pyloric mucosa is perforated and repaired during surgery, nasogastric drainage is recommended for 24 hours before feeding is started.
 - b. **Intussusception** is an invagination of proximal intestine into adjacent distal bowel with resultant obstruction of the lumen. If prolonged, this obstruction may compromise the mesenteric venous return and, eventually, the arterial supply. It is a frequent cause of bowel obstruction in infants, toddlers, and young children, but the highest incidence is at 5–10 months of age. In 30% of cases, an antecedent viral gastroenteritis or upper respiratory infection may exist. Ileocolic intussusceptions are the most common. A lead point of the intussusception is identified in only 5% of patients and is most commonly a Meckel's diverticulum (see [section V.A.1.e](#)).
 1. **Diagnosis** of intussusception should be considered in a previously healthy infant who **presents** with a history of periods of abrupt crying and pulling the legs up to the abdomen. These attacks usually subside over a few minutes but recur every 10–15 minutes. Some children become lethargic between attacks. Normal stool is passed early on, but, eventually, dark red, mucoid stool, referred to as “currant jelly,” may be passed. Vomiting is common. On **physical examination**, hyperperistaltic rushes may be heard on auscultation of the abdomen during an attack. A sausage-shaped abdominal mass may be palpated or the tip of the intussusception may be felt on rectal examination. If the presentation is early, the vital signs usually are within normal limits. However, a delay in obtaining medical attention can lead to dehydration, sepsis, or shock. A **barium or air-contrast enema** confirms the diagnosis by demonstrating a “coiled spring” sign.
 2. **Management** of a patient with intussusception should involve surgical consultation before an attempt at nonoperative reduction. Before obtaining a barium or air-contrast enema, a nasogastric tube should be placed, intravenous fluid resuscitation started, and broad-spectrum antibiotics given. **Pneumatic or hydrostatic reduction** of the intussusception by air insufflation under radiographic guidance should be attempted if no evidence of peritonitis exists and the patient is stable. This technique has a 90% success rate. The maximum, safe intraluminal air pressure for young infants is 80 mm Hg, and that for older children is 110–120 mm Hg. After successful pneumatic or hydrostatic reduction, the child should be admitted for 24 hours

of observation. A liquid diet can be started once the child is awake and alert, and the diet is advanced as tolerated. Recurrent intussusception occurs in 8–12% of patients treated nonoperatively, and most of these patients should be managed the same as for an initial presentation. If a recurrence happens in an older child, however, surgery is advised because small-bowel tumors, which can serve as the lead point for the intussusception, occur more frequently in this age group. **Surgery** is required when nonoperative reduction fails or the child presents with peritonitis, sepsis, or shock. A transverse incision can be made to deliver the bowel. Gentle retrograde pressure is applied to the telescoped portion of the intestine in an attempt at manual reduction. Proximal and distal segments should not be pulled apart because of the risk of injury to the bowel. If manual reduction is not possible, resection of the involved segment and primary anastomosis should be done. An incidental appendectomy should also be performed. Recurrence of intussusception after operative treatment is approximately 1%.

- c. **Distal intestinal obstruction syndrome (DIOS)**, formerly known as *meconium ileus equivalent*, is an intestinal obstruction caused by impaction of inspissated intestinal contents in older patients with cystic fibrosis. This problem occurs in 10–40% of patients with cystic fibrosis who are followed long term.
1. **Diagnosis** of DIOS should be suspected in a child with cystic fibrosis who presents with chronic or recurrent abdominal pain and distention, vomiting, and constipation. An inciting cause, such as abrupt cessation of pancreatic enzyme supplementation, dehydration, dietary change, or an exacerbation of respiratory symptoms, may precipitate an acute presentation. Plain abdominal radiographs show the typical ground-glass appearance of the intestine. Dilated small-bowel and air-fluid levels may be present. Diagnosis may be confirmed by water-soluble contrast enemas, which demonstrate the inspissated intestinal contents.
 2. **Management** of a patient with DIOS most often is accomplished at the time of diagnostic enema, which induces an osmotic diarrhea to flush the intestine and relieve the obstruction. Only rarely, when intussusception or volvulus complicates DIOS, is surgery required.
- B. **Jaundice** is a physical sign that reflects the presence of hyperbilirubinemia. In infants, when jaundice is present, the total serum bilirubin usually exceeds 7 mg/dL. This elevated bilirubin may reflect a rise in either the conjugated (direct) or unconjugated (indirect) bilirubin, or both. This distinction is key in establishing a differential diagnosis for jaundice.
1. **Unconjugated hyperbilirubinemia** results when bilirubin that has not been metabolized in the liver rises above normal serum values. The most common causes of this condition are hemolytic disorders, breast-feeding, and physiologic jaundice of the newborn. Treatment with phototherapy or, in severe hyperbilirubinemia, exchange transfusions, should be considered in infants with these conditions. Less common causes of unconjugated hyperbilirubinemia include meconium ileus, Hirschsprung's disease, and pyloric stenosis, which indirectly cause an increase in the enterohepatic circulation of bilirubin. Surgical treatment of the underlying disorder corrects the jaundice in these patients.
 2. **Conjugated hyperbilirubinemia** is an elevation of the serum bilirubin, which has been linked to monoglucuronides and diglucuronides in the liver. This abnormality can be caused by biliary obstruction; hepatitis of infectious, toxic, or metabolic etiology; and conditions of chronic bilirubin overload. Because of these various causes, serologic titers for the TORCH (*toxoplasmosis, rubella, cytomegalovirus, and herpes simplex virus*) infections and screening tests for inherited etiologies of elevated direct bilirubin should be part of the initial evaluation. However, only the obstructive causes are amenable to surgical therapy and are discussed here.
 - a. **Biliary atresia** is the most common cause of infantile jaundice that requires surgical correction. The etiology is unknown, but the disease is characterized by a dynamic and progressive obliteration and sclerosis of the biliary tree. As the infant gets older, obliteration of extrahepatic bile ducts, proliferation of intrahepatic bile ducts, and fibrosis or cirrhosis of the liver worsen at an unpredictable rate.
 1. **Diagnosis** of biliary atresia often is difficult to make because clinical and laboratory information is nonspecific. Typically, these infants are well nourished and appear otherwise healthy but present with jaundice, acholic stools, dark urine, and hepatomegaly. **Percutaneous liver biopsy** usually is the first test obtained. Some biopsy specimens demonstrate unequivocally the pattern of biliary atresia. More often, however, the histology cannot be differentiated from that in alpha₁-antitrypsin deficiency or neonatal hepatitis. An alpha₁-antitrypsin level can rule out this disorder. Subsequently, the distinction between liver parenchymal disease and biliary obstructive disease can be made using **hepatobiliary imaging** with technetium-99m iminodiacetic acid. In biliary atresia, the liver readily takes up this tracer molecule, but no excretion into the duodenum is seen. **Ultrasonography** of the biliary tree can provide some structural information. Patients with biliary atresia characteristically have shrunken extrahepatic ducts and a noncontractile gallbladder by ultrasonography.
 2. **Management** of biliary atresia begins with repeat liver biopsy by open technique and an intraoperative cholangiogram. The common bile duct is visualized by cholangiography in only 25% of patients with biliary atresia. Cholangiography in the remaining 75% of patients demonstrates an atretic biliary tree. After diagnosis has been confirmed, operative correction is undertaken by performing a Kasai procedure (hepatopertoenterostomy). In this operation, the obliterated extrahepatic ducts are excised, and a hepaticojejunostomy is performed. When the distal common bile duct is patent, a choledochojejunostomy is constructed. Postoperatively, patients require supplementation with medium-chain triglycerides and the fat-soluble vitamins A, D, E, and K until normal bile flow is restored. In large series, the overall results for children treated with a Kasai procedure are that one-third show long-term improvement, one-third obtain temporary benefit, and one-third experience treatment failure. Survival for infants treated before 2.5 months of age with a Kasai procedure is 50% at 5 years (*Ann Surg* 210:289, 1989). Approximately one-fourth of these same patients survive until adolescence. When liver failure results or the Kasai procedure fails, liver transplantation is the only option available.
 - b. **Choledochal cyst** is a spectrum of diseases characterized by cystic dilatation of the extrahepatic and intrahepatic biliary tree. Types I–V plus a *forme fruste* variant are recognized. However, type I (fusiform cystic dilatation of the common bile duct) and type IV (cystic disease of the intrahepatic and extrahepatic bile ducts) are the most common forms. Although the pathogenesis of choledochal cysts is unclear, an embryologic malformation of the pancreaticobiliary system is believed to be the origin of this disease.
 1. **Diagnosis** of more than 50% of choledochal cysts is made during the first 10 years of life. Infants most commonly present with unremitting, asymptomatic jaundice, whereas older children and adults manifest mild intermittent but long-standing jaundice. Approximately one-third of older patients present with the symptom triad of abdominal pain, jaundice, and an abdominal mass. Cholangitis and pancreatitis also are possible presentations for this disease. Previously unrecognized choledochal cysts may even present as cholelithiasis, cirrhosis, portal hypertension, hepatic abscess, biliary carcinoma, or, infrequently, cyst rupture. Ultrasonography is the recommended initial test to identify a suspected choledochal cyst. Hepatobiliary scintigraphy can reveal intrahepatic cystic dilatation as well as biliary obstruction. Transhepatic cholangiography or endoscopic retrograde cholangiopancreatography are superior to ultrasound or scintigraphy for delineating intrahepatic and extrahepatic ductal disease.
 2. **Management** of a choledochal cyst is most commonly accomplished by cyst excision. This operation involves removing the entire cyst or shelling out the inner wall and its contents. It is important to identify the entrance of the pancreatic duct into the biliary tree before the excision is complete. After the cyst is removed, a choledochojejunostomy is constructed for biliary drainage. In rare cases, hepatic resection may be necessary when the disease is intrahepatic and is limited to a lobe or segment of the liver. If the intrahepatic component of the disease is diffuse or extensive, liver transplantation may be required.
- C. **Groin masses** in the pediatric population are most often hernias or hydroceles, but the examiner must exclude less common causes, such as acute testicular torsion, an undescended or retractile testicle, idiopathic scrotal edema, epididymitis, and inguinal lymphadenitis.
1. The **indirect inguinal hernia** is more common in children than are direct or femoral hernias. This inguinal defect occurs in 1–5% of children, and boys outnumber girls 8:1. Prematurity may increase the incidence of inguinal hernia to 7–30%. The true incidence of bilateral hernias is difficult to quantitate exactly, but it probably ranges from 10% to 40%. Bilateral hernias occur more frequently in premature babies and in girls.
 - a. **Diagnosis** of an inguinal hernia is made most often on physical examination by identification of a groin bulge extending toward the scrotum or vulva. Frequently, a child is referred with a history of a hernia, but no mass can be found on examination. If the patient can be induced to laugh, cry, or stand, the hernia may be reproduced. If these maneuvers prove ineffective, the presence of a thickened spermatic cord with a clear history from a reliable observer is sufficient to proceed with hernia repair.
 - b. **Management** of an inguinal hernia begins with assessment for incarceration. Most hernias reduce spontaneously with relaxation. Some hernias require manual reduction, however, which may be accomplished with gentle direct pressure on the hernia. Simultaneously applying caudal traction on the testicle in addition to direct pressure on the hernia may be necessary in some cases. If the hernia cannot be reduced, emergent operative repair is indicated. A child referred with an incarcerated hernia that is then reduced should be admitted to the hospital and scheduled for herniorrhaphy the next day. Premature infants in the nursery should have the hernia repair a few days before discharge from the hospital. Repair of a hernia is accomplished by high surgical ligation of the processus vaginalis through an inguinal incision. The hernia sac is found lying anterior and medial to the spermatic cord in boys. Sac contents can include small bowel, omentum, or ovary. In these cases, the viability of the sac contents must be ensured before repair.
 2. **Hydroceles** are fluid collections within the processus vaginalis that envelop the testicle. This fluid may communicate freely with the peritoneal cavity if the processus is patent and can thus be regarded as a hernia. Alternatively, after the portion of the processus that traverses the inguinal canal obliterates normally, any fluid accumulation remains confined to the scrotum as a noncommunicating hydrocele. Hydroceles occur in approximately 6% of full-term male newborns. A newly discovered hydrocele, a history of a change in size of this mass, or persistence of the hydrocele for longer than 12 months suggests the presence of a patent processus vaginalis. In these cases, elective repair of the hydrocele should be scheduled to prevent subsequent incarceration. Noncommunicating hydroceles are self-limiting and usually resolve in 6–12 months.
- D. **Abdominal pain** is a very common complaint in the pediatric age group. There are multiple causes of abdominal pain, some of which are unrelated to an intraabdominal process. In constructing a differential diagnosis for acute abdominal pain, one must consider the age and gender of the patient as well as the characteristics of the pain itself, such as onset, duration, or modifying factors. The following is a list of causes of abdominal pain in children, according to prevalence.

1. Differential diagnosis of acute abdominal pain

a. Very common

1. Acute appendicitis
2. Viral infection, nonspecific
3. Gastroenteritis
4. Constipation
5. Urinary tract infection

b. Less common

1. Intussusception
2. Lower-lobe pneumonia
3. Intestinal obstruction
4. Urinary tract obstruction
5. Inguinal hernia
6. Meckel's diverticulum
7. Cholecystitis
8. Intraabdominal tumors or masses

c. Rare

1. Henoch-Schönlein purpura
2. Primary peritonitis (nephrotic syndromes)
3. Pancreatitis
4. Hepatitis
5. Diabetic ketoacidosis
6. Lead poisoning
7. Acute porphyria
8. Herpes zoster
9. Sickle cell anemia
10. Hemophilia (retroperitoneal hematoma)

2. A **history** of the present illness can often be difficult to obtain from a child, and consequently the examiner must rely on the parents for accurate information. Several characteristics of the pain can be helpful in guiding the investigation of the symptom. The quality of the pain, such as whether it is sharp or dull, episodic or constant, is valuable information. The onset, location, duration, and presence of exacerbating or relieving factors for the pain also are important descriptors. Associated symptoms, such as vomiting (bilious vs. nonbilious) or diarrhea, help to elucidate the cause of the abdominal pain.
3. The **physical examination** should be performed with the child in a comfortable position and with full access to his or her abdomen and groin. Early in the examination, it is imperative to determine whether the child has peritonitis. Palpation of the abdomen may reveal guarding or rebound tenderness when the peritoneum is involved in the disease process. Guarding is best assessed by palpating lateral to the rectus abdominis muscles bilaterally. In a child who does not relax for examination, percussion of the abdomen also should produce guarding and rebound tenderness if peritonitis is present. Tenderness usually is most impressive in the region of underlying pathology. Abdominal pain either on manipulation of the hip or with deep respiratory movements suggests peritoneal irritation. In addition, on rectal examination, movement of the peritoneal reflection causes severe pain if peritonitis is present. A technique for performing a rectal examination in a child includes positioning the patient supine with the knees and hips maximally flexed and the legs spread apart. Younger children may pose a challenge to the examiner who attempts to perform a rectal examination. In these cases, diverting the child's attention or examining the child while asleep or cuddling with the mother may help facilitate the task. Whereas in adults auscultation of the abdomen is important, this technique is of limited value in infants and young children.

E. Tumors and neoplasms

1. **Neuroblastoma** is a neoplasm of the sympathochromaffin system. It has an incidence of approximately eight new cases per million children per year. This tumor is the most common extracranial tumor of childhood and accounts for 10% of all pediatric malignancies. The median age at diagnosis is 2 years, with 85% of the tumors being diagnosed before age 5.
 - a. **Diagnosis** of neuroblastoma usually is made during radiographic studies performed for other reasons or after the finding of an abdominal mass by the parents or pediatrician. Occasionally, children present with symptoms of fever, malaise, or abdominal pain. At the time they are discovered, up to 75% of neuroblastomas are metastatic. The most common sites of spread are to regional lymph nodes, liver, skin, and bone. Metastasis to the orbits may produce the raccoon's-eye appearance.
 - b. **Management** of neuroblastoma includes initial staging of the disease because age and tumor stage at the time of diagnosis are the most significant predictors of outcome. Several staging systems are available; however, the proposed International Neuroblastoma Staging System incorporates clinical, radiographic, and surgical information to define tumor stage ([Table 38-1](#)). Plain radiographs of the chest and skull along with a bone scan and bone marrow aspirate should be performed. Computed tomographic (CT) scanning or MR scanning studies are useful in delineating tumor surrounding the spinal canal. Operative evaluation may also be necessary for accurate staging. Children younger than 12 months at the time of diagnosis have a better prognosis for cure, whereas in older patients with disseminated disease, the prognosis remains poor (International criteria for diagnosis, staging, and response to treatment in patients with neuroblastoma. In: *Progress in clinical and biological research*. New York: Alan R. Liss, 1988). Surgical treatment for local disease is complete excision of the tumor with lymph node sampling. A liver biopsy should also be performed if the tumor is intraabdominal. When bulky or metastatic disease is present, a tumor biopsy is performed, followed by chemotherapy, radiotherapy, or both. N- *myc* amplification in the tumor is associated with a worse prognosis. A good response to chemotherapy is indicated by shrinkage of the tumor. Delayed resection then is undertaken.

Table 38-1. International Neuroblastoma Staging System

2. **Wilms' tumor** is the most common renal malignancy in children. The annual incidence is approximately 5.0–7.8 per million children younger than 15 years. It is bilateral in 5% of cases and accounts for 6% of all malignancies in children. The gender distribution is equal, and most Wilms' tumors are diagnosed between 1 and 3 years of age.
 - a. **Diagnosis** of Wilms' tumor is made from a combination of clinical, radiographic, and pathologic information.
 1. The **history** reveals abdominal pain in almost one-half of patients with Wilms' tumor; one-fourth of patients have fever, hematuria, or a urinary tract infection.
 2. On **physical examination**, 85% of these children have a palpable flank or abdominal mass. Anomalies associated with Wilms' tumor include hemihypertrophy (2%), aniridia (1%), and genitourinary anomalies (5%).
 3. **Abdominal ultrasonography** usually can distinguish an intrarenal mass, more consistent with a Wilms' tumor, from an extrarenal mass, more characteristic of a neuroblastoma. This test also is useful in ruling out tumor extension into the renal vein or vena cava.
 4. **Abdominal and chest CT scans** are necessary for staging, particularly to evaluate the contralateral kidney and to screen for pulmonary metastases.
 5. **Histologic examination** of tissue obtained at surgery confirms the diagnosis.
 - b. **Management** of patients with Wilms' tumor combines surgery and chemotherapy, which together result in a better than 90% chance for cure. A radical nephrectomy with sampling of the paraaortic lymph nodes is performed through a transabdominal approach. Isolation of the hilar vessels and examination of the contralateral kidney also are required. If Wilms' tumor is found initially to be unresectable because of size or bilaterality, a second-look operation can be done after chemotherapy. Vincristine, doxorubicin, and dactinomycin are the most effective chemotherapeutic agents. Radiotherapy is used for advanced stages of Wilms' tumor.
3. **Hepatic tumors** in children are malignant in 70% of cases, but they comprise fewer than 5% of all intraabdominal malignancies.
 - a. **Hepatoblastoma** accounts for 39% of liver tumors. Ninety percent occur before the age of 3 years, and 60% are diagnosed by 1 year of age.
 - b. **Hepatocellular carcinoma** usually presents in older children. Approximately one-third of these patients have cirrhosis secondary to an inherited metabolic abnormality.
 - c. **Diagnosis** of a hepatic tumor often begins with a child who presents with an enlarging abdominal mass that may be painful. The mass is best imaged by a CT scan or MR scan. Angiography can be useful in distinguishing benign from malignant tumors. Most patients with hepatoblastoma have elevated serum AFP levels; this is a useful parameter to monitor for recurrence after resection.
 - d. **Management** of hepatic malignancies is to resect the primary tumor. If the tumor is deemed unresectable at the initial operation, however, chemotherapy

is administered, followed by surgical reexploration at 4 months. Lymph node sampling and frozen-section analysis of the liver margins is necessary to confirm complete removal of the tumor. Survival is dismal for patients in whom the tumor cannot be completely resected.

4. **Teratomas** are composed of tissues from all germ layers—that is, endoderm, ectoderm, and mesoderm. In neonates, sacrococcygeal teratomas are the most common. These tumors are much more common in girls (4:1). A family history of twinning is observed in 10% of cases.
 - a. **Diagnosis** is usually made on prenatal ultrasound. If the tumor is large, delivery by cesarean section may be needed. The tumor presents as a mass extending from the sacrum. Ultrasound of the tumor may show extension of the tumor into the pelvis and abdomen.
 - b. **Management** includes elective resection through a chevron-shaped buttocks incision during the first week of life. Occasionally, arteriovenous shunting in the tumor produces a shocklike syndrome with metabolic acidosis. This situation requires emergency resection. Principles important in resection of the tumor include preservation of the rectal sphincter muscles, resection of the coccyx with the tumor, and early control of the midsacral vessels that supply the tumor. The latter helps to control hemorrhage, the most common complication of this operation. Failure to resect the coccyx results in a high recurrence rate.
 - c. **Malignancy** is rarely seen in neonates but increases with the age of the child. If the tumor is malignant, a thorough search for metastases is in order, and combination chemotherapy is given, which may shrink the tumor and allow for resection.
5. **Soft-tissue sarcomas** account for 6% of childhood malignancies, more than one-half of which are rhabdomyosarcomas.
 - a. **Diagnosis** of a soft-tissue sarcoma is best accomplished with a multidisciplinary approach. The mass should be imaged by CT scan or MR scan. An incisional biopsy usually is required to determine the histologic type preoperatively. Consultation with a radiotherapist and oncologist before initiating therapy is advised.
 - b. **Management** of nonrhabdomyosarcomas includes wide surgical excision. If the tumor is a rhabdomyosarcoma, however, treatment is determined by the location of the tumor. Complete resection of head and neck tumors is rarely possible, and they usually are treated with biopsy followed by chemotherapy. Trunk and retroperitoneal tumors are treated with wide excision whenever possible. Rhabdomyosarcomas of the extremity also are treated with wide excision, but resection of muscle groups and the use of radiotherapy or brachytherapy should also be considered (*Surg Clin North Am* 72:1417, 1992). A biopsy of the regional lymph nodes should be included in the procedure.

39 Neurosurgical Emergencies

James J. Lu and Ralph G. Dacey, Jr.

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Introduction

Neurosurgical emergencies involve a broad spectrum of illness, including traumatic injury to the head and spine. Several nontraumatic settings also require emergent intervention, among these intracranial hemorrhage, elevated intracranial pressure (ICP), cord compression, and infections. In many cases, rapid assessment with immediate neurosurgical consultation and intervention can prevent significant neurologic injury and can even be lifesaving.

Neurosurgical Trauma

I. Intracranial trauma

- A. **Evaluation.** Initial management of head injury focuses on hemodynamic stabilization with establishment of an adequate airway, ventilation, and support of circulation, followed by rapid diagnosis and treatment of intracranial injuries.
1. **Airway and ventilation.** Severe head injury frequently leads to failure of oxygenation, ventilation, or airway protection; therefore, intubation in these cases is essential. A rapid neurologic assessment is performed before sedation and paralysis are induced. A low threshold for intubation must be present for agitated patients requiring sedation. Even with adequate oxygenation (e.g., by pulse oximetry), inadequate ventilation can occur. Intubation, when clinically indicated, should not be delayed for arterial blood gas analysis.
 2. When possible, cervical spine films should be assessed before intubation. In situations of acute compromise, intubation should proceed, even before complete clearance of the cervical spine. Two-person in-line intubation is performed, with the second individual securing the patient's neck with axial traction to avoid extension of the neck during intubation. Nasal intubation can be performed if craniofacial injuries do not otherwise contraindicate this procedure. Associated cervical spine injuries should be assumed in the patient with a head injury until they are ruled out.
 3. **Circulatory support** requires aggressive fluid resuscitation for treatment of arterial hypotension, followed by blood products, if necessary, and identification of the etiology of the hypotension. Head injury with intracranial hemorrhage is almost never the sole cause of systemic hypotension (in the absence of profuse scalp bleeding). When clinical signs of adequate oxygen perfusion are present, patients with head injuries should not be overresuscitated with fluids. Impairment in mental status cannot be adequately assessed until mean arterial pressure (MAP), and thus cerebral perfusion pressure (cerebral perfusion pressure = MAP – ICP), is corrected.
 4. **Neurologic evaluation**
 - a. **A rapid, but systematic**, neurologic examination is performed on the scene and is repeated frequently during transport and on initial presentation to the emergency room. Frequent neurologic checks and recording of the **Glasgow Coma Scale** (GCS) continue throughout evaluation ([Table 39-1](#)). Examination focuses on the three components of the GCS: eye opening, verbal response, and motor response (*Lancet* 2:81, 1974). This score indicates injury severity and measures changes in the impairment of consciousness. Pupillary response, extraocular movements, facial movements, and the remainder of the cranial nerve examination should also be performed. The **pupils** of a head-injured patient should never be artificially dilated in the acute setting because changes in pupillary reaction can be early signs of herniation. Strength and symmetry of the extremities should be noted to search for evidence of cerebral or spinal cord damage. The sensory examination should be performed as thoroughly as the level of consciousness permits. Landmarks for sensory dermatomes include the nipple (T4 level) and the umbilicus (T10 level). The C4 and T2 dermatomes meet at the clavicle, with intervening levels distributed along the upper extremity. Reflexes and sphincter tone should also be assessed. Finally, the head-injured patient has a high incidence of associated injuries. Cervical spine evaluation is obligatory (see [section II.A](#)). Thorough examination is essential, even in patients thought to have an isolated closed head injury. Unnecessary sedation and prolonged pharmacologic paralysis in head-injured patients can make neurologic assessment difficult and should be avoided.
 - b. **Systemic causes** of mental status impairment must be ruled out. Metabolic (electrolyte or acid-base abnormalities, hypo- and hyperglycemia), toxic (drugs, uremia), hypothermic, or respiratory (hypoxia or hypercapnia) derangements can underlie mental status changes, even if closed head injury is present. Recent seizures or cardiac arrest can also impair neurologic function severely. Corrective measures, such as dextrose for hypoglycemia, thiamine (100 mg i.m.) in the setting of alcohol abuse, naloxone for suspected opioid overdose, and oxygenation or ventilatory support should be initiated as immediately indicated.
 5. **Radiographic evaluation** begins with cervical spine plain X-rays, including anteroposterior, lateral, and open-mouth (odontoid) views. The initial emergency room evaluation should proceed rapidly to head computed tomographic (CT) scanning. Mass lesions are not diagnosed or excluded reliably by clinical examination. Delay caused by evaluation of non-life-threatening injuries should be avoided until the patient's head is imaged. Centers without this capability should stabilize the vital signs, secure the airway, and arrange for rapid transfer of the patient to a facility with head CT scanning and neurosurgical facilities.
 6. **Initial therapies**
 - a. **If elevated ICP** is suspected, such as with signs of herniation or acute neurologic deterioration, therapy should be initiated until the ICP can be measured. **Mannitol** (0.25–1.0 g/kg i.v. bolus) is effective in controlling raised ICP acutely. A Foley catheter should be placed to follow the osmotic diuresis closely after mannitol administration. Fluid replacement may be necessary to avoid hypotension. Systemic causes for neurologic deterioration must also be ruled out. Ventilatory support to maintain a mildly hypocapnic partial pressure of carbon dioxide (PCO_2 ; ~35 mm Hg) should be instituted. For refractory elevated ICP, **hyperventilation** (PCO_2 ; ~30 mm Hg) may be used only in the acute setting for **brief** periods. Prophylactic hyperventilation should be avoided in severe brain injury. Prolonged use of hyperventilation should not be used to control elevated ICP and may worsen ischemia by compromising cerebral blood flow. No proven role for steroids in the management of acute head injury exists.
 - b. **Seizures** should be controlled rapidly in patients with head injury. Intravenous lorazepam (Ativan) can be administered in 1-mg boluses and repeated until seizures are controlled. Airway protection must be available if significant doses of benzodiazepines are to be given. Phenytoin (Dilantin) should also be administered for seizures and may be indicated for seizure prophylaxis in patients at high risk for early posttraumatic seizures (GCS \leq 10, intracranial hematoma, depressed skull fracture, cortical contusion visible on CT, penetrating or open injuries). Duration of prophylactic antiepileptic therapy should be no more than 7 days (*N Engl J Med* 323:497, 1990). The use of prophylactic phenytoin in other patients with head injury is controversial. A loading dose of 15–18 mg/kg phenytoin sodium is given by slow (<50 mg per minute) intravenous infusion to avoid cardiovascular complications, such as hypotension. Alternatively, fosphenytoin (Cerebyx) may be administered more rapidly than phenytoin and may be useful in patients with poor intravenous access or status epilepticus. A loading dose of fosphenytoin, 15–18 mg phenytoin sodium equivalents, is given either intravenously or intramuscularly. The rate of intravenous infusion of fosphenytoin should be no greater than 150 mg phenytoin sodium equivalents per minute to avoid hypotension. With either phenytoin or fosphenytoin, close monitoring of the ECG, blood pressure, and respiratory function should be performed during administration. Maintenance doses of phenytoin should then be started (4–6 mg/kg) and drug levels followed to guide dosing.
- B. **Types of head injury**
1. **Focal (mass) lesions** are best diagnosed by emergent CT scan of the head without contrast. Hemiparesis, unilateral pupillary dysfunction (the fixed and dilated pupil), or both, can herald brainstem herniation from mass lesions, but these are imperfect localizing signs (*Neurosurgery* 34:840, 1994); therefore, CT scan is imperative. Indications for surgical evacuation include neurologic symptoms referable to the mass lesion, midline shift greater than 5 mm, and

elevated ICP that is refractory to medical management. Posterior fossa mass lesions can be particularly dangerous because brainstem herniation may have very few specific warning signs before death occurs (see [section III.A.2](#)).

- a. **Epidural and subdural hematomas (SDHs)** can cause rapid deterioration and usually require surgical evacuation. Classically, epidural hematomas present with a "lucid interval" after injury, which precedes rapid deterioration. This sign is inconsistent and nonspecific and may also be seen with SDHs. Epidural hematomas appear as biconvex hyperdensities on head CT scan that typically respect the suture lines and are operated on emergently if they are causing significant mass effect, symptoms, or both. A frequent association with simultaneous SDH exists.
- b. **Acute SDHs** typically appear as hyperdense crescents on head CT scan as the blood spreads around the surface of the brain. However, hyperacute SDHs can appear hypodense on head CT. Acute SDHs that are less than 1 cm thick with minimal midline shift and without localizing neurologic dysfunction can be observed; however, emergent evacuation is otherwise indicated. Often, SDHs are associated with severe intracranial injury. If surgical evacuation is indicated and delayed for more than 4 hours, these lesions have a high mortality (*J Neurosurg* 74:212, 1991; *N Engl J Med* 304:1511, 1981).

SDHs also can present, especially in the elderly, days to weeks after head injury. These **chronic SDHs** can cause focal neurologic deficit, mental status changes, and seizures. A symptomatic chronic SDH large enough to be drained should be treated with burr-hole drainage or twist-drill craniostomy with subdural drain placement (*J Neurosurg* 65:183, 1986). Prophylactic anticonvulsants are considered, and steroids may be beneficial. Diagnosis is best made with CT scan because MR scan can give an inconsistent picture of lesion size.

- c. **Contusions**, or bruising, of the brain tissue may be apparent on initial head CT scanning. Areas of brain that are at risk for striking bone during acceleration-deceleration injuries (e.g., temporal pole, frontal pole, and occipital lobe) are most susceptible to contusion. Delayed hemorrhage into contused brain parenchyma can occur hours to days after the initial injury. Delayed deterioration in the neurologic examination after head injury requires an emergent head CT scan and possible surgical evacuation of any significant hemorrhage.
2. Nonfocal sequelae of head injury include **cerebral edema** and **diffuse axonal injury (DAI)**. Hallmarks of cerebral edema on head CT scan include obliteration of the basal cisterns and coronal sulci and loss of differentiation of the gray and white matter. In DAI, severe head injury is associated with minimal changes on head CT scan (*J Neurosurg* 56:26, 1982). DAI represents the pathologic result of shearing forces on the brain. Often, small hemorrhages are seen in corpus callosum, midbrain, deep white matter, or other deep cerebral structures. Microscopic injury to axons may result in severe neurologic dysfunction. Radiographic evidence of diffuse edema or a severely impaired neurologic condition may be an indication for monitoring and treatment of elevated ICP.
 3. **Open injuries and fractures**
 - a. **Open skull fractures** require operative irrigation, debridement of nonviable tissues, and dural closure. Prophylactic antibiotics may reduce the risk of infection. Surgical treatment of **depressed skull fractures** usually is required for depressions greater than the thickness of the skull table. The bony defect is elevated, and underlying dural tears are repaired. Seizure prophylaxis usually is indicated in this setting. Open, depressed skull fractures require elevation and debridement of depressed skull fragments as well as devitalized tissue, followed by a course of antibiotics.
 - b. Fractures through the paranasal air **sinuses**, especially with associated pneumocephalus and dural tears, may require repair. Often, repair is performed in combination with repair of associated facial fractures. The prophylactic use of broad-spectrum antibiotics to prevent meningitis in these cases is controversial.
 - c. **Basilar skull fractures** can be complicated by **cerebrospinal fluid (CSF)** leaks, which can be managed nonoperatively without prophylactic antibiotics. If drainage continues or recurs, a lumbar drain or surgical repair may be required because persistent leakage can lead to meningitis. Temporal bone fractures can be associated with damage to the seventh and eighth cranial nerves, the middle ear apparatus, or both.
 - d. **Missile injuries** require debridement, closure, and prophylactic antibiotics similar to those used for other open head injuries. However, injuries from gunshot wounds present several associated problems. Shock waves can result in widespread destruction of brain tissue and vasculature. Operative management must address removal of accessible foreign bodies and bone fragments, evacuation of intracranial hematomas, debridement of entrance and exit wounds, and closure of dura and scalp. Overaggressive debridement near large vessels should be avoided to prevent further damage to vascular structures.

C. Management of elevated ICP

1. Monitoring

- a. **Indications.** ICP monitoring generally is recommended in head injury if serial neurologic examinations cannot be used as a reliable indicator of progressive intracranial pathology. This usually corresponds to a GCS score of less than 9. Head CT scans demonstrating mass lesions, diffuse cerebral edema, or other pathology associated with elevated ICP are also indications for monitoring or for empiric treatment until monitoring is available.
- b. **ICP pressure monitors** are of several types. The **parenchymal bolt** consists of a fiberoptic or strain gauge catheter tip that measures ICP at the brain surface. Placement usually is over the right frontal lobe in the midpupillary line 2 cm anterior to the coronal suture. This avoids placement over essential cortex. **Intraventricular catheters** (ventriculostomy) are placed in the lateral ventricle with the tip at the foramen of Monro. The entry point is identical to the site of subdural bolt placement, with the catheter passed 6 cm toward the nasion medially and the external auditory meatus posteriorly. ICPs are transduced through the CSF column. Additionally, intraventricular catheters allow for the drainage of CSF in the treatment of elevated ICP (see [section I.C.3](#)). Other monitors include subdural catheters, usually placed intraoperatively. Also used are extradural sensors, which are considered less accurate but may have a lower complication rate.

2. Treatment

- a. In addition to initial treatment of elevated ICP (e.g., mannitol, mild hypocapnia), **simple measures** are taken, such as head elevation to 30 degrees, a neutral head position to enhance venous drainage, avoidance of circumferential taping around the patient's neck when securing the endotracheal tube, appropriate fitting of Philadelphia C collars if indicated, and adequate sedation before any stimulation, such as intubation. Elevated intrathoracic pressures (as with coughing, straining, or high positive end-expiratory pressure) can elevate ICP. Fever can also exacerbate ICP, and aggressive treatment with antipyretics and cooling blankets should be instituted to prevent hyperthermia.
- b. Although **hyperventilation for brief periods** may be necessary in the setting of acute deterioration, prolonged hyperventilation with PCO₂ less than 35 mm Hg can reduce cerebral blood flow and should be avoided. Prophylactic hyperventilation is not recommended after severe head injury.
- c. Further treatment is aimed at keeping ICP less than 20 mm Hg (*J Neurosurg* 75:S59, 1991), usually with **osmotic and loop diuretics** (mannitol, 25- to 50-g boluses i.v. every 6 hours, or furosemide). Fluid balance, serum electrolytes, and serum osmolarity should be carefully monitored; euvolemia must be maintained. Diuretics generally are held if serum osmolarity exceeds 320 mOsm/L. In addition, hypotension should be avoided in these patients, and there are some data that suggest better outcome with maintenance of cerebral perfusion pressure (MAP – ICP) of 70 mm Hg or greater (see [section I.D.1](#)).
- d. **Sedation** can also be used to control ICP. Benzodiazepine or narcotic (e.g., fentanyl) infusions can be given and titrated to effect (Ramsay 3). Intubation and mechanical ventilation usually are needed. Refractory elevation in ICP may require neuromuscular paralysis or even barbiturate coma with invasive hemodynamic monitoring.

