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A LIPPINCOTT MANUAL®

The Washington Manual™ of Surgery

SEVENTH EDITION

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Washington University in St. Louis® SCHOOL OF MEDICINE

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Foreword

Welcome to the seventh edition of *The Washington Manualª of Surgery.* Over the past 100 years, an important focus of our Department of Surgery has been medical education of students, residents, fellows, and practicing surgeons. This commitment is clearly evident in the current edition of *The Washington Manualª of Surgery.*

The educational focus of our Department of Surgery has a rich tradition. The first full-time head of the Department of Surgery at Washington University was Dr. Evarts A. Graham (1919Ñ1951). Dr. Graham was a superb educator. Not only was he an outstanding technical surgeon, but his insightful comments at conferences and ward rounds were well known and appreciated by a generation of surgeons who learned at his elbow. Dr. Graham was a founding member of the American Board of Surgery and made many seminal contributions to the management of surgical patients. His work in the development of oral cholecystography actually helped establish the Mallinckrodt Institute of Radiology at Washington University. Dr. Graham was among the first to identify the epidemiological link of cigarette smoking to lung cancer and was instrumental in raising public health consciousness about the deleterious effect on health from cigarette smoke.

Dr. Carl Moyer (1951Ñ1965) succeeded Dr. Graham. Dr. Moyer is still regarded as a legendary educator at Washington University. He was particularly known for his bedside teaching techniques, as well as for linking pathophysiology to patient care outcomes. Dr. Walter Ballinger (1967-1978) came from the Johns Hopkins University and incorporated the Halsted tradition of resident education. Dr. Ballinger introduced the importance of laboratory investigation and began to foster development of the surgeon/scientist in our department. Dr. Samuel A. Wells (1978-1997) is credited with establishing one of the most accomplished academic departments of surgery in the United States. Not only did he recruit world-class faculty, but he increased the focus on research and patient care. Dr. Wells also placed a great emphasis on educating the future academic leaders of surgery.

As in previous editions, this seventh edition of *The Washington Manualª of Surgery* combines authorship of residents, ably assisted by faculty coauthors and our senior editor, Dr. Mary Klingensmith, who is vice-chair for education in our department. Dr. Klingensmith is joined in this edition by a new senior editor, Dr. Chandu Vemuri. This combination of resident and faculty participation has helped to focus the chapters on issues that will be particularly helpful to the trainee in surgery. This new edition of the manual provides a complete list of updated references that will serve medical students, residents, and practicing surgeons who wish to delve more deeply into a particular topic. This manual does not attempt to extensively cover pathophysiology or history, but it presents brief and logical approaches to the management of patients with comprehensive surgical problems. In each of the chapters, the authors have attempted to provide

the most up-to-date and important diagnostic and management information for a given topic, as well as algorithms for quick reference. We have attempted to standardize each of the chapters so that the reader will be able to most easily obtain information regardless of subject matter.

The seventh edition has undergone a reorganization of chapters with an emphasis on clarity and consistency. As with the past edition, evidence-based medicine has been incorporated into each of the chapters, with updated information and references to reflect current knowledge and practice. All of the sections have been updated

and rewritten to reflect the most current standards of practice for each topic. These updates have been carefully edited and integrated so that the volume of pages remains approximately the same. Our goal is to keep this volume concise, portable, and userfriendly. I am truly indebted to Drs. Klingensmith and Vemuri for their passion for education and devotion to this project. Additionally, I am proud of the residents in the Department of Surgery at Washington University who have done such an outstanding job with their faculty co-authors in this seventh edition. I hope that you will find *The Washington Manual® of Surgery* a reference you commonly utilize in the care of your patient with surgical disease.

Timothy J. Eberlein, MD
St. Louis, Missouri

Preface

As with the previous six editions, this seventh edition of *The Washington Manualª of Surgery* is designed to complement *The Washington Manual of Medical Therapeutics.* Written by resident and faculty members of the Department of Surgery, it presents a brief, rational approach to the management of patients with surgical problems. The text is directed to the reader at the level of the second- or third-year surgical resident, although surgical and nonsurgical attendings, medical students, physician assistants, nurse practitioners, 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 of diagnosis and therapy; this is not an exhaustive surgical reference. Coverage of pathophysiology, the history of surgery, and extensive reference lists have been excluded from most areas.

This is the seventh edition of the manual; the first edition was published in 1997, followed by editions in 1999, 2002, 2005, 2007, and 2012. New to this volume is a set of multiple-choice review questions at the end of each chapter so that readers can self-assess their knowledge. Additionally, we have added chapters on ÒBiostatistics for the General Surgeon,Ó ÒQuality Improvement and Patient Safety,Ó and ÒFundamentals of Endoscopic, Laparoscopic, and Robotic SurgeryÓ; many chapters have been consolidated and reorganized to best reflect the nature of surgical practice. In addition, chapters have been updated with evidence-based medicine, with the latest information and treatment algorithms in each section. In many chapters, additional treatment algorithms have been added for quick reference. As with previous editions, this seventh edition includes updates on each topic as well as substantial new material.

This is a resident-prepared manual. Each chapter was extensively updated and revised (or authored) by a resident with assistance from a faculty coauthor. Editorial oversight for the manual was shared by four senior resident coeditors (Lola Fayanju, MD, Chapters 35, 36, 38, 39, 40, 41, 42, 43, 44, 45 and 46; Pamela Samson, MD, Chapters 6 and 7, 26, 27, 28, 29, 30 and 31, 33 and 34; Dominic Sanford, MD, Chapters 15, 16, 17. 18, 19, 20, 21, 22, 23, 24 and 25 and 37; and Jason Robertson, MD, Chapters 1, 2, 3, 4 and 5, 8, 9, 10, 11, 12, 13 and 14 and 32). Additionally, Dr. Chandu Vemuri, assistant professor and vascular surgeon extraordinaire, provided some editorial and leadership assistance for this edition. The tremendous effort of all involvedÑ residents and faculty members and particularly the senior resident coeditorsÑis reflected in the quality and consistency of the chapters.

I am indebted to the former senior editor of this work, Gerard M. Doherty, MD, who developed and oversaw the first three editions, then handed over to me an exceptionally well-organized

project. I am grateful for the continued tremendous support from Wolters Kluwer Health, who have been supportive of the effort and have supplied dedicated assistance. Keith Donnellan has been tremendously helpful, and Brendan Huffman has been a terrific developmental editor, keeping me in line and on schedule.

Finally, I am grateful to have a fantastic mentor and leader in my department chair, Timothy J. Eberlein, MD. He is an inspiration for his leadership, and dedication. To my family, thank you for all you do to keep it all fun and interesting.

M. E. K.

1

General and Perioperative Care of the Surgical Patient

Joshua D. Sommovilla

Mary E. Klingensmith

I. PREOPERATIVE EVALUATION AND MANAGEMENT

A. General Evaluation of the Surgical Patient. The goals of preoperative evaluation are to (1) identify the patient's medical problems and functional status; (2) determine if further information is needed to characterize the patient's medical status; (3) estimate the patient's level of risk for the planned procedure; and (4) establish if the patient's condition is medically optimized. Much of this can be accomplished with a thorough history and physical examination. For minor surgical procedures and procedures on young, healthy patients, routine diagnostic testing is often unnecessary. For patients with existing comorbidities, or in patients undergoing certain complex procedures, preoperative laboratory studies and imaging should be decided on an individual basis.

B. Specific Considerations in Preoperative Management

1. Cardiovascular disease is one of the leading causes of death after noncardiac surgery. Patients who experience a myocardial infarction (MI) after noncardiac surgery have a hospital mortality rate of 15% to 25% (*CMAJ.* 2005;173:627). A study of 4,315 patients older than 50 years of age undergoing nonemergent, noncardiac surgery with expected postoperative stays greater than 48 hours found that major perioperative cardiac events occur in 1.4% of patients *(Circulation.* 1999;100:1043). Risk stratification for major adverse cardiac events (MACE, defined as death, Q-wave MI, and need for revascularization) by the operating surgeon, anesthesiologist, and consulting internist is important.

a. Risk factors. A number of patient factors have been identified and are associated with perioperative cardiac morbidity and mortality. These include age above 70 years, unstable angina, recent (prior 6 months) MI, untreated CHF, diabetes mellitus, valvular heart disease, cardiac arrhythmias, peripheral vascular disease, and functional impairment. Factors related to the surgical procedure under consideration also convey risk. In their most recent guidelines published in 2014, the American Heart Association has condensed procedures into two risk levels: *low* risk (MACE risk <1%) and *elevated* risk (MACE risk >1%). The category of intermediate risk is no longer used, as the management of patients undergoing these and elevated risk procedures is similar.

b. Cardiac risk indices/calculators. Several tools have been created to aid in predicting preoperative risk of a MACE. The Revised Cardiac Index is one such tool, and its criteria are shown in Table 1-1. The American College of Surgeons NSQIP Surgical Risk Calculator combines cardiac and noncardiac factors to calculate risk of overall postoperative complications and can be found at riskcalculator.facs.org.

c. Functional status. Patients with poor functional status are at significantly elevated risk of perioperative cardiac events. This can usually be assessed from a patient's activities of daily living (ADLs) and is often expressed in metabolic equivalents (METs), with 1 MET equaling the resting oxygen consumption of an average 40-year-old male (Table 1-2). Functional capacity can be classified as excellent (>10 METs), good (7 to 10 METs), moderate (4 to 6 METs), or poor (<4 METs). Moderate functional capacity is classified as the ability to perform usual ADLs.

d. Preoperative testing. Specific preoperative workup is based on several factors including medical history, urgency of surgery, risk

of surgical procedure, patient functional status, and goals of care. A treatment algorithm guiding the preoperative cardiac workup is shown in Figure 1-1. When it is determined that a patient requires further testing prior to surgery, a multidisciplinary approach including a cardiologist is employed to determine which noninvasive or invasive measures should be taken to optimize the patient.

TABLE 1-1 Revised Cardiac Risk Index[a]

Risk Factor	Comment
High-risk surgery	Intrathoracic, intraperitoneal, major vascular
Ischemic heart disease	History of myocardial infarction, positive exercise stress test, angina, nitrate therapy, electrocardiogram with abnormal Q waves
History of CHF	History of CHF, pulmonary edema, or paroxysmal nocturnal dyspnea, bilateral rales, S_3 gallop, chest x-ray showing pulmonary vascular redistribution

History of cerebrovascular disease	History of transient ischemic attack or stroke
Preoperative insulin therapy for diabetes	
Preoperative serum creatinine >2 mg/dL	

[a]Rates of major cardiac complication with 0, 1, 2, or 3 of these factors were 0.4%, 0.9%, 7.0%, and 11.0%, respectively.
Adapted from Lee TH, Marcantonio ER, Mangione CM, et al. Derivation and prospective validation of a simple index for prediction of cardiac risk of major noncardiac surgery. *Circulation.* 1999;100:1043.

TABLE 1-2 Assessment of Functional Status

Functional Capacity	MET Range	Example Activities
Poor	<4	Sleeping, writing, watching TV, walking 2-3 mph on flat land, golfing with a cart
Moderate	4-7	Climbing a flight of steps, slow bicycling, sexual activity
Good	7-10	Jogging, calisthenics
Excellent	>10	Rope jumping

e. Preoperative management

(1) Patients with pacemakers should have their pacemakers turned to the uninhibited mode (e.g., DOO) before surgery. In addition, bipolar cautery should be used when possible in these patients. If unipolar cautery is necessary, the dispersive electrode should be placed away from the heart.

(2) Patients with internal defibrillators should have these devices turned off during surgery.

(3) Perioperative beta-blockade should be considered as part of a thorough evaluation of each patient's clinical and surgical risk. Preoperative evaluation should involve identification of active cardiac conditions that would require intensive management and may result in delay or cancellation of nonemergent operations. Over the past 15 years, there has been conflicting and poorly supported evidence regarding the efficacy of beta-blockers in reducing perioperative cardiac events. However, recent studies, including the PeriOperative ISchemic Evaluation (POISE) trial, suggest that beta-blockers reduce perioperative ischemia and may reduce the risk of MI and cardiovascular death in high-risk patients (*Lancet.* 2008;371:1839-1847). Routine administration of higher-dose, long-acting metoprolol on the day of surgery should be avoided in beta-blocker na•ve patients, as its use is associated with an overall increase in mortality. Beta-blockers should ideally be started in appropriate patients days to weeks before elective surgery. Preoperatively, each patient's dose should be titrated to achieve adequate heart rate control to benefit from beta-blockade while avoiding the risks of hypotension and bradycardia (*Circulation.* 2009;120:2123-2151).

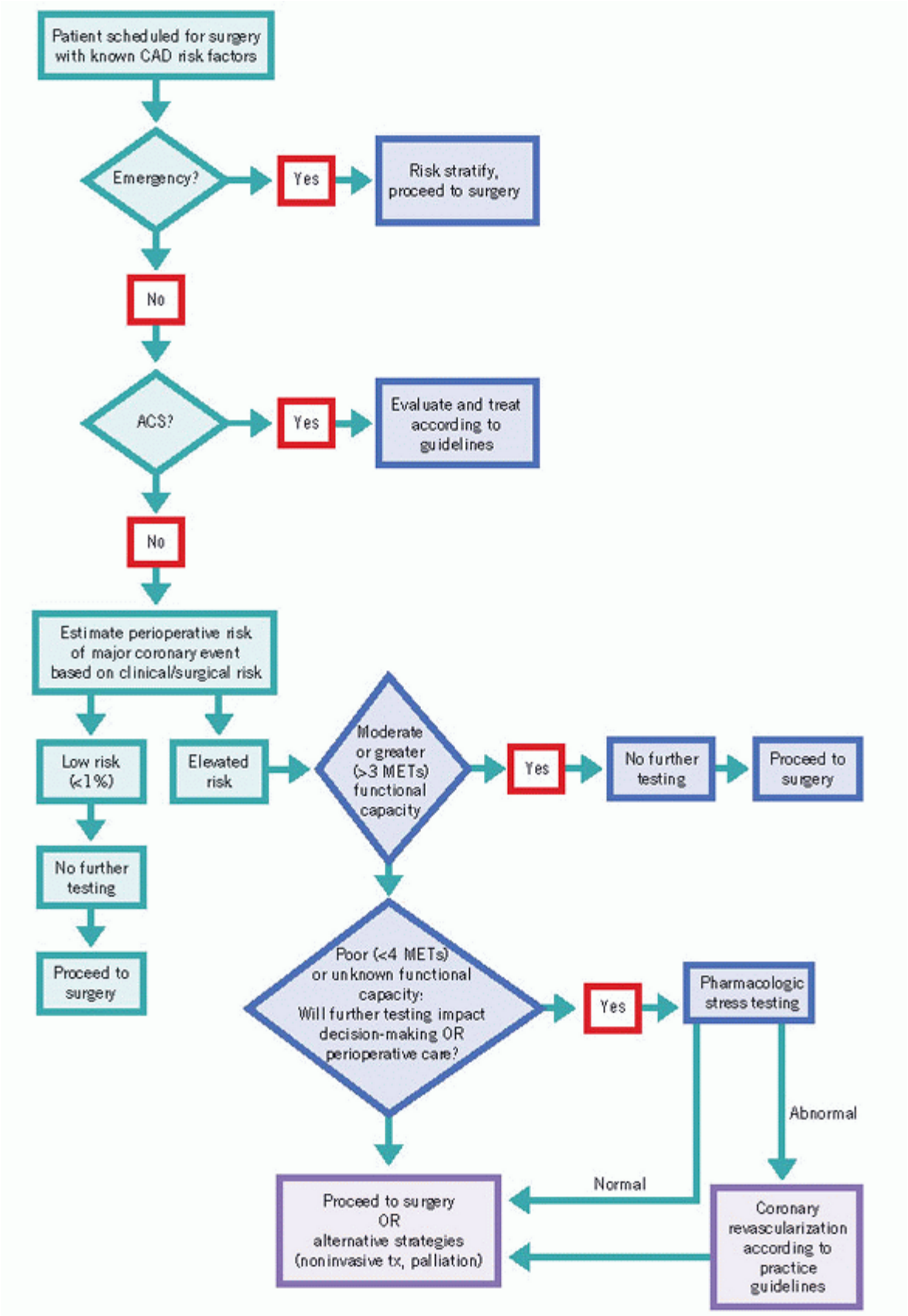

Figure 1-1 Algorithm for preoperative workup of cardiac disease. (Adapted from Flelisher LA, Fleischmann KE, Auerbach AD, et al. 2014 ACC/AHA guideline on perioperative cardiovascular evaluation and management of patients undergoing noncardiac surgery: a

report of the American College of Cardiology/American Heart Association Task Force on practice guidelines. *J Am Col Cardiol.* 2014;64(22):e77-e137.)

(4) Patients with recent angioplasty or stenting. Over the past two decades, use of coronary angioplasty and stenting has increased dramatically. Several studies have shown a high incidence of cardiovascular complications when noncardiac surgery is performed shortly after coronary angioplasty or stenting. Current guidelines are to delay noncardiac surgery at least 6 weeks after coronary angioplasty or placement of bare metal stents, which require 6 weeks of dual antiplatelet therapy with aspirin and clopidogrel. In contrast, dual antiplatelet therapy should be continued for at least 12 months following placement of a drug-eluting stent (DES), which can affect timing of elective operations. For all patients, the risk of bleeding and thrombosis need to be weighed against each other. Surgery in an open body space such as the abdomen is possible on patients taking these medications, albeit with an elevated bleeding risk.

2. Pulmonary disease. Preexisting lung disease confers a dramatically increased risk of perioperative pulmonary complications. Risk factors for pulmonary complications include chronic obstructive pulmonary disease, smoking, asthma, obstructive sleep apnea, advanced age, obesity, surgical site located near the diaphragm, smoking, and functional status.

a. Preoperative evaluation and screening

(1) Physical examination should be performed carefully, with attention paid to signs of lung disease (e.g., wheezing, prolonged expiratoryÑinspiratory ratio, clubbing, or use of accessory muscles of respiration).

(2) Diagnostic evaluation

(a) A **chest x-ray (CXR)** should only be performed for acute symptoms related to pulmonary disease, unless it is indicated for the specific procedure under consideration.

(b) An **arterial blood gas (ABG)** can be considered in patients with a history of lung disease or smoking to provide a baseline for comparison with postoperative studies, but is not reliable to accurately predict postoperative pulmonary complications.

(c) Preoperative pulmonary function testing is controversial and probably unnecessary in stable patients with previously characterized pulmonary disease undergoing nonthoracic procedures.

b. Preoperative prophylaxis and management

(1) Pulmonary toilet. Increasing lung volume 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

emergent surgery is required, patients with acute pulmonary infections should receive intravenous (IV) antibiotic therapy.

(3) Cessation of smoking. All patients should be encouraged to and assisted in smoking cessation before surgery. There has been debate over timing of smoking cessation, in particular over whether smoking cessation within weeks of surgery may paradoxically increase pulmonary complications. This concern, however, is not supported by evidence, and current guidelines favor smoking cessation prior to surgery regardless of timeframe.

(4) Bronchodilators. In the patient with obstructive airway disease and evidence of a significant reactive component, bronchodilators may be required in the perioperative period. Elective operation should be postponed in the patient who is actively wheezing.

3. Renal disease

a. Preoperative evaluation of patients with existing renal insufficiency

(1) Evaluation

(a) History. Patients with hypertension or diabetes and CRI are at a substantially increased risk of perioperative morbidity and mortality. The timing and quality of the patient's last dialysis session, the amount of fluid removed, and the preoperative weight provide important information about the patient's volume status. In nonanuric patients, the amount of urine made on a daily basis should also be documented.

(b) Physical examination should be performed 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 electrolyte and bicarbonate levels should be measured, as well as blood urea nitrogen (BUN) and creatinine. A complete blood cell count (CBC) should be obtained to evaluate for significant anemia or a low platelet level. Normal platelet numbers can mask platelet dysfunction in patients with chronic uremia.

(2) Management

(a) Timing of dialysis. Dialysis should be performed within 24 hours of the planned operative procedure.

(b) Intravascular volume status. Cardiac events are the most common cause of death in patients with CRI. Both hypovolemia and volume overload are poorly tolerated, and invasive monitoring in the intraoperative and postoperative periods may assist in optimizing fluid balance.

b. Preventing perioperative renal dysfunction

(1) Risk factors. Patients without preexisting CRI ranges may be at risk of developing postoperative acute renal failure (ARF), depending on certain patient and procedure risk factors. Incidence of postoperative ARF ranges from 1.5% to 2.5% for

cardiac surgical procedures to more than 10% for patients undergoing repair of supraceliac abdominal aortic aneurysms (AAAs). Other risk factors for the development of ARF include elevated preoperative BUN or creatinine, CHF, advanced age, intraoperative hypotension, sepsis, aortic cross-clamping, intravascular volume contraction, and use of nephrotoxic and radionucleotide agents.

(2) Prevention

(a) Intravascular volume expansion. Adequate hydration is the most important preventive measure for reducing the incidence of ARF.

(b) Radiocontrast dye administration. Patients undergoing radiocontrast dye studies have an increased incidence of postoperative renal failure. Fluid administration (1 to 2 L of isotonic saline) alone appears to confer protection against ARF. Additional commonly used but unproven measures for reducing the incidence of contrast dye-mediated ARF include the use of low-osmolality contrast agents, a bicarbonate drip, and oral *N*-acetylcysteine.

(c) Other nephrotoxinsÑincluding aminoglycoside antibiotics, nonsteroidal anti-inflammatory drugs (NSAIDs), and various anesthetic drugsÑcan predispose to renal failure, as well, and should be avoided in patients at high risk for postoperative renal failure.

4. Infectious complications. Infectious complications are a major cause of morbidity and mortality following surgery. They may arise at the surgical site itself or in other organ systems. It is impossible to overemphasize the importance of frequent handwashing or antiseptic foam use by all healthcare workers to prevent the spread of infection. In addition to impacting the patient, rates of postsurgical infections are closely monitored by hospitals and healthcare providers, and are increasingly being used as a metric by which hospitals, departments, and surgeons are measured.

a. Assessment of risk. Risk factors for infectious complications after surgery can be grouped into procedure-specific and patient-specific risk factors.

(1) Procedure-specific risk factors include the type of operation, the degree of wound contamination (whether the case is classified as clean, cleanÑcontaminated, contaminated, or dirty), and the duration and urgency of the operation.

(2) Patient-specific risk factors include age, diabetes, obesity, immunosuppression, malnutrition, preexisting infection, and other chronic illness.

b. Prophylaxis

(1) Surgical site infection. Several modifiable factors under control of various members of the

surgical team have been identified as preventable contributors to surgical site infections. A recent review in 2011 provided updated guidelines to prevent surgical

site infections, which have been embraced by the American College of Surgeons. These updated guidelines, in the form of a checklist, are shown in Table 1-3. Perioperative antibiotic recommendations for specific procedures are shown in Table 1-4.

(2) Respiratory infections. Risk factors and measures for preventing pulmonary complications are discussed in Section I.B.2.

(3) Genitourinary infections may be caused by instrumentation of the urinary tract or placement of an indwelling urinary catheter. Preventive measures include avoiding catheterization for short operations, sterile insertion of the catheter, and removal of the catheter on postoperative day 1. Some operations that include a low pelvic dissection, will require longer catheterization because of local trauma.

TABLE 1-3 Recommendations for Prevention of Surgical Site Infection

- The guidelines provided by the CDC and accrediting agents have been followed. These include techniques for asepsis, air handling, cleaning of surfaces, sterilization techniques, and activities and attire of the surgical team
- Members of the operative team have double gloved and changed gloves when any perforation is identified
- Preoperative showering with chlorhexidine the night prior and few hours of the operation was done and preoperative cleansing of the site with a chlorhexidine-impregnated cloth just before entering the operating room
- Clippers used for hair removal shortly before operation
- Reduction of skin organisms of patient and surgical team done with a combination of alcohol and chlorhexidine, or iodophors
- Antimicrobial impregnated adherent drape used at operative site
- Suture material resistant to infection used wherever possible
- Dead spaces obliterated, where possible
- Minimal trauma to the wound itself with limited use of electrocautery, with devitalized tissue removed
- Drainage through a working incision not used
- Prophylactic topical antibiotics used by pressure irrigation during operation and prior to closure in all but simplest cases
- Prophylactic systemic antibiotics used according to guidelines in all cases with

incidence of infection >0.5% and all cases with foreign body implantation

- Core temperature maintained above 36¡C or higher throughout perioperative period
- Inspired oxygen given to maintain SpO_2 >96%
- All diabetic and hyperglycemic patients received tight glucose control (blood glucose <180 mg/dL) during perioperative period and for 2-3 days afterward in high-risk patients
- Transfusion of blood products limited
- Patients have stopped smoking for at least 4 weeks prior to any highly elective operation

Adapted from Alexaner JW, et al. *Annals of Surgery.* 2011:1082-93.

TABLE 1-4 Recommendations for Antibiotic Prophylaxis

Operation	Likely Pathogens	Recommended Antibiotics
Cardiac: Prosthetic valve and other procedures	Staphylococci, corynebacteria, enteric Gramnegative bacilli	Vancomycin and Cefazolin Vancomycin and Aztreonam
Thoracic	Staphylococci	Cefazolin Vancomycin
Vascular: Peripheral bypass or aortic surgery with prosthetic graft	Staphylococci, streptococci, enteric Gram-negative bacilli, clostridia	Cefazolin Vancomycin and Aztreonam[a]
Orthopedic: Total joint replacement or internal fixation of fractures	Staphylococci	Cefazolin Vancomycin

Gastrointestinal		
Upper GI and hepatobiliary	Enteric Gramnegative bacilli, enterococci, clostridia	Cefazolin Cefotetan Cefoxitin
Colorectal	Enteric Gramnegative bacilli, anaerobes, enterococci	Cefoxitin Cefotetan Ertapenem Cefazolin and Metronidazole
Appendectomy (no perforation)	Enteric Gramnegative bacilli, anaerobes, enterococci	Cefoxitin Cefotetan
Obstetrics/gynecology	Enteric Gramnegative bacilli, anaerobes, group B streptococci, enterococci	Cefotetan Cefoxitin Cefazolin Clindamycin and Gentamicin

[a] IV, intravenous.

From Casabar E, Portell J. *The Tool Book: Drug Dosing and Treatment Guidelines, Barnes-Jewish Hospital.* 12th ed. St. Louis, MO: Department of Pharmacy, Barnes-Jewish Hospital; 2014.

5. Diabetes mellitus. Diabetic patients are at increased risk of morbidity and mortality. Vascular disease is common in diabetics, and MI, often with an atypical presentation, is the leading cause of perioperative death among diabetic patients.

a. Preoperative evaluation. All diabetic patients should have their blood glucose measured in pre-op holding and intraoperatively to prevent unrecognized hyperglycemia or hypoglycemia.

(1) Patients with **diet-controlled diabetes mellitus** can be maintained safely without food or glucose infusion before surgery.

(2) Oral hypoglycemic agents should be discontinued the evening before scheduled surgery. Long-acting agents such as chlorpropamide or glyburide should be discontinued 2 to 3 days prior.

(3) Insulin-dependent diabetics 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 intravenously. Subsequent insulin administration by either

subcutaneous (SC) sliding-scale or insulin infusion is guided by frequent blood glucose determinations. SC insulin pumps should be inactivated the morning of surgery.

6. Anticoagulation. The most common indications for warfarin therapy are atrial fibrillation, venous thromboembolism (VTE), and mechanical heart valves. Warfarin's anticoagulant effect endures for several days following cessation of the drug. Recommendations for the management of anticoagulation in the perioperative period require weighing the risks of thromboembolic events (Table 1-5) against the risk of perioperative bleeding.

a. Preoperative anticoagulation. Surgery is generally safe when the international normalized ratio (INR) value is below 1.5. Patients whose INRs are maintained between 2.0 and 3.0 normally require withholding of the medication for 5 days preoperatively.

b. Patients with high risk of thrombotic complications should be managed with **bridging anticoagulation.** This can consist of transitioning as an outpatient with low-molecular-weight heparin (LMWH, stopped 24 hours prior to surgery) or as an inpatient with an unfractionated heparin (UFH, stopped 4 to 6 hours prior to surgery) infusion when Coumadin is stopped.

c. Postoperative anticoagulation. Coumadin requires several days to reach therapeutic levels, so therapy can be resumed on postoperative days 1 or 2. High-risk patients should be bridged with therapeutically dosed SC LMWH or IV UFH until their INR is therapeutic; moderate-risk patients can be bridged with therapeutically dosed SC LMWH, therapeutically dosed IV UFH, or prophylactically dosed SC LMWH. Low-risk patients do not need to be bridged.

d. Emergent procedures. In urgent or emergent situations in which there is no time to reverse anticoagulation before surgery, plasma products, such as fresh frozen plasma (FFP), must be administered. Vitamin K can be administered, but its effects will not be seen for 8 hours if given orally and it will continue to counteract Coumadin given postoperatively.

TABLE 1-5 Risk Stratification for Perioperative Thromboembolism

	Indication for Anticoagulation		
Risk Level	**Mechanical Heart Valve**	**Atrial Fibrillation**	**VTE**
High	Any mitral valve prosthesis; recent stroke or TIA; high	$CHADS_2$ score 5 or 6; stroke	Recent (<3 mo) VTE; severe thrombophilia

	risk aortic prostheses	or TIA within 3 mo; Rheumatic heart valve disease	(protein C or S deficiency, anti-phospholipid syndrome)
Moderate	Bileaflet aortic prosthesis plus one of: atrial fibrillation (AF), prior stroke or TIA, hypertension, diabetes, congestive heart failure, age >75	$CHADS_2$ score 3 or 4	VTE within 3-12 mo; less severe thrombophilia (Factor V Leiden, prothrombin mutation); recurrent VTE; active cancer
Low	Bileaflet aortic prosthesis with no other stroke risk factors	$CHADS_2$ score of 0-2	VTE >12 mo and no other risk factors

Adapted from Douketis JD, et al. *Chest.* 2012;141:e326S-e30S.

II. POSTOPERATIVE CARE OF THE PATIENT

A. Routine Postoperative Care

1. Intravenous fluids. The intravascular volume of surgical patients is depleted by both insensible fluid losses and redistribution into the third space. As a general rule, patients should be maintained on IV fluids until they are tolerating oral intake. Extensive open abdominal procedures are associated with a loss of 500 to 1,000 mL/hour and require aggressive resuscitation.

2. Deep venous thrombosis prophylaxis. It is important to provide prophylactic therapy to nonambulatory patients to reduce the risk of deep venous thrombosis (DVT) and pulmonary embolism (PE) (Table 1-6). Risk of DVT development depends on both patient and procedure risk factors. Surgery for major trauma, hip or leg fractures, spinal cord injury, intra-abdominal cancer, joint replacement, and bariatric surgery are particularly of high risk. The most significant patient risk factor is a prior history of DVT. Other patient risk factors include malignancy, thrombophilias, oral contraceptive therapy, obesity, immobility, and indwelling central venous lines. Prophylaxis should be started

preoperatively in patients undergoing major procedures because of venous stasis and relative hypercoagulability occur during the operation. Prophylaxis and management of patients with a

history of DVT or PE are discussed in Chapter 29.

TABLE 1-6 Recommendations for VTE Prophylaxis

	Risk and Consequences of Major Bleeding	
Risk of Symptomatic VTE	**Average**	**High (>2% or Severe Consequences)**
Very low (<0.5%)	No specific prophylaxis	
Low (1.5%)	Mechanical prophylaxis, preferably with IPC	
Moderate (3.0%)	LDUH, LMWH, plus mechanical prophylaxis with ES or IPC	Mechanical prophylaxis, preferably with IPC
High (6%)	LDUH, LMWH, plus mechanical prophylaxis with ES or IPC	Mechanical prophylaxis, preferably with IPC, until bleeding risk allow addition of pharmacologic prophylaxis
High risk (6%) undergoing cancer surgery	LDUH, LMWH, plus mechanical prophylaxis with ES or IPC, and extended-duration prophylaxis with LMWH post discharge	As above
High risk (6%) contraindication to LDUH and LMWH	Fondaparinux of lowdose aspirin (160 mg); mechanical prophylaxis	As above

LDUH, low-dose unfractionated heparin; LMWH, low-molecular-weight heparin; ES, elastic stockings; IPC, intermittent pneumatic compression.

Modified from Gould MK, et al. *Chest.* 2012:e227s.

3. Pulmonary toilet. Pain and immobilization in the postoperative patient decrease the clearance of pulmonary secretions and the recruitment of alveoli. Patients with inadequate pulmonary toilet can develop fevers, hypoxemia, and pneumonia. Early mobilization, incentive spirometry, and cough and deep breathing exercises are indispensable to avoid these complications.

CHAPTER 1: PERIOPERATIVE CARE

Multiple Choice Questions

1. Which of the following factors is associated with the highest elevated cardiac risk?

a. Diabetes controlled with metformin and glyburide

b. Mild renal impairment with a preoperative creatinine level of 1.7 mg/dL

c. History of a transient ischemic attach 9 months ago

d. History of hypertension controlled with three medications

View Answer

2. Classify the functional status of a patient who is able to golf with a cart and climb two flights of steps but unable to jog or do push ups:

a. Poor

b. Moderate

c. Good

d. Excellent

View Answer

3. Which of the following is a recommendation endorsed by the American College of Surgeons to reduce the risk of surgical site infection?

a. Hair removal from surgical site by shaving

b. Tight glucose control perioperatively with goal of <200 mg/dL

c. Core body temperature maintained above 35.5°C

d. Use of supplementary oxygen during surgery to maintain SpO_2 greater than 96%

View Answer

4. Which of the following patients with a history of venous thromboembolism (VTE) is at highest risk for recurrent VTE when undergoing a surgical procedure?

a. A patient with a spontaneous VTE 3 years ago

b. A patient with VTE 6 months ago who was diagnosed with Factor V Leiden deficiency

c. A patient with a VTE 4 months ago from Protein C deficiency

d. A patient with a diagnosis of ovarian cancer who was diagnosed with a pulmonary embolism 9 months ago

View Answer

5. Which patient with a prosthetic heart valve is at greatest risk of perioperative thromboembolism?

a. A diabetic with a bileaflet aortic prosthesis

b. An asymptomatic patient with mitral prosthesis

c. A patient with CVA diagnosed 1 year ago who has a bileaflet aortic prosthesis

d. A patient with atrial fibrillation who has a bileaflet aortic prosthesis

View Answer

6. Which of the following patients would require pharmacologic stress testing prior to surgery?

a. A patient presenting with sepsis from perforated diverticulitis who has known coronary artery disease

b. A patient with history of coronary artery disease and three-vessel CABG with moderate functional status presenting for elective knee replacement

c. A diabetic set to undergo peripheral arterial bypass who has no dyspnea on exertion but for whom claudication limits walking to ˜10 paces

d. An elderly male with coronary artery disease and diabetes who is able to bicycle several miles without dyspnea and is scheduled for major liver resection

View Answer

7. For an elective operation, how many days prior to surgery should Coumadin be discontinued?

a. It need not be stopped

b. 3 days

c. 5 days

d. 7 days

View Answer

8. Which of the following is an indication for postoperative hemodialysis?

a. A potassium level of 6.2 in an oliguric patient with no EKG changes

b. Removal of fluid in an intubated and anuric patient with pulmonary edema

c. Oliguria and sepsis in a patient with creatinine 2× baseline and a moderate metabolic acidosis

d. A severely under-resuscitated patient with creatinine on 6.8

View Answer

9. A 53-year-old male undergoes emergent exploratory laparotomy for perforated sigmoid diverticulitis. He is not septic and makes urine throughout the case, but the procedure lasts for 5 hours and the patient receives over 4 L of intravenous crystalloid. When should this patient's Foley catheter be removed following surgery?

a. Immediately following the procedure

b. On postoperative day 1

c. On postoperative day 3 if no hematuria is present and ureteral injury ruled out

d. When patient is ambulatory

View Answer

2
Common Postoperative Problems

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Isaiah R. Turnbull

This chapter explores common postoperative problems and initial stages of their management. The initial evaluation of all postoperative patients should include assessment of hemodynamic stability (ÒA, B, C'sÓ of airway, breathing, and circulation). The next consideration should be the need to transfer to a higher level of care. Patients with new onset failure of more than one organ system (e.g., new respiratory insufficiency and new renal failure) or patients with an acute problem that does not respond to initial intervention (e.g., hypovolemia unresponsive to fluid challenge) should be considered for transfer to an intensive care unit (ICU). The stable patient can be efficiently evaluated and treated in the inpatient ward. This chapter offers descriptions of commonly encountered postoperative complaints, their initial workup and treatment.

I. NEUROLOGIC COMPLICATIONS

A. Diagnostic Considerations. The physiologic changes from surgical stress can alone affect neurologic function. The patient in postoperative day 0 is recovering from general anesthesia, the effects of which can last up to 48 hours. In addition, after major surgery, patients are placed in unfamiliar surroundings, are woken throughout the night, and are administered powerful medications to which they may not have been previously exposed. When evaluating neurologic concerns, initial differentiation should be made between the patient with altered sensorium characterized by somnolence, confusion, disorientation, and other deficits in executive function and the patient with focal neurologic changes such as slurred speech, changes in sensation or motor function, or cranial nerve deficits. This delineation will guide the development of a differential diagnosis. Altered sensorium primarily results from systemic problems such as hypoxemia, shock, or delirium. Focal neurologic deficits are concerning for an acute neurologic process such as stroke.

B. Basic Differential Diagnosis: Respiratory insufficiency, hypoglycemia, stroke, hypotension, arrhythmia, seizure, delirium, alcohol withdrawal, infection, medication related, and electrolyte abnormalities.

C. Initial Workup. A full set of vital signs including pulse oximetry and a fingerstick blood glucose should be immediately obtained. For somnolent

patients, consider inadvertent or unknown extra administration of narcotic agents; a dose of 0.04 mg of naloxone is a reasonable treatment in this setting. A complete blood cell count and a basic metabolic profile

should be obtained to evaluate for hemorrhage, early signs of infection, and electrolyte disturbances. An arterial blood gas (ABG) can be obtained to determine if the patient needs positive pressure ventilation to recover from carbon dioxide narcosis. An electrocardiogram should be obtained to evaluate for arrhythmias and myocardial infarction (MI). Abnormalities may prompt serial serum troponins. All patients with new focal neurologic findings should undergo an emergent computed tomographic (CT) scan of the head to evaluate for intracranial hemorrhage; although, ischemic stroke may not be evident on acute CT imaging.

D. Special Considerations

1. Immediate postoperative patients will likely be drowsy but should be arousable to voice or light touch. Common causes of somnolence in the acute perioperative period are narcotic overdose and hypoxemia. If the patient's respiratory rate is depressed, stimulate him or her and encourage deep breathing. If the patient is obtunded in the early postoperative period, consider naloxone injection and continuous infusion. If the patient responds to stimulation with combativeness, he or she is usually calmed with reorientation. The amnestics administered during general anesthesia can cause a patient to repeatedly lose orientation. A close friend or family member can be effective in re-orientation.

2. Elderly patients have less neurologic reserve and are the largest population to suffer from mental status changes. Their other organ systems are also delicate, often requiring ICU admissions that may prompt ICU delirium. The effects of sedatives and pain medications can be quite prolonged in this population and additional administration should only be done with careful consideration.

E. Perioperative Stroke

1. Presentation. Strokes usually present with acute onset of focal neurologic dysfunction (unilateral weakness or clumsiness, sensory loss, speech disorder, diplopia, or vertigo). Massive strokes can present with altered mental status, but mildly altered sensorium alone without focal neurologic changes is unlikely to represent a stroke.

2. Examination. A thorough neurologic examination should be part of the initial encounter.

3. Unique evaluation. CT scan of the head should be obtained urgently to rule

out hemorrhagic stroke. For patients with focal neurologic changes or imaging findings concerning for stroke, emergent consultation from a neurologist is warranted. Further studies including echocardiography, carotid ultrasound, electrocardiogram, and magnetic resonance imaging (MRI) may be needed.

4. Treatment. General supportive measures include supplemental oxygen and IV fluid. Aspirin (325 mg orally) should be given immediately in ischemic stroke. Thrombolysis has been proven effective in improving outcomes from ischemic strokes, but it may be contraindicated in postoperative patients and should only be initiated in close consultation with a neurologist and the operative surgeon.

F. Seizures

1. Precipitating factors. Most seizures in postoperative patients occur due to perioperative medication changes in patients with a history of seizure. Common causes of new seizure in postoperative patients are metabolic derangements including electrolyte abnormalities (e.g., hyponatremia and hypocalcemia), alcohol withdrawal, hypoglycemia, fever, and drugs (e.g., imipenem).

2. Examination. Complete physical and neurologic examination should focus on BLS and then on any sequelae of seizure, including trauma, aspiration, or rhabdomyolysis. A careful patient history will help determine whether a true seizure was witnessed. If so, its type, characteristics (i.e., general vs. focal), and similarity to any previous seizures should be noted. A focally abnormal neurologic examination, especially in the setting of a new-onset focal seizure, suggests a possible cerebrovascular event.

3. Unique evaluation. Serum chemistries should include calcium and magnesium. Serum levels of anticonvulsants should be measured in patients who normally take these medications. Patients with new-onset seizures who do not have identifiable metabolic or systemic causes warrant further evaluation with a head CT scan followed by a lumbar puncture and EEG.

4. Treatment:

a. The patient's airway should be stabilized initially with a soft oral or nasal airway, but endotracheal intubation may ultimately be required to protect the airway. Cardiopulmonary parameters should be monitored and IV access established immediately.

b. A single, nonrecurring seizure with identifiable metabolic or systemic causes usually requires only correction of the underlying abnormality.

c. Recurrent seizures warrant neurologic consultation. A regimen beginning with a 15- to 20-mg/kg load of phenytoin, given parenterally in three divided doses, and followed by maintenance dosing of 5 mg/kg/day in three divided

doses is typically prescribed. Status epilepticus, defined as a seizure lasting more than 5 minutes or a series of multiple, continuous seizures without return to baseline mental status, is a medical emergency and a neurology consult should be obtained immediately. Treatment regimens include:

(1) Lorazepam (2 to 4 mg IV at a rate of 2 mg/minute) should be given for generalized convulsions lasting longer than 5 minutes. Either lorazepam or fosphenytoin may be given intramuscularly in emergent situations. Results are usually seen within 10 minutes.

(2) Fosphenytoin (prescribed in phenytoin equivalents) administered parenterally is the first choice to supplement benzodiazepines in this setting and should be started concurrently.

(3) Phenobarbital is a second-line agent and should be used when fosphenytoin is contraindicated (e.g., heart block) or ineffective. A loading dose of 20 mg/kg IV can be given at 100 mg/minute.

Maintenance doses of 1 to 5 mg/kg/day intravenously or orally are required to achieve therapeutic plasma levels. Institution of a phenobarbital coma should be considered if status epilepticus continues.

G. Delirium

1. Presentation. Symptoms include impaired memory, altered perception, and paranoia, often alternating with periods of lucency. Altered sleep patterns result in drowsiness during the day with wakefulness and agitation at night (i.e., sundowning). Disorientation and combativeness are common.

2. Evaluation. Generally considered a diagnosis of exclusion, management begins with eliminating the possibility of an underlying physiologic or metabolic derangement, with particular attention paid to infection. Infection should be evaluated with a CBC and urinalysis. Hypoxemia and hypercarbia should be assessed by ABG. Other testing, including ECG or CXR, is dictated by clinical suspicion. Medications should be reviewed carefully, paying particular attention to anticholinergic agents, opiate analgesics, and antihistamines.

3. Treatment. Mood-altering medications including narcotics and anticholinergics should be minimized and any metabolic abnormalities corrected. Benzodiazepines, antihistamines such as diphenhydramine, and sleep aids such as zolpidem and eszopiclone, ought to be strictly avoided. Good sleep hygiene is also important and should be encouraged by keeping the lights off at night and on during the day, minimizing nighttime laboratory draws and nursing care and getting the patient out of bed during waking hours. Patients also benefit from an exterior view when possible. For recalcitrant delirium, Quetiapine 25 to 50 mg at bedtime has been shown to be an effective adjunct *(J Hosp Med.*

2013;8[4]:215-220). Haloperidol (1 to 5 mg orally or intramuscularly) can also be used for hyperactive delirium. In some cases, physical restraints might be necessary to prevent self-harm, but should be used as a last resort and in concert with pharmacologic treatment.

H. Alcohol Withdrawal

1. Presentation. Symptoms of minor withdrawal can begin 8 hours after blood alcohol levels normalize and are characterized by insomnia, anxiety, tachycardia, tachypnea, fever, and hypertension. These symptoms peak at 72 hours. Alcohol withdrawal seizures occur 12 to 48 hours after normalization of blood alcohol level. They are usually brief and self-limited. Delirium tremens (DT) typically occurs 72 to 96 hours or longer after cessation of alcohol intake and is characterized by disorientation, hallucinations, and autonomic lability that include tachycardia, hypertension, fever, and profuse diaphoresis. Mortality for hospitalized patients with DT is 1% to 4%. Patients with acute DT should be monitored in an ICU *(N Engl J Med.* 2014;371[22]:2109-2113).

2. Evaluation. The diagnosis is clinical in nature. While one should have a high index of suspicion, in absence of a strong social history, alcohol withdrawal should also be considered a diagnosis of near exclusion in most cases.

3. Treatment

a. Nutritional supplements. Thiamine 500 mg intravenously for 3 days followed by 100 mg orally every day should be given to all suspected alcoholic patients to prevent development of Wernicke encephalopathy. Many chronic alcoholics have hypomagnesemia; if present, magnesium sulfate should be administered to patients with normal renal function. Folate 1 mg should be given daily.

b. Benzodiazepines such as chlordiazepoxide, 25 to 100 mg orally every 6 hours; oxazepam, 5 to 15 mg orally every 6 hours; or diazepam, 5 to 20 mg orally or IV every 6 hours, can be used as prophylaxis in alcoholics who have a history of withdrawal or to alleviate symptoms of minor withdrawal. Benzodiazepines are most helpful in preventing recurrent seizures. Patients with DT should be given diazepam, 5 to 10 mg IV every 10 to 15 minutes, to control symptoms. Oversedation must be avoided through close monitoring. The dose 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.

c. Clonidine, 0.1 mg orally four times a day or **atenolol** 50 to 100 mg orally a day, can be used to treat tachycardia or hypertension resulting from autonomic hyperactivity. Close hemodynamic monitoring is required during therapy.

II. CARDIOVASCULAR COMPLICATIONS

A. Postoperative Hypotension

1. Diagnostic considerations. In the postoperative patient, hypotension should immediately raise concerns of postoperative bleeding. A full set of vital signs should be obtained, as should a CBC. Severe hypovolemia can cause hypotension and is usually preceded by oliguria and tachycardia. The notable exception to this is the patient on beta-blockade, in whom tachycardia is blunted. In the early postoperative period (postop days 0 to 2) hypovolemia from bleeding or under resuscitation is the most common cause of hypotension. Later in the hospital course, sepsis must be strongly considered.

2. Basic differential diagnosis: Bleeding, under-resuscitation, sepsis, anesthetics and analgesics (especially via epidural catheter), and administration of antihypertensive medication.

3. Treatment. In postoperative patients without a history of congestive heart failure (CHF), an initial fluid challenge is usually warranted. A Foley catheter should be placed to monitor urine output and the patient should have adequate intravenous access for resuscitation. If an epidural catheter is in place, the infusion should be slowed or discontinued completely. In patients with an upper extremity central venous catheter (internal jugular or subclavian) a measurement of central venous oxygen saturation can be helpful in distinguishing between septic and hypovolemic shock (see Chapter 7). If hypotension is not quickly resolved by IV fluid infusion, blood transfusion, if indicated,

or drug cessation, the patient should be transferred to a higher level of care.

B. Hypertension

1. Diagnostic considerations. Postoperative hypertension should be defined by the patient's preoperative blood pressure (BP). Chronic hypertension causes a shift in the cerebral autoregulatory system that may not allow for adequate cerebral perfusion at normotensive BPs. A reasonable goal of therapy for acute postoperative hypertension is within 10% of the patient's normal BP.

2. Basic differential diagnosis: Essential hypertension, hypertensive urgency, pain, ethanol withdrawal, hypoxemia, hypothermia, and acidosis.

3. Treatment. Underlying causes of hypertension such as acute pain or alcohol withdrawal should be identified and addressed first. Initial treatment for hypertension should be to resume home antihypertensive agents when possible. Acute hypertension can be managed with labetalol (10 to 20 mg intravenously

every 10 minutes, to a total dose of 300 mg), hydralazine (10 to 20 mg intravenously every 6 hours), or clonidine (0.1 mg orally every 6 hours). Patients with symptomatic hypertension (hypertensive urgency or emergency) should be transferred to an ICU for further care.

C. Myocardial Ischemia and Infarction

1. Presentation. The presentation of myocardial ischemia in the postoperative patient is often subtle. Frequently, perioperative MI is silent or presents with dyspnea, hypotension, or atypical pain, including abdominal pain. Close questioning of the patient on the pain characteristics often narrows the differential significantly. Postoperative MIs classically occur on postoperative day 2 and any new development of chest pain should prompt a full workup for MI.

2. Basic differential diagnosis: MI, pulmonary embolism (PE), pleuritis, pneumonia, pericarditis, incisional pain, aortic dissection, pneumothorax, pneumomediastinum, and GERD.

3. Evaluation. Most chest pain complaints warrant a new set of vital signs, serum electrolytes, hemoglobin, and a chest radiograph. An EKG is necessary in virtually all cases of postoperative chest pain, and it should be compared to prior tracings. Sinus tachycardia is one of the most common rhythms associated with MI. An elevated troponin I level in the setting of EKG changes is diagnostic of MI. A series of three samplings of troponin I 6 to 9 hours apart has a sensitivity and specificity of greater than 90% for detecting myocardial injury *(N Engl J Med.* 2009;361:868-877). However, clinical factors such as global shock and renal failure can lead to false positives. Further diagnostic evaluation (including echocardiography) should be pursued as indicated by the initial workup.

4. Treatment. If the troponin levels are elevated or there are EKG findings consistent with ischemia (ST-segment or new conduction system changes), an emergent cardiology consultation should be obtained. The patient should be placed on telemetry monitoring and have oxygen applied to keep saturations >90%. Morphine may be administered

to manage the pain and to decrease the sympathetic drive (1 to 4 mg IV every hour), and nonenteric coated aspirin administration can be lifesaving (325 mg). In the absence of hypotension, initial management for cardiac chest pain includes sublingual nitroglycerin (0.4 mg) every 5 minutes until the pain resolves. Hemodynamically stable patients without CHF, significant bradycardia, or heart block should also receive beta-blockade, usually metoprolol 15 mg IV in 5 mg doses every 5 minutes, as this has been shown to improve patient

outcomes (*Am J Cardiology.* 1999;84:76). Patients with any sign of hemodynamic changes should be urgently transferred to an ICU pending expert consultation.

D. Congestive Heart Failure

1. Presentation. CHF exacerbations typically present with shortness of breath or hypoxia. Physical examination often reveals signs of fluid overload. CHF can occur in the immediate postoperative period as a result of excessive intraoperative administration of fluids or 24 to 48 hours postoperatively related to mobilization of fluids that are sequestered in the extracellular space. Patients frequently have a history of asymptomatic heart failure.

2. Evaluation. Bedside evaluation includes pulse oximetry and assessment of net fluid balance and weight for the preceding days. Laboratory studies include troponin I, B-type natriuretic peptide (BNP), ABG, CBC, electrolytes, and renal function tests. CXR and echocardiogram are frequently indicated.

3. Differential diagnosis: Pneumonia, atelectasis, PE, reactive airway disease (asthma, COPD exacerbation), and pneumothorax.

4. Treatment is aimed at maximizing cardiac perfusion and efficiency.

a. Supplemental oxygen should be administered. Mechanical ventilation is indicated in patients with refractory hypoxemia.

b. Diuresis should be initiated with furosemide (20 to 40 mg IV push), with doses up to 200 mg every 6 hours as necessary. Furosemide drips can be effective in promoting adequate diuresis. Fluid intake should be limited, and serum potassium should be monitored closely. If contraction alkalosis occurs, acetazolamide may be substituted for furosemide.

c. Arterial vasodilators. To reduce afterload and help the failing heart in the acute setting, ACE inhibitors can be used to lower the systolic BP to 90 to 100 mm Hg. Negative inotropes such as calcium channel blockers and beta-blockade should be avoided.

d. Inotropic agents. Digoxin increases myocardial contractility and can be used to treat patients with mild failure. Patients with florid failure may need invasive monitoring and continuous inotrope infusion in an intensive care setting.

III. PULMONARY COMPLICATIONS

A. Dyspnea

1. Diagnostic considerations: Shortness of breath is often thought of as being a primary respiratory problem, but it can be a symptom

of systemic illness such as CHF and PE. Differential diagnoses include atelectasis, lobar collapse, pneumonia, CHF, COPD, asthma exacerbation, pneumothorax, PE, and aspiration. Shortness of breath can also be a result of MI, intra-abdominal complications, systemic sepsis, and fever. Additional factors that help to differentiate disease entities include smoking history, fever, chest pain, and the time since surgery.

2. Examination may reveal jugular venous distention, abnormal breath sounds (wheezing, crackles), asymmetry, and increased respiratory effort.

3. Evaluation. CBC, pulse oximetry, ABG, and CXR are mandatory for all persistently dyspneic patients. ECGs should be obtained for any patient older than 30 years with significant dyspnea or tachypnea to exclude myocardial ischemia and in any patient who is dyspneic in the setting of tachycardia.

4. Treatment

a. Atelectasis commonly occurs in the first 36 hours after operation and typically presents with dyspnea and hypoxia. Therapy is aimed at re-expanding the collapsed alveoli. For most patients, deep breathing, coughing, and incentive spirometry are adequate. Postoperative pain should be controlled so that pulmonary mechanics are not impaired. In patients with 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. Gastric aspiration usually presents with acute dyspnea and fever. CXR might be normal initially but subsequently demonstrate a pattern of diffuse interstitial infiltrates. Therapy is supportive, and antibiotics are typically not given empirically.

c. 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.

d. Volume overload, pneumonia, and PE are discussed elsewhere in this chapter.

B. COPD and Asthma Exacerbations

1. Reactive airways are common in postoperative smokers and asthmatic patients. The local trauma of an endotracheal tube can induce bronchospasm *(Anesth Analg.* 1995;80:276).

2. Presentation may include wheezing, dyspnea, tachypnea, hypoxemia, and possibly hypercapnia.

3. Treatment

a. 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 to 6 hours via nebulization). Beta-adrenergic agonists are indicated primarily for acute exacerbations rather than for long-term use.

b. Anticholinergics such as ipratropium bromide (Atrovent, 2 puffs every 4 to 6 hours) can also be used in the perioperative period, especially if the patient has significant pulmonary secretions.

c. Patients with severe asthma or COPD may benefit from **parenteral steroid therapy** (methylprednisolone, 50 to 250 mg intravenously every 4 to 6 hours) as well as **inhaled steroids** (beclomethasone metered-dose inhaler, 2 puffs four times a day), but steroids require 6 to 12 hours to take effect.

IV. RENAL COMPLICATIONS

A. Oliguria is defined as urine output of less than 0.5 mL/kg/hour. The most common early perioperative cause of oliguria is hypovolemia from under resuscitation or bleeding. Other important considerations include preoperative renal dysfunction, home diuretic use, and perioperative urinary retention due to general anesthesia. Initial evaluation of the patient with oliguria should include a serum electrolyte panel, a hematocrit or serum hemoglobin level, and an ultrasound scan of the bladder to assess for urinary retention. Any patient with persistent oliguria should have a Foley catheter placed. For patients with normal cardiac and renal function, a fluid challenge with 0.5 to 1 L of crystalloid IV can be diagnostic and therapeutic for hypovolemia.

B. Urinary Retention. Perioperative patients are at risk for acute urinary retention. Urinary retention can present as failure to void or with acute pain due to an overdistended bladder. In the perioperative patient, failure to void within 6 hours should prompt a workup for oliguria as described above, including an ultrasound bladder scan. Patients with subjective symptoms of bladder distension or patients with greater than 500 *cc* of urine on bladder ultrasound should undergo bladder catheterization. An initial trial of bladder decompression with immediate removal of the catheter (“straight cath”) is reasonable, although others advocate for a short duration (24 hours) of bladder decompression with an indwelling Foley catheter. Treatment with alpha-blockade (Tamsulosin 0.4 mg daily) may decrease the probability of a second episode of urinary retention *(Rev Urol.* 2005;7 Suppl 8:S26-33).

C. Acute Kidney Injury (AKI)

1. Presentation. AKI is defined by an increase in serum creatinine level by 0.3 mg/dL or 1.5-fold above baseline in the setting of oliguria. The etiologies of AKI

can be classified as prerenal, intrinsic renal, and postrenal (Table 2-1).

a. Prerenal azotemia results from decreased renal perfusion that might be secondary to hypotension, intravascular volume contraction, or decreased effective renal perfusion.

b. Intrinsic renal causes of AKI include drug-induced acute tubular necrosis, pigment-induced renal injury, radiocontrast dye administration, acute interstitial nephritis, rhabdomyolysis, and prolonged ischemia from suprarenal aortic cross-clamping.

TABLE 2-1 Laboratory Evaluation of Oliguria and Acute Renal Failure

Category	FE_{Na}	U_{Osm}	RFI	U_{Cr}/P_{Cr}	U_{Na}
Prerenal	<1	>500	<1	>40	<20
Renal (acute tubular necrosis)	>1	<350	>1	<20	>40
Postrenal	>1	<50	>1	<20	>40

FE_{Na}, fractional excretion of sodium; RFI, renal failure index; U_{Cr}/P_{Cr}, urine:plasma creatinine ratio; U_{Na}, urine sodium; U_{Osm}, urine osmolality.

c. Postrenal causes of AKI 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, obstruction of the bladder from an enlarged prostate, narcotic use for management of postoperative pain, or an obstructed urinary catheter can contribute to postrenal ARF.

2. Evaluation. Urinalysis with microscopy and culture (as indicated) can help in differentiating between etiologies of AKI. In addition, urinary indices including fractional excretion of sodium (FE_{Na}), renal failure index, and fractional

extraction of urea (FE_{Ur}) help to classify AKI into the above listed categories. In the setting of diuretic administration, FE_{Ur} is favored over FE_{Na}. Renal ultrasonography can be used to exclude obstructive uropathy, assess the chronicity of renal disease, and evaluate the renal vasculature with Doppler ultrasonography. Radiologic studies using IV contrast are contraindicated in patients with suspected AKI due to potential exacerbation of renal injury.

3. Treatment

a. Prerenal. In most surgical patients, oliguria is caused by hypovolemia. Initial management includes a fluid challenge (e.g., a normal saline bolus of 500 mL). Patients with adequate fluid resuscitation and CHF may benefit from invasive monitoring and optimization of cardiac function.

b. Intrinsic renal. Treat the underlying cause, if possible, and manage volume status.

c. Postrenal. Ureteral injuries or obstruction can be treated with percutaneous nephrostomy tubes and generally are managed in consultation with an urologist. Urinary retention and urethral obstruction can be managed by placement of a Foley catheter or, if necessary, a suprapubic catheter.

d. In all cases, careful attention to intravascular volume is paramount. Patients should be weighed daily and have careful records on intakes and outputs. Hyperkalemia, metabolic acidosis, and hyperphosphatemia are common problems in patients with AKI and should be

managed as discussed in Chapter 4. Medication doses should be adjusted appropriately and potassium removed from maintenance IV fluids.

e. Dialysis: Indications for dialysis include intravascular volume overload, electrolyte abnormalities, especially hyperkalemia, metabolic acidosis, and complications of uremia (encephalopathy, pericarditis).

V. GASTROINTESTINAL COMPLICATIONS

A. Postoperative Nausea and Vomiting

1. Presentation. On postoperative days 0 and 1, postanesthesia nausea can affect up to 30% of patients *(Anesthesiology.* 1992;77:162). Other common causes of nausea in the early perioperative period include medication side-effects (especially from opiate analgesics), perioperative gastroparesis, and paralytic ileus. Patients who have undergone extensive intra-abdominal procedures and are more than 24 hours out from anesthesia should be evaluated for the underlying causes of nausea before administration of antiemetics. Up to 20% of these patients will suffer an ileus requiring

nasogastric decompression *(Dis Colon Rectum.* 2000;43:61).

2. Treatment. Aggressive management with antiemetics can be employed. Multimodal therapy with Ondansetron, Phenergan, Compazine, Scopolamine, and Decadron can be required.

B. Postoperative Paralytic Ileus

1. Presentation. Paralytic ileus typically presented with obstipation, persistent nausea despite antiemetic use, intolerance of oral diet, belching, abdominal distension with mild discomfort, and absence of flatus.

a. Differential diagnosis: Bowel obstruction, constipation, Ogilvie syndrome, intra-abdominal infection, and retroperitoneal bleeding.

b. Evaluation. Upright and lateral decubitus radiographs of the abdomen should be obtained to evaluate for dilated stomach and loops of bowel. Air should be seen in the colon, thus helping to differentiate from bowel obstruction. When this imaging is insufficient to rule out bowel obstruction, abdominal CT with oral contrast is both sensitive and specific (90% to 100%), though is less reliable for partial than complete small bowel obstructions. If the diagnosis remains uncertain, an upper gastrointestinal study with water-soluble contrast material may be necessary.

2. Treatment. Patients with an ileus should be made NPO and started on IVF. Strong consideration should be made for placing a decompressive nasogastric tube, even in the absence of gastric distension on plain film. A patient with an NG tube in place who complains of nausea should have the NG tube manipulated until functioning properly. This may even require replacement with a larger-bore NG tube. Deficiencies of potassium and magnesium as well as excess opioids can prolong ileus. Since the etiology is nonmechanical factors, patience must then be employed as one awaits return of bowel function. Should the ileus persist beyond 7 days, a nutrition consultation should be placed to evaluate for TPN.

VI. INFECTIONS COMPLICATIONS.

Infection can manifest as obvious signs and symptoms such as erythema, induration, drainage, necrosis, or tenderness on examination, but it can also manifest as more subtle signs and symptoms such as chills, malaise, hypothermia, or unexplained leukocytosis. While attention to the multitude of peri- and postoperative infections complications is paramount, it is beyond the scope of this chapter. The discussion below is designed to serve as an initial starting point and will touch briefly on management of specific infectious etiologies.

A. Prevention and the Surgical Care Improvement Project (SCIP). Undoubtedly, the key

to management of postoperative infectious complications is prevention. The mainstays of infection prevention have been careful attention to sterile techniques, prophylactic antibiotic administration, selected use of Foley catheters, early mobilization, and pulmonary toilet. The SCIP was launched in 2005 as a quality improvement partnership aimed at reducing significant surgical complications and improving surgical outcomes nationally. In addition to the direct effect on patients, these quality metrics are being used by the Joint Commission and the Centers for Medicare and Medicaid Services as a measurement for hospital and clinician performance. Of the 2014 SCIP core measurements, seven of the nine indicators relate to infection prevention. They are as follows:

1. Prophylactic antibiotics received within 1 hour prior to surgical incision in order to obtain bactericidal serum level.

2. Prophylactic antibiotic selection for surgical patients (Table 2-2) to encourage good stewardship as well as targeted coverage.

3. Prophylactic antibiotics discontinued within 24 hours after surgery end time (with the exception being cardiac surgery which is within 48 hours). There are no data to suggest an increased benefit after these time frames. The goal is decreased risk of developing *Clostridium difficile* and multidrug resistant organisms.

4. Surgery patients receive appropriate hair removal, favoring clippers over razors, in order to prevent skin abrasions.

5. Urinary catheter removal on postoperative day 1 or 2 to prevent urinary tract infections.

6. Perioperative temperature management, since hypothermia increases the risk of infection and impairs healing.

B. Generalized Fever

1. Presentation. In the immunocompetent adult, fever is defined as a body temperature greater than 38¡C. Evaluation of fever should take into account the amount of time that has passed since the patient's most recent operation.

a. Intraoperative fever may be secondary to malignant hyperthermia, a transfusion reaction, or a pre-existing infection.

b. Fever in the first 24 hours usually occurs as a result of atelectasis. A high fever (>39¡C) is commonly the result of a streptococcal or clostridial wound infection, aspiration pneumonitis, or a pre-existing infection. However, fever in this time period can also be seen in trauma or burn patients as a part of the SIRS response in the absence of infection.

TABLE 2-2 Recommendations for Antibiotic Prophylaxis

Nature of Operation	Likely Pathogens	Recommended Antibiotics	Adult Dose Before Surgery[a]
Cardiac: Prosthetic valve and other procedures	Staphylococci, coryne-bacteria, enteric Gram-negative bacilli	Vancomycin and Cefazolin	1-1.5 g IV
		Vancomycin and Aztreonam[a]	1-3 g IV
Device insertion		Cefazolin or Vancomycin	1-1.5 g IV 1-2 g IV
Thoracic	Staphylococci	Cefazolin	1-3 g IV
		Vancomycin[a]	1-1.5 g IV
Vascular: Peripheral bypass or aortic surgery with prosthetic graft	Staphylococci, strepto-cocci, enteric Gram-negative bacilli, clostridia	Cefazolin	1-3 g IV
		Vancomycin and Aztreonam[a]	1-1.5 g IV 1-2 g IV
Abdominal wall hernia	Staphylococci	Cefazolin	1-3 g IV
		Clindamycin[a]	900 mg IV
Orthopedic: Total joint replacement or internal fixation of fractures	Staphylococci	Cefazolin +/− Vancomycin	1-3 g IV 1-1.5 g IV
		Vancomycin and Aztreonam[a]	1-1.5 g IV 1-2 g IV
Gastrointestinal			

Upper GI and hepatobiliary	Enteric Gram-negative bacilli, enterococci, clostridia	Cefotetan Cefoxitin Clindamycin and Gentamicin[a] Ciprofloxacin and Metronidazole[a]	1-2 g IV 1-2 g IV 900 mg IV 5 mg/kg IV 400 mg IV 500 mg IV
Colorectal	Enteric Gram-negative bacilli, anaerobes, enterococci	Cefoxitin Cefotetan Ertapenem Cefazolin and Metronidazole[a]	1-2 g IV 1-2 g IV 1 g IV 1-3 g IV 500 mg IV
Appendectomy (no perforation)	Enteric Gram-negative bacilli, anaerobes, enterococci	Cefoxitin Cefotetan Ciprofloxacin and Metronidazole[a]	1-2 g IV 1-2 g IV 400 mg IV 500 mg IV
Obstetrics/gynecology	Enteric Gram-negative bacilli, anaerobes, group B strepto-cocci, enterococci	Cefotetan Cefoxitin Cefazolin Clindamycin and Gentamicin[a]	1-2 g IV 1-2 g IV 1-3 g IV 900 mg IV 1.5-5 mg/kg IV

For Vancomycin, dose of 1 g is recommended for patients <80 kg, 1.5 g is recommended for >80 kg.
For cefazolin, cefotetan, cefoxitin, and aztreonam, pre- and intraoperative dosing of 1 g is suggested for patients weighing <80 kg, 2 g for patients weighing >80 kg and <120 kg, and 3 g for patients weighing >120 kg.

In obese patients, single dose gentamicin should be dosed at 5 mg/kg of adjusted body weight (ABW = IBW + 0.4[TBW Ð IBW]).

[a] Indicated for patients with penicillin/cephalosporin allergy. IV, intravenous.
Adapted from Casabar E, Portell J. *The Tool Book: Drug Dosing and Usage Guidelines, Barnes-Jewish Hospital.* 12th ed. St. Louis, MO: Department of Pharmacy, Barnes-Jewish Hospital; 2014.

c. Fever that occurs more than 72 hours after surgery has a broad differential diagnosis, including but not limited to the following: wound infection (including fascial or muscle infections), pneumonia, gastroenteritis, infectious colitis (including *C. difficile),* abscesses, peritonitis, UTI, infected prosthetic materials or catheters, DVT, thrombophlebitis, drug allergy, or devastating neurologic injury. In immunocompromised hosts, viral and fungal infections should also be considered. Transfusion reactions can be confused for infection due to the presence of fever; although, the treatment is vastly different and will not be discussed here.

2. Evaluation. The new onset of fever or leukocytosis without an obvious source of infection requires a thorough history and physical examination, including inspection of all wounds, tubes, and catheter sites. A CBC, a urinalysis, and a chest x-ray should all be obtained. Gram stain/cultures of the blood, sputum, urine, and/or wound should be dictated

by the clinical situation. Imaging such as an ultrasound or CT should be chosen based on clinical context, usually to evaluate for a deep space infection in the cavity where surgery was performed.

3. Treatment

a. Empiric antibiotics may be initiated after collection of cultures, with therapy directed by clinical suspicion, but are not always warranted. Therapy usually begins with broad spectrum IV antibiotics and narrows as more information is known about the infection source and organisms.

C. Surgical Site Infections (SSIs) are the second leading cause of nosocomial infections, leading to significant patient care costs, longer length of hospital stays, and increased rates of readmission. They typically present with erythema, pain, induration, and drainage. Fever and leukocytosis may be present.

1. Prevention of SSI begins with appropriate selection of prophylactic antibiotics as discussed above (Table 2-2). In addition, consideration should be given to the classification of the preoperative field, which places a patient at increased risk for SSI despite antibiotic therapy (Table 2-3).

2. Treatment is to open the wound to allow drainage and culture if possible. Parenteral antibiotics are used only if extensive erythema or a deeper infection is present and are not

required for superficial infections. Wound infections in the perineum or after bowel surgery are more likely to be caused by enteric pathogens and anaerobes. More aggressive infections involving underlying fascia require operative dŽbridement and broad-spectrum IV antibiotics. Streptococcal wound infections present with severe local erythema and incisional pain. Penicillin G or ampicillin is effective adjuvant treatment. Patients with a severe necrotizing clostridial infection present with tachycardia and signs of systemic illness, pain, and crepitus near the incision. Treatment includes emergent operative debridement and metronidazole (500 mg IV every 6 hours) or clindamycin (600 to 900 mg IV every 8 hours).

D. Respiratory Infections. Pneumonia is diagnosed by the presence of fever, leukocytosis, purulent sputum production, and an infiltrate on CXR. After Gram stain and culture of the sputum and blood is performed, empiric antibiotics are started and aimed at nosocomial organisms in postoperative patients. Steps to help prevent pneumonia in the postoperative patient include incentive spirometry/pulmonary toilet, adequate pain control, early ambulation, and early extubation. Consideration should be given to viral causes of pneumonia as well.

E. *Clostridium difficile* **Infection (CDI)**

1. Definition. A diagnosis of CDI requires a positive stool *C. difficile* A or B toxin assay and clinically significant diarrhea *or* ileus. Successful treatment is demonstrated by decreased stool output to baseline and resolution of symptoms. Refractory CDI is defined as persistent symptoms despite 6 days of adequate treatment. Recurrence is classified as return of symptoms within 60 days after completion of full course of treatment plus either toxin positive stool or findings of pseudomembranes on colonoscopy.

TABLE 2-3 Classification of Surgical Wounds

Wound Class	Definition	Examples of Typical Procedures	Wound Infection Rate (%)	Usual Organisms
Clean	Nontraumatic, elective surgery; no entry of GI, biliary, tracheobronchial, respiratory, or GU tracts	Wide local excision of breast mass, inguinal hernia repair	2	*Staphylo-coccus aureus*

Clean-contami-nated	Respiratory, genitourinary, GI tract entered but minimal contamination	Gastrectomy, hysterectomy	<10	Related to the viscus entered
Contaminated	Open, fresh, traumatic wounds; uncontrolled spillage from an unprepared hollow viscus; minor break in sterile technique	Ruptured appendix; resection of unprepared bowel	20	Depends on underlying disease
Dirty	Open, traumatic, dirty wounds; traumatic perforated viscus; pus in the operative field	Intestinal fistula resection	28-70	Depends on underlying disease

GI, gastrointestinal.

2. Presentation. CDI may present in any patient who has received antibiotics, even after prophylactic perioperative dosing. There should be a low threshold for performing an assay for the *C. difficile* organism or toxin in postoperative patients with diarrhea. However, while less common, CDI can manifest as ileus with other systemic symptoms. CDI should be thought of as a spectrum of disease severity which then guides treatment:

a. Mild: Diarrhea, minimal symptoms

b. Moderate: IV fluids needed, abdominal pain, mucus or blood in stool, WBC 10 to 20K, low-grade fever, colitis on colonoscopy

c. Severe: Hypotension, peritonitis, WBC >20K, fever <38.5¡C

d. Life-threatening: Perforation, toxic megacolon, ischemia, transfusion requirement from colonic bleeding, pressor requirement

3. Treatment. Initial therapy includes fluid resuscitation, cessation of unnecessary antibiotics,

stopping pro-motility or antidiarrheal agents, and contact isolation precautions.

a. First time CDI with mild to moderate symptoms require administration of oral metronidazole (500 mg every 8 hours for 14 days) or oral vancomycin (125 mg every 6 hours for 10 to 14 days) if intolerant of metronidazole. Oral metronidazole is more effective than IV for CDI.

b. First time CDI with severe to life-threatening symptoms in patients tolerating oral medications is treated with oral metronidazole and oral vancomycin (dosed as above). Rectal vancomycin is not indicated. Consultations should be sought from gastroenterology, infectious disease, and colorectal surgery.

c. First time CDI with severe to life-threatening symptoms in patients intolerant of oral medications are treated with IV metronidazole (500 mg IV every 8 hours) and per tube vancomycin (500 mg per tube every 6 hours). In consultation with infectious disease, these measures are often supplemented by rectal or cecal vancomycin enemas. Little data exists to support the use of cecostomies or diverting loop ileostomies to facilitate administration of vancomycin enemas. Transition to oral treatment is preferable when tolerated. In severe cases, total abdominal colectomy may be required.

d. In the case of **CDI recurrence,** the first recurrence is treated identically to the initial episode. Subsequent episodes of recurrence require infectious disease consultation and usually require a prolonged, tapered course of oral vancomycin. Prolonged courses of metronidazole should be avoided.

e. For **Refractory CDI,** total abdominal colectomy may be required. Fidaxomicin has been used, but its use requires ID approval and guidance.

F. Intra-abdominal Abscesses often presents with asymptomatic leukocytosis, but may also exist with fever, abdominal pain, and tenderness. If generalized peritonitis is present, 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 intra-abdominal abscess is

drainage. Depending on size and location, percutaneous drainage under radiologic guidance may be an option. In other situations, operative debridement and drainage are required. Empiric antibiotic therapy should cover enteric pathogens and anaerobes. Empiric antifungal coverage should be considered in patients who have undergone recent GI surgery and present with severe sepsis or shock, have a recent history of TPN, have yeast present on Gram stain, or present with necrotizing pancreatitis. Consider broader coverage in patients with known MRSA or VRE. Duration of empiric treatment should not exceed 4 to 7 days and should be de-escalated as soon as possible.

G. Genitourinary Infections are the most common nosocomial infections in the postoperative patient. Foley catheters are a major risk factor and the likelihood of suffering from a UTI has

been shown to increase if catheters are left in place for more than 2 days (see ÒSCIP measuresÓ above). Treatment begins with obtaining a urine specimen for urinalysis and culture, followed by removal of the Foley catheter. After the urine is cultured, simple lower-tract infections can be managed with oral antibiotics. Ill patients or those with pyelonephritis require more aggressive therapy.

H. Prosthetic-deviceÑrelated Infections may present with fever, leukocytosis, and systemic bacteremia. Infection of prosthetic valves may present with a new murmur. Management may require removal of the infected device and the use of long-term antibiotics.

I. Catheter-related Infections are also diagnosed by the presence of fever, leukocytosis, and systemic bacteremia. Local erythema and purulence may sometimes be present around central venous catheter insertion sites. Management includes removal of the catheter and IV antibiotic coverage. In rare instances, the risk associated with line removal outweighs the benefits, and salvage treatment is, therefore, initiated (i.e., for hemodialysis catheters, pheresis catheters, and implanted ports). Line salvage should be undertaken only with the guidance of an infectious disease consultant.

J. Fungal Infections (primarily with *Candida* species) occur most commonly with long-term antibiotic administration and indwelling catheters. Evaluation of persistent fever without an identified bacterial source should include several sets of routine and fungal blood cultures, removal of all IV catheters, and examination of the retina for *Candida endophthalmitis.* Therapy includes amphotericin B, fluconazole, or micafungin.

VII. THROMBOTIC COMPLICATIONS

A. Deep Venous Thrombosis (DVT)

1. Presentation. Symptoms of DVT vary greatly, although classically they include pain and swelling of the affected extremity distal to the site of venous obstruction. DVTs may sometimes present as an unexplained leukocytosis or fever, particularly in patients who have poor functional status or have had prolonged ICU stays.

2. Examination. Findings may include edema, erythema, warmth, a palpable cord, or calf pain with dorsiflexion of the foot (Homan sign). Physical examination alone is notoriously inaccurate in the diagnosis of DVT.

3. Evaluation is primarily with noninvasive studies of the venous system, most notably B-mode ultrasonography plus color Doppler (duplex scanning). Reported sensitivity and specificity of this test for the detection of proximal DVT are greater than 90% with nearly 100% positive predictive value. This modality is less reliable in the detection of infrapopliteal thrombi, and a negative study in symptomatic patients should be followed by repeat examination in 48 to 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.

4. Prevention and Treatment. See Chapter 27 for a discussion of treatment of venous and lymphatic disease. Prevention should be paramount on any surgical service. Guidelines exist to risk stratify patients for DVT in order to guide mechanical and chemical prophylaxis (Table 2-4 and Fig. 2-1).

B. Pulmonary Embolism

1. PE is a very common postoperative occurrence, and autopsy studies demonstrate that it is more common than clinicians appreciate *(Chest.* 1995;108:978). Chest pain with a sensation of shortness of breath and low oxygen saturations should raise the possibility of PE.

2. Presentation. Symptoms of PE are neither sensitive nor specific. Mental status changes, dyspnea, pleuritic chest pain, and cough can occur, 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.

3. Evaluation

a. Preliminary studies. Findings that are suggestive of PE include arterial oxygen desaturation; nonspecific ST-segment or T-wave changes on ECG; and atelectasis, parenchymal abnormalities, or pleural effusion on CXR. Such classic signs as $S_1Q_3T_3$ on ECG or a prominent central pulmonary artery with decreased pulmonary vascularity (Westermark sign) on CXR are uncommon. ABG determination is a helpful adjunctive test; a decreased arterial oxygen tension (PaO_2) (<80 mm Hg), an elevated alveolar—arterial oxygen gradient, or a respiratory alkalosis may support clinical suspicion. Data obtained from these initial studies collectively may corroborate clinical suspicion but none of these alone is either sensitive or specific for PE. D-Dimer assays have a high negative predictive value; however, positive values, particularly in the setting of recent surgery, are less helpful because the postoperative period is one of many conditions that can cause an elevation of this test.

b. Spiral CT scan is becoming the primary diagnostic modality for PE. The advantages of CT scans for PE include increased sensitivity, the

ability to simultaneously evaluate other pulmonary and mediastinal abnormalities, greater after-hours availability, and the ability to obtain a CT venogram with the same dye load. This study subjects the patient to a contrast

dye load and requires a large (18 g or higher) IV in the antecubital vein, but it is not invasive. There are still wide variations in technology and institutional expertise with this modality, with reported sensitivities ranging from 57% to 100% and specificity ranging from 78% to 100%. If the patient is in extremis, consider intubating prior to obtaining the CT.

TABLE 2-4 Levels of Thromboembolism Risk and Recommended Thromboprophylaxis in Hospital Patients

Level of Risk		Approximate DVT Risk w/o Prophylaxis (%)	Suggested Thromboprophylaxis Options
Low	Minor surgery in mobile patients Medical patients who are fully mobile	<10	Early and ÒaggressiveÓ ambulation
Moderate	Most general, open gynecologic, or urologic surgery patients Medical patients who are on bed rest, ÒsickÓ	10-40	LMWH, unfractionated SC heparin BID or TID, fondaparinux
	Moderate VTE risk plus high bleeding risk		Mechanical throm-boprophylaxis
High	THA, TKA, HFS Major trauma Spinal cord injury	40-80	LMWH, fondaparinux, warfarin (INR 2-3)

High VTE risk plus high bleeding risk	Mechanical thromboprophylaxis

THA, total hip arthroplasty; TKA, total knee arthroplasty; HFS, hip fracture surgery; LMWH, low-molecular-weight heparin; VTE, venous thromboembolism; INR, international normalized ratio.
Adapted with permission from Geerts WH, et al. *Prevention of Venous Thromboembolism: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines.* 8th ed. *Chest.* 2008;133:381S-453S.

c. A **V/Q 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, 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, a high-probability V/Q scan should prompt treatment. Likewise, a normal scan virtually excludes PE (96%). Scans of intermediate probability require additional confirmatory tests.

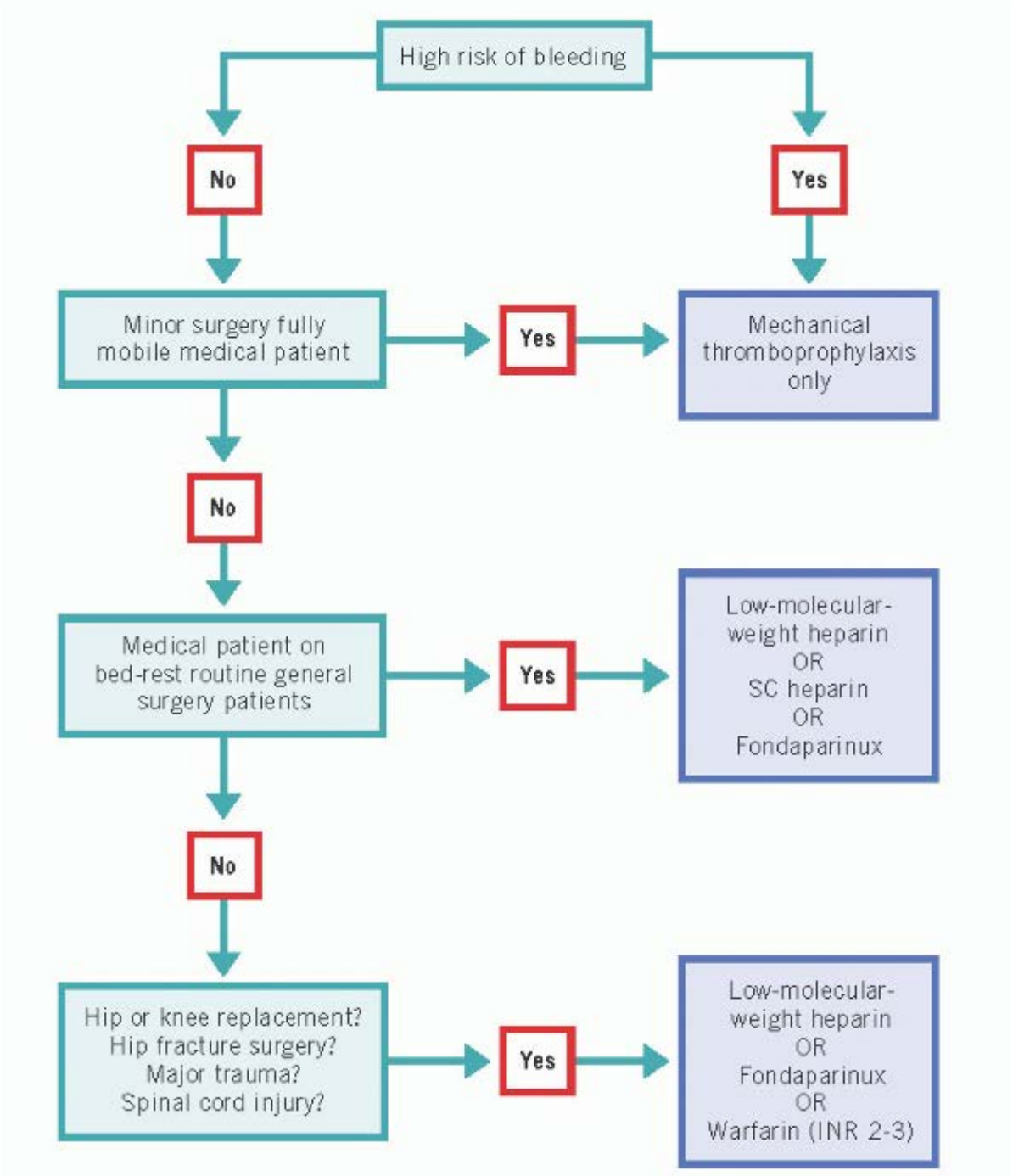

Figure 2-1 Algorithm for the selection of appropriate DVT prophylaxis.

d. Pulmonary angiography is the reference standard for the diagnosis of PE, but it is an invasive test with some element of risk. This test is rapidly being supplanted by spiral CT for most circumstances. Its use 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 without access to spiral

CT; 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.

4. Treatment

a. Supportive measures include administration of oxygen to correct hypoxemia and use of IV fluids to maintain BP. Hypotensive patients with high clinical suspicion of PE (i.e., high-risk patients, patients with acute right heart failure or right ventricular ischemia on ECG) require immediate transfer to an ICU, where hemodynamic monitoring and vasoactive medications may be required.

b. Anticoagulation with intravenous unfractionated heparin (UFH) or subcutaneous low-molecular-weight heparin (LMWH) should be started immediately with a target activated partial thromboplastin time (PTT) of 50 to 80 seconds. Oral warfarin can be started concurrently while heparin is continued until a therapeutic INR is achieved. Anticoagulation should continue for 6 months unless risk factors persist or DVT recurs.

c. 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.

d. Surgical patients with shock secondary to angiographically proven massive PE that is refractory to anticoagulation should be considered for either **transvenous embolectomy, open pulmonary embolectomy, or percutaneous removal via extracorporeal venous bypass.** These aggressive measures are rarely successful.

e. Inferior vena caval filter placement is indicated when a contraindication to anticoagulation exists, a bleeding complication occurs while receiving anticoagulation, or a DVT or PE recurs during anticoagulation therapy.

VIII. DIABETIC COMPLICATIONS

A. Diabetic Ketoacidosis (DKA)

1. Presentation. DKA may occur in any diabetic patient who is sufficiently stressed by illness or surgery. DKA patients who require an operation should be provided every attempt at correction of metabolic abnormalities before surgery, 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.

2. Evaluation should include blood glucose, CBC, serum electrolytes, serum

osmolarity, and ABG.

3. Treatment. Management should emphasize volume repletion, correction of acidosis and electrolyte abnormalities, and regulation of blood glucose with insulin infusion.

a. Restoration of intravascular volume should be initiated with isotonic (0.9%) saline or lactated Ringer solution without glucose. Patients without cardiac disease should receive 1 L or more of fluid per hour until urine output is greater than 30 mL/hour and hemodynamics have stabilized. Invasive hemodynamic monitoring may be required to guide fluid replacement in some circumstances (i.e., CHF, MI, and renal failure). Once intravascular volume has been restored, maintenance fluids of 0.45% NaCl with potassium (20 to 40 mEq/L) can be instituted. Dextrose should be added to fluids when the blood glucose is less than 400 mg/dL.

b. 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. Three ampules (150 mEq $NaHCO_3$) of bicarbonate can be added to 0.45% NaCl and given during the initial resuscitation in lieu of isotonic saline or LR.

c. Potassium replacement should be instituted immediately unless hyperkalemia with ECG changes exists, as there is significant total body hypokalemia in the setting of acidosis. In nonoliguric patients, replacement should begin with 30 to 40 mEq/hour of KCl for serum potassium of less than 3; 20 to 30 mEq/hour of KCl for serum potassium of 3 to 4; and 10 to 20 mEq/hour of KCl for potassium of greater than 4 mEq/L.

d. Blood glucose can be controlled with 10 units of insulin as an IV bolus followed by insulin infusion at 2 to 10 units per hour to a target range of 200 to 300 mg/dL. When the blood glucose falls below 400 mg/dL, 5% dextrose should be added to the IV fluids. Therapy is guided by hourly blood glucose determinations.

B. Nonketotic Hyperosmolar Syndrome

1. Presentation. This typically presents with severe hyperglycemia and dehydration without ketoacidosis. This occurs most often in elderly noninsulin-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 osmolarity of greater than 350 mOsm/L.