3. **Surgical interventions** are directed primarily at removal of **mass lesions**, if present. In the absence of a mass lesion, uncontrollable ICP and a deteriorating neurologic examination may require **craniectomy** with removal of a large bone flap to relieve pressure on the intracranial contents (*Neurosurgery* 29:62, 1991). Removal of CSF by **ventriculostomy** can reduce ICP; however, the small intracranial volume occupied by the CSF limits this effect.

D. Frequently associated complications

1. **Cardiac considerations.** Adequate blood pressure should be maintained in the setting of elevated ICP, with care taken to avoid hypotension (systolic blood pressure <90 mm Hg), which has been associated with worse outcome in severely head-injured patients (*Br J Neurosurg* 7:267, 1993). Maintenance of cerebral perfusion pressure greater than 70 mm Hg (or MAP >90 mm Hg) can be used as a treatment guideline. Administration of isotonic crystalloid, colloid, and/or easily titratable vasopressors, such as phenylephrine or dobutamine, can be used to maintain an adequate perfusion pressure.
2. **Respiratory considerations.** Early airway control is essential in the head-injured patient. Severe head injury is also associated with ventilation-perfusion mismatch and development of pulmonary edema or adult respiratory distress syndrome. Delayed complications of pneumonia and pulmonary embolism are common, as are associated injuries, such as pneumothorax or pulmonary contusions.
3. **Gastrointestinal (GI) considerations.** Patients with severe head injury and increased ICP are at risk of developing Cushing's (stress) ulcers and GI bleeding. These patients should undergo prophylaxis with H₂ antagonists or sucralfate.
4. **Fluid and electrolytes.** Head-injured patients are at risk for development of either diabetes insipidus or the syndrome of inappropriate antidiuretic hormone. Initially, the use of isotonic saline (with glucose and, if necessary, potassium) avoids exacerbating cerebral edema. Close monitoring of electrolytes is essential because alterations in sodium and water balance are common. **Diabetes insipidus** can develop rapidly and must be treated aggressively. Fluid hydration should match output. Often, the process is self-limiting, but persistent output of large amounts (>300 mL per hour) of urine with a low specific gravity may necessitate vasopressin treatment [desmopressin (DDAVP), 1 µg i.v. every 12 hours]. If the syndrome of inappropriate antidiuretic hormone with hyponatremia develops, treatment with restriction of free water intake usually is sufficient, although infusion of hypertonic (1.5% NaCl) saline may be necessary. As was mentioned previously, treatment with osmotic diuretics may require large-volume fluid replacement. In these cases, electrolytes (including potassium, magnesium, and phosphorus) should be monitored closely.
5. **Hematologic considerations.** Disseminated intravascular coagulopathy can occur with severe head injury, such as missile injuries, often developing several

hours after the disruption of brain tissue. Coagulopathies should be aggressively treated with fresh frozen plasma and vitamin K (10 mg per day i.m.), especially if intracranial hemorrhage is present.

6. **Nutrition.** Nutritional demands are increased in head injury (*Neurosurg Clin North Am* 2:301, 1991). High-osmolarity tube feedings can reduce the risk of cerebral edema and provide adequate caloric intake. If tube feedings are not tolerated, parenteral nutrition may be necessary. Replacement of 140% of the expected resting metabolism expenditure in nonparalyzed patients and 100% of the expenditure in paralyzed patients by the seventh day after injury can be used as a guideline.
 7. **Associated injuries.** A thorough assessment for other systemic and orthopedic injuries is essential. The unconscious patient should always be assumed to have a cervical spine injury until this is ruled out with appropriate radiographic tests.
- E. **Minor head injury.** Management of the patient with a minor head injury focuses on detecting and rapidly treating those at risk for subsequent deterioration (*J Neurosurg* 65:203, 1986). Minor injury usually refers to a presenting GCS score of 13–15, often with associated loss of consciousness and minimal CT findings. Selection of those at higher risk for delayed mass lesion, associated fracture, elevated ICP, and CSF leaks is needed. In this group of patients, those with an associated skull fracture on radiographic studies are at particular risk of developing neurosurgical complications. A normal CT scan without altered level of consciousness, neurologic deficit, or open injuries may allow for the patient's discharge to home with reliable supervision. Any exceptions may indicate a more severe injury with a higher risk of associated or delayed lesions and may require that the patient be admitted for observation. A postconcussive syndrome, even weeks to months after injury, is described with vague symptoms, including headache, fatigue, inattention, dizziness, vertigo, and memory difficulties. In the acute setting, these symptoms may be difficult to discern from delayed elevation of ICP owing to cerebral edema or hemorrhage.

II. Spinal trauma

- A. **Evaluation** for spinal injury is indicated if focal pain, neurologic examination, or mechanism of injury warrants. Cervical spine injuries are common in motor vehicle accidents, falls, or other injuries in which axial loading, flexion-extension forces, or both, are exerted. Thoracic and lumbar spine evaluation should also be performed if local pain, tenderness, or deformity is found on physical examination. Neurologic deficit involving the lower extremities after trauma may require evaluation of the entire spine to find an injury.
1. **Initial support**
 - a. **Airway and breathing.** Intubation should be performed early in patients demonstrating respiratory fatigue or otherwise requiring airway protection or ventilatory support. In the presence of cervical spine injury, fiberoptic intubation requires less manipulation and reduces the risk of further neurologic injury. When this is not available, two-person intubation with in-line stabilization of the neck can be performed safely.
 - b. **Circulation.** Nonneurologic sources of hypotension must be pursued thoroughly. In patients with obvious external hemorrhage or suspected hypovolemic shock, standard fluid resuscitation should be initiated. In the quadriplegic patient, if no blood loss is suspected and fluid challenges do not improve perfusion, diagnosis of **spinal shock** should be considered. Hypotension associated with the loss of sympathetic tone seen in some high thoracic and cervical spine injuries does not respond to fluids alone. Pressors (e.g., dopamine) reduce peripheral vasodilatation and improve cardiac output. Central hemodynamic monitoring, including placement of a pulmonary artery catheter, may be helpful. Excessive fluid administration can worsen respiratory difficulty and spinal cord edema.
 - c. A careful **neurologic examination** includes assessment of motor and sensory function and deep tendon reflexes. Multiple sensory modalities (light touch, pinprick, temperature sensation, and joint position sense) should be assessed, especially in the patient with an incomplete lesion. Deep tendon reflexes, sphincter tone, and cremasteric and bulbocavernosus reflexes should be documented. Incomplete lesions are common and, as with sacral sparing of sensory function, carry prognostic significance (*Neurosurgery* 20:742, 1987).
 2. **Radiographic evaluation**
 - a. Standard radiographic evaluation of the cervical spine includes **anteroposterior, lateral, and open-mouth (odontoid)** views. A **swimmer's** view may be necessary to visualize C7 and the C7-T1 interspace, which is essential in complete clearance of the cervical spine. CT scanning is necessary if adequate plain films cannot be obtained, although plain films are the best survey for fractures or subluxations. Anteroposterior and lateral views of the thoracic and lumbar spine are obtained as indicated. If a spinal fracture is found, other spine injuries should be sought, owing to the high rate of coincident injuries (*J Spinal Disord* 5:320, 1992).
 - b. Other imaging modalities include **CT scans** to further evaluate known or suspected fractures. Intraspinous hemorrhage can often be detected. **MR scan** may reveal protruded intervertebral discs (*J Neurosurg* 79:341, 1993), which can cause deficit with or without bony abnormalities seen on plain films. Damage to the vertebral artery can occur with cervical injuries, especially if fracture of the foramen transversarium occurs. Angiography usually is pursued if neurologic damage is referable to vertebral artery injury.
 3. **Instability.** Spinal instability must be suspected until ruled out. Ligamentous injury can occur in the absence of fracture, and instability can occur with normal plain films. Malalignment (subluxation >3.5 mm, abnormal angulation >11 degrees), extensive fracture involving multiple spinal column segments, or neurologic deficit usually indicates instability. Severe pain or apprehension of neck movement also warrants careful assessment. In the minimally symptomatic, alert patient, cervical flexion-extension films can aid in the assessment of spinal stability. Flexion and extension must be done under the patient's own power, and motion should be stopped if pain or other symptoms arise.
- B. **Treatment**
1. **Steroids** are known to improve outcome from spinal cord injury if given within 8 hours (*N Engl J Med* 322:1405, 1990). Patients seen between 3–8 hours after injury should be treated emergently with **methylprednisolone, 30 mg/kg i.v. bolus** over 15 minutes, followed 45 minutes later by a **5.4-mg/kg per hour i.v. infusion** continued over the next 48 hours (*JAMA* 277:1597, 1997). If steroids are administered within 3 hours of injury, intravenous infusion is given for only 24 hours. If patients are seen more than 8 hours after injury, this protocol is not of proven benefit and is contraindicated.
 2. **Immobilization and reduction.** Suspected or known spine injuries require immobilization, initially with a rigid cervical collar and long backboard. Cervical spine subluxations and dislocations must be reduced under neurosurgical supervision. Gardner-Wells skull tongs are placed 2 cm above the pinna, and axial traction is applied. The initial weight should be 2 lb in upper cervical levels and in the elderly to avoid overdistraction. Higher weights are used in lower cervical injuries. Weights can be advanced by 5-lb increments until reduction is seen on X-ray or fluoroscopy. Muscle relaxants (diazepam, 5 mg i.v. or p.o.) may aid in reduction. Traction (5–10 lb) also is useful for ligamentous instability, even if reduced radiographically, to maintain adequate alignment. If reduction cannot be achieved externally, operative reduction is required. Fractures may be treated by external immobilization (e.g., halo external fixation) or by operative fusion, depending on the nature of the fracture and degree of instability.
- Thoracic and lumbar spine fractures are managed with operative stabilization or immobilization with an orthosis. Injuries with associated neurologic deficit, with excessive displacement or angulation of the spinal column, or with loss of vertebral body height are more likely to require operative stabilization. Before stabilization, patients are managed with bedrest with frequent logrolling. Although early surgical decompression and stabilization promote mobilization of the patient, the timing of surgery and the role of emergency surgery in the patient with acute neurologic deficit remain somewhat controversial (*Neurosurgery* 35:240, 1994).
3. **Cervical spine injuries are associated** with the development of adult respiratory distress syndrome, hyponatremia, hypotension, bradyarrhythmias, ileus, and urinary retention. Patients with cervical spine injuries may require cardiorespiratory monitoring, isotonic fluid support, nasogastric decompression, urinary catheter placement, laxatives, and stool softeners to address these issues. Bladder and bowel dysfunctions also are seen with lower spine injuries, although autonomic problems are rare in injuries below the upper thoracic spine.
- C. **Penetrating injuries** to the neck and torso may result in fractures of the spine or penetration of the spinal canal, in association with other injuries.
1. **Initial evaluation** includes airway control with cervical and thoracolumbar spine immobilization, circulatory support, and radiographic evaluation for potential spine injury. Penetrating injuries to the neck may be associated with injury to the esophagus, airway, or great vessels in the neck. **Diagnostic evaluation** in these patients may include angiography, esophagography, and fiberoptic endoscopy in addition to plain films of the spine. Operative intervention for related injuries can be complicated by the need to stabilize the cervical spine.
 2. **Treatment.** Aggressive **débridement** usually is not indicated unless the spinal canal is compromised. **Dural tears** with leakage of CSF may require direct repair, although such leaks usually resolve with observation or temporary CSF drainage through a lumbar drain (*Neurosurgery* 30:241, 1992). High-dose steroid administration has not been shown to be of proven benefit in these patients and is not indicated (*J Trauma* 37:576, 1994).

III. Other emergencies

- A. **Nontraumatic intracranial hypertension and herniation syndromes**
1. **Etiology.** Elevation of the ICP may lead to compression of neurologic structures and irreversible neurologic damage. Nontraumatic causes include hemorrhagic and nonhemorrhagic mass lesions.
 - a. Spontaneous **intraparenchymal hemorrhages** may occur owing to hypertension, vascular malformations (arteriovenous malformations or aneurysms), tumor, angiopathy, vasculitis, or secondary hemorrhage into a large infarction.
 - b. **Nonhemorrhagic lesions** include tumor, infection, and mass effect from edema after cerebral infarction.
 2. Clinical **herniation syndromes** exist with presentations referable to the location of the lesion.

- a. **Supratentorial** sites of herniation include uncal, central transtentorial, and subfalcine herniation. Progressive lethargy, unilateral pupillary dysfunction, and/or hemiparesis suggest uncal herniation. Compression of vascular and brain structures requires immediate intervention. **Uncal** herniation often is the result of temporal lobe lesions. **Central transtentorial** herniation results from downward compression of brainstem structures. Unresponsiveness, deep coma, and cranial nerve dysfunction are observed. Central transtentorial herniation suggests a bilateral process or an interhemispheric lesion. **Subfalcine** herniation results from lesions causing a shift across the inferior aspect of the falx and can present with only lethargy or lower-extremity weakness. Both falcine and uncal herniation can progress to a central transtentorial picture as further structures are compressed.
 - b. **Infratentorial** herniation results from posterior fossa masses compressing the brainstem or from herniation of the cerebellar tonsils through the foramen magnum. Signs of infratentorial herniation include lower cranial nerve dysfunction and the rapid onset of respiratory or cardiac arrest with little warning.
3. **Treatment** of herniation requires control of the ICP. The obtunded patient must have his or her airway controlled. Mannitol can be used, often with ICP monitoring (see [section I.C.1](#)) to guide medical treatment. Mass lesions may need to be evacuated. The underlying cause of a hemorrhagic lesion must be determined if time permits. Unplanned entrance into an undetected arteriovenous malformation can be catastrophic. Angiography may help clarify the etiology. Evacuation of deep basal ganglia hemorrhages (commonly hypertensive) is avoided unless it is necessary for ICP control because the risk of worsening the deficit—especially language deficits for dominant hemisphere lesions—is high.
- B. Nonhemorrhagic lesions**
1. Diffuse cerebral edema also can produce ICP elevations that require treatment. **Metabolic derangements**, such as those seen in hepatic encephalopathy, can elevate the ICP.
 2. Edema can develop also secondary to large **infarctions** in the cerebral hemispheres, leading to delayed deterioration. Management of elevated ICP may be indicated in large cerebral infarctions, whether hemorrhagic or nonhemorrhagic, possibly even requiring hemicraniectomy for large nondominant hemispheric infarctions (*Stroke* 21:874, 1990). Posterior fossa infarctions are a special category because even nonhemorrhagic infarctions in the cerebellum can result in enough swelling to compress the brainstem and the fourth ventricle, the latter leading to hydrocephalus. Emergent decompression and resection of edematous cerebellum can be lifesaving (*Arch Neuroi* 22:490, 1970).
 3. **Brain tumors** rarely are surgical emergencies. Presenting symptoms include progressive headache, seizures, and localizing neurologic deficit. Uncommonly, acute neurologic deterioration is seen, usually suggestive of hemorrhage into the tumor. Evaluation and treatment are similar to those for any other acute intracranial mass lesion (see [section I](#)), with airway control, hyperventilation, and osmotic diuresis. **Contrast-enhanced CT or MR scanning** should be performed if a tumor is suspected. High-dose **steroids** may have a potent effect on the brain edema associated with tumors. Urgent **surgical evacuation** of the mass lesion occasionally is required.
- C. Hydrocephalus** may result from a variety of causes and can lead to rapid neurologic deterioration.
1. **Cerebellar tumors** or other mass lesions may compress the cerebral aqueduct and cause fourth ventricle obstruction without preceding symptoms. Tuberculosis and bacterial meningitis also can cause hydrocephalus. Patients classically present with lethargy, headache, papilledema, sixth nerve palsy, or abnormalities of upward gaze (“setting-sun” sign). **Treatment** involves urgent placement of a catheter for external **ventricular drainage** (see [section I.C.1.b](#)) to relieve the buildup of CSF. Posterior fossa mass lesions may further compress the brainstem if the elevated ICP is relieved too rapidly, leading to upward herniation (see [section III.A.2](#)).
 2. **Shunt malfunction.** Internal ventricular drainage systems (shunts) require special attention. Malfunction can present with warning symptoms, such as headache or nausea, or with rapid deterioration of mental status. Imaging, such as an emergency CT scan, can help in the diagnosis, but emergent operative revision may be indicated if mental status deterioration is present and shunt malfunction is suspected. A shunt series (plain films of the head, neck, chest, and abdomen) can visualize the entire course of the shunt and its connections, possibly leading to identification of a malfunction.
- D. Intracranial hemorrhage.** Spontaneous intracranial hemorrhage requires emergent intervention in addition to addressing the sequelae of the associated mass effect (see [section III.A](#)).
1. **Subarachnoid hemorrhage (SAH)** secondary to aneurysmal hemorrhage is a common neurosurgical emergency.
 - a. The clinical **presentation** usually includes a history of severe sudden headache, nuchal rigidity, photophobia, lethargy, agitation, or a comatose state. Acute blood from an SAH appears bright on CT scan. Blood usually is seen in the cisterns or sylvian fissure. Lumbar puncture can make the diagnosis if the history is suggestive, even with a normal CT scan, but is not required when the scan is diagnostic. Patients with SAH require intensive neurologic monitoring, airway protection if mental status deteriorates, anticonvulsant therapy, and cerebral angiography to determine the origin of the SAH. The principal goal of treatment is the prevention of rebleeding by surgical clipping or endovascular techniques. Without treatment, rebleeding occurs in 50% of patients with a ruptured aneurysm in the first 6 months (*J Neurosurg* 62:321, 1985).
 - b. **Complications**
 1. **Vasospasm** of the cerebral arteries can follow aneurysmal SAH, leading to further neurologic deterioration. The calcium channel blocker nimodipine (60 mg p.o. every 4 hours) can reduce the risk of vasospasm and is given for a period of 21 days if aneurysmal SAH is suspected (*N Engl J Med* 308:619, 1983). After surgical clipping or endovascular treatment of the aneurysm, the prevention and treatment of cerebral vasospasm include maintenance of adequate volume status and systemic arterial pressure. Central hemodynamic monitoring, including central venous and pulmonary artery pressures, may be required.
 2. **Hydrocephalus** can also develop acutely or chronically after SAH, especially if large amounts of blood are seen on CT scan. Neurologic deterioration after diagnosis of SAH should prompt a repeat head CT scan to evaluate for the development of hydrocephalus. External ventriculostomy may be required, although serial lumbar punctures may be sufficient to treat some cases of hydrocephalus, with resolution over several days to weeks.
 2. **Spontaneous intraparenchymal hemorrhage** (see [section III.A.1.a](#)).
 3. **Spontaneous intraventricular hemorrhage** may result from vascular malformations, hypertensive hemorrhage, or extension of intraparenchymal hemorrhage. Angiography can be used in determining the etiology. Patients are observed for the development of hydrocephalus, which requires external CSF drainage (ventriculostomy).
 4. **Pituitary apoplexy** occurs after hemorrhage into a pituitary neoplasm. Patients typically present with acute headache and visual symptoms, such as decreased acuity, visual field cut, ptosis, or diplopia, which result from compression of nearby cranial nerves. Life-threatening panhypopituitarism can occur. Treatment involves hormonal replacement, correction of any electrolyte abnormalities, and emergent CT scan or MR scan to evaluate for hemorrhagic pituitary lesions. Emergent evacuation of hematoma may preserve vision.
- E. Infections**
1. **Cerebral abscesses** can result from hematogenous or local traumatic spread of a septic process. Underlying abnormalities are common, such as an immunocompromised state or systemic arteriovenous shunting. Presenting symptoms include those of increased ICP, focal deficits, or seizures. A head CT scan with intravenous contrast classically shows a ring-enhancing lesion. Early-stage cerebral abscess may respond to medical management alone. If no mass effect is present, a lumbar puncture can be safely performed, usually demonstrating an infectious profile (increased white blood cell count, increased protein level, decreased glucose level). Abscesses larger than 3 cm, failure of medical management, and need for tissue diagnosis are common indications for surgical drainage by open craniotomy or stereotactic drainage. Prolonged intravenous antibiotics are indicated.
 2. **Intracranial epidural abscesses** can result from skull fractures or contiguous spread from sinusitis, otitis, or mastoiditis. Associated osteomyelitis may also occur. Treatment involves operative drainage and long-term antibiotics.
 3. **Subdural empyema** should be suspected in the patient who presents with fever, nuchal rigidity, and focal neurologic deficit (e.g., hemiparesis). Seizures and decreased level of consciousness also are seen more commonly with subdural empyema than with epidural abscesses. A subdural hypodense mass with enhancement and, often, associated cerebral edema is seen on contrast CT scanning or MR scanning. The infectious process can spread along the subdural space, making evacuation emergent. Anaerobic organisms are more common, and long-term antibiotics are necessary after surgical drainage.
 4. **Spinal epidural abscesses** can become a surgical emergency. Severe neck or back pain in the setting of fever should raise concern. Although neurologic deficit may not occur initially, often progressive evidence of cord compression exists. MR scan or myelography demonstrates the lesion. Surgical evacuation is most often necessary, although antibiotics alone can be attempted if neither neurologic compromise nor a large collection is present. Even with surgical drainage, an extended course of antibiotics is required. Disc space infection and spinal osteomyelitis can occur in association with or separately from spinal epidural abscesses. Antibiotic therapy usually is effective. An elevated erythrocyte sedimentation rate generally is present and falls with effective treatment.
- F. Spinal cord compression**
1. **Diagnosis.** Nontraumatic spinal cord compression can result from metastatic tumor or another adjacent mass lesion. Patients present initially with pain followed by progressive or sudden neurologic symptoms. Lung, breast, and prostate are the most common sources of metastases (*Neurosurgery* 21:676, 1987). Examination usually reveals weakness, long-tract signs (spasticity, hyperactive reflexes, upgoing toes), or sphincter dysfunction. It is important to distinguish myelopathy from radiculopathy. The latter presents with pain, sensory changes, and weakness in a dermatomal pattern. Emergent MR scan or myelography to demonstrate the presence and level of the lesion confirms diagnosis of cord compression. Imaging should extend to higher spinal levels if no lesion is found because, for example, a cervical lesion can present with only lower-extremity symptoms; alternatively, multilevel involvement can be present.
 2. **Treatment** begins with **steroids** (dexamethasone, 10 mg i.v., followed by 4–10 mg p.o. or i.v. every 6 hours; higher doses frequently are given for severe deficits), which are administered immediately if spinal cord compression is suspected. The neurosurgical priorities include **decompression** if a deficit is present and spinal **stabilization and fusion** if bony destruction is prominent (*Neurosurgery* 17:424, 1985). Vertebral corpectomy and reconstruction with internal fixation often are required (simple laminectomy may not be helpful). Emergent **radiation therapy** to the area of compression may be preferable to surgical intervention in some cases.

Treatment of Orthopedic Injuries

I. Initial assessment

II. Radiologic examination

III. Fractures and dislocations

IV. Orthopedic emergencies

V. Soft-tissue injury

VI. Practical procedures

Treatment of Orthopedic Injuries

I. Initial assessment

- A. **Priorities of management. ABCs** (airway, breathing, and circulation) take precedence over extremity injuries, but multisystem-injured patients benefit from aggressive treatment of extremity and pelvic trauma.
- B. **History.** An understanding of the mechanism of injury with details of the accident helps to direct assessment and management of patients with musculoskeletal injuries. Knowledge of patient age, associated medical conditions, and preinjury functional status is also helpful.
- C. **Examination**
 1. **Remove all of the patient's clothing.** Observe the patient's entire affected extremity for deformity and asymmetry compared to the opposite side. **Palpate** all extremities, noting tenderness, crepitus, deformity, or instability. Assess joint **range of motion**. In suspected cervical spine injury, maintain the patient in a cervical collar until radiographs are obtained and the patient is able to comply with a physical examination. Logroll the patient to examine the back.
 2. **Assess vascular status** by checking pulses, body temperature, and color and comparing these to the opposite side. Remember that normal pulses do not rule out compartment syndrome.
 3. **Sensorimotor evaluation.** Little value exists in grading muscle strength in the acute setting, except for spinal cord injury in which the neurologic status is evolving. If an abnormality is detected, a more detailed examination is needed. Sensory examination should be performed with light touch and pinprick, along with two-point discrimination in upper-extremity injuries.
 4. **Associated injuries.** Relate the location of the trauma to structures present at the same level. If the patient is unconscious, spinal and pelvic injuries must be ruled out.

II. Radiologic examination. All trauma patients and unconscious patients must have screening chest, pelvis, and lateral cervical spine radiographs. The lateral cervical spine radiograph must include all cervical vertebrae down through the C7-T1 junction. Assessment of extremity fractures and dislocations should include two views, 90 degrees to each other, of the affected area and should include the joints above and below the injured area. Dislocations of the knee and any other joint (i.e., ankle) that involve neurovascular or soft-tissue compromise should be reduced before radiographs.

III. Fractures and dislocations

A. Terminology and classification

1. **Anatomic location** refers usually to the proximal, middle, or distal portion of the bone. *Epiphyseal, metaphyseal, and diaphyseal* or *head, base, and shaft* are also acceptable descriptive terms.
2. **The direction of fracture is transverse** if perpendicular to the long axis of the bone and **oblique** if angled 45–60 degrees to the bone. **Spiral** fractures are intuitively spiral in appearance and are fractures caused by a torsional mechanism. **Comminuted** injuries have more than two fragments.
3. **Alignment** refers to the amount of angulation between the proximal (closest to the trunk) fragment and distal (closest to the end of the extremity) fragment. **Apposition** denotes the amount of cortical contact between fragments. **Displacement** denotes the distance between cortical surfaces. Intraarticular fractures involve the joint surface.
4. **Stable** fractures do not displace after reduction, whereas **unstable** injuries do displace.
5. **Soft-tissue injury.** **Closed** fractures are those with the overlying skin intact. **Open (compound)** fractures occur when the overlying skin is disrupted and the fracture communicates with the external environment. **Complicated** fractures are those with associated neurovascular, ligamentous, or muscular injury.
6. **Subluxation** refers to joint disruption with partial contact between joint surfaces. **Dislocation** refers to complete loss of contact between joint surfaces. Both are described by the position of the distal bone in relation to its proximal articulation.

B. General management principles

1. **Dislocation**
 - a. All dislocated joints, especially in the setting of **neurovascular compromise**, should be reduced in the emergency room if adequate and safe anesthesia can be administered (see [section VI](#)). Successful reduction reduces the risk of soft-tissue injury (e.g., pressure necrosis) and neurovascular compromise.
 - b. **Hip and knee** dislocations require immediate reduction in an attempt to avoid secondary complications, including avascular necrosis (hip) or neurovascular compromise (knee). Postreduction angiography is performed after knee dislocations when pulses are asymmetric before or after reduction, or both.
 - c. **Wrist, ankle, and foot** dislocations often are associated with a fracture, thus creating an unstable joint. In this setting, reduction may not be maintained in a splint or cast, necessitating definitive surgical intervention.
2. **Fracture** is diagnosed by history of the injury and symptoms of pain, loss of motion, and swelling. Physical examination findings of fracture include crepitus, tenderness, swelling, and/or deformity. Because any of these signs may be absent, a high index of suspicion is needed. Confirmation of fracture is obtained via radiographs.
 - a. **Treatment** is directed toward reduction, if indicated, and immobilization. Uncontrolled movement is avoided to prevent further injury. In open fractures, the wound is irrigated thoroughly and covered with a sterile dressing soaked with normal saline (see [section IV](#)). Direct pressure over pulsatile bleeding or clamping of obvious bleeding vessels is usually effective. Tourniquets are generally not used. Gentle realignment of the limb before radiographic assessment is beneficial and mandatory when the overlying skin or distal blood flow is compromised.
 1. **Stable fractures** do not displace without additional trauma but are immobilized to decrease pain and allow healing.
 2. **Unstable fractures** require immobilization and, often, surgical stabilization. Intraarticular fractures with more than 2 mm articular displacement are treated similarly.
 3. **Closed reduction** is performed as described in [section VI](#). In the case of skin, soft-tissue, or neurovascular compromise, immediate reduction to improve alignment is performed. Reduction may not be attainable because of the fracture pattern or soft-tissue interposition. In these cases, the fracture may be immobilized in the best attainable position until early surgical stabilization, especially when neurovascular, soft-tissue, or skin compromise is present.
 - b. **Open fractures** (see [section IV](#)).
 - c. **Pediatric musculoskeletal fractures**
 1. **Younger patients have a greater potential for bony remodeling**, and therefore a greater amount of angulation is acceptable. However, often some reduction of deformity is beneficial and encouraged to decrease the risk of permanent deformity.
 2. **Physeal plate injuries** are common because this is the weakest part of the bone. The physeal plate is the cartilage region responsible for longitudinal growth. The Salter-Harris classification categorizes these fractures into five types. Type I injuries involve a fracture through the growth plate without any bony involvement. Type II injury occurs when disruption of the growth plate is associated with a metaphyseal fracture. Type III involves growth-plate disruption and an epiphyseal fracture. Fracture through the metaphysis and across the growth plate and epiphysis is a type IV injury. Type V occurs with a crush injury to the growth plate.
 3. **Only qualified personnel should perform reductions**, unless skin or neurovascular compromise mandates immediate improvement of position. Adequate immobilization of the injured area and the joints above and below is necessary.
 - d. **Hazards in fracture management.** Observe the patient closely for possible compartment syndrome, especially in leg and forearm injuries. Neurovascular compromise after plaster immobilization usually is secondary to swelling, and splints and bandages should be loosened to accommodate anticipated swelling. Circumferential casting in the acute setting should generally be avoided. If the patient is to return home, he or she should be instructed on the

warning signs of early compartment syndrome and told to return to the hospital if these symptoms develop (see [section IV](#)).

C. Upper extremity

1. **Clavicle.** Injury usually occurs after a fall on an outstretched hand or shoulder and, less commonly, secondary to a direct blow. Deformity may be clinically apparent, and pain occurs with motion. Close attention to the neurovascular status is needed because subclavian structures may be damaged. These fractures may be treated with a sling to counter the weight of the arm or a figure-of-eight splint. Most clavicle fractures heal with nonoperative treatment.
2. **Sternoclavicular joint.** Injury ranges from simple sprains to subluxation or dislocations. Computed tomographic (CT) scan is often necessary to determine the direction of dislocation. Anterior dislocations are treated with a sling or shoulder immobilizer if the skin is not compromised. Posterior dislocations can compress the mediastinal structures, causing hoarseness, dyspnea, dysphagia, and engorged neck veins, and they require emergent reduction. To facilitate reduction, a rolled towel is placed between the scapulae, posterior pressure is applied to both shoulders, and the clavicle is pulled from the retrosternal area. A towel clip can be used to grasp the proximal clavicle to facilitate reduction under general anesthesia. This should be performed with a general or thoracic surgeon in case there is injury to the lung or great vessels. A figure-of-eight bandage is applied.
3. **Acromioclavicular joint.** Injury occurs after a direct blow to the acromion or a fall on the extremity, with injury ranging from sprain to complete dislocation. Pain, swelling, tenderness to palpation, and limited motion are present. Stress views are obtained with 5–10 lb of weight tied to each hand. Sprains and subluxations are treated with a sling and early motion, but dislocations may require reduction and fixation, especially if the skin is compromised.
4. **Shoulder**
 - a. **Dislocations.** Anterior dislocations (most common) occur after forced abduction or external rotation (or both). Posterior dislocations result from a direct blow or seizure and often are missed on initial examination. Inferior dislocations occur after hyperabduction injuries. Pain with motion and shoulder asymmetry are present, with the humeral head often palpable anteriorly, posteriorly, or inferiorly. Neurovascular status, especially deltoid function and sensation about the lateral proximal arm to assess axillary nerve function, must be adequately documented before reduction. Anteroposterior, scapular lateral, and axillary radiographs are needed to look for humeral head defects and associated fractures. Reduction is performed as described in [section VI](#). Reduction is confirmed with radiographs, and neurovascular status is documented. A sling or shoulder immobilizer is applied, and motion is initiated early (3–5 days). Anterior dislocations are stable in internal rotation, whereas posterior dislocations are stable in external rotation and may require prolonged immobilization. Dislocations in older patients are associated with acute tears of the rotator cuff, causing weakness or pain with abduction and rotation. The redislocation rate is inversely proportional to the patient's age.
 - b. **Soft-tissue injury** (see [section V](#)).
5. **Humerus**
 - a. **Proximal fractures** commonly occur in older patients after a fall on the extremity. Examination reveals swelling, tenderness, pain with motion, and ecchymosis of the shoulder and lateral chest wall. When the fracture is nondisplaced, treatment involves a sling and early circumduction exercises. Displaced fractures require reduction and can be associated with neurovascular injury. Fracture-dislocations of the glenohumeral joint often are irreducible by closed means, requiring emergent surgical intervention. Attempts at closed reduction can result in further displacement and neurovascular injury.
 - b. **Diaphyseal fractures** usually are in the middle third of the humerus. Neurovascular status is checked closely. The radial nerve is especially vulnerable and should be evaluated by checking for active wrist and thumb extension and intact sensation in the dorsal first web space. Transverse fractures of the middle third are most commonly associated with radial neuropraxia, whereas spiral fractures of the distal third present a higher risk of laceration or entrapment of the radial nerve. Treatment involves immobilization in a coaptation splint or hanging arm cast and eventual stabilization in a fracture brace. In the acute setting, operative fixation is indicated in cases of multiple trauma, inadequate closed reduction, radial nerve palsy after reduction, open fractures, segmental fracture, bilateral humerus fractures, or ipsilateral forearm fractures.
 - c. **Distal (supracondylar) fractures** are most common in children and older individuals and occur after a fall on the elbow. Careful examination for neurovascular compromise should be performed because these injuries may constitute a surgical emergency. In adults, closed reduction and repositioning in the emergency room are performed to improve or protect neurovascular status. Definitive treatment in the operating room is required for displaced fractures in children and comminuted fractures with intraarticular extension in adults.
6. **Elbow**
 - a. **Olecranon fractures** usually are evident by lack of active elbow extension and a palpable defect, if displaced. Nondisplaced fractures are treated with a posterior splint with the elbow flexed to 90 degrees, and displaced fractures are reduced and stabilized surgically.
 - b. **Elbow dislocations** usually occur posteriorly, after a fall on an outstretched arm, but can also occur anteriorly, medially, or laterally. Examination reveals asymmetry between sides, and neurovascular status should be checked closely. Radiographs confirm the diagnosis and reveal the direction and any associated fracture. Treatment involves immediate reduction (see [section VI](#)). After reduction, the elbow is assessed for stability, and neurovascular status is documented. The joint is splinted in a stable position. When immobilized, the elbow has the propensity to become stiff, and, thus, for stable reductions, early motion exercises are mandatory.
7. **Forearm**
 - a. **Radial head fractures** commonly occur after a fall on an outstretched hand; the patient presents with tenderness over the proximal radius and pain with forearm rotation. To facilitate examination and confirm the diagnosis, the joint should be aspirated to check for lipohearthrosis (indicative of a fracture) and injected with lidocaine (see [section VI](#)). Elbow motion should then be documented. Motion block, articular fractures involving more than 25–30% of the joint surface, or those fractures with severe comminution or displacement require surgical intervention. Otherwise, treatment involves sling immobilization for 3–5 days and early motion exercises.
 - b. **Fracture of the proximal ulna** can be associated with dislocation of the radial head (Monteggia's fracture). This fracture commonly occurs in children, and closed reduction and splinting usually are successful. When reduction is unsuccessful or when Monteggia's fracture occurs in adults, it is treated operatively, with open reduction and internal fixation of the ulna.
 - c. **Diaphyseal (radial and ulnar) fractures** may be associated with compartment syndrome, and careful examination is needed (see [section IV](#)). In children, closed reduction and casting usually are effective, but in adults, open reduction and surgical fixation are required. For either group, nondisplaced fractures are placed in a posterior long-arm splint. Fractures of the distal half of the radius can be associated with disruption of the distal radioulnar joint (Galeazzi's fracture). If the distal radioulnar cannot be reduced and the reduction is unstable, open reduction and fixation are indicated.
 - d. **Distal fractures** occur usually after a fall on an outstretched hand. Volar or dorsal displacement of the distal fragment may occur. These fractures can be severely comminuted or have intraarticular extension. Children should be assessed for possible physeal involvement. Closed reduction and splinting above the elbow are indicated. Operative reduction and fixation are necessary in cases of unstable fractures, inadequate closed reduction, or displaced intraarticular fragments.
8. **Wrist fractures**
 - a. **Scaphoid fractures** present with painful wrist motion and anatomic "snuffbox" tenderness. Even if a fracture is not evident on X-ray, a thumb spica splint is applied until repeat radiographic examination is performed. Nondisplaced fractures are treated similarly. Fractures with more than 1 mm displacement require surgical intervention.
 - b. **Lunate and perilunate injuries** usually occur after wrist hyperextension and present with pain, tenderness, limitation of motion, and possible fullness on the volar wrist. Median neuropathy may also be present. Reduction is performed by applying longitudinal traction, hyperextending of the wrist, and then applying pressure over the lunate. Surgical stabilization is often needed to maintain reduction and reestablish normal ligamentous anatomy.
 - c. **Carpometacarpal dislocations** are rare and are difficult to diagnose clinically because of considerable swelling. Radiographic evaluation usually is diagnostic. Reduction, if needed, and splinting are performed to prevent further injury or displacement.
9. **Hand.** Although the position of immobilization ultimately depends on the particular injury treated, the "safe position" can always be used (see [section VI](#)).
 - a. **Extraarticular fractures** are treated with reduction, if needed, and immobilization in a splint. Radial or ulnar gutter or thumb spica splints are used for metacarpal fractures, depending on their location. Prefabricated aluminum splint material usually is adequate for phalangeal fractures. Intraarticular and unstable fractures often require surgical intervention.
 - b. **Dislocations** are treated with closed reduction and immobilization. Dorsal metacarpophalangeal joint dislocations irreducible by closed technique require immediate open reduction in the operating room. Fracture dislocations can occur at any joint, especially the base of the first or fifth metacarpal. These injuries are often unstable and treated surgically.
 - c. **Fingertip injuries.** Painful **subungual hematomas** are decompressed by burning a hole in the nail with a hand-held portable electrocautery or by penetrating the nail with a large-bore needle after a digital block. When concurrent fracture of the distal phalanx is present, nail-bed repair may be needed. Small areas of skin loss require dressing changes and possible skin graft. Larger, full-thickness loss without nail involvement may require a graft or local coverage. **Amputations** through bone are treated with skeletal shortening and closure.

D. Pelvic fractures

1. **History.** Pelvic fractures are typically high-energy injuries and are a common cause of death associated with trauma secondary to associated soft-tissue injury and bleeding. Many patients are in shock, and it is often difficult to distinguish intraabdominal bleeding from pelvic hemorrhage. Pelvic bleeding may result in a loss of 2–3 L of blood or more, and replacement must be handled aggressively.
2. **Examination**
 - a. **A high index of suspicion is needed**, especially when tenderness, crepitus, or instability with compression or distraction of the iliac wings is present. A screening anteroposterior pelvic radiograph is essential in the initial examination. Minimizing manipulation of the pelvis when radiographs demonstrate

significant fracture or dislocation decreases the risk of bleeding, dislodging clots, or causing further soft-tissue damage.

- b. **Pelvic and rectal examination** are performed to check for blood, masses, a high-riding prostate, and open communication with fracture. Open pelvic fractures are associated with a high morbidity and mortality. Although placement of an indwelling urinary catheter helps monitor volume status, genitourinary tract injury is common and is suspected when blood is present at the urethral meatus, when a high-riding prostate is found, or when symphysis pubis diastasis is present. In these cases, a retrograde urethrogram and a cystogram should be performed before placing a Foley catheter. Once stabilized, the patient should undergo further X-rays, including inlet and outlet views, to better assess the pelvic injury. CT is extremely helpful in assessing sacroiliac joint and sacral injuries.

3. Treatment

- a. **Maintenance of blood volume, fracture fragment reduction, and immobilization** are essential first steps to decrease further hemorrhage. If the patient is hemodynamically stable, surgical intervention can be performed electively to allow complete assessment of associated injuries and resuscitation of the patient. If, however, the patient is hemodynamically unstable after fluid resuscitation and has an unstable pelvic injury, he or she should undergo temporary manipulation of the pelvis (i.e., tying a sheet around the pelvis, laying the patient on his/her side, or applying a pelvic C-clamp) to reduce pelvic volume until anterior pelvic external fixation can be applied. An intraabdominal source of continued bleeding must then be ruled out before pelvic angiography and embolization are considered.
- b. **Fractures not involving weightbearing regions (i.e., pubic rami) or without associated pelvic ring disruption** are treated symptomatically, with increased weightbearing as pain resolves. Pelvic ring disruptions or fractures involving weightbearing areas require restricted weightbearing, traction, and, possibly, surgical intervention.

E. Lower-extremity fractures and dislocations

1. Hip dislocation

- a. **Hip dislocation usually occurs secondary to severe trauma** in association with considerable soft-tissue damage. Examination reveals pain with attempted range of motion. Anterior dislocations occur with forced abduction, leaving the extremity abducted, externally rotated, and flexed. Posterior dislocations occur when a force is applied to a flexed knee, such as striking the dashboard, leaving the extremity internally rotated, shortened, and adducted. Dislocations may be associated with a fracture of the femoral head or acetabulum. Central fracture-dislocation results from a blow to the greater trochanter, by which the extremity is shortened. In all cases, radiographs are essential to confirm the direction of dislocation and assess for associated femoral or acetabular fracture.
- b. **Treatment.** Immediate closed reduction, and open reduction if necessary, is performed after careful assessment and documentation of the patient's neurovascular status (see [section VI](#)). Skeletal traction through the distal femur should then be applied when femoral head or acetabular fractures are identified. Postreduction X-rays (including anteroposterior and Judet views) are mandatory to assess joint congruity and rule out the presence of intraarticular fragments. CT scans are obtained to rule out the presence of incarcerated fragments and to assess femoral head fractures or acetabular fractures, particularly when incongruence is noted on radiographs.
- c. **Complications.** Hip dislocations are associated with neurovascular compromise (peroneal division of the sciatic nerve), avascular necrosis, and posttraumatic osteoarthritis. Immediate congruent reduction is imperative.

2. Femur

- a. **Femoral neck fractures** usually occur after low-energy injuries, such as a stumble or fall in older patients, but are generally the result of high-energy injuries in younger individuals. A high index of suspicion is needed because patients often present with minimal groin or medial knee pain. If routine radiographs do not reveal any abnormality in the older patient, MR scan or bone scan may be needed. Displaced fractures cause shortening and external rotation of the extremity. In younger patients, stress fractures and nondisplaced fractures are treated with non-weightbearing ambulation and, possibly, surgical stabilization. Displaced fractures require immediate anatomic reduction and internal fixation to minimize the risk of avascular necrosis. Older patients generally require surgical intervention with reduction and percutaneous screw fixation for nondisplaced fractures or hemireplacement for displaced fractures.
- b. **Peritrochanteric fractures.** Older patients usually present after a fall or a direct blow to the hip. The extremity is shortened and externally rotated, and there is pain on attempted motion. Treatment consists of gentle skin traction to decrease pain until surgery is performed. Younger patients sustain subtrochanteric fractures secondary to direct high-energy trauma. Surgical stabilization is needed. The patient is maintained in skeletal traction until operation, with careful monitoring of hemodynamic status and the limb's neurovascular status. Methods of stabilization include plate and screw fixation or use of a reconstruction intramedullary nail.
- c. **Femoral shaft fractures** occur with significant trauma and are associated with considerable bleeding, soft-tissue damage, and other extremity injuries. The injury is obvious, but adequate examination of the remainder of the extremity is necessary. Ipsilateral injuries to the knee are common. X-ray evaluation must include the hip and knee joint to detect the presence of other fractures. Thomas splints provide immediate stabilization, but skeletal traction and surgical stabilization are needed. The gold standard of treatment is an antegrade, reamed, statically locked intramedullary nail.
- d. **Supracondylar fractures.** Careful assessment of the neurovascular status is essential. Nondisplaced fractures can be treated with splint and cast immobilization, but displaced fractures typically are treated with surgical intervention.

3. Knee

- a. **Patellar fracture or dislocation.** Nondisplaced fractures in association with an intact extensor mechanism are treated with an immobilizer or a cylinder cast. Displaced fractures require surgical intervention because the extensor mechanism is usually disrupted. Patellar dislocation is evident clinically and is reduced with sedation, extension, and pressure to push the patella toward the midline. Radiographs confirm reduction, and a knee immobilizer is applied.
- b. **Knee dislocations** are rare but require immediate reduction and careful assessment of the neurovascular status. When pulses are diminished before or after reduction, immediate arteriography is needed to rule out vascular injury. If vascular repair is required, a spanning external fixator can be applied to stabilize the knee. After repair of vascular injury, prophylactic fasciotomy should be performed. Often, reconstruction of soft-tissue structures is necessary to restore knee stability.

4. Tibia

- a. **Plateau fractures** can be displaced, nondisplaced, or comminuted. The knee must be examined for stability. If the fracture is nondisplaced, early mobilization but limited weightbearing should be instituted. Displaced fractures of greater than 2 mm require surgical intervention to restore articular congruity and joint stability.
- b. **Diaphyseal fractures** may occur with either torsional, low-energy mechanisms or high-energy direct blows. Given that the tibia is subcutaneous, open fractures are common (see [section IV](#)). Soft tissues should be adequately assessed, and neurovascular status must be checked frequently to rule out the development of a compartment syndrome. Skin compromise secondary to severe angulation requires immediate realignment and splint immobilization. Treatment by either splint/cast immobilization or intramedullary nailing depends on the fracture pattern and the amount of comminution, shortening, rotation, and angulation.
- c. **Distal tibia articular fractures (Pilon fractures)** are associated with severe soft-tissue injury and involve the joint surface. In the case of displaced and shortened fractures, initial management includes closed reduction and application of a temporary external fixator, which acts as a form of portable traction. External fixation is maintained until soft tissues can tolerate formal open reduction and internal fixation.

5. **Ankle fracture.** The precise location of pain, swelling, and tenderness must be assessed. Radiographs, including anteroposterior, lateral, and mortise views, are obtained when history and physical examination suggest fracture. Bimalleolar (medial and lateral) fractures represent unstable ankle injuries and require surgical intervention. Isolated lateral malleolar fractures without medial tenderness are stable; they are treated with splint immobilization and early weightbearing. When lateral malleolar fractures occur together with medial tenderness on examination, a mortise stress X-ray (stabilizing the distal tibia and externally rotating the patient's foot) is obtained to assess joint stability. Widening of more than 2 mm of the medial joint space represents an unstable ankle. Initial management of ankle fractures includes closed reduction, splinting, and strict elevation to help avoid soft-tissue complications or swelling.

6. Foot

- a. **Calcaneus fractures** usually occur after a fall from a height. These injuries are often bilateral and can be associated with lumbosacral spine injuries as well. Examination of these areas is necessary, as is radiologic assessment. CT scans help to further delineate these fractures and to direct treatment. These injuries are associated with considerable swelling and blister formation. A well-padded splint should be applied and the limb elevated and observed. Fractures with severe subtalar joint depression and comminution may require open reduction and internal fixation. Surgery is typically delayed for 10–14 days to allow recovery of the soft tissues.
- b. **Talus**
 1. **Peritalar dislocation** occurs after varying degrees of forced foot inversion. **Skin disruption or fracture of the talar neck** may occur. Examination often reveals deformity, with the patient's foot medial to his or her leg and the lateral skin tented, with the talus palpable under the skin. Emergent reduction is performed to decrease the chance of avascular necrosis and eliminate skin compromise. Soft-tissue interposition can prevent closed reduction, in which case immediate open reduction is required. Associated fractures must be anatomicly reduced and stabilized.
 2. **Fractures of the talar neck** occur with forced ankle flexion or extension. A high index of suspicion is needed because these fractures may not be apparent on examination or radiographs. Displaced fractures of greater than 2 mm require anatomic reduction and fixation to minimize the risk of avascular necrosis. Nondisplaced fractures can be treated with strict non-weightbearing, immobilization, and close follow-up.
- c. **Fracture dislocations of the tarsometatarsal joints** (Lisfranc's fracture-dislocation) are complex and difficult to treat. These injuries are diagnosed

radiographically by incongruity between the medial base of the second metatarsal and the medial edge of the middle cuneiform, which normally are collinear. Treatment consists of splinting, elevation, non-weightbearing, and operative reduction and fixation.

- d. **Lesser metatarsal fractures** are treated with a pressure dressing and elevation until pain and swelling subside. First metatarsal fractures, if displaced, may require surgical intervention. Transverse fractures of the diaphysis of the fifth metatarsal ("Jones" fracture) requires strict non-weightbearing and immobilization, and, possibly, surgery, whereas avulsion fractures of the proximal fifth metatarsal metaphysis ("pseudo-Jones") can be treated with early weightbearing.
- e. **Toe injuries** are best treated by "buddy" taping to the adjacent digit. The patient may then ambulate in a hard-soled shoe.

IV. Orthopedic emergencies

A. **Infection** must be considered in any patient with localized findings (pain, redness, swelling, and warmth) or systemic findings (malaise, fever, or tachycardia).

1. **Acute infections** often respond to prompt assessment, medical treatment with appropriate antibiotic therapy (cellulitis), and surgical decompression (abscess, septic bursitis).
 - a. **Septic arthritis** usually occurs secondary to immunosuppression, systemic infection, preexisting joint disease, or previous arthroscopy.
 1. **Examination** reveals tenderness, effusion, increased warmth, and pain with motion. The patient may have an elevated erythrocyte sedimentation rate, C-reactive protein, and/or white blood cell (WBC) count. Diagnosis is confirmed by needle aspiration and laboratory analysis of synovial fluid. Inflammatory and noninflammatory processes (e.g., gout) are ruled out by analyzing the synovial fluid for cell count and differential, Gram stain, routine aerobic and anaerobic cultures, and crystals.
 2. **Baseline radiographs** (significant changes occur late, 7–10 days) should be obtained and broad-spectrum intravenous antibiotics (first-generation cephalosporin and gentamicin) initiated after adequate specimens are obtained and the limb immobilized. If septic arthritis is diagnosed, some means of cleansing the joint should be performed (serial aspirations, arthroscopic débridement, or arthroscopy) to prevent cartilage degradation and further systemic illness.
 - b. **Osteomyelitis.** Childhood osteomyelitis is most common secondary to hematogenous spread of the bacteria to the metaphysis. Adult osteomyelitis typically occurs from direct inoculation via surgery, an open fracture, or chronic soft-tissue ulcers. Hematogenous spread may occur in cases of intravenous drug abuse, sickle cell disease, or immunosuppression.
 1. **Physical examination** findings are similar to those in septic arthritis but also may reveal bony tenderness and drainage.
 2. **Imaging** studies including bone scan, MR scan, or both may confirm the diagnosis, along with needle aspiration, bone biopsy, elevated erythrocyte sedimentation rate, elevated C-reactive protein, elevated WBC count, and culture.
 3. **Treatment** typically involves débridement and assessment of soft-tissue coverage followed by an extended course of intravenous antibiotics.
 - c. **Suppurative flexor tenosynovitis.** Patients present with tenderness along the flexor sheath, a semiflexed finger position, pain on extension, and fusiform swelling of the entire finger. Skin wounds may appear innocuous. The patient's entire hand must be examined because infection may extend into other spaces. Immediate surgical decompression, irrigation, and débridement are indicated.
 - d. **Abscess**
 1. **Hand.** Numerous potential spaces exist that can be infected. All present with pain, tense swelling, induration, and tenderness to palpation. Systemic signs of infection may or may not be present. Immediate surgical drainage is mandatory.
 2. **Others.** Infection in the soft tissue may occur anywhere and presents with pain, tenderness, swelling, possible fluctuation, and induration. Fluid collections are readily localized with large-bore needle aspiration. Surgical drainage is performed on a semielective basis unless systemic involvement compromises patient health or management.

B. Fat embolism

1. Fat embolism occurs within 3 days of a long-bone fracture and is characterized by mental status changes, tachycardia, dyspnea, and petechiae.
2. **Laboratory data** reveal a decrease in platelet count ($<15,000 \mu\text{L}$), an arterial oxygen tension of less than 60 mm Hg, increased serum lipase, and, possibly, fat in the urine. Patchy infiltrates appear on the chest X-ray. Electrocardiography can reveal tachycardia, inverted T waves, right bundle-branch block, and depressed S-T segments.
3. **Treatment** involves respiratory support to keep the arterial oxygen tension at 50–100 mm Hg; intubation may be necessary. Positive end-expiratory pressure helps inhibit pulmonary edema. Corticosteroid administration has also been used to treat these complications.

C. **Compartment syndrome** is characterized by an increase in tissue pressure within a closed osteofascial space, which may compromise microcirculation, leading to irreversible muscle injury and eventual rhabdomyolysis. Acute renal failure may then ensue.

1. **Location.** Most frequently, compartment syndrome occurs in the anterior, lateral, or posterior compartments of the leg or the volar or dorsal compartments of the forearm.
2. **Causes.** Bleeding after fracture, crush, or vascular injury is a likely cause. Increased capillary permeability secondary to postischemic swelling, trauma, and burns may also contribute to compartment syndrome. Muscle hypertrophy, tight dressings, and pneumatic antishock garments (i.e., military antishock trousers) are less common causes.
3. **Examination.** Patients at risk for compartment syndrome should be identified early and examined frequently. Signs to watch for are as follows:
 - a. **Pain** out of proportion to the injury and not controlled with appropriate narcotics.
 - b. **Pain with passive motion** of involved muscles or tendons traversing the suspicious compartment.
 - c. **Paresthesias** in the distribution of the peripheral nerves traversing the suspicious compartment.
 - d. **Paralysis, pallor, and pulselessness** are very late signs and likely indicate irreparable soft-tissue injury; if pulses are altered or absent, major arterial occlusion rather than compartment syndrome should be considered in the diagnosis.
 - e. **Elevated compartment pressures** should be measured in high-risk patients or when the clinical examination is suspicious. Infusion, wick, or injection techniques are used to measure the pressure of all compartments in the involved extremity. Several measurements should be taken in different locations, and comparison to pressures in uninvolved compartments can be helpful.
4. **Treatment.** Circumferential bandages, splints, or casts should be removed. For pressures of less than 30 mm Hg, or pressures of 30–40 mm Hg without clear evidence of the syndrome clinically, observation with hourly examination and pressure measurements is appropriate. If examination is equivocal or worrisome, fasciotomy should be performed. For pressures of 30–40 mm Hg with equivocal clinical examination, or those exceeding 40 mm Hg regardless of examination, fasciotomy is in order.

D. **Open fractures and joints.** Lacerations or wounds near fractures or joints often communicate. If exposed bone is not evident, wounds should be probed to determine if communication to fracture is present. Joints may be distended with sterile saline to check for extravasation from adjacent wounds, which would indicate open communication. Air in the joint on X-ray and fat droplets in blood from the wound also confirm communication with a joint or fracture.

1. **Treatment**
 - a. **Assess wounds.**
 - b. **Irrigate grossly contaminated wounds with normal saline.**
 - c. **Apply moist saline-soaked dressing, reduce the fracture or joint, and splint the extremity.**
 - d. **Administer tetanus prophylaxis and intravenous antibiotics.** Grade I injuries with a skin opening less than 1 cm require a first-generation cephalosporin. With grade II and III injuries (skin opening >1 cm and significant soft-tissue stripping), an aminoglycoside should be added. Farm injuries require administration of penicillin to cover for *Clostridium perfringens*.
 - e. **Check the neurovascular status closely and frequently.**
2. **Gunshot injuries**
 - a. The **weapon caliber and type** must be identified. High-energy injuries (shotgun, rifle, or high-caliber handguns) require operative débridement secondary to severe soft-tissue or bony damage. With low-energy injuries (low-caliber handguns), débridement is not always needed because less damage occurs.
 - b. The **neurovascular status** should be checked closely and frequently, especially if the wound is near major neurovascular structures, and radiographs to assess bony damage should be obtained. If the wound is near a joint without evident intraarticular involvement, aspirate for hemarthrosis, sterilely distend the joint capsule with saline, and look for extravasation from the wound.
 - c. **Treatment.** Clean the skin, débride the wound edges, and irrigate thoroughly. Apply a dressing and splint the extremity if a fracture is found. If no neurovascular compromise or compartment syndrome exists, isolated soft-tissue injury is treated with local wound care and oral antibiotics.

E. Traumatic amputation

1. **A team approach is needed** to evaluate for possible replantation, and all necessary consultants should be contacted early.
2. **The proximal stump is cleaned**, and a compressive dressing is applied. Tourniquets are not used. Amputated parts are wrapped in moist gauze, placed in a bag, and cooled (avoid freezing) by placing on ice.

V. Soft-tissue injury

A. Principles of management

1. **In general**, isolated soft-tissue injuries, such as ligament sprains and muscle strains, are treated with rest, ice, compression bandage, and elevation (RICE therapy) with or without immobilization.
2. **Skin**. All devitalized tissue should be débrided. If the wound cannot be closed owing to excessive tension, it should be covered with a moist saline dressing, and a delayed primary closure or skin grafting should be planned.
3. **Muscle**
 - a. **Trauma to the musculotendinous unit** usually is secondary to violent contraction or excessive stretch and ranges from stretch of the fibers to a complete tear and loss of function.
 - b. **Swelling, tenderness, and pain with movement occur**. A defect may be palpable. Immobilization of the extremity and any associated joints on which the muscle acts is adequate for less severe injuries.
4. **Tendon**. Lacerated, ruptured, or avulsed tendons, especially of the upper extremity, should be surgically repaired because failure to do so results in loss of function. Examination reveals loss of motion or weakness. If associated with a laceration, the wound is irrigated thoroughly, débrided, and closed primarily. In grossly contaminated wounds, incision and débridement in the operating room are needed. Splints are applied with the extremity in a functional position.
5. **Ligament**. Injury ranges from mild stretch to complete tear. Pain, localized tenderness, abnormal joint motion or position, or instability may be present on examination. Radiographs may reveal joint incongruence. If the joint is clinically or radiographically unstable, treatment involves immobilization in a reduced position. If no evidence of instability is present, treatment with the RICE principle is used, and early range of motion is encouraged.

B. Common injuries

1. **Rotator cuff tear**. Acute rupture is most commonly associated with traumatic dislocation of the glenohumeral joint, especially in the older patient. It can also be associated with avulsion of the tuberosity. Weakness and pain with attempted shoulder abduction or rotation occurs.
2. **Rupture of the pectoralis major and biceps brachii tendon** often occurs with heavy lifting or exertion and is diagnosed by pain, weakness, and a visible, palpable defect. Initial treatment consists of placement of the extremity in a sling and initiation of early motion.
3. A **ruptured heel cord** usually occurs during running, jumping, or vigorous activity, with sudden pain and difficulty in walking. Examination reveals a palpable defect, weak plantar flexion, and no passive ankle plantar flexion on squeezing the patient's calf (positive "Thompson's sign"). Older patients often can be treated in a splint, with the ankle plantar flexed. Serial casting with progressive dorsiflexion of the foot is then initiated. In younger patients, rerupture rates are less with surgical repair of the tendon.
4. **Ankle sprains** commonly occur with inversion or external rotation of the ankle. Patients present with swelling, ecchymosis, and maximal tenderness along all injured ligaments. Radiographs are normal or reveal clinically insignificant, small cortical avulsions. Initial treatment with RICE is adequate, followed by physical therapy for proprioceptive training.
5. **Knee ligament disruption**. Dislocation must be considered. Stability is assessed. An effusion may be aspirated to examine for lipohemarthrosis, to confirm the presence of a fracture. If no fracture or dislocation exists, an immobilizer is applied for comfort with weightbearing, and early range of motion is emphasized. Common knee injuries include anterior cruciate ligament tears, medial collateral ligament tears, and meniscal tears.

VI. Practical procedures

A. **Common splints**. Splints limit further soft-tissue injury and swelling and help to minimize pain. They also facilitate further clinical and radiographic evaluation. Air splints are used only in the emergency setting because they increase pressure in the extremity and compromise blood flow.

1. **Preparation and application**. Prefabricated splints and immobilizers can be used, if available. Plaster splints consist of plaster and cast padding. The required length, to include a joint above and below the injury, is measured from the uninjured side. Two layers of soft roll are applied against the skin, and extra padding is placed over bony prominences. A ten-layer-thick stack of plaster splint material is wetted in cold to lukewarm water and squeezed until damp. The splint is applied over the soft-roll padding and wrapped lightly with an elastic bandage. The extremity is held in the appropriate position until the plaster is firm.
2. **Upper-extremity splints**. Remove all of the patient's jewelry!
 - a. **Commercial shoulder immobilizers, Velpeau dressing, and sling and swathe** are used for shoulder dislocations, humerus fractures, and some elbow fractures. A pad is placed in the axilla to prevent skin maceration.
 - b. **Figure-of-eight slings** can be used for clavicle fracture stabilization but are often poorly tolerated by adults.
 - c. **Posterior and sugar tong splints** are used in elbow, forearm, and wrist injuries. They are applied with the patient's elbow flexed to 90 degrees, wrist extended 20–30 degrees, and forearm in neutral rotation.
 - d. **Thumb spica, ulnar/radial gutter, and volar/dorsal forearm splints** are used for forearm, hand, and wrist injuries. Finger injuries may be treated with prefabricated aluminum splint material. For wrist and hand injuries, immobilization is performed with the patient's hand and wrist in a so-called **safe position**: the wrist in 20–30 degrees of extension, the metacarpophalangeal joints in 70–80 degrees of flexion, and the interphalangeal joints extended.
3. **Lower-extremity splints**
 - a. **Thomas splints** are used in femur fractures. Traction is applied by an ankle hitch, with countertraction across the ischial tuberosity. The splint is not left in place for longer than 2 hours because sloughing of the skin can occur about the ankle.
 - b. A **Jones dressing** with or without plaster reinforcements is used in acute knee, ankle, calcaneus, and some tibial fractures, where considerable swelling is expected. The injured extremity is wrapped with cotton, followed by a lightly wrapped elastic bandage. Plaster splints can be applied to the posterior, medial, and lateral aspects for added stability. Circumferential plaster should be avoided.
 - c. **Short leg splints** are used in acute leg or foot trauma. They extend from below the knee to the toes and incorporate posterior, medial, and lateral plaster slabs. The ankle should be immobilized in the neutral position.
4. **Precautions**. Bony prominences are padded. Casts or circumferential splints are avoided in acute trauma when swelling is anticipated, unless they are bivalved or split (including padding) to allow swelling. To split the cast, cut a 1-inch strip of plaster from the full length of the cast, split the padding, and spread the cast edges.

B. Anesthesia for fracture and joint reduction

1. **Local anesthesia**. Appropriate sterile technique must be used.
 - a. **Peripheral nerve block**. The peripheral nerves of the hands and feet can be easily blocked using 1% lidocaine without epinephrine (sometimes mixed with 0.25–0.50% bupivacaine for longer-lasting analgesia). The digital nerves of the fingers or toes can be blocked by infiltrating 2–5 mL into the web spaces adjacent to the injured digit. An ankle block may be performed by infiltrating (often with 0.5% bupivacaine) (1) 10 mL posterior to the medial malleolus to block the posterior tibial nerve, (2) 5 mL s.c. from the medial malleolus to the lateral malleolus dorsally to block the saphenous and superficial peroneal nerves, (3) 2.5 mL posterior to the lateral malleolus to block the sural nerve, and, finally, (4) 2.5 mL posterior to the extensor hallucis longus tendon anteriorly to block the deep peroneal nerve. At the wrist, the median nerve and ulnar nerves may be blocked by infiltrating 5 mL local anesthetic into the carpal tunnel and the space dorsal to the flexor carpi ulnaris tendon, respectively.
 - b. **Hematoma block** is especially effective with fractures of the distal radius. A 21-gauge needle is inserted into the fracture site through the dorsal forearm. Aspiration of blood confirms appropriate position of the needle in the fracture site. Approximately 8–10 mL 1% lidocaine without epinephrine is then infiltrated. A hematoma block in conjunction with light sedation often provides excellent analgesia and relaxation for reduction.
 - c. **Intraarticular injection**. To provide analgesia for reduction of fractures and joints, and to position joints for splinting, some joints may be easily injected with local anesthetic. Aspiration of blood in the case of fracture and easy flow of the anesthetic confirm entry into the joint. The ankle may be infiltrated anteriorly adjacent to either malleolus. The elbow may be entered laterally in the triangle formed by the lateral epicondyle, radial head, and olecranon. Finally, the shoulder is infiltrated either anteriorly lateral to the coracoid process or posteriorly 2 cm distal and 1 cm medial to the posterolateral edge of the acromion.
2. **Sedation**. Midazolam (Versed) and morphine are administered intravenously slowly over 2–5 minutes to achieve easily arousable sedation and pain control (usually, a total of 2–5 mg midazolam is needed). Close observation of the respiratory status and monitoring with a pulse oximeter are required. Midazolam sedation is readily reversed with flumazenil (Romazicon) and morphine with naloxone hydrochloride (Narcan). Patients are monitored for 60 minutes after manipulation.

C. Technique for reduction of fracture or dislocation

1. **Dislocation**. After adequate analgesia and sedation as noted, longitudinal traction of the affected extremity (avoiding sudden, forceful movements) is applied, initially gently and progressively with increasing force until reduction is achieved. The dislocated fragment is manipulated by applying pressure in the direction of reduction. Gentle rotation may help but is performed cautiously because long-bone fracture may occur.
2. **Fracture**. Traction is applied first in the direction of the angulation (recreating the injury to release the impaction of the bony ends) and then in line with the long axis of the limb to correct the alignment, rotation, and length. Pressure is applied to the distal fragment in the direction of the reduced position. Postreduction X-rays are obtained in all cases, and the joint or extremity is immobilized.

41 Urologic Surgery

Robert L. Grubb and Robert S. Figenshau

Evaluation of Hematuria

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Genitourinary Trauma

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IV. Genital and urethral injuries

Evaluation of Hematuria

I. **Hematuria** is the hallmark of disease in the genitourinary tract and warrants a thorough investigation. It can be classified into two categories.

- A. **Gross hematuria** typically is associated with urologic pathology, including tumors, urolithiasis, urinary tract infection, benign prostatic hyperplasia (BPH), hemorrhagic cystitis, and trauma.
- B. **Microscopic hematuria** [>3 red blood cells (RBCs)/high-power field] may be a marker for medical renal disease or urologic disease. The hallmark of medical disease is proteinuria (100–300 mg/dL or 2+ to 3+ by dipstick). Dysmorphic RBCs, red cell casts, glucosuria, and azotemia often are present as well. Referral to a nephrologist may be appropriate in these patients.
- C. **Evaluation of hematuria**
 1. **Specific gravity** is noted because RBC rupture occurs in hypotonic urine (specific gravity <1.008). Urinary pH can aid in the diagnosis of disease. Microscopic analysis evaluates RBC morphology, casts, crystals, white blood cell (WBC) count, and bacteria. The presence and character of clots can be revealing; upper tract bleeding may cause vermiform clots, whereas bladder clots may be amorphous.
 2. **Complete blood cell (CBC) count, coagulation parameters, and serum creatinine** are essential.
 3. **Radiologic evaluation** of the upper urinary tract with an intravenous pyelogram (IVP) or retrograde pyelogram is mandatory.
 4. The lower urinary tract is visualized with **cystoscopy**.
 5. **Urine is sent for culture.**
 6. **Urine is sent for cytology.**
 7. **If these tests do not render a diagnosis**, renal parenchymal imaging with ultrasonography or computed tomographic (CT) scanning is necessary. The use of CT scan as a lone modality for hematuria is still controversial. If a diagnosis still is not made, a repeat urinalysis in 6 months is necessary, with another complete workup if the hematuria persists. Evaluation of microscopic hematuria can be performed on an outpatient basis. Anticoagulation at therapeutic levels does not predispose patients to hematuria; these patients require complete evaluation.
- D. **Treatment.** Gross hematuria requires urgent evaluation.
 1. **Patients passing blood clots** may require irrigation and initiation of continuous bladder irrigation with normal saline via a three-way Foley catheter [22–26 French (Fr.)]. Prostatic bleeding may be controlled with gentle catheter traction.
 2. **Bladder irrigation** with 1% alum or 1% silver nitrate can alleviate persistent bleeding. It is imperative that the bladder is free of clots before initiating alum or silver nitrate irrigation. Silver nitrate and alum are astringents that act by protein precipitation over the bleeding surfaces. Initiation of intravenous epsilon-aminocaproic acid (Amicar; 5 g in 250 mL D5W infused over 1 hour, then 1 g per hour continuous infusion) can also be used to help control bleeding. Epsilon-aminocaproic acid is an inhibitor of fibrinolysis and can be associated with thromboembolic complications. It should be used judiciously. It may also be given orally or administered intravesically.
 3. **Persistent bleeding** on continuous bladder irrigation or significant gross hematuria in the unstable patient requires immediate cystoscopic evaluation to localize and control bleeding.

Diseases of the Kidney

I. Evaluation of renal masses

- A. Increased use of abdominal CT scanning and ultrasonography has resulted in detection of more asymptomatic **renal masses**. These masses must be characterized as benign or malignant.
- B. **The vast majority of masses are benign cysts** (*Radiology* 179:307, 1991).
- C. **Renal cysts** occur in one-half of persons older than 50 years. Other **benign lesions** include infarction, abscess, hemangioma, angiomyolipoma, and adenoma. **Most solid renal masses** (85–90%) are renal cell carcinoma. Up to 25% of these cancers measure less than 3 cm in diameter and are diagnosed incidentally by imaging obtained for other reasons (*Radiology* 170:699, 1989). Improved imaging and early diagnosis have dramatically increased the number of patients who present with curable disease. **Other malignant lesions** include transitional cell carcinoma, oncocytoma (benign in the vast majority of cases), sarcoma, lymphoma, leukemia, and metastatic tumor (lung, breast, gastrointestinal, prostate, pancreatic, and melanoma).
- D. **Presentation.** The historical triad of flank pain, hematuria, and flank mass occurs less than 10% of the time.
 1. Ten percent to 40% of renal cell carcinomas are associated with **paraneoplastic syndromes**.
 - a. Hypertension from **renin overproduction** is common.
 - b. **Stauffer's syndrome**, or nonmetastatic hepatic dysfunction, is seen in some patients and resolves after tumor removal.
 - c. **Hypercalcemia** from parathyroid hormone–like protein produced by the tumor also may occur.
 - d. **Erythrocytosis** can occur as a result of production of erythropoietin by the tumor.
- E. **Staging.** Adequate staging of renal cell carcinoma is imperative to properly guide therapy. In addition to complete history, thorough physical examination, and blood work [CBC, electrolytes (including calcium), creatinine, and liver function tests], a chest radiograph and cross-sectional abdominal imaging (CT or MR scan with contrast) should be obtained. Radionuclide bone scan is not necessary in patients with normal alkaline phosphatase and serum calcium who do not have symptoms of skeletal involvement. Symptoms and results of these basic tests should guide additional radiographic staging. The TNM (tumor, node, metastasis) staging system is outlined in [Table 41-1](#).