2. Treatment. 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.

CHAPTER 2: COMMON POSTOPERATIVE PROBLEMS

Multiple Choice Questions

1. Postoperative oliguria (<0.5 cc/kg/hour) with a fractional excretion of sodium of less than 1% is most consistent with:

a. Prerenal acute renal failure

b. Intrinsic acute renal failure

c. Chronic renal insufficiency

d. Acute tubular necrosis

e. None of the above

View Answer

2. Which of the following is NOT an appropriate treatment for acute delirium?

a. Minimizing nighttime disruptions such as laboratory draws

b. Benzodiazepines as needed for anxiety

c. Haloperidol for acute hyperactive delirium

d. Quetiapine at bedtime for sleep

e. Encouraging patient to be up out-of-bed during the day

View Answer

3. An otherwise healthy 65-year-old man is 16 hours postoperative from laparoscopic left colectomy for stage II colon adenocarcinoma. You are called by the nurse because over the last 3 hours his urine output has been less than 15 cc/hour. On evaluation, he reports feeling moderately anxious. His heart rate is 102 bpm and his blood pressure is 120/80. His physical examination is unremarkable. The appropriate immediate next step(s) in the workup and treatment of the patient include:

a. Obtain a chest x-ray

b. Measure hematocrit

c. Give a 1 L fluid bolus

d. Emergent transfer to the intensive care unit

e. Return to the operating room for re-exploration

f. B, C, and E

g. B and C

View Answer

4. The appropriate maximum duration of antibiotic coverage after routine uncomplicated general surgery is:

a. A single dose given in the operating room

b. IV antibiotics until the patient is afebrile and has a normal white blood cell count

c. A single intraoperative dose of antibiotics then oral antibiosis until discharge

d. 24 hours

e. 48 hours

View Answer

5. To prevent postoperative infectious complications, the Surgical Care Improvement Project recommends all of these interventions EXCEPT:

a. Prophylactic antibiotics within one hour of incision

b. Placing antibiotic-impregnated central venous catheters for complicated cases

c. Using clippers (not shaving) to remove body hair

d. Removing urinary catheters on postoperative day 1 or 2

e. Preventing perioperative hypothermia

View Answer

6. A 75-year-old female is in the postoperative care unit after a left hemicolectomy. You are called to evaluate her because she is suddenly disoriented and agitated. What is your next step?

a. Naloxone administration

b. Arterial blood gas

c. Electrocardiogram

d. Vital signs and pulse oximetry

e. Computed tomographic scan

View Answer

7. Your 69-year-old male patient is postoperative day 1 from a small bowel resection for small bowel obstruction. It is 10 pm, and for the second night in a row he believes that he is in the White House in spring of 1960. He is very agitated. Realizing that he is delirious you

order sleep hygiene and which of the following?

a. Dilaudid

b. Quetiapine

c. Benadryl

d. Zolpidem

e. Lorazepam

View Answer

8. A patient with a known history of alcohol use is now postoperative day 3. He begins to see ants on the walls and ceiling of his hospital room, is sweating profusely, and his heart rate increases from an average of 75 to 110 beats per minute. His most likely diagnosis is:

a. Wernicke encephalopathy

b. Alcoholic seizure

c. Delirium tremens

d. Benzodiazepine overdose

e. Delirium

View Answer

9. Which of the following causes of hypovolemia usually will not be present in a postoperative day 0 patient?

a. Bleeding

b. Anesthetic/analgesics

c. Antihypertensive medications

d. Under-resuscitation

e. Sepsis

View Answer

10. What day do most postoperative myocardial infarctions take place?

a. Postoperative day 0

b. Postoperative day 1

c. Postoperative day 2

d. Postoperative day 3

e. Postoperative day 4

View Answer

11. A 60-year-old patient with a history of congestive heart failure is 2 days postoperative from an exploratory laparotomy after a motor vehicle accident. He was extubated at the end of the surgery but now is complaining of shortness of breath. His pulse oximeter shows his oxygen saturation has decreased to around 89%. What are your next steps?

a. Supplemental oxygen, furosemide

b. Supplemental oxygen, digoxin

c. Intubation, furosemide

d. Intubation, digoxin

e. Supplemental oxygen, nicardipine

View Answer

12. A 70-year-old male is 3 days out from a right hemicolectomy, and he is starting to sip on clear liquids. Later that day he complains of nausea and has two bouts of emesis. Soon after, he complains of difficult breathing. After obtaining a chest x-ray, which is normal, you check his vital signs and note his temperature is 38.5°C. Which of the following is your top differential diagnosis?

a. Atelectasis

b. Gastric aspiration

c. Pneumonia

d. Congestive heart failure

e. Bowel perforation

View Answer

13. A 50-year-old male undergoes a right inguinal hernia repair as an outpatient. Six hours later, he is unable to void despite multiple attempts. What is the next step in his care?

a. Fluid challenge with 1 L normal saline

b. Foley insertion for 14 days

c. Flomax (tamsulosin)

d. Bladder scan, consider Foley placement

e. Discharge home

View Answer

14. A 68-year-old male status post a total abdominal colectomy with end ileostomy is postoperative day 2 when his creatinine increases from 1.2 to 1.8. His urine output is low normal and you decide to check an FENa, which comes back equal to 0.8%. What is his diagnosis?

a. Postrenal failure

b. Antibiotic nephrotoxicity

c. Urinary tract infection

d. Prerenal failure

e. Intrinsic renal failure

View Answer

15. Patients with nausea that is more than 24 hours out from surgery should be evaluated for an ileus, which may require which of the following treatments?

a. Return to the operating room for a revision

b. Ondansetron

c. Daily enemas

d. Valium

e. Nasogastric decompression

View Answer

3

Nutrition for the Surgical Patient

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Sara A. Buckman

Dietary nutrition supplies carbohydrates, lipids, and proteins that drive cellular metabolism. The chemical processes that maintain cellular viability consist of catabolic (breakdown) and anabolic (synthesis) reactions. Catabolism produces energy, whereas anabolism requires energy. While both processes occur concomitantly, our collective metabolism can be driven in either direction to balance our energy needs. Feeding drives synthesis and storage, whereas starvation promotes the mobilization of energy. In preparation to Òfight or flight,Ó physiologic stressors also mobilize energy stores. Populations stressed by surgery are at a unique metabolic disadvantage since they are often nutritionally restricted perioperatively. A thorough understanding of metabolism and its influences is necessary to assess for nutritional adequacy in surgical patients.

NUTRIENT METABOLISM

I. CARBOHYDRATES.

Carbohydrates are the primary energy source for the body, providing 30% to 40% of calories in a typical diet. Brain and red blood cells rely almost exclusively on a steady supply of glucose to function. Each gram of enteral carbohydrate provides 4 kcal of energy, whereas parenteral formulations are hydrated and thus provide only 3.4 kcal/g.

A. Glucose Stores. During fed states, hyperglycemia leads to insulin secretion, promoting glycogen synthesis. About 12 hours worth of glycogen is available in the liver and skeletal muscles, which can provide a steady supply of glucose between meals. During starvation and stress, depleted glycogen stores cause the release of glucagon, which promotes hepatic gluconeogenesis from amino acids. If dietary carbohydrates are not resumed, glucagon promotes ketone body formation from lipids, which the brain can utilize. A minimum intake of 400 calories of carbohydrate per day minimizes protein breakdown.

B. Carbohydrate digestion is initiated by the action of salivary amylase, and absorption is generally completed within the first 1 to 1.5 m of small intestine. Salivary and pancreatic amylases cleave starches into oligosaccharides. Surface oligosaccharides then hydrolyze and transport these molecules across the gastrointestinal (GI) tract mucosa. Diseases that result in generalized mucosal flattening (e.g., celiac sprue, Whipple disease, and hypogammaglobulinemia) may cause diminished uptake of carbohydrates because of resultant deficiencies in

oligosaccharides.

II. LIPIDS.

Fatty acids are the functional units of lipid metabolism. They comprise 25% to 45% of calories in the typical diet. During starvation, lipids

provide the majority of energy in the form of ketone bodies converted by the liver from long-chain fatty acids. Each gram of lipid provides 9 kcal of energy.

A. Lipid Storage. Lipids are important energy sources for the heart, liver, and skeletal muscle. Free fatty acids are bound to a glycerol backbone and join to form triacylglycerols during fed states. Triglycerides are stored in adipocytes and can be mobilized in times of stress or starvation. Lipolysis is stimulated by steroids, catecholamines, and glucagon, whereas insulin promotes synthesis and storage.

B. Digestion and absorption of lipids is complex and utilizes nearly the entire GI tract. Coordination between biliary and pancreatic secretions, as well as a functional jejunum and ileum are necessary. Fat in the duodenum stimulates cholecystokinin and secretin release, leading to gallbladder contraction and pancreatic enzyme release, respectively. Pancreatic secretions contain a combination of lipase, cholesterol esterase, and phospholipase A2. In the alkaline environment of the duodenum, lipase hydrolyzes triglycerides to one monoglyceride and two fatty acids. Bile salts emulsify these fats into micelles, thereby facilitating absorption across the intestinal mucosal barrier by creating a hydrophilic outer coating. Bile salts are then reabsorbed in the terminal ileum to maintain the bile salt pool **(i.e., the enterohepatic circulation).** Consequently, major ileal resection may lead to depletion of the bile salt pool and subsequent fat malabsorption. Clinical lipid 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.

III. PROTEIN.

Amino acids are the functional units of protein metabolism. Proteins are important for the biosynthesis of enzymes, structural molecules, and immunoglobulins. When energy needs are unmet by nutrition, muscle breakdown yields amino acids for hepatic gluconeogenesis, which can lead to wasting and deconditioning in severe circumstances. Each gram of protein can be converted into 4 kcal of energy.

A. Digestion of proteins yields dipeptides and single amino acids, which are actively absorbed. Gastric pepsin initiates the process of digestion. Pancreatic proteases, activated on exposure to enterokinase found throughout the duodenal mucosa, are the principal effectors of protein degradation. Once digested, almost 50% of protein absorption occurs in the duodenum, and complete protein absorption is achieved by the midjejunum.

B. Metabolism of absorbed amino acids occurs initially in the liver where portions of amino acids are extracted to form circulating proteins.

STRESS METABOLISM

Alterations in metabolism due to physiologic stress share similar patterns with simple starvation. Regardless of the stimulus, our conserved response to stress is the sameÑ catabolic shifts mobilize energy stores in order to prepare us for Òfight or flight.Ó

I. SIMPLE STARVATION.

After an overnight fast, liver glycogen is rapidly depleted as glucagon responds to falling serum glucose levels. Carbohydrate stores are exhausted after 24 hours. For the first few days during starvation, caloric needs are met by fat and protein degradation. Most of the protein is from breakdown of skeletal and visceral muscle, which is converted to glucose via hepatic gluconeogenesis. The brain preferentially uses this endogenously produced glucose, with the remainder consumed by red blood cells and leukocytes. Within approximately 10 days of starvation, the brain adapts and uses fat in the form of ketoacids as its fuel source. Use of ketoacids has a protein-sparing effect.

II. PHYSIOLOGIC STRESS.

The interaction of metabolic and endocrine responses that result from major operation, trauma, or sepsis can be divided into three phases.

A. 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). Following 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 reduction in insulin.

B. The early anabolic phase is also called the *corticoid withdrawal phase* as the body shifts from catabolism to anabolism. The timing of this event is variable, depending on the severity of stress, 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, including 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.

C. 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 higher caloric

content of fatÑ the primary energy stores deposited during the early anabolic phaseÑas compared to protein.

NUTRITIONAL ASSESSMENT

Nutrition plays a vital and often underappreciated role in the recovery of patients from surgery. It is estimated that between 30% and 50% of hospitalized patients are malnourished. While most healthy patients can tolerate 7 days of starvation, patients affected by major trauma, surgery, sepsis, or other critical illnesses require nutritional intervention earlier. Poor nutrition has deleterious effects on wound healing and immune function, which increases postoperative morbidity and mortality. Identification of those at risk for malnutrition is made through ongoing clinical assessments by vigilant clinicians.

BMI Categories (kg/m^2)

Underweight	<18.5
Normal	18.5-24.9
Overweight	25-29.9
Obese	30-39.9
Morbid obesity	>40

I. TYPES OF MALNUTRITION

A. Overnutrition. Obesity as defined by body mass index (BMI) >30.

B. Undernutrition

1. Caloric

a. Marasmus is characterized by inadequate protein *and* caloric intake, typically caused by illness-induced anorexia. It is a chronic nutritional deficiency marked by losses in weight, body fat, and skeletal muscle mass. Visceral protein stores remain normal, as do most laboratory indices.

2. Noncaloric

a. Kwashiorkor is characterized by catabolic protein loss, resulting in hypoalbuminemia and generalized edema. This form of malnutrition develops with prolonged starvation or severe stress. Even in a well-nourished patient, a severe stress (e.g., major burn or prolonged sepsis) may rapidly lead to depletion of visceral protein stores and impairment in immune function.

b. Vitamins and trace elements. In addition to the principal sources of energy, our metabolic machinery also requires various other substances in order to function efficiently. Vitamins are involved with wound healing and healthy immune function while many trace elements are important as cofactors and enzymatic catalysts. These substances cannot be synthesized *de novo* and therefore must be part of dietary intake. Deficiencies can have a multitude of detrimental effects (Tables 3-1 and 3-2).

II. CLINICAL ASSESSMENT

A. History. Every good clinical assessment should begin with a thorough history from the patient. Specific inquiries pertinent to nutritional status include recent history of weight fluctuation with attention as to the timing and intent.

Recent weight loss (5% in the last month or 10% over 6 months) or a current body weight of 80% to 85% (or less) of ideal body weight suggests severe malnutrition. Energy intake ²50% of estimated energy requirement

for ³5 days also indicates severe malnutrition. Anorexia, nausea, vomiting, dysphagia, odynophagia, gastroesophageal reflux, or a history of generalized muscle weakness should prompt further evaluation. A complete history of current medications is essential to alert caretakers to potential underlying deficiencies as well as drugÑnutrient interactions.

TABLE 3-1 Vitamins

Vitamin	Function	Deficiency State
Fat Soluble		
A (Retinol)	Rhodopsin synthesis	Xerophthalmia, keratomalacia
D (Cholecalciferol)	Intestinal calcium absorption, bone remodeling	Rickets (children), osteomalacia (adults)

E (α-Tocopherol)	Antioxidant	Hemolytic anemia, neurologic damage
K (Naphthoquinone)	γ-Carboxylation of glutamate in clotting factors	Coagulopathy (deficiency in factors II, VII, IX, and XI)
Water Soluble		
B_1 (Thioamide)	Decarboxylation and aldehyde transfer reactions	Beriberi, neuropathy, fatigue, heart failure
B_2 (Riboflavin)	Oxidation-reduction reactions	Dermatitis, glossitis
B_5 (Niacin)	Oxidation-reduction reactions	Pellagra (**d**ermatitis, **d**iarrhea, **d**ementia, **d**eath)
B_6 (Pyridoxal phosphate)	Transamination and decarboxylation reactions	Neuropathy, glossitis, anemia
B_7 (Biotin)	Carboxylation reactions	Dermatitis, alopecia
B_9 (Folate)	DNA synthesis	Megaloblastic anemia, glossitis
B_{12} (Cyanocobalamin)	DNA synthesis, myelination	Megaloblastic anemia, neuropathy
C (Ascorbic acid)	Hydroxylation of hormones, hydroxylation of proline in collagen synthesis, antioxidant	Scurvy

B. Physical examination may identify muscle wasting (especially thenar and temporal

muscles), loose or flabby skin (indicating loss of subcutaneous fat), and peripheral edema and/or ascites (as a result of hypoproteinemia). Subtler findings of nutritional deficiency include skin rash, pallor, glossitis, gingival lesions, hair changes, hepatomegaly, neuropathy, and dementia. Hand grip strength has also been used as a functional parameter to assess malnutrition. Limitations include heavily sedated patients or those unable to perform a valid hand grip (*Nutr Clin Pract.* 2013;28:639-650). In addition, clinical signs of inflammation including fever, hypothermia, tachycardia, or hyperglycemia may reveal acute disease or injury-related malnutrition (*J Parenter Enteral Nutr.* 2012;36:197-204).

TABLE 3-2 Minerals

Trace Element	Function	Deficiency
Chromium	Promotes normal glucose utilization in combination with insulin	Glucose intolerance, peripheral neuropathy
Copper	Component of enzymes	Hypochromic microcytic anemia, neutropenia, bone demineralization, diarrhea
Fluorine	Essential for normal structure of bones and teeth	Caries
Iodine	Thyroid hormone production	Endemic goiter, hypothyroidism, myxedema, cretinism
Iron	Hemoglobin synthesis	Hypochromic microcytic anemia, glossitis, stomatitis
Manganese	Component of enzymes, essential for normal bone structure	Dermatitis, weight loss, nausea, vomiting, coagulopathy

Molybdenum	Component of enzymes	Neurologic abnormalities, night blindness
Selenium	Component of enzymes, antioxidant	Cardiomyopathy
Zinc	Component of enzymes involved in metabolism of lipids, proteins, carbohydrates, nucleic acids	Alopecia, hypogonadism, olfactory and gustatory dysfunction, impaired wound healing, acrodermatitis enteropathica, growth arrest

C. Laboratory tests are nonspecific indicators of the *degree of illness* rather than strict markers of nutrition. Albumin, prealbumin, and transferrin vary with nutritional status, as well as with the body's response to inflammation: Hepatic metabolism decreases synthesis and diluted serum levels result from capillary leak (*J Am Diet Assoc.* 2004;104:1258-1264). As such these levels should be interpreted with caution. Other laboratory indicators of inflammation include C-reactive protein (CRP), white blood cell count, and blood glucose levels.

D. Imaging tests can also provide valuable information on nutritional assessment, particularly in patients requiring serial imaging. Cross-sectional area of specific muscle groups can be measured using computed tomography (CT) or ultrasonography.

III. ESTIMATION OF ENERGY NEEDS

A. Indirect calorimetry remains the gold standard in measuring energy expenditure in the clinical setting. It measures CO_2 production and O_2 consumption during rest and exercise at steady-state to calculate total energy expenditure (TEE).

B. Basal energy expenditure (BEE) can be predicted by using the Harris-Benedict equation (in kilocalories per day):

For **men** equals

66 + [13.7 × weight (kg)] + [5 × height (cm)] Ð [6.8 × age (years)].

For **women** equals

655 + [9.6 × weight (kg)] + [1.8 × height (cm)] Ð [4.7 × age (years)].

This equation is generally accurate in healthy subjects when compared to indirect calorimetry; however, it is much less accurate in critically ill patients and those at the extremes in weight. BEE must be adjusted for activity and injury level (Table 3-3).

C. Respiratory quotient (RQ) represents the ratio of expired CO_2 to O_2 consumed. This ratio can provide valuable information regarding the primary energy substrate being utilized. An RQ of 1 indicates glucose oxidation and 0.8 indicates protein utilization, while 0.7 indicates fat metabolism. Patients receiving excess carbohydrates thus have higher CO_2 production that could lead to difficulty weaning from the ventilator.

D. BMI. Patient's BMI can be used to estimate caloric requirements in hospitalized patients. BMI can be calculated by the following equations:

BMI	Daily Energy Needs (kcal/kg/day)
<15	35-40
15-19	30-35
20-25	20-25
26-29	15-17
>29	15

TABLE 3-3 Basal Energy Expenditure Activity and Injury Factor Adjustments

Activity Factors

Bedbound = 1.2
Out of bed = 1.3
Healthy normal activity =1.5

Injury Factors

Surgery	Minor = 1.0 to 1.2 Major =1.1 to 1.3

Infection	Mild = 1.0 to 1.2 Moderate = 1.2 to 1.4 Severe = 1.4 to 1.8
Major skeletal or blunt trauma = 1.35	
Head trauma = 1.6 to 1.8	
Sepsis = 1.6 to 1.8	
Burn	<20% BSA=1.2 to 1.5 20-40% BSA = 1.5 to 1.8 >40% BSA = 1.8 to 2.0

Adapted from *Handbook of Medical Nutrition Therapy: The Florida Diet Manual. 2000.* Tallahassee, FL: Florida Dietetic Association; 2000. p I1.7.

E. Estimates of Protein Requirements. Patients who are nonstressed should receive 0.8 to 1.2 g/kg/day of protein. Those who are critically ill generally require 1.2 to 1.5 g/kg/day, and burn, septic, and obese patients may require 1.5 to 2 g/kg/day.

NUTRITION ADMINISTRATION

Surgical patients present a unique set of challenges to clinicians who must determine when, how, and what to feed them. Safe administration of an oral diet requires that the patient should have an intact chewing/swallowing mechanism along with a functioning alimentary tract. The timing, route, and type of nutrition are important considerations in surgical patients. Figure 2-1 provides an algorithm for selecting nutritional support for the hospitalized patient.

I. INITIAL TIMING OF ADMINISTRATION.

Open abdominal surgery produces a paralytic ileus of variable length that alters the digestion and absorption of nutrients. Resolution, marked by the passage of flatus, occurs in most patients within 72 hours of surgery and is symptomatic of functional GI continuity. Return of bowel function begins with the small intestine within hours of surgery, is followed by the stomach at 48 hours, and finally by the colon, typically at 72 hours. Traditionally, postoperative patients were maintained on dextrose-containing IV fluids and kept NPO for up to 7 days until evidence of bowel function returned; however, several strategies have recently emerged to shorten

postoperative ileus.

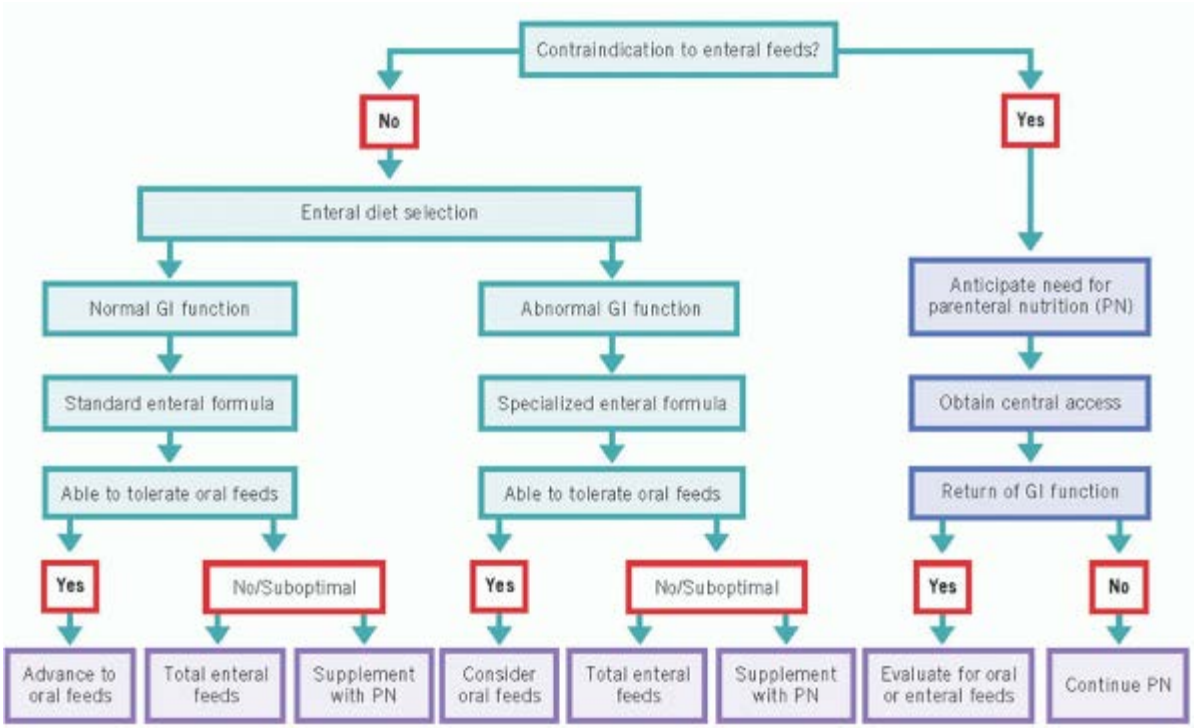


Figure 3-1 Nutritional approach to the hospitalized patient. Adapted from Ukleja A, Freeman KL, Gilbert K, et al.; Task Force on Standards for Nutrition Support: Adult Hospitalized Patients, and the American Society for Parenteral and Enteral Nutrition Board of Directors. Standards for Nutrition Support: Adult Hospitalized Patients. *Nutr Clin Pract.* 2010;25:403.

A. Strategies to Hasten GI Recovery Following Abdominal Surgery:

1. Laparoscopic surgery is less traumatic and has been associated with shorter periods of ileus versus open approaches.

2. Epidural analgesia with an infusion of local anesthetic minimizes dependence on narcotics for pain control, thus limiting their adverse effects on gut motility (*Cleve Clin J Med.* 2009;76:641-648).

3. Enhanced Recovery After Surgery (ERAS) is a multimodal perioperative care pathway that makes use of preoperative counseling, optimization of nutrition with early enteral feeding, a peripherally acting μ-opioid antagonist (i.e., alvimopan), standardized analgesic and anesthetic regimens, and early mobilization to achieve early recovery for patients undergoing major surgery. It has been associated with shorter hospital stays (up to 2.5 days shorter than non-ERAS

patients) and reductions in postoperative complications by up to 50% (*Clin Nutr.* 2010;29:434-440).

II. ROUTE OF ADMINISTRATION.

Oral administration of nutrition is the preferred route since it is the most physiologic and the least invasive. In patients with a functioning GI tract, several requirements must still be met, however, before initiating an oral diet.

A. Mental Alertness and Orientation. Patients who have altered mentation are at increased risk for aspiration and should not begin an oral nutrition regimen.

B. Intact Chewing/Swallowing Mechanism. Patients who have had a stroke or undergone pharyngeal surgery may have difficulty swallowing. They may be candidates for modified oral diets, such as mechanical, soft, or pureed.

III. DIET SELECTION

A. Transitional diets minimize digestive stimulation and colonic residue while providing more calories than IV fluids alone in patients recovering from postoperative ileus. Advancement to the next stage should be predicated on frequent assessment of the patient's bowel function in the absence of nausea, vomiting, or distention.

1. Clear liquids provide fluids mostly in the form of sugar and water. Indications for a clear liquid diet include short-term use after an acute illness or surgery when there is an intolerance for foods; to restrict undigested material in the GI tract; and to prepare the bowel for surgery or a GI procedure. This diet provides between 700 and 1,000 kcal per day. Examples include carbonated beverages, clear gelatin, fruit ices and popsicles, most juices, coffee, tea, and clear broth.

2. Full liquids include foods that are liquid at body temperature, such as gels and frozen liquids. In addition, full liquids contain dairy products and would not be appropriate in patients who are lactose intolerant. Transition to full liquids is good for patients who have undergone head and neck surgery and thus may have some difficulty swallowing postoperatively. Full liquids provide approximately 1,200 kcal and 40 g

of protein per day; however, providing between meal supplementation will increase the caloric and protein intake *(Manual of Clinical Nutrition Management, Morrison, Inc,* 2013).

3. Regular diet represents an unrestricted regimen that includes various foods designed to meet all caloric, protein, and elemental needs.

B. Surgery-specific Diets

1. Dumping syndrome diet/postgastrectomy diet. Procedures that reduce the reservoir capacity of the stomach, compromise the pyloric sphincter, or alter secretion of GI hormones can produce a Òdumping syndromeÓ postoperatively. When undigested, hyperosmolar, food reaches

the jejunum, massive fluid shifts into the intestine to equalize osmotic pressure, leading to nausea, diaphoresis, tachycardia, bloating, abdominal cramping, and diarrhea. This diet encourages small, frequent meals limiting beverages and liquids at meals, in addition to limiting the intake of simple carbohydrates and sugars. This diet is moderate in fat but high in protein. Fiber is gradually introduced and may help slow gastric emptying *(Manual of Clinical Nutrition Management, Morrison, Inc,* 2013).

Procedures That Can Cause Dumping Syndrome

Standard Whipple procedure

Partial/total gastrectomy/antrectomy

Esophagectomy

Pyloromyotomy

2. Postgastric bypass. Patients who undergo bariatric surgery have unique postoperative nutritional needs as well as long-term goals of sustained weight loss. **Postbariatric surgery transitional diets** emphasize small meals without added sugar to avoid stretching the pouch and dumping syndrome, respectively. Patients progress from clear liquid to the regular bariatric diet in approximately 6 weeks. During this time, patients are taught to eat three to five small meals per day slowly over a period of 45 minutes in order to stay fuller longer. The focus should be on chewing foods longer and avoiding drinking beverages at the same time. Protein should provide 25% of intake, fats 25% to 30%, and carbohydrates 50% while limiting the amount of concentrated sugars. After a full-liquid diet starts, supplementation with a chewable multivitamin with minerals should be initiated. Additional supplementation with iron, calcium, and vitamin B_{12}, may be required.

3. Low-residue diet provides <10 g/day of dietary fiber. It is used to prevent the formation of an obstruction when there is intestinal narrowing, delay intestinal transit, reduce fiber in the colon pre- and postoperatively, and allow bowel rest in times of colonic inflammation and/or irritation. Patients who benefit from a low-fiber diet include

those going through an acute phase of inflammatory bowel disease (IBD), diverticulitis, or radiation enteritis.

4. Open abdomen guidelines from the Western Trauma Association advocate early enteral feeding (after resuscitation is complete) since this is associated with increased fascial closure rates, decreased complication rates, and decreased mortality *(J Trauma Acute Care Surg.* 2012;73: 1380-1387).

NUTRITIONAL SUPPORT

The need for nutritional support should be assessed continually in patients both preoperatively and postoperatively. Most elective surgical patients have adequate fuel reserves to withstand common catabolic stresses and partial starvation for up to 7 days and do not benefit from perioperative nutritional support (*Nutrition.* 2000;16:723Ñ 728). For these patients, IV fluids with appropriate electrolytes and a minimum of 100 g glucose daily (to minimize protein catabolism) is adequate. However, even well-nourished patients can quickly become malnourished following a major operation or trauma. Without nutritional intervention, these patients may suffer complications related to impaired immune function and poor wound healing from depleted visceral protein stores. Patients with a significant degree of preoperative malnutrition have less reserve, tolerate catabolic stress and starvation poorly, and are at higher risk for postoperative complications.

I. ROUTES OF NUTRITIONAL SUPPORT

A. Enteral. In general, the enteral route is preferred to the parenteral route. Enteral feeding is simple, physiologic, and relatively inexpensive. Enteral feeding maintains the GI tract cytoarchitecture and mucosal integrity (via trophic effects), absorptive function, and normal microbial flora. This results in less bacterial translocation and endotoxin release from the intestinal lumen into the bloodstream. Choice of appropriate feeding site, administration technique, formula, and equipment may circumvent these problems. Enteral feeding is indicated for patients who have a functional GI tract but are unable to sustain an adequate oral diet, and it is contraindicated in patients with an intestinal obstruction, upper GI bleeding, severe diarrhea, intractable vomiting, enterocolitis, a high-output enterocutaneous fistula, and severe IBD. Understanding of appropriate feeding sites, administration techniques, formulas, and equipment is necessary.

1. Feeding tubes. Nasogastric, nasoduodenal or jejunal, gastrostomy, and jejunostomy tubes are available for the administration of enteral feeds. Percutaneous gastrostomy tubes can be placed endoscopically or under fluoroscopy.

2. Enteral feeding products. Various enteral formulas are commercially available. Standard solutions provide 1 to 2 kcal/mL. The available dietary formulations for enteral feedings can be classified as standard, elemental, or semi-elemental. Standard (polymeric) formulas include synthetic and blenderized formulas. There are specialized formulas that are disease specific such as formulas for renal disease, hepatic disease, pulmonary disease, and diabetes mellitus. The

formulas include those containing fiber, omega-3 fatty acids, arginine, or glutamine.

3. Enteral feeding protocols. It is recommended to start with a fullstrength formula at a slow rate, which is steadily advanced. This reduces the risk of microbial contamination and achieves goal intake earlier. Conservative initiation and advancement are recommended for patients who are critically ill, those who have not been fed for some time, and those receiving a high osmolarity or calorie-dense formula.

a. Bolus feedings are reserved for patients with gastrostomy feeding tubes. Feedings are administered by gravity, begin at 50 to 100 mL every 4 hours, and are increased in 50-mL increments until goal intake is reached (usually 240 to 360 mL every 4 hours). Tracheobronchial aspiration is a potentially serious complication because feedings are prepyloric. This type of feeding should be avoided in those with a high risk of aspiration, disorders of glucose metabolism, or fluid management concerns. To reduce the risk of aspiration, the patient's head and body should be elevated to 30 to 45 degrees during feeding and for 1 to 2 hours after each feeding. The gastric residual volume should be measured before administration of the feeding bolus. If this volume is greater than 50% of the previous bolus, the next feeding should be held. The feeding tube should be flushed with approximately 30 mL of water after each use. Free water volume can be adjusted as needed to treat hypo- or hypernatremia.

b. Continuous infusion administered by a pump is generally required for nasojejunal, gastrojejunal, or jejunal tubes. Feedings are initiated at 20 mL/hour and increased in 10- to 20-mL/hour increments every 4 to 6 hours until the desired goal is reached, with 30 mL of water flushes every 4 hours. Feedings should be held or advancement should be slowed if abdominal distension or pain develops. For some patients, the entire day's feeding can be cycled over 8 to 12 hours at night to allow the patient mobility free from the infusion pump during the day.

4. Conversion to oral feeding. When supplementation is no longer needed, an oral diet is resumed gradually. In an effort to stimulate appetite, enteral feeding can be modified by the following measures:

a. Providing fewer feedings.

b. Holding daytime feedings.

c. Decreasing the volume of feedings. When oral intake provides approximately 60% of the patient's energy requirements and 100% of the patient's fluid requirements, the physician should consider discontinuing tube feeding.

5. Complications

a. Metabolic derangements. Abnormalities in serum electrolytes, calcium, magnesium, and phosphorus can be minimized through vigilant monitoring. Hypernatremia may lead to the development of mental lethargy or obtundation. This is treated with the slow administration of

free water by giving either dextrose 5% in water (D_5W) intravenously or additional water in the tube feedings.

Hyperglycemia may occur in patients receiving tube feeds and is particularly common in preexisting diabetics or in the setting of sepsis. A sliding scale insulin protocol along with long-acting agents should be used to treat hyperglycemia in tube-fed patients.

b. Refeeding syndrome is a potentially lethal complication in patients who are severely malnourished. Alterations in phosphate, potassium, magnesium, and thiamine can be seen which can lead to harmful effects on the cardiac, respiratory, hepatic, neuromuscular, and hematologic systems (*Eur J Clin Nutr.* 2008;62:687-694).

c. Clogging can usually be prevented by careful routine flushing of the feeding tube. Instillation of carbonated soda or pancreatic enzyme replacement is sometimes useful for unclogging feeding tubes. Tubes should be flushed both before and after medication administration and liquid medications should be used whenever possible due to the possibility of crushed meds clogging tubes.

d. Tracheobronchial aspiration of tube feeds may occur with patients who are fed into the stomach or proximal small intestine and can lead to major morbidity. Patients at particular risk are those with central nervous system abnormalities and those who are sedated. Precautions include frequent assessment of gastric residuals as well as head of bed elevation.

e. High gastric residuals 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 the underlying cause. Gastroparesis frequently occurs in diabetic or head-injured patients. Promotility agents such as metoclopramide or erythromycin may aid in gastric emptying. 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.

f. Diarrhea occurs in 10% to 20% of patients; however, other causes of diarrhea (e.g., *Clostridium difficile* colitis) should be considered. Diarrhea may result from an overly rapid increase in the volume of hyperosmolar tube feedings, medications, or substances (e.g., lactulose or sorbitol). If other causes of diarrhea can be excluded, the volume or concentration of tube feedings should be decreased. Soluble fiber may also be added but should be used with caution within the first 7 days after an intestinal operation. If no improvement occurs, a different formula should be used. Antidiarrheal agents such as loperamide should be reserved for patients with severe diarrhea who have had infectious etiologies excluded.

B. Parenteral nutrition is indicated for patients who require nutritional support but cannot meet their needs through oral intake and for whom enteral feeding is contraindicated or not tolerated, including extensive small bowel resection, perforated small bowel, high output

enterocutaneous fistula, severe emesis or diarrhea, bowel obstruction, severe GI bleed, or hemodynamic instability with high pressor requirements.

1. Peripheral parenteral nutrition (PPN) is administered through a peripheral IV catheter. The osmolarity of PPN solutions generally is limited to 900 mOsm (÷12% dextrose solution) to avoid phlebitis. Consequently, unacceptably large volumes (>2,500 mL) are necessary to meet the typical patient's nutritional requirements. Temporary nutritional supplementation with PPN may be useful in selected patients but is not typically indicated.

2. Total parenteral nutrition (TPN) provides complete nutritional support. The solution, volume of administration, and additives are individualized on the basis of an assessment of the nutritional requirements.

a. Access. TPN solutions must be administered through a central venous catheter, preferably placed in either the subclavian or internal jugular vein.

b. TPN solutions are generally administered as a three-in-one admixture of protein, as amino acids (10%, 4 kcal/g); carbohydrate, as dextrose (70%, 3.4 kcal/g); and fat, as a lipid emulsion of soybean, safflower, or olive oil (9 kcal/g). Alternatively, the lipid emulsion can be administered as a separate IV ÒpiggybackÓ infusion. Typical dosing may be calculated using the patient's BMI to estimate the total daily caloric requirement. Patients who are not stressed typically need 0.75 g/kg/IBW of protein/day. Patients who are critically ill without renal or hepatic dysfunction need 1 to 1.5 g/kg/IBW of protein/day. For the nonamino acid calories, it is feasible to start with 60% to 70% as dextrose and 30% to 40% as fat emulsion, but not more than 1 g/kg/day of fat.

c. Additives. Other elements can be added to the basic TPN solutions.

(1) Electrolytes (sodium, potassium, chloride, acetate, calcium, magnesium, phosphate) should be adjusted daily. The number of cations and anions must balance: This is achieved by altering the concentrations of chloride and acetate. The calcium:phosphate ratio must be monitored to prevent salt precipitation.

(2) Medications such as H_2-receptor antagonists and insulin can be administered in TPN solutions. Regular insulin should initially be administered subcutaneously on the basis of the blood glucose level. After a stable insulin requirement has been established, insulin can then be administered via TPN solutionÑ generally at two-thirds the daily subcutaneous insulin dose.

(3) Vitamins and trace elements are added daily using a commercially prepared mixture that includes copper, chromium, selenium, manganese, and zinc. Vitamin K is not included in most multivitamin mixtures and must be added separately.

C. Administration of TPN is most commonly a continuous infusion. A new three-in-one admixture bag of TPN is administered daily at a constant infusion rate over 24 hours. Additional maintenance IV fluids are unnecessary, and total infused volume should be kept constant while

nutritional content is increased. Serum electrolytes should be obtained and TPN adjusted until the patient can be maintained on a stable regimen.

1. 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 IV access who require administration of other medications, 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 to 16 hours, most commonly at night. This should not be done until metabolic stability has been demonstrated for patients on standard, continuous TPN infusions.

D. Discontinuation of TPN should take place when the patient can consistently satisfy 60% of their caloric and protein needs with oral intake or enteral feeding and 100% of the daily fluid needs. The calories provided by TPN can be decreased in proportion to calories from the patient's increasing enteral intake. To discontinue TPN when hypoglycemia is a concern, the infusion rate should be halved for 1 hour, halved again the next hour, and then discontinued. Tapering in this manner prevents rebound hypoglycemia from hyperinsulinemia. It is not necessary to taper the rate if the patient demonstrates glycemic stability when TPN is abruptly discontinued (i.e., cycled TPN) or receives less than 1,000 kcal/day.

E. Complications Associated with TPN

1. Catheter-related complications can be minimized by strict aseptic technique and routine catheter care.

2. Metabolic complications include electrolyte abnormalities and glucose homeostasis. While it was previously thought that strict maintenance of serum glucose levels below 110 mg/dL improves mortality, it was shown in the NICE-SUGAR study that intensive glucose control actually increased mortality. Therefore blood glucose levels should be kept below 180 mg/dL *(N Engl J Med.* 2009;360:1283-1297).

3. Cholestasis is another common metabolic complication of long-term parenteral nutrition. This is due to the lack of enteral stimulation for gallbladder contraction. Cholestatic liver disease may ultimately lead to biliary cirrhosis, which is treated with transplantation.

CHAPTER 3: NUTRITION

Multiple Choice Questions

1. After a patient eats a large meal of carbohydrates, which of the following occurs?

a. Ketone bodies form

b. Oligosaccharides are cleaved by salivary amylase

c. Cholecystokinin is released

d. Muscle breakdown occurs

e. Glycogen is depleted

View Answer

2. During simple starvation, how long does it take for carbohydrate stores to be exhausted?

a. 48 hours

b. 6 hours

c. 18 hours

d. 12 hours

e. 10 days

View Answer

3. A 21-year-old male is involved in a motor vehicle collision and suffers multiple rib fractures, a humerus fracture, and a femur fracture. He is intubated in the intensive care unit. How much protein does he require?

a. 1.2 to 1.5 g/kg/day

b. 0.8 to 1 g/kg/day

c. >2 g/kg/day

d. 1.5 to 2 g/kg/day

e. <0.8 g/kg/day

View Answer

4. A 45-year-old male presents to the ER today with perforated diverticulitis; however, he has been ill for the last week. Clinical signs of acute disease-related inflammation include which of the following?

a. Hypoglycemia

b. Hypothermia

c. Bradycardia

d. Dyspnea

e. Chest pain

View Answer

5. A 36-year-old female undergoes a laparoscopic cholecystectomy for biliary colic and suffers a common bile duct injury. She is approximately 5 days postop and is about to start a diet. Her preoperative weight is

150 lb and she is 5 feet 6 inches tall. What is her BMI and daily energy needs?

a. 30, 15 kcal/kg/day

b. 21.5, 35 kcal/kg/day

c. 28, 16 kcal/kg/day

d. 18, 40 kcal/kg/day

e. 24.2, 25 kcal/kg/day

View Answer

6. A 56-year-old male undergoes a pancreaticoduodenectomy for an ampullary tumor after receiving neoadjuvant treatment. His operative course is complicated by a large volume blood loss after a portal vein injury. On POD 7 he is ready to have his diet advanced past clear liquids. Which of the following symptoms may he experience if he receives the wrong diet?

a. Vomiting

b. Bloating

c. Constipation

d. Fever

e. Hypoglycemia

View Answer

7. A 47-year-old alcoholic male presents with a 3-day history of hematemesis. He undergoes an EGD and is found to have several varices with stigmata of recent bleeding, although no active bleeding is found. He is in the intensive care unit with an NGT in place, is not receiving vasoactive medications, and is on 4-L oxygen via nasal cannula. Which of the following is a contraindication to starting enteral feeds on this patient?

a. Being in the intensive care unit

b. Alcohol withdrawal

c. Recent EGD

d. UGI bleeding

e. Lack of enteral access

View Answer

8. A 70-year-old male suffers a CVA and fails his swallow study. He also has a history of a large hiatal hernia with intermittent large volume episodes of emesis before his stroke. He is currently being fed via a small bowel feeding tube, and a surgery consult is obtained for long-term feeding access. Which is the best choice for a feeding tube in this patient who is close to discharge to a skilled rehabilitation facility?

a. Leave his small bowel feeding tube in place

b. Place a nasogastric tube

c. Gastrostomy tube

d. Jejunostomy tube

e. Give him swallowing exercises to improve his swallow mechanism

View Answer

9. A 55-year-old alcoholic male presents with a 2-day history of chest pain after forceful vomiting. He is taken to the OR for a thoracotomy after he is found to have an esophageal rupture. A jejunal feeding tube is placed and he is started on tube feeds on POD 1. You are worried about refeeding syndrome given his severe malnutrition. What electrolyte abnormality do you expect to see?

a. Hypophosphatemia

b. Hyperphosphatemia

c. Hypernatremia

d. Hyponatremia

e. Hyperkalemia

View Answer

10. A 42-year-old female is admitted to the ICU after being operated on for a perforated duodenal ulcer. Her past medical history is significant for steroid-dependent COPD. Given the degree of contamination in her abdomen the decision is made to wait at least 1 week before obtaining an UGI and starting enteral feeds. She will be started on TPN and has vascular access with a right IJ triple lumen catheter. She is currently on low-dose norepinephrine and mechanically ventilated. She weighs 50 kg and is 150 cm tall. She does not have any renal or hepatic dysfunction. What should the composition of her TPN be?

a. 35 kcal/kg/day, 0.75 g/kg/IBW of protein/day

b. 25 kcal/kg/day, 1.5 g/kg/IBW of protein/day

c. 20 kcal/kg/day, 2 g/kg/IBW of protein/day

d. 20 kcal/kg/day, 0.75 g/kg/IBW of protein/day

e. 25 kcal/kg/day, 0.5 g/kg/IBW of protein/day

View Answer

11. What is the blood glucose goal for critically ill patients on TPN?

a. <100 mg/dL

b. <150 mg/dL

c. <180 mg/dL

d. <110 mg/dL

e. There is no goal blood glucose level

View Answer

12. A 60-year-old TPN-dependent male with short gut syndrome and diarrhea presents with a nonhealing leg wound. Which trace element may he need supplementation with?

a. Manganese

b. Fluorine

c. Selenium

d. Copper

e. Zinc

View Answer

13. A 22-year-old female presents with an acute flair of ulcerative colitis. When an oral diet is started, which is the preferred diet?

a. Low-fiber diet

b. Regular diet

c. High-fat, high-protein diet

d. Tube feeding

e. Dumping syndrome diet

View Answer

14. Which of the following additives should be adjusted daily in a TPN?

a. Vitamin K

b. H_2-receptor antagonists

c. Potassium

d. Copper

e. Zinc

View Answer

15. A 50-year-old male is started on tube feeds 3 days after a bowel resection and has profuse diarrhea. A stool sample for *Clostridium difficile* is sent and is negative. What can be done to slow down the diarrhea?

a. Start antibiotics

b. Increase the concentration of the tube feeding formula

c. Add soluble fiber

d. Start an antidiarrheal agent such as loperamide

e. Start parenteral nutrition

View Answer

4

Fluid, Electrolytes, and Acid-Base Disorders

Wen Hui Tan

Stephanie L. Bonne

I. INTRODUCTION.

The surgical patient is at risk for multiple derangements of fluid balance and electrolyte composition. As a result, knowing how to manage these derangements is essential for optimal postop management.