Table 41-1. American Joint Committee on Cancer staging for kidney cancer

F. Imaging modalities

1. **CT scan** with and without intravenous contrast is the preferred diagnostic study to evaluate a renal mass. Precontrast images may be hypodense, isodense, or hyperdense compared to normal renal parenchyma; renal cell carcinomas generally enhance, but to a lesser degree than surrounding parenchyma. CT also provides staging information, including local extent of the tumor, presence of regional lymphadenopathy, or presence of distant metastatic lesions (lung, liver, adrenal gland).
 2. **Ultrasonography** is the modality of choice in determining whether a lesion is solid or cystic.
 - a. **Bosniak classification** (*Radiology* 179:307, 1991).
 1. **Category I: simple cyst.**
 2. **Category II: high-density cyst; thin, smooth septa; or linear calcification.**
 3. **Category III: indeterminate lesions.** Numerous or thick septa, or both; thick calcification. These lesions require surgical management.
 4. **Category IV: high probability of malignancy with cystic component, irregular margins, and solid vascular elements.** These lesions require surgical management.
 - b. **Doppler ultrasonography** may be useful for evaluating the extent of vena caval involvement.
 3. **MR scan** is useful to stage renal tumors (especially in patients with renal insufficiency or allergies to contrast dye) and to detect tumor thrombus in the renal vein and inferior vena cava.
 4. **Angiography** rarely is used in evaluating an indeterminate renal mass, but it may be helpful in planning nephron-sparing surgery (partial nephrectomy). Renal cell carcinoma is characteristically hypervascular on arteriography.
 5. **IVP** remains the standard diagnostic modality to evaluate the upper urinary tract of patients presenting with hematuria (38% of renal cell cancers). Renal masses may distort the collecting system. Tomograms may detect 85% of lesions greater than 3 cm.
- G. **Surgery** remains the most effective treatment modality. Ten-year survival after nephrectomy for Robson stage I–II lesions was greater than 78% in one modern series (*J Urol* 159:192, 1998). Nephron-sparing surgery (partial nephrectomy) has been proven to have results similar to those of radical nephrectomy for small (<4 cm) lesions (*Urology* 46:149, 1995). Laparoscopic nephrectomy increasingly is being performed for renal cell cancer.
- H. **Renal cell carcinoma is resistant to radiation and chemotherapy.** Immunotherapy protocols using interferon have demonstrated increased survival after nephrectomy for metastatic renal cell carcinoma (*J Urol* 163:154S, 2000).

Diseases of the Ureter

I. Ureteropelvic junction obstruction (UPJO)

- A. UPJO is often a **congenital anomaly** that results from a stenotic segment of ureter. Acquired lesions may include tortuous or kinked ureters as a result of vesicoureteral reflux, benign tumors such as fibroepithelial polyps, or scarring as a result of stone disease, ischemia, or previous surgical manipulation of the urinary system. The role of crossing vessels (present in one-third of cases) has not been firmly established, although their presence may be associated with treatment failures.
- B. **Presentation.** Although UPJO can be a congenital problem, patients may present at any age. Common symptoms are flank pain, which may be intermittent, hematuria, infection, or, rarely, hypertension.
- C. **Radiographic studies** should determine the site and functional significance of the obstruction. Useful studies include IVP, diuretic renal scintigraphy, and retrograde pyelography. Ultrasound may demonstrate a hydronephrosis, but this is not diagnostic of functional obstruction.
- D. **Open pyeloplasty** is the gold standard treatment, with success rates of greater than 90%. Recently, a variety of minimally invasive procedures have been developed to avoid the morbidity of open surgery. Options include laparoscopic pyeloplasty, percutaneous endopyelotomy, and ureteroscopic or retrograde endopyelotomy with a balloon-cutting device.

II. Urolithiasis

- A. **Epidemiology.** The peak incidence of urinary calculi is in the third to fifth decade. Stones are more prevalent in men than in women. Stone incidence is increased during the late summer months. The United States, especially the Southeast, has a high incidence of stone disease. Dietary factors leading to stone formation include low water intake and high protein or oxalate (leafy green vegetable) consumption. Calcium restriction is not recommended to prevent stone formation; however, a low-sodium diet may decrease calciuria. Citrus juices, particularly lemonade, may increase urinary levels of citrate, an inhibitor of stone formation. Various drugs, including high-dose vitamin C and D, acetazolamide, triamterene, and some protease inhibitors (Indinavir) [*Lancet* 349(9061):1294, 1997], have been associated with stone formation. Disease states, such as inflammatory bowel disease, and metabolic disorders, such as type I renal tubular acidosis or cystinuria, also contribute to stone formation.
- B. **Clinical features.** Acute onset of severe flank pain or renal colic, often associated with nausea and vomiting, results from urinary obstruction by the stone. Common locations for stones to become impacted include renal infundibulum, the ureteropelvic junction, the crossing of the iliac vessels, and the ureterovesical junction, which is the most constricted area through which the stone must pass. Patients may present with microscopic or gross hematuria. Up to 15% of patients may have no hematuria.
- C. **Types of calculi**
 1. **Calcium stones** make up approximately 70% of all stones. Disorders of calcium metabolism, such as increased intestinal absorption or increased renal excretion of calcium or oxalate, can cause calcium stones. Systemic disorders, such as hyperparathyroidism, sarcoidosis, immobilization (causing calcium resorption from bone), or type I renal tubular acidosis, can lead to these derangements of calcium metabolism.
 2. **Uric acid stones** make up approximately 10% of all stones. They occur as a result of hyperuricosuria, persistently acidic pH, and low urine volumes.
 3. **Cysteine stones** account for 4% of stones. They are caused by a defect in tubular reabsorption of cysteine that is inherited in an autosomal recessive manner. Hexagonal crystals in the urine are highly suggestive of cysteine stones.
 4. **Magnesium ammonium phosphate or struvite stones** account for 15% of stones and are associated with urinary tract infection, commonly with urea-splitting organisms, and a chronically alkaline urinary pH (>7.2). Urinalysis may demonstrate rectangular “coffin lid” crystals.
- D. **Evaluation of urinary calculi**
 1. **Urinalysis**, including microscopic examination and urine culture, should be performed on all patients suspected of having calculi.
 2. **Serum electrolytes**, including calcium, with creatinine levels also are part of the standard workup. Additionally, uric acid levels and parathyroid hormone levels may be helpful. A CBC with differential can be obtained in patients with signs of concurrent infection.
 3. **KUB (kidneys, ureters, bladder)** should be the initial radiographic study. Calcium phosphate and calcium oxalate stones are the most radiopaque, whereas uric acid stones are radiolucent.
 4. **Noncontrast spiral CT** has replaced IVP as the diagnostic study of choice in the acute setting (*J Urol* 160:679, 1998). Spiral CT is quick and easy to obtain, and it does not require the use of contrast. For patients with suspected nephrolithiasis but atypical symptoms, CT may elucidate other causes of abdominal pain. Signs of obstruction include hydroureteronephrosis and perinephric fat stranding. Contrast may be given to delineate the course of the ureter, to aid in stone localization, and to evaluate the degree of obstruction.
- E. **Management of urinary calculi**
 1. **Hydration.** Intravenous fluids are required if the patient is nauseated and cannot take oral fluids. Normal saline usually is initiated at 150 mL per hour in appropriate patients.
 2. **Pain management.** Patients whose pain is not adequately managed with oral analgesics require hospitalization for administration of parenteral narcotics. Parenteral nonsteroidal compounds, such as ketorolac, can be effective in reducing the pain of renal colic but should not be used in patients who may undergo lithotripsy. Shock-wave lithotripsy is contraindicated within 72 hours of administration of any nonsteroidal analgesics to minimize the risk of renal hematoma.
 3. **Urine should be collected and strained** to retrieve the stone. The stone should be analyzed for composition.
 4. **Any patient found to have an obstructing stone in the presence of infection or fever needs emergent decompression** with percutaneous nephrostomy tube or stent placement. This situation can deteriorate quickly into a life-threatening crisis, particularly in the diabetic or immunosuppressed patient.
 5. **Ninety-five percent of stones 4 mm or smaller in size pass spontaneously** (*J Urol* 158:1915, 1997). Patients may be given up to 4 weeks to pass a partially obstructing stone. Patients with stones larger than 4 mm or with intractable symptoms of pain, nausea, or vomiting may need early surgical treatment to relieve obstruction with a ureteral stent, shock-wave lithotripsy, or ureteroscopy with stone ablation or retrieval.

Diseases of the Urinary Bladder

I. **Bladder cancer.** Bladder cancer is found in up to 10% of patients with microscopic hematuria.

- A. **Transitional cell carcinoma** accounts for up to 90% of bladder tumors in the United States, whereas squamous cell carcinoma and adenocarcinoma are less common. Transitional cell carcinoma has been linked directly to cigarette smoking, aniline dyes, aromatic amines, and chronic phenacetin use. Patients who have received cyclophosphamide are at increased risk for developing transitional cell carcinoma; use of the uroprotectant mesna may reduce this risk as well as reducing the risk of hemorrhagic cystitis.
- B. **Transitional cell cancer is categorized as superficial or invasive.** Staging is outlined in [Table 41-2](#).

Table 41-2. American Joint Committee on Cancer staging for bladder cancer

1. **Superficial tumors** are exophytic papillary lesions that do not invade the muscular bladder wall. These tumors can be treated with transurethral resection. Sixty-five percent to 85% of superficial tumors recur; therefore, diligent follow-up is necessary. Recurrent tumors are treated with transurethral resection and intravesical therapy (bacillus Calmette-Guérin, mitomycin C, or thiotepa). Ten percent to 15% of patients with superficial bladder tumors progress to muscle-invasive disease; this is dependent on stage and grade.
2. **Muscle-invasive transitional cell carcinoma** is treated with radical cystectomy and urinary diversion. Appropriate metastatic evaluation for patients with invasive bladder cancer includes chest radiograph, IVP, CT scan of the abdomen and pelvis, bone scan, and liver function tests. Bladder-sparing protocols combining external-beam radiation therapy and chemotherapy can be used for patients who desire to keep their native bladder or for those who are not operative candidates. Despite this aggressive management, only 50% of patients with invasive bladder cancer are rendered completely free of tumor because many have occult metastases at the time of surgery.
3. **Chemotherapy** [methotrexate, vinblastine, doxorubicin (Adriamycin), and cis-platinum (MVAC)] and radiotherapy are used for the treatment of advanced or residual disease. Gemcitabine is a new agent that appears to be effective against transitional cell carcinoma with less renal toxicity than platinum-based chemotherapy (*J Clin Oncol* 17:3068, 2000).

II. Urinary incontinence

- A. Urinary incontinence is usually classified into three types: **stress incontinence, urge incontinence, and mixed incontinence**. Stress incontinence is incontinence associated with activity that causes increased abdominal pressure, such as coughing, sneezing, standing, and exercising. Urge incontinence is associated with a sudden, uncontrollable urge to urinate leading to incontinence. Overflow incontinence is a special category that is associated with urinary obstruction or detrusor hypotonicity leading to urinary spillage from a full bladder.
- B. **Evaluation** of incontinence begins with a thorough history and a focused physical examination. The goal of the history is to try to classify the patient's symptoms into one of the three categories. Important points include timing and onset of incontinence, obstetric history, voiding symptoms, associated factors and general medical history including neurologic diseases, systemic illnesses such as diabetes, and a history of pelvic irradiation or surgery.
- C. **Physical examination** for incontinence should focus on neurologic and genitourinary examinations. For women, a full pelvic examination should be performed. A speculum can be used to examine for pelvic organ prolapse. The patient should cough or strain to try to reproduce stress incontinence. A Q-tip test can be used to check for urethral hypermobility; a lubricated Q-tip is placed in the urethra, and if the angle of deflection is greater than 30 degrees with straining, the test is considered positive for urethral hypermobility. Perineal sensation should be evaluated, and the bulbocavernosus reflex should be checked to test for the integrity of S2, S3, S4 spinal cord segments. A postvoid residual urine can be checked to rule out urinary retention.
- D. **Laboratory testing** should include urinalysis and urine culture to rule out infection. If indicated by symptoms or in cases with hematuria, cystoscopy or upper tract imaging may be necessary. Formal urodynamic testing can be performed if the patient's history and examination do not clearly demonstrate the etiology of the incontinence or if surgical intervention is planned.
- E. **Correctable causes** of incontinence, such as urinary tract infection, should be treated. Recent guidelines recommend initial behavioral and pharmacologic interventions before considering surgery to treat incontinence. Conservative measures help 50–65% of patients. These nonoperative interventions include behavioral modifications such as timed voiding, fluid restriction, and dietary modification. Biofeedback techniques, such as Kegel exercises and vaginal weighted cones, are designed to strengthen the pelvic floor musculature that provides urethral support involved in continence. Electrical stimulation and sacral nerve root stimulation attempt to alter bladder and urethral function by stimulation of pudendal and sacral nerves.
- F. A variety of **surgical procedures** are available for the correction of urinary incontinence. Indications and applications are beyond the scope of this chapter.

Diseases of the Prostate

I. Prostate cancer

- A. **Prostate examination. Digital rectal examination (DRE)** of the prostate is an important part of the physical examination.
1. The **normal prostate** is chestnut sized and measures 3.5 cm wide at the base, 2.5 cm long, and 2.5 cm deep; it weighs approximately 20 g. The prostate should feel smooth, having the consistency of the contracted thenar eminence of the thumb. The prostate is best examined with the patient standing with the knees slightly flexed and elbows resting on a table, or in the lateral decubitus position with the hips flexed.
 2. **Prostate nodules** usually are small (pea-sized) or larger firm areas within the peripheral zone of the prostate. They can represent prostate cancer and must be evaluated with transrectal ultrasonography (TRUS) and prostate biopsy. In men older than 50 years, 15% have a suspicious rectal examination, and 21% of those men have cancer. Serum prostate-specific antigen (PSA) is a more sensitive test than DRE for detection of prostate cancer, but the tests should be used together to maximize cancer detection (*J Urol* 151:1283, 1994).
- B. **Prostate cancer** is the most common noncutaneous malignancy in American men and the second leading cause of cancer death. Twenty percent of men with prostate cancer die of the disease. Prostate cancer rarely causes symptoms until it becomes locally advanced or metastatic and is no longer curable.
- C. **Current American Urological Association and American Cancer Society Guidelines** recommend that men age 50 and older begin prostate cancer screening with a yearly DRE and PSA measurement. African-American men and men with a family history should begin screening at age 45 (*Oncology* 14:267, 2000). Abnormalities in either the DRE (manifest as indurated nodules) or the PSA (>4.0 ng/mL) should be evaluated by TRUS and needle biopsy of the prostate.
- D. **New assays for measuring the percentage of free PSA** have the potential to categorize men with intermediate levels of total PSA (4–10 ng/mL) into low and high cancer risk groups. Prostate cancer is more likely to be found on biopsy in men with a low percentage of free PSA (<25%). This could result in a reduction of unnecessary prostate biopsies in men with a high percentage of free PSA (*JAMA* 279:1542, 1998).
- E. **Appropriate staging workup includes DRE and PSA.** [Table 41-3](#) outlines current staging for prostate cancer. Prostatic acid phosphatase may be used as a marker for metastatic disease. Bone scan is not necessary for patients with well- or moderately differentiated tumors and a PSA less than 10. CT is of limited value for patients with well- or moderately differentiated tumors and a PSA less than 20.

Table 41-3. American Joint Committee on Cancer staging for prostate cancer

- F. **Treatment options** for men with organ-confined prostate cancer include radical prostatectomy, external-beam radiation therapy, and interstitial radiotherapy (brachytherapy).
1. For **radical prostatectomy**, the overall 5-year freedom from PSA progression is 61–87% in several published series (*Urol Clin North Am* 20:713, 1993). Overall, 10-year disease-specific survival following radical prostatectomy for clinically localized prostate cancer is 85% (81–87%, 95% confidence interval)

(*JAMA* 276: 615, 1996).

2. **External-beam radiotherapy** achieves disease-free rates of 45–85% for localized disease (*N Engl J Med* 331:996, 1994).
3. **Technical improvements in dosimetry and implantation** combined with reports of low morbidity have led to a renewed interest in brachytherapy. Some authors have suggested that brachytherapy alone may have a higher rate of PSA progression than other treatment modalities (*Urology* 51:884, 1998). The optimal form of treatment for clinically localized prostate cancer has not been conclusively determined.
4. **Hormonal therapy** with either bilateral orchiectomy or luteinizing hormone–releasing hormone agonists usually is reserved for men with locally advanced or metastatic disease.
5. Watchful waiting is appropriate for men with a life expectancy of fewer than 10 years and low-stage, low-grade prostate cancer (*J Urol* 159:1431, 1998).

II. Prostatitis is a diagnosis that spans a spectrum of disease entities. The classification and diagnostic criteria for the different forms of prostatitis recently have been changed in an effort to standardize diagnosis to improve research and clinical treatment.

- A. **Signs and symptoms of urinary tract infection** mark acute bacterial prostatitis; many patients have significant voiding complaints, fevers, and malaise. Antibiotics are the mainstays of treatment. Those patients with high fevers may require admission for intravenous antibiotics. Drainage of the urinary bladder via a suprapubic tube or a small urethral catheter may be required for those patients in urinary retention.
- B. **Chronic bacterial prostatitis** is differentiated from other categories by the presence of documented recurrent bacterial infection of expressed prostatic secretions, postprostatic massage urine, or semen. Treatment is with antibiotics; fluoroquinolones have excellent prostatic penetration.
- C. Historically, **patients with chronic pelvic pain**, but without culture-proven infection of the lower urinary tract, were given the diagnosis of prostatitis, along with patients from the first two categories. However, in the new classification scheme, these patients are considered to have chronic pelvic pain syndrome. These patients are further stratified based on the presence of WBCs in postprostatic massage urinalysis. Treatments for this group are less well defined, and the optimal treatment modality may not be the same for all patients. Alpha-blockers, antibiotics, nonsteroidal antiinflammatory drugs, finasteride, biofeedback, and muscle relaxants are all therapies that have been used alone or in varying combinations for this difficult-to-treat entity (*Infect Urol* 13:22S, 2000).

III. Prostatic abscess can be difficult to diagnose.

- A. **Patients typically have acute urinary retention, fever, dysuria, urinary frequency, and perineal pain.**
- B. **Prostatic enlargement** is the most common finding on examination; other findings, such as tenderness and fluctuance, are more variable (*Rev Infect Dis* 10:239, 1988).
- C. **CT scan or TRUS** confirms the diagnosis.
- D. **Treatment** is with percutaneous or transurethral drainage and appropriate parenteral antibiotics.

IV. Urinary retention may result from BPH, prostate cancer, or urethral stricture disease. Retention also can be associated with pelvic trauma, neurologic conditions, or various medications or the postoperative setting.

- A. **Evaluation.** A history usually elicits the cause of retention. Common signs and symptoms of BPH include hesitancy, decreased force of stream, frequency, urgency, postvoid dribbling, double voiding, incomplete bladder emptying, and nocturia. Patients with BPH who are treated with decongestants containing an alpha-agonist may develop urinary retention from increased smooth-muscle tone at the bladder neck and the prostate. Anticholinergic medications can cause urinary retention through relaxation of the detrusor muscle.
- B. **Physical examination** reveals a distended lower abdomen. Prostatic enlargement is common on DRE. Serum electrolytes and creatinine levels, urinalysis, and urine culture should be obtained. Serum PSA concentration obtained during acute urinary retention often is spuriously elevated and is best measured at least 4–6 weeks after the acute event.
- C. **Treatment**
 1. **Acute management of retention is catheter placement.** Sterile technique is used. The proper technique of urethral catheter placement involves passing the catheter to the hub and inflating the balloon *only* after the return of urine.
 2. **When a standard Foley catheter cannot be passed easily**, sterile 2% viscous lidocaine can be injected through the urethra. This anesthetizes and relaxes the sphincter, allowing gentle passage of a 16–22 Fr. Coudé tip catheter. The viscous lidocaine is given 5–10 minutes before passing the catheter. The catheter is passed gently with the tip directed toward the patient's head. If the Coudé tip catheter does not pass easily, a urology consultation is required.
 3. **Catheterization should not be attempted when a urethral injury is suspected.** Urethral stricture requires calibration and dilation or placement of a suprapubic tube by a urologist. Urinary clot retention usually requires bladder irrigation.
- D. **Patients should be monitored for postobstructive diuresis.** This is a physiologic response to a hypervolemic state. Occasionally, it can become a pathologic diuresis and may warrant hospital observation with fluid and electrolyte replacement. 5% dextrose in 0.45% saline should be used for hydration. Urine output greater than 200 mL per hour for more than 2 hours should be replaced with 0.5 mL intravenous 0.45% saline for each 1 mL urine. Electrolytes should be checked every 6 hours initially and replaced as needed.
- E. **BPH** is most commonly treated medically with alpha-blockers, such as doxazosin, terazosin, or tamsulosin. Another class of medications, 5-alpha-reductase inhibitors, such as finasteride, may help improve voiding symptoms.

Diseases of the Penis

I. Priapism is a persistent erection not associated with sexual stimulation or detumescence that does not occur after orgasm. The corpora cavernosa are affected, but the corpus spongiosum usually is spared. Priapism can be classified as low flow or high flow.

- A. **Low-flow priapism** is an ischemic state in the corpora, secondary to prolonged erection and resultant edema of the cavernosal trabeculae. Symptoms include pain and tenderness. Stasis, thrombosis, fibrosis, and scarring of the corpora cavernosa eventually can result in erectile dysfunction (ED). The diagnosis is made by penile corporal aspiration, which demonstrates dark, crankcase-like oil blood with an acidic pH. Ischemia and acidosis appear after approximately 6 hours.
 1. **Priapism** may occur in association with sickle cell anemia, with a 6% incidence among sickle cell patients. Patients may have a history of stuttering priapism lasting 2–6 hours, usually at night. Initial treatment should include aggressive hydration with intravenous fluid supplemented with 1 ampule of NaHCO₃/L and oxygen to prevent further sickling. If these measures are unsuccessful, corporal aspiration injection as described below may be necessary.
 2. **Iatrogenic priapism** can occur secondary to intracavernous injection of vasoactive substances (prostaglandin E₁, phentolamine, or papaverine) used to treat ED. Oral alpha-agonists, such as terbutaline or pseudoephedrine, may be effective up to one-third of the time in iatrogenic priapism. If oral treatment fails, aspiration and irrigation of the corpora (see below) should be performed. Psychotropic agents, antihypertensive agents (hydralazine, guanethidine, and prazosin), and alcohol account for 20% of priapism. The antidepressant trazodone has been shown to induce priapism (*Med Lett Drugs Ther* 26:35, 1984).
 3. **Neoplasm (especially leukemia)**, with venous occlusion, stasis, and emboli, can result in priapism. Treatment is with chemotherapy and radiotherapy.
4. **Treatment**
 - a. **First-line treatment** involves aspiration of old blood from the corpora via a 21-gauge needle.
 - b. An **alpha-adrenergic agent** (phenylephrine, 250–500 µg) can then be injected. The solution is prepared by mixing 1 mL phenylephrine, 10 mg/mL in 19 mL sterile normal saline. Each mL contains 500 µg phenylephrine. Doses can be repeated every 5–10 minutes. Patients should be monitored for the possible hemodynamic effects of phenylephrine. Topical or subcutaneous injection of lidocaine before therapeutic injection or irrigation can be helpful for patient comfort. Injections and aspiration should be performed laterally at the 3 o'clock and 9 o'clock positions to avoid the dorsal blood supply of the penis.
 - c. If evacuation of old blood and injection of alpha-adrenergic agents fails, **surgical shunting** should be considered.
 1. The **Winter shunt** involves creation of a fistula between the glans penis (corpus spongiosum) and corpus cavernosum. This is done with a core (Tru-Cut) needle after a penile block.
 2. The **Al-Ghorab procedure** is a more aggressive open surgical modification of the Winter shunt. It involves a glandular incision, exposing the tips of the corpora; a 5-mm ellipse of the tunica albuginea is removed to create a cavernospongiosal shunt.
 3. Finally, the more proximal **side-to-side cavernospongiosal shunt (Quackels shunt)** or the **cavernosaphenous shunt** may be necessary when distal shunting procedures fail. Circumferential compressive dressings should never be used after shunting because they can obstruct venous drainage, resulting in further tissue ischemia.
- B. **High-flow priapism** is a nonischemic state usually brought about by perineal or genital trauma. A traumatic pudendal arterial fistula or cavernosal artery laceration may give rise to a high-flow state. Diagnosis is confirmed by aspiration of bright-red, well-oxygenated blood. Blood gas analysis can be helpful in

differentiating low-flow priapism from high-flow priapism. Treatment is accomplished by embolization of the ipsilateral branch of the pudendal artery.

II. ED recently has received increasing attention from the public and lay media as a result of new treatment modalities. Minimal, moderate, or complete ED may affect up to 50% of men aged 40–70 years; incidence increases with age.

- A. **Initial evaluation** includes complete history and physical examination, with a focus on eliciting possible underlying causes of ED, including heart disease, hypertension, diabetes, renal insufficiency, and endocrine abnormalities (hypogonadism, hyperprolactinemia). Smoking alone or in combination with any of these risk factors can increase the incidence of ED.
1. **Attention should be paid to medications**, such as antihypertensives [central-acting agents (clonidine), alpha-adrenergic blocking agents (prazosin), beta-blocking agents], antipsychotics, tricyclic antidepressants, and histamine H₂ blockers (cimetidine), which may be associated with ED. Cigarette smoking and heavy use of alcohol can also lead to ED. Previous pelvic or penile surgery may be associated with ED. The timing of onset of ED (sudden vs. gradual) should be noted, as should the presence of nocturnal or morning erections. Loss of libido may signal hormonal disturbances.
 2. **Physical examination** should focus on genital development and signs of endocrinologic or neurologic abnormalities.
 3. **Appropriate laboratory testing** includes serum chemistries, including creatinine, CBC count, urinalysis, and, when indicated, a limited hormonal evaluation (testosterone and prolactin).
- B. **For the majority of patients**, ED is multifactorial, and no single cause is identified. In these cases, it is appropriate to counsel patients about available nonsurgical options for treatment and to allow them a trial of one or more of these methods until a satisfactory solution is found. Available medical options include the following:
1. **Hormone replacement.** Exogenous testosterone is available in a variety of delivery methods, including parenteral preparations and transdermal therapy. Liver function tests should be monitored. This treatment is best suited for patients with a low libido and documented hypogonadism. The effect of testosterone on erectile function is variable.
 2. **Oral therapies.** Sildenafil citrate (Viagra) is a selective type 5-phosphodiesterase inhibitor that inhibits the breakdown of cyclic guanosine monophosphate, allowing smooth-muscle relaxation in the corpus cavernosum. Typical doses are 50–100 mg taken 1 hour before sexual activity. Sildenafil is effective in 60% of men, regardless of etiology. Side effects include headache, facial flushing, and dyspepsia. Sildenafil citrate is contraindicated in patients who are taking nitrates because of a synergistic effect, which results in excessive reduction in blood pressure. Other, older oral therapies, such as yohimbine, have not been consistently effective in treating ED.
 3. **Intracavernosal therapy.** Injection of vasoactive medications, such as alprostadil (prostaglandin E₁), directly into the corpus cavernosum is effective in 70–80% of patients. Side effects are pain with injection, hematoma or ecchymosis, and priapism. An intraurethral alprostadil suppository is also available and is effective in some men.
 4. **Medical devices.** For men who do not desire or who are not candidates for medical therapy, there are several devices that may aid in getting and maintaining an erection. A vacuum pump is efficacious, but many couples find it cumbersome and uncomfortable. Patients with difficulty in maintaining an erection due to cavernosal venous insufficiency may benefit from a constriction band.
 5. **Surgical options.** For patients who are refractory to noninvasive therapy, consideration may be given to a surgically placed penile implant. These devices have a high degree of success but have the disadvantage of being irreversible, and there are potential complications, such as infection (2%) and mechanical malfunction (2%).

Diseases of the Scrotum and Testicles

I. **Management of scrotal emergencies.** Acute scrotal pathology can result in significant morbidity, testicular loss, and infertility. The diagnosis can be difficult to make and may require scrotal exploration.

- A. **Testicular torsion** develops most often in the peripubertal (12–18 years old) age group, although it can occur at any age.
1. The **clinical picture** is one of acute onset of testicular pain and swelling, commonly associated with nausea and vomiting. Some patients give a history of a prior episode that spontaneously resolved (intermittent torsion). There usually is no history of voiding complaints or dysuria, fever, or exposure to sexually transmitted diseases.
 2. **Physical examination** reveals an extremely tender, swollen testicle high in the scrotum with a transverse lie. The cremasteric reflex (elicited by stroking the inner thigh) is absent on the affected side. In contrast to epididymitis, elevation of the scrotum does not provide relief of pain (Prehn's sign) in torsion. Normal urinalysis and the absence of leukocytosis help to rule out epididymitis.
 3. When the **clinical diagnosis** is equivocal, radiographic studies may be helpful. Depending on availability, nuclear scintigraphy and color Doppler ultrasound have similar sensitivity in evaluating testicular torsion (*Radiol Clin North Am* 35:959, 1997).
 4. **Treatment** should not be delayed to obtain imaging. If testicular torsion is suspected, urgent scrotal exploration is indicated. Manual detorsion of the testicle may be attempted in the emergency room, but orchiopexy is still indicated. Testicular viability is a function of the reestablishment of perfusion ([Table 41-4](#)). Contralateral testicular fixation should be performed at the time of surgery.

Table 41-4. Rate of salvage in testicular torsion

- B. **Torsion of testicular appendage (appendix testis)** can present with symptoms similar to those of torsion of the testicle, usually in a prepubertal boy. The onset commonly is over 12–24 hours.
1. **Extreme tenderness over the appendage** exists, usually on the superior aspect of the testicle. The “blue dot” sign may be present when the ischemic appendage can be seen through the scrotal skin. The testicle has a normal position and lie. Careful examination reveals that the testicle and the epididymis are not diffusely tender or swollen. The cremasteric reflex usually is present. Imaging studies may be necessary if the clinical diagnosis is unclear (see above).
 2. **Torsion of the testicular appendage** usually is managed expectantly. Pain is best controlled with antiinflammatory agents and gradually resolves over 7–14 days.
- C. **Mild epididymitis** usually presents with a 1- to 2-day onset of unilateral testicular pain and swelling associated with dysuria or urethral discharge.
1. Typically, the **findings** include a painful, indurated epididymis and pyuria. Urinalysis, urine culture, and CBC count are obtained. When clinically indicated, urethral swabs for gonococci and chlamydia are sent for culture.
 2. With **appropriate antibiotic coverage**, these patients can be managed as outpatients. For patients in whom the etiology is gonococcal or chlamydial, ceftriaxone (125–250 mg i.m.) is given in the emergency room, followed by doxycycline (100 mg p.o. b.i.d. for 7 days). Azithromycin, 1 g p.o. as a one-time dose, is as effective as doxycycline in the treatment of chlamydial infections [*MMWR Morb Mortal Wkly Rep* 47(RR-1):51, 1998]. In older men (>35 years of age), enterobacteria are more common, and a fluoroquinolone, such as ciprofloxacin (500 mg p.o. b.i.d.), provides broad coverage until culture sensitivities can be obtained. Nonsteroidal analgesics and scrotal elevation can reduce inflammation and provide symptomatic relief. Moderate to severe cases of epididymitis may require hospital admission. Symptoms usually have been present for several days. Fever and leukocytosis are present. Broad-spectrum antibiotics and supportive measures of bedrest with scrotal elevation should be instituted. Ultrasonography can be useful to rule out abscess formation and assess testicular perfusion.
- D. **Fournier's gangrene** is a severe polymicrobial soft-tissue infection involving the genitals and perineum. Although the term *Fournier's gangrene* usually is applied to men, necrotizing fasciitis of this area can occur in women. Prompt diagnosis and institution of treatment may be lifesaving. Roughly 25% of the patients have a genitourinary source, 25% have an anorectal source, up to 10% have an intraabdominal source, and nearly 40% have an unidentified source. Diabetic, alcoholic, and other immunocompromised patients appear more susceptible. The clinical course is one of abrupt onset with pruritus, rapidly progressing to edema, erythema, and necrosis, often within a few hours. Fever, chills, and malaise are accompanying signs.
1. **Physical examination** reveals edema and erythema of the skin of the scrotum, phallus, and perineal area. This may progress rapidly to frank necrosis of the skin and subcutaneous tissues, with extension to the skin of the abdomen and back, reaching as high as the clavicles and down the thighs. Crepitus in the tissues suggests the presence of gas-forming organisms.
 2. **Laboratory evaluation** should include a CBC count, serum electrolytes, creatinine, arterial blood gas, coagulation parameters, urinalysis, urine, and blood cultures. A KUB plain film may reveal subcutaneous gas.
 3. **The patient should be stabilized and prepared emergently for the operating room.** Broad-spectrum antibiotics that are active against both aerobes

(including *Staphylococcus aureus*, beta-hemolytic *Streptococcus*, *Pseudomonas* species, *Escherichia coli*, and *Klebsiella*) and anaerobes (including *Bacteroides* and *Clostridium* species) should be started immediately. Aerobic and anaerobic wound cultures are usually polymicrobial.

4. **Wide débridement** is required with aggressive postoperative support. The testicles are often spared, as they have a blood supply discrete from the scrotum; orchiectomy is rarely indicated. Wound closure and dermal coverage often is an extensive process, and recovery requires intense physical therapy and wound care.
5. Despite improvements in critical care, antibiotics, and surgical technology, **mortality** still exceeds 30% in some recent series (*Urology* 47:935, 1996).

II. Nonacute scrotal masses

- A. **Hydroceles** generally are asymptomatic fluid collections around the testicle that transilluminate. Ultrasound evaluation is recommended to rule out serious underlying causes such as testicular malignancies. If hydroceles do enlarge and become symptomatic, they can be repaired by a variety of transscrotal techniques. Hydroceles in infants may be associated with a patent processus vaginalis; parents give a history of intermittent scrotal swelling. These hydroceles usually resolve by 1 year of age. Those that persist can be repaired by an inguinal approach.
- B. **Spermatoceles** are benign cystic dilatations involving the tail of the epididymis or proximal vas deferens.
- C. **Varicoceles** are an abnormal tortuosity and dilation of the testicular veins within the spermatic cord. On physical examination they feel like a “bag of worms.” The varicocele may diminish in size when the patient is supine. Because the left gonadal vein drains directly into the renal vein, varicoceles are much more common on the left side. Right-sided varicoceles may be associated with obstruction of the inferior vena cava. Varicocele is the most common surgically correctable cause of male infertility; however, most men with varicoceles remain fertile. Varicocele repair results in improved semen quality in approximately 70% of patients. Treatment of varicoceles is indicated for diminished testicular growth in adolescents, infertility, or significant symptoms. Varicoceles may be treated by spermatic vein embolization or spermatic vein ligation via a laparoscopic, open, or microvascular approach.

III. Testicular tumors are the most common solid tumors in 15- to 35-year-old men. The estimated lifetime risk for testicular malignancy is 1 in 500. Due to improved multimodality therapy, overall 5-year survival for testis cancer is now 95%.

- A. **The typical clinical finding is a painless testicular mass**, although one-third of patients may present with pain. Pulmonary or gastrointestinal complaints or the presence of an abdominal mass may reflect advanced disease. Scrotal sonography is mandatory; seminomas appear as a hypoechoic lesion, and nonseminomatous tumors appear inhomogeneous. Alpha-fetoprotein, beta-human chorionic gonadotropin, and lactic acid dehydrogenase are serum tumor markers that help to identify the tumor type and to completely stage the tumor. The markers are used to monitor the effectiveness of therapy and to screen for recurrence.
- B. **Staging of testicular tumors** is outlined in [Table 41-5](#). Serum tumor markers have recently been added to the staging system.

Table 41-5. American Joint Committee on Cancer staging of testicular cancer

- C. **Initial therapy** for all testicular tumors is radical inguinal orchiectomy. The type of tumor and the stage of the disease determine further therapy.
- D. **Seminomas** constitute 60–65% of germ cell tumors. Low-stage seminomas are treated with adjuvant radiation therapy to the retroperitoneum. Advanced disease is usually treated with a platinum-based chemotherapy regimen.
- E. **Nonseminomatous tumors** include the histologic types of embryonal carcinoma, teratoma, choriocarcinoma, and yolk sac elements, alone or in combination. Nonseminomatous tumors are more likely to present with advanced disease. Patients with clinically negative retroperitoneal nodes with normal tumor markers are treated with retroperitoneal lymph node dissection, prophylactic chemotherapy, or close observation. Patients with high-stage disease with elevated markers receive platinum-based chemotherapy followed by retroperitoneal node dissection if there is residual disease.

Genitourinary Trauma

Genitourinary injuries should be identified during the secondary survey after life-threatening injuries have been addressed and initial resuscitation has been undertaken.

I. Renal injury is a component of approximately 10% of abdominal traumas. Blunt trauma accounts for 80–90% of renal injuries. Penetrating trauma occurs as a result of gunshot wounds or stab wounds and accounts for 10–20% of renal injuries. The grading system for renal injuries is shown in [Table 41-6](#).

Table 41-6. Renal injury scale of the American Association for the Surgery of Trauma

- A. **Blunt renal trauma** should be suspected in patients with abdominal tenderness, lower rib fractures, vertebral fractures, or flank contusions.
 1. **Microscopic hematuria** (>3 RBC/high-power field) or gross hematuria is present in more than 95% of patients with a renal injury. A voided specimen is best for urinalysis, but if the patient cannot void or is unconscious and no blood is at the meatus, a well-lubricated urethral catheter should gently be passed.
 2. All patients with **gross hematuria and blunt trauma** should be evaluated with a CT scan using intravenous contrast. Patients with microscopic hematuria and shock (systolic blood pressure <90 mm Hg) should be imaged with a CT scan after they stabilize. Patients with microscopic hematuria, no shock, and no evidence of significant deceleration or renal injury do not need radiographic evaluation of their urinary system (*J Urol* 141:1095, 1989).
 3. It is important to understand that the **degree of hematuria** does not correlate with the severity of the injury (*J Urol* 120:455, 1978) and that any patient with a suspected renal injury due to rapid deceleration requires radiographic evaluation. Disruption of the ureteropelvic junction should be considered in children with deceleration or hyperextension injuries.
 4. The majority of **blunt renal injuries** can be managed conservatively; fewer than 10% of blunt renal injuries require surgery (*World J Urol* 17:71, 1999).
- B. **Penetrating renal trauma** with microscopic hematuria (>3 RBC/high-power field) or gross hematuria requires radiographic assessment with CT scan or an IVP. Preferably, this is done before exploration to evaluate the injured kidney and to confirm function of the contralateral kidney. The presence of a normal contralateral kidney may influence the surgeon's decision (repair vs. nephrectomy) on management of the injured kidney.
 1. A **high-dose, single-shot IVP** with a 2 mL/kg bolus injection of contrast followed by a single film at 10 minutes can be performed in the trauma suite or in the operating room without interfering with other critical elements of the trauma evaluation and resuscitation.
 2. **Indications for intraoperative renal exploration** include an expanding, pulsatile, or unconfined retroperitoneal hematoma; all grade V renal injuries; and renal injuries that have not been completely staged.

II. Ureteral injuries account for approximately 3% of all urologic traumas. A high index of suspicion often is necessary to make the diagnosis, and many have a delayed presentation.

- A. The most common scenario is **penetrating trauma with multiple associated injuries**. The absence of gross or microscopic hematuria has been documented in 30% of patients.
- B. **Radiographic findings** include extravasation, or more commonly delayed function; proximal dilation; or deviation of the ureter. A CT may demonstrate medial extravasation; delayed images are necessary to assess ureteral patency. Retrograde pyelography is the most sensitive diagnostic tool but may be difficult to obtain in the setting of acute trauma.
- C. **Adequately visualizing the ureter during laparotomy** is important in diagnosing ureteral injury; intravenous or intraureteral injection of indigo carmine or methylene blue may help to assess the integrity of the urothelium.

- D. For purposes of determining the **type of repair**, the ureter is divided into thirds.
1. Injuries to the distal one-third of the ureter are best managed by **ureteral reimplantation**. Additional length to provide a tension-free anastomosis may be gained by using a Psoas hitch and, if necessary, a Boari bladder flap.
 2. Injuries of the middle or upper third of the ureter are best managed by **ureteroureterostomy**. An omental wrap may be used to protect the repair. Stents and drains are recommended for all ureteral repairs (*World J Urol* 17:78, 1999).

III. Bladder injuries result from blunt trauma, penetrating trauma, and iatrogenic injury during surgical procedures.

- A. Ninety-five percent of bladder injuries present with **gross hematuria**. Anyone with a history of blunt or penetrating trauma, gross hematuria, and difficulty in urinating should be evaluated with a cystogram.
1. A **Foley catheter** is placed if a urethral injury is not suspected (see below). A scout film is obtained. Standard radiographic contrast is diluted to a 50:50 mix with saline and is infused under gravity. When 100 mL has been instilled, an anteroposterior radiograph is taken. If no extravasation is seen, the bladder is filled to 350 mL under gravity, and an anteroposterior and oblique radiograph is obtained. A postdrainage film is mandatory.
 2. **Upper tract imaging** can then be done. If a CT scan is obtained, a CT cystogram may be substituted for a plain radiographic or fluoroscopic cystogram. The bladder should be filled retrograde by gravity via an indwelling Foley catheter with 350 mL dilute (3–5%) contrast. Postdrainage films are not necessary; merely clamping the Foley to allow bladder filling with excreted contrast does not constitute an adequate study. An IVP or CT scan alone is not adequate to evaluate bladder trauma.
- B. **Management**
1. All patients with penetrating trauma to the bladder and intraperitoneal extravasation of contrast require **surgical exploration and repair** of the bladder (*World J Urol* 17:84, 1999).
 2. Often, patients with blunt trauma and extraperitoneal extravasation of contrast can be managed nonoperatively with **catheter drainage** for 10 days.

IV. Genital and urethral injuries

- A. **Urethral injuries** occur in 5% of patients with pelvic fractures. Posterior urethral injuries involve the prostatic and membranous urethra to the level of the urogenital diaphragm. These injuries are caused mainly by blunt trauma.
- B. **Urethral injury** should be suspected when blood is at the meatus or the mechanism of injury is such that urethral injury might have occurred. Physical examination in patients with urethral injury may reveal penile and scrotal edema and ecchymosis. Rectal examination can reveal a high-riding prostate or boggy hematoma in the expected position of the prostate.
- C. A **retrograde urethrogram** is performed by placing a 14 or 16 Fr. Foley catheter into the urethra so that the balloon is 2–3 cm beyond the meatus. The balloon is inflated with 1–2 cc to seat it in the fossa navicularis. With the patient in a 30-degree oblique position, 25–30 mL half-strength contrast medium is injected through the catheter. The radiograph is exposed when the contrast is nearly completely injected. If the urethra is normal, the balloon can be deflated, the catheter advanced into the bladder, and a cystogram performed.
- D. **Anterior urethral injuries** include the bulbous and penile urethra distal to the urogenital diaphragm. Straddle injuries and penetrating trauma are the most common causes of these types of injuries. Injuries contained by Buck's fascia often have a characteristic "sleeve of penis" pattern, whereas urethral or penile injuries where Buck's fascia is disrupted are contained by Colles' fascia and have a "butterfly" appearance on the perineum.
- E. **Complete disruptions** of the posterior urethra can be managed by primary endoscopic realignment or by suprapubic diversion and delayed repair. **Partial disruptions** may be managed with catheter drainage for 14–21 days (*World J Urol* 16: 69, 1998). These injuries are often associated with formation of urethral strictures and with impotence.
- F. **Minor penile lacerations and contusions** can be managed in the emergency room. **Serious blunt or penetrating trauma** with injury to the corpus cavernosum is best managed with surgical exploration, débridement, and repair of the corporal injury. A retrograde urethrogram is necessary to rule out urethral injury. Broad-spectrum antibiotics should be given, particularly in human bite injuries.
- G. **Testicular injury** may occur as a result of blunt or penetrating trauma. History and physical examination are the keys to diagnosis of testicular rupture. The presentation is marked by acute and severe pain, often with associated nausea and vomiting. Physical examination may reveal a hematoma or ecchymosis of overlying skin. Ultrasonography has a specificity of 75% and a sensitivity of 64% in combined series. In cases in which the suspicion is high, scrotal exploration should be undertaken. The orchiectomy rate is below 10% for ruptured testicles explored within 72 hours after injury. Repair consists of hematoma evacuation, débridement of the necrotic tubules, and closure of the tunica albuginea (*World J Urol* 17:101, 1999).
- H. **Scrotal avulsion and skin loss** are most often a result of motor vehicle accidents. Because of the redundancy and vascularity of scrotal skin, a variety of options are available for local flaps and coverage of the testicles. Wounds should be copiously irrigated and débrided; clean wounds may be closed in layers, whereas grossly contaminated wounds should be cleaned and packed with sterile gauze dressings.

Obstetric and Gynecologic Disorders

I. Vaginal bleeding

II. Abdominal pain

III. Nonobstetric surgery in the pregnant patient

IV. Trauma in pregnancy

V. Gynecologic malignancies

Obstetric and Gynecologic Disorders

I. Vaginal bleeding to an abnormal degree may result from a variety of causes. A thorough history, including pattern and intensity of bleeding, and physical examination frequently are sufficient to determine the etiology. A pregnancy test must be performed in all women of reproductive age. Hemoglobin (Hgb) and hematocrit (Hct) should be determined if the abnormal bleeding is chronic or heavy. Endometrial biopsy should be performed in all postmenopausal women to rule out endometrial carcinoma or if hormonal intervention is planned. Evaluation and treatment depend on the presentation and etiology.

A. **Obstetric etiologies.** Approximately 30–40% of all pregnancies are associated with some vaginal bleeding, and approximately half of these are spontaneously aborted.

1. Terminology

- Threatened abortion:** any vaginal bleeding during the first half of pregnancy, cervix closed
- Missed abortion:** fetal death with retention of products of conception, cervix closed
- Inevitable abortion:** cervical dilatation with or without ruptured membranes
- Incomplete abortion:** partial passage of products of conception, cervix open
- Complete abortion:** expulsion of all products of conception from the uterine cavity, cervix closed

2. **Presentation and clinical features.** Patients classically present with vaginal bleeding and crampy midline lower abdominal pain. Bleeding from the urethra or rectum or from lacerations of the cervix or vagina should be excluded. Complaints of passage of tissue may represent a complete or incomplete abortion.

3. **Physical examination.** Vital signs are within normal ranges unless extensive vaginal bleeding or septic abortion occurs, with resultant tachycardia and hypotension. Septic abortions can cause elevated temperatures, marked suprapubic tenderness, or purulent discharge through the cervical os.

4. Laboratory investigation

- Hgb and Hct.** Plasma volume expansion in pregnancy may result in a lower mean Hgb during the second trimester. With acute blood loss, the Hgb and Hct can be normal until compensatory mechanisms restore normal plasma volume.
- White blood cell (WBC) count with differential** is useful to evaluate elevated temperatures. Septic abortion is associated with a left shift and elevated WBC.
- Blood type and screen** are useful to identify Rh-negative patients at risk for isoimmunization (see [section I.A.6.g](#)).
- Quantitative beta subunit of human chorionic gonadotropin (hCG).** The sensitivity of a pregnancy test (urine or serum) can vary greatly depending on the type of test performed (i.e., latex agglutination, enzyme-linked immunosorbent assays, radioimmunoassay). Most institutions use very sensitive serum pregnancy tests; however, the sensitivity of the urine pregnancy test may be more variable. A urine pregnancy test has the advantage of requiring only a few drops of urine and gives a rapid result. Unlike the serum pregnancy test, however, the urine pregnancy test is unable to be quantified, which is helpful when evaluating the status of a pregnancy. Serial quantitative (serum) hCG values may be used along with ultrasonography to distinguish an early viable pregnancy from an abnormal pregnancy. In most normal intrauterine pregnancies near 6 weeks' gestation, hCG increases by at least 66% every 48 hours (*ACOG Prac Bull* 3, December 1998). Patients with stable clinical examinations can be followed with serial hCG values until reaching the sonographic threshold values at which ultrasound visualization of an intrauterine pregnancy is possible (see [section I.A.5](#)).
- Progesterone levels** in excess of 15 ng/mL usually are associated with a normal intrauterine pregnancy. Below this range, pregnancy is likely abnormal, but location may be intrauterine or extrauterine.

5. **Imaging studies.** Ultrasonography may be useful in demonstrating a viable pregnancy. The sonographic threshold value of hCG for vaginal probe ultrasonography is generally greater than 2,000 mIU/mL, International Reference Preparation, although you should be able to see a gestational sac at 1,500 mIU/mL. For abdominal ultrasonography, the threshold is greater than 6,000 mIU/mL. Cardiac activity can be seen at 10,000 mIU/mL.

6. Treatment

- Threatened abortion.** Patients with a pregnancy that is viable or of indeterminate viability, vaginal bleeding, and a closed internal cervical os are followed expectantly with repeat ultrasound in 7 days or repeat hCG in 48 hours, or both.
- Missed abortion.** Patients may be followed expectantly or undergo evacuation. If they are followed expectantly for more than 3–4 weeks, coagulation studies [i.e., complete blood cell (CBC) count, prothrombin time, partial thromboplastin time, fibrinogen, and fibrin degradation products] should be monitored because of the risk of disseminated intravascular coagulopathy. Counsel patients to bring any tissue passed back to the hospital for pathologic verification. Generally, after 3 weeks evacuation is suggested, and the individual should be scheduled as an outpatient.
- For inevitable abortion,** the uterus usually begins to contract, resulting in expulsion of products before infection develops. Patients occasionally are followed expectantly with monitoring for infection but typically undergo uterine evacuation. If fever develops, intravenous antibiotics with polymicrobial coverage are administered, followed by evacuation of the uterus. These patients require admission and careful monitoring of coagulation factors.
- For incomplete abortion,** bleeding, cramping, and an open internal os usually are found. Uterine evacuation is indicated. If products of conception are not recovered, ectopic pregnancy should be considered.
- For complete abortion,** all products of conception have been expelled and the cervix is closed. Bleeding and cramping are minimal. Only short-term observation for stability is necessary.
- Evacuation of the uterus.** Suction curettage is done safely in the first trimester and can be performed in the emergency room if significant cervical dilatation exists. A stable patient with a first-trimester missed abortion that requires dilation of the cervix undergoes dilation and curettage (D&C) as an outpatient. In the second trimester, a dilation and evacuation or medical induction of labor under gynecologic consultation is performed. After curettage, prophylactic antibiotics (doxycycline, 100 mg p.o. b.i.d. for 7 days), ergot alkaloids (methylergonovine maleate, 0.2 mg p.o. t.i.d. for 2–3 days), and antiprostaglandins (ibuprofen, 800 mg p.o. every 8 hours as needed for pain) commonly are prescribed. If heavy vaginal bleeding, abdominal pain, or fever occurs after evacuation, investigation for retained products, uterine perforation, and endometritis is warranted.
- Rho immune globulin (RhoGAM)** is given to any pregnant patient with vaginal bleeding who is Rh negative and has a negative antibody screen. The recommended dose of RhoGAM is 300 µg i.m. after the first trimester. For first-trimester events, 50 µg i.m. is sufficient (*ACOG Prac Bull* 4, May 1999).
- Pathology.** Any tissue passed or obtained on uterine evacuation must be evaluated for chorionic villi. If villi are not identified, further investigation is necessary to exclude ectopic pregnancy or incomplete abortion.

B. **Nonobstetric etiologies of vaginal bleeding** ([Table 42-1](#)).

Table 42-1. Nonobstetric causes of vaginal bleeding

II. Abdominal pain. The differential diagnosis for nongynecologic causes includes appendicitis, gastroenteritis, ischemic bowel, ureteral colic, urinary tract infection, and cholecystitis. Pregnancy should be excluded in all women of reproductive age.

A. **Ectopic pregnancy** occurs when the blastocyst implants outside the uterine cavity; 97% of cases occur in a fallopian tube (tubal pregnancy).

- Presentation and clinical features.** Although more than 90% of patients with tubal pregnancies have abdominal or pelvic pain, some may be asymptomatic. Early (unruptured) pregnancies often present with amenorrhea, vaginal spotting, and colicky, vague lower abdominal pain, whereas ruptured ectopics often

- have severe pain, syncope, dizziness, and lightheadedness. Pleuritic chest pain and shoulder pain from diaphragmatic irritation can also occur.
2. **Physical examination.** Vital signs vary greatly, from normal blood pressure and pulse to hypotension and tachycardia from cardiovascular collapse secondary to hemorrhage. Whereas patients with unruptured ectopic pregnancy may demonstrate only mild tenderness, peritoneal signs, including marked tenderness, rigidity, guarding, and rebound, may be found. Pelvic masses sometimes are palpable, but lack of a mass does not exclude ectopic pregnancy. The uterus may appear small for dates.
 3. **Laboratory investigation**
 - a. **CBC.** Hgb and Hct may indicate the degree of hemorrhage except in acute cases. The WBC count typically is normal or slightly elevated and lacks a left shift.
 - b. **Blood type and screen** should be obtained to identify Rh-negative patients and to cross-match blood for possible transfusion (see [section I.A.6.g](#)).
 - c. **hCG.** Although ectopic pregnancy can occur with any quantitative serum hCG value, these data can be useful for ultrasound interpretation. Serial hCG values that do not increase appropriately are suspicious for an ectopic pregnancy (see [section I.A.4.d](#)).
 - d. **Progesterone** values rarely aid in the diagnosis of ectopic pregnancy (see [section I.A.4.e](#)).
 4. **Imaging studies.** Ultrasonography is most useful in excluding a tubal pregnancy by demonstrating an intrauterine gestational sac or fetus. With a quantitative hCG less than 1,500 mIU/mL, this may not be possible, and stable patients should be followed with serial hCG titers. However, with an hCG less than 2,500 mIU/mL, absence of a gestational sac in the uterus indicates either nonviable intrauterine or ectopic pregnancy. Ultrasound findings consistent with ectopic pregnancy include a uterus without a well-formed gestational sac, possibly free intraperitoneal fluid, and, sometimes, an adnexal mass representing the tubal pregnancy.
 5. **Diagnostic studies**
 - a. **Culdocentesis** is useful to detect hemoperitoneum but is painful and contraindicated in the presence of a cul-de-sac mass or bleeding diathesis. It is used infrequently because sonographic evaluation for free intraperitoneal fluid often is sufficient. A culdocentesis is performed with a speculum in the vagina and a single-toothed tenaculum on the posterior cervical lip to lift the cervix anteriorly and expose the uterosacral ligaments. An 18-gauge spinal needle with a 10-mL syringe is placed aseptically in the midline of the posterior vaginal fornix. Aspiration of clear yellow fluid is normal. If no fluid is obtained, the test is nondiagnostic. If clotting blood is obtained, an intravascular source is likely, and no conclusions can be drawn. If nonclotting blood is obtained, especially with an Hct above 15%, there is a hemoperitoneum, consistent with (but not diagnostic of) ruptured ectopic pregnancy.
 - b. **D&C** can be performed to differentiate between ectopic pregnancy and incomplete abortion after excluding a normal early intrauterine pregnancy. Chorionic villi are suggested if curettage products float in saline. If villi are not identified, laparoscopy to exclude ectopic pregnancy is indicated.
 6. **Treatment**
 - a. **Surgical therapy.** The majority of ectopic pregnancies are treated surgically.
 1. **Laparoscopy** is preferred for diagnosis and treatment of tubal pregnancy; however, laparotomy is indicated if the patient is hemodynamically unstable.
 2. **Conservative surgical therapy** is recommended in patients who wish to preserve reproductive potential. **Linear salpingotomy** in the antimesosalpinx portion of the tube performed with fine-tip electrocautery is preferable when the ectopic pregnancy is unruptured and is located in the ampulla of the tube. After removal of the pregnancy from the tube, the base is irrigated, and hemostasis is achieved with cautery. The tube is left to heal by secondary intention. **Segmental resection** often is performed when the tube is ruptured and the ectopic pregnancy is in the isthmic (proximal) portion of the tube; interval reanastomosis can be performed.
 3. **Nonconservative surgical therapy** includes **salpingectomy** (removal of tube) for tubal rupture or severe hemorrhage and **cornual resection** for interstitial pregnancies. Pregnancy rates after salpingectomy have been shown to be equivalent to those following linear salpingotomy, although the incidence of recurrent ectopic pregnancy may be slightly higher with salpingostomy.
 4. **Follow-up.** Patients treated with conservative surgical management, or after rupture or spillage of trophoblastic tissue, have a 5% occurrence of persistent viable trophoblastic tissue. Weekly quantitative hCG values should be followed until negative. If the levels plateau or increase, reevaluation is indicated.
 - b. **Medical therapy** with methotrexate, a folic acid antagonist, can be used in compliant outpatients who are hemodynamically stable. They should have a mass less than 3.5 cm in diameter located in the isthmus or ampulla of the tube, an intact tube, no fetal heart motion or evidence of hemoperitoneum, and an hCG less than 10,000 mIU/mL. Quantitative hCG values are followed until negative. Approximately 20% of patients have an inappropriate fall in hCG levels and require a repeat dose of methotrexate or laparoscopic evaluation.
- B. Pelvic inflammatory disease (PID)** is a polymicrobial infection of the upper genital tract. The majority of cases occur in sexually active young women between the ages of 15 and 30 years. It rarely occurs in nonmenstruating women and is extremely rare during pregnancy. Less commonly, PID follows a procedure that breaks the cervical mucus barrier, such as hysteroscopy, D&C, endometrial biopsy, or hysterosalpingography. Risk factors include a history of sexually transmitted diseases or PID, multiple sexual partners, and age less than 25 years.
1. **Presentation and clinical features.** Patients with PID typically have lower abdominal and pelvic pain, which may be constant, dull, sharp, or, occasionally, crampy. PID is aggravated by movement and often occurs around or during menses. Approximately 75% of patients have purulent vaginal discharge, fewer than 50% have abnormal vaginal bleeding, and only 33% manifest fever. Nausea and vomiting with associated ileus may occur but usually are late symptoms.
 2. **Physical examination** reveals lower abdominal tenderness, including peritoneal signs of guarding and rebound, adnexal tenderness, and cervical motion tenderness; an adnexal fullness or a mass may be found. PID is often accompanied by a purulent vaginal discharge. Cervical motion tenderness is a nonspecific sign of peritoneal irritation that can be elicited in any female patient with peritonitis of any cause, and, therefore, it is not pathognomonic of PID.
 3. **Laboratory investigation.** Pregnancy must be excluded. Elevated WBC count and elevated erythrocyte sedimentation rate suggest a severe infection. A wet-smear examination of a drop of vaginal discharge in a few drops of isotonic saline under high magnification usually reveals numerous WBCs. A cervical swab for DNA probe analysis of *Neisseria gonorrhoeae* and *Chlamydia trachomatis* should be obtained. Bloody samples may give false-negative results. Alternatively, cultures for *N. gonorrhoeae* and *C. trachomatis* can be obtained by using a urine assay (uriprobe).
 4. **Imaging studies.** Ultrasonography is used to detect tubo-ovarian abscess if a mass is palpated on examination or if no improvement is noted after 48 hours of antibiotic treatment. In general, the management of patients with PID is rarely modified by ultrasound results. Abdominal plain films, ultrasonography, or computed tomographic (CT) scan also may be used to investigate other etiologies of a patient's pain if the diagnosis is uncertain.
 5. **Diagnostic studies**
 - a. The Centers for Disease Control and Prevention (CDC) has issued guidelines for diagnosis of acute PID serving as clinical criteria for initiating treatment. **Minimum criteria** include low abdominal tenderness, adnexal tenderness, and cervical motion tenderness. **Additional criteria** include temperature greater than 101°F (38.3°C), abnormal cervical/vaginal discharge, elevated erythrocyte sedimentation rate, elevated C-reactive protein, and documented infection with *N. gonorrhoeae* or *C. trachomatis*. **Definitive criteria** include histopathologic evidence of endometritis on endometrial biopsy, transvaginal ultrasound showing fluid-filled tubes or tubo-ovarian abscesses (TOAs), and laparoscopic abnormalities consistent with PID.
 - b. **Culdocentesis** seldom is necessary to make the diagnosis of PID. However, aspiration of purulent material confirms an infectious process (see [section II.A.5.a](#)).
 - c. **Laparoscopy** revealing erythema and edema of the fallopian tubes and purulent material confirms the diagnosis and provides the opportunity to collect direct cultures of infected organs.
 6. **Treatment** of PID depends on the severity of the infection.
 - a. **Inpatient therapy** is indicated for patients with nausea and vomiting, possible surgical emergencies, pregnancy, suspicion for TOA, immunodeficiency, or failed outpatient therapy, or for patients in whom preservation of reproductive potential is very important. According to CDC guidelines from 1998, inpatient treatment is provided by cefotetan, 2 g i.v. every 12 hours, or cefoxitin, 2 g i.v. every 6 hours, and doxycycline, 100 mg p.o. or i.v. every 12 hours (alternatively, clindamycin, 900 mg i.v. every 8 hours, and gentamicin, 2 mg/kg i.v. load followed by 1.5 mg/kg i.v. every 8 hours, can be used). Other alternative regimens include ofloxacin, 400 mg i.v. every 12 hours, and metronidazole, 500 mg i.v. every 8 hours; ampicillin/sulbactam, 3 g i.v. every 6 hours, and doxycycline, 100 mg i.v. or p.o. every 12 hours; or ciprofloxacin, 200 mg i.v. every 12 hours, and doxycycline, 100 mg i.v. or p.o. every 12 hours, and metronidazole, 500 mg every 8 hours. Inpatient treatment is continued until the patient is afebrile for 48 hours and has decreased pain on pelvic examination. Patients then are treated with oral doxycycline for a total of 14 days.
 - b. **Outpatient therapy** includes one dose of ceftriaxone, 250 mg i.m., followed by doxycycline, 100 mg p.o. b.i.d. for 14 days (alternatively, ofloxacin, 400 mg p.o. b.i.d., and metronidazole, 500 mg p.o. b.i.d. for 14 days) [*MMWR Morb Mortal Wkly Rep* 47(RR-1):1, 1998]. Patients should be followed up within 48–72 hours to ensure adequate progress.
 - c. In patients with TOAs who are not responding to intravenous antibiotics after 48 hours, **surgery** should be considered. Generally, surgery should be reserved for patients with symptomatic pelvic masses or ruptured TOAs and for draining TOAs in patients failing intravenous antibiotics. Ultrasound-guided transvaginal aspiration of TOAs is another option that may benefit patients who are not responding to intravenous antibiotics.
- C. Corpus luteal cysts** develop from mature follicles in the ovary. Intrafollicular bleeding can occur 2–4 days after ovulation, creating a hemorrhagic cyst. Corpus luteal cysts usually are 4 cm or less in diameter but can be larger than 12 cm. These can occur in the pregnant or nonpregnant state. Diagnosis can be difficult in pregnancy, as a cyst may be confused with ectopic pregnancy.
1. **Presentation and clinical features.** Patients can be asymptomatic or may present with unilateral dull lower abdominal and pelvic pain. If the cyst has ruptured, the patient may complain of sudden, severe lower abdominal pain.

2. **Physical examination** reveals adnexal enlargement or tenderness, or both; if the cyst has ruptured, signs of peritoneal irritation can be present. Hemodynamic stability must be ensured, as some patients can bleed significantly from a hemorrhagic cyst.
 3. **Laboratory investigation.** A CBC count and an hCG should always be obtained. Ultrasound can aid in visualizing a cyst or free fluid in the pelvis indicative of recent cyst rupture. Culdocentesis may be performed to search for blood in the cul-de-sac in cases of suspected cyst rupture (see [section II.A.5.a](#)).
 4. **Treatment** usually is conservative, allowing for spontaneous resolution. Oral contraceptive pills (OCPs) to suppress ovulation and future cyst formation, as well as analgesia with nonsteroidal antiinflammatory drugs or short courses of narcotics, can be prescribed. Surgical treatment with laparoscopic cystectomy is rarely indicated unless significant intraperitoneal hemorrhage is present.
- D. **Adnexal torsion** occurs when the ovary, tube, or both, twist on the infundibulopelvic ligament. Incomplete torsion results in occlusion of the venous and lymphatic channels, causing cyanotic and edematous adnexa. With complete torsion, the arterial supply is interrupted, with subsequent ischemia and necrosis of the adnexa. Adnexal torsion occurs most commonly in the reproductive age group, more frequently on the right side, and typically with large ovaries or ovarian masses.
1. **Presentation and clinical features.** Patients with torsion present with acute and severe, sharp, intermittent unilateral lower abdominal or pelvic pain. The pain is proportional to the degree of vascular obstruction. Intermittent torsion may present with periodic pain for days to weeks from twisting and untwisting of the adnexa. The pain often is related to a sudden change in position. Approximately two-thirds of patients experience nausea, whereas few have fever.
 2. **Physical examination** can reveal tachycardia or bradycardia (from vagal stimulation) and fever if there is necrosis. Unilateral abdominal tenderness or a tender adnexal mass often is found on pelvic examination.
 3. **Diagnostic studies.** Ultrasonography may visualize an adnexal mass. Doppler ultrasound has moderate sensitivity and specificity in diagnosing torsion. Blood flow around the adnexa is reassuring but does not exclude the diagnosis of torsion. Laparoscopy confirms the diagnosis.
 4. **Treatment** involves immediate surgical intervention with removal of the involved adnexa and necrotic tissue. If only partial torsion or no evidence of tissue necrosis exists and the patient desires future fertility, preservation of adnexa with untwisting and stabilization is possible once underlying malignancy has been excluded.
- E. **Fibroids or leiomyomas** are benign tumors of uterine smooth muscle that vary in size from less than 1 cm to greater than 20 cm.
1. **Presentation and clinical features.** Although most fibroids are asymptomatic, one-third of patients have dysmenorrhea or abnormal menstrual bleeding, including menorrhagia (heavy menses) or metrorrhagia (intermenstrual bleeding). Symptoms also can result from local compression of the bladder or rectum.
 2. **Physical examination** reveals an enlarged, irregular uterus.
 3. **Diagnostic studies.** Ultrasonography confirms uterine size and the presence of myomas. In patients older than 35 years with abnormal bleeding, an endometrial biopsy should be performed to rule out endometrial pathology including hyperplasia and carcinoma.
 4. **Treatment** is determined by symptoms and the patient's desire for future fertility. Prostaglandin synthetase inhibitors, including ibuprofen (200–800 mg every 6–8 hours) or naproxen sodium (220–550 mg every 6 hours); hormonal therapy, including OCPs and medroxyprogesterone acetate to regulate bleeding; and gonadotropin-releasing hormone agonist to shrink the tumor can be used. Surgery (myomectomy or hysterectomy) is reserved for patients with failed medical management.
- F. **Dysmenorrhea** is painful lower abdominal cramping that occurs just before and during menses. It affects 40% of women of reproductive age and often is accompanied by other symptoms, including diaphoresis, tachycardia, headache, nausea, vomiting, and diarrhea.
1. The **etiology** of dysmenorrhea is thought to be related to increased prostaglandin levels.
 2. **Treatment** includes prostaglandin synthetase inhibitors, such as ibuprofen (400–800 mg) or naproxen sodium (220–550 mg p.o. every 6 hours) beginning before the onset of menses, or OCPs to suppress ovulation.
- G. **Adnexal masses** are often found incidentally either on pelvic examination or intraoperatively. To guide the surgeon in their management, criteria have been developed that aid in assessing the malignant potential of adnexal masses.
1. **Presentation and clinical features.** Most adnexal masses are asymptomatic unless they are associated with torsion or large enough to compress surrounding structures.
 2. **Physical examination** reveals adnexal fullness or a discrete mass. Attention should be paid to its mobility.
 3. **Diagnostic studies.** Ultrasound can reveal size and characteristics of adnexal masses. CT is less useful in visualizing the pelvis.
 4. **Treatment.** A very large (>10 cm) adnexal mass warrants laparotomy. Smaller masses in premenopausal women may be observed for 6–8 weeks unless certain factors make it likely to be malignant. If it grows or persists on repeat ultrasound, surgery is indicated. Controversy exists regarding the management of small, asymptomatic adnexal masses in postmenopausal women. Small, unilocular cysts may be observed for some time, although most practitioners have a lower threshold for surgery in this age group. Laparoscopy has been shown to be equally effective and safe for removal of masses less than 10 cm as laparotomy, with decreased associated morbidity and shorter hospital stays. Fixed and solid masses or those associated with ascites require laparotomy and a gynecologic oncologist on standby. Benign adnexal masses frequently encountered include benign cystic teratomas (dermoid cysts), endometriomas, and serous and mucinous cystadenomas. Dermoids and serous cystadenomas are frequently bilateral and warrant close inspection of the contralateral ovary. Mucinous cystadenomas can be associated with pseudomyxoma peritonei. In those cases, inspection of the appendix is important, as appendiceal mucocèles and carcinomas can also result in pseudomyxoma. Other benign, solid ovarian tumors include Brenner tumors, fibromas, and thecomas.

III. Nonobstetric surgery in the pregnant patient. Common indications for major surgery in the pregnant patient are appendicitis, adnexal mass, and cholecystitis. It is preferable to treat pregnant patients conservatively, if possible, deferring surgery to the postpartum period; however, delay of an indicated surgical procedure can be devastating. If nonemergent surgery is required, it is safest to proceed in the second trimester. Surgery in the first trimester carries a risk of fetal loss and malformation because organogenesis occurs during this period. Surgery in the third trimester carries a risk of inducing preterm labor.

- A. **Preoperative considerations.** Patients are at increased risk for aspiration because of upward displacement of the stomach and the inhibitory effects of progesterone on gastrointestinal motility. A nonparticulate antacid should be given shortly before induction of anesthesia. In the second half of pregnancy, the left lateral position should be used to decrease venacaval and aortic compression. Fetal heart tones should always be documented pre- and postoperatively.
- B. Trocar placement for **laparoscopy in pregnancy** is best accomplished by an open technique, with the additional trocars inserted under direct visualization. Laparoscopy in pregnancy has primarily been used for cholecystectomy. The second trimester is the maximum gestational age for safe laparoscopy in pregnancy because the uterus is not large enough to obstruct visualization and room for manipulation exists. Intraabdominal carbon dioxide insufflation appears safe up to 14 mm Hg and is unlikely to alter the acid-base balance in the fetus. Preliminary data suggest that laparoscopic surgical procedures may result in decreased morbidity and mortality, a lower overall cost with shorter hospitalizations, and a decreased risk of thromboembolic disease compared with laparotomy.
- C. **Anesthetic** selection is based on the maternal condition and the planned surgical procedure. For general anesthesia, all patients should be intubated using cricoid pressure to minimize the risk of aspiration. Anesthetic inhalation agents and narcotic analgesia commonly are used. Regional anesthesia may be complicated by episodes of hypotension, which is potentially poorly tolerated by patient and fetus. Most commonly administered anesthetics are acceptable for use in pregnancy.
- D. **Postoperatively**, fetal-uterine monitoring and tocolytic agents are used, depending on gestational age and degree of maternal symptoms.

IV. Trauma in pregnancy

- A. **Presentation and clinical features.** Trauma is a frequent event during pregnancy; it is the leading cause of morbidity and mortality in the United States in women younger than 40 years of age. Initial interventions are aimed at stabilization of the mother according to advanced cardiac trauma life support protocols. After initial survey of the mechanism and extent of injury, establishment of fetal age and fetal heart tone monitoring (if feasible) are undertaken.
- B. **Types of abdominal trauma**
 1. **Penetrating trauma** places the uterus and fetus at great risk during the later stages of pregnancy. Evaluation and treatment are similar to those in the nonpregnant patient with the usual necessity of surgical exploration. Amniocentesis to establish fetal lung maturity or to detect bacteria or blood may be helpful if time permits.
 2. **Blunt trauma** in pregnancy is associated most often with motor vehicle accidents. Despite the concern for abdominal seat-belt injuries, restrained pregnant patients fare better than those who are unrestrained. Intrauterine or retroplacental hemorrhage must be considered because 20% of the cardiac output in pregnancy is delivered to the uteroplacental unit.
 - a. **Abruptio placentae** occurs in 1–5% of minor and 40–50% of major blunt traumas. Focal uterine tenderness, vaginal bleeding, hypertonic contractions, and fetal compromise frequently occur. Disseminated intravascular coagulopathy may occur in almost one-third of abruptions, and a disseminated intravascular coagulation panel including fibrinogen and a Kleihauer-Betge screen to assess for fetal-maternal hemorrhage in Rh-negative patients should be ordered.
 - b. **Management of abruptio placentae** depends on fetal age and degree of placental separation and blood loss as estimated on ultrasonography. At viability, continuous fetal heart monitoring is done, and route of delivery is dictated by both fetal and maternal cardiovascular stability, with immediate cesarean section undertaken if evidence of severe compromise exists.
- C. **Special considerations of trauma in pregnancy**
 1. **Fetal-uterine monitoring** is effective in determining fetal distress, abruptio placentae, and preterm labor caused by trauma. These measures are instituted for gestations greater than 20 weeks. Dopplers are sufficient for previable pregnancies after 12 weeks of gestation.