A. Definition of Body Fluid Compartments. Water constitutes 50% to 70% of lean body weight. Total body water is divided into an intracellular fluid compartment and an extracellular fluid compartment, which consists of an intravascular compartment and an interstitial compartment, as illustrated in Figure 4-1. 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+}, and the principal intracellular anions are phosphates and negatively charged proteins.

B. Osmolality and Tonicity. *Osmolality* refers to the number of osmoles of solute particles per kilogram of water, and is comprised of both effective and ineffective components. Effective osmoles cannot freely permeate cell membranes and are therefore restricted to either the intracellular or extracellular fluid compartments. The asymmetry in effective osmoles between these compartments causes the movement of water across the cell membrane. The effective osmolality of a solution is equivalent to its tonicity, and in turn, tonicity is the parameter the body attempts to regulate.

II. PARENTERAL FLUID THERAPY

A. Principles of Fluid Management. A normal individual consumes an average of 2,000 to 2,500 mL of water daily. Daily water losses include approximately 1,000 to 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 of insensible water loss occurs daily via the skin and respiratory tract. Insensible losses increase with hypermetabolism, fever, and hyperventilation. The composition of commonly used parenteral fluid is presented in Table 4-1.

1. Maintenance. Maintenance fluids should be administered at a rate that is sufficient to maintain a urine output of 0.5 to 1 mL/kg/hour in adults. Maintenance fluid requirements can be approximated on the basis of body weight as follows: 100 mL/kg/day for the first 10 kg, 50 mL/kg/day for the second 10 kg, and 20 mL/kg/day for each

subsequent 10 kg. Maintenance fluids in general should contain Na^+ (1 to 2 mmol/kg/day) and K^+ (0.5 to 1 mmol/kg/day [e.g., D5 0.45% NaCl + 20 to 30 mmol K^+/L]).

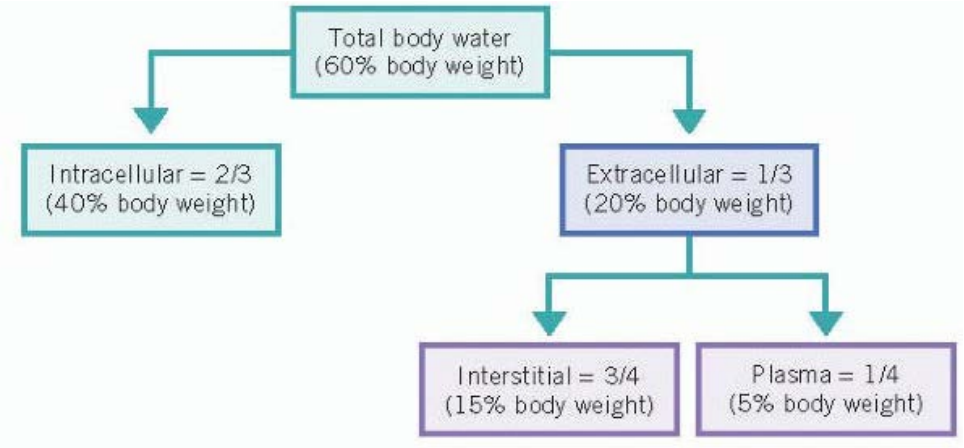


Figure 4-1 Body fluid compartments.

2. Intraoperative fluid management requires replacement of preoperative deficit as well as ongoing losses. Intraoperative losses include maintenance fluids for the length of the case, hemorrhage, and Òthird-space losses.Ó 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 depend on the size of the incision and the extent of tissue trauma. Small incisions with minor tissue trauma (e.g., inguinal hernia repair) result in third-space losses of approximately 1 to 3 mL/kg/hour. Medium-sized incisions with moderate tissue trauma (e.g., uncomplicated sigmoidectomy) result in third-space losses of approximately 3 to 7 mL/kg/hour. Larger incisions and operations with extensive tissue trauma and dissection (e.g., pancreaticoduodenectomy) can result in third-space losses of approximately 9 to 11 mL/kg/hour or greater.

3. Postoperative fluid management requires careful evaluation of the patient, and should generally be titrated to maintain an adequate urine output (0.5 to 1.0 mL/kg/hour). Sequestration of extracellular fluid into the sites of injury or operative trauma can continue for 12 or more hours after operation. GI losses that exceed 250 mL/day from nasogastric tube suction should be replaced with an equal volume of crystalloid. Mobilization of perioperative third-space fluid losses typically begins 2 to 3 days after operation. In general, gastric losses should be replaced with D5 1/2 NS with 20 mEq K and pancreatic, biliary, largest intestine losses (e.g., diarrhea) and small intestine losses should be replaced with Lactated Ringer (LR) solution.

B. Crystalloids are solutions that contain sodium as the major particle. Crystalloids are inexpensive and used for volume expansion, maintenance infusion, and correction of electrolyte disturbances.

TABLE 4-1 Composition of Common Parenteral Fluids[a]

Solution	Volume[b]	Na^+	K^+	Ca^{2+}	Mg^{2+}	Cl^-	HCO_3 (as Lactate)	Dextrose (g/L)	mOsm/L
Extracellular fluid	Ñ	142	4	5	3	103	27	Ñ	280-310
Lactated	Ñ	130	4	3	Ñ	109	28	Ñ	273

Ringer									
0.9% NaCl	Ñ	154	Ñ	Ñ	Ñ	154	Ñ	Ñ	308
0.45% NaCl	Ñ	77	Ñ	Ñ	Ñ	77	Ñ	Ñ	154
D5W	Ñ	Ñ	Ñ	Ñ	Ñ	Ñ	Ñ	50	252
D5/0.45% NaCl	Ñ	77	Ñ	Ñ	Ñ	77	Ñ	50	406
D5LR	Ñ	130	4	3	Ñ	109	28	50	525
3% NaCl	Ñ	513	Ñ	Ñ	Ñ	513	Ñ	Ñ	1,026
7.5% NaCl	Ñ	1,283	Ñ	Ñ	Ñ	1,283	Ñ	Ñ	2,567
6% hetastarch	500	154	Ñ	Ñ	Ñ	154	Ñ	Ñ	310
10% dextran-40	500	0/154[c]	Ñ	Ñ	Ñ	0/154[c]	Ñ	Ñ	300
6% dextran-70	500	0/154[c]	Ñ	Ñ	Ñ	0/154[c]	Ñ	Ñ	300
5% albumin	250, 500	130-160	<2.5	Ñ	Ñ	130-160	Ñ	Ñ	330
25% albumin	20, 50, 100	130-160	<2.5	Ñ	Ñ	130-160	Ñ	Ñ	330
Plasma protein fraction	250, 500	145				145			300

[a]Electrolyte concentrations in mmol/L.[b] Available volumes (mL) of colloid solutions.[c] Dextran solutions available in 5% dextrose (0 Na^+, 0 Cl) or 0.9% NaCl (154 mmol Na^+, 154 mmol Cl).
D5LR, 5% dextrose in lactated Ringer solution; D5/0.45% NaCl, 5% dextrose per 0.45% NaCl; D5W, 5% dextrose in water.

1. Isotonic crystalloids include LR solution and 0.9% NaCl (NS). Isotonic crystalloids distribute uniformly throughout the extracellular fluid compartment so that after 1 hour, only 25% of the total volume infused remains in the intravascular

space. LR 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, LR and NS can be used interchangeably. However, NS 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 resuscitation fluids for patients with shock or burns. These fluids were initially appealing because, relative to isotonic crystalloids, smaller quantities are required for resuscitation. Infusion of hypertonic saline solutions in patients suffering from traumatic hemorrhagic shock has been found to reduce neutrophil and endothelial cell activation, potentially inhibiting posttraumatic inflammation (*Shock.* 2012;38(4):348). However, most human clinical trials to date have not shown any statistically significant difference in survival or multiple organ dysfunction syndrome in patients receiving hypertonic saline or isotonic solutions *(Anesthesiol Clin.* 2013;31(1):17). The possible side effects of hypertonic solutions include hypernatremia, hyperosmolality, hyperchloremia, hypokalemia, and central pontine demyelination with rapid infusion and therefore they should be administered with caution until more conclusive research becomes available.

3. 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 (as in hypernatremia).

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. In fact, the SAFE (Saline versus Albumin Fluid Evaluation) study, which randomized 6,997 patients in the ICU to receive either 4% albumin or normal saline for fluid resuscitation, found no significant difference in outcomes, including mortality and organ failure, between the two groups *(N Engl J Med.* 2004;350:2247). Since colloid solutions are substantially more expensive than crystalloids, their routine use is controversial. The most recent *Surviving Sepsis* guidelines advocate the use of crystalloids as the initial fluid of choice in the resuscitation of patients with septic shock. The use of colloids is indicated when substantial amounts of crystalloids fail to sustain plasma volume (*Intensive Care Med.* 2013;39:165-228).

1. Albumin preparations ultimately distribute throughout the extracellular space, although the initial location 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 to 500 mL). Albumin 25% is indicated in the edematous patient to mobilize interstitial fluid into the intravascular space. 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 expands 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 and interference with blood cross-matching). There is no clear benefit to the use of dextrans over crystalloid solutions *(Ann Intensive Care.* 2014;4:38).

3. Hydroxyethyl starch (hetastarch) is a synthetic molecule resembling glycogen that is available as a 6% solution in 0.9% NaCl. Hetastarch, like 5% albumin, increases the intravascular volume by an amount equal to or greater than the volume infused. A recent meta-analysis, however, found that fluid resuscitation with hydroxyethyl starch is associated with an increased incidence of acute kidney injury, transfusion of packed red blood cells, need of renal replacement therapy, and 90-day mortality in patients with sepsis *(J Crit Care.* 2014:29(1):185). Increasing literature in specific patient populations also shows either no benefit or detrimental effects of hetastarch. For these reasons, use of crystalloid is preferred over hydroxyethyl starch.

III. DIAGNOSIS AND TREATMENT OF COMMON ELECTROLYTE

DISORDERS

A. Sodium

1. Physiology. The normal individual consumes 3 to 5 g of NaCl (130 to 217 mmol Na^+) daily. Sodium balance is maintained primarily by the kidneys. Normal Na^+ concentration is 135 to 145 mmol/L (310 to 333 mg/dL). Potential sources of significant Na^+ loss include sweat, urine, and gastrointestinal secretions (Table 4-2).

2. Hyponatremia

a. Clinical manifestations. Symptoms associated with hyponatremia are predominantly neurologic and result from hypo-osmolality, and include lethargy, confusion, nausea, vomiting, seizures, and coma. Chronic hyponatremia is often asymptomatic until the serum Na^+ concentration falls below 110 to 120 mEq/L (253 to 276 mg/dL). An acute drop in the serum Na^+ concentration to 120 to 130 mEq/L (276 to 299 mg/dL), conversely, may produce symptoms.

b. Causes, diagnosis, and treatment. The diagnostic approach to hyponatremia is illustrated in Figure 4-2, and a treatment algorithm is detailed in Figure 4-3. It is first necessary to measure the serum osmolality to evaluate patients with hyponatremia to determine if there is an associated tonicity imbalance.

TABLE 4-2 Composition of Gastrointestinal Secretions

Source	Volume (mL/24 hr)[a]	Na^+ (mmol/L)[b]	K^+ (mmol/L)[b]	Cl^- (mmol/L)[b]	HCO_3 (mmol/L)[b]
Salivary	1,500 (500-2,000)	10 (2-10)	26 (20-30)	10 (8-18)	30
Stomach	1,500 (100-4,000)	60 (9-116)	10 (0-32)	130 (8-154)	0
Duodenum	(100-2,000)	140	5	80	0
Ileum	3,000	140 (80-150)	5 (2-8)	104 (43-137)	30
Colon		(100-9,000)	60	30	40
Pancreas	(100-800)	140 (113-185)	5 (3-7)	75 (54-95)	115
Bile	(50-800)	145 (131-164)	5 (312)	100 (89-180)	35

[a] Average volume (range).

[b] Average concentration (range).

Reprinted with permission from Faber MD, Schmidt RJ, Bear RA, et al. Management of fluid, electrolyte, and acid-base disorders in surgical patients.
In: Narins RG, ed. *Clinical Disorders of Fluid and Electrolyte Metabolism.* New York: McGraw-Hill; 1994:1424.

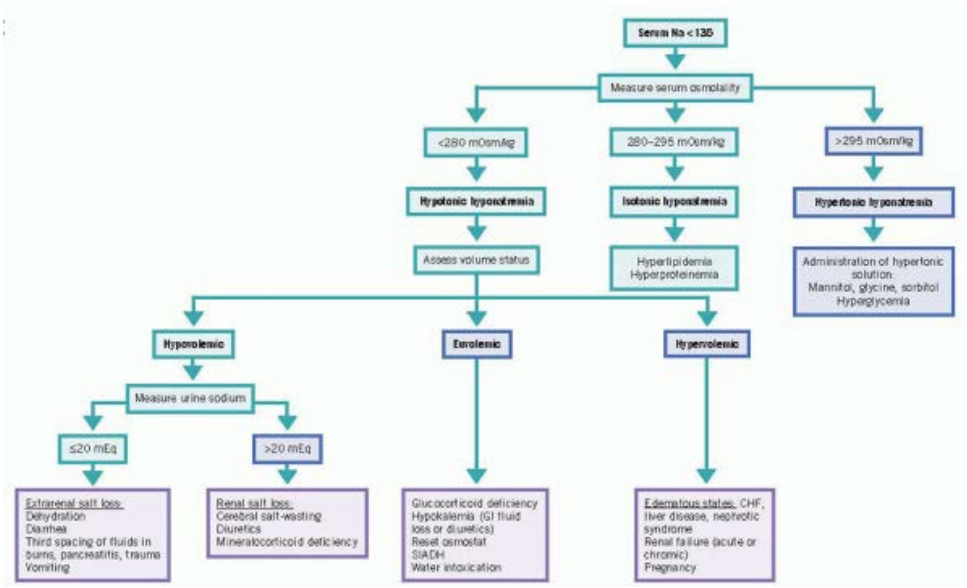


Figure 4-2 Diagnostic approach for hyponatremia. CHF, congestive heart failure; GI, gastrointestinal; SIADH, syndrome of inappropriate antidiuretic hormone secretion.

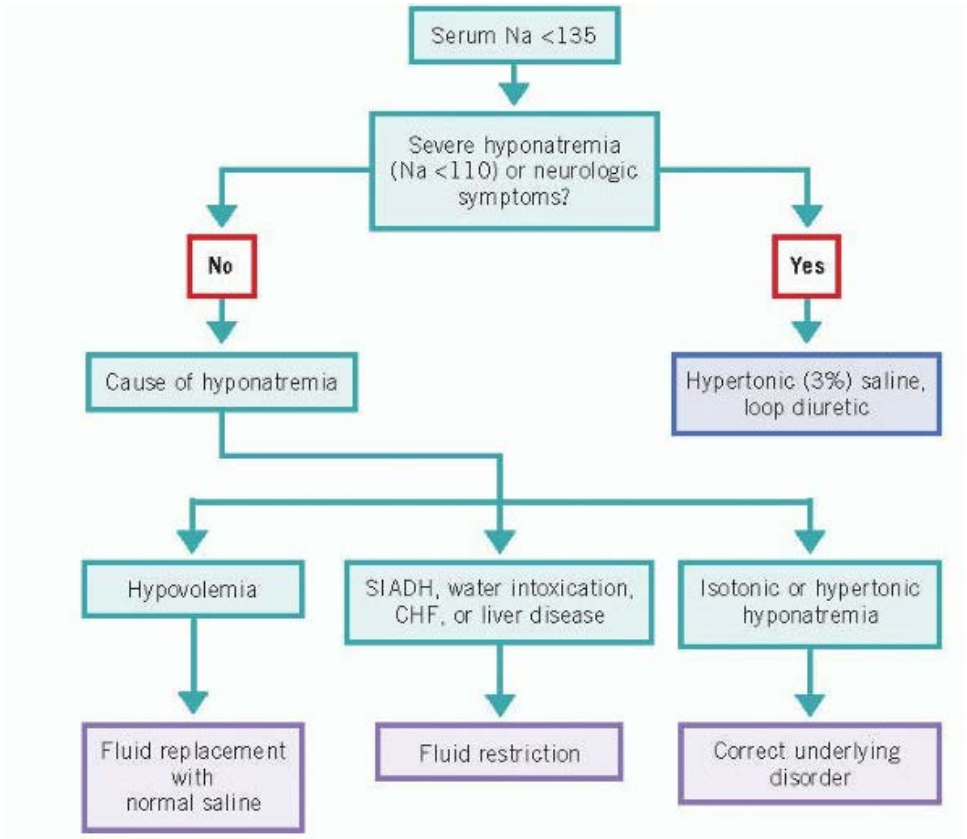


Figure 4-3 Treatment algorithm for hyponatremia. CHF, congestive heart failure; SIADH, syndrome of inappropriate antidiuretic hormone secretion.

(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, although total body Na^+ remains unchanged. Isotonic, sodium-free solutions of glucose, mannitol, and glycine are restricted initially to the extracellular fluid and may similarly result in transient hyponatremia. Isotonic hyponatremia corrects with resolution of the underlying disorder.

(2) Hypertonic hyponatremia. Hyperglycemia may result in a transient fluid shift from the intracellular to the extracellular compartment, thereby diluting serum Na^+ concentration. Rapid infusion of hypertonic solutions of glucose, mannitol, or glycine may have a similar effect on Na^+ concentration. Hypertonic hyponatremia corrects with resolution of the underlying disorder.

(3) Hypotonic hyponatremia is classified on the basis of extracellular fluid volume.

(a) Hypovolemic hypotonic hyponatremia in the surgical patient most commonly results from replacement of sodium-rich fluid losses (e.g., from the GI tract, skin, or lungs) with an insufficient volume of hypotonic fluid (e.g., D5W and 0.45% NaCl). Hypovolemic hyponatremia can be managed with administration of 0.9% NaCl to correct volume deficits and replace ongoing losses.

(b) Hypervolemic hypotonic hyponatremia. The edematous states of congestive heart failure, liver disease, and nephrosis occur in conjunction with inadequate circulating blood volume. This serves as a stimulus for the renal retention of sodium and water. Disproportionate accumulation of water results in hyponatremia. Hypervolemic hyponatremia may respond to water restriction (1,000 mL/day) to return Na^+ to greater than 130 mmol/L (299 mg/dL). In cases of severe congestive heart failure, optimizing cardiac performance 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 to 200 mg intravenously every 6 hours) while replacing urinary Na^+ losses with 3% NaCl. Hypertonic saline should not be administered to these patients without concomitant diuretic therapy. Administration of synthetic brain natriuretic peptide (BNP) is also useful therapeutically in the setting of acute heart failure because it inhibits Na^+ reabsorption at the cortical collecting duct and inhibits the action of antidiuretic hormone (ADH) on water permeability at the inner medullary collecting duct.

(c) Isovolemic hypotonic hyponatremia

(i) Water intoxication typically occurs in the patient who consumes large quantities of water and has 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 renal failure. Water intoxication responds to fluid restriction (1,000 mL/day).

(ii) Hypokalemia, either from GI fluid loss or secondary to diuretics, may result in isovolemic hyponatremia due to cellular exchange of K^+ and Na^+.

(iii) Reset osmostat. Normally, the serum ÒosmostatÓ is set at 285 mOsm/L. In some individuals with chronic disease (e.g., cirrhosis), the osmostat is ÒresetÓ downward, thus maintaining a lower serum osmolality. These patients respond normally to water loads with suppression of ADH secretion and excretion of free water.

(iv) Syndrome of inappropriate ADH (SIADH) is characterized by low plasma osmolality (<280 mOsm/L), hyponatremia (<135 mmol/L), low urine output with

concentrated urine (>100 mOsm/kg), elevated urine sodium (>20 mEq/L), and clinical euvolemia. The major causes of SIADH include pulmonary disorders (e.g., atelectasis and respiratory failure), central nervous system disorders (e.g., trauma and meningitis), drugs (e.g., cyclophosphamide and cisplatin), and ectopic ADH production (e.g., small-cell lung carcinoma). For SIADH, water restriction (1,000 mL/day) should be attempted initially. The addition of a loop diuretic (furosemide) or an osmotic diuretic (mannitol) may be necessary.

(4) Transurethral resection syndrome refers to hyponatremia 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.

(5) In the presence of symptoms or **extreme hyponatremia** (Na^+ <110 mmol/L [253 mg/dL]), hypertonic saline (3% NaCl) is indicated. Serum Na^+ should be corrected to approximately 120 mmol/L (276 mg/dL). The quantity of 3% NaCl that is required to increase serum Na^+ to 120 mmol/L (276 mg/dL) can be estimated by calculating the Na^+ deficit:

$$\text{Na deficit} = 0.60 \times \text{lean body weight (kg)} \times \left[120 - \text{measured Na}\left(\frac{\text{mmol}}{\text{L}}\right)\right]$$

(Each liter of 3% NaCl provides 513 mmol Na^+.) The use of a loop diuretic (furosemide, 20 to 200 mg intravenously every 6 hours) may increase the effectiveness of 3% NaCl administration. Central pontine demyelination can occur in the setting of correction of hyponatremia. The risk factors for demyelination are controversial but appear to be related to the chronicity of hyponatremia (>48 hours) and the rate of correction. In patients with severe acute hyponatremia, the initial correction rate should not exceed 1 to 2 mmol/L/hour. In the first 48 hours, normo- or hypernatremia should be avoided. The patient's volume status should be carefully monitored over this time, and the serum Na^+ should be measured frequently (every 1 to 2 hours). Once the serum Na^+ concentration reaches 120 mmol/L (276 mg/dL) and symptoms have resolved, administration of hypertonic saline can be discontinued.

3. Hypernatremia

a. Clinical manifestations. Symptoms of hypernatremia include lethargy, weakness, and irritability and may progress to fasciculations, seizures, coma, and irreversible neurologic damage.

b. Diagnosis and treatment. Patients are categorized on the basis of their extracellular fluid volume status. The diagnostic and treatment approaches to hypernatremia are illustrated in Figure 4-4.

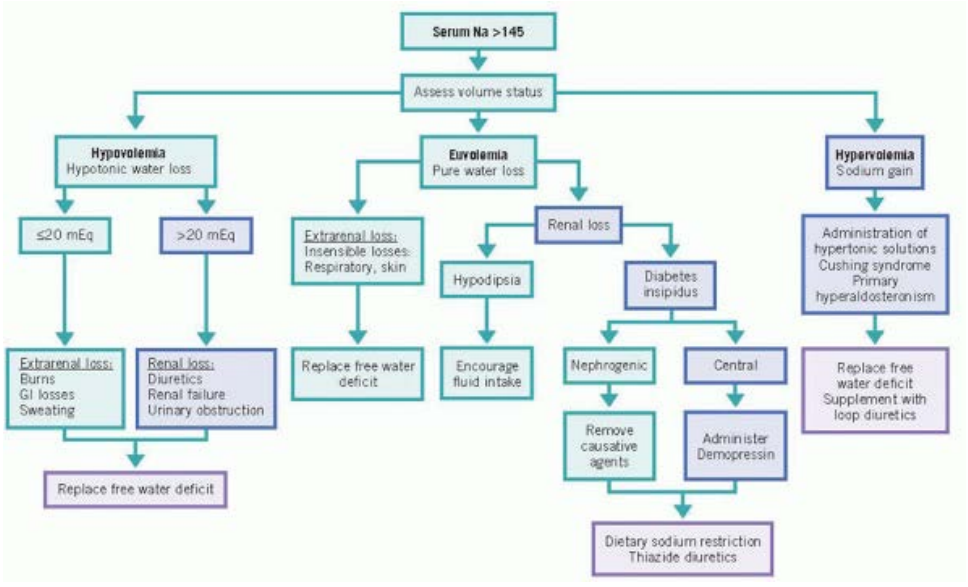


Figure 4-4 Diagnostic and treatment algorithm for hypernatremia. GI, gastrointestinal.

(1) Hypovolemic hypernatremia. Common causes in the surgical patient include diuresis, as well as GI, respiratory, and cutaneous (e.g., burns) fluid losses. Chronic renal failure and partial urinary tract obstruction also may cause hypovolemic hypernatremia.

(a) Replacement of free water is the main goal of treatment. The **water deficit** associated with hypernatremia can be estimated using the following equation where TBW = Total body weight:

$$\text{Water deficit (L)} = 0.60 \times \text{TBW (kg)} \times \left[\frac{\text{serum } Na^+ \left(\frac{\text{mmol}}{\text{L}}\right)}{140} - 1\right]$$

Rapid correction of hypernatremia can result in cerebral edema and permanent neurologic damage. Consequently, only one-half of the water deficit should be corrected over the first 24 hours, with the remainder being corrected over the following 2 to 3 days. Oral fluid intake is acceptable for replacing water deficits. If oral intake is not possible, D5W or D5 0.45% NaCl can be substituted.

(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, and nutrition). It can also be the result of aldosteronism, Cushing disease (secondary hypercortisolism), or mineralocorticoid excess. In cases of hypervolemic hypernatremia, free water replacement can be supplemented with a loop diuretic.

(3) Isovolemic hypernatremia

(a) Hypotonic losses. Evaporative losses from the skin and respiratory tract, in addition to ongoing urinary free water losses, require the administration of approximately 750 mL of 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 polyuria and polydipsia in association with hypotonic urine (urine osmolality <200 mOsm/kg or a specific gravity of <1.005) and a high plasma osmolality (>287 mOsm/kg). *Central diabetes insipidus* (CDI) describes a defect in the hypothalamic secretion of ADH while *nephrogenic diabetes insipidus* (NDI) describes renal insensitivity to normally secreted ADH. CDI can be treated with desmopressin acetate-synthetic ADH. 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 to 100 mg/day orally).

(c) Therapeutic. Hypertonic saline may be administered for deliberate hypernatremia to control elevated intracranial pressure (ICP) and cerebral edema after head injury.

B. Potassium

1. Physiology. K^+ is the major intracellular cation, with only 2% of total body K^+ located in the extracellular space. The normal serum concentration is 3.3 to 4.9 mmol/L (12.9 to 19.1 mg/dL). Approximately 50 to 100 mmol (195 to 390 mg/dL) K^+ is ingested and absorbed daily. Ninety percent of K^+ is renally excreted, with the remainder eliminated in stools.

2. Hypokalemia

a. Clinical manifestations. Mild hypokalemia (K^+ >3 mmol/L [11.7 mg/dL]) is generally asymptomatic. Symptoms occur with severe K^+ deficiency (K^+ <3 mmol/L [11.7 mg/dL]) and are primarily cardiovascular. Early electrocardiogram (ECG) manifestations include ectopy, T-wave depression, and prominent U waves. Severe depletion increases susceptibility to re-entry arrhythmias.

b. Causes. K^+ depletion from inadequate intake alone is rare. Common causes of K^+ depletion in the surgical patient include GI losses, renal losses, and cutaneous losses (e.g., burns). Other causes of hypokalemia include conditions

associated with acute intracellular K^+ uptake, such as insulin excess, metabolic alkalosis, myocardial infarction, delirium tremens, hypothermia, and theophylline toxicity. Hypokalemia may also occur in refeeding syndrome.

c. Treatment. 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 to 100 mmol (156 to 390 mg) potassium chloride 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 (156 mg/dL), and the rate of administration should not exceed 20 mmol (78 mg)/hour. However, higher K^+ concentrations (60 to 80 mmol/L [234 to 312 mg/dL]) administered more rapidly (with cardiac monitoring) are indicated in cases of severe hypokalemia, for cardiac arrhythmias, and in the management of diabetic ketoacidosis.

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. Abnormal redistribution of K^+ from the intracellular to the extracellular compartment may occur as a result of insulin deficiency, β-adrenergic receptor blockade, acute acidemia, rhabdomyolysis,

cell lysis (after chemotherapy), digitalis intoxication, reperfusion of ischemic limbs, and succinylcholine administration.

b. Clinical manifestations. Mild hyperkalemia is generally asymptomatic. Signs of significant hyperkalemia 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.

c. Treatment

(1) Mild hyperkalemia (K^+= 5 to 6 mmol/L [19.5 to 23.4 mg/dL]) can be treated conservatively by the reduction in daily K^+ intake and, if needed, the addition of a loop diuretic (e.g., furosemide) to promote renal elimination. Any medication that is capable of impairing K^+ homeostasis (e.g., nonselective β-adrenergic antagonists, angiotensin-converting enzyme inhibitors, K^+-sparing diuretics, and nonsteroidal anti-inflammatory drugs) should be discontinued, if possible.

(2) Severe hyperkalemia (K^+ >6.5 mmol/L [25.4 mg/dL])

(a) Temporizing measures produce shifts of potassium from the extracellular to the intracellular space.

(i) Calcium gluconate 10% (5 to 10 mL intravenously over 2 minutes) should be administered to patients with profound ECG changes who are not receiving digitalis preparations. Calcium functions to stabilize the myocardium.

(ii) $NaHCO_3$ (1 mmol/kg or 1 to 2 ampules [50 mL each] of 8.4% $NaHCO_3$) can be infused intravenously over a 3- to 5-minute period. This dose can be repeated after 10 to 15 minutes if ECG abnormalities persist.

(iii) Dextrose (0.5 g/kg body weight) infused with insulin (0.3 unit of regular insulin/g of dextrose) transiently lowers serum K^+ (the usual dose is 25 g dextrose, with 6 to 10 units of regular insulin given simultaneously as an intravenous bolus).

(iv) Inhaled β-agonists (e.g., albuterol sulfate, 2 to 4 mL of 0.5% solution [10 to 20 mg] delivered via nebulizer) have been shown to lower plasma K^+, with a duration of action of up to 2 hours.

(b) Therapeutic measures to definitively decrease total body potassium by increasing potassium excretion:

(i) Sodium polystyrene sulfonate (Kayexalate) is a Na^+Ñ K^+ exchange resin. A decrease in serum K^+ level typically occurs 2 to 4 hours after administration; however, we caution its use in surgical patients, as it has been associated with bowel necrosis.

(ii) Hydration with 0.9% NaCl in combination with a loop diuretic (e.g., furosemide, 20 to 100 mg intravenously) should

be administered to patients with adequate renal function to promote renal K excretion.

(iii) Dialysis is definitive therapy in severe, refractory, or lifethreatening hyperkalemia.

C. Magnesium

1. Physiology. Mg^{2+} (normal serum concentration: 1.3 to 2.2 mEq/L or 0.65 to 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. 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. We recommend maintaining a patient's magnesium at the upper limit of normal (2 to 2.5 mEq/L) to prevent QT prolongation and arrhythmias.

b. Causes. Hypomagnesemia on the basis of dietary insufficiency is rare. Common etiologies include excessive GI or renal Mg^{2+} loss. Urinary loss occurs with marked diuresis, primary hyperaldosteronism, renal tubular dysfunction (e.g., renal tubular acidosis), chronic alcoholism, or as a drug side effect (e.g., loop diuretics, cyclosporine, amphotericin B, aminoglycosides, and cisplatin). Hypomagnesemia may also result from shifts of Mg^{2+} from the extracellular to the intracellular space, particularly in conjunction with acute myocardial infarction, 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 refeeding syndrome and in the trauma patient.

c. Treatment

(1) Parenteral therapy is preferred for the treatment of severe hypomagnesemia (Mg^{2+} <1 mEq/L or 0.5 mmol/L) or in symptomatic patients. In cases of life-threatening arrhythmias, 1 to 2 g (8 to 16 mEq) of $MgSO_4$ can be administered over 5 minutes, followed by a continuous infusion of 1 to 2 g/hour for the next several hours. The infusion subsequently can be reduced to 0.5 to 1 g/hour for maintenance. In less urgent situations, $MgSO_4$ infusion may begin at 1 to 2 g/hour for 3 to 6 hours, with the rate subsequently adjusted to 0.5 to 1 g/hour for maintenance. Mild hypomagnesemia (1.1 to 1.4 mEq/L or 0.5 to 0.7 mmol/L) in an asymptomatic patient can be treated initially with the parenteral administration of 50 to 100 mEq (6 to 12 g) of $MgSO_4$ daily until body stores are replenished. Treatment should be continued for 3 to 5 days, at which time the patient can be switched to an oral maintenance dose.

(2) Oral therapy. Magnesium oxide is the preferred oral agent. Each 400 mg tablet provides 241 mg (20 mEq) of Mg^{2+}. Other formulations include magnesium gluconate (each 500-mg tablet provides 27 mg [2.3 mEq] of Mg^{2+}) and magnesium chloride (each 535-mg tablet provides 64 mg [5.5 mEq] of Mg^{2+}). Depending on the level of depletion, oral therapy should provide 20 to 80 mEq of Mg^{2+}/day 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 to 0.45 mEq/kg of Mg^{2+}/day (i.e., by adding 8 to 16 mEq [1 to 2 g] of $MgSO_4$ to each liter of intravenous fluids).

3. Hypermagnesemia

a. Clinical manifestations. Mild hypermagnesemia (Mg^{2+} 5 to 6 mEq/L or 2.5 to 3 mmol/L) is generally asymptomatic.

Severe hypermagnesemia (Mg >8 mEq/L or 4 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.

b. Causes. Hypermagnesemia occurs infrequently, is usually iatrogenic, and is seen most commonly in the setting of renal failure.

c. Treatment. Cessation of exogenous Mg^{2+} is necessary. Calcium gluconate 10% (10 to 20 mL over 5 to 10 minutes intravenously) is indicated in the presence of life-threatening symptoms to antagonize the effects of Mg^{2+}. A 0.9% NaCl (250 to 500 mL/hour) infusion with loop diuretic (furosemide, 20 mg intravenously every 4 to 6 hours) in the patient with intact renal function promotes renal elimination. Dialysis is the definitive therapy in the presence of intractable symptomatic hypermagnesemia.

D. Phosphorus

1. Physiology. Extracellular fluid contains less than 1% of total body stores of phosphorus at a concentration of 2.5 to 4.5 mg/dL (0.81 to 1.45 mmol/L). Phosphorus balance is regulated by a number of hormones that also control calcium metabolism. As a consequence, derangements in concentrations of phosphorus and calcium frequently coexist. Phosphate is necessary to produce ATP, which cells use for energy, and is thus critical to many metabolic processes. The average adult consumes 800 to 1,000 mg of phosphorus daily, which is predominantly excreted through the kidneys.

2. Hypophosphatemia

a. Clinical manifestations. Moderate hypophosphatemia (phosphorus 1 to 2.5 mg/dL or 0.32 to 0.81 mmol/L) is usually asymptomatic. Severe hypophosphatemia (phosphorus <1 mg/dL or 0.32 mmol/L) may result in respiratory muscle dysfunction, diffuse weakness, and flaccid paralysis.

b. 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, during hyperglycemia as a result of osmotic diuresis, and after large liver resections *(Ann Surg.* 2009;249(5):824-827).

(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.

c. Treatment. Adequate repletion of phosphorus is especially important in critically ill patients, who are more likely to experience adverse physiologic consequences from hypophosphatemia, including the inability to be weaned from the ventilator, organ dysfunction, and death. Phosphorus replacement should begin with intravenous therapy, especially for moderate (1 to 1.7 mg/dL) or severe (<1 mg/dL) hypophosphatemia *(J Am Coll Surg.* 2004;198:198) (Table 4-3). Risks of intravenous therapy include hyperphosphatemia, hypocalcemia, hypotension, hyperkalemia (with potassium phosphate), hypomagnesemia, hyperosmolality, metastatic calcification, and renal failure.

Five to seven days of intravenous repletion may be required before intracellular stores are replenished. Once the serum phosphorus level exceeds 2 mg/dL (0.65 mmol/L), oral therapy can be initiated with a sodiumÑpotassium phosphate salt (e.g., Neutra-Phos, 250 to 500 mg [8 to 16 mmol phosphorus] orally four times a day; each 250-mg tablet of Neutra-Phos

contains 7 mmol each of K^+ and Na^+).

TABLE 4-3 Phosphorus Repletion Protocol

Phosphorus Level	Weight 40-60 kg	Weight 61-80 kg	Weight 81-120 kg
1 mg/dL	30 mmol Phos IV	40 mmol Phos IV	50 mmol Phos IV
1-1.7 mg/dL	20 mmol Phos IV	30 mmol Phos IV	40 mmol Phos IV
1.8-2.2 mg/dL	10 mmol Phos IV	15 mmol Phos IV	20 mmol Phos IV

If the patient's potassium is <4, use potassium phosphorus.
If the patient's potassium is >4, use sodium phosphorus.
IV, intravenous; Phos, phosphorus.
Adapted with permission from Taylor BE, Huey WY, Buchman TG, et al. Effectiveness of a protocol based on patient weight and serum phosphorus levels in repleting hypophosphatemia in a surgical ICU. *J Am Coll Surg.* 2004;198:198-204.

3. Hyperphosphatemia

a. Clinical manifestations, in the short term, include hypocalcemia and tetany. In contrast, soft tissue calcification and secondary hyperparathyroidism occur with chronicity.

b. 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.

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 and diuresis (acetazolamide, 500 mg every 6 hours orally or intravenously). Phosphate binders (aluminum hydroxide, 30 to 120 mL orally every 6 hours) minimize intestinal phosphate absorption and can induce a negative balance of greater than 250 mg of phosphorus daily, even in the absence of dietary phosphorus. Hyperphosphatemia secondary to conditions that cause phosphorus redistribution (e.g., diabetic ketoacidosis) resolves with treatment of the underlying condition and requires no specific therapy. Dialysis can be used to correct hyperphosphatemia in extreme conditions.

E. Calcium

1. Physiology. Serum calcium (8.9 to 10.3 mg/dL or 2.23 to 2.57 mmol/L) exists in three forms: Ionized (45%), protein bound (40%), and in a complex with freely diffusible compounds (15%). Only free ionized Ca^{2+} (4.6 to 5.1 mg/dL or 1.15 to 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.

2. Hypocalcemia

a. Clinical manifestations. Tetany is the major clinical finding and may be demonstrated by Chvostek sign (facial muscle

spasm elicited by tapping over the branches of the facial nerve). The patient may also complain of perioral numbness and tingling. In addition, hypocalcemia can be associated with QT-interval prolongation and ventricular arrhythmias.

b. 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. Hypocalcemia may occur in conjunction with 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. As 40% of serum calcium is bound to albumin, hypoalbuminemia may decrease total serum calcium significantlyÑa 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.

c. Treatment

(1) Parenteral therapy. Asymptomatic patients do not require parenteral therapy. Symptoms such as overt tetany, laryngeal spasm, or seizures are indications for parenteral calcium. Approximately 200 mg of elemental calcium is needed to abort an attack of tetany. Initial therapy consists in the administration of a calcium bolus (10 to 20 mL of 10% calcium gluconate over 10 minutes) followed by a maintenance infusion of 1 to 2 mg/kg elemental calcium/hour. Calcium chloride contains three times more elemental calcium than calcium gluconate; one 10-mL ampule of 10% calcium chloride contains 272 mg (13.6 mEq) elemental calcium, whereas one 10-mL ampule of 10% calcium gluconate contains only 90 mg (4.6 mEq) elemental calcium. The serum calcium level typically normalizes in 6 to 12 hours with this regimen, at which time the maintenance rate can be decreased to 0.3 to 0.5 mg/kg/hour. In addition to monitoring calcium levels frequently during therapy, one should check Mg^{2+}, phosphorus, and K^{+} levels and replete as necessary.

(2) Oral therapy. Calcium salts are available for oral administration (calcium carbonate, calcium gluconate). Each 1,250-mg tablet of calcium carbonate provides 500 mg of elemental calcium (25.4 mEq), and a 1,000-mg tablet of calcium gluconate has 90 mg (4.6 mEq) of elemental calcium. In chronic hypocalcemia, with serum calcium levels of 7.6 mg/dL (1.9 mmol/L) or higher, the daily administration of 1,000 to 2,000 mg of 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 of calciferol, 0.4 mg of dihydrotachysterol, or 0.25 to 0.50 µg of 1,25-dihydroxyvitamin D_3 orally. Subsequent therapy should be adjusted as necessary.

3. Hypercalcemia

a. Clinical manifestations. Mild hypercalcemia (calcium <12 mg/dL or <3 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.

b. 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.

c. Treatment of hypercalcemia depends on the severity of the symptoms (Fig. 4-5). Mild hypercalcemia (calcium <12 mg/dL or <3 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 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 to 500 mL/hour) with furosemide (20 mg intravenously every 4 to 6 hours) can be administered initially. The rate of 0.9% NaCl infusion and the dose of furosemide should subsequently be adjusted to maintain a urine output of 100 to 150 mL/hour. Serum Mg^{2+}, phosphorus, and K^{+} levels should be monitored and repleted as necessary. The inclusion of KCl (20 mmol) and $MgSO_4$ (8 to 16 mEq or 1 to 2 g) in each liter of fluid may prevent hypokalemia and hypomagnesemia. This treatment may promote the loss of as much as 2 g of calcium over 24 hours.

(2) Salmon calcitonin, in conjunction with adequate hydration, is useful for the treatment of hypercalcemia associated with either malignancy or primary hyperparathyroidism. Salmon calcitonin can be administered either subcutaneously or intramuscularly. Skin testing by subcutaneous injection of 1 IU is recommended before progressing to the initial dose of 4 IU/kg intravenously or subcutaneously every 12 hours. A hypocalcemic effect may be seen as early as 6 to 10 hours after administration. The dose may be doubled if unsuccessful after 48 hours of treatment. 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 associated with malignancy. For moderate hypercalcemia (calcium 12

to 13.5 mg/dL or 3 to 3.38 mmol/L), 60 mg of 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.

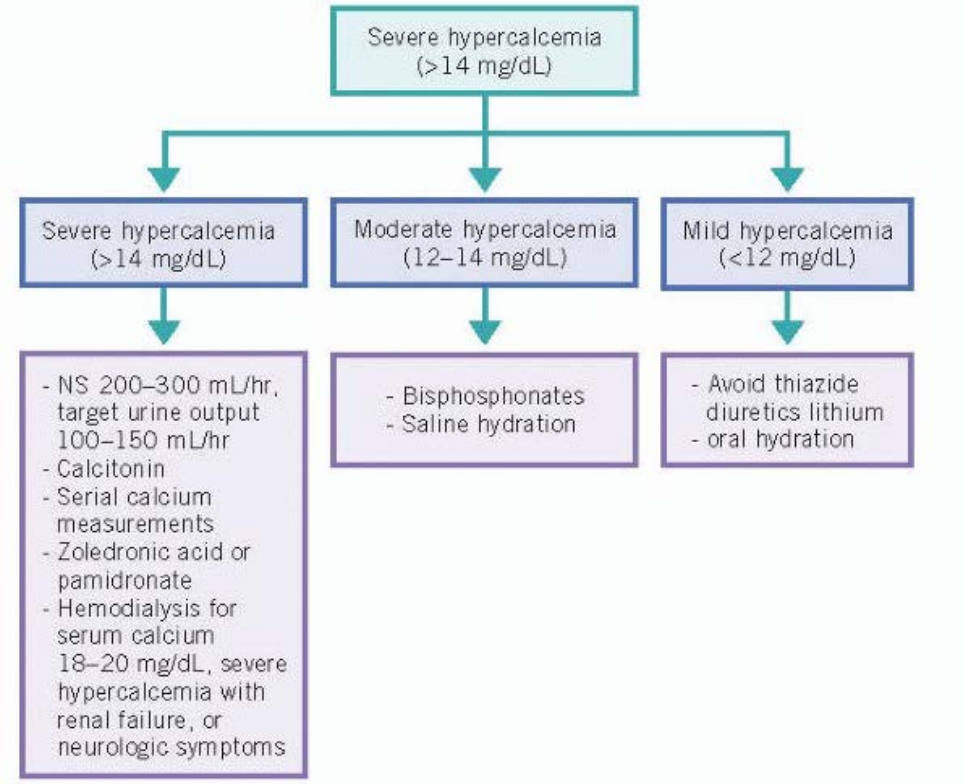


Figure 4-5 Basic treatment algorithm for hypercalcemia. Treatment specifics for unique causes of hypercalcemia is beyond the scope of this chapter.

(4) Plicamycin (25 µg/kg, diluted in 1 L of 0.9% NaCl or D5W, infused over 4 to 6 hours each day for 3 to 4 days) is useful for treatment of hypercalcemia associated with malignancy. The onset of action is between 1 and 2 days, with a

duration of action of up to 1 week.

IV. ACID-BASE DISORDERS

A. Diagnostic Approach

1. General concepts

a. AcidÑbase homeostasis represents equilibrium among the concentration of H^+, partial pressure of CO_2 (Pco_2), and HCO_3^-. Clinically, H^+ concentration is expressed as pH.