2. **Ultrasonography** is an effective tool for establishing gestational age, fetal viability, placental state, and location.
3. In **positioning** the pregnant patient, avoid placing her supine to optimize venous return. During cardiopulmonary resuscitation, a 10- to 15-degree wedge into the left lateral decubitus position should be used, if possible.
4. **Tetanus prophylaxis** should be administered in the same manner and for the same indications as in the nonpregnant patient.
5. **Peritoneal lavage**, usually by the open, supraumbilical technique, can be used to detect intraperitoneal hemorrhage while avoiding the uterus, which is localized by examination and ultrasonography (*J Trauma* 29:1628, 1989).
6. **Radiation** in the form of diagnostic studies places the fetus at potential risk of spontaneous abortion (first several weeks of pregnancy), teratogenesis (weeks 3–12), and growth retardation (>12 weeks' gestation). These effects and secondary childhood cancers are unlikely at doses of less than 10 rads (chest film, <1 rad; CT scan of abdomen and pelvis, 5–8 rads). Uterine shielding should be used when possible. Studies should be ordered judiciously, but X-rays that are deemed important for evaluation should not be omitted (*ACOG Educ Bull* 236, April 1997).
7. **Perimortem or postmortem cesarean section** should be accomplished within 10 minutes of maternal cardiopulmonary arrest to optimize neonatal prognosis and maternal response to resuscitation.
8. **Isoimmunization** must be considered in the Rh-negative patient, and RhoGAM is administered when fetal maternal hemorrhage is suspected (see [section I.A.6.g](#)).
9. All blood product **transfusions should be screened** and negative for cytomegalovirus.
10. **Prophylactic cephalosporins** are safe in all trimesters of pregnancy.

V. Gynecologic malignancies. The female genital tract accounts for more than 80,000 new cases of invasive carcinoma annually in the United States, resulting in 26,300 deaths projected for 2001. Mortality would be reduced by earlier detection because 5-year survival rates approach 90% with proper treatment when these cancers are diagnosed as stage I (confined to primary organ) but fall to 30–50% when diagnosed at advanced stages. A brief overview of vulvar, cervical, endometrial, and ovarian cancers is presented, with emphasis on diagnosis and initial management. A complete discussion of gynecologic malignancies, including less common cancers (vaginal, fallopian tube, and gestational trophoblastic disease), is beyond the scope of this manual. Patients should be referred to a gynecologic oncologist for specialized care and comprehensive management.

- A. **Vulvar carcinoma.** Vulvar cancer is primarily a disease of postmenopausal women, with an average age at diagnosis of 68 years. The etiology has been linked to human papillomavirus infection in younger patients, and vulvar cancer is associated with chronic vulvar conditions, including dystrophies, lichen sclerosis, and condylomata. Squamous cell histology predominates (90%), followed by melanoma and adenocarcinoma.
 1. **Presentation and clinical features.** Vulvar cancer often presents as a hyper- or hypopigmented lesion. It may be ulcerating, pruritic, painful, or asymptomatic and may have been treated with a variety of antibiotics and ointments before diagnosis.
 2. **Diagnosis.** Accurate diagnosis requires biopsy and histopathologic evaluation of suspicious areas.
 3. **Treatment** of vulvar cancer depends on the stage. Surgery ranging from local excision to radical vulvectomy with bilateral groin lymph node dissection generally is the primary treatment. Local, groin, and pelvic adjuvant radiation is administered, depending on the surgical pathologic findings. In cases of basal cell carcinoma, wide local excision is sufficient.
 4. **Prognosis** depends on the extent of disease. Patients with stage I tumors experience 5-year survivals of 90% or greater, whereas those with positive lymph nodes have less than a 40% 5-year survival, depending on the number and location of positive lymph nodes.
- B. **Cervical carcinoma.** The incidence of invasive cervical cancer has dramatically decreased (but preinvasive disease has markedly increased) in the United States owing largely to widespread institution screening by cervical cytology (Papanicolaou smears). This success in cervical screening has not been duplicated worldwide; cervical cancer remains by far the leading cause of mortality among gynecologic malignancies in less developed countries. The goal is to diagnose and treat disease of the cervix in the preinvasive state. This is accomplished by aggressive evaluation of abnormal Papanicolaou smears with colposcopy-guided biopsies. Risk factors for cervical cancer include a history of sexually transmitted diseases, HIV infection, human papillomavirus, multiple sex partners, lower socioeconomic status, and smoking.
 1. **Presentation and clinical features.** Patients may be asymptomatic or present with irregular or postcoital vaginal bleeding. A foul-smelling or watery discharge may also be present. Advanced stages may present with leg pain (sciatic nerve involvement), flank pain (ureteral obstruction), or renal failure.
 2. **Diagnosis** is by biopsy via speculum examination of a visible or palpable nodule, or both. Staging remains clinical and is based on a thorough bimanual and rectovaginal examination, cystoscopy, and proctoscopy. Appropriate adjuvant radiographs include chest X-ray, intravenous pyelogram, and barium enema. Stage is never changed by intraoperative findings and remains the most important prognostic factor, with 88% 5-year survival for stage I and 38% 5-year survival for stage III disease ([Table 42-2](#)).

Table 42-2. Cervical cancer staging

3. **Treatment** depends on stage and lymph node status.
 - a. **Microinvasive disease** (stage IA1, depth of invasion <3 mm, diameter <7 mm, negative lymph vascular space invasion) can be treated with cervical conization alone or with extrafascial hysterectomy. Lesions with greater depth of invasion, multifocal disease, or upper vaginal involvement (stages IA2–IIA) require a radical hysterectomy, which removes the parametria and upper vagina and includes a complete pelvic and sometimes paraaortic lymphadenectomy. Although radical hysterectomy is only appropriate for a subset of patients, radiotherapy is applicable to any patient with early-stage cervical cancer. The most common complication after radical hysterectomy is bladder dysfunction. Ureteral fistulas, infection, hemorrhage, and lymphocyst formation are more rare.
 - b. **Radiotherapy** is the appropriate treatment for advanced-stage disease. Combined surgery and radiotherapy for advanced stages does not increase survival but dramatically increases complication rates in the form of obstruction, strictures, and fistula formation. The prescription of radiation is based on stage, lesion size, and lymph node status. Both external-beam (teletherapy) and intracavitary (brachytherapy) radiation are used in various combinations. Complications from radiotherapy depend on dose, volume, and tissue tolerance. Acute complications include transient nausea and diarrhea. Early complications occur within the first 6 months after treatment and include skin ulceration, cystitis, and proctitis. Late complications may occur any time thereafter and include bowel obstruction secondary to strictures, fistulas, and chronic proctosigmoiditis. Recent studies indicate that adding cisplatin to radiation in locally advanced (stage IIB–IVA) and early-stage high-risk disease (IB–IIA with pelvic node involvement, parametrial involvement, or positive surgical margins) decreases the risk of dying from cervical cancer by 30–50% over radiation alone.
 - c. Patients with **pelvic recurrence** after radical hysterectomy are often treated with radiation. Following primary radiation treatment, patients with central recurrence may be candidates for pelvic exenteration. Five-year survival ranges from 20% to 62% after exenteration with an operative mortality of 10%. Response to chemotherapy in recurrent cervical cancer is generally poor.
 - d. **Uncontrolled bleeding** from cervical cancer per vagina occasionally is encountered in the emergency department. In most cases, bleeding can be stabilized with tight vaginal packing, after which a transurethral Foley catheter should be placed. Acetone-soaked gauze is the most effective packing for vessel sclerosis and control of hemorrhage from necrotic tumor. Emergent radiotherapy may be necessary.
- C. **Endometrial carcinoma.** Endometrial carcinoma is the most common gynecologic malignancy in the United States, and 70% of the patients present with disease confined to the uterus. Most (approximately 90%) of these tumors are adenocarcinomas arising from the lining of the uterus. Risk factors include Caucasian race, obesity, early menarche and late menopause, nulliparity, and other factors leading to unopposed estrogen exposure. Atypical endometrial hyperplasia is a precursor lesion and progresses to carcinoma in 23% of cases if left untreated. Only 1–3% of hyperplasia without atypia progresses to carcinoma. Hyperplasia can be treated conservatively with progestins and close observation. Extrafascial hysterectomy is suggested for persistent hyperplasia in patients who have completed their childbearing.
 1. **Presentation and clinical features.** The most common symptom is abnormal vaginal bleeding, often in the form of postmenopausal bleeding. Approximately three-fourths of patients present with stage I disease.
 2. **Physical examination** may reveal evidence of hyperestrogenism. The uterus may be enlarged or of normal size.
 3. **Diagnosis** is by transcervical aspiration (e.g., Pipelle), which usually is performed as an office procedure, or by D&C, which is performed in the operating room. Ultrasound can help suggest an intrauterine abnormality. Staging is done surgically.
 4. **Treatment** generally consists of extrafascial total hysterectomy and bilateral salpingo-oophorectomy with selective pelvic and paraaortic lymphadenectomy depending on tumor histology, grade, spread, and depth of invasion ([Table 42-3](#)). To assess the latter, the uterus is bivalved in the operating room and the tumor is inspected. Pelvic fluid cytology and sometimes omentectomy are also performed. Adjuvant radiotherapy is used postoperatively in patients with poor prognostic factors who are at high risk for recurrence. Hormonal therapy or chemotherapy is used for advanced disease, but response to chemotherapy has been poor.

Table 42-3. Endometrial cancer staging

5. **Prognosis** generally is favorable, with 5-year survival greater than 90% for patients with surgical stage I tumors. Prognosis depends on the grade of tumor as well as the depth of myometrial invasion, adnexal involvement, pelvic fluid cytology, and lymph node spread. Less common histologies, such as clear-cell or papillary serous cancers, and the sarcomas arising from the wall of the uterus do not enjoy the overall good prognosis of early-stage adenocarcinomas.
- D. **Ovarian carcinoma.** Ovarian cancer is the deadliest of all the gynecologic malignancies; more than two-thirds of patients in whom epithelial ovarian cancer is diagnosed eventually die from this disease (13,900 per year in the United States) (*CA Cancer J Clin* 51:23, 2001). Besides the most common epithelial histologies that arise from the ovarian coelomic surface, germ cell (often in younger patients) and stromal primary tumors can occur. Two-thirds of epithelial ovarian cancers are diagnosed at advanced stages, with extraovarian metastasis. Incidence increases steadily with advancing age to a total lifetime incidence of 1 in 57. Risk factors include nulliparity, late menopause, early menarche, and high dietary fat intake. OCP use is protective secondary to inhibition of ovulation.
 1. **Presentation and clinical features.** Women with early-stage disease are generally asymptomatic. In later stages, patients may present with vague abdominal pain or pressure, nausea, early satiety, weight loss, or swelling.
 2. **Diagnosis** of ovarian cancer at early stages has proved clinically difficult ([Table 42-4](#)). No cost-effective screening test has yet been proved reliable in detecting stage I disease (confined to the ovaries). Bimanual examination remains the most effective means of screening, followed by surgery for histologic diagnosis. Ultrasonography of the pelvis (preferably transvaginal) and CT scan are effective adjuncts. CA 125 antigen is not effective for mass screening but serves as an effective tumor marker once diagnosis has been established and treatment is initiated.

Table 42-4. Ovarian cancer staging

3. **Treatment** is primarily surgical, with aggressive debulking and complete staging, even if inspection intraoperatively suggests that disease may be confined to one ovary. Complete staging is done surgically and includes pelvic washings on entering the peritoneum, total abdominal hysterectomy, bilateral salpingo-oophorectomy, omentectomy, pelvic and paraaortic lymph node dissection, and peritoneal biopsies. Optimal cytoreduction (residual disease <1 cm) improves response to adjuvant chemotherapy and overall survival. In young women with early-stage disease, fertility-sparing surgery can often be performed, with removal of the uterus and contralateral ovary after childbearing is completed. However, complete staging at initial surgery is still necessary. Patients with disease outside the ovary are treated with six cycles of paclitaxel and platinum-based chemotherapy.
4. **Prognosis** correlates directly with stage and with the residual disease remaining after debulking. Median survival depends on optimal cytoreduction with removal of all possible tumor at initial laparotomy. Median survival for optimally debulked advanced-stage tumors is nearly 3 years, but overall survival at 5 years falls to approximately 30%.

43 Common Surgical Procedures

Randall P. Scheri and Gerard M. Doherty

Catheterization, Drainage, and Emergency Airway Procedures

I. Central venous catheterization

II. Thoracic drainage procedures

III. Peritoneal drainage procedure

IV. Emergency airways

Catheterization, Drainage, and Emergency Airway Procedures

I. Central venous catheterization. The ability to gain access to the central venous circulation rapidly and safely is essential for the management of surgical patients. Central venous catheterization is an important diagnostic tool for the measurement of central venous pressure, pulmonary wedge pressure, cardiac output, or mixed venous blood gases, as well as an important therapeutic tool in the delivery of intravenous fluids, blood products, medications, or total parenteral nutrition. Hemodialysis, plasmapheresis, chemotherapy, and the placement of inferior vena cava filters are also indications for central venous access. Several approaches to the central venous system exist, and, depending on the indication, each has its advantages and disadvantages.

Absolute and relative contraindications for central venous catheterization exist. Venous thrombosis is an absolute contraindication to catheter placement in that vein. This may not become evident until the vein has already been cannulated, but once noted, an alternative approach should be used. Relative contraindications include coagulopathies (international normalized ratio >2 or partial prothrombin time >55 seconds), thrombocytopenia (platelet count $<50,000/\mu\text{L}$), ongoing sepsis, or the restless, combative patient in whom safe placement of a central venous catheter is difficult. The needle used to cannulate the venous system has the potential to cause significant damage to blood vessels, lung parenchyma, nerves, and soft tissue. Therefore, it is very important for the surgeon to set a limit on the number of unsuccessful passes to be made with the needle before abandoning the procedure and obtaining assistance.

A. Internal jugular approach

- 1. Indications.** The internal jugular vein is easily and rapidly accessible in most surgical patients. Several advantages to obtaining central venous access through the internal jugular vein exist: decreased risk of pneumothorax compared to the subclavian approach, direct access to the vessels in the case of bleeding, and the fact that the right internal jugular offers the most direct route to the right atrium. Therefore, it is the preferred route of access for short-term catheters; for thin, frail patients; and for the coagulopathic or thrombocytopenic patient. The right internal jugular vein is the preferred site for the placement of Swan-Ganz catheters because it offers the most direct path to the right atrium and pulmonary vasculature. In contrast, the left internal jugular vein has an indirect route to the pulmonary vasculature; therefore, Swan-Ganz catheters should be placed here only if the other approaches have been exhausted.
- 2.** For the unsedated or ambulatory patient, this is a difficult site to maintain. An internal jugular catheter is an uncomfortable site and may hinder the patient's neck movement. Keeping the insertion site dressed and sterile can be difficult. It is a site that should not be used for long-term access.
- 3. Technique.** The pulse of the common carotid artery is palpated at the medial border of the sternocleidomastoid (SCM) at midneck. The internal jugular vein is located lateral to the common carotid artery and courses slightly anterior to the artery as it joins the subclavian vein ([Fig. 43-1](#)). Two equally effective approaches to the internal jugular vein are advocated: the central and posterior approaches. The patient is placed in the Trendelenburg position at an angle of 10–15 degrees with his or her head flat on the bed and turned contralateral to the side of the line placement. The skin is prepared with povidone-iodine, and 1% lidocaine is infiltrated subcutaneously over the belly and lateral border of the SCM. The physician stands at the head of the bed for either approach. For the central approach, a 21-gauge “seeker” needle is introduced approximately 1 cm lateral to the carotid pulse into the belly of the SCM. At a 45-degree angle to the floor, the needle is slowly advanced toward the ipsilateral nipple. For the posterior approach, the seeker needle is introduced at the lateral edge of the SCM and directed toward the sternal notch at a 45-degree angle to the floor. The vein should be entered within 5–7 cm with both approaches. If the vein is not entered, the needle should be withdrawn and redirected for another pass. Redirection of the needle should be done just below the surface of the skin because the tip of the needle is capable of lacerating the vessels in the area. As previously mentioned, a limit on the number of passes should be set. Constant negative pressure is exerted on the syringe at all times, and entry into the vein is confirmed by the return of venous blood.

Fig. 43-1. Anatomy of the upper chest and neck, including the vasculature and important landmarks. SVC, superior vena cava.

- 4.** A 14-gauge needle is then introduced just inferior to the seeker needle and is advanced along the same path until dark, venous blood is aspirated. The **Seldinger technique** is carried out, whereby a flexible guidewire is passed into the vein through the 14-gauge needle, the needle is removed over the wire, and a nick is made in the skin at the puncture site to allow passage of the dilator. It is very important to maintain control of the guidewire at all times. The dilator creates a tract for the passage of the less rigid central venous catheter. The catheter is introduced over the wire and advanced to 15–20 cm so that its tip is at the junction of the superior vena cava (SVC) and the right atrium. In the operating room, fluoroscopy may be used to assess the position of the catheter tip immediately. Aspiration of blood from all ports and subsequent flushing with saline confirm that the catheter is positioned in the vein and that all its ports are functional. The catheter is then secured to the patient's neck at a minimum of two sites, and a sterile dressing is applied. A chest X-ray is obtained to confirm the location of the catheter tip and to rule out the presence of a pneumothorax.
- 5. Complications.** All percutaneously placed neck lines carry a risk of pneumothorax. This should be evaluated immediately after placement of the catheter with an anteroposterior chest X-ray. Every attempt at placement of a central venous catheter, successful or unsuccessful, should be followed by a chest X-ray before the catheter is used or line placement is attempted at another site. Inadvertent carotid artery puncture is usually tolerated in the noncoagulopathic patient. This is treated by direct pressure over the carotid artery. If the carotid artery is punctured, no further attempts at central venous access should be made on either side of the patient's neck. The patient's neck must be watched for the next 10–15 minutes for the development of a neck hematoma. In the hemodynamically unstable or poorly oxygenating patient, it is not always possible to distinguish venous blood from arterial blood by appearance. This can lead to inadvertent cannulation of the carotid artery. When this complication is recognized, the dilator or catheter should not be removed because this can lead to life-threatening hemorrhage, and help should be sought. The dilator is a rigid, large-bore device that is capable of injuring the subclavian or brachiocephalic vein during its advancement; therefore, great care must be taken when it is introduced and advanced. Advancement of guidewire into the right atrium can cause an arrhythmia, which usually resolves once the wire is withdrawn from the right atrium. Venous stenosis can occur at the site where the catheter enters the vein, which can lead to thrombosis of the vessel. Because the upper extremities and neck have extensive venous collaterals, stenosis or thrombosis is usually well tolerated. If the patient develops symptoms from a stenosis, the lesion can either be stented or undergo venoplasty. Air embolus, perforation of the right atrium or ventricle with resultant hemopericardium or cardiac tamponade injury to the trachea, esophagus, thoracic duct, vagus nerve, phrenic nerve, or brachial plexus can all complicate the placement of neck catheters.
- 6.** Treatment of catheter-associated infections and sepsis is the removal of the catheter and institution of appropriate antibiotic therapy. Some advocate the routine replacement of indwelling central venous catheters every 3–7 days to prevent line sepsis (*Ann Surg* 208:651, 1988). Other studies have failed to show significant reduction of catheter-associated infections with the prophylactic exchange of catheters versus replacing them as needed (*Crit Care Med* 25:257, 1997). One study demonstrated that catheters made of polyurethane and impregnated with chlorhexidine and silver sulfadiazine reduced the incidence of catheter-related infections and increased the time that they could be left in place (*Ann Intern Med* 127:257, 1997). Cuffs impregnated with antibiotics are advocated in some centers to prevent the migration of skin flora along the catheter tract in long-term catheters (*Am J Infect Control* 16:79, 1988).

B. Subclavian vein approach

- 1. Indications.** The subclavian approach to the central venous system is the site least cumbersome to the patient and is the easiest to maintain. Therefore, it is the preferred site for long-term indwelling catheters. Because the left subclavian vein offers a direct route to the right atrium and the pulmonary vasculature, it is the second best approach for the placement of a Swan-Ganz catheter. The clavicle overlies the subclavian vessels and does not allow direct access to the vessels; therefore, this approach should not be used in the coagulopathic or thrombocytopenic patient. Because of the significant risk of pneumothorax and lack of ability to control the vessels, the subclavian approach should be discouraged in emergency situations.
- 2. Technique.** The subclavian vein courses posterior to the clavicle, where it joins the internal jugular vein and the contralateral veins to form the SVC ([Fig.](#)

43-1). The subclavian artery and the apical pleura lie just posterior to the subclavian vein. The patient is placed in the Trendelenburg position with a rolled towel between the scapulas, which allows the shoulders to fall posterior. The skin is prepared with povidone-iodine, and 1% lidocaine is infiltrated subcutaneously in the infraclavicular space near the middle and lateral third of the clavicle. The infusion is carried into the deep soft tissue and to the periosteum of the clavicle. A 14-gauge needle is introduced at the middle third of the clavicle in the deltopectoral groove. Keeping the needle parallel to the plane of the floor, the needle is slowly advanced toward the sternal notch, deep to the clavicle. Constant negative pressure is applied to the syringe. Once the needle enters the subclavian vein, the guidewire, dilator, and catheter are introduced using the **Seldinger technique**, as previously described (see [section I.A.4](#)). As with the other approaches, all catheter ports are aspirated and flushed to ensure that they are functional. A chest X-ray is obtained to confirm the location of the catheter tip and to evaluate for pneumothorax. A supraclavicular approach to the subclavian vein has also been described (*South Med J* 83:1178, 1990).

3. **Complications.** The complications of subclavian venous catheterization include those described in [section I.A.5](#). Puncture of the subclavian artery can be troublesome because the clavicle prevents the application of direct pressure to achieve homeostasis. Therefore, this approach should be avoided in the patient with coagulopathy. If the artery is punctured, the patient should be monitored for the next 30–45 minutes to ensure that bleeding is not ongoing. Inadvertent cannulation of the subclavian artery with the dilator or catheter is a potentially fatal complication. The dilator or catheter should be left in place, and removal of the catheter and repair of the arteriotomy are to be done in the operating room. Particular attention must be paid to the placement of left subclavian catheters to avoid injuring the thoracic duct, brachiocephalic vein, and SVC with the needle or dilator because of the course of these vessels. Attention must be paid to the final position of the catheter tip when placed on the left side to avoid abutting the SVC wall, which poses the immediate or delayed risk of SVC perforation (*Chest* 101:1633, 1992).

C. Femoral vein approach

1. **Indications.** The femoral vein is the easiest site to obtain central access and is therefore the preferred approach for central venous access during cardiopulmonary resuscitation. This approach does not interfere with the other procedures of cardiopulmonary resuscitation. It should be remembered that a femoral vein catheter does not actually reach the central circulation and may not be ideal for the administration of vasoactive drugs. This may be the only site available in patients with upper-body burns. The femoral vein catheter inhibits patient mobility, and the groin is a difficult area in which to maintain sterility. Therefore, it should not be used in elective situations, except when upper-extremity and neck sites are not available.
2. **Technique.** The femoral artery crosses the inguinal ligament approximately midway between the anterosuperior iliac spine and the pubic tubercle. The femoral vein runs medial to the artery as they cross the inguinal ligament ([Fig. 43-2](#)). If possible, placing the patient in the reverse Trendelenburg position may help with venous cannulation. The skin is prepared with povidone-iodine, and 1% lidocaine is infiltrated in the subcutaneous tissue medial to the femoral artery and inferior to the inguinal ligament. The pulse of the femoral artery is palpated below the inguinal ligament, and a 14-gauge needle is introduced medial to the pulse at a 30-degree angle to the floor. It is directed cephalad with constant negative pressure until the vein is entered. The catheter is placed using the **Seldinger technique** described in [section I.A.4](#). When a femoral pulse cannot be palpated, as in cardiopulmonary arrest, the position of the femoral artery can be estimated to be at the midpoint between the anterosuperior iliac spine and the pubic tubercle, and the vein then lies 1–2 cm medial to this point. Once the catheter is successfully placed, all three ports are aspirated and flushed to ensure that they are functional.

[Fig. 43-2](#). Anatomy of the femoral vessels.

3. **Complications.** Injury to the common femoral artery or its branches during cannulation of the femoral vein can result in an inguinal or retroperitoneal hematoma, a pseudoaneurysm, or an arteriovenous fistula. The femoral nerve can be also be damaged. Injury to the inguinal lymphatic system can result in a lymphocele. The possibility of injuring peritoneal structures also exists, particularly if an inguinal hernia is present. Errant passages of the guidewire and the rigid dilator run the risk of perforating the pelvic venous complex and causing retroperitoneal hemorrhage. Late complications include infection and thrombosis of the femoral vein.

D. Cephalic vein cutdown

1. **Indications.** Cannulation of the cephalic vein under direct visualization has several advantages over the percutaneous approach to the central venous system. A decreased risk of pneumothorax exists, and bleeding complications are easily controlled. It is indicated in the patient with coagulopathy, the patient who cannot tolerate the Trendelenburg position, and the patient who would tolerate pneumothorax poorly. When repeated percutaneous attempts at vascular access have failed, cephalic vein cutdown should be considered. Some consider this the preferred approach for the placement of long-term vascular access devices. This approach should be attempted under strict sterile conditions, such as the operating room; bedside cephalic vein cutdown is discouraged.
2. **Technique.** The cephalic vein courses anterior along the deltoid muscle. It is reached most easily where it passes through the deltopectoral groove before turning posterior to enter the axillary vein ([Fig. 43-1](#)). Once the area over the lateral third of the clavicle has been prepared with povidone-iodine, 1% lidocaine is infiltrated subcutaneously over the deltopectoral groove, just medial to the coracoid process. A 3- to 4-cm incision is made in the skin parallel to the deltopectoral groove. The incision is carried down to the level of the clavicopectoral fascia. This fascia is incised, and the cephalic vein should then be easily identified. The vein is mobilized and ligated distally. A transverse venotomy is made, and the catheter is passed into the vein under direct visualization, with or without the assistance of a vein pick. The catheter position is confirmed via radiography or fluoroscopy.
3. **Complications.** Compared to the percutaneous approach, no significant difference in the complications of cephalic vein cutdown exists. Risk of pneumothorax, bleeding, air embolus, damage to the vessels, thrombosis, and infection remains, but the risk of pneumothorax and arterial injuries is significantly lower. Inadequate vein size and difficulty in cannulating the vein does lead to prolonged operative times and increases the need for additional cutdown sites (*Am Surg* 208:651, 1984). Because the vein is ligated, its future use is compromised.

II. Thoracic drainage procedures

A. Thoracentesis

1. **Indications.** A diagnostic thoracentesis is indicated for any pleural effusion of unknown etiology. Pleural effusions are generally categorized as transudative or exudative. This differentiation of pleural fluid is based on its microscopic, gross, and biochemical characteristics. A wide variety of laboratory studies are available to study pleural fluid. The patient's clinical presentation should guide the studies obtained. A pleural fluid–serum lactate dehydrogenase (LDH) ratio greater than 0.6 and a fluid–serum protein ratio greater than 0.5 indicate an exudative effusion, whereas a fluid–serum LDH ratio less than 0.6 and a fluid–serum protein ratio less than 0.5 indicate a transudative effusion. Fluid glucose level, amylase level, lipid level, and pH should also be measured when analyzing pleural fluid. Cytologic examination for malignant cells should be obtained when a malignant effusion is considered. If an infectious etiology is suspected, Gram stain and culture for bacteria and fungi are necessary (*Ches* 111:970, 1997). Therapeutic thoracentesis is indicated to relieve shortness of breath or discomfort from large pleural effusions. When repeated therapeutic thoracentesis is needed to treat recurrent pleural effusions, chest tube drainage and pleurodesis should be considered (*Am Rev Respir Dis* 140:257, 1989).
2. **Technique.** The site for thoracentesis depends on the location of the effusion, which can be determined by physical and radiographic examination. For free-flowing effusions, the patient is seated upright and slightly forward. The thorax should be entered posteriorly, 4–6 cm lateral to the spinal column and one to two interspaces below the cessation of tactile fremitus and where percussion is dull. Loculated effusions can be localized by ultrasonography, and the site for thoracentesis is marked on the skin. The site is prepared with povidone-iodine and draped with sterile towels. Lidocaine 1% is infiltrated into the subcutaneous tissue covering the rib below the interspace to be entered. The infiltration is carried deep to the periosteum of the rib. Next, placing negative pressure on the syringe, the needle is advanced slowly over the top of the rib, taking care to avoid injury to the neurovascular bundle. The needle is advanced until pleural fluid is returned; then, it is withdrawn a fraction, and lidocaine is injected to anesthetize the pleura. Lidocaine is then infiltrated into the intercostal muscles as the needle is withdrawn.

Most thoracentesis kits contain a long 14-gauge needle inserted into a plastic catheter with an attached syringe and stopcock. The needle-catheter apparatus is introduced at the level of the rib below the interspace to be entered. With negative pressure applied to the syringe, the needle is slowly advanced over the top of the rib and into the pleural cavity until fluid is returned. Aspiration of air bubbles indicates puncture of the lung parenchyma; the needle should be promptly removed under negative pressure. Once the needle is in the pleural space, the catheter is advanced over the needle toward the diaphragm. Special attention is taken not to advance the needle as the catheter is being directed into the pleural space. A drainage bag is attached to the stopcock to remove the pleural fluid. The amount of fluid removed depends on the indication for the thoracentesis. A diagnostic tap requires 20–30 mL fluid for the appropriate tests, and a therapeutic tap can drain 1–2 L fluid. A chest X-ray should be obtained after the procedure to evaluate for pneumothorax and resolution of the effusion.

3. **Complications.** Pneumothorax is the most common complication of thoracentesis. It must be treated with a tube thoracostomy and negative suction until the air leak seals. Reexpansion pulmonary edema is common after therapeutic thoracentesis when a large amount of fluid is removed at one time. To minimize

the occurrence of this complication, no more than 1.5–2.0 L should be removed at a time. Hemothorax, infection, injury to the neurovascular bundle, and subcutaneous hematoma are other complications that can follow this procedure.

B. Tube thoracostomy

- 1. Indications.** Tube thoracostomy is indicated for a pneumothorax, hemothorax, recurrent pleural effusion, chylothorax, and empyema. The need for a chest tube may be emergent (e.g., a trauma situation) or elective (e.g., the drainage of a recurrent malignant effusion). Under either circumstance, a good understanding of thoracic anatomy is needed to prevent injuries to the lung parenchyma, diaphragm, intercostal neurovascular bundles, and mediastinum. The lung may have adhesions to the chest wall that make insertion and advancement of the thoracostomy tube difficult. The intercostal neurovascular bundle runs in a groove on the inferior aspect of each rib; therefore, the tube should be passed over the top of the rib to avoid injury. Because, during normal respiration, the diaphragm can rise to the level of the fourth intercostal space, insertion of the chest tube lower than the sixth interspace is to be discouraged. Profound coagulopathy is a relative contraindication to the placement of chest tubes, and all efforts should be made to correct the coagulopathy before the tube is placed.
- 2. Technique.** The size of thoracostomy tube needed depends on the material to be drained. Generally, a 24–28 French tube directed apically is used for a pneumothorax, and a 28–32 French tube directed basally is used for the evacuation of a hemothorax and a dependent pleural effusion. The patient is placed in the lateral position with the unaffected side down, and the head of the bed is inclined 10–15 degrees. The patient's arm on the affected side is extended forward or above the head. With the skin prepared, 1% lidocaine is infiltrated over the fifth or sixth rib in the middle or anterior axillary line by the technique described in [section II.A.2](#). A 2- to 3-cm transverse incision is made through the skin and subcutaneous tissue. A curved clamp is used to bluntly dissect an oblique tract to the rib ([Fig. 43-3A](#)). With careful spreading, the clamp is advanced over the top of the rib. The parietal pleura is punctured with the clamp, and an efflux of air or fluid is usually encountered. A finger is introduced into the tract to ensure passage into the pleural space and to lyse any adhesions at the point of entry ([Fig. 43-3B](#)). With the clamp as a guide, the chest tube is introduced into the pleural space ([Fig. 43-3C](#)). It is directed apically for pneumothorax and basally and posteriorly for dependent effusions. A clamp placed at the free end of the chest tube prevents drainage from the chest until the tube can be connected to a closed suction or water-seal system. The chest tube is advanced until the last hole of the tube is clearly inside the thoracic cavity. When the tube is positioned properly and functioning adequately, it is secured to the skin with two heavy silk sutures and covered with an occlusive dressing to prevent air leaks. Some surgeons place a U-stitch around the chest tube to be used as a purse-string suture when the tube is removed. A chest X-ray is obtained after the procedure to assess reexpansion of the lung and the tube position. The trocar insertion technique has been discouraged because of a high incidence of complications, but safe modifications of the technique have been described (*J Am Coll Surg* 179:230, 1994).

Fig. 43-3. Tube thoracostomy placement. **A:** Pleural space entered by blunt spreading of the clamp over the top of the adjacent rib. **B:** A finger is introduced to ensure position within the pleural space and to lyse adhesions. **C:** The chest tube is placed into the tunnel and directed with the help of a Kelly clamp. The tube is directed caudal for an effusion or hemothorax and cephalad for a pneumothorax.

- 3. Complications.** The majority of complications are due to improper insertion technique and tube placement. Low placement of a chest tube can result in injury to the diaphragm with associated injury to the liver or spleen. Management of a punctured diaphragm or intraperitoneal chest tube must be tailored to the particular situation. Some tubes may be removed without significant consequence, and others may require operative management. Failure to guide the tube into the pleural space can result in dissection of the extrapleural plane. This can be a difficult diagnosis, but anteroposterior and lateral chest radiographs should reveal a lung that has failed to reexpand and suggest a chest tube placed outside the thorax. The tube should be removed and placed within the thoracic cavity to reexpand the lung. Parenchymal or hilar injuries or cardiac contusions can occur with overzealous advancement of the tube or dissection of pleural adhesions. Other complications include subcutaneous emphysema, reexpansion pulmonary edema, phrenic nerve injury, esophageal perforation, contralateral pneumothorax, and neurovascular bundle injury. Late complications include empyema, infection along the chest tube tract, and abscess.

III. Peritoneal drainage procedure

A. Paracentesis

- 1. Indications.** Bedside paracentesis is a useful diagnostic and therapeutic tool. A diagnostic paracentesis is indicated in the case of ascites with an unknown etiology. Measurement of the fluid protein, LDH, amylase, specific gravity, red blood cell count, white blood cell count with differential, and fibrinogen can help establish a diagnosis. Cytology, Gram stain, and culture are other useful laboratory tests. A therapeutic paracentesis is indicated for patients with respiratory compromise or discomfort caused by tense ascites and in patients with ascites refractory to medical management. Relative contraindications include previous abdominal surgery, pregnancy, coagulopathy, and progressive liver failure with encephalopathy or hepatorenal syndrome.
- 2. Technique.** The patient's bladder is emptied, either by the patient or by the placement of a Foley catheter. With the patient lying supine, the level of the ascites can be determined by locating the transition from dullness to tympany with percussion. Depending on the height of the ascites, a midline or lateral approach can be used. Care must be taken with the midline approach because the air-filled bowel tends to float on top of the ascites. The skin at the site of entry should be prepared and draped. One percent lidocaine is infiltrated subcutaneously and is carried to the level of the peritoneum. For the midline approach, a needle is introduced at a point midway between the umbilicus and the pubis symphysis. For the lateral approach, the point of entry can be in the right or left lower quadrant in the area bound by the lateral border of the rectus abdominis muscle, the line between the umbilicus and the anterior iliac spine, and the line between the anterior iliac spine and the pubis symphysis. A simple diagnostic tap can be achieved by inserting a 22-gauge needle into the peritoneal cavity and aspirating 20–30 mL fluid. Constant negative pressure should be applied to the syringe, and care is taken not to advance the needle beyond the point where ascites is encountered. For a therapeutic paracentesis, a 14-gauge needle fit with a catheter allows for efficient drainage of larger volumes of ascites. With either the midline or the lateral approach, once ascites is returned, the catheter is advanced over the needle and directed toward the pelvis. A drainage bag is attached to the catheter to collect and measure the fluid removed. Intravenous volume replacement with 10 g 25% albumin for each liter of ascites removed helps prevent hypovolemia and hypotension with large-volume taps.
- 3. Complications.** Injuries to the bowel or bladder can occur with percutaneous paracentesis. Emptying of the bladder, avoiding the insertion of the needle near surgical scars, and maintaining control of the needle once inside the peritoneum help to minimize these injuries. Intraperitoneal hemorrhage from injury to a mesenteric vessel can occur. Laceration of the inferior epigastric vessels can lead to a hematoma of the rectus sheath or the abdominal wall. In patients with large, recurrent ascites, a persistent leakage of ascites from the site of entry can result. These fistulas usually close spontaneously, but they can persist and develop into difficult management issues. Late complications include peritonitis and abdominal wall abscess.

B. Diagnostic peritoneal lavage (DPL)

- 1. Indications.** DPL is most useful in the diagnosis of intraabdominal injury associated with stab wounds and blunt trauma. In general, it is indicated when the injured patient has (1) an equivocal abdominal examination; (2) an abdominal examination that is unreliable because of coma, intoxication, spinal cord injury, or anesthesia; (3) unexplained hypotension; or (4) other injuries that require urgent treatment, thus making observation impractical. Ultrasonography and computed tomography are also effective modalities to detect intraabdominal injury associated with blunt trauma. The uses, advantages, and disadvantages of the various diagnostic modalities are discussed in [Chapter 28](#). DPL has the advantage of being performed easily and rapidly, and it has been reported to be 90% sensitive for detecting intraabdominal hemorrhage (*Arch Surg* 132:909, 1997). The only absolute contraindication is an existing indication for exploratory laparotomy. Relative contraindications include previous abdominal surgery, pregnancy, and morbid obesity.
- 2. Technique.** The DPL can be performed either open or closed. The closed technique is not advocated because of the high risk of injuring an intraabdominal structure. Therefore, only the open technique is presented here. The patient is placed in the supine position and the bladder decompressed. The midline, infraumbilical area is prepared and draped. After the subcutaneous tissue is infiltrated with 1% lidocaine, a short midline incision is made below the umbilicus. The incision is carried through the linea alba to the level of the peritoneum. Once the peritoneum is identified, it is elevated with forceps, and entry is gained under direct visualization ([Fig. 43-4](#)). A peritoneal dialysis catheter is introduced and directed deep into the pelvis. The direct aspiration of 5–10 mL gross blood, feculent material, or bile is a positive lavage and is an indication for emergent exploration. In the absence of a bloody, feculent, or bilious aspirate, 1 L saline is run into the peritoneal cavity. The drainage bag is lowered to the ground, and the lavage fluid is recovered by gravity. One hundred thousand red blood cells/ μ L or 500 white blood cells/ μ L is considered a positive lavage. If pelvic fracture or a retroperitoneal hematoma is suspected, a supraumbilical approach helps to minimize false-positive results caused by hematomas expanding into the infraumbilical region. If the lavage is negative, the fascial layer is closed with interrupted figure-of-eight stitches with a 0 synthetic monofilament suture. The skin can be closed with simple stitches or a running subcuticular stitch.

[Fig. 43-4](#). Open technique for peritoneal lavage. The peritoneal envelope is entered under direct vision after sharply traversing the midline infraumbilical layers.

3. **Complications.** Complications are the same as those of paracentesis, including laceration of the urinary bladder or visceral organs, bowel perforation, hemorrhage, and infection. An incisional hernia can develop at the site where the peritoneum was entered. Creation of a false path in the suprapertoneal plane or bleeding from the entry site can lead to false-positive results. The use of the open technique helps to minimize these complications as much as possible.

IV. Emergency airways

A. Endotracheal (ET) intubation

1. **Indications.** Establishment of a secure airway is the first priority in the management of an acutely ill patient. The airway can be secured either mechanically, with an ET tube, or surgically, with a tracheostomy or cricothyroidotomy. ET intubation is indicated when a patient is unable to adequately oxygenate and ventilate. Cardiac arrest, pneumonia, adult respiratory distress syndrome, pulmonary edema, inhalation injury, multisystem trauma, sepsis, postoperative hemodynamic instability, and altered mental status owing to trauma, anesthesia, or coma are reasons for failure to oxygenate or ventilate. The oral approach under direct vision is the most common method to intubate the trachea. Other approaches include nasotracheal and endoscopic intubation; however, only orotracheal intubation is presented here. Relative contraindications to orotracheal intubation include maxillofacial trauma, laryngeal injury, and cervical spine injury.
2. **Technique.** Whether orotracheal intubation is emergent or elective, the principles are the same. Preoxygenation with a bag-valve-mask apparatus and 100% oxygen suction, adequate sedation, and muscle relaxation; an appropriately sized ET tube; and a functional laryngoscope are required. Two types of laryngeal scope blades are available: a straight blade (Miller) and a curved blade (Macintosh). The straight blade may provide better visualization in children, and the curved blade may be better for patients with short, thick necks. The physician should be comfortable using either blade. With the physician at the patient's head, the head is positioned so that the pharyngeal and laryngeal axes are in alignment ([Fig. 43-5A](#)). The patient's head and neck are fully extended into the "sniffing" position. With the nondominant hand, the physician opens the patient's mouth with the thumb and index finger on the patient's lower and upper teeth, respectively. Using the middle finger, the physician sweeps the patient's tongue to the side. The oropharynx is inspected, and foreign bodies or secretions are removed. The blade of the laryngoscope is introduced and advanced with gentle traction of the scope upward and toward the patient's feet. Once the epiglottis is visualized, the tip of the blade is positioned in the vallecula. Great care must be taken not to use the handle as a lever against the patient's teeth and lips. The glottic opening and vocal cords should come into view ([Fig. 43-5B](#)). If not, gently increase the upward and caudal traction or have an assistant place external pressure on the cricoid and thyroid cartilage. If still unable to visualize the glottic opening, remove the blade, oxygenate and reposition the patient, and try again. Once the glottic opening is adequately visualized, the ET tube is advanced under direct vision until the cuff passes through the vocal cords. The cuff is inserted roughly 2 cm past the vocal cords, and the patient's incisors should rest at the 19- to 23-cm markings on the tube. The stylet and laryngoscope are carefully removed while maintaining control and the position of the ET tube. The cuff is inflated, and proper position is confirmed by auscultating bilateral breath sounds or monitoring end-tidal carbon dioxide. Once position is confirmed, the ET tube is secured to the patient. An anteroposterior chest X-ray is obtained to confirm position. When in the ideal position, the tip of the ET tube is 2–4 cm above the carina.

[Fig. 43-5](#). Orotracheal intubation. **A:** Fully extending the patient's head into the "sniffing" position aligns the pharyngeal and laryngeal axes. This allows for the best visualization of the airway. **B:** View of the larynx and airway during oral intubation of the trachea.

3. **Complications.** Unsuccessful attempts to intubate the trachea must be followed by face-mask ventilation and oxygenation with 100% oxygen. Do not allow the patient to go for more than 30 seconds without ventilation and oxygen when attempting to intubate the trachea. If the patient cannot be manually ventilated or the most experienced person is unable to intubate the trachea, an emergent surgical airway must be secured (discussed in the following section). Intubation of the esophagus or right main-stem bronchus can readily be diagnosed by absent breath sounds associated with epigastric gargling on manual ventilation and right-sided breath sounds with absent left-sided breath sounds, respectively. Chipped teeth, emesis, vocal cord injury, laryngospasm, and soft-tissue injury to lips, tongue, and gums may all complicate ET intubation.

B. Cricothyroidotomy

1. **Indications.** Cricothyroidotomy is indicated when attempts at oral or nasal intubation have failed or when maxillofacial injury prohibits oral or nasal intubation. In cases of trauma, blood or maxillofacial injuries may prevent direct visualization of the larynx. In this instance, cricothyroidotomy is the procedure of choice. Other indications for a surgical airway include cervical spine injuries and difficulty intubating the patient because of his or her location. In extreme cases, a large-bore needle can be placed through the cricothyroid membrane as a temporary mode of ventilation until a more secure airway can be obtained. Laryngeal tracheal separation and laryngeal trauma are contraindications to this procedure. Percutaneous dilational cricothyroidotomy is a safe and effective method to emergently obtain an airway, and it is gaining widespread use (*Intensive Care Med* 22:937, 1996).
2. **Technique.** Because the vast majority of cricothyroidotomies are done in emergent situations, an excellent understanding of the anatomy in the region of the trachea is necessary to minimize complications. The thyroid cartilage is easily identified in the midline of the neck ([Fig. 43-6](#)). The cricoid, the only complete cartilaginous ring, is the first ring inferior to the thyroid cartilage. The cricothyroid membrane joins these two cartilages and is an avascular membrane. Inferior to the cricoid and straddling the trachea is the isthmus of the thyroid gland. The thyroid lobes lie lateral to the trachea, and the superior poles can extend to the level of the thyroid cartilage. If time permits, the area is prepared, draped, and anesthetized with 1% lidocaine. The cricoid cartilage is identified and held firmly and circumferentially in the physician's nondominant hand until the end of the procedure. Using a No. 11 or 15 blade, a small 3- to 5-cm transverse incision is made over the cricothyroid membrane. The incision is carried deep until the airway is entered through the cricothyroid membrane. The index finger of the physician's nondominant hand can be used to identify landmarks as the dissection proceeds. Using a clamp, tracheal dilator, or the end of the scalpel handle, widen the tract. Insert the tracheostomy tube along its curve into the trachea, inflate the cuff, and check for bilateral breath sounds. If breath sounds are present, secure the tracheostomy to the skin by suturing the tabs to the skin with heavy, nonabsorbable, monofilament suture.

[Fig. 43-6](#). Anatomy of the larynx.

3. **Complications.** Creation of a false passage when inserting the tracheostomy tube is the most common complication. This should become evident by the absence of breath sounds and the development of subcutaneous emphysema. Pneumothorax can also occur. Injury to surrounding structures, such as the thyroid, parathyroids, esophagus, anterior jugular veins, and recurrent laryngeal nerves, can occur in situations of extreme urgency. Subglottic stenosis and granuloma formation are potential long-term complications.

A Commonly Used Formulas

Randall P. Scheri

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Cardiovascular Formulas

Formula	Normal range
Arterial carbon dioxide tension (PaCO ₂)	35–45 mm Hg
Arterial carbon dioxide content (CaCO ₂)	48–50 mL/dL
Arterial oxygen content (CaO ₂)	20 vol%
CaO ₂ = 1.36 [hemoglobin (Hgb)] (SaO ₂) + 0.003 (PaO ₂)	
Arterial oxygen saturation (SaO ₂)	93–98%
Arterial oxygen tension (PaO ₂)	70–100 mm Hg
Body-surface area (BSA)	1.73 m ² for 70-kg adult
Carbon dioxide production (VCO ₂)	2.3 mL/kg per min
Heart rate (HR)	60–100 beats per min
Maximum age-adjusted HR = 220 – age in yr	
Cardiac output (CO)	4–8 L per min
Fick equation: Equation	
Physiologic: CO = HR × SV	
Cardiac index (CI)	2.5–4.0 L per min
CI = CO/BSA	
Central venous pressure (CVP)	0–8 mm Hg
Fraction of inspired oxygen (FIO ₂)	0.21–1.0
Left atrial pressure (LAP)	3–12 mm Hg
Left ventricular ejection fraction (LVEF)	55–70%
Left ventricular end-diastolic volume (LVEDV)	70 mL/m ²
Left ventricular systolic pressure (LVS)	100–140 mm Hg
Mean arterial pressure (MAP)	70–105 mm Hg
MAP = [(SBP – DBP)/3] + DBP	
SBP = systolic blood pressure	
DBP = diastolic blood pressure	
Mixed venous carbon dioxide tension Equation	41–51 mm Hg
Mixed venous oxygen content Equation	15 vol%
Equation	
Mixed venous oxygen saturation Equation	75%
Mixed venous oxygen tension Equation	35–40 mm Hg
Oxygen consumption Equation	250 mL per min
Oxygen delivery (O ₂ del)	640–1,000 mL per min at rest
O ₂ del = CO × CaCO ₂	
Pulmonary artery pressure, mean (PAP)	5–10 mm Hg
Pulmonary artery diastolic pressure (PAD)	5–12 mm Hg
Pulmonary artery systolic pressure (PAS)	15–30 mm Hg
Pulmonary capillary wedge pressure (PCWP)	5–12 mm Hg
Pulmonary vascular resistance (PVR)	<3 dynes × sec/cm ³
PVR = (PAP – PCWP)/CO	
Pulse pressure (PP)	40 mm Hg
PP = SBP – DBP	
Right atrial pressure (RAP)	0–8 mm Hg
Right ventricular diastolic pressure (RVD)	0–8 mm Hg
Right ventricular systolic pressure (RVS)	15–28 mm Hg
Stroke volume (SV)	60–100 mL/beat
SV = CO/HR	
Stroke volume index (SVI)	33–47 mL/beat/m ²
SVI = 1,000 (CO)/(HR) (BSA)	
Systemic vascular resistance (SVR)	<20 dynes × sec/cm ³
SVR = (MAP – RAP)/CO	

Pulmonary Formulas

Formula	Normal range
Alveolar arterial oxygen gradient (P[A-a] O ₂)	3–16 mm Hg
Age correction: A-a gradient = 2.5 + age/4	
Alveolar oxygen tension (PAO ₂)	Room air: 100 mm Hg
PAO ₂ = 713 (FIO ₂) – (PaCO ₂ /0.8) at sea level	100%: 673 mm Hg
Alveolar carbon dioxide tension (PAO ₂)	40 mm Hg
Equation	
Alveolar ventilation (VA)	4 L per min
Equation	
Arterial blood gas format	
pH/PaCO ₂ /PaO ₂ /HCO ₃ ⁻ , FIO ₂	
pH	7.35–7.45
PaCO ₂	35–45 mm Hg
PaO ₂	70–100 mm Hg
HCO ₃ ⁻	24–30 mmol/L
FIO ₂	21% room air
Expiratory reserve volume (ERV)	25% VC
ERV = FRC – RV	
Forced expiratory volume in 1 sec (FEV ₁)	FEV ₁ = 83%
Forced expiratory volume in 2 sec (FEV ₂)	FEV ₂ = 94%

Functional residual capacity (FRC) FRC = ERV + RV	1.8–3.4 L
Henderson-Hasselbalch equation pH = pK + log (base/acid) H ₂ O + CO ₂ « H ₂ CO ₃ « HCO ₃ ⁻ + H ⁺ pH = 6.1 + log (HCO ₃ ⁻ /CO ₂)	pH 7.4
Inspiratory capacity (IC) IC = inspiratory reserve volume (IRV) + V _T	1.0–2.4 L
Minute ventilation (V _E) V _E = V _T × RR (respiratory rate)	90 mL/kg per min
Negative inspiratory force (NIF)	60–100 cm H ₂ O
Pulmonary blood flow (QT)	5 L per min
Pulmonary shunt blood volume (QS)	150 mL per min
Pulmonary shunt fraction (QS/QT)	2–3%
Residual volume (RV)	1.0–2.4 L
Tidal volume (V _T)	6–7 mL/kg
Total lung capacity (TLC) TLC = VC + RV	4–6 L
Vital capacity (VC) VC = IRV + ERV + V _T	3–5 L

Renal Formulas

Creatinine Clearance (Cl_{Cr})

[Equation](#)
[Equation](#)

Degree of Renal Impairment Indicated by Creatinine Clearance (Cl_{Cr})

Cl _{Cr} (mL per min)	Degree of renal impairment
>100	Normal
40–60	Mild
10–40	Moderate
<10	Severe

Fractional Excretion of Sodium (FeNa⁺)

$$\text{FeNa}^+ = (\text{urine Na}^+ \times \text{serum Cr}) / (\text{urine Cr} \times \text{serum Na}^+) \times 100\%$$

Fractional excretion of X [X = K⁺, blood urea nitrogen (BUN), amylase, and so forth] can be calculated by replacing Na⁺ with X in preceding formula.

Free Water Clearance [Equation](#)

Normal value = -20

[Equation](#)

where V = urine volume/time (mL/min), U_{osm} = measured urine osmolality, and P_{osm} = plasma osmolality.

Glomerular Filtration Rate (GFR)

GFR = ultrafiltrate/time

Normal GFR = 80 L per day

Daily Renal Solute and Water Exchange for a Normal Adult Man

	Filtered	Excreted	% Reabsorbed
Na ⁺ (mEq)	26,000	100–250	>99
Cl ⁻ (mEq)	21,000	100–250	>99
K ⁺ (mEq)	800	40–120	85–95
HCO ₃ ⁻ (mEq)	4,800	0	100
Glucose (mmol/L)	900	0	100
Urea (mmol/L)	900	400	44
Creatinine (mmol/L)	15	15	0
H ₂ O (L)	180	0.5–3.0	98–99

Urine Output (UO)

UO = 0.5–1.0 mL/kg/h

Oliguria = <500 mL/day

Metabolic and Fluid Formulas

	Normal range
Anion gap (AG) AG = Na ⁺ - (Cl ⁻ + HCO ₃ ⁻)	8–12 mEq/L
Plasma osmolality (calculated) Equation	270–300 mOsm/L
Sodium deficit = total body water × [140 - Na ⁺ (mmol)]	
Total body water (TBW)	Male, 0.6 × lean body wt (kg) Female, 0.5 × lean body wt (kg)
Extracellular fluid (ECF)	1/3 TBW
Interstitial fluid (ISF)	3/4 ECF

Plasma
 Whole blood
 Intracellular fluid (ICF)

$\frac{1}{4}$ ECF
 75 mL/kg
 $\frac{2}{3}$ TBW

Bodily Fluid Composition (mEq/L)

Fluid	Na ⁺	Cl ⁻	K ⁺	HCO ₃ ⁻ H ⁺	Volume (L/day)	
Saliva	30-60	15-40	20	15-50	—	0.5-1.5
Gastric	60-100	90-140	10	—	30-100	1.0-4.0
Duodenum	140	80	550	—	—	1.0-3.0
Bile	140	100	550	—	—	0.5-1.5
Pancreas	140	75	590	—	—	1.0
Jejunum	100	100	510	—	—	1.0-2.0
Ileum	130	110	1030	—	—	1.0-2.0
Colon	60	40	3020	—	—	—

Composition of Commonly Used Intravenous Fluids (mEq/L)

Fluid	Na ⁺	Cl ⁻	K ⁺	Ca ²⁺	Base	Glucose	pH	mOsm/L
0.9% normal saline	154	154	—	—	—	—	—	5.0292
5% dextrose in normal saline	154	154	—	—	—	50	—	5.0565
Lactated Ringer's solution	130	109	4	28	—	—	—	6.5277
D5W	—	—	—	—	—	50	—	4.0274
5% albumin	154	154	—	—	—	—	—	7.0-300
Hetastarch	154	154	—	—	—	60	—	5.5310

Nutrition Formulas

Actual Energy Expenditure (AAE)

AAE = basal energy expenditure (BEE) × [metabolic activity factor (MAF) + 1.0]

Basal Energy Expenditure in kcal/day (Harris-Benedict Equation)

Men: kcal/24 hr = 66 + [13.7 × wt (kg)] + [5 × ht (cm)] - (6.8 × age)

Women: kcal/24 hr = 655 + [9.6 × wt (kg)] + [1.8 × ht (cm)] - (4.7 × age)

Estimate: 25-30 kcal/kg/day

Calorie Conversion

Substrate	kcal
Carbohydrate	1 g = 3.4
Fat, long chain	1 g = 9.0
Fat, medium chain	1 g = 7.1
Protein	1 g = 4.0

Ideal Body Weight (Estimate)

Men: 50 kg for 5 ft + 2.3 kg for every 1 in. thereafter

Women: 45.5 kg for 5 ft + 2.3 kg every 1 in. thereafter

Ideal calorie-nitrogen ratio = 135-200:1

Metabolic Activity Factor

Activity factor + injury factor + fever factor + growth factor

Condition	Activity factor
Bedrest	0.2
Moderately active	0.35
Active	0.5

Condition	Injury factor
Minor surgery	0.2
Major surgery	0.35
Major burn	1.0

Condition	Growth factor
Moderate weight loss	0.05
Severe weight loss	0.13

Fever factor: 0.13°C above 37°C

Protein Requirement and Nitrogen Balance

Must be adjusted for renal insufficiency.

Nitrogen (N₂): 0.8-2.0 g/kg/24 hr

6.25 g protein = 1 g

N_2 balance = protein uptake (g)/6.25 – [UUN + 4 g (insensible loss)]

Urinary urea nitrogen (UUN) = collected urinary N_2 in 24 hr

Respiratory Quotient

Equation

Oxidized substrate	Value
Pure fat	0.7
Pure protein	0.8
Pure carbohydrate	1.0
Fat synthesis	1.3

Unit Conversions

Length

1 in. = 2.54 cm
1 cm = 0.3973 in.

Pressure

1 mm Hg = 0.735 cm H_2O
1 cm H_2O = 1.36 mm Hg

Temperature

$^{\circ}F = 9/5 ^{\circ}C + 32$
 $^{\circ}C = 5/9 (^{\circ}F - 32)$

Weight

1 lb = 0.454 kg
1 kg = 2.204 lb

Volume

1 qt = 0.943 L
1 L = 1.06 qt

Selected Drug Relative Potencies

Bumetanide: 1 mg = furosemide, 20–40 mg

Methylprednisolone: 4 mg = prednisone, 5 mg = hydrocortisone, 20 mg = cortisone

Reference Values and Differential

	Current units	SI units
Complete blood cell count		
Erythrocyte sedimentation rate	0–30 mm per hr	0–30 mm per hr
Hematocrit		
Male	40.7–50.3%	0.407–0.503
Female	36.1–44.3%	0.361–0.443
Hemoglobin		
Male	13.8–17.2 g/dL	8.56–10.70 mmol/L
Female	12.1–15.1 g/dL	7.50–9.36 mmol/L
Leukocytes	4–10 × 10 ³ /μL	4–10 × 10 ⁹ /L
Neutrophils	1.8–6.6 × 10 ³ /μL	1.8–6.6 × 10 ⁹ /L
Lymphocytes	1.2–3.3 × 10 ³ /μL	1.2–3.3 × 10 ⁹ /L
Monocytes	0.2–0.7 × 10 ³ /μL	0.2–0.7 × 10 ⁹ /L
Eosinophils	0.0–0.5 × 10 ³ /μL	0.0–0.5 × 10 ⁹ /L
Basophils	0.0–0.2 × 10 ³ /μL	0.0–0.2 × 10 ⁹ /L
Mean corpuscular volume	80.0–97.6 μm ³	80.0–97.6 fl
Platelet count	190–405 × 10 ³ /μL	190–405 × 10 ⁹ /L
Reticulocyte count	0.5–1.5%	0.005–0.015
Coagulation profile		
Activated partial thromboplastin time	25–36 sec	25–36 sec
Bleeding time	2.5–9.5 min	150–570 sec
Fibrinogen	150–360 mg/dL	1.5–3.6 g/L
Fibrin degradation products	<8 μg/mL	<8 mg/L
Prothrombin time	11–14 sec	11–14 sec
Thrombin time	11.3–18.5 sec	11.3–18.5 sec
Basic metabolic panel		
Carbon dioxide	22–31 mmol/L	22–31 mmol/L
Chloride	97–110 mmol/L	97–110 mmol/L
Creatinine	0.7–1.4 mg/dL	62–124 μmol/L
Glucose	65–110 mg/dL	3.58–6.05 mmol/L
Potassium	3.3–4.9 mmol/L	3.3–4.9 mmol/L
Sodium	135–145 mmol/L	135–145 mmol/L
Urea nitrogen	8–25 mg/dL	2.9–8.9 mmol/L
Complete metabolic panel		
Albumin	3.6–5.0 g/dL	36–50 g/L
Aspartate aminotransferase	11–47 IU/L	0.18–0.78 μkat/L
Bilirubin, total	0.2–1.3 mg/dL	3.4–22.2 μmol/L
Calcium	8.9–10.3 mg/dL	2.23–2.57 mmol/L
Chloride	97–110 mmol/L	97–110 mmol/L
Creatinine	0.7–1.4 mg/dL	62–124 μmol/L
Glucose	65–110 mg/dL	3.58–6.05 mmol/L
Phosphatase, alkaline	38–126 IU/L	0.63–2.10 μkat/L
Potassium	3.3–4.9 mmol/L	3.3–4.9 mmol/L
Protein, total	6.2–8.2 mg/dL	62–82 g/L
Sodium	135–145 mmol/L	135–145 mmol/L
Urea nitrogen	8–25 mg/dL	2.9–8.9 mmol/L
Enzymatic activities and chemistries, other		
Alanine aminotransferase	7–53 IU/L	0.12–0.88 μkat/L
Ammonia	19–43 μmol/L	19–43 μmol/L
Amylase	35–118 IU/L	0.58–1.97 μkat/L
Bilirubin, direct (conjugated)	0.0–0.2 mg/dL	0.0–3.4 μmol/L
Calcium, ionized	4.6–5.1 mg/dL	1.15–1.27 mmol/L
Carboxyhemoglobin		
Nonsmoker	0.0–1.5%	
Moderate smoker	1–5%	
Heavy smoker	5–9%	
Ceruloplasmin	21–53 mg/dL	210–530 mg/L
Cholesterol		
High	>240 mg/dL	>6.20 mmol/L
Borderline	200–239 mg/dL	5.18–6.19 mmol/L
Desirable	<200 mg/dL	<5.18 mmol/L
Copper	11.0–24.3 μmol/L	11.0–24.3 μmol/L
Creatine kinase		
Male	30–220 IU/L	0.50–3.67 μkat/L
Female	20–170 IU/L	0.33–2.83 μkat/L
MB fraction	0–12 IU/L	0.00–0.20 μkat/L
Ferritin		
Male	36–262 ng/mL	81–590 pmol/L
Female	10–155 ng/mL	23–349 pmol/L
Folate, plasma	3.9–28.6 nmol/L	3.9–28.6 nmol/L
Gamma-glutamyl transpeptidase		
Male	20–76 IU/L	0.33–1.27 μkat/L
Female	12–54 IU/L	0.2–0.9 μkat/L
Glycated hemoglobin	4.4–6.3%	0.044–0.063
Haptoglobin	44–303 mg/dL	0.44–3.03 g/L
High-density lipoprotein	27–98 mg/dL	0.70–2.54 mmol/L
Iron	50–175 g/dL	9.0–31.3 μmol/L
Binding capacity	250–450 μg/dL	44.8–80.6 μmol/L
Transferrin saturation	20–50%	0.20–0.50
Lactate, plasma	0.3–1.3 mmol/L	0.3–1.3 mmol/L

Lactate dehydrogenase (LDH)	90–280 IU/L	1.50–4.67 μ kat/L
Lipase	2.3–20.0 IU/L	0.38–3.33 μ kat/L
Low-density lipoprotein		
High	>160 mg/dL	>4.144 mmol/L
Borderline	130–159 mg/dL	3.367–4.144 mmol/L
Desirable	<130 mg/dL	<3.367 mmol/L
Magnesium	1.3–2.2 mEq/L	0.65–1.10 mmol/L
5'-Nucleotidase	2–16 IU/L	0.03–0.27 μ kat/L
Osmolality, serum	270–290 mOsm/L	270–290 mmol/kg
Phosphatase, acid	0.0–0.7 IU/L	0.0–11.6 nkat/L
Phosphorus	2.3–4.3 mg/dL	0.74–1.39 mmol/L
Triglycerides, fasting	<200 mg/dL	<2.83 mmol/L
Uric acid	3.0–8.0 mg/dL	179–476 μ mol/L
Vitamin B ₁₂	200–800 pg/mL	148–590 pmol/L
Selected serum hormones and tumor markers		
Adrenocorticotrophic hormone, fasting, a.m.	<60 pg/mL	<13.2 pmol/L
Aldosterone	10–160 ng/mL	28–443 mmol/L
Alpha-fetoprotein	0.0–8.9 ng/mL	0.0–8.9 μ g/L
Beta-human chorionic gonadotropin		
Nonpregnant	0.0–5.0 mIU/mL	0.0–5.0 IU/L
Pregnant	5–200 \times 10 ³ mIU/mL	5–200 \times 10 ³ IU/L
Carcinoembryonic antigen (CEA)		
Nonsmoker	0.0–3.0 ng/mL	0.0–3.0 μ g/L
Smoker	0.0–5.0 ng/mL	0.0–5.0 μ g/L
Cortisol, a.m.	8–25 μ g/dL	0.22–0.68 μ mol/L
Follicle-stimulating hormone		
Male	2.4–19.9 IU/L	2.4–19.9 IU/L
Female		
Follicular	3.1–19.7 IU/L	3.1–19.7 IU/L
Luteal	1.7–11.2 IU/L	1.7–11.2 IU/L
Midcycle	10.4–23.1 IU/L	10.4–23.1 IU/L
Postmenopausal	18–126 IU/L	18–126 IU/L
Gastrin, fasting	0–130 pg/mL	0–130 ng/L
Growth hormone, fasting	<8 ng/mL	<8 g/L
17-Hydroxyprogesterone		
Prepubertal		
Male adult	3–90 ng/dL	0.1–2.7 nmol/L
Female adult	27–199 ng/dL	0.8–6.0 nmol/L
Follicular	15–70 ng/dL	0.5–2.1 nmol/L
Luteal	35–290 ng/dL	1.1–8.8 nmol/L
Insulin, fasting	5–25 mU/L	36–180 pmol/L
Luteinizing hormone		
Male	0.0–8.9 IU/L	0.0–8.9 IU/L
Female		
Follicular	1.4–11.5 IU/L	1.4–11.5 IU/L
Luteal	0.1–16.1 IU/L	0.1–16.1 IU/L
Midcycle	20.1–73.9 IU/L	20.1–73.9 IU/L
Postmenopausal	8.4–46.5 IU/L	8.4–46.5 IU/L
Parathyroid hormone	12–72 pg/mL	12–72 ng/L
Progesterone		
Male	<0.5 ng/mL	<1.6 nmol/L
Female		
Follicular	0.1–1.5 ng/mL	0.32–4.80 nmol/L
Luteal	2.5–28.0 ng/mL	8–89 nmol/L
First trimester	9–47 ng/mL	29–149 nmol/L
Third trimester	55–255 ng/mL	175–811 nmol/L
Postmenopausal	<0.5 ng/mL	<1.6 nmol/L
Prolactin		
Male	2–12 ng/mL	2–12 μ g/L
Female	2–20 ng/mL	2–20 μ g/L
Prostate-specific antigen	0.0–4.0 ng/mL	0.0–4.0 ng/mL
Renin activity, plasma	0.9–3.3 ng/mL per hr	0.90–0.33 μ g/L per hr
Testosterone, free		
Male	52–280 pg/mL	150–970 pmol/L
Female	1.1–6.3 pg/mL	4–22 pmol/L
Testosterone, total		
Male	350–1,030 ng/dL	12.1–35.6 nmol/L
Female	10–55 ng/dL	0.4–1.9 nmol/L
Thyroid-stimulating hormone	0.45–6.20 μ U/mL	0.45–6.20 mU/L
Thyroxine, free	1.0–2.3 ng/dL	12.3–29.7 pmol/L
Thyroxine, total (T ₄)	3.0–12.0 μ g/dL	38.7–155.0 nmol/L
Triiodothyronine	80–200 ng/dL	1.2–3.1 nmol/L
T ₄ index	0.85–3.50	0.85–3.50
Tumor uptake	20–40%	0.2–0.4
Vitamin D, 1,25-hydroxy	20–76 pg/mL	48–182 pmol/L
Vitamin D, 25-hydroxy	10–55 ng/mL	25–137 nmol/L
Immunology		
Complement, total	118–226 U/mL	118–226 U/mL
C3	77–156 mg/dL	0.77–1.56 g/L
C4	15–39 mg/dL	0.15–0.39 g/L
Immunoglobulin (Ig)		
IgA	91–518 mg/dL	0.91–5.18 g/L
IgG	805–1,830 mg/dL	8.05–18.30 g/L
IgM	61–355 mg/dL	0.61–3.55 g/L
Urinalysis		
Macroscopic		
Bilirubin (qualitative)	Negative	
Glucose (qualitative)	Negative	
Ketones (qualitative)	Negative	
Occult blood (qualitative)	Negative	
pH	4.5–8.0	4.5–8.0
Protein	0–150 mg per day	0.000–0.150 g per day
Specific gravity	1.003–1.040 g/mL	1.003–1.040 g/mL

Urobilinogen	0.1–1.0 mg/dL	0.1–1.0 mg/dL
Microscopic		
Casts	None	
Bacteria	0–1+	
Red blood cells (RBCs)	0–3/high-power field	
White blood cells (WBCs)	0–5/high-power field	
Chemistries		
Amylase	0.04–0.30 IU per min	0.67–5.00 nkat per min
Calcium	0–250 mg per day	0.00–6.25 mmol per day
Copper	15–50 µg per day	0.24–0.78 µmol per day
Coproporphyrin	0–72 µg per day	0–110 nmol per day
Cortisol, free	20–90 µg per day	55–248 nmol per day
Creatinine		
Male	1.0–2.0 g per day	8.8–17.7 mmol per day
Female	0.6–1.5 g per day	5.3–13.3 mmol per day
Dopamine	100–440 µg per day	653–2,900 nmol per day
Delta-amino-levulinic acid	1.3–7.0 mg per day	9.9–53.0 µmol per day
Epinephrine	<15 µg per day	<82 nmol per day
5-Hydroxyindoleacetic acid	<9 mg per day	<47 µmol per day
Hydroxyproline, total	25–77 mg per day	191–588 µmol per day
Metanephrine	<0.9 mg per day	<4.9 µmol per day
Norepinephrine	11–86 µg per day	0.24–0.78 µmol per day
Oxalate		
Male	7–44 mg per day	80–502 µmol per day
Female	4–31 mg per day	46–353 µmol per day
Protein	0–150 mg per day	0.000–0.150 g per day
Uroporphyrin	0–27 µg per day	0–32 nmol per day
Vanillylmandelic acid	2–10 mg per day	10–51 µmol per day
Therapeutic drug levels (serum)		
Amikacin		
Trough	5–10 mg/L	8.6–17.0 µmol/L
Peak	20–30 mg/L	34–51 µmol/L
Amitriptyline	100–250 µg/L	361–903 nmol/L
Carbamazepine	4–10 mg/L	17–42 µmol/L
Clonazepam	10–80 µg/L	32–254 nmol/L
Cyclosporin A	125–300 ng/mL	125–300 ng/mL
Desipramine	100–300 µg/L	375–1,125 nmol/L
Digoxin	0.5–2.0 mg/L	0.6–2.6 nmol/L
Disopyramide	2–5 mg/L	6–15 µmol/L
Ethosuximide	40–100 mg/L	283–708 µmol/L
5-Fluorocytosine		
Trough	20–60 mg/L	20–60 µg/mL
Peak	50–100 mg/L	50–100 µg/mL
Gentamicin		
Trough	<2 mg/L	<4.2 µmol/L
Peak	4–8 mg/L	8.4–16.7 µmol/L
Imipramine	150–300 µg/L	536–1,071 nmol/L
Ketoconazole		
Trough	£1 mg/L	
Peak	1–4 mg/L	
Lithium	0.6–1.2 mmol/L	0.6–1.2 mmol/L
N-Acetyl procainamide	6–20 mg/L	25.4–84.6 µmol/L
Nortriptyline	50–175 µg/L	190–665 nmol/L
Phenobarbital	10–40 mg/L	43–172 µmol/L
Phenytoin	10–20 mg/L	40–79 µmol/L
Primidone	5–15 mg/L	23–69 µmol/L
Procainamide	4–8 mg/L	17–34 µmol/L
Quinidine	1.7–6.1 mg/L	5.2–18.8 µmol/L
Salicylate	20–290 mg/L	0.14–2.10 mmol/L
Sulfamethoxazole		
Trough	75–120 mg/L	
Peak	100–150 mg/L	
Theophylline	10–20 mg/L	55–110 µmol/L
Tobramycin		
Trough	0.5–1.5 mg/L	1.1–3.2 µmol/L
Peak	6–8 mg/L	12.8–17.0 µmol/L
Trimethoprim		
Trough	2–8 mg/L	
Peak	5–15 mg/L	
Valproic acid	50–100 mg/L	346–693 µmol/L
Vancomycin		
Trough	5–10 mg/L	
Peak	20–35 mg/L	