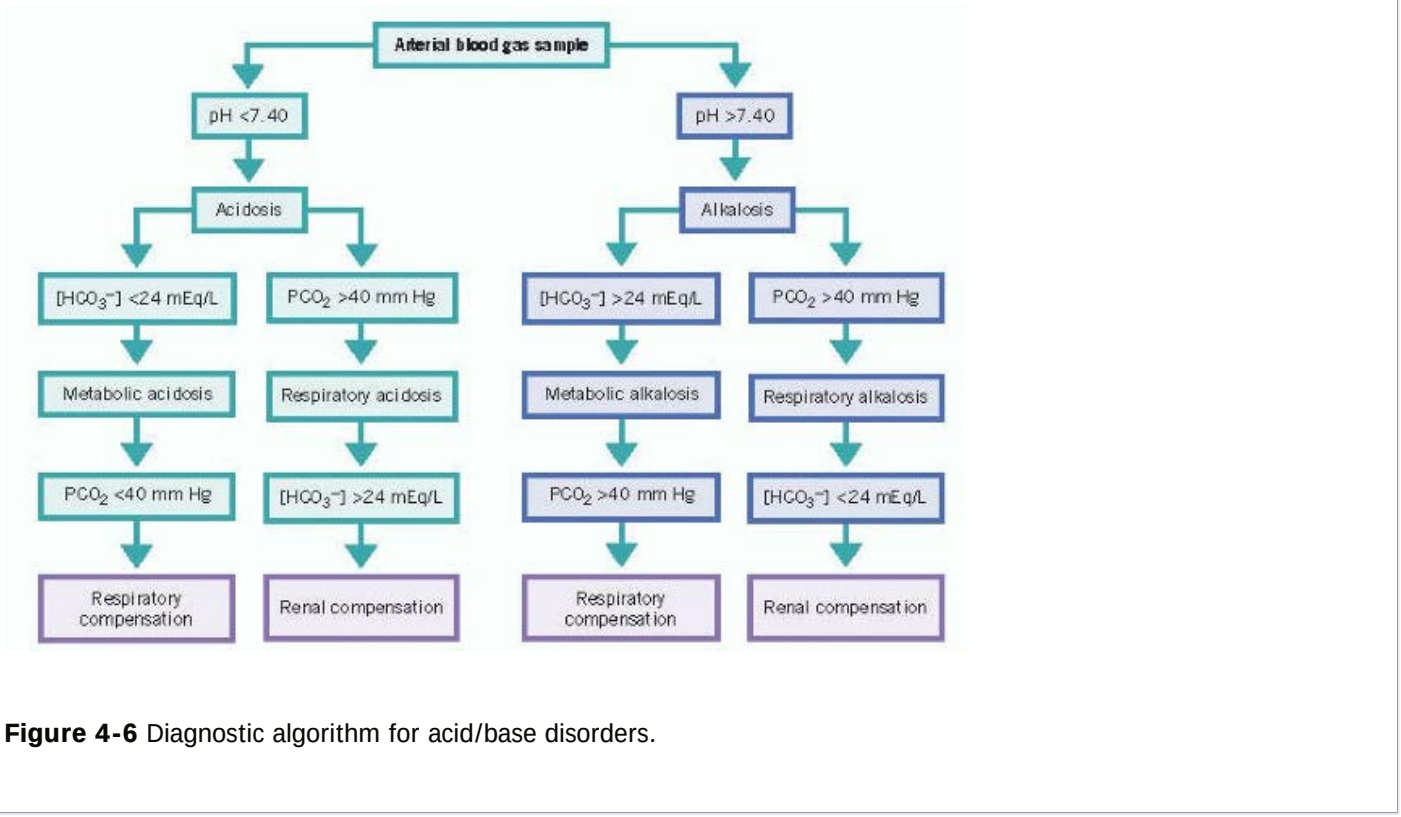


Figure 4-6 Diagnostic algorithm for acid/base disorders.

b. Initial evaluation of acidÑbase disorders should include an arterial blood gas and serum electrolytes (Fig. 4-6). Normal blood pH is 7.35 to 7.45. **Acidemia** refers to pH of less than 7.35, and **alkalemia** refers to pH of greater than 7.45.

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, and vice versa. By convention, these compensating changes are termed *secondary, 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 acidÑbase disturbance.

B. Primary Metabolic Disorders

1. Metabolic acidosis results from the accumulation of nonvolatile acids, reduction in renal acid excretion, or loss of alkali. The most common causes of metabolic acidosis are listed in Table 4-5. The appropriate diagnosis depends on the clinical setting and laboratory tests. It is useful diagnostically to classify metabolic acidosis into increased or normal AG metabolic acidosis. **The anion gap** (AG; normal =12 ± 2 mmol/L) represents the anions, other than Cl^- and HCO_3^-, which are

necessary to counterbalance Na^+ electrically (all values are in mmol/L): AG = Na^+ Ð [Cl^- + HCO_3^-]. For

causes of increased and normal (hyperchloremic) AG metabolic acidosis, see Table 4-5.

TABLE 4-4 Expected Compensation for Simple Acid-Base Disorders

Primary Disorder	Initial Change	Compensatory Response	Expected Compensation
Metabolic acidosis	HCO_3^- decrease	Pco_2 decrease	Pco_2 decrease = 1.2 × ΔHCO_3^-
Metabolic alkalosis	HCO_3^- increase	Pco_2 increase	Pco_2 increase = 0.7 × ΔHCO_3^-
Respiratory acidosis	Pco_2increase	HCO_3^- increase	Acute: HCO_3^- increase = 0.1 × ΔPco_2 Chronic: HCO_3^- increase = 0.35 × ΔPco_2
Respiratory alkalosis	Pco_2decrease	HCO_3^- decrease	Acute: HCO_3^- decrease = 0.2 × ΔPco_2 Chronic: HCO_3^- decrease = 0.5 × ΔPco_2

a. Treatment of metabolic acidosis must be directed primarily at the underlying cause of the acidÑbase 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 using the following equation:

$$\text{HCO}_3^- \text{ deficit}\left(\frac{\text{mmol}}{\text{L}}\right) = \text{body weight (kg)} \times 0.4 \times \left[\text{desired HCO}_3^-\left(\frac{\text{mmol}}{\text{L}}\right) - \text{measured HCO}_3^-\left(\frac{\text{mmol}}{\text{L}}\right)\right]$$

In nonurgent situations, the estimated HCO_3^- deficit can be repaired by administering a continuous intravenous infusion over 4 to 8 hours. A 50-mL ampule of 8.4% $NaHCO_3$ solution, which provides 50 mmol HCO_3^-, can be added to 1 L of D5W or 0.45% of NaCl. In urgent situations, the entire deficit can be repaired by administering a bolus over several minutes. The goal of HCO_3^- therapy should be to raise the arterial blood pH to 7.20.

2. Metabolic alkalosis (Table 4-6)

a. Causes

(1) 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 and 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. Since replenishment of Cl^- corrects the metabolic alkalosis in these conditions, each is classified as Cl^--responsive metabolic alkalosis.

TABLE 4-5 Causes of Metabolic Acidosis

Increased anion gap
Increased acid production
Ketoacidosis
Diabetic
Alcoholic
Starvation
Lactic acidosis
Toxic ingestion (salicylates, ethylene glycol, methanol)
Renal failure
Normal anion gap (hyperchloremic)
Renal tubular dysfunction
Renal tubular acidosis
Hypoaldosteronism
Potassium-sparing diuretics
Loss of alkali
Diarrhea
Ureterosigmoidostomy

Carbonic anhydrase inhibitors

Administration of HCl (ammonium chloride, cationic amino acids)

TABLE 4-6 Causes of Metabolic Alkalosis

Associated with extracellular fluid volume (chloride) depletion

Vomiting or gastric drainage

Diuretic therapy

Posthypercapnic alkalosis

Associated with mineralocorticoid excess

Cushing syndrome

Primary aldosteronism

Bartter syndrome

Severe K^+ depletion

Excessive alkali intake

(2) Chloride-unresponsive metabolic alkalosis is encountered less frequently in surgical patients and usually results from mineralocorticoid excess.

b. Diagnosis. Although the cause of metabolic alkalosis is usually apparent in the surgical patient, measurement of the urinary chloride concentration may be useful for differentiating these disorders. A 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. A urine Cl^- concentration of 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 replacing Cl^-, K^+, and volume deficits. Rapid correction of this disorder usually is not necessary because metabolic alkalosis generally is well tolerated.

(1) Initial therapy should include the correction of volume deficits (with 0.9% NaCl) and hypokalemia.

(2) Edematous patients. Chloride administration does not enhance HCO_3 excretion because it does not correct the reduced effective arterial blood volume. Acetazolamide (5 mg/kg/day intravenously or orally) facilitates fluid mobilization while decreasing renal HCO_3^- reabsorption.

(3) 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 and 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 cases of acute respiratory acidosis, 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, and bronchospasm), chronic hypoxia (e.g., cyanotic heart disease and anemia), and respiratory center stimulation (e.g., anxiety, fever, Gram-negative sepsis, salicylate intoxication, central nervous system disease, cirrhosis, and pregnancy). Excessive ventilation may also cause respiratory alkalosis in the mechanically ventilated patient.

D. Mixed AcidÑBase Disorders. When two or three primary acidÑbase 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 acidÑbase disturbances. The diagnosis of mixed acidÑbase disorders depends principally on evaluation of the clinical setting and on interpretation of acidÑbase patterns. However, even normal acidÑbase patterns may conceal mixed disorders.

TABLE 4-7 Common Causes of Mixed Acid-Base Disorders

Metabolic acidosis and respiratory acidosis
Cardiopulmonary arrest
Severe pulmonary edema
Salicylate and sedative overdose
Pulmonary disease with superimposed renal failure or sepsis
Metabolic acidosis and respiratory alkalosis
Salicylate overdose

Sepsis

Combined hepatic and renal insufficiency

Chronic pulmonary disease, with superimposed:

- Diuretic therapy
- Steroid therapy

Vomiting

Reduction in hypercapnia by mechanical ventilation

Metabolic alkalosis and respiratory alkalosis

Pregnancy with vomiting

Chronic liver disease treated with diuretic therapy

Cardiopulmonary arrest treated with bicarbonate therapy and mechanical ventilation

Metabolic acidosis and alkalosis

Vomiting superimposed on

- Renal failure
- Diabetic ketoacidosis
- Alcoholic ketoacidosis

CHAPTER 4: FLUID, ELECTROLYTES, AND ACID-BASE DISORDERS

Multiple Choice Questions

1. In which group of patients is there a clinically proven reduction in mortality following the administration of hypertonic saline?

a. A 65-year-old female with mild to moderate dehydration

b. A 35-year-old male with moderate hyponatremia from psychogenic polydipsia

c. A 40-year-old female with traumatic hemorrhagic shock following a motor vehicle accident and splenic laceration

d. A 5-year-old child with renal insufficiency

e. Hypertonic saline has not been shown to decrease mortality in any patient population

View Answer

2. A patient with severe sepsis secondary to cholangitis has received 4 L of crystalloid resuscitation over the last 6 hours. His MAP remains below 65, but he is fluid responsive. Which of the following fluids should be administered?

a. 0.9% NS, 1 L over 1 hour

b. 0.45% NS, 2 L over 1 hour

c. 5% albumin, 500 cc over 1 hour

d. Dextran 40, 500 cc over 2 hours

e. Hetastarch, 6% solution, 1 L over 1 hour

View Answer

3. A patient with a known history of coronary artery disease presents to the emergency room with shortness of breath and extensive lower extremity edema. Initial laboratory studies reveal a sodium of 124 mmol/L. What is the initial therapy?

a. Administration of 1 L of 0.9 NS

b. Fluid restriction to 1 L of free water per day

c. Administration of 500 cc 3% NaCl

d. Fluid restriction to 2 L of free water per day

e. Administration of 500 cc lactated Ringer solution

View Answer

4. You are caring for a head injured patient in the intensive care unit who has a large volume urine output and who, you suspect, may have central diabetes insipidus. What confirmatory test can you order in order to support your diagnosis?

a. Urine specific gravity

b. Serum sodium and urine sodium

c. 24-hour urine for electrolytes

d. Serum potassium and urine sodium

e. Serum glucose level

View Answer

5. You are informed by the laboratory that a patient you are caring for has a potassium of 6.0 on routine laboratory tests. What is the correct order of steps to manage this issue?

a. Order a confirmatory whole blood K level, order an EKG, administer insulin and glucose

b. Order an EKG, order a confirmatory whole blood K level, administer insulin and glucose

c. Order an EKG, administer insulin and glucose, place a dialysis catheter, order a confirmatory whole blood K

d. Order an EKG, place a dialysis catheter, administer albuterol, administer insulin and glucose, order a confirmatory whole blood K

e. Consult renal service for dialysis management, order an EKG, administer insulin and glucose

View Answer

6. Which of the following is a manifestation of hypomagnesemia?

a. Flaccid paralysis

b. Renal insufficiency

c. Insomnia

d. Ventricular arrhythmias

e. Vertigo

View Answer

7. What is the most common cause of metabolic alkalosis in the postoperative patient?

a. General anesthetic reaction

b. Urinary losses

c. Associated hypomagnesemia

d. Acute blood loss

e. Inadequate fluid resuscitation

View Answer

8. Bicarbonate therapy for metabolic acidosis is appropriate for which of the following patients?

a. A 36-year-old hemodynamically stable patient with salicylate poisoning

b. A 65-year-old female who remains severely acidemic despite correction of her lactic acidosis and underlying anemia

c. A 22-year-old trauma patient who has exsanguinated from acute blood loss and is now receiving ACLS protocol

d. A 72-year-old male in renal failure with mixed acid-base disorder

e. A 44-year-old male who has just arrived to the emergency department with an acidemia of unknown origin

View Answer

9. You are caring for a patient who recently had a thyroidectomy. She complains of perioral numbness and has a positive Chvostek sign. While sending her blood for laboratory examination, she has a seizure. What treatment is indicated?

a. 0.9 NS, 1 L bolus

b. 0.9 NS, 1 L bolus, and a loop diuretic therapy

c. 20 mL of calcium gluconate intravenously over 20 minutes

d. Oral calcium carbonate

e. 4 IU/kg subcutaneous salmon calcitonin

View Answer

10. You have a postsurgical patient who is dehydrated with hypernatremia. You calculate a free water deficit of 3 L. How much free water should be given in the first 24 hours?

a. 1 L

b. 1.5 L

c. 2 L

d. 2.5 L

e. It is safe to correct the entire deficit over 24 hours

View Answer

5
Anticoagulation, Hemostasis, and Transfusions

Christopher P. Lawrance

Robert D. Winfield

There are two main goals of hemostasis: (1) To prevent bleeding from defects in vessel walls via the temporary formation of localized, stable clot and (2) repair of injured vessel walls.

I. MECHANISMS OF HEMOSTASIS.

Hemostasis is centered on the creation and destruction of a fibrin-cross-linked platelet plug (thrombus). Thrombus formation is limited to the area of vessel injury and is temporary in nature. This involves a complex interplay of thrombotic, anticoagulant, and fibrinolytic processes that occur simultaneously. Injury, disease, medications, and scores of other factors can tip the homeostatic balance resulting in life-threatening hemorrhagic or thrombotic complications.

A. Thrombus formation occurs in response to endothelial damage that exposes collagen and tissue factor (TF) to circulating blood (*N Engl J Med.* 2008;359:938). Two critical and interdependent events occur simultaneously to create a stable, fibrin-cross-linked thrombus: (1) Platelet plug formation and (2) blood coagulation.

1. Platelet plug formation. Exposed subendothelial collagen interacts with glycoprotein (GP) Ia/IIA and VI on platelets leading to tethering at an injured site. Platelet adhesion is reinforced by von Willebrand factor (vWF) interaction with GP Ib/V/IX. Engaged GP receptors further activate platelets leading to release of vasoactive agents and expression of important adhesion molecules, such as GP IIb/IIIa, which is involved in fibrin cross-linking of platelets.

2. Blood coagulation refers to the generation of fibrin via thrombin as the end-product of activation of serine proteases known as coagulation Òfactors.Ó These include both enzymatic proteins and cofactors (e.g., factors V and VIII). As with platelet plug formation, blood coagulation is initiated by endothelial disruption with uncovering of TF, a membrane protein expressed on multiple cell types including vascular cells such as fibroblasts and medial smooth-muscle cells. Importantly, TF is the sole initiator of thrombin generation and therefore fibrin formation. This coagulation network has traditionally been divided into intrinsic and extrinsic pathways (Fig. 5-1).

B. Endogenous anticoagulants are important to restrict coagulation to the specific area of vascular injury and prevent pathologic thrombosis.

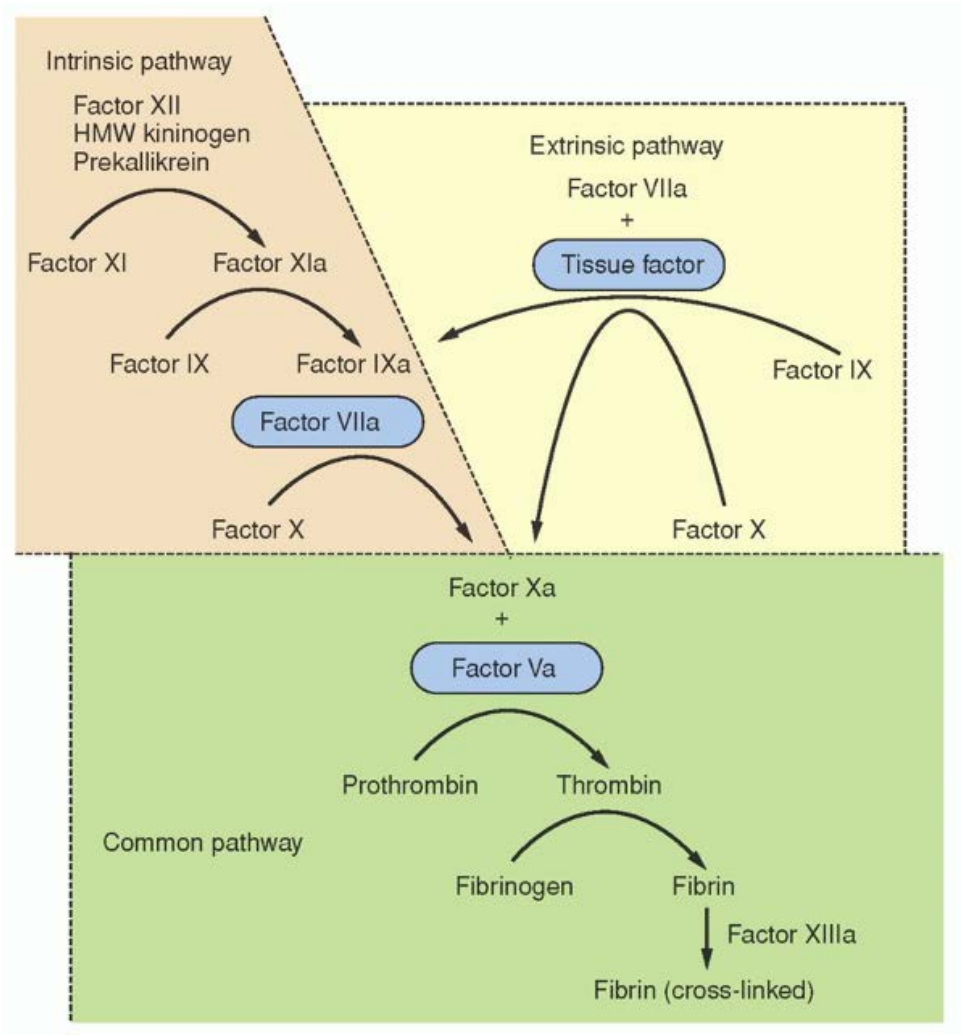


Figure 5-1 Blood coagulation cascade. Plasma zymogens are sequentially converted to active proteases (*arrows*). Nonenzymatic protein cofactors (*ovals*) are required at several stages of the cascade. Factors IX and X and prothrombin are activated on phospholipid surfaces. Thrombin cleaves fibrinogen, yielding fibrin monomers that polymerize to form a clot. HMW, high molecular weight.

1. The endothelium serves as a physical barrier sequestering subendothelial factors (TF, collagen) from platelets and circulating coagulation factors. Endothelial cells actively release antiplatelet factors (nitric oxide, prostacyclin [PGI_2]) and express surface enzymes that degrade adenosine diphosphate (ADP), a platelet-activating factor. Intact endothelial cells are also coated with thrombomodulin (see below) and heparin-like glycosaminoglycans that facilitate antithrombin (AT) activation.

2. AT (previously known as AT III) inhibits coagulation by binding several clotting factors (e.g., thrombin and factor Xa) and producing

complexes that are cleared from the circulation. Heparin markedly accelerates AT-induced factor inhibition, increasing factor clearance, and leading to anticoagulation.

3. The thrombomodulinÑprotein CÑprotein S system. Thrombomodulin, an endothelial membrane protein, binds with thrombin creating Òanticoagulant thrombinÓ that accelerates the activation of protein C, a vitamin K-dependent proenzyme. Activated protein C inactivates factors Va and VIIIa in the presence of protein S.

4. Other anticoagulant factors include TF pathway inhibitor (TFPI) that inactivates the TF/VIIa/Xa complex.

C. Fibrinolysis involves the dissolution and remodeling of thrombus. Plasminogen is a plasma zymogen that is incorporated into a developing thrombus. Tissue plasminogen activator (tPA) converts plasminogen to its active form, plasmin; breaks down clot; and allows for subsequent wound healing. Negative feedback includes (1) α-2 antiplasmin in blood, (2) plasminogen activator inhibitor (PAI-1) from platelets and endothelial cells, and (3) thrombin-activated fibrinolytic inhibitor (TAFI).

II. EVALUATION OF HEMOSTASIS.

A detailed history and physical examination constitute the most important screening tools for disorders of hemostasis in surgical patients (Table 5-1). A family history of bleeding or bleeding disorders should be elicited. Although surgical patients can have hereditary disorders of hemostasis, acquired defects and medications affecting hemostasis are most common. Below, evaluation is divided into evaluation of coagulation, platelets, and global hemostasis.

A. Evaluation of Coagulation

1. Laboratory evaluation

a. Prothrombin time (PT) is the clotting time measured after the addition of thromboplastin, phospholipids, and calcium to citrated plasma. This test assesses the extrinsic and common pathways and is most sensitive to factor VII deficiency. Test reagents vary in their responsiveness to warfarin-induced anticoagulation; therefore, the **international normalized ratio (INR)** is used to standardize PT reporting between laboratories.

b. Partial thromboplastin time (PTT) is the clotting time for plasma that is pre-incubated in particulate material (causing contact activation) followed by the addition of phospholipid and calcium. Inhibitors or deficiencies of factors in the intrinsic or common pathways cause prolongation 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 PTT prolongation due to inhibitors remains abnormal.

c. Activated clotting time (ACT) assesses the clotting time of whole blood. A blood sample is added to a diatomite-containing tube, leading to activation of the intrinsic pathway. The ACT is used to follow coagulation in patients requiring high doses of heparin (i.e., vascular procedures, percutaneous coronary interventions, extracorporeal membrane oxygenation [ECMO], or cardiopulmonary bypass). Automated systems are available for intraoperative use, allowing accurate and rapid determinations of the state of anticoagulation. Normal ACT is less than 130 seconds, with therapeutic target values ranging from 400 to 480 seconds for cardiac bypass procedures and 250 to 350 seconds for noncardiac vascular procedures.

TABLE 5-1 Preoperative Evaluation of Hemostasis, Bleeding Disorder, and Anemia

	History	Physical Examination	Drugs	Laboratory Values

Platelets	• Easy bruising
	• Frequent nosebleeds
	• Prolonged bleeding after:
	  ◦ Minor injury
	  ◦ Dental procedures
	  ◦ Surgery
	  ◦ Childbirth
		• Mucosal bleeding
		• Petechiae
		• Purpura
			• ASA
			• Clopidogrel
			• NSAIDs
				CBC with differential

Coagulation	After unrecognized injury, **delayed** development of: ◦ Hematomas ◦ Hemarthrosis • Nutritional status (i.e., vitamin K) • Family history of males with bleeding disorder (i.e., hemophilia) • Atrial fibrillation, DVT/PE, or other conditions requiring anticoagulant therapy	Joint fullness, bruising • Hematomas • Broad/large scar formation	Warfarin • Heparin, LMWH • Herbs, supplements	PT/INR PTT
Global	• Need for previous transfusions • Melena, hematochezia • Hematemesis • Hemoptysis • Family history of bleeding disorders	• Skin, conjunctival pallor • Tachycardia • Hypotension • Flow murmur		CBC Reticulocyte count Iron studies

ASA, Aspirin; CBC, complete blood count; DVT, deep vein thrombosis; LMWH, low-molecular-weight heparin; NSAID, nonsteroidal anti-inflammatory drug; PE, pulmonary embolism; PT/INR, prothrombin time/international normalized ratio; PTT, partial thromboplastin time.

d. Factor assays. Factor Xa activity may be used to assess the effect of low-molecular-weight heparin (LMWH). Factor VIII and IX levels are assessed in hemophiliacs prior to an operative procedure to guide transfusion of appropriate blood products. Fibrinogen level can be measured directly by functional or immunologic quantitative assays. Fibrin degradation product (FDP) elevations occur in many disease states characterized by increased fibrinogen turnover, including DIC and thromboembolic events, as well as during administration of fibrinolytic therapy. D-dimer levels reflect fibrinolysis and thus, the utility in surgical patients is less clear due to its nonspecific elevation in response to inflammation.

B. Evaluation of Platelets

1. Laboratory evaluation

a. Platelet count. Abnormalities in platelet number should be confirmed with a peripheral smear. It should be noted that the average life span of a normal platelet is 7 days.

b. Platelet function. No single test is adequate for screening platelet dysfunction due to limited sensitivity; therefore, the risk or severity of surgical bleeding cannot be reliably assessed. Bleeding time measures how long it takes bleeding to stop after a standardized superficial cut. Qualitative platelet disorders, von Willebrand disease (vWD), vasculitides, and connective tissue disorders prolong bleeding time.

C. Evaluation of Global Hemostasis

1. Thromboelastography (TEG)/Thromboelastometry (TEM). Traditional in vitro tests such as PT/INR, PTT, and ACT are useful in the diagnosis and management of bleeding diathesis; however, abnormal values obtained in a test tube are not always indicative or representative of underlying hemostatic perturbation. TEG and TEM provide broad functional assessments of the coagulation system. Briefly, a small amount of whole blood is added to a cup in which a pin is immersed in the blood. Either the cup (TEG) or pin (TEM) rotates and transmits kinetic information to the sensor, indicating the dynamic state of clot formation and breakdown within the sample (Fig. 5-2).

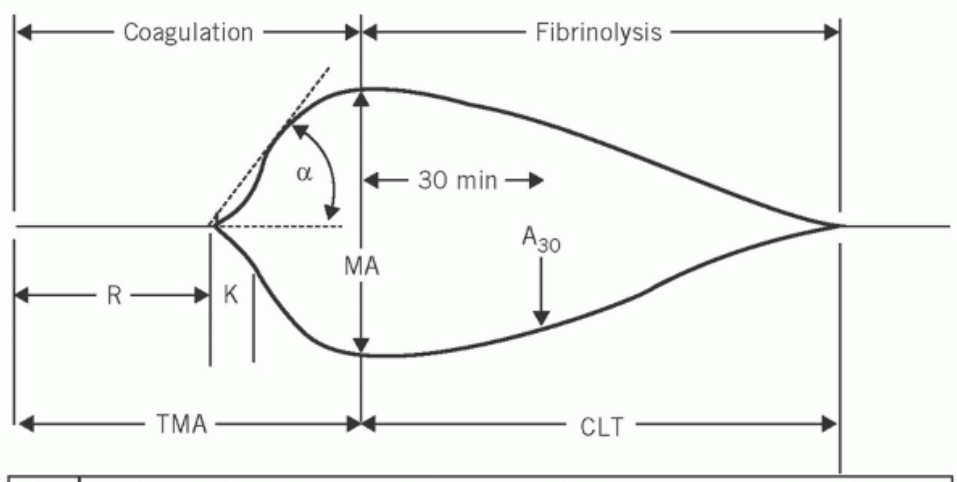
III. DISORDERS OF COAGULATION AND PLATELET FUNCTION

A. Disorders of Coagulation

1. Acquired factor deficiencies

a. Vitamin K deficiency leads to the production of inactive, noncarboxylated forms of **factors II (prothrombin), VII, IX, X, and**

proteins C and S. The diagnosis should be considered in a patient with a prolonged PTT that corrects with a 50:50 mixture of normal plasma. Vitamin K deficiency can occur in patients without oral intake within 1 week, with biliary obstruction, with malabsorption, and in those receiving antibiotics or warfarin.



R	Period of latency from when blood is placed into the TEG analyzer until the inital fibrin formation. Represents the enzymatic portion of coagulation.
K	Measures the speed to reach a certain level of clot strength (20 mm aboce baseline). This represents clot kinetics.
α	Measures the rapidity of fibrin built up and cross-linking (clot strengthening). This represents fibrinogen level.
MA	Maximum amplitude, is a direct function of the maximum dynamic properties of the fibrin and platelet binding via GPIIb/IIIa and represents the ultimate strength of the fibrin clot. This represents platelet function/aggregation.
LY30	Measures the rate of amplitude reduction 30 min after MA. This represents clot lysis.

Figure 5-2 Thromboelastogram (TEG). (Image modified from *Interact Cardiovasc Thorac Surg.* 2006;560-563.)

b. Liver dysfunction leads to complex alterations in coagulation through decreased synthesis of most clotting and anticlotting factors with the notable exceptions of factor VIII and vWF (from endothelium). Coagulopathy is worsened by uremic platelet dysfunction as well as thrombocytopenia from portal hypertension-associated hypersplenism. Spontaneous bleeding is infrequent, but coagulation defects should be corrected prior to invasive procedures. Fresh-frozen plasma (FFP) administration often improves the coagulopathy transiently.

c. Sepsis overstimulates the coagulation cascade resulting in decreased levels of anticoagulant factors such as protein C, protein S, and AT. This imbalance in hemostasis causes the formation of microvascular thrombi. These thrombi further amplify injury resulting in distal tissue ischemia and hypoxia.

d. Hemophilia is an inherited factor deficiency of either factor VIII (hemophilia A) or factor IX (hemophilia B, Christmas disease). The

diagnosis is suggested by patient history (Table 5-1) and an elevated PTT, normal PT, and normal bleeding time. Factor activity assays confirm the diagnosis and are an indicator of disease severity. Minor bleeding can often be controlled locally

without the need for factor replacement therapy. DDAVP stimulates the release of vWF into the circulation, which increases factor VIII levels two- to sixfold. This may control minor bleeding in patients with mild disease. Major bleeding (e.g., during a surgical procedure) requires factor VIII replacement. Recombinant factor VIIa is FDA approved for the treatment of patients who have developed inhibitors to factor VIII or IX (Section III.B.3). Cryoprecipitate contains factor VIII, vWF, and fibrinogen (Table 5-2) and can be used to treat patients with hemophilia A for control of bleeding. Purified factor IX is the treatment of choice for hemophilia B. Consultation with a hematologist is important prior to operating on individuals with hemophilia.

e. vWD is the most common inherited bleeding disorder with a prevalence as high as 1% of the general population. DDAVP is effective to increase plasma vWF in type 1 disease but is ineffective for types 2 and 3. These disorders require replacement via blood products with high amounts of vWF and factor VIII, such as cryoprecipitate (Table 5-2).

f. Inherited hypercoagulable disorders are defined as either type I (loss of inhibition) or type II (gain of procoagulant). These disorders put patients at risk of both venous and arterial thrombosis and may require lifelong anticoagulation. For most inherited disorders, unprovoked DVT treatment should continue for 6 months without the need for lifelong anticoagulation. Recommendations for lifelong anticoagulation include: (a) Homozygosity or two separate heterozygous thrombophilic defects, (b) an initial life-threatening thrombosis (massive PE, mesenteric thrombosis), (c) >2 spontaneous thrombosis (*Ann Intern Med.* 2001;135(5):367). Many clinicians will also choose to recommend lifelong anticoagulation for individuals with protein S, AT, and protein C deficiency as well since these three disorders have the highest lifetime risk of spontaneous thrombosis compared to normal individuals (8.5, 8.1, and 7.3 times more likely respectively) (*Blood.* 1998;92(7):2353). Specific thrombophilias are discussed below.

(1) Type I (Loss of inhibition)

AT deficiency is an autosomal-dominant disorder (prevalence 1:500) that presents with recurrent venous and occasionally arterial thromboembolism, usually in the second decade of life. Assays for AT levels are typically decreased in the setting of acute thrombosis and also if the patient is receiving heparin. Patients with acute thromboembolism or previous history of thrombosis are typically anticoagulated. AT-deficient patients should have the AT level restored to more than 80% of normal activity with AT concentrate prior to operation or childbirth. Many recommend starting lifelong anticoagulation after a single unprovoked DVT in patients with AT deficiency given the high lifetime risk of developing DVTs (8.1 times more likely).

TABLE 5-2 Blood Products

Blood Product	Volume (mL)	Additional Factors	Expected Response	Common Use
PRBC 1 unit	200-250	Fibrinogen: 10-75 mg Clotting factors: none	Increase: 1 mg/dL Hgb 3% HCT	ABLA MTP Surgical blood loss
Platelets SDP (apheresis) RDP[a]	300-500 50 per unit	Fibrinogen: 2-4 mg/mL (360-900 mg) Clotting factors: equivalent of 200-250 mL of plasma (hemostatic level) Ò6 packÓ of pooled RDP similar to SDP	Increase: 30-60 K/mm^3 Increase: 7-10 K/mm^3 per unit	Plt count <10 K MTP Bleeding with known qualitative plt defect

FFP[b] 1 unit	180-300	Fibrinogen: 400 mg Clotting factors: 1 mL contains 1 active unit of each factor (II, V, VII, IX, X, XI)	Decrease: PT/INR PTT	Coagulopathy Warfarin overdose DIC
Cryo 10 pack		Fibrinogen: 1,200-1,500 Clotting factors: VIII, vWF, XII	Decrease: PT/INR PTT Increase: fibrinogen level	vWD DIC Hemophilia A

[a] 4-10 RDP units are pooled prior to transfusion.

[b] Note: Duration of FFP effect is approximately 6 hours. INR of FFP is 1.6 to 1.7.

ABLA, acute blood loss anemia; Cryo, cryoprecipitate; FFP, fresh-frozen plasma; Hbg, hemoglobin; HCT, hematocrit; MTP, massive transfusion protocol; PRBC, packed red blood cells; plt, platelet; RDP, random donor platelets; SDP, single donor platelets.

Protein C deficiency and protein S deficiency are risk factors for venous thrombosis. In a state of protein C or S deficiency, factors Va and VIIIa are not adequately inactivated, thereby allowing unchecked coagulation. Besides the inherited type, protein C deficiency is encountered in patients with liver failure and in those who are receiving warfarin therapy. Symptomatic patients are treated with heparin (or LMWH) anticoagulation followed by warfarin therapy. In individuals with diminished protein C activity, effective heparin anticoagulation must be confirmed before warfarin initiation because warfarin transiently lowers protein C levels further and potentially worsens the hypercoagulable state manifested as warfarin-induced skin necrosis (see below). Patients with protein C or S deficiency but with no history of thrombosis typically do not require prophylactic anticoagulation; however, many will elect to start lifelong treatment after a single unprovoked event given the high lifetime risk of developing a DVT.

(2) Type II (Gain of procoagulant)

Activated protein C resistance (factor V Leiden) is the most common hereditary coagulation disorder accounting for 40% to 50% of inherited hypercoagulable disorders. Factor V Leiden is caused by a genetic mutation in factor V that renders it resistant to breakdown by activated protein C leading to venous thrombosis. Routine preoperative screening in asymptomatic patients is unnecessary. Therapy for venous thrombosis consists of anticoagulation with heparin followed by warfarin therapy and lifelong anticoagulation is typically not required.

Prothrombin G201210A is a mutation of the prothrombin gene at nucleotide 201210 which causes heterozygous carriers to have 30% more plasma prothrombin levels compared to those without the mutation. This results in a lifetime DVT risk of 2.8 times that of normal individual. Individuals with this mutation alone do not generally require lifelong anticoagulation.

Hyperhomocysteinemia is a rare autosomal recessive disorder resulting in elevated homocysteine levels. Homocysteine is an intermediate amino acid formed in the conversion of methionine to cysteine and usually results from a decrease in activity of methylene tetrahydrofolate reductase (MTHFR). Vitamin supplementation with folate has been shown to lower homocysteine levels and elevated homocysteine levels are a risk factor for VTEs; however, lowering homocysteine levels with vitamin supplementation has not resulted in a lower incidence of VTEs (*Blood.* 2007;109(1):139).

g. Acquired hypercoagulable disorders

(1) Antiphospholipid antibodies are immunoglobulins that are targeted against antigens composed in part of platelet and

endothelial cell phospholipids. Antiphospholipid antibody disorders may be detected by lupus anticoagulant, anticardiolipin, or other antiphospholipid antibodies. Patients with these antibodies are at risk for arterial and venous thrombosis, recurrent

miscarriages, and thrombocytopenia.

(2) Other acquired hypercoagulable states include malignancies, pregnancy or the use of estrogen therapy, intravascular hemolysis (e.g., hemolytic anemia or that seen after cardiopulmonary bypass), and the localized propensity for thrombosis in arteries that have recently undergone endarterectomy, angioplasty, or placement of prosthetic vascular grafts.

B. Platelet Disorders

1. Thrombocytopenia is defined as a platelet count of less than 140,000/μL. If platelet function is normal, thrombocytopenia is infrequently the cause of bleeding unless counts are below 50,000/μL. Severe spontaneous bleeding may occur with platelet counts under 10,000/μL. Intramuscular injections, rectal examinations, suppositories, or enemas should be limited in this circumstance. Occult liver disease must be considered for thrombocytopenia of unknown etiology.

2. Drug-induced thrombocytopenia. Many drugs can affect platelet production or cause increased platelet destruction. Common offenders include antibiotics (e.g., penicillin, linezolid, and sulfonamides), thiazide diuretics, and chemotherapeutic agents. Increased destruction is most commonly the result of an immune mechanism in which platelets are destroyed by complement activation following formation of drug-antibody complexes. All nonessential drugs should be discontinued until the cause of the thrombocytopenia is identified. Drug-induced thrombocytopenia typically resolves within 7 to 10 days after cessation and clearance of the offending agent. Prednisone (1 mg/kg/day orally) may facilitate recovery of platelet counts.

3. Heparin-induced thrombocytopenia (HIT) is a unique form of drug-induced thrombocytopenia in which two different forms have been recognized.

a. HIT type I is a nonimmune, heparin-associated thrombocytopenia that typically begins within 4 days of initiation of heparin therapy. The incidence ranges from 5% to 30% and may not require cessation of heparin.

b. HIT type II is a severe immune-mediated syndrome caused by heparin-dependent antiplatelet antibodies (anti-PF4-heparin complex) occurring 5 to 10 days after initial exposure to heparin but within hours after re-exposure. Platelet counts are often less than 100,000/μL or drop by more than 30% from baseline. In a minority of cases, thrombotic events ensue, including extensive arterial and venous thrombosis (*N Engl J Med.* 2006;355:809). If HIT is suspected, all heparin products should be stopped immediately until the diagnosis is refuted, including occult sources of heparin (e.g., flushes and heparin-coated catheters).

c. Laboratory tests. HIT is a clinical diagnosis with laboratory findings. The enzyme-linked immunosorbent assay (ELISA, HIT panel) and serotonin release assay (SRA) may suggest the diagnosis if the associated clinical features are present. The ELISA is a sensitive test that is useful for screening but has low specificity. The SRA is the gold standard due to its high sensitivity and specificity and often used as a confirmatory test.

d. Treatment. Since thrombotic complications can continue even after heparin cessation, anticoagulation with nonheparin anticoagulants such as direct thrombin inhibitor (Section III.D.3) is recommended if there are no contraindications. Platelet transfusion will exacerbate the process and is contraindicated. If HIT is present, warfarin administration can potentiate a hypercoagulable state and has been associated with the development of venous limb gangrene. Therefore, warfarin therapy should not be initiated until (1) the platelet count is >150 K and (2) the patient is therapeutically anticoagulated with another agent. Until the platelet count is >150 K, patients are in a procoagulant state. Warfarin therapy causes an initial decrease in the antithrombotic factors, protein C and protein S. This results in hypercoagulation during procoagulant states as there is now a state of uninhibited activated thrombin.

e. Dilutional thrombocytopenia can occur with rapid blood product replacement for massive hemorrhage. No formula predicts accurate platelet requirements in this setting (Section III.D.3).

f. Other causes of thrombocytopenia include disseminated intravascular coagulation (DIC), sepsis, immune thrombocytopenic purpura (ITP), thrombotic thrombocytopenic purpura (TTP), hemolytic uremic syndrome (HUS), dialysis, and hematopoietic disorders.

g. Thrombocytosis is defined as a platelet count greater than 600,000/μL. Essential thrombocytosis is caused by myeloproliferative disease. Secondary thrombocytosis occurs with splenectomy, iron deficiency, malignancy, or chronic

inflammatory disease. Aspirin therapy (81 mg/day orally) is useful in prevention of thrombotic events in patients with myeloproliferative disorders and in decreasing fetal loss in pregnant women, but secondary thrombocytosis usually requires no specific therapy.

h. Qualitative platelet dysfunction

(1) Acquired defects of platelets are caused by uremia, liver disease, or cardiopulmonary bypass. Desmopressin acetate (DDAVP, 0.3 µg/kg intravenously, administered 1 hour before an operation) may limit bleeding from platelet dysfunction, particularly in uremic patients. Conjugated estrogens (0.6 mg/kg/day intravenously for 5 days) also can improve hemostatic function.

(2) Hereditary defects of platelet dysfunction (e.g., vWD, Bernard-Soulier syndrome, Glanzmann thrombasthenia, and storage pool defects) are less common and usually warrant consultation with a hematologist.

4. Global disorders of hemostasis

a. DIC has many inciting causes, including sepsis and extensive trauma or burns. The pathogenesis involves inappropriate generation of thrombin within the vasculature, leading to platelet activation, formation of fibrin thrombi, and increased fibrinolytic activity. DIC often presents with complications from microvascular thrombi that involve the vascular beds of the kidney, brain, lung, and skin. In some patients, the consumption of coagulation factors, particularly fibrinogen, and the activation of the fibrinolytic pathway can lead to bleeding. Laboratory findings in DIC include thrombocytopenia, hypofibrinogenemia, increased FDPs, and prolonged PTT. Therapy begins with treatment of the underlying cause. Correction of coagulopathy with platelet transfusions, FFP, and cryoprecipitate should be undertaken for bleeding complications but should not be empirically given the potential risk of worsening inappropriate coagulation due to DIC.

IV. ANTICOAGULATION AND ANTIPLATELET MEDICATIONS

A. Anticoagulation Medications

1. 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. Table 5-3 summarizes selected anticoagulant medications. Relative contraindications to anticoagulation therapy include recent surgical intervention, severe trauma, intracranial or other sites of active bleeding, and in patients with an increased risk of falling.

2. Heparin

a. Unfractionated heparin

(1) Administration. Heparin is administered parenterally. PTT should be measured before initiation of heparin, 6 hours after initiation of the drip and 6 hours after each change in dosing. Use of heparin boluses is not generally recommended for surgical patients. Platelet counts should be measured daily until a maintenance dose of heparin is achieved and periodically thereafter to monitor for development of HIT.

(2) Complications that occur with heparin therapy include bleeding and HIT. If bleeding occurs, heparin should be discontinued, and immediate assessment of the PT, PTT, and complete blood count (CBC) should be undertaken. Gastrointestinal (GI) bleeding that occurs while a patient is therapeutically anticoagulated suggests an occult source and warrants further evaluation.

(3) Heparin clearance is rapid and dose dependant. Initially heparin is cleared by the reticuloendothelial system. Once saturated, clearance is renally dependant and takes longer. Reversal can be achieved more quickly with intravenous protamine sulfate. Each milligram of protamine sulfate reverses approximately 100 units of heparin. The PTT or ACT can be used to assess the adequacy of the reversal. Protamine should be used with caution because it can induce anaphylactoid reactions, systemic hypotension from splanchnic vasodilation, and other complications.

TABLE 5-3 Anticoagulant Medications

Drug	Mechanism	Metabolism	Dose for DVT Prophylaxis	Dose for Therapeutic Anticoagulation	Therapeutic Target	Reversal Agent
Heparin	Potentiates antithrombin: IIa, Xa, IXa, XIa, XIIa inhibition	Hepatic, RES and 50% renal excretion	5,000 U SC twice to thrice daily	Bolus = 80 µ/kg Infusion = 18 µ/kg/hr; adjust to target PTT	aPTT = 60-80 s	Protamine: start with 25-50 mg
LMWH (e.g., Enoxaparin [Lovenox])	Potentiates antithrombin: Xa inhibition	Mainly renal excretion	40 mg SC once daily	1 mg/kg SC twice daily	Chromogenic anti-Xa assay: 0.6-1 anti-Xa U/mL	None
Fondaparinux (Arixtra)	Potentiates antithrombin: Xa inhibition	Renal	2.5 mg SC once daily	5 or 7.5 or 10 mg SC once daily	Chromogenic anti-Xa assay 0.6-1 anti-Xa U/mL	None
Rivaroxaban (Xarelto)	Direct Xa inhibition	Likely liver	10 mg PO once daily		Chromogenic anti-Xa assay 0.6-1 anti-Xa U/mL	None
Apixaban (Eliquis)	Direct Xa inhibition	Liver	5 mg PO BID		Chromogenic anti-Xa assay 0.6-1 anti-Xa U/mL	None
Warfarin (Coumadin)	Prevents carboxylation of X, IX, VII, II, protein C and S	Hepatic, marked genetic variability		2-10 mg PO daily; adjust to target INR	INR = 2-4	Vitamin K: 1-10 mg PO or plasma; start with 2-4 units
Lepirudin (Refludan)	Direct thrombin inhibition	Renal		Bolus = 0.4 mg/kg Infusion = 0.15 mg/kg/hr; adjust to target PTT	aPTT = 60-80 (1.5-2.5 times control)	None

Bivalirudin (Angiomax)	Direct thrombin inhibition	Proteolytic cleavage and renal (20%)		Bolus = 1 mg/kg Infusion = 0.2 mg/kg/hr; adjust to target PTT	aPTT = 60-80 s	None
Desirudin (Iprivask)	Direct thrombin inhibition	Renal	10-15 mg SC twice daily		Prolongs the aPTT	None
Argatroban (Acova)	Direct thrombin inhibition	Hepatic		Infusion = 2 µg/kg/min; adjust to target PTT	aPTT = 60-80 s; may prolong INR	None
Dabigatran etexilate (Pradaxa)	Direct thrombin inhibition	Renal (unchanged) and some conjugation with glucuronic acid	150 mg PO once daily	150 mg PO twice daily	Prolongs aPTT	None

b. LMWH preparations include enoxaparin, dalteparin, and tinzaparin. The anticoagulant effect of LMWH is predominantly due to factor Xa inhibition via potentiation of AT, and use of LMWH results in less thrombin inhibition than unfractionated heparin. The advantages of LMWH include a more predictable anticoagulant effect, less platelet interaction, and a longer half-life. LMWH may be used for longer-term therapy in patients with a contraindication to oral anticoagulant treatment (e.g., pregnant patients who cannot take warfarin); however, this practice has been called into question. Because LMWH has a longer half-life and no effective antidote, it must be used with caution in surgical patients and in those in whom a bleeding risk has been substantiated. Furthermore, because LMWH is renally cleared, adjustments need to be made for patients in renal insufficiency and should not be given to patients in renal failure.

3. Direct thrombin inhibitors are a class of compounds that bind to free and fibrin-bound thrombin. These agents inhibit thrombin activation of clotting factors, fibrin formation, and platelet aggregation.

a. Lepirudin (recombinant hirudin) binds irreversibly to thrombin, providing effective anticoagulation. The drug is approved in patients with HIT but may be considered in other severe clotting disorders. **Bivalirudin** is a truncated form of recombinant hirudin that targets only the active site of thrombin. Bivalirudin is FDA approved for HIT as well as for use during percutaneous coronary angioplasty and stenting. Because these agents are cleared by the kidneys, they should not be used in patients with renal failure.

b. Argatroban is a synthetic thrombin inhibitor that is also approved for treatment of HIT. It is cleared by the liver and should not be used in patients with hepatic failure.

4. Warfarin is an oral vitamin K antagonist that causes anticoagulation by inhibiting 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, so the full warfarin anticoagulant effect is not apparent for 5 to 7 days. When immediate anticoagulation is necessary, heparin or another agent must be used initially.

a. Administration. Warfarin usually is initiated with a loading dose of 5 to 10 mg/day for 2 days, followed by dose adjustment based on daily INR results. Elderly patients, those with hepatic insufficiency, and those who are receiving parenteral nutrition or broad-spectrum antibiotics, should be given lower initial doses. A daily dose of warfarin needed to

achieve therapeutic anticoagulation ranges from 2 to 15 mg/day. Assistance with dosing can be obtained from www.warfarindosing.org, keeping in mind that this website should not replace good clinical judgment. An INR of 2 to 3 is therapeutic for most indications, but patients with prosthetic heart valves should be maintained with an INR of 2.5 to 3.5. Once a stable INR is obtained on a given warfarin dose, it can be monitored biweekly or monthly.

b. Complications. The bleeding risk in patients who are treated with warfarin is estimated to be approximately 10% per year. The risk of bleeding correlates directly with the INR. Warfarin-induced skin necrosis, caused by dermal venous thrombosis, occurs in patients with preexisting protein C and S factor deficiencies who receive warfarin without systemic anticoagulation. In these patients, warfarin initiation results in a prothrombotic state as protein C and S levels are the first to significantly decrease. Warfarin can produce significant birth defects and fetal death and should not be used during pregnancy. Changes in medications and diet may affect warfarin or vitamin K levels and require more vigilant INR monitoring and dose adjustment.

c. Reversal of warfarin-induced anticoagulation requires up to 1 week after discontinuation of therapy. Vitamin K administration can be used to reverse warfarin anticoagulation within 1 to 2 days, but the effect can last for up to 1 week longer. The appropriate vitamin K dose depends on the INR and the urgency with which correction must be accomplished. For patients with bleeding or extremely high INR levels (>10), 10 mg of vitamin K should be administered intravenously. In addition, FFP or factor concentrates can be administered to patients with ongoing hemorrhage or in need of rapid reversal.

(1) Indirect factor Xa inhibitors (Fondaparinux) are small, synthetic, heparin-like molecules that enhance AT-mediated inhibition of factor Xa. Fondaparinux has been shown to be as effective in preventing DVT after hip and knee replacement. Monitoring of coagulation parameters is usually not necessary.

(2) Direct factor Xa inhibitors (rivaroxaban, apixaban) are a newer class of anticoagulation medications with rapid onset and fewer food/drug interactions when compared to warfarin. They are indicated for patients with atrial fibrillation and for DVT prophylaxis in patients receiving hip and knee surgery. This class of anticoagulants does not require monitoring, but no effective reversal agents are available if bleeding occurs.

B. Antiplatelet Medications

1. Aspirin irreversibly acetylates cyclooxygenase, inhibiting platelet synthesis of thromboxane A2 and causing decreased platelet function. It is often used in the prevention and treatment of acute transient ischemic attacks, stroke, myocardial infarction, and coronary and vascular graft occlusion. Aspirin should be discontinued about 1 week before elective nonvascular operations to allow new, functional platelets to form.

2. Clopidogrel (Plavix) is a thienopyridine that irreversibly inhibits platelet function by binding to the ADP receptor that promotes aggregation and secretion. It is used to decrease thrombotic events in percutaneous coronary and vascular stenting and in patients with unstable angina. Although the half-life of clopidogrel is 8 hours, the bleeding time remains prolonged over 3 to 7 days as the platelet population is turned over. Therefore, patients should discontinue clopidogrel therapy 7 days

prior to elective operations to decrease the risk of bleeding complications. Clopidogrel has become more prevalent with the increased use of coronary artery stents that require at least a full year of antiplatelet treatment for drug-eluting stents and at least 4 weeks with bare metal stents (*Chest.* 2008;133:776S). Discontinuation of clopidogrel within this time period carries a significant risk of stent thrombosis with resultant myocardial infarction. Continuation of aspirin, consultation with a cardiologist, and consideration of preoperative ÒbridgeÓ with GP IIb/IIIa inhibitors (see below) should be considered.

3. GP IIb/IIIa inhibitors including abciximab (ReoPro), tirofiban (Aggrastat), and eptifibatide (Integrilin) function by blocking platelet adhesion to fibrin. These agents are used in preventing coronary artery thrombosis after coronary angioplasty or in unstable angina. Although they have relatively short half-lives (0.5 to 2.5 hours), the bleeding time may remain elevated for longer periods. It is recommended that surgery be delayed 12 hours after discontinuing abciximab and 4 hours after discontinuing tirofiban or eptifibatide. Zero-balance ultrafiltration may be beneficial in patients who need immediate surgical interventions (*Perfusion.* 2002;17:33).

4. Other medications: Dextran is used to reduce perioperative thrombotic events such as bypass graft occlusion because of its ability to decrease platelet aggregation and adhesion. **Nonsteroidal anti-inflammatory drugs (NSAIDs)** such as ketorolac (Toradol) inhibit cyclooxygenase, reversibly inhibiting platelet aggregation. This may result in clinically relevant bleeding, particularly in patients taking warfarin, aspirin, or clopidogrel (*Ann Intern Med.* 2014;18:161).

C. Fibrinolytic Therapy

1. Thrombolytic therapy is most often used for clinically significant arterial and venous thromboses. Contraindications to fibrinolytic therapy are listed in Table 5-4. tPA (Alteplase) or a recombinant analog (Reteplase), as well as urokinase (Abbokinase), are used for lysis of catheter, venous, and peripheral arterial thrombi.

V. TRANSFUSION THERAPY AND REVERSAL OF COAGULOPATHY.

The risks and benefits of transfusion therapy must be considered carefully in each situation. Informed consent should be obtained before blood products are administered. The indications for transfusion should be noted in the medical record. Before elective procedures that are likely to require blood transfusion, the options of autologous or directed blood donation 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 and improve the oxygen-carrying capacity of the blood. A hemoglobin level of 7 to 8 g/dL is adequate for tissue oxygenation in most normovolemic patients. However, therapy must be individualized based on the clinical situation rather than a hemoglobin level. The patient's age, cardiovascular and pulmonary status, volume status, the type of transfusion

(i.e., homologous vs. autologous), and the expectation of further blood loss should guide transfusion decisions. Of note, algorithm-guided approaches to transfusion that utilize laboratory and POC values (i.e., TEG) have been shown to be safe and decrease the number of blood products transfused in cardiac surgery (*Br J Anesth.* 2004;92:178) and are being evaluated for trauma (*Ann Surg.* 2010;251:604).

TABLE 5-4 Contraindications to Fibrinolytic Therapy

Absolute Contraindications
Intolerable ischemia (for arterial thrombosis)
Active bleeding (not including menses)
Recent (<2 mo) stroke or neurosurgical procedure
Intracranial pathology such as neoplasm
Relative Contraindications
Recent (<10 d) major surgery, major trauma, parturition, or organ biopsy
Active peptic ulcer or recent gastrointestinal bleeding (within 2 wk)

Uncontrolled hypertension (blood pressure >180/110 mm Hg)

Recent cardiopulmonary resuscitation

Presence or high likelihood of left heart thrombus

Bacterial endocarditis

Coagulopathy or current use of warfarin

Pregnancy

Hemorrhagic diabetic retinopathy

2. Transfusions in critically ill patients. Critically ill patients may be at increased risk for the immunosuppressive complications of transfusions and may benefit from a more restrictive transfusion protocol. This was demonstrated in the Transfusion Requirements in Critical Care (TRICC) trial, a randomized, controlled trial that showed significantly lower mortality rates with a restrictive transfusion strategy (transfusion for hemoglobin less than 7 g/dL) (*N Engl J Med.* 1999;340:409). Patients with active cardiac ischemia or infarction may benefit from a

higher hemoglobin level of >10 g/dL to improve oxygen delivery (*Am Heart J.* 2013;165(6):964).

3. Preparation. Before administration, both donor blood and recipient blood are tested to decrease transfusion reactions. Blood typing tests the recipient's RBCs for antigens (A, B, and Rh) and screens the recipient's serum for the presence of antibodies to a panel of known RBC antigens. Each unit to be transfused is then cross-matched against the recipient's serum to check for preformed antibodies against antigens on the donor's RBCs. In an emergency situation, type O/Rh-negative blood that has been prescreened for reactive antibodies may be administered prior to blood typing and cross-matching. After blood typing, type-specific blood can be given. Immunocompromised patients and those receiving blood from first-degree relatives should be given irradiated blood to prevent graft versus host disease (GVHD).

4. Administration (Table 5-2). Packed RBCs should be administered through a standard filter (170 to 260 μm) and an 18-gauge or larger intravenous catheter. The rate is determined by the clinical situation; typically, however, each unit of blood must be administered within 4 hours to prevent infection. Patients are monitored for adverse reactions during the first 5 to 10 minutes of the transfusion and frequently thereafter.

5. Alternatives to homologous transfusion exist and may provide advantages in safety and cost when used in elective procedures with a high likelihood of significant blood loss.

a. Autologous predonation is the preferred alternative for elective transfusions. Up to 20% of patients still require allogeneic transfusion, however, and transfusion reactions may still result from clerical errors in storage.

b. Isovolemic hemodilution is a technique in which whole fresh blood is removed and crystalloid is simultaneously infused in the immediate preoperative period. The blood is stored at room temperature and reinfused after acute blood loss has ceased. Moderate hemodilution (hematocrit 32% to 33%) is as effective as autologous predonation in reducing the need for allogeneic transfusion, and it is much less costly.

c. Intraoperative autotransfusion (Cell Saver) in which blood from the operative field is returned to the patient, can decrease allogeneic transfusion requirements. Equipment to separate and wash recovered RBCs is required. Contraindications include neoplasm and enteric or purulent contamination.

d. Erythropoietin may be effective in decreasing allogeneic transfusion requirements when given preoperatively. The

appropriate dose can be calculated based on anticipated transfusion requirements and is administered weekly over 2 to 4 weeks. Adjunctive use with autologous pre-donation has not consistently been shown to be effective. Chronic anemia, particularly anemia due to renal disease, is usually treated with erythropoietin (50 to 100 U/kg subcutaneously three times a week) rather than with transfusions. Erythropoietin should be used with caution in critically ill patients since it is associated with an increased risk of thrombotic events (*N Engl J Med.* 2007;357:965).

B. Transfusion Products for Coagulopathy (Table 5-2)

1. FFP contains all the coagulation factors. However, factors V and VIII may not be stable through the thawing process and are not reliably recovered from FFP. Therefore, it can be used to correct coagulopathies that are due to deficiencies of any other coagulation factor and is particularly useful when multiple factor deficiencies exist (e.g., liver disease or massive transfusion). FFP's effects are immediate and typically last about 6 hours.

2. Cryoprecipitate is the cold-insoluble precipitate of fresh plasma and is rich in factor VIII and vWF as well as fibrinogen, fibronectin, and factor XIII. Cryoprecipitate may be used as second-line therapy in vWD or hemophilia but is most often used to correct fibrinogen deficiency in DIC or during massive transfusion.

3. Recombinant human factor VIIa (rhFVIIa, NovoSeven) is FDA approved for the treatment of hemophilia with inhibitors of factors VIII or XI. The recommended dose for this indication is 90 to 120 µg/kg, which can be repeated every 2 hours for 24 hours. The off-label use of rhFVIIa has been utilized as Òrescue therapyÓ for patients with severe or dangerous bleeding. rhFVIIa used in blunt trauma was shown to reduce the overall blood transfusion requirement and the incidence of multisystem organ failure and acute respiratory distress syndrome (*Crit Care.* 2006;10:R178; *J Trauma.* 2006;60:242); however it has fallen out of favor due to increased risks of arterial and venous thrombosis (*N Engl J Med.* 2010;363:1791).

4. Factor concentrates are an emerging option for rapid reversal of anticoagulation. These agents (e.g., Prothrombin Complex Concentrate [PCC]) contain high levels of factors II, IX, and X. Factor VII may also be a component of these products or may be administered separately. In addition to providing rapid reversal, these agents require lower volumes than FFP administration and thus may reduce cardiac complications in patients with CHF or dysrhythmias.

5. Platelet transfusions are used to control bleeding that is caused by thrombocytopenia or platelet dysfunction and to prevent spontaneous bleeding in situations of severe thrombocytopenia. In cases of bleeding or for minor surgical procedures, the transfusion threshold is often increased to a platelet count of less than 50,000/µL. Preparations, volumes, and expected response are summarized in Table 5-2.

C. Complications of Transfusions

1. Infections. Current methods of blood screening have greatly reduced the transmission rate of viral disease. Hepatitis B transmission is in the range of 1 in 205,000 units transfused. The risk of HIV or hepatitis C transmission is in the range of 1 in 2 million units transfused. Cytomegalovirus (CMV) transmission is a risk in CMV-negative, immunocompromised patients. Bacteria and endotoxins can be infused with blood products, particularly in platelets that are stored at room temperature with coagulase-negative staph being the most common organism. Parasitic infections also can be transmitted, although rarely, with blood products.

2. Transfusion reactions

a. Allergic reactions are the most common type of transfusion reactions and occur when the patient reacts to donated plasma proteins in the blood. Symptoms include itching or hives and can often be treated with antihistamines such as diphenhydramine (25 to 50 mg orally or intravenously). Prophylactic administration of diphenhydramine (Benadryl) and prednisone prior to a transfusion may be considered in patients with a previous history of allergic reaction. Rarely, severe reactions may involve bronchospasm or laryngospasm, which should prompt discontinuation of the infusion. Steroids and subcutaneous epinephrine may also be required.

b. Febrile nonhemolytic reactions involve the development of a high fever during or within 24 hours of a transfusion. This reaction is mediated by the body's response to WBCs in donated blood. General malaise, chills, nausea, or headaches may accompany the fever. Because fever can be the first manifestation of a more serious transfusion reaction, the situation

must be promptly evaluated. Patients with a previous history of a febrile reaction should receive leukoreduced blood products.

c. Acute immune hemolytic reactions are the most serious transfusion reactions, in which patient antibodies react to transfused RBC antigens causing intravascular hemolysis. This typically occurs with ABO or Rh incompatibility. Symptoms include nausea, chills, anxiety, flushing, and chest or back pain. Anesthetized or comatose patients may show signs of excessive incisional bleeding or oozing from mucous membranes. The reaction may progress to shock or renal failure with hemoglobinuria. If a transfusion reaction is suspected, the infusion should be stopped immediately. Identities of the donor unit and recipient should be rechecked because clerical error is the most common cause. A repeat cross-match should be performed in addition to a CBC, coagulation studies, and serum bilirubin. 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/hour using volume resuscitation and possibly diuretics if resuscitation is adequate.

d. Delayed hemolytic reactions result from an anamnestic antibody response to antigens other than the ABO antigens to which the recipient has been previously exposed. Transfused blood cells may take days or weeks to hemolyze after transfusion. Typically there are few signs or symptoms other than a falling RBC count or elevated bilirubin. Specific treatment is rarely necessary, but severe cases should be treated like acute hemolytic reactions, with volume support and maintenance of urine output.

e. Transfusion-related acute lung injury (TRALI) may be one of the most common causes of morbidity and mortality associated with transfusion. TRALI typically occurs within 1 to 2 hours of transfusion but can occur any time up to 6 hours later. Support can vary from supplemental oxygen to intubation and ventilation. Although most cases resolve on their own, severe cases can be fatal.

f. 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. It has an associated mortality of greater than 80%. Irradiation of donor blood from first-degree relatives of immunocompetent patients and all blood for immunocompromised patients prevents this complication.

g. Volume overload after blood transfusion can occur in patients with poor cardiac or renal function, or as a component of the transfusion-associated circulatory overload (TACO) phenomenon. Careful monitoring of the volume status and judicious use of diuretic therapy can reduce the risk of this complication.

h. Alloimmunization occurs in 50% to 75% of patients receiving repeated platelet transfusions and presents as a failure of the platelet count to increase significantly after a transfusion. This occurs in immunocompetent individuals who mount an immune response to platelet specific antigens which include class I human leukocyte antigens (HLA). Therefore, in patients who need long-term platelet therapy, HLA-matched single-donor platelets slow the onset of alloimmunization.

i. Posttransfusion purpura is a rare complication of platelet transfusions seen in previously transfused individuals and multiparous women. It is usually caused by antibodies that develop in response to a specific platelet antigen PI^{A1} from the donor platelets. This condition presents with severe thrombocytopenia, purpura, and bleeding occurring 7 to 10 days after platelet transfusion. Although fatal bleeding can occur, the disease is typically self-limiting. Plasmapheresis or an infusion of intravenous immunoglobulin may be helpful.

D. Massive transfusion, usually 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. Coagulopathy might arise as a result of platelet or coagulation factor depletion. This has led to the use of transfusion ratios in the trauma setting that involve the transfusion of platelets and FFP in concert with packed red blood cells (PRBCs). No definitive ratio has been established, but ratios of 1:1 or 1:2 FFP:PRBC are common. Many centers have established massive transfusions protocols (MTPs) which make use of these ratios. The algorithm for an MTP at Barnes-Jewish Hospital is shown in Figure 5-3. These prepacked boxes serve to maintain the proper ratio of clotting factor and platelets to RBCs during situations requiring rapid volume expansion, and they remove the confusion and human error that can result when having to ÒthinkÓ about proper ratios during an emergency. Hypothermia can result from massive volume resuscitation with chilled

blood products but can be prevented by using blood warmers. Hypothermia can lead to cardiac dysrhythmias and coagulopathy. Citrate toxicity can develop after massive transfusion in patients with hepatic dysfunction. Hypocalcemia can be treated with intravenous administration of 10% calcium gluconate. Electrolyte abnormalities, including acidosis and hyperkalemia, can rarely occur after massive transfusions, especially in patients with preexisting hyperkalemia.

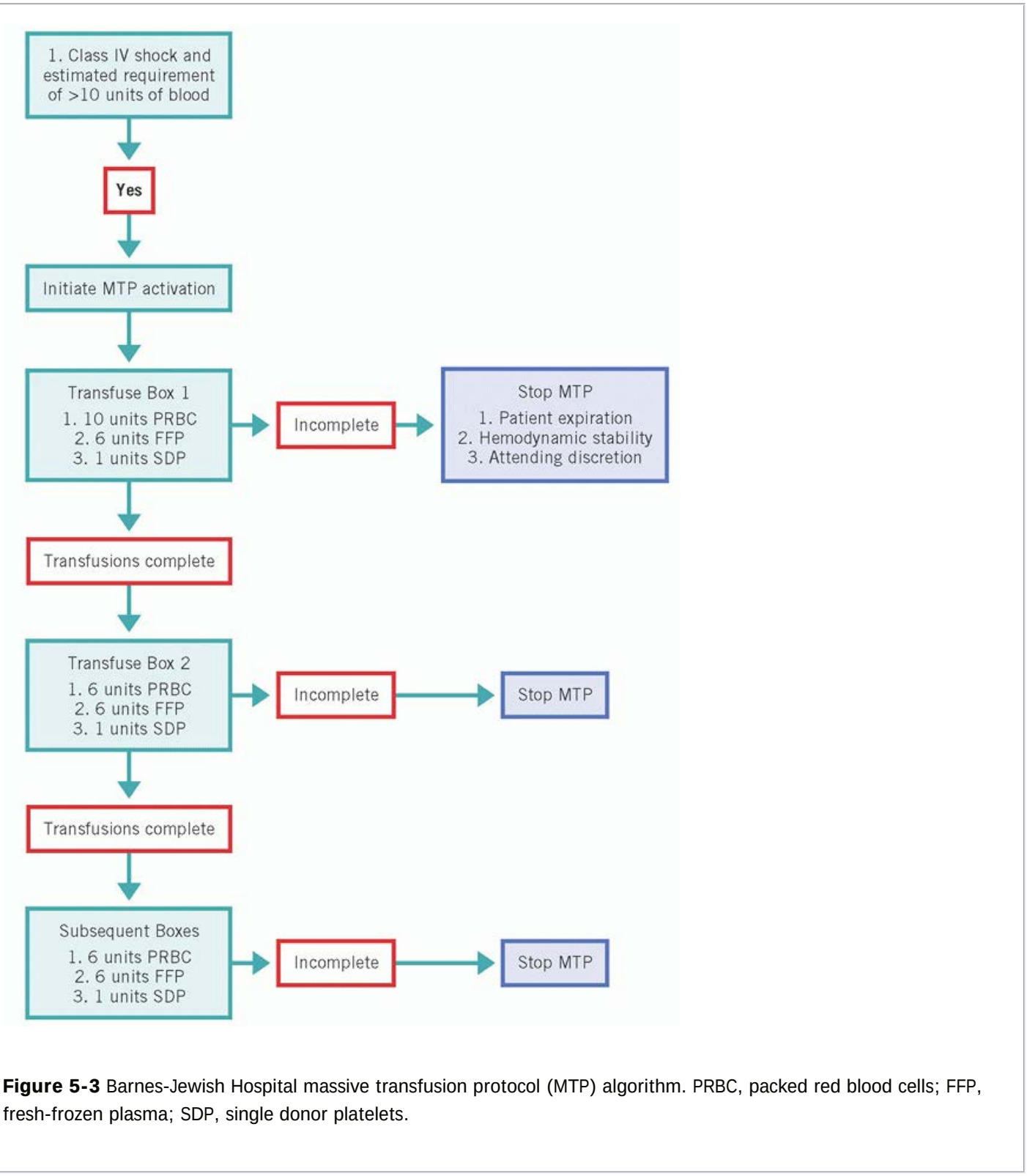


Figure 5-3 Barnes-Jewish Hospital massive transfusion protocol (MTP) algorithm. PRBC, packed red blood cells; FFP, fresh-frozen plasma; SDP, single donor platelets.

VI. LOCAL HEMOSTATIC AGENTS.

Local hemostatic agents promote hemostasis by providing a matrix for thrombus formation, and these agents can aid in the intraoperative control of mild to moderate surgical bleeding, such as that from needle punctures, vascular suture lines, or areas of extensive tissue dissection. Generally speaking, Gelfoam, Surgicel, and Helistat are reasonable agents when direct pressure can be applied, including for superficial solid organ injuries as part of hepatorrhaphy or splenorrhaphy. Avitene, Hemotene, topical thrombin, Tisseel, and Evicel are best used when there is more diffuse bleeding from a large dissection bed (i.e., following a retroperitoneal dissection). Floseal is particularly useful for areas where there is a small cavity with mild hemorrhage (such as a penetrating hepatic injury), as it can be used to fill the cavity as it is injected. Bleeding that is pulsatile, visibly discrete, or severe in nature will not respond to topical hemostatics. Anastomotic bleeding usually is best controlled with local pressure or a simple suture. Each agent is described below.

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 to 6 weeks without a 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 slowly resorbed and creates a foreign body reaction similar to that of cellulose.

D. Microfibrillar collagen (e.g., Avitene and Hemotene) can be sprayed onto wounds and anastomoses for hemostasis, particularly in areas that are difficult to reach. It stimulates platelet adhesion and promotes thrombus formation. Since microfibrillar collagen can pass through autotransfusion device filters, it should be avoided during procedures that utilize the cell saver.

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. 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. Fibrin sealants (e.g., Tisseel and Evicel) are prepared by combining human thrombin and human fibrinogen. These components are separated prior to administration and are mixed during application to tissue via a dual-syringe system. An insoluble, cross-linked fibrin mesh is created which provides a matrix for thrombus formation.

CHAPTER 5: HEMOSTASIS, ANTICOAGULATION, AND TRANSFUSION THERAPY

Multiple Choice Questions

1. A 57-year-old male who developed atrial fibrillation is noticed to have a drop in his platelets to 60,000/μL 7 days after initiation of a heparin drip. He goes on to develop lower-extremity swelling, and the presence of a new DVT is confirmed with ultrasound. What if any changes should be made to address his anticoagulation regimen and thrombocytopenia?

- **a.** Continue heparin drip
- **b.** Discontinue all anticoagulation and place an IVC filter
- **c.** Start the patient on a bivalirudin drip
- **d.** Start platelet transfusion
- **e.** Obtain a hematology consult

View Answer

2. A 45-year-old male is septic and found to have DIC with hypofibrinogenemia. Which of the following products would be appropriate to administer?

- **a.** FFP
- **b.** PRBCs
- **c.** DDAVP
- **d.** Cryoprecipitate
- **e.** Whole Blood

View Answer

3. Patients with Hemophilia A:

- **a.** Commonly present with spontaneous bleeding
- **b.** Should receive factor IX prior to surgery
- **c.** May produce factor VII inhibitors
- **d.** Are deficient in platelet membrane receptors
- **e.** Are mostly female

View Answer

4. Which of the following is not an absolute contraindication to fibrinolytic therapy?

- **a.** Intolerable ischemia
- **b.** Active bleeding
- **c.** Recent stroke or neurosurgical procedure
- **d.** Intracranial neoplasm
- **e.** Active menses

View Answer

5. Which of the following facts concerning von Willebrand disease is correct?

- **a.** It is the third most common inherited bleeding disorder.
- **b.** It is characterized by low levels of vWF alone.
- **c.** It is characterized by ineffective vWF alone.
- **d.** Type 2 is treated with DDAVP.
- **e.** Type 3 is treated with cryoprecipitate.

View Answer

6. Which of the following concerning factor XIIIa is correct?

- **a.** It is involved in the activation of platelets.
- **b.** It is involved in the cross-linking of fibrin.
- **c.** It is not involved in coagulation cascade.
- **d.** It is deficient in Christmas disease.
- **e.** It is found in the prothrombinase assembly which involves factors Va, Xa, and calcium.

View Answer

7. A deficiency of all of the following will result in hypercoagulability except:

- **a.** Prekallikrein
- **b.** Protein C

c. Protein S

d. Plasminogen

e. Antithrombin III

View Answer

8. Which of the following is correct regarding antithrombin III?

a. It is a necessary cofactor for heparin.

b. It is activated by argatroban.

c. It is secreted by endothelial cells.

d. Its synthesis is affected by warfarin.

e. It is inhibited by fondaparinux.

View Answer

9. Which of the following regarding the prothrombinase complex is correct?

a. It is inhibited by heparin.

b. It is inhibited by argatroban.

c. It does not require ionized calcium.

d. It is inhibited by clopidogrel.

e. It is inhibited by aspirin.

View Answer

10. Cryoprecipitate includes:

a. Factor II

b. Factor VII

c. Factor IX

d. Factor X

e. vWF

View Answer

6

Anesthesia

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Tracey Wagner Stevens

Thorough preoperative assessment of the surgical patient is an integral component of comprehensive anesthesia care and is critical to patient safety. It includes a complete history including previous anesthetic complications, physical examination including airway and vascular access evaluation, optimization of patient comorbidities, and perioperative management of home medications. Please see Chapter 1 for a detailed discussion of preoperative patient evaluation.

ANESTHESIA TECHNIQUES AND MEDICATIONS

Anesthesia care can be broadly categorized into four major types: Local, regional, monitored anesthesia care (MAC), and general anesthesia. Multimodal anesthesia strategies employ combinations of these techniques to improve patient outcomes while minimizing the adverse effects of anesthetic medications (e.g., less sedation, earlier ambulation, faster return of bowel function).

I. BASIC ANESTHESIA MONITORING STANDARDS.

These apply to patients undergoing all types of anesthesia. Minimum standards for patient monitoring include continuous evaluation of oxygenation, ventilation, circulation, and temperature.

A. Oxygenation

1. Oxygen analyzer measurement of inspired gas

2. Pulse oximetry with variable pitch pulse tone and low threshold alarm

B. Ventilation

1. Continuous end-tidal CO_2 analysis when endotracheal tube or laryngeal mask is used, noninvasive analysis with moderate or deep sedation

C. Circulation

1. Blood pressure

2. Pulse

3. Continuous electrocardiogram

D. Body temperature when clinically significant changes are anticipated. (American Society of Anesthesiologists (ASA) Standards for Basic Anesthesia Monitoring, July 2011)

II. LOCAL ANESTHESIA.

Local anesthesia refers to the blockade of sensory nerve impulses by injection or application near the surgical site. Local anesthetics are categorized into two groups: **Amino esters (one i)** include tetracaine, procaine, cocaine, and chloroprocaine; **amino amides (two i's)** include lidocaine, bupivacaine, ropivacaine, and mepivacaine. Characteristics of commonly used local anesthetic agents are summarized in Table 6-1.

TABLE 6-1 Local Anesthetics for Infiltration[a]

	Maximum Dose (mg/kg)		Length of Action (hr)	
Agent	**Plain**	**With Epinephrine[a]**	**Plain**	**With Epinephrine[b]**
Procaine	7	9	0.5	0.5-1
Lidocaine	4	7	0.5-1	2
Mepivacaine	4	7	0.75-1.5	2
Bupivacaine	2.5	3	2-4	3-4
Ropivacaine	3	4	2-4	3-4
Tetracaine	1.5		24	

[a] See Ch. 36 Local Anesthetics in *Miller's Anesthesia,* Saunders, 2015 for dosing recommendations for regional anesthesia applications.

[b] 1:200,000.

A. Mechanism of Action

1. The **mechanism of action** of local anesthetics is blockade of voltagegated sodium channels, thereby inhibiting neuronal depolarization and axonal conduction.

2. Local tissue acidosis (e.g., from infection) slows the onset and decreases the intensity of analgesia by causing local anesthetic molecules to become positively charged and less able to diffuse into the neuron.

B. Toxicity (Dose Dependent, Except for Hypersensitivity Reactions)

1. Central nervous system (CNS) toxicity includes mental status changes, dizziness, perioral numbness, a metallic taste, tinnitus, and visual disturbances. Seizures can result from overdose or inadvertent intravascular injection.

2. Cardiovascular toxicity ranges from decreased cardiac output to hypotension and cardiovascular collapse. Most local anesthetics cause CNS toxicity before cardiovascular toxicity. Bupivacaine (Marcaine) is an exception, and its intravascular injection can result in severe cardiac compromise.

3. Treatment of local anesthetic systemic toxicity (LAST) involves airway support and ventilation with 100% oxygen. Benzodiazepines are preferred for seizure suppression; propofol should be avoided in patients with cardiovascular instability. Modified BLS and ACLS protocols should be followed for management of cardiac arrhythmias, with avoidance of vasopressin, Ca^{2+}-channel blockers, and beta blockers, and reduction of individual epinephrine doses to <1 µg/kg. **Lipid emulsion therapy** should be used based on the clinical severity and rate of progression of LAST, and is given as a 1.5 mL/kg IV bolus of 20% lipid emulsion followed by

continuous infusion at 0.25 mL/kg/min. Bolus doses may be repeated up to two times, with a recommended upper limit of 10 mL/kg over the first 30 minutes (see American Society of Regional Anesthesia Checklist for Treatment of LAST, *Reg Anesth Pain Med.* 2012;37(1):16-18).

4. 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 anaphylactic reactions are questionable.

a. Signs and symptoms can range from urticaria to bronchospasm, hypotension, and anaphylactic shock.

b. Treatment is similar to that for hypersensitivity reactions from other etiologies. Urticaria responds to diphenhydramine, 25 to 50 mg IV. Bronchospasm is treated with inhaled bronchodilators (e.g., albuterol) and oxygen. Hypotension is treated with fluid resuscitation and vasopressors or small incremental doses of epinephrine as required. Anaphylactic cardiovascular collapse should be treated with epinephrine, 0.5 to 1 mg, administered as an IV 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, and penis).

III. REGIONAL ANESTHESIA.

Regional anesthesia refers to either neuroaxial or peripheral nerve blockade with local anesthetic to inhibit the sensation of pain in a certain area of the body.

A. General Considerations

1. Supplements to regional anesthesia. Local infiltration by the surgeon may be required if there is an incomplete block. IV sedation using short-acting agents can also be helpful. Conversion to general anesthesia may be required when a regional technique provides inadequate analgesia or surgical positioning is not tolerated.

2. Ultrasound imaging is replacing landmark-based and nerve stimulation techniques as a guidance tool for peripheral nerve blockade, resulting in more consistent blockade and decreased complications.

B. Neuroaxial Blockade

1. Spinal anesthesia involves the injection of low-dose local anesthetic solution into the subarachnoid space at the level of the lumbar spine.

a. Level of analgesia is affected by multiple variables. The **baricity** of the agent and the **position** of the patient immediately after injection are the major determinants of level. The **total dose injected** and the **total volume injected** are also important factors.

b. Onset and duration of analgesia are primarily determined by the **specific characteristics of the local anesthetic** used. Variability in the length of analgesia is significant, ranging from as little as 30 minutes (lidocaine) to up to 6 hours (tetracaine with epinephrine).

c. Complications

(1) Hypotension occurs as a result of sympatholytic-induced vasodilation. It is more severe in hypovolemic patients or in those with preexisting cardiac dysfunction. Treatment includes IV fluids, vasopressors, and positive inotropic and chronotropic drugs. Leg elevation and Trendelenburg positioning can be used to increase venous return to the heart. It is advisable to administer 500 to 1,000 mL of crystalloid prior to spinal block to avoid hypotension due to spinal anesthesia.

(2) High spinal blockade. Inadvertently high levels of spinal blockade may result in hypotension (blocking dermatomes T1ÑT4: Preganglionic cardioaccelerator nerves), dyspnea (loss of chest proprioception or intercostal muscle function, diaphragmatic paralysis due to C3ÑC5

blockade), or apnea (decreased medullary perfusion secondary to hypotension). Treatment consists of ventilatory support and/or intubation, IV fluids, and chronotropic and inotropic support.

(3) Headache can result from leakage of CSF at the dural puncture site. 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 bed rest, abdominal binders, oral or IV fluids, oral analgesics, and caffeinated beverages. Severe refractory headache may require placement of an epidural blood patch to prevent ongoing leakage of CSF.

(4) CNS infection after spinal anesthesia, although extremely rare, may result in meningitis, epidural abscess, or arachnoiditis.

(5) Permanent nerve injury is exceedingly rare and is seen with the same frequency as in general anesthesia.

(6) Urinary retention with bladder distention can sometimes occur in patients with spinal anesthesia whose bladders are not drained by urethral catheters.

d. Contraindications

(1) Absolute contraindications to spinal anesthesia are lack of consent, localized infection at the planned puncture site, increased intracranial pressure, generalized sepsis, and coagulopathy.

(2) Relative contraindications include hypovolemia, preexisting CNS disease, chronic low back pain, platelet dysfunction, and preload-dependent valvular lesions such as aortic and mitral stenosis.

2. Epidural anesthesia is similar to spinal anesthesia except that the needle remains in the epidural space (between the ligamentum flavum and dura mater) and is not advanced through the dura. A flexible catheter is often advanced into the space to allow for repeat bolus doses or continuous infusion of local anesthetics and opioids.

a. Level of analgesia is primarily determined by the **volume** of injection, as well as by patient position, age, and **area of placement.**

b. Onset and duration of analgesia

(1) Epidural anesthesia develops more slowly than does spinal anesthesia because the local anesthetic solution must diffuse further. The rate of onset of sympathetic blockade and hypotension also is slowed, providing for less acute hemodynamic effects compared with spinal anesthesia.

(2) The **dosing interval** and **duration of action** depends on the agent used.

c. Complications are similar to those encountered with spinal anesthesia, and additionally

include:

(1) The management of postoperative epidural-associated **hypotension** includes:

(a) IV fluid bolus

(b) Decreased epidural infusion rate

(c) Initiation of vasopressor such as phenylephrine

(d) Evaluation for other causes of postoperative hypotension (e.g., bleeding, sepsis)

(e) Modifying infusion to remove local anesthetic

(2) Postdural puncture (spinal) headache (PDPH) may result from inadvertent perforation of the dura. Treatment is the same as with spinal anesthesia.

(3) Unintended dural puncture may be managed by withdrawing the catheter and repeating the procedure at a different level, or by intrathecal placement of the epidural catheter, which avoids a repeat procedure, ensures adequate anesthesia, and may reduce the incidence of CSF leak and PDPH. Care must be taken, however, to avoid high block and inappropriate drug administration.

(4) Intravascular catheter placement (usually in epidural veins) is potentially devastating due to the potential for intravascular injection of local anesthetic and risk of systemic toxicity. The ability to aspirate blood or a positive response to a test dose warrants removal of the epidural catheter placement.

(5) 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.

d. Combined spinal and epidural anesthesia

(1) A **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 for administration of anesthetic to the subarachnoid space prior to placement of the epidural catheter. This procedure combines the quick onset of spinal analgesia with the continuous dosing advantages of epidural analgesia.

C. Peripheral Nerve BlockadeÑUpper Extremity

1. Brachial plexus blockade. Blockade of the upper extremity is achieved by injection of local anesthetic into the brachial plexus sheath by one of several approaches.

a. Interscalene blockade targets the trunks of the brachial plexus and is used for shoulder and upper arm surgery because it reliably blocks the shoulder. The lower trunk is often missed making interscalene blockade unsuitable for distal arm surgery. Ipsilateral recurrent laryngeal nerve, stellate ganglion, and phrenic nerve blockade can result in hoarseness, Horner syndrome, and

dyspnea from diaphragmatic paralysis, respectively.

b. Supraclavicular blockade targets the divisions and is used for arm surgery. Supplementation with supraclavicular nerve block allows for shoulder coverage. Risks are similar to interscalene blockade. The risk of pneumothorax is reduced with the use of ultrasound guidance.

c. Infraclavicular blockade targets the cords. It is suitable for arm and hand surgery. Phrenic nerve block is unlikely and it is therefore the preferred option for patients with severe pulmonary disease.

d. Other regional techniques for anesthesia of the upper extremity include **axillary blockade,** distal blocks of the **radial, median, and ulnar nerves, digital blockade, and intravenous regional anesthesia (Bier block).**

D. Peripheral Nerve BlockadeÑLower Extremity

1. Femoral nerve blockade is used for anterior thigh, femur, and knee surgery by blocking the femoral nerve at the groin. Complications are rare but include femoral arterial puncture and hematoma.

2. Other regional techniques for anesthesia of the lower extremity include blockade of the **popliteal nerve,** the **saphenous nerve,** and the **ankle.**

E. Miscellaneous Regional Anesthesia Techniques

1. Intercostal nerve block is indicated after thoracotomy or before chest tube placement. Local anesthetic is injected just below the rib in the posterior axillary line, usually for a distance of five interspaces surrounding the interspace of interest. Complications include pneumothorax and intravascular injection. Injection into the nerve sheath with retrograde spread to the spinal cord can produce a high spinal or epidural block.

2. Paravertebral nerve block targets the spinal nerves at the level of the paravertebral space. It is most commonly performed at the thoracic level for breast surgery, thoracotomy, or rib fractures.

3. Transversus abdominis plane (TAP) block targets the cutaneous branches of the low thoracic and lumbar spinal nerves that travel in the plane between the transversus abdominis and internal oblique muscles. It may be used for lower abdominal surgery such as appendectomy, inguinal hernia repair, caesarean section, and prostatectomy.

4. Interested readers are referred to the New York Society of Regional Anesthesia website for excellent reviews of regional anesthesia techniques (www.nysora.com).

IV. MONITORED ANESTHESIA CARE.

This describes a multimodal approach to anesthesia for minor procedures and surgeries that do

anesthesia. MAC involves a combination of local and/or regional anesthesia of the operative site with mild sedation and analgesia. Full monitors are applied and supplemental oxygen is administered via nasal cannula or face mask. Patients should maintain spontaneous respirations and the ability to respond to the anesthesia provider. Vigilance is essential to ensure the avoidance of apnea and airway obstruction. Medications commonly used during MAC are summarized in Table 6-2.

TABLE 6-2 Medications for Short-term Sedation and Analgesia during Procedures

Agent	Route	Dose (as Needed)	Comments
Dexmedetomidine (Precedex)	IV	1 µg/kg over 10 min, then 0.2-1 µg/kg/hr	May cause bradycardia and hypotension, transient hypertension during loading
Fentanyl	IV	25-50 µg q5-10min	Opioids provide analgesia with unpredictable sedative effects
Ketamine (Ketalar)	IV/IM	0.2-0.8 mg/kg IV q10-15min 4-6 mg/kg IM	Provides sedation and analgesia, co-administer midazolam to reduce emergence reactions
Midazolam (Versed)	IV	0.5-1 mg q15min	Benzodiazepines do not provide analgesia
Propofol (Diprivan)	IV	10-20 mg over 3-5 min q10min, or 25-75 µg/kg/min	May cause hypotension, especially with bolus dosing

IV, intravenous; q, every.

V. GENERAL ANESTHESIA.

A balanced approach to general anesthesia provides unconsciousness, amnesia, analgesia, and skeletal muscle relaxation.

A. Premedication is often used in the immediate preoperative period for anxiolysis and amnesia. Common agents include benzodiazepines (e.g., midazolam) or opioids (e.g., fentanyl).

B. Induction of General Anesthesia. IV agents are most widely used owing to rapid onset and ease of administration. All patients must be fully monitored and preoxygenated with 100% oxygen prior to induction.

1. Propofol (Diprivan), a phenol derivative (1 to 3 mg/kg IV), is a commonly used agent for both induction and maintenance of anesthesia. Onset of action is immediate. Propofol decreases systemic vascular resistance and blood pressure, and should be used with caution in patients with hypotension or active coronary ischemia. The incidence of postoperative nausea and vomiting (PONV) is low. The pharmacokinetics are not changed by chronic hepatic or renal failure. Pain on injection can be attenuated with a low-dose IV lidocaine injection.

2. Etomidate (Amidate), an imidazole derivative (0.3 mg/kg IV), has an onset of 30 to 60 seconds and has only mild direct hemodynamic depressant effects. **Adrenal insufficiency** may result from a single administration.

3. Ketamine (Ketalar), a phencyclidine derivative (1 to 4 mg/kg IV), provides dissociative anesthesia and is also an excellent analgesic. It increases cardiac output and BP in patients who are not catecholamine depleted, and is an ideal induction agent for patients with bronchospasm. It does, however, raise intracranial pressure and should not be used in patients with head trauma. Ketamine can be given with midazolam for its amnestic properties, to address the side effects of **emergence delirium** and **hallucinations.** It is often used in the pediatric population with the advantage that it can be given **intramuscularly.**

C. Neuromuscular blockade is achieved with acetylcholine receptor antagonists that act on postsynaptic receptors in the neuromuscular junction to produce muscle relaxation. Agents are categorized as either depolarizing or nondepolarizing (Table 6-3). Blockade can facilitate endotracheal intubation and may improve operating conditions for many surgical procedures. Its use, however, increases the risk for intraoperative awareness and postoperative neuromuscular weakness. It should only be used when clinically indicated, and normal neuromuscular function should be ascertained prior to extubation or stopping the anesthetic. While nerve monitors are not a standard ASA monitoring requirement, they should be used whenever neuromuscular blockade is performed.

1. Depolarizing agents include only **succinylcholine (Anectine, Quelicin)** in current clinical use, a rapidly acting (60 seconds) and rapidly metabolized agent that allows return of

neuromuscular function in 5 to 10 minutes. In certain patients, the normally mild hyperkalemic response can be greatly exaggerated, possibly leading to cardiac arrest. Its use is therefore usually contraindicated in patients with severe burns, trauma, paralysis, neuromuscular disorders, or prolonged bed rest. It can also cause increased intraocular, intracranial, and gastric pressures. It is contraindicated in those with a personal or family history of **malignant hyperthermia.** Prolonged neuromuscular blockade can occur in patients with pseudocholinesterase deficiency.

2. Nondepolarizing agents can be divided into short-, intermediate-, and long-acting (Table 6-3). Associated hemodynamic effects and elimination pathways vary. These agents are distinguished from succinylcholine by reversibility and reduced risk of malignant hyperthermia.

TABLE 6-3 Agents Producing Neuromuscular Blockade

Agent	Initial Dose (mg/kg)	Duration (min)	Elimination	Associated Effects
Depolarizing				
Succinylcholine	1-1.5	3-5	Plasma cholinesterase	Fasciculations, increase or decrease in heart rate, transient hyperkalemia, known malignant hyperthermia trigger agent
Nondepolarizing				
Atracurium	0.2-0.4	20-35	Ester hydrolysis	Histamine release
Cisatracurium	0.1-0.2	20-35	Ester hydrolysis	

Vecuronium	0.1-0.2	25-40	Hepatic and renal	Histamine release
Rocuronium	0.6-1.2	30	Hepatic and renal	
Pancuronium	0.04-0.1	45-90	Primarily renal	Increase in heart rate, mean arterial BP, and cardiac output

BP, blood pressure.

3. Reversal of neuromuscular blockade with **acetylcholinesterase inhibitors** (neostigmine, 0.06 to 0.07 mg/kg, and edrophonium, 0.1 mg/kg) 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; therefore a spontaneously ventilating patient **may still be unable to protect the airway.** Dosage response should be monitored by a peripheral nerve stimulator (train-of-four) to assess twitches. Strength can also be assessed by having the patient raise their head from the bed for 5 seconds or more. A muscarinic anticholinergic agent such as atropine or glycopyrrolate is used to counteract the side effects of these reversal agents.

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 procedures that do not require neuromuscular relaxation. Nasopharyngeal and oral airways can relieve obstruction and make mask ventilation more effective.

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 fiber-optic bronchoscope. Newer optical and video laryngoscopes are also helpful devices in difficult intubations.

3. Supraglottic airway devices are alternative airway support devices that are positioned above the larynx. The laryngeal mask airway (LMA), the most commonly used supraglottic device, is inserted blindly into the pharynx to form a low-pressure seal around the laryngeal inlet. Although supraglottic devices allow ventilation with gentle positive pressure, they do not

definitively protect the airway from aspiration. Their use is contraindicated in nonfasted patients, morbidly obese patients, and patients with obstructive or abnormal lesions of the oropharynx.

4. Management of the difficult airway. Several adjuncts to endotracheal intubation may facilitate management of the difficult airway, including supraglottic devices, intubating stylets, optical or video laryngoscopy, fiber-optic bronchoscopy, and invasive airway access (see American Society of Anesthesiologists Difficult Airway Algorithm (p. 257), *Anesthesiology.* 2013;118(2):251-270).

5. Rapid sequence intubation (RSI) is an algorithm that guides the approach to endotracheal intubation in emergent scenarios and in patients at increased risk for aspiration. It can be applied in preoperative, emergency department, and critical care settings (see RSI Algorithm (p. 1408), *Chest.* 2005;127(4):1397-1412).

E. Maintenance of Anesthesia

1. Inhalational agents

a. Volatile anesthetics include **isoflurane (Forane), sevoflurane (Ultane),** and **desflurane (Suprane).** They provide unconsciousness

and amnesia with less cardiovascular depression than previously used agents. Isoflurane has a relatively slow rate of metabolism. Sevoflurane is nonirritating to the airway and is the preferred agent for inhalational induction. Desflurane is less fat soluble and therefore metabolized more quickly; however, it is a pungent airway irritant and should not be used for induction or in patients with severe reactive airway disease.

b. 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, suspected pneumothorax, or those undergoing ophthalmologic procedures.

2. Intravenous agents may be used alone (total intravenous anesthesia [TIVA]) or as a supplement to inhalational agents for anesthesia maintenance, and include opioids, benzodiazepines, propofol, **dexmedetomidine (Precedex),** and ketamine. TIVA carries an increased risk of intraoperative awareness, and the use of electroencephalogram (EEG) or processed EEG (e.g., bispectral index) monitoring is recommended.

F. Recovery from General Anesthesia

1. 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.

2. Patients recover from the effects of anesthesia in the **postanesthesia care unit (PACU).**

Patient readiness for PACU discharge can be evaluated with tools such as the **Aldrete Post Anesthetic Recovery Score** (*Anesth Analg.* 1970;49:924-933).

G. Complications of General Anesthesia

1. Intraoperative awareness includes intraoperative consciousness and/or explicit recall of intraoperative events, and places patients at risk of developing psychological sequelae. Risk factors include the use of TIVA, neuromuscular blockade, and anesthetic underdosing due to technical, surgical, or patient-related factors. Management includes recognition and avoidance of risk factors, and prompt referral for psychological evaluation in patients reporting awareness.

2. Delayed emergence from general anesthesia may be attributable to residual medication effect, hypercarbia or hypoxia, hypoglycemia, hypothermia, electrolyte abnormalities, or neurologic complications. Treatment may include a trial of naloxone, flumazenil, or physostigmine for anesthetic reversal, followed by laboratory and radiologic evaluation for alternative causes.

3. Malignant hyperthermia is a hypermetabolic disorder of skeletal muscle that is characterized by intracellular hypercalcemia and rapid adenosine triphosphate consumption. An acute episode is a life-threatening emergency. This condition is initiated by exposure to a triggering agent,

such as desflurane, enflurane, halothane, isoflurane, sevoflurane, and succinylcholine. Signs and symptoms may occur in the operating room or more than 24 hours postoperatively and include tachycardia, tachypnea, hypertension, hypercapnia, hyperthermia, acidosis (metabolic with/without respiratory component), and skeletal muscle rigidity. Treatment involves immediate cessation of triggering agents and administration of **dantrolene (Dantrium)** (1 mg/kg IV up to a total dose of 10 mg/kg). Acidosis and hyperkalemia should be monitored and treated appropriately. Intensive care monitoring for 48 to 72 hours is indicated after an acute episode of malignant hyperthermia to evaluate for recurrence, acute tubular necrosis, pulmonary edema, and disseminated intravascular coagulation. Readers are referred to the Malignant Hyperthermia Association of the United States (www.mhaus.org, (800) 644-9737 for 24-hour emergency assistance).

4. Hypothermia occurs by increased heat losses due to peripheral vasodilation during general anesthesia. Hypothermia is more pronounced in the elderly and may lead to prolonged emergence, cardiac arrhythmias, and coagulopathy. Treatment should be preventative, including warming the operating room prior to the patient's arrival and minimizing unnecessary patient exposure prior to draping. Active warming with forced-air convective warmers is effective, but care should be taken to avoid use on ischemic extremities.

5. Laryngospasm may occur due to noxious stimulation of the vocal cords by the endotracheal tube, blood, or other oral secretions. Forceful apposition of the vocal cords restricts or completely prevents airflow through the larynx. This can cause airway compromise and lead to negative-pressure pulmonary edema. Treatment involves the use of positive-pressure ventilation by mask

to break the spasm. Succinylcholine may be required in refractory cases to allow successful ventilation.

6. PONV occurs in approximately 30% of patients undergoing general anesthesia, and is more common in preadolescents, women, and obese patients. Cortical (pain, hypotension, hypoxia), visceral (gastric distention, visceral traction), vestibular, and chemoreceptor trigger zone (opioids) afferent stimuli can all play a role in the mechanism. Medications including opioids, etomidate, inhalational gasses, and reversal agents such as neostigmine have also been implicated. Commonly used agents for the treatment of PONV include **ondansetron (Zofran), prochlorperazine (Compazine), promethazine (Phenergan),** and **diphenhydramine (Benadryl).** Agents better used for prophylaxis of PONV include **dexamethasone (Decadron)** and transdermal **scopolamine (Scopace).**

7. Postanesthesia shaking/shivering may be uncomfortable or painful to the patient, and significant metabolic effects may result, including acidosis and myocardial ischemia. The clonic component from residual inhalational anesthetic is exacerbated by hypothermia. Shivering may be relieved by administration of **meperidine (Demerol)** or other opioids, although these are less effective.

8. Urinary retention, although not uncommon with spinal anesthesia, occurs in only 1% to 3% of cases involving general anesthesia. It most commonly occurs after pelvic operations and in conjunction with benign prostatic hypertrophy (BPH). Early urinary catheter removal, early ambulation, and resumption of BPH medications may reduce the occurrence. Treatment may require urinary catheter placement.

9. Nerve injury can occur secondary to improper positioning of the patient on the operating table or insufficient padding of dependent regions. Resulting nerve palsies can be long lasting and debilitating. Prophylactic padding of sensitive regions and attention to proper positioning remain the most effective preventative therapies.

POSTOPERATIVE ANALGESIA AND COMPLICATIONS

I. POSTOPERATIVE ANALGESIA.

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, IV, or epidural route. Consultation with a dedicated Pain Management service is recommended for patients whose postoperative pain is difficult to manage.

A. Opioids are the most commonly used agents for postoperative analgesia. **Fentanyl, morphine,** and **hydromorphone (Dilaudid)** are most often administered in IV form by nursing staff or via a **patient-controlled analgesia (PCA)** device. With PCA, the patient has the ability to self-deliver analgesics

within preset safety parameters. Continuous “basal” infusions are rarely used in the surgical population due to the risk of respiratory compromise with opioid toxicity. **Hydrocodone** and **oxycodone** are most often administered by the oral route.

B. Nonnarcotic adjuncts for postoperative analgesia include continuous infusion local anesthetic devices (e.g., On-Q Pain Relief System), **acetaminophen (Tylenol, Ofirmev),** nonsteroidal anti-inflammatory drugs (NSAIDs; e.g., **ketorolac [Toradol]** and **ibuprofen [Motrin**]), and **gabapentin (Neurontin).**

C. Epidural and peripheral nerve infusions are also useful adjuncts for postoperative pain caused by thoracotomy, extensive abdominal incisions, or extremity procedures (see Section III. Regional Anesthesia).

D. Side Effects and Complications

1. Oversedation and respiratory depression

a. Arousable, spontaneously breathing patients should be given supplemental oxygen and be 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),** 0.04 mg IV repeated every 30 to 60 seconds until the patient is arousable. Excess naloxone may result in severe pain and/or severe hypertension with possible

pulmonary edema. Adequate ventilation should be confirmed by an 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 recurrence of sedation as the effects of naloxone dissipate.

2. Apnea

a. Treatment involves immediate supportive mask ventilation and possible intubation if no improvement in clinical status. Naloxone administration should be considered.

3. Nausea and vomiting: Consider decreasing dosage, alternate medication, and/or giving opioid relief with ondansetron.

4. Pruritus

a. Symptomatic relief may be provided with diphenhydramine or **hydroxyzine (Vistaril).**

5. Serotonin toxicity can result from combination of **monoamine oxidase**

inhibitors (e.g., isocarboxazid, phenelzine) with **phenylpiperidine derivative opioids** such as meperidine, tramadol, methadone, and fentanyl. This interaction may result in severe hemodynamic swings, respiratory depression, seizures, diaphoresis, hyperthermia, and coma. Meperidine has been most frequently implicated and should be avoided.

CHAPTER 6: ANESTHESIA

Multiple Choice Questions

1. A 56-year-old, otherwise healthy male is undergoing elective right inguinal hernia repair under local anesthesia. Immediately following anesthetic injection under the external oblique aponeurosis, the patient acutely becomes unconscious, hypotensive, and begins convulsing. Which of the following is a correct statement about the management of local anesthetic systemic toxicity?

a. Propofol is the preferred medication for seizure suppression.

b. Vasopressin should be used to counteract the reduction in vascular tone.

c. Cardiovascular collapse should be treated with epinephrine according to standard ACLS protocol dosing.

d. Lipid emulsion therapy should be implemented based on the clinical severity and rate of progression of symptoms.

e. Monitoring may be discontinued 2 hours after treatment for local anesthetic toxicity.

View Answer

2. A 44-year-old woman is brought to the operating room for elective laparoscopic cholecystectomy and undergoes uncomplicated induction of general anesthesia and endotracheal intubation. The minimum standards for basic monitoring of a patient undergoing general anesthesia include all of the following parameters EXCEPT:

a. Electrocardiogram

b. Cerebral oximetry

c. Temperature

d. Blood pressure

e. End-tidal CO_2

View Answer

3. A 65-year-old man with coronary artery disease and chronic obstructive pulmonary disease, requiring 2 L of home oxygen therapy, is being prepared to undergo operative fixation of a right distal humerus fracture. Which of the following regional anesthesia techniques is preferred to reduce the risk of postoperative pulmonary complications in this patient?

a. Cervical blockade

b. Interscalene blockade

c. Supraclavicular blockade

d. Infraclavicular blockade

e. Axillary blockade

View Answer

4. Which of the following muscle groups demonstrates the earliest recovery from neuromuscular blockade following administration of an anticholinesterase reversal agent?

a. Adductor pollicis

b. Diaphragm

c. Geniohyoid

d. Pharyngeal

e. Flexor hallucis

View Answer

5. Minutes after receiving anesthesia induction with sevoflurane and succinylcholine, a 23-year-old male undergoing elective inguinal hernia repair develops a temperature of 39.2°C, heart rate of 148 bpm, blood pressure of 186/104 mm Hg, and muscle rigidity. Which of the following is the most appropriate FIRST step in management?

a. Administration of IV dantrolene

b. Hyperventilation with 100% oxygen

c. Cessation of volatile anesthetics

d. Obtain STAT potassium level and electrocardiogram

e. Obtain full family medical history

View Answer

6. Which of the following is a risk factor for intraoperative awareness

during general anesthesia?

a. Use of neuromuscular blockade

b. Use of inhalational anesthesia

c. Older patient age

d. Elective surgery

e. Hypothyroidism

View Answer

7. In which of the following situations would the use of succinylcholine be preferred to a nondepolarizing neuromuscular blocking agent?

a. Severe burns

b. Rapid sequence intubation

c. Muscular dystrophy

d. Hyperkalemia

e. Family history of malignant hyperthermia

View Answer

7
Critical Care

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This chapter focuses on the monitoring of critically ill patients, the most common reasons for surgical ICU admissions (respiratory or circulatory failure) and sepsis. It also addresses the topics of sedation and analgesia, stress-induced ulcer prophylaxis, and the role of transfusion and glucose control in the critically ill.

I. MONITORING OF THE CRITICALLY ILL PATIENT

A. Temperature Monitoring. Critically ill patients should have their temperature measured at least every 4 hours. The fever cut-off is traditionally ³38.5iC. Some suggest avoidance of antipyretics as a strategy of temperature control because it may lead to an increased mortality (*Crit Care.* 2012;16:R33).

B. Electrocardiographic (ECG) Monitoring. Continuous ECG monitoring with telemetry allows for rapid detection of dysrhythmias and assessment of heart rate and rhythm.

C. Arterial Pressure Monitoring

1. Indirect. Blood pressure measurement should be performed at least hourly with a noninvasive blood pressure cuff or more often during vasoactive drip titration.

2. Direct. Intra-arterial catheters allow for the continuous measurement of arterial pressures and provide convenient access for frequent arterial bloodÑgas measurement, and blood draws for laboratory tests. They are utilized in patients with vasoactive medication requirements or a tenuous respiratory status. The most common site is the radial artery, chosen because of accessibility and collateral blood flow. Alternative sites are the axillary or brachial artery. The femoral artery can be used, though it is more inconvenient and usually only cannulated in an emergency. The extremity distal to the catheter should be assessed prior to and after insertion for ischemia.

D. Central Venous Pressure (CVP) Monitoring. Central venous catheters provide access to measure CVP, $SCvO_2$, and to administer vasoactive medications and TPN. CVP is often used as a surrogate for volume status and ventricular preload. Though many question its accuracy, there is probable utility in some situations such as large volume resuscitation or tamponade.

E. Pulmonary artery (PA) catheters determine CVP, cardiac output (CO), PA pressures, systemic vascular resistance (SVR), and mixed venous oxygen saturation (SvO_2). They can be used in unstable patients with rapid changes in hemodynamic status to assess responses to treatment with fluid

and cardioactive agents. Importantly, the use of PA catheters has not been demonstrated to improve mortality, in part, due to error in interpretation and variations in management (*JAMA.* 2005;294:1625). However, this has not been evaluated sufficiently through randomized controlled trials in surgical patients, specifically after cardiac surgery. Nonetheless, the ongoing popularity of PA catheters in cardiac surgery patients should be reconsidered in view of data from observational research suggesting lack of benefit (*Anesth Analg.* 2011;113:994). Furthermore, the hemodynamic measurements of the PA catheter are operator dependent (e.g., volume and rate of saline injection) and error prone, especially when there is tricuspid regurgitation or intracardiac shunts. The mixed venous oxygen saturation measurement is attractive as it requires no operator action and provides a useful indicator of adequacy of global oxygen delivery.

1. Complications. Prior to PA catheter placement a left-bundle-branch block must be ruled out because PA catheter placement can induce a transient **right-bundle-branch block.** Thus, PA catheter insertion with a left-bundle-branch block can cause life-threatening bradycardia. A **balloon rupture** results in the risk of air and balloon fragment emboli. Balloon rupture is confirmed by aspiration of blood from the balloon port. If either occurs, the catheter should be removed. **PA perforation** presents with hemoptysis, typically after balloon inflation. Management of this life-threatening complication requires positioning the patient

with the involved side in the dependent position and emergent thoracic surgical consultation. Other complications include malposition (e.g., coronary sinus), right ventricular rupture, and cardiac tamponade.

F. Esophageal Doppler and Pulse Contour Analysis have been introduced as less invasive alternatives to PA catheters for advanced hemodynamic monitoring. An esophageal Doppler measures descending aortic blood velocity and, based on assumptions regarding aortic diameter, calculates descending aortic blood flow, from which CO is extrapolated. Though it has shown utility, its utilization of the descending aorta for flow measurement assumes a constant and specific percentage of blood flow distributed to the heart, head, and upper extremities, an assumption which may often not be true. Pulse contour analysis uses an arterial pressure waveform to calculate the CO, with multiple systems available.

G. Respiratory Monitoring

1. Pulse oximetry should be used in all critically ill patients to provide a continuous assessment of arterial oxygen saturation (SaO_2). An elevated carboxyhemoglobin falsely raises the measurement and methemoglobinemia results in a persistent reading of 85%.

2. Capnography provides a quantitative, continuous assessment of expired CO_2 concentrations. A rise in $ETCO_2$ can indicate a decrease in alveolar ventilation or an increase in CO_2 production, as seen with sepsis and fever. An acute fall in $ETCO_2$ may indicate an increase in alveolar ventilation or an increase in dead space, as seen with massive pulmonary embolism (PE), endotracheal tube (ET), or mainstem bronchus obstruction.

H. Neurologic Monitoring

1. Intracranial pressure monitoring. See Chapter 10.

2. Processed electroencephalogram monitors (e.g., bispectral Index and SedLine) use proprietary algorithms to analyze the EEG waveform and provide a dimensionless number that is intended to indicate sedation or anesthetic depth. These monitors can be particularly useful in the ICU while patients are receiving neuromuscular blocking agents.

II. SEDATION AND ANALGESIA.

Sedation may be necessary for ICU patients who require invasive or poorly tolerated interventions. For example, sedation is frequently required to enable pressure-controlled ventilation. Pain should be treated to alleviate suffering and to promote deep breathing and early rehabilitation. However, both sedative and analgesic medications have side effects and should be given sparingly. Recent evidence suggests that outcomes are improved when sedation is decreased. This can often be achieved by using a sedation scale, such as the Richmond Agitation-Sedation Scale (RASS) (Table 7-1). The dose of the chosen agent is then titrated by the nurse to maintain the sedation goal.

A. Control of Agitation

1. Benzodiazepine's effects are mediated through γ-aminobutyric $acid_A$ ($GABA_A$) receptors. The two benzodiazepines commonly used for

sedation are midazolam and lorazepam. **Midazolam** is hepatically metabolized, but has renally cleared active metabolites, which may accumulate after prolonged infusions and take days to clear. **Lorazepam** has a slower onset and longer half-life. While it does not have active metabolites, it may still accumulate if utilized for a long duration. Benzodiazepines are associated with higher rates of delirium, especially in older patients (*Crit Care.* 2010;14:R38). Benzodiazepines are also indicated for alcohol or benzodiazepine withdrawal.

TABLE 7-1 Richmond Agitation-Sedation Scale (RASS)[a]

Score	Characteristics

+4	Combative: Danger to self or staff
+3	Very agitated: Aggressive, pulling at tubes
+2	Agitated: Frequent nonpurposeful movements
+1	Restless: Anxious, movements vigorous but not aggressive
0	Calm and alert
-1	Drowsy: In response to voice; eye contact sustained >10 sec
-2	Light sedation: In response to voice; eye contact sustained <10 sec
-3	Moderate sedation: In response to voice; movement but without eye contact
-4	Deep sedation: In response to physical stimulation; any movement
-5	Unarousable: No response to verbal or physical stimulation

[a]*JAMA.* 2003;289:2983.

2. Propofol is short acting and also acts through potentiation of $GABA_A$ receptors. There is a rapid onset due to its lipophilicity allowing it to cross the bloodÑbrain barrier quickly. It has no analgesic properties and it is typically used with an opioid in postoperative patients. Propofol induces significant hypotension due to myocardial depression, vasodilation, and increased venous capacitance. Respiratory depression and bradycardia are also common side effects (*Crit Care Med.* 2014;42:1696). Profound hypotension has been described when propofol is administered to patients receiving Rifampin (*Anesth Analg.* 2013;117:61). Prolonged use can lead to an elevation of triglycerides and subsequent pancreatitis. Although this is rare, triglyceride levels should be checked periodically. Propofol infusion syndrome is a rare but lethal complication, presenting with arrhythmia, rhabdomyolysis, and lactic acidosis (*Injury.* 2014;452:245). It should be suspected when patients receiving propofol have an unexplained metabolic acidosis.

3. Dexmedetomidine is a centrally acting α_2-adrenoreceptor agonist with sedative and analgesic properties. It is becoming increasingly popular as a sedative agent in the ICU as it might be associated with less delirium than other sedative agents, has analgesic properties, and does not depress respiration. Dexmedetomidine may be useful during a breathing trial to decrease anxiety while other anxiolytics are discontinued. As an agonist of the α_2-adrenoreceptor, similar to clonidine, it may induce hypotension and bradycardia (*Crit Care Med.* 2014;42:1696).

4. Ketamine is a dissociative anesthetic agent which activates the sympathetic nervous system and maintains mean arterial pressure and CO. For these reasons it may be used in patients with depressed cardiac function which may be present after cardiac surgery.

B. Management of Delirium. Delirium is a common manifestation of acute illness and is associated with increased mortality (*Crit Care.* 2010;14:R210). The cardinal features of delirium are waxing and waning inattention and disorganized thinking coupled with an acutely altered level of consciousness. Most patients have hypoactive delirium. No pharmacologic intervention has been shown to improve outcomes with delirium.

1. Antipsychotics. Haloperidol is an antipsychotic used to treat hyperactive delirium if the patient is self-destructive. Major toxicities include hypotension, prolongation of the QT interval, and extrapyramidal symptoms. **Quetiapine** is a second-

generation antipsychotic which might be used as alternative to haloperidol (*Crit Care Med.* 2010; 38:419).

2. Benzodiazepines can be life-saving for delirium tremens.

3. Dexmedetomidine. The role of dexmedetomidine for preventing delirium and for treating patients with hyperactive delirium is under investigation.

C. Control of Pain

1. Opioids control pain as CNS μ-receptor agonists. **Morphine** is the prototypical opioid and is commonly utilized for pain control. Morphine may not be the best opioid for critically ill patients as its active metabolite, morphine-6-glucuronide, is renally cleared. **Fentanyl** is commonly administered as a continuous infusion postoperatively. If given for prolonged periods (i.e., hours), fentanyl accumulates in tissue stores and has a prolonged duration of action. **Hydromorphone** is another popular option, with many favoring it for patients with renal impairment. **Meperidine,** while no longer typically used as an analgesic, is occasionally used for patients with postanesthetic shivering.

2. Nonsteroidal Anti-Inflammatory Drugs (NSAIDs) and Acetaminophen are commonly used adjuncts to narcotics. IV acetaminophen use has increased because it is well tolerated with a small side effect profile. It is a good alternative, or addition to, narcotics for surgical patients who are NPO. As with NSAIDs, its use decreases opioid use, though neither has been shown to reduce narcotic-induced adverse effects. NSAID use has been associated with an increased risk of postsurgical bleeding (*Br J Anaesth.* 2011;106:292). Further, NSAIDs are often avoided owing to concerns about nephrotoxicity.

3. Infusion of local anesthesia via thoracic or lumbar epidural catheters decreases the need for intravenous narcotics, and can improve compliance with respiratory therapy. Subcutaneous heparin should be held for at least 2 hours before and after removing the epidural catheter. One adverse effect is hypotension which can be managed by decreasing the infusion rate, removing the local anesthetic, giving volume, or by infusing a vasopressor.

D. The use of a **sedation protocol,** titrated by the bedside nurse, has been shown to decrease the number of days on mechanical ventilation and the ICU length of stay (*Crit Care Med.* 1999;27:2609). For patients who require long-term sedation and analgesia, a daily interruption of sedation to wakefulness produces decreased time on mechanical ventilation and shorter ICU stays (*N Engl J Med.* 2000;342:1477).

III. RESPIRATORY FAILURE

A. Etiology. Respiratory failure results from inadequate gas exchange caused by ventilation/perfusion (V/Q) mismatch, hypoventilation, or impaired systemic delivery/extraction. Dead space ventilation refers to airflow within the lung that does not equilibrate with bloodÑgas content; this occurs in chronic obstructive pulmonary disease and PE. In contrast, intrapulmonary shunts result from perfusion of lung tissue that is poorly ventilated, such as severe pulmonary edema, acute respiratory distress syndrome (ARDS),

or pneumonia. Hypoventilatory hypoxemia may be caused by a failure of mechanical ventilation, which results in hypercapnia and hypoxemia.

B. Diagnosis. Signs or symptoms of respiratory distress should prompt pulse oximetry and an ABG. Oxygen saturation readings less than 90% can be reflective of impaired tissue oxygenation. An acute rise in $PaCO_2$ accompanied by a decrease in pH (respiratory acidosis) implies a significant imbalance between carbon dioxide production and elimination. **It is important to note that adequate oxygenation does not guarantee adequate ventilation.** In addition to a physical examination and consideration of the patient's recent history, a chest x-ray, EKG, and chest CT with PE protocol should be considered.

C. Treatment. The urgency of the situation may necessitate management prior to a diagnosis. Treatment may consist of inhaled oxygen, noninvasive positive pressure ventilation (NIPPV), endotracheal intubation, chest tube placement, or extracorporeal membrane oxygenation (ECMO).

1. Oxygen therapy. Supplemental oxygen can be administered to increase the alveolar oxygen concentration. At increasing concentrations of oxygen delivery, these methods include nasal cannula, simple face mask, or face mask with a reservoir (Table 7-2).

TABLE 7-2 Oxygen Delivery Systems

Type	FiO_2 Capability	Comments
Nasal cannula	24-48%	Flow rates of 1-8 L/min; true FiO_2 uncertain and highly dependent on minute ventilation; simple, comfortable, and can be worn during eating or coughing
Simple face mask	35-55%	Flow rates of 6-10 L/min
High-humidity mask	Variable from 28% to nearly 100%	Flow rates should be 2-3 times minute ventilation; levels >60% may require additional oxygen bleed-in
Nonrebreather	90-95%	Flow rates of 12-15 L/min; incorporates valve to reduce room air entrainment and rebreathing of expired air
Ventimask	24%, 28%, 31%, 35%, 40%, or 50%	Provides controlled FiO_2; useful in chronic obstructive pulmonary disease patients to prevent depression of respiratory drive; poorly humidified gas at maximum FiO_2

FiO_2, fraction of inspired oxygen.

2. Airway management. If uncertainty exists about whether the airway is patent or protected from aspiration, ET intubation is indicated. **Unless the physician is skilled in artificial airway placement, bag-mask ventilation should be performed until an expert arrives.**

a. ET intubation. Once placed, the adequacy of ventilation is confirmed with a CO_2 indicator and bilateral auscultation. A chest x-ray confirms correct ET tube position, midway between the clavicles and carina.

b. NIPPV. Biphasic positive airway pressure (BiPAP) is a form of ventilation that is delivered by a tight-fitting mask over the mouth and nose. It is useful in patients with mild-to-moderate respiratory insufficiency of short duration and may prevent intubation in patients with rapidly reversible respiratory failure. BiPAP may result in gastric distension, increasing the risk of aspiration.

c. Tracheostomy should be considered urgently in the presence of severe maxillofacial injury. It should also be considered electively if prolonged intubation is anticipated. Tracheostomy provides a more secure airway, improves patient comfort and oral hygiene, increases patient mobility, and enhances secretion removal. In one study, the placement of an early tracheostomy decreased the duration of mechanical ventilation and the length of ICU and hospital stay (*Crit Care Med.* 2005;33:2513) while a more recent trial showed no difference in survival or ICU length of stay for early over late tracheostomy *(JAMA.* 2013;309:2121). Timing of tracheostomy placement remains controversial. **If a tracheostomy is inadvertently removed prior to the development of an adequate tract, approximately 2 weeks, an orotracheal tube should be placed rather than blind attempts at tracheostomy replacement.** If orotracheal intubation is not possible, a bronchoscope can aid in tracheostomy reinsertion. If the tracheostomy tube is inadvertently placed in the pretracheal tissue, the subsequent attempts at ventilation will create subcutaneous emphysema which can make tracheal intubation more difficult.

d. Cricothyroidotomy is utilized to obtain an airway in emergent situations when attempts to ventilate by facemask or laryngeal mask airway (LMA), and ET tube placement are unsuccessful.

3. Modes of mechanical ventilation are divided into volume- or pressure-controlled modes. Volume-controlled modes deliver a set tidal volume ensuring adequate alveolar ventilation; airway pressure varies depending on compliance. Pressure-controlled modes deliver a set airway pressure; tidal volume varies depending on compliance (the change in volume divided by the change in pressure, $\Delta V/\Delta P$).

a. Volume-controlled modes

(1) Intermittent mandatory ventilation (IMV) delivers a preset tidal volume over a set time, and the pressure is varied. The ventilator is synchronized to assist with any patient-initiated breaths up to a set rate, but any additional breaths initiated by the patient will be unassisted. If the patient does not initiate enough breaths to fulfill the set respiratory rate, the ventilator

will initiate additional breaths. This is well tolerated with minimal sedation.

(2) Assist-control (A/C) ventilation delivers a preset tidal volume at a set rate over a set time. As the machine senses each inspiratory effort by the patient, it delivers the set tidal volume over a set time. If the patient's respiratory rate is below the machine's set rate, ventilator-initiated breaths are delivered to make up the difference. A/C ventilation minimizes the work of breathing because the ventilator assists all breaths; however, this mode is uncomfortable in the minimally sedated patient if the breaths are dyssynchronous.

b. Pressure-controlled modes

(1) Pressure-support ventilation (PSV) delivers a preset inspiratory pressure but at no set rate. Constant inspiratory pressure continues until the inspiratory flow falls below a predetermined level and the exhalation valve opens, delivering tidal volumes only when the patient is breathing spontaneously. Therefore, this mode is for spontaneously breathing patients. It does increase the amount of work necessary because each breath is patient initiated and the duration of pressure support is also dependent on ongoing patient effort. As such PSV is effort cycled. Low pressures (5 to 8 cm H_2O) are set routinely to overcome the resistance caused by the ET tube and the inspiratory demand valves. This mode is often utilized to evaluate for extubation.

(2) Pressure-controlled ventilation (PCV) delivers a preset inspiratory pressure at a set rate with each breath delivered over a set time. As such, PCV is time cycled. This mode allows the physician to set the airway pressure and minimize barotrauma. The disadvantage is that the tidal volume varies depending on compliance and any increase in airway resistance can decrease the tidal volume to dangerously low levels. This mode is used in patients with poor lung compliance which requires a higher pressure.

c. Advanced Ventilator Settings. For patients in whom conventional mechanical ventilation fails to achieve adequate oxygenation, open lung ventilation may be considered. It minimizes shearing forces due to alveolar collapse by stenting alveoli open at end expiration.

(1) Airway pressure release ventilation (BiLevel) is pressure support with an inverse I:E ratio. It increases the mean airway pressure without increasing the peak. It is managed with four variables, a time at a high pressure (T_{high}) and lower pressure (T_{low}) and the pressure high (P_{high}) and low (P_{low}). It is set to have a higher T_{high} to recruit alveoli with ventilation occurring with spontaneous breaths over the P_{high} and during the pressure release to P_{low}.

(2) High-frequency oscillatory ventilation (HFOV) uses higher rates (180 to 300/minute) and smaller tidal volumes than conventional modes. Adjustable variables include oscillatory

frequency (Hz), FiO_2, amplitude or power (tidal volume), and inspiratory time. When utilizing HFOV, often as the final option on the ventilator algorithm, the patient must be deeply sedated and paralyzed.

4. Ventilator management

a. FiO_2 should be adjusted to ensure adequate oxygenation with the lowest possible FiO_2 to prevent pulmonary oxygen toxicity.

b. Tidal volume. It has been shown that a lung-protective strategy, during an abdominal operation, with lower volume ventilation may improve clinical outcomes (*N Engl J Med.* 2013;369:428), whereas a strategy to prevent atelectasis with a high

PEEP leads to higher vasoactive drug requirements during the operation without reducing postoperative complications (*Lancet.* 2014;384:495). With ARDS, a randomized trial demonstrated improved survival in patients who were ventilated with low tidal volumes (6 mL/kg ideal body weight) compared with high tidal volumes (12 mL/kg) (*N Engl J Med.* 2000;342: 1301). As a result, the tidal volume should be decreased to maintain plateau pressures <30 cm H_2O and to minimize barotrauma but >20 cm H_2O to minimize atelectasis.

c. Ventilatory rate. Once the tidal volume has been determined, the rate is chosen to provide adequate minute ventilation and adjusted to optimize arterial pH and $PaCO_2$.

d. Inspiratory-expiratory (I:E) ratio. The normal I:E ratio is 1:2 to 1:3. Longer expiratory times allow patients with obstructive lung disease to exhale fully and prevent breath stacking. 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. This improves gas exchange by progressive alveolar recruitment with a higher mean airway pressure. It is used most commonly with PCV.

e. PEEP increases functional residual capacity and improves V/Q matching by opening terminal airways and recruiting alveoli. A PEEP of 5 cm H_2O is considered minimal; higher levels are used with hypoxemia. PEEP levels >15 cm H_2O significantly increase the risk of barotrauma and spontaneous pneumothorax. **Continuous positive airway pressure (CPAP)** is PEEP applied to the spontaneously ventilating patient without additional inspiratory support.

f. Sedation and neuromuscular paralysis is often necessary in mechanically ventilated patients to control anxiety, allow for rest, and synchronize breathing. However, a recent clinical trial demonstrated that patients who receive no sedation have more ventilator-free days and shorter ICU lengths of stay (*Lancet.* 2010;375:475). The minimum sedation necessary should be used. The need for paralysis is rare, except in patients with severe respiratory failure and decreased pulmonary compliance. An evaluation of patients with severe ARDS found that a continuous infusion of cisatracurium was associated with improved survival (*N Engl J Med.* 2010;363:1107). If paralytics are necessary, they should be discontinued as soon as

possible and the patient must be adequately sedated. The extent of paralysis should routinely be assessed with neuromuscular monitoring, and adequacy of anesthesia should be ensured with a processed electroencephalogram.

g. Prone positioning is a rescue strategy for patients with severe ARDS. Patients are placed in a prone position for a scheduled period of time daily; theoretical benefits include recruitment of dorsal lung units, improved mechanics, decreased V/Q mismatch, and increased secretion drainage (*JAMA.* 2005;294:2889). Recently, the PROSEVA study demonstrated a survival advantage with early use of prolonged prone positioning for patients with severe ARDS (*N Engl J Med.* 2013;368:2159).

h. Weaning from mechanical ventilation. The patient who has required prolonged ventilatory support may require several days to weeks to wean because of marginal respiratory muscle strength and the time required for lung recovery. In general, hemodynamic instability or high work of breathing are contraindications to weaning. Reduction in the FiO_2 to 0.40 and PEEP to 5 cm H_2O is accomplished first. At Washington University in Saint Louis, patients receive daily PSV trials to assess suitability for extubation. A common tool is the rapid shallow breathing index (RSBI), the respiratory rate/tidal volume, with a value >105 suggesting that discontinuation of assisted ventilation is unlikely to succeed.

5. Complications

a. ET tube dislodgment and patient self-extubation can become an emergency. For this reason, restraint of the patient's upper extremities is frequently required.

b. ET tube cuff leaks lead to a decreased airway pressure and return of expired volume. It may indicate that the ET tube needs to be advanced.

c. Respiratory distress may occur during mechanical ventilation due to an acute change in the patient's status or to ventilator malfunction. The first priority is to switch to bag ventilation using 100% oxygen to ensure adequate ventilation and oxygenation. Increased airway pressures may indicate obstruction of the tube with secretions, a kink, bronchospasm, pneumothorax, inadequate sedation, or migration of the ET tube into a mainstem bronchus. Check the ET tube for patency; 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 reintubate the patient. Listen closely for any change in breath sounds consistent with a pneumothorax, new lung consolidation, or pleural

fluid collection. A less common but important cause of respiratory distress is PE. The results of an ABG and a chest x-ray are frequently helpful. In addition, a CT or bronchoscopy can be performed.

d. Barotrauma from high peak airway pressures can lead to subcutaneous emphysema, pneumomediastinum, and pneumothorax. A pneumothorax that develops while on positive-pressure ventilation

is at risk for becoming a tension pneumothorax and is treated with a tube thoracostomy.

e. Oxygen toxicity refers to levels of intra-alveolar oxygen high enough to cause lung damage. The precise mechanism is unknown, but likely involves oxidation of cell membranes due to oxygen radicals. FiO_2 should be weaned as soon as possible.

f. Tracheoinnominate fistula is caused by erosion of a tracheostomy tube into the innominate artery. It leads to accumulation of blood within the airway and hemorrhage. Emergent treatment consists of insertion of a finger into the tracheostomy and applying ventral pressure to compress the artery. Orotracheal intubation should be performed and a thoracic surgical consult obtained. This complication can be minimized by maintaining a cuff pressure <25 mm Hg.

6. ECMO may be the only remaining option if all other modes fail. It can be utilized as either veno-venous (VV) for pulmonary support or as veno-arterial (VA) for pulmonary and cardiac support. This method of support, with its necessary anticoagulation, is fraught with complications. In the critical care setting, it may be necessary as temporary support for a patient with reversible myocardial damage after surgery (Chapter 30). Another potential indication is VV-ECMO for ARDS, with a survival to discharge of 63% (*Lancet.* 2009;374:1351).

IV. CIRCULATORY FAILURE: SHOCK

A. Shock is defined by global tissue hypoxia and occurs when the supply of oxygen is insufficient to meet metabolic demands.

B. Classification and Recognition of Shock. Early recognition and prompt intervention is critical (Table 7-3). Note that some of these values require a PA catheter, which is frequently unnecessary. The patient's recent history, laboratory values, and physical examination are usually sufficient for determining the etiology.

1. Hypovolemic shock results from loss of circulating blood volume caused by acute hemorrhage, fluid depletion, or dehydration. Patients are peripherally vasoconstricted, tachycardic, and have low jugular venous pressure.

2. Distributive shock is a hyperdynamic state consisting of tachycardia, vasodilation, decreased SVR, and increased CO. The most common causes include sepsis, neurogenic shock, adrenal insufficiency, and liver failure. **Neurogenic shock** results from interruption of the spinal cord at or above the thoracolumbar sympathetic nerve roots, which produces loss of sympathetic tone, causing vasodilation. Patients are peripherally vasodilated and tachycardic. Jugular venous pressure is low.

3. Obstructive shock results from etiologies that prevent adequate CO but are not intrinsically cardiac in origin. This may be caused by PE, tension pneumothorax, or cardiac tamponade. Jugular venous pressure is elevated while the peripheral tissues demonstrate vasoconstriction.

TABLE 7-3 Clinical Parameters in Shock

Shock Classification	Skin	Jugular Venous Distention	Cardiac Output	Pulmonary Capillary Wedge Pressure	Systemic Vascular Resistance	Mixed Venous Oxygen Content
Hypovolemic	Cool, pale	↓	↓	↓	↑	↓
Cardiogenic	Cool,	↑	↓	↑	↑	↓

	pale					
Septic						
Early	Warm, pink	↑↓	↑	↓	↓	↑
Late	Cool, pale	↓	↓	↓	↑	↑↓
Neurogenic	Warm, pink	↓	↓	↓	↓	↓

4. Cardiogenic shock results from inadequate CO due to intrinsic cardiac failure. Diagnosis may require echocardiography. These patients typically are peripherally vasoconstricted and tachycardic with an elevated jugular venous pressure.

C. Interventions Common to All Types of Shock. The goal of therapy is to ensure adequate oxygen delivery. Because oxygen delivery is proportional to SaO_2, hemoglobin concentration, and CO, each should be optimized.

1. SaO_2. Supplemental oxygen should be administered or an airway placed to achieve a SaO_2 >92%.

2. Hemoglobin concentration. For most critically ill patients, a transfusion trigger of 7 g/dL is appropriate, except with an ongoing myocardial infarction or severe ischemic cardiomyopathy (*N Engl J Med.* 1999;340:409).

3. CO. A continuous cardiac monitor provides the heart rate and indirect clues about stroke volume. The atrial contraction provides approximately 15% to 25% of preload; therefore, atrial fibrillation can significantly reduce CO. Other tachyarrhythmias decrease diastolic ventricular filling and may reduce CO despite the elevated HR, typically occurring at a HR >140. With the exception of the patient in pulmonary edema, patients in circulatory shock should initially receive 10 to 20 mL/kg bolus of a crystalloid solution.

4. Palpable pedal pulses and urine output exceeding 1 mL/kg/hour indicate good CO. A metabolic acidosis can reflect 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 7.15 because it may worsen intracellular pH as it is converted to CO_2 at the tissue level.

D. Specific Therapy

1. Hypovolemic shock. Therapy focuses on control of ongoing loss and restoration of intravascular volume. Patients with blood losses of up to 20% can be resuscitated using crystalloid solutions. However, because salt solutions equilibrate with the interstitial space, volume replacement 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 transfusion. To achieve rapid infusion rates, short, large-bore intravenous catheters in a peripheral vein are best. If this is not possible, an 8.5-French sheath in a central vein is highly effective. In addition, if intravenous attempts are unsuccessful, intraosseous access can be quickly obtained. **A multilumen central line is not effective for rapid volume resuscitation since resistance to flow is proportional to catheter length and inversely proportional to catheter lumen radius raised to the fourth power.** Hypothermia is aggravated by rapid infusion of room temperature crystalloid and refrigerated blood, impairing oxygen unloading and compromising coagulation; therefore fluids and blood products should be warmed. With adequate volume resuscitation, vasoactive agents can usually be avoided.

2. Distributive shock

a. Septic shock (see Section V.C).

b. Systemic Inflammatory Response Syndrome (SIRS) may result from noninfectious causes of inflammation. Treatment

is supportive until the inflammatory process resolves.

c. Critical illness-related corticosteroid insufficiency (CIRCI) can result from adrenal insufficiency or glucocorticoid resistance. The diagnosis and treatment of adrenal insufficiency in septic shock are evolving. The use of corticosteroids is not without risk as it does increase the risk of infection (*N Engl J Med.* 2008;358:111). Per ACCM 2008 guidelines, adrenal insufficiency is best diagnosed by an increase in cortisol level of <9 µg/dL after a cosyntropin stimulation test or random total cortisol level <10 µg/dL. These guidelines state that patients with primary adrenal insufficiency or those with septic shock refractory to fluid resuscitation and vasopressors, without performing a cosyntropin stimulation test, should be treated with moderate-dose hydrocortisone due to a faster resolution of shock seen in multiple studies and a survival advantage (*Crit Care Med.* 2008;36:1937). Corticosteroids in the critically ill remains a topic of controversy. A recent meta-analysis evaluating the use of steroids for septic shock did not show a survival advantage, though again, there was faster resolution of shock (*Anesth Analg.* 2014;118:346). Future studies will need to clarify the utility of corticosteroids and any subgroups where they are beneficial.

d. Neurogenic shock. The initial intervention is volume infusion. A peripheral vasoconstrictor, phenylephrine or norepinephrine, is administered to increase vascular tone if hypotension is refractory to volume infusion. Dopamine is used in patients with neurogenic shock and bradycardia. Because patients with spinal shock tend to equilibrate body temperature with their environment, fluids and room temperature must be kept warm.

3. Obstructive shock. Tension pneumothorax is treated by needle decompression followed by tube thoracostomy. Pericardial tamponade is treated by needle decompression, often with catheter placement for drainage. The treatment of a PE varies based on the degree of hemodynamic compromise. Options include systemic anticoagulation, thrombolysis, and surgical clot removal. IVC filters are used in patients with a contraindication to anticoagulation or with progression of thrombus on therapeutic anticoagulation.

4. Cardiogenic shock. Management is directed toward maintaining adequate myocardial perfusion and CO with volume expansion and vasoactive medications (Table 7-4). Initial treatment is often guided by CVP measurements or PA catheter data, while the precipitating cause is identified and treated. CVP in this setting is useful for assessment of RV function and not as a marker of volume status (*Chest.* 2008;134:172). Intra-aortic balloon counterpulsation may be necessary before and during recovery from definitive surgical treatment. If perfusion remains inadequate, the only remaining option is mechanical circulatory support.

V. SEPSIS

A. Definition. Sepsis is defined as SIRS with a documented or presumed infection. The clinical definition of SIRS requires two of the following: Body temperature >38¡C or <36¡C, heart rate >90 beats/minute, respiratory rate >20/minute or $Paco_2$ <32, and WBC count >12 or <4 or >10% bands. Severe sepsis is multiple-organ dysfunction or hypoperfusion (septic shock) resulting from infection.

B. Diagnosis

1. Cultures should be obtained as part of the initial evaluation, at least one of which should be drawn percutaneously, prior to the initiation of antibiotics. This will allow a more specific antibiotic regimen once susceptibilities return.

C. Treatment

1. Infection

a. Antibiotic therapy

(1) Broad-spectrum intravenous antibiotics should be initiated within the first hour (*Chest.* 2000;118:146). The use of antifungal therapies and agents directed at highly resistant Gram-negative rods, methicillin-resistant *Staphylococcus aureus,* vancomycin-resistant enterococcus, and resistant pneumococcus should be guided by the clinical situation and local susceptibility patterns.

TABLE 7-4 Inotropes and Vasoactive Drugs and Their Specific Actions

Class and Drug	Blood Pressure	Systemic Vascular Resistance	Cardiac Output	Heart Rate	Inotrope: Low Dose	Inotrope: High Dose	Renal Blood Flow	Coronary Blood Flow	Mvo_2
Alpha Only									
Phenylephrine	↑↑	↑↑↑↑	↓	↓	±	±	↓↓↓↓	±↑↑	↑
Alpha and Beta									
Norepinephrine	↑↑↑	↑↑↑↑	↑↑↑	↑ ±	↑	↑	↓↓↓↓	↑↑	↑↑
Epinephrine	↑↑↑	↑↑↑↑	↑↑↑↑	↑↑↑	↑↑	↑↑↑	↓±	↑↑	↑↑↑
Dopamine	↑↑	↑↑	↑↑↑	↑↑	±	↑↑	↑↑↑	↑↑	↑↑
Beta Only									
Dobutamine	±	↓↓↓	↑↑↑↑	↑↑	↑↑↑	↑↑↑	±	↑↑↑	↑↑↑
Beta-blocker									
Metoprolol	↓	↓	↓↓	↓↓↓	↓↓	↓↓↓	±	↓↓	↓↓
Other									
Nitroglycerine	± ↓	↓↓	↑↑	±	±	±	± ↑	↓	↓↓
Hydralazine	↓↓	↓↓↓	↑↑	↑↑	±	±	± ↑	↓	↓↓
Nitroprusside	↓↓↓	↓↓↓	↓↓↓	± ↑	±	±	↑↑	±	↓↓

Mvo_2, mixed venous oxygen saturation.

(a) The following increase risk for infection with resistant organisms:

(i) Prior antibiotic treatment.

(ii) Prolonged hospitalization.

(iii) Presence of invasive devices.

(2) For a hospitalized patient who becomes septic with a presumed pneumonia, a common initial broad-spectrum regimen consists of vancomycin and cefepime. For intra-abdominal infections, therapies commonly start with vancomycin and piperacillin/tazobactam with the possible addition of an antifungal.

b. Source control, drainage, debridement, or removal of the infectious source, is imperative.

2. Circulatory support

a. Volume resuscitation. Early goal-directed therapy consists of volume resuscitation for a CVP of 8 to 12, MAP >65, UOP >0.5 mL/kg/hour, and mixed SvO_2 >70% (*N Engl J Med.* 2001;345:1368). This has been practiced for septic patients for a decade; however, a recent randomized trial showed no difference in survival with this protocol over standard medical judgment (*N Engl J Med.* 2014;370:1683). Volume resuscitation remains a cornerstone, but can be used more judiciously.

b. Vasoactive medications. Septic patients who fail to achieve rapid hemodynamic stability with fluids are started on a vasoactive medication. Most practitioners favor norepinephrine for its vasoconstrictive properties as well as its ability to increase CO. Dopamine is still used by some, but it is associated with higher rate of dysrhythmias (*N Engl J Med.* 2010;362:779; *Shock.* 2010;33:375). The addition of low-dose vasopressin increases MAP, SVR, and urine output in septic patients who are hyporesponsive to catecholamines. This may spare patients from high-dose norepinephrine, although its impact on survival is unclear.

VI. UPPER GASTROINTESTINAL HEMORRHAGE PROPHYLAXIS.

Patients in the ICU are at increased risk for stress-induced mucosal ulceration and GI hemorrhage. Risk factors include head injury (Cushing ulcers); burns (Curling ulcers); prolonged mechanical ventilation; history of peptic ulcer disease; NSAIDs or steroids; and the presence of shock, renal failure, portal hypertension, or coagulopathy. An H_2-receptor antagonist should be used to maintain mucosal integrity in these patients. Proton-pump inhibitors are preferred by some, and they should be used in patients who bleed despite H_2-receptor antagonists. The use of stress ulcer prophylaxis for a patient without an above-listed risk factor should be avoided due to an increased risk of *Clostridium difficile*-associated diarrhea (*J Crit Care.* 2014;696:e11).

VII. ANEMIA.

The prospective Transfusion Requirements in Critical Care (TRICC) trial reported that **transfusing all patients to a hemoglobin of**

10 mg/dL either has no effect or may actually decrease survival in the critically ill (*N Engl J Med.* 1999;340:409). A restrictive transfusion strategy (hemoglobin <7 mg/dL) is recommended in critically ill patients; except in those with acute coronary syndrome, severe hypoxemia, or active hemorrhage. A randomized controlled trial demonstrated that administration of recombinant erythropoietin did not reduce the rate of transfusion or reduce mortality in critically ill patients, but increased the risk of thrombotic events (*N Engl J Med.* 2007;357:965).

VIII. BLOOD GLUCOSE CONTROL.

A study of randomly assigned surgical patients to tight glucose control (blood glucose goal: 80 to 110 mg/dL) versus conventional control (blood glucose goal: 180 to 200 mg/dL) showed nearly a twofold decrease in mortality in the tight-glucose-control group and reduced in-hospital mortality, bloodstream infections, acute renal failure, number of red cell transfusions, and critical illness polyneuropathy (*N Engl J Med.* 2001;345:1359). However, a follow-up study in medical patients did not demonstrate such a benefit (*N Engl J Med.* 2006;354:449). Hypoglycemia remains a major risk of tight glucose control and has been associated with increased mortality. A goal blood sugar of less than 140 mg/dL seems safe and beneficial; further studies are required to determine the response of different patient populations to varying intensities of insulin.

IX. MEDICATIONS.

Commonly used drugs and doses (Table 7-5).

TABLE 7-5 Drugs Commonly Used in the Intensive Care Unit

Drug	Dilution (Concentration)	Loading Dose	Dose	Comments
Diltiazem	125 mg/125 mL 0.9% NaCl or D5W (1 mg/mL)	0.25 mg/kg (followed by 0.35 mg/kg if needed)	5-15 mg/hr	May cause hypotension
Dobutamine	250 mg/100 mL 0.9% NaCl (2,500 µg/mL)		2-20 µg/kg/min	Selective inotropic (beta) effect; may cause tachycardia and arrhythmias
Dopamine	400 mg/250 mL 0.9% NaCl or D5W (1,600 µg/mL)		Dopa, 1-3 µg/kg/min; alpha, 3-10 µg/kg/min; beta, 10-20 µg/kg/min	Clinical response is dose and patient dependent; may cause arrhythmias and tachycardia
Epinephrine	5 mg/500 mL 0.9% NaCl or D5W, or 4 mg/100 mL 0.9% NaCl or D5W		0.01-0.05 µg/kg/min	Mixed alpha and beta effects; use central line; may cause tachycardia and hypotension
Esmolol	2.5 g/250 mL 0.9% NaCl or D5W (10 mg/mL)	500 µg/kg/min for 1 min (optional)	50-300 µg/kg/min	Selective $beta_1$-blocker; $T_{1/2}$ 9 min; not eliminated by hepatic or renal routes; may cause hypotension
Heparin	25,000 units/250 mL 0.45% NaCl (100 units/mL)	60 units/kg	14 units/kg/hr	Obtain PTT every 4-6 hr until PTT is 1.5-2 times control; may cause thrombocytopenia
Lidocaine	2 g/500 mL D5W (4 mg/mL)	1 mg/kg (can repeat two times if needed)	1-4 mg/min	Dose should be decreased in patients with hepatic failure, acute MI, CHF, or shock
Nitroglycerin	50 mg/250 mL D5W (200 µg/mL)		5-20 µg/min	Use cautiously in right-sided MI
Nitroprus-side	50 mg/250 mL D5W (200 µg/mL)		0.25-10 µg/kg/min	Signs of toxicity include metabolic acidosis, tremors, seizures, and coma; thiocyanate may accumulate in renal failure

Norepinephrine	8 mg/500 mL D5W (16 µg/mL)	0.01-0.1 µg/kg/min	Potent alpha effects; mainly $beta_1$ effects at lower doses; use central line
Phenylephrine	10 mg/250 mL 0.9% NaCl or D5W (40 µg/mL)	10-100 µg/min	Pure alpha effects; use central line; may cause reflex bradycardia and decreased cardiac output
Vasopressin	20 units/100 mL NS (0.2 units/mL)	0.04 units/min	Do not titrate; higher doses may cause myocardial ischemia.

CHF, congestive heart failure; D5W, 5% dextrose in water; max, maximum; MI, myocardial infarction; PTT, partial thromboplastin time; $T_{1/2}$, terminal half-life.

CHAPTER 7: CRITICAL CARE

Multiple Choice Questions

1. A patient develops significant hemoptysis and shortness of breath minutes after insertion of a pulmonary artery catheter into a branch of the left pulmonary artery and measurement of the wedge pressure. Management should be:

a. Emergent tracheostomy.

b. Thrombolytics followed by systemic anticoagulation or IVC filter placement.

c. Changing to pressure-controlled ventilation with a reverse I:E ratio.

d. Placing the patient with the side of the PA catheter in left lateral decubitus position and urgent thoracic surgery consult.

e. Placing the patient with the side of the PA catheter in right lateral decubitus position and urgent thoracic surgery consult.

View Answer

2. Concerning the sedated patient for mechanical ventilation:

a. Sedation should be deep in order to minimize any discomfort when there is no chance of extubation.

b. The chosen method of sedation and goal level of sedation should be communicated to the bedside nurse who will titrate dosage.

c. For patients receiving neuromuscular blockade, a BIS of <90 is considered sufficient.

d. Due to a lack of analgesic properties, propofol often leads to hypertension.

e. Ketamine should be avoided in patients who have depressed cardiac function.

View Answer

3. A 72-year-old male has been admitted to the surgical ICU for 16 days after surgical repair of a spontaneous duodenal perforation due to steroids for his SLE. He developed pneumonia and has required mechanical ventilation since his operation. He underwent tracheostomy placement on POD 6. Yesterday he had a small amount of blood from his tracheostomy which stopped spontaneously. He now develops significantly more hemoptysis through his tracheostomy and his respiratory status is rapidly decompensating. Your next step should be to:

a. Remove the tracheostomy, place your finger through the tracheostomy site, and apply pressure to the innominate artery. The patient should be intubated.

b. Urgent CT to evaluate for potential PE followed by systemic anticoagulation or thoracic surgical consult for emergent thrombectomy.

c. Urgent ENT consult for bleeding likely from the nasopharynx.

d. Tube thoracostomy placement.

e. Transfuse 2 units of PRBCs through a level I infuser into a large peripheral IV followed by FFP and platelets as with a massive transfusion protocol.

View Answer

4. The ventilator mode airway pressure release ventilation (APRV) or BiLevel:

a. Increases the peak airway pressure to open alveoli.

b. Provides additional time for ventilation to eliminate CO_2.

c. Usually utilizes an I:E of 1:2.

d. Increases the mean airway pressure without increasing the peak.

e. Requires a paralyzed patient.

View Answer

5. A tracheostomy is placed for a patient who is anticipated to have a prolonged ventilatory course. He does better than anticipated and is weaned from the ventilator to tracheostomy collar 2 days after placement. He is in bed and during a roll his tracheostomy collar gets caught and pulls his tracheostomy tube out. His respiratory status declines quickly. The next step is:

a. Bag-mask ventilation over the tracheostomy site.

b. Replacement of the tracheostomy tube and bag-mask ventilation until an appropriate oxygen saturation is reached.

c. Intubation from above with appropriate sedation.

d. Bronchoscopy to clear any mucus plugs.

e. Blocking the tracheostomy site to prevent air leakage so the patient can breathe normally.

View Answer

6. A patient who comes into the emergency department with unknown history is hypotensive. Initial physical examination findings are increased jugular venous distention and cool skin. After PA catheter placement you see decreased cardiac output with increased wedge pressure and increased systemic vascular resistance. This patient is most likely suffering from:

a. Hypovolemic shock.

b. Neurogenic shock.

c. Late septic shock.

d. Early septic shock.

e. Cardiogenic shock.

View Answer

7. Steroid administration for septic shock:

a. Should be given if the cosyntropin stimulation test has a δ >9 µg/dL.

b. Should not be given if the random total cortisol level is <10 µg/dL.

c. Has been shown to decrease duration of sepsis and improve survival in all studies.

d. Should be high-dose dexamethasone.

e. Should be given to patients who do not respond to volume and vasoactive medications without evaluation of cortisol level.

View Answer

8. A patient on a mechanical ventilator for ARDS has required increasing pressure for oxygenation. He suddenly develops respiratory distress with desaturation, tachycardia, and hypotension. Initially you increase the FiO_2 and the pressure, but the situation continues to deteriorate. On physical examination you note severely diminished breath sounds on the right and you notice the CVP is much higher. Your next step is:

a. Bronchoscopy to remove a mucus plug.

b. Needle decompression followed by tube thoracostomy.

c. Volume resuscitation and more advanced ventilator settings.

d. Chest CT scan to evaluate for PE.

e. Decompressive laparotomy for abdominal compartment syndrome.

View Answer

9. Stress ulcer prophylaxis:

a. Should be administered only in patients with risk factors admitted to the ICU.

b. Should be administered to all patients admitted to the ICU.

c. Should be given to patients who have an NG tube.

d. Does not increase the risk of *C. difficile* infection.

e. Is not necessary in patients receiving corticosteroids.

View Answer

10. For which of the following patients, currently in the ICU, is blood transfusion indicated?

a. A 26-year-old male admitted after a motorcycle accident with femur fracture s/p ORIF POD 2 with a hemoglobin of 7.4 mg/dL with low UOP.

b. A 76-year-old patient with ESRD who underwent brachiocephalic graft placement complicated by a postoperative pneumonia with a hemoglobin of 8.2.

c. An 84-year-old male POD 2 after a femoral-popliteal bypass complicated by postoperative NSTEMI and a hemoglobin of 7.9 mg/dL.

d. A 56-year-old male after left hemicolectomy with the intraoperative course complicated by significant blood loss with a hemoglobin of 8.6 mg/dL and a small norepinephrine requirement.

e. A 94-year-old female admitted after hepaticojejunal bypass for a mass obstructing the duodenum with a hemoglobin of 8.2 mg/dL.

View Answer

8
Burns

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Burns result from thermal injury to the skin. They compromise the skin's function as a barrier to injury and infection and as a regulator of body temperature and fluid loss. Like trauma, mortality from burns occurs in a bimodal pattern: Immediately after the injury or weeks later from sepsis and multiorgan failure. Most burns are preventable and, thus, prevention strategies are of utmost importance. Detailed guidelines for the management of burns may also be found in the American Burn Association Consensus Statements (*J Burn Care Res.* 2013;34;4).

ASSESSMENT AND MANAGEMENT OF BURN INJURIES

I. ASSESSMENT

(Fig. 8-1)

A. Mechanism of Injury. Identify burn source, duration of exposure, time of injury, and environment. 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 eardrums and lungs and may also cause blunt trauma.

B. Primary survey should follow the guidelines established by the American College of Surgeons' Advanced Trauma Life Support Course. Burned patients should be evaluated and treated as victims of multisystem trauma because there is significant morbidity associated from missed injuries secondary to an explosion, falls, and so forth.

1. Airway assessment and security are the foremost 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, and presence of carbon in the oropharynx. The decision to intubate the trachea for airway protection should be made early and is preferable to cricothyroidotomy in the edematous and swollen neck. Awake intubation or intubation over a bronchoscope is the safest approach if there is any question about the ease or adequacy of airway exposure (*Curr Opin Anaesthesiol.* 2003;16:183).

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. Most severely burned patients develop early pulmonary insufficiency and respiratory failure. The etiology of this failure can be direct thermal injury to the upper airways or, more commonly, indirect acute lung injury secondary to activation of systemic inflammation. In addition, the decreased pulmonary compliance and chest wall rigidity of burn patients can lead to iatrogenic ventilator-induced lung injury. The use of lower tidal volumes, permissive hypercapnia, and the Òopen lungÓ approach to ventilation can significantly improve outcome (*N Engl J Med.* 2000;342:1301).

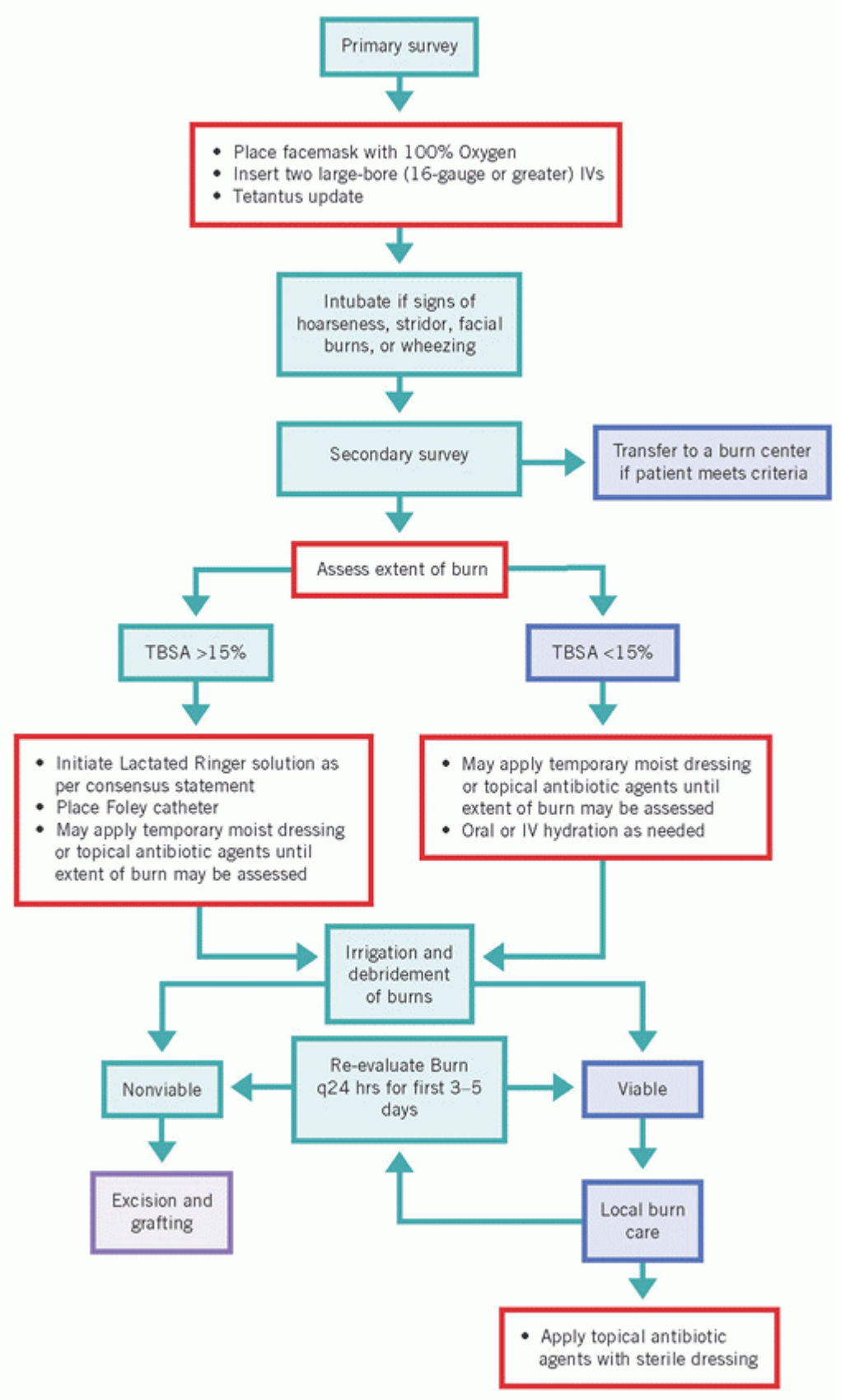

Figure 8-1 Algorithm for the evaluation and management of burns.

3. Circulation. Circulatory support in the form of aggressive and prompt fluid resuscitation is a cornerstone of early burn management. Burn injury causes a combination of hypovolemic and distributive shock characterized by the release of inflammatory mediators, dynamic fluid shifts from the intravascular compartment to the interstitium, and exudative and evaporative water loss from the burn injury. Full-thickness circumferential extremity or neck burns require escharotomy if circulation distal to the injury is impaired; however, escharotomies are rarely needed within the first 6 hours of injury.

4. Exposure. 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.

C. Associated injuries can result from explosions, falls, or jumping during escape attempts.

D. Patient age is a major determinant on outcome. Infants and elderly patients are at highest risk. Burns are a common form of child abuse and need to be considered in every child. Suggestive physical examination findings include stocking/glove injury patterns, lack of splash marks, and dorsally located contact burns of the hands (*Forensic Sci Int.* 2009;187:81). Elderly patients often have comorbid medical problems and decreased physiologic reserve.

E. State of Health. Preexisting medical problems should be noted, with particular attention paid to cardiac, pulmonary, renal, and gastrointestinal systems.

F. Burn-specific Secondary Survey

1. Depth of burn (Table 8-1)

a. 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 to 4 days.

b. Second-degree burns, which are subdivided into **superficial or deep partial-thickness burns,** are limited to the dermal layers of the skin. **Superficial** partial-thickness burns involve the papillary dermis. 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 dermal appendages (e.g., nerves, sweat glands, or hair follicles). Hence, such burns can be less sensitive or hairs may be easily plucked out. Nonetheless, the only definitive method of differentiating superficial and deep partial-thickness burns is by length of time to heal. Furthermore, any partial-thickness burn can convert to full-thickness injury over time, especially if early fluid resuscitation is inadequate or infection ensues.

TABLE 8-1 Treatment Algorithm for the Three Clinically Important Burn Depths[a]

Burn Depth[b]	Level of Injury	Clinical Features	Treatment	Usual Result
Superficial partial-thickness	Papillary dermis	Blisters Erythema Capillary refill Intact pain sensation	Tetanus prophylaxis Cleaning (e.g., with chlorhexidine gluconate) Topical agent (e.g., 1% silver sulfadiazine) Sterile gauze dressing[c] Physical therapy Splints as necessary	Epithelialization in 7-21 days Hypertrophic scar rare Return of full function
Deep partial-thickness	Reticular dermis	Blisters pale white or yellow color Absent pain sensation	As for superficial partial-thickness burns Early surgical excision and skin grafting an option	Epithelialization in 21-60 days in the absence of surgery Hypertrophic scar common Earlier return of function with surgical therapy
Full-thickness	Subcutaneous fat, fascia, muscle, or bone	Blisters may be absent Leathery, in classic, wrinkled	As for superficial partial-thickness	Functional limitation more frequent Hypertrophic

	appearance over bony prominences No capillary refill Thrombosed subcutaneous vessels may be visible Absent pain sensation	burns Wound excision and grafting at earliest feasible time	scar mainly at graft margins

[a]Epidermal (first-degree) burns present clinically with cutaneous erythema, pain, and tenderness; they resolve rapidly and generally require only symptomatic treatment.

[b] No clinically useful objective method of measuring burn depth exists; classification depends on clinical judgment.

[c] Sterile gauze dressings are frequently omitted on the face and neck.

Reprinted with permission from Monafo WW. Initial management of burns. *N Engl J Med.* 1996;335:1581.

c. Full-thickness (third- or fourth-degree) burns involve all layers of the skin and some subcutaneous tissue. In **third-degree** burns, all skin appendages and sensory fibers are destroyed. This results in an initially painless, insensate dry surface. **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.

G. Percentage of Body Surface Area (BSA) Estimation. The accurate and timely assessment of BSA is a critical aspect of the initial evaluation of burned patients. It will determine whether transfer to a specialized burn center is required as well as the magnitude of initial fluid resuscitation and nutritional requirements (*J Burn Care Res.* 2007;28:42).

1. Small areas: The area of patient's hand (including palm and extended fingers) equals 1% of BSA (*Burns.* 2001;27:591).

2. Large areas: Òrule of ninesÓ: Regions of the body approximating 9% BSA or multiples thereof are shown in Figure 8-2. Note that infants and babies have a proportionally greater percentage of BSA in the head and neck region and less in the lower extremities than adults (*Burns.* 2000;26:156).

II. MANAGEMENT

A. Emergency Room

1. Resuscitation. A surgical consultation is initiated for all patients with major injury.

a. Oxygen should be provided to patients with all but the most minor injuries. A 100% oxygen high-humidity facemask for those with possible inhalation injury assists the patient's expectoration from dry airways and treats carbon monoxide poisoning.

b. Intravenous access. All patients with burns of 15% or greater BSA require intravenous fluids. Two 16-gauge or larger peripheral venous catheters 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 sites are unavailable and may be sutured in place. Avoid lowerextremity catheters, if possible, to prevent phlebitic complications.

c. Fluid. Improved survival in the era of modern burn care is largely attributable to early and aggressive volume resuscitation. Intravenous fluid in excess of maintenance fluids is administered to all patients with burns of 15% or greater BSA in adults (³10% BSA in children) and generally follows established guidelines and formulas. In particular, fluid resuscitation based on the consensus formula is widely used

and has decreased the occurrence of burn-induced shock (*J Burn Care Res.* 2008;29:257).

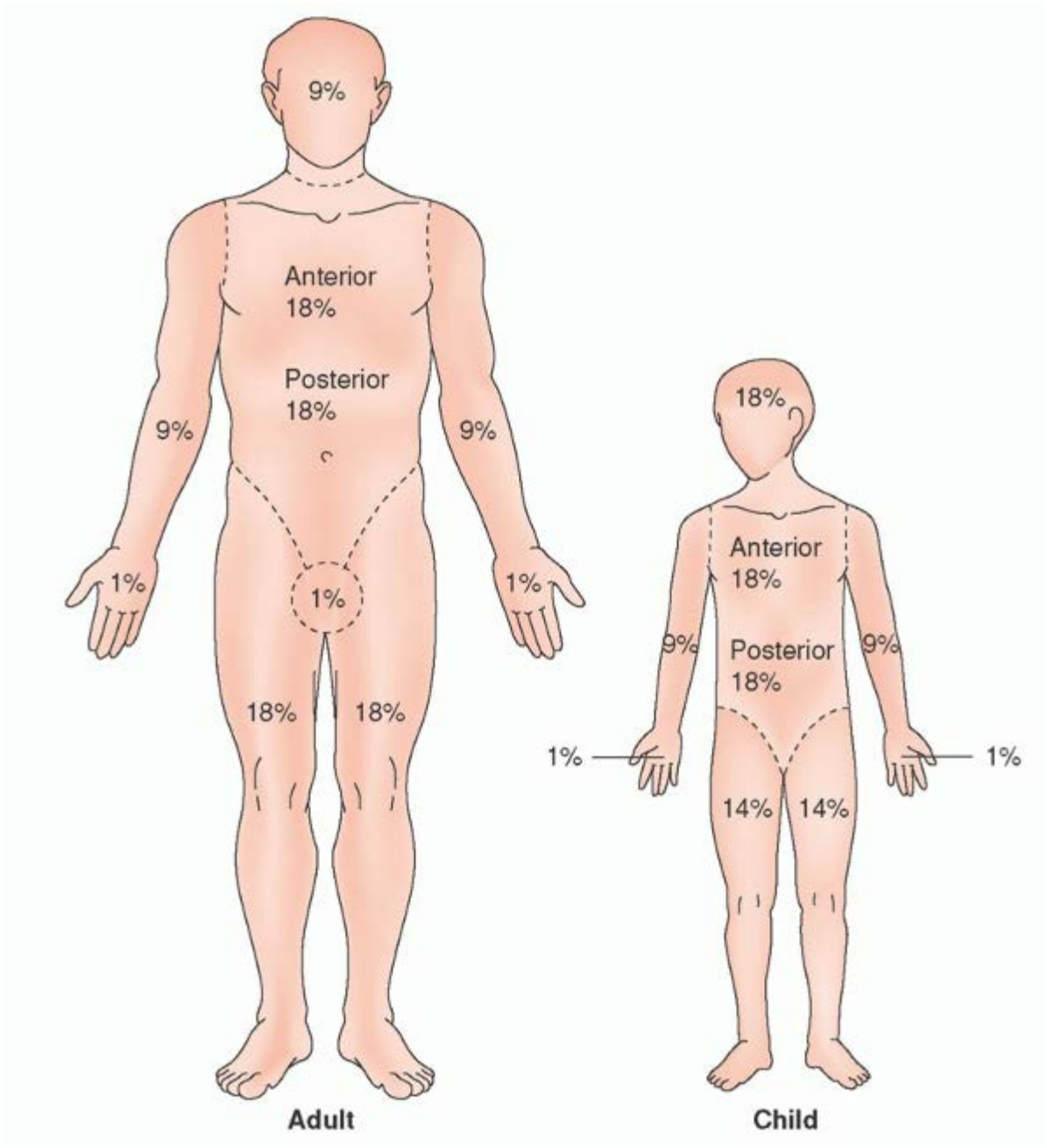

Figure 8-2 Estimation of percent area burned in adults and children using the rule of nines.

(1) Consensus formula. The estimated crystalloid requirement for the first 24 hours after injury is calculated on the basis of patient weight and BSA burn percentage. Lactated Ringer solution volume in the first 24 hours = 2 to 4 mL × %BSA (second-, third-, and 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. Furthermore, for children weighing 30 kg or less, 5% dextrose in one-quarter normal saline maintenance fluids should supplement the Parkland formula 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 should be avoided as an intravenous therapy until after the first 12 to 24 hours postburn, at which time capillary leak diminishes. A study of 7,000 critically ill (nonburned) patients found that while colloid resuscitation resulted in less volume administered, it did not improve organ failure rates, ventilator days, or mortality (*N Engl J Med.* 2004;350:2247). A Cochrane review also found that the relative risk of death was 2.4 times higher in burned patients who received albumin than in those who were given only crystalloid fluids (*Cochrane Database Syst Rev.* 2002;4:CD001208).

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/hour in children (weighing ²30 kg) and 0.5 mL/kg/hour in adults is the guideline for adequate intravenous infusion.

e. Nasogastric tube insertion with low suction is performed if patients are intubated or develop nausea, vomiting, and abdominal distention consistent with adynamic ileus. Virtually all patients with burns of greater than 25% BSA will have ileus.

f. Escharotomy may be necessary in full-thickness circumferential burns of the neck, torso, or extremities when increasing tissue edema impairs peripheral circulation 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 aspects of the extremities and the anterior axillary lines of the chest (*J Burn Care Res.* 2009;30:759). 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.

2. Continuous pulse oximetry to measure oxygen saturation is useful. One caveat is that falsely elevated levels can be observed in carbon monoxide poisoning.

3. Laboratory evaluation includes a baseline complete blood cell count, type and crossmatch, electrolytes and renal panel, β-human chorionic

gonadotropin (in women), arterial carboxyhemoglobin, arterial bloodÑgas evaluation, and urinalysis. An electrocardiogram is useful initially, particularly in elderly patients or those with electrical burns. Fluid and electrolyte fluxes during resuscitation and later mobilization of

thirdspace edema can result in arrhythmias and interval electrocardiogram 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, to avoid hypothermia. Cold water can also cause vasoconstriction and can extend the depth and surface area of injury.

5. Analgesia is given intravenously every 1 to 2 hours to manage pain but in small doses to guard against hypotension, oversedation, and respiratory depression.

6. Early irrigation and debridement 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 small blisters overlying superficial partial-thickness burns intact because they permit healing in a sterile environment and offer some protection to the underlying dermis. However, in larger and deeper partial-thickness burns, debridement of burn blisters should be done to relieve tension and purge inflammatory mediators. Nonviable tissue in the burn wound should be debrided early because the dead tissue provides a bacterial medium putting the patient at risk for both local and systemic infections. Early excision and grafting has been shown to benefit survival, blood loss, incidence of sepsis, and length of stay compared with serial debridement (*Burns.* 2006;32:145). If the burns resulted from liquid chemical exposure, they are irrigated continuously for 20 to 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.

7. Topical antimicrobial agents are the mainstay of local burn wound management (Table 8-2). Prior to the use of topical antimicrobial agents, the most common organisms causing burn wound infections were *Staphylococcus aureus* and group A streptococci (*J Trauma.* 1982;22:11). Subsequent to the development of topical agents, Gram-negative organisms, particularly *Pseudomonas aeruginosa,* and fungi are the most common causes of invasive burn wound sepsis (*J Burn Care Res.* 2011;32:324). Systemic antibiotics are not administered prophylactically but are reserved for documented infection. Bacterial proliferation may occur underneath the eschar at the viableÑnonviable interface, resulting in subeschar suppuration and separation of the eschar. Microorganisms can invade the underlying tissue, producing invasive burn wound sepsis. The risk of invasive infection is higher in patients with multiorgan failure or burns greater than 30% BSA (*World J Surg.* 2004;22:135). When the identity of the specific organism is established, antibiotic therapy is targeted to that 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. Wound infection is defined by more than 10^5 organisms per gram of tissue. Treatment requires infected eschar excision and appropriate topical/systemic antibiotic therapy.

TABLE 8-2 Topical Antimicrobial Agents for Burns Agent

Agent	Advantages	Disadvantages
Silver sulfadiazine (Silvadene)	Broad spectrum (Gram positive, gram negative, some fungal) Nonirritating Easy to use Few adverse side effects Formulated as a cream, which minimizes water and heat loss, thereby diminishing caloric requirements	*Pseudomonas* residence Poor eschar penetration Occasional transient leukopenia 3 to 5 days after use (harmless, resolves regardless of cessation of treatment)
Mafenide acetate (Sulfamylon)	Broad spectrum (includes *pseudomonas* and *enteroccocus* species) Good eschar penetration	Painful Allergic rash Can cause metabolic acidosis via carbonic anhydrase inhibition, limiting its use to small, full thickness burns
Polymyxin B sulfate (Polysporin), neomycin, bacitracin, mupirocin	Painless Allows wound observation Tolerated well on facial burns Do not discolor skin Mupirocin-improved activity against methicillin-resistant *S. aureus* and gram negative bacteria	Poor gram negative coverage Poor eschar penetration
Silver nitrate	Painless, application as a soaked gauze Good antimicrobial coverage Safe in sulfa allergy	Stains tissue gray to black (makes wound monitoring difficult) Hypotonic (causes severe electrolyte abnormalities)

Acticoat	Easy application Good antimicrobial coverage from impregnated silver ions	Expensive Can only be left in place for 3 days

8. Tetanus prophylaxis. If a patient's last booster was administered greater than 5 years prior, 0.5 mL of tetanus toxoid is given intramuscularly. If immunization status is unknown, 250 to 500 units of human tetanus immunoglobulin (Hyper-Tet) are given intramuscularly.

9. Critical care issues with burns. Issues include burn wound infection, pneumonia, sepsis, ileus, Curling ulcer (gastroduodenal), acalculous cholecystitis, and superior mesenteric artery syndrome.

a. Stress ulcer prophylaxis (e.g., H_2 blockers or proton-pump inhibitors) should be provided for patients who have major burns (Eastern Association for the Surgery of Trauma Stress Ulcer Prophylaxis Guidelines. 2008).

b. Venous Thromboembolism (VTE). Burn patients are at increased risk for VTE and should receive pharmacologic prophylaxis (*Burns.* 2004;30:591). Increased TBSA and need for ICU admission also increase the risk of VTE (*J Burn Care Res.* 2011;32:6).

c. Sepsis. In patients who survive the first 24 hours after injury, burn sepsis is the leading cause of mortality (*Burns.* 2006;32:545). The evidence-based recommendations of the Surviving Sepsis Campaign (*Crit Care Med.* 2008;36(1):296) include antibiotic therapy, source control, crystalloid resuscitation, vasopressor use, a hemoglobin transfusion trigger of 7 g/dL, an open-lung/low-tidal-volume ventilatory strategy, and maintenance of blood glucose less than 180 mg/dL.

B. Outpatient. Only minor first-degree or partial-thickness injuries should be considered for outpatient management. The decision to use outpatient management 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.

1. Dressings are often managed by the patient when the injury is easily accessible. Silver sulfadiazine is often applied as a light coating, followed by sterile dressings once or twice daily.

2. Antibiotics are not prescribed prophylactically. Their use is limited to documented wound infections.

3. Follow-up usually occurs 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 (with the application of

foam tape or Jobst garments), dry skin (with unscented lotion massage), and pruritus (with antihistamines). Hyperpigmentation can be prevented with avoidance of direct sunlight and use of sunscreen.

Rehabilitation potential and need for therapy (physical, occupational, social, and psychological) are also evaluated.

C. Inpatient

1. Transfer to a burn center should follow the guidelines of the American Burn Association (www.ameriburn.org). These criteria reflect multiple studies showing that age and BSA burn percentage remain the two most important prognostic factors. Criteria include:

a. Partial-thickness burns greater than 10% BSA.

b. Any full-thickness burn.

c. Burns that involve the face, hands, feet, genitalia, perineum, or major joints.

d. Any inhalation, chemical, or electrical injury (including lightning).

e. Burn injury in patients with preexisting medical conditions that could complicate management, prolong recovery, or affect mortality.

f. Burns in combination with significant associated mechanical trauma. Note, if the traumatic injury poses a greater threat to life, the patient should be stabilized at a trauma center before transfer to a burn unit.

g. Burned children in hospitals without qualified personnel or equipment for the care of children.

h. Patients requiring specialized rehabilitation, psychological support, or social services (including suspected neglect or child abuse).

2. Nutrition. Severe burns induce a hypermetabolic state proportional to the size of the burn up to 200% the normal metabolic rate. The daily estimated metabolic requirement (EMR) in burn patients can be calculated from the Curreri formula: EMR = [25 kcal × body weight (kg)] + (40 kcal × %BSA). In children, formulas based on BSA are more appropriate. Protein losses in burn patients from both an increased oxidation rate and burn wound extravasation should be replaced by supplying 1.5 to 2 g/kg of protein/day (*Lancet.* 2004;363:1895). Therapeutic strategies should target prevention of body weight loss of more than 10% of the patient's baseline weight. Losses of more than 10% of lean body mass may lead to impaired immune function and delayed wound healing. Losses of more than 40% lead to imminent death (*Shock.* 1998;10:155).

a. 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 decrease the catabolic response, reduce infectious complications, and decrease the length of ICU stay (*J Burn Care Res.* 2011;32:104). Increasing feedings beyond

the EMR results in overfeeding and is associated with difficulty weaning patients from the ventilator, the development of fatty liver, and hyperglycemia, all of which have a negative influence on outcome in burned patients (*Surg Clin North Am.* 2011;91:609).

b. Total parenteral nutrition should be initiated after fluid resuscitation only if the patient is unable to tolerate enteral feeding.

c. Daily vitamin supplementation in adults should include 1.5 g of ascorbic acid, 500 mg of nicotinamide, 50 mg of riboflavin,

50 mg of thiamine, and 220 mg of zinc. Although results from high-dose antioxidant therapy are promising, further clinical trials are needed to define its role in burn patients (*J Burn Care Rehabil.* 2005;26:207).

d. Anabolic adjuncts, including growth hormone (*Ann Surg.* 2009 Sep 2;Epub), insulin-like growth factor, insulin, testosterone (*Crit Care Med.* 2001;29:1936), oxandrolone (*Pharmacotherapy.* 2009;29:213), and propranolol (*N Engl J Med.* 2001;345:1223), have been shown to improve protein synthesis after severe injury, but caution is advised as growth hormone therapy has been found to increase mortality in critically ill patients. However, patients with burns and sepsis were excluded from that study (*N Engl J Med.* 1999;341:785).

3. Wound care

a. Analgesia and **sedation** for dressing changes are necessary for major burns. Benzodiazepines can be used with or without ketamine for sedation. Ketamine can cause tachycardia, hypotension, and arrhythmias. Alternatively, in patients with a secure airway, intravenous propofol has the desired effects of ease of titration and quick onset/offset of action. Either of these sedative regimens in concert with narcotic analgesia is well tolerated.

b. 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.

c. Debridement of all nonviable tissue should take place using sterile technique and instruments once demarcation occurs. Partial-thickness eschar can be abraded lightly using wet gauze. Enzymatic treatments can be useful in dissolving eschar to develop granulation tissue for tissue grafting. All full-thickness eschar should be identified early, excised, and closed or covered before the development of wound colonization and infection.

d. Temporary dressings for massive burns with limited donor sites give stable coverage without painful dressing changes.

(1) Biologic dressings include allograft (cadaver skin) and xenograft (pig skin). These dressings provide the advantages of ease of acquisition and application while providing barrier protection and a biologic bed under which dermis can granulate. After several days, the allograft can be removed, and a meshed autograft may be replaced for definitive coverage. The use of

cultured autologous epithelium (keratinocytes) has shown encouraging results, particularly for patients with massive burns (>80% BSA) and limited donor sites (*J Cell Mol Med.* 2005;9:592). However, this technology is currently limited by the time needed to grow the autograft (2 to 3 weeks) and the relatively lower take rate (50% to 75%).

(2) Synthetic dressings have become an attractive alternative for early wound coverage. **Biobrane** is a collagen-coated silicone membrane that prevents moisture loss, but, therefore, can trap

infection. It is relatively painless and can be easily peeled from the wound after epithelialization. It is useful for superficial partial-thickness burns and skin graft donor sites. **Trancyte** is similar to Biobrane, but also has growth factors from cultured fibroblasts to theoretically aided wound healing. **Integra** consists of an epidermal analogue (silastic film) and a dermal analogue (collagen matrix), making it useful for full-thickness burns. Once adequate vascularization is seen through the silicone layer, the film is removed, and an ultrathin autograft is placed onto the artificial dermis, which allows more rapid reharvesting from the donor site (*J Burn Care Rehabil.* 2003;24:42).

4. Operative management

a. Early tangential excision of burn eschar to the level of bleeding capillaries should follow the resuscitation phase. A meta-analysis of six randomized control trials found decreased morality and decreased length of hospital stay in burn patients with early excision (*Burns.* 2006;32:145); however, this difference was only found in those without inhalational injury. Excision can be performed using a 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 of operating time. Even within such limits, aggressive debridement frequently produces profound blood loss and hypothermia.

b. Split-thickness skin grafts are harvested at a thickness of 0.012 to 0.015 inch (*Clin Dermatol.* 2005;23:332). For