Selected Bodily Fluid Normal Chemistries and Cellular Contents

Ascitic fluid	Transudate (SI)	Exudate (SI)
Amylase	—	Elevated
CEA	—	>10 ng/mL (>10 µg/mL)
CEA (ascites)/CEA (serum)	—	>2
Glucose	—	<60 mg/dL
Glucose (ascites)/glucose (serum)	<1	>1
LDH	—	Elevated
LDH (ascites)/LDH (serum)	—	>0.6
Lipase	—	Elevated
Protein	<3.0 g/dL (<30 g/L)	>3.0 g/dL (>30 g/L)
Protein (ascites)/protein (serum)	—	> 0.5
RBCs	—	Elevated
Triglycerides	—	Elevated
WBCs	<300–500/µL (<0.3–0.5 × 10 ⁹ /L)	>500/µL (>0.5 × 10 ⁹ /L)

Normal values

Cerebrospinal fluid

Current units

SI units

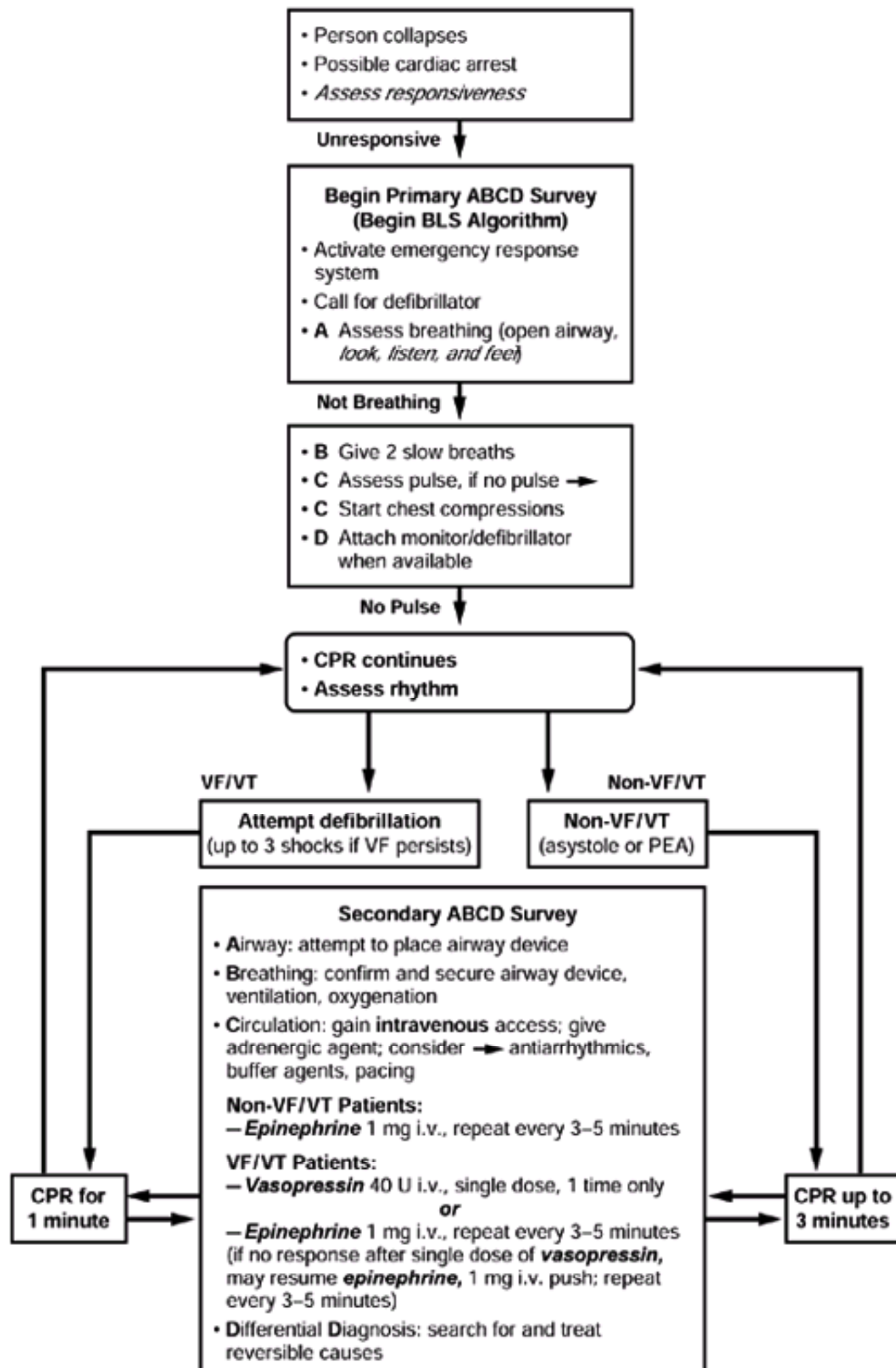
Glucose	45–80 mg/dL	2.5–4.4 mmol/L
Pressure	70–180 mm H ₂ O	70–180 mm H ₂ O
Protein	15–45 mg/dL	150–450 mg/L
Total WBCs	0–10/μL	0–1 × 10 ⁶ /L
Pleural fluid	Transudate (SI)	Exudate (SI)
Amylase	—	Twice serum amylase
Glucose	—	0–60 mg/dL (0.0–3.3 mmol/L)
LDH	<200 IU (<3.33 μkat/L)	>200 IU (>3.33 μkat/L)
LDH (pleural)/LDH (serum)	<0.6	>0.6
pH	7.4	<7.4
Protein	<3.0 g/dL (<30 g/L)	>3.0 g/dL (>30 g/L)
Protein (pleural)/protein (serum)	<0.5	>0.5
RBCs	<10,000/μL (<10 × 10 ⁹ /L)	>10,000/μL (>10 × 10 ⁹ /L)
Specific gravity	<1.016	>1.016
WBCs	<1,000/μL (<1 × 10 ⁹ /L)	>1,000/μL (>1 × 10 ⁹ /L)

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Gerard M. Doherty, Jennifer K. Lowney, John E. Mason, Scott I. Reznik, Michael A. Smith
The Washington Manual of Surgery

$$\text{TLC} = \frac{\% \text{ lymphocytes} \times \text{WBC}}{100}$$

$$\text{CHI} = \frac{\text{actual 24-hour creatinine excretion}}{\text{expected creatinine excretion}}$$

$$N_2 \text{ balance} = N_2 \text{ intake} - N_2 \text{ losses}$$



Primary ABCD survey**(Begin basic life support algorithm)**

Activate emergency response system.

Call for defibrillator.

A Airway: Open airway; assess breathing (open airway, look, listen, feel).

B Breathing: Give two slow breaths.

C Cardiopulmonary resuscitation: Check pulse; if no pulse →

C Start chest compressions.

D Defibrillator: Attach automatic external defibrillator or monitor/defibrillator when available.

Secondary ABCD survey

A Intubate as soon as possible.

B Confirm tube placement; use two methods to confirm.

Primary physical examination criteria *plus*

Secondary confirmation device (qualitative and quantitative measures of end-tidal carbon dioxide)

B Secure tracheal tube.

Prevent dislodgment; purpose-made tracheal tube holders are recommended over tie-and-tape approaches.

If the patient is at risk for transport movement, cervical collar and backboard are recommended.

B Confirm initial oxygenation and ventilation.

End-tidal carbon dioxide monitor

Oxygen saturation monitor

C Oxygen, i.v., monitor, fluids → rhythm-appropriate medications

C Vital signs: temperature, blood pressure, heart rate, respirations

D Differential diagnoses

Adapted from A guide to the International ACLS algorithms. *Circulation* 2000;102[Suppl 1]:I-142-I-157.

Primary ABCD Survey
Focus: basic CPR and defibrillation

- **Check** responsiveness
- **Activate** emergency response system
- **Call** for defibrillator

A Airway: open the airway
B Breathing: provide positive-pressure ventilations
C Circulation: give chest compressions
D Defibrillation: assess for and shock VF/pulseless VT, up to 3 times (200 J, 200–300 J, 360 J, or equivalent *biphasic*) if necessary

Rhythm after first 3 shocks?

Persistent or recurrent VF/VT

Secondary ABCD Survey
Focus: more advanced assessments and treatments

- A Airway:** place airway device as soon as possible
- B Breathing:** confirm airway device placement by exam plus confirmation device
- B Breathing:** secure airway device; purpose-made tube holders preferred
- B Breathing:** confirm effective oxygenation and ventilation
- C Circulation:** establish i.v. access
- C Circulation:** identify rhythm → monitor
- C Circulation:** administer drugs appropriate for rhythm and condition
- D Differential Diagnosis:** search for and treat identified reversible causes

- **Epinephrine**, 1 mg i.v. push, repeat every 3–5 minutes
- **Vasopressin**, 40 U i.v., **single dose**, 1 time only

Resume attempts to defibrillate
 1 x 360 J (or equivalent *biphasic*) within 30–60 seconds

Consider antiarrhythmics:
amiodarone (IIb), *lidocaine* (Indeterminate)
magnesium (IIb if hypomagnesemic state),
procainamide (IIb for intermittent/recurrent VF/VT)
Consider buffers

Resume attempts to defibrillate

Pulseless Electrical Activity
(PEA = rhythm on monitor, without detectable pulse)

Primary ABCD Survey
Focus: basic CPR and defibrillation

- **Check** responsiveness
- **Activate** emergency response system
- **Call** for defibrillator

A Airway: open the airway

B Breathing: provide positive-pressure ventilations

C Circulation: give chest compressions

D Defibrillation: assess for and shock VF/pulseless VT

Secondary ABCD Survey
Focus: more advanced assessments and treatments

A Airway: place airway device as soon as possible

B Breathing: confirm airway device placement by exam plus confirmation device

B Breathing: secure airway device; purpose-made tube holders preferred

B Breathing: confirm effective oxygenation and ventilation

C Circulation: establish i.v. access

C Circulation: identify rhythm → monitor

C Circulation: administer drugs appropriate for rhythm and condition

C Circulation: assess for occult blood flow ("pseudo-EMT")

D Differential Diagnosis: search for and treat identified reversible causes

Review for most frequent causes

• Hypovolemia	• "Tablets" (drug OD, accidents)
• Hypoxia	• Tamponade, cardiac
• Hydrogen ion — acidosis	• Tension pneumothorax
• Hyper-/hypokalemia	• Thrombosis, coronary (ACS)
• Hypothermia	• Thrombosis, pulmonary (embolism)

Epinephrine, 1 mg i.v. push, repeat every 3–5 minutes

Atropine, 1 mg i.v. (if PEA rate is **slow**), repeat every 3–5 minutes as needed, to a total dose of 0.04 mg/kg

Asystole

Primary ABCD Survey
Focus: basic CPR and defibrillation

- **Check** responsiveness
 - **Activate** emergency response system
 - **Call** for defibrillator
- A Airway:** open the airway
B Breathing: provide positive-pressure ventilations
C Circulation: give chest compressions
C Confirm true asystole
D Defibrillation: assess for VF/pulseless VT; shock if indicated
- Rapid scene survey:** any evidence personnel should **not** attempt resuscitation?

Secondary ABCD Survey
Focus: more advanced assessments and treatments

- A Airway:** place airway device as soon as possible
B Breathing: confirm airway device placement by exam plus confirmation device
B Breathing: secure airway device; purpose-made tube holders preferred
B Breathing: confirm effective oxygenation and ventilation
C Circulation: confirm true asystole
C Circulation: establish i.v. access
C Circulation: identify rhythm → monitor
C Circulation: give medications appropriate for rhythm and condition
D Differential Diagnosis: search for and treat identified reversible causes

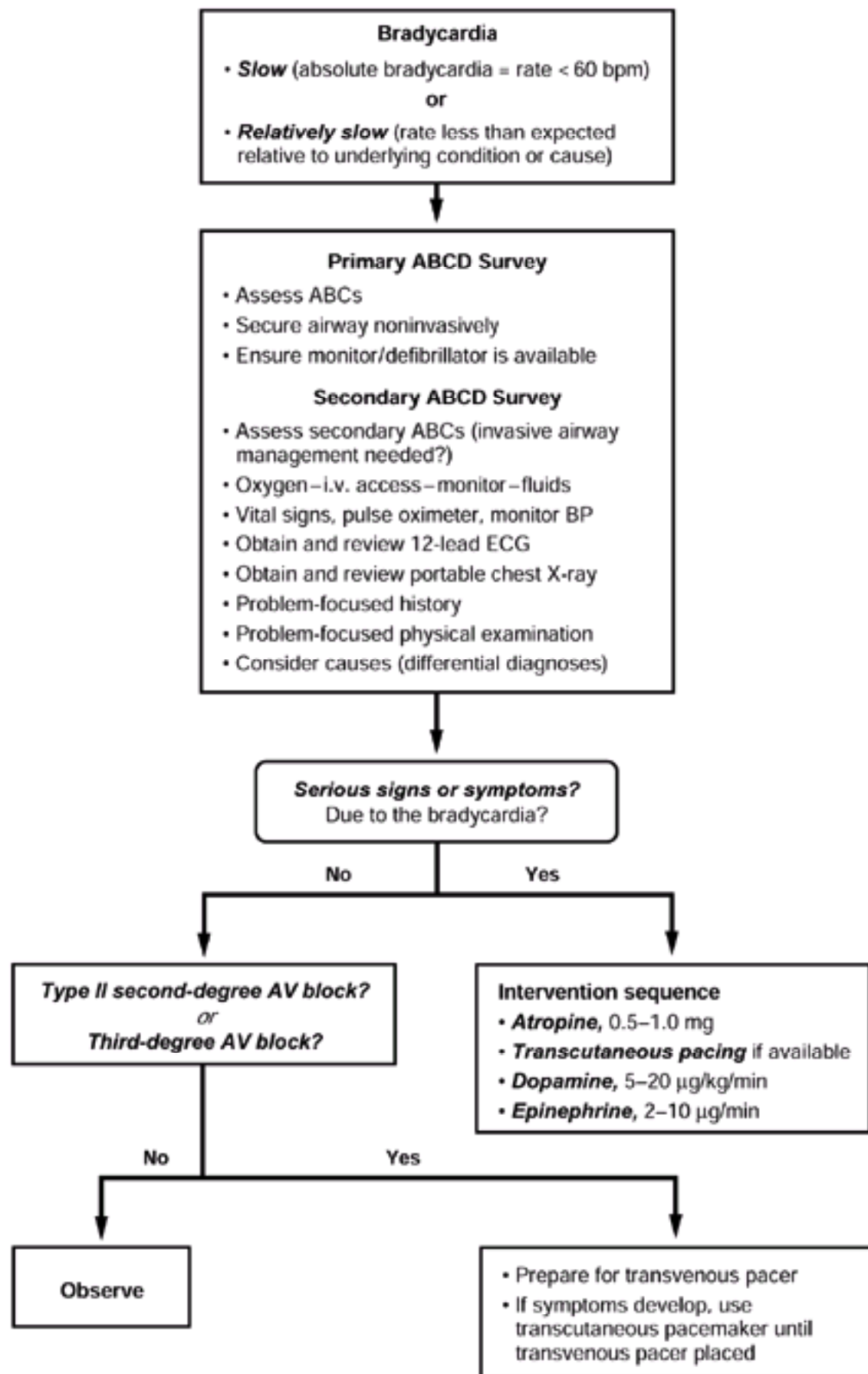
Transcutaneous pacing
If considered, perform immediately

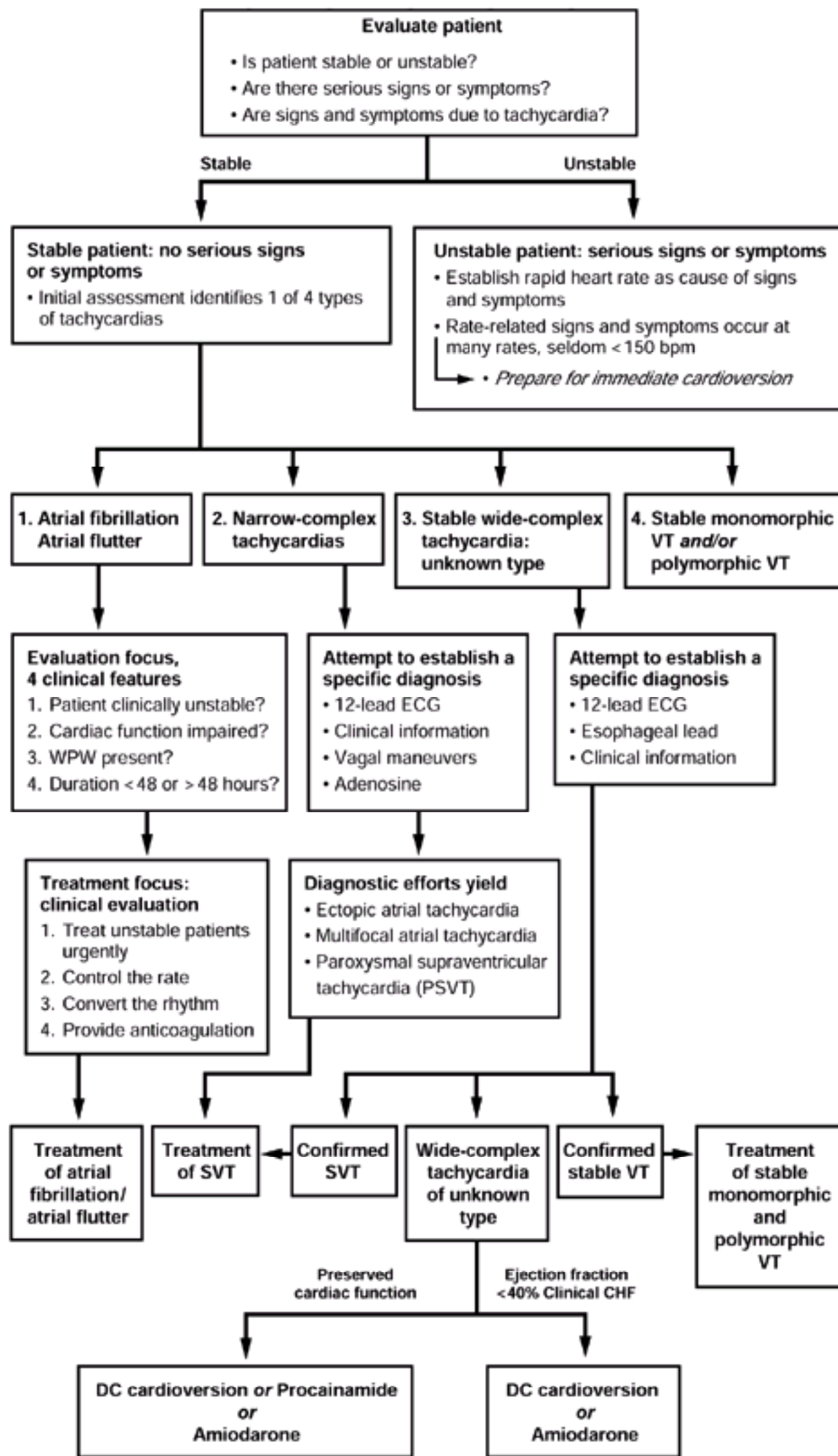
Epinephrine, 1 mg i.v. push, repeat every 3–5 minutes

Atropine, 1 mg i.v., repeat every 3–5 minutes
up to a total of 0.04 mg/kg

Asystole persists
Withhold or cease resuscitation efforts?

- Consider quality of resuscitation?
- Atypical clinical features present?
- Support for cease-efforts protocols in place?





$$P_{\text{osm}} (\text{mOsm/kg}) = 2[Na^+ (\text{mmol/L}) + K^+ (\text{mmol/L})] + \left[\frac{\text{blood urea nitrogen (mg/dL)}}{2.8} \right] + \left[\frac{\text{glucose (mg/dL)}}{18} \right]$$

Na^+ deficit (mmol) = $0.60 \times \text{lean body weight (kg)} \times$
 $[120 - \text{measured serum Na}^+ \text{ (mmol/L)}]$

Water deficit (L) = 0.60 × total body

$$\left[\left(\frac{\text{serum Na}^+ [\text{mmol/L}]}{140} \right) - 1 \right]$$

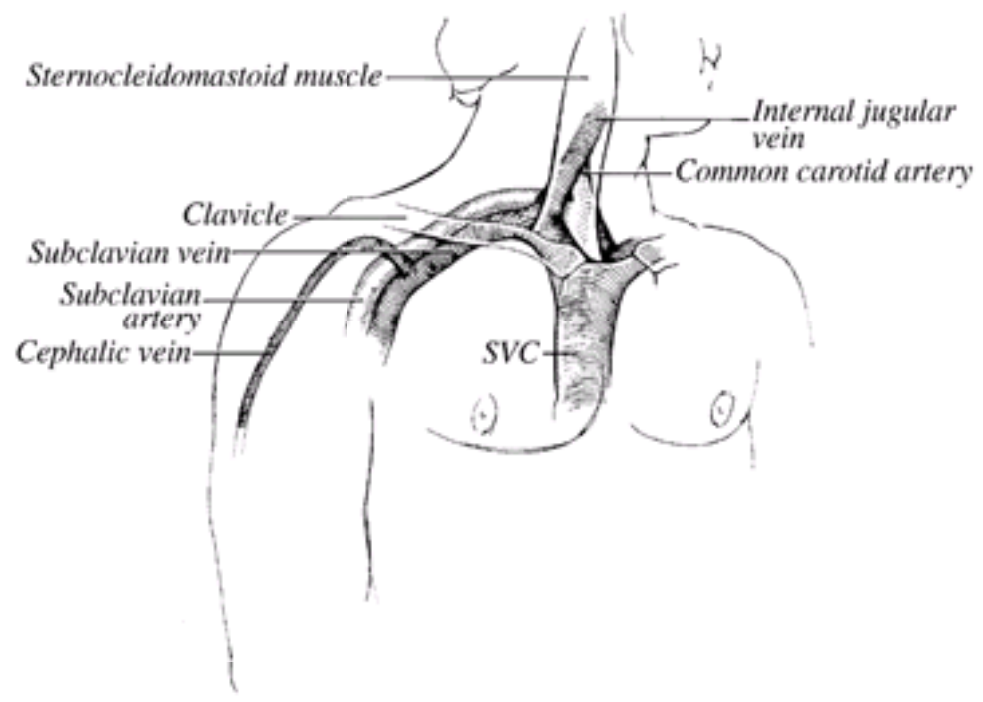
$$\text{AG (mmol/L)} = \text{Na}^+ \text{ (mmol/L)} - [\text{Cl} \text{ (mmol/L)} + \text{HCO}_3^- \text{ (mmol/L)}]$$

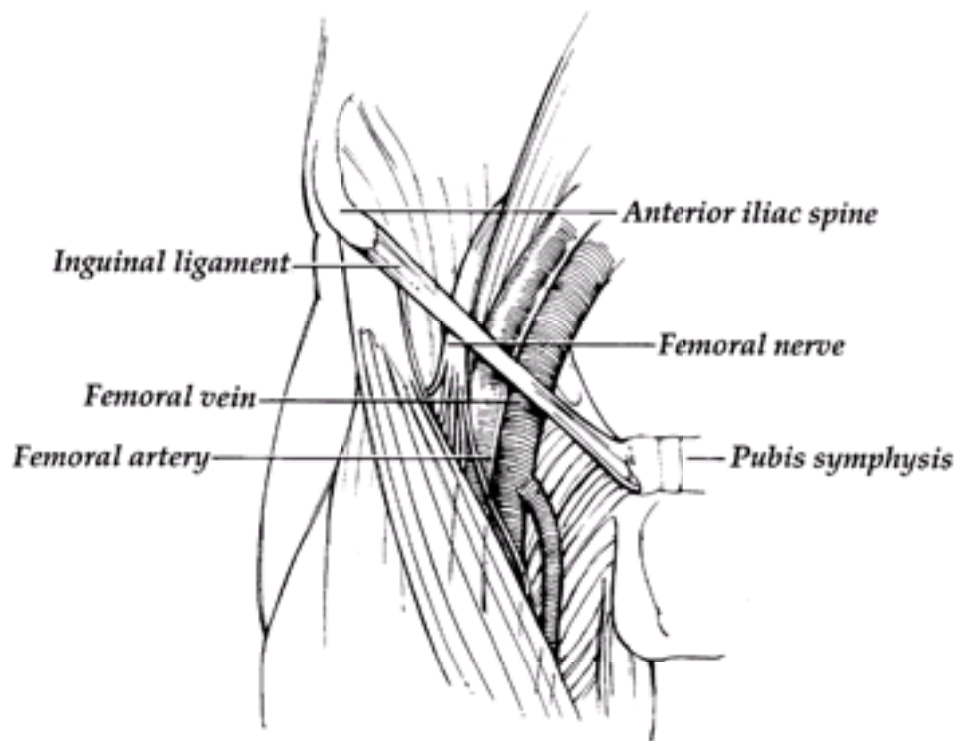
$$\text{HCO}_3^- \text{ deficit (mmol/L)} = \text{body weight (kg)} \times 0.4 \times$$
$$[(\text{desired HCO}_3^- \text{ [mmol/L]}) - (\text{measured HCO}_3^- \text{ [mmol/L]})]$$

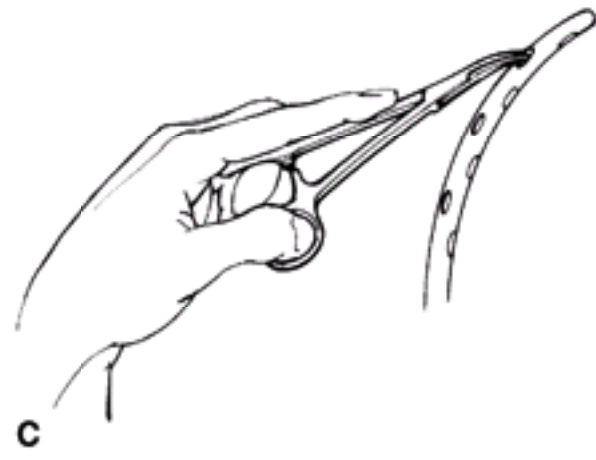
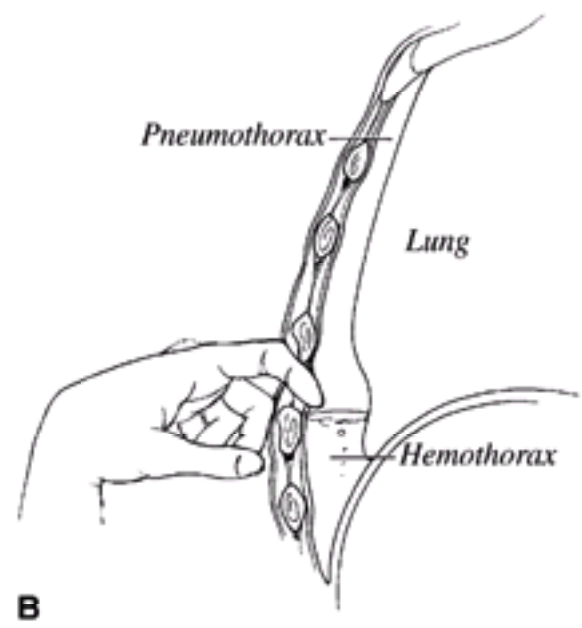
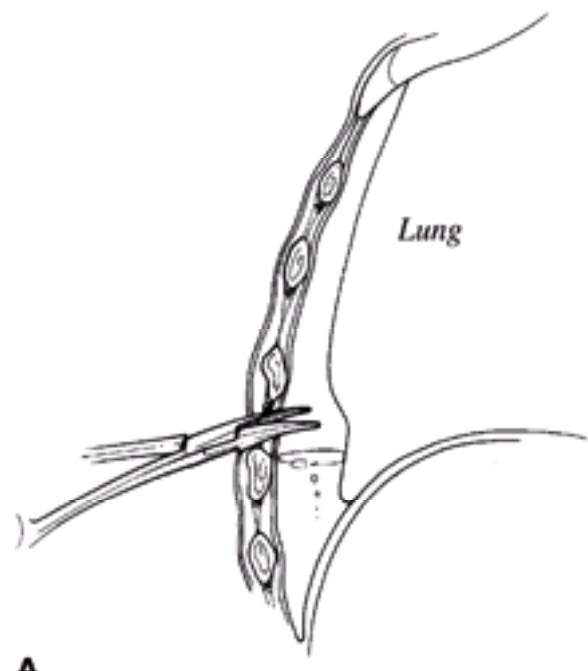
$$\text{NH}_4\text{Cl (mmol)} = 0.2 \times \text{weight (kg)} \times [103 - \text{serum Cl}^- \text{ (mmol)}]$$

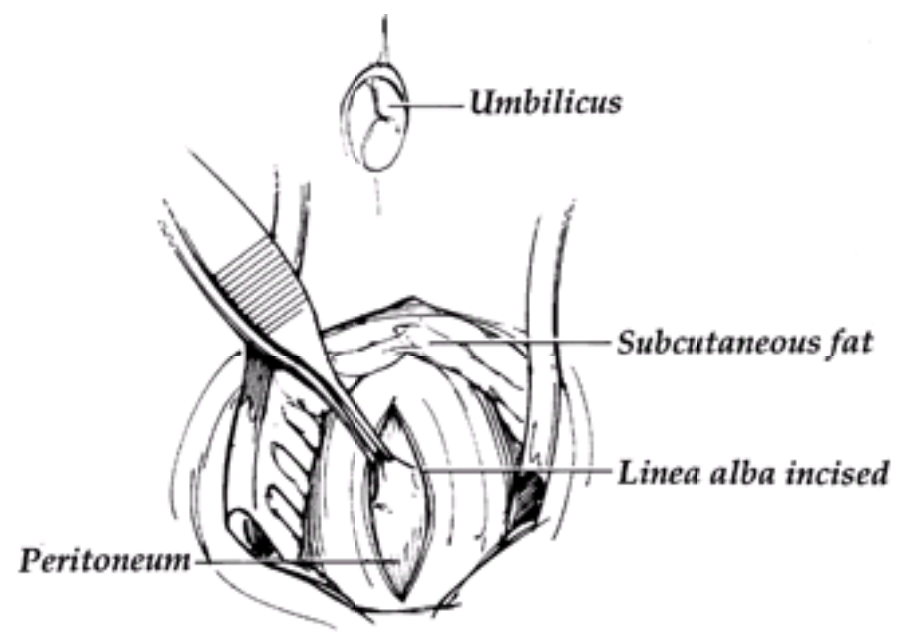
$$\text{H}^+ \text{ (mmol)} = 0.5 \times \text{weight (kg)} \times [103 - \text{serum Cl}^- \text{ (mmol/L)}]$$

$$Fe_{Na} = (U_{Na}/P_{Na})/(U_{Cr}/P_{Cr})$$



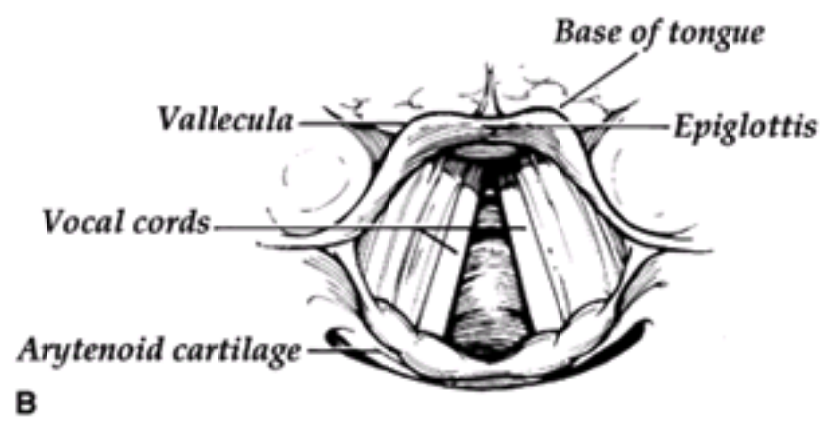




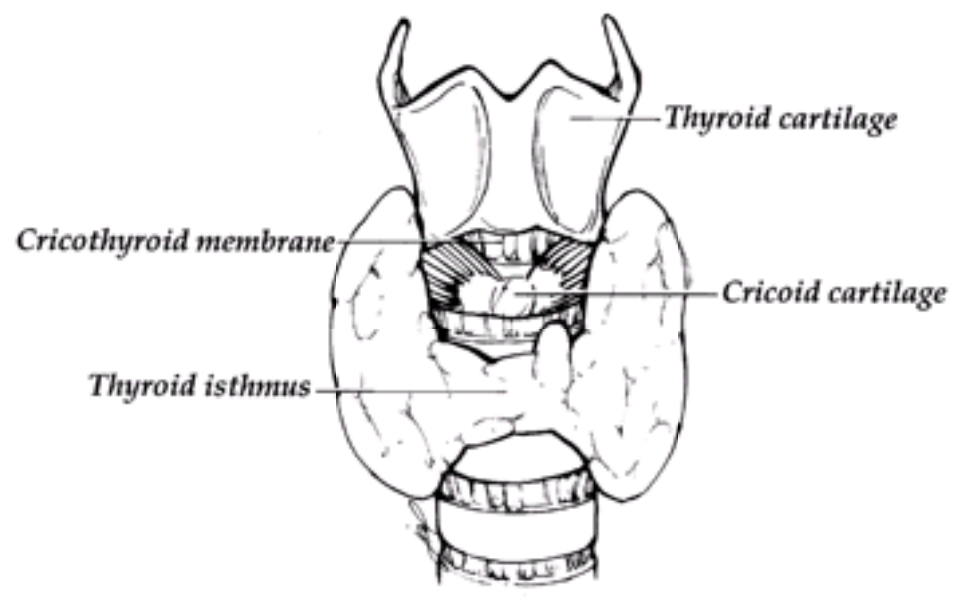




A



B



$\dot{V}O_2/8.5$ ($CaO_2 - CVO_2$)

(P_vCO₂)

(CVO₂)

$$CvO_2 = 1.36 (\text{Hgb}) (SvO_2) + 0.003 (MvO_2)$$

(SVO₂)

(MVO₂)

($\dot{V}O_2$)

$$P_{ACO_2} = \dot{V}_{CO_2} \times 0.863 / VA$$

$$VA = 0.863 (\dot{V}CO_2)/Paco_2$$

$$Cl_{Cr} \text{ (mL/min)} = \frac{\text{urine Cr (mg/dL)} \times \text{volume (mL)}}{\text{plasma Cr (mg/dL)} \times \text{time (min)}}$$

$$Cl_{H_2O} = V - (U_{osm} \times V) / P_{osm}$$

$$P_{\text{osm}} = \frac{2[\text{Na}^+ (\text{mEq/dL})] + \text{glucose (mg/dL)}}{18} + \frac{\text{BUN (mg/dL)}}{2.8} + \frac{\text{mannitol (mg/dL)}}{18} + \frac{\text{ethanol (mg/dL)}}{4.6}$$

$\dot{V}_{CO_2}/\dot{V}_{O_2}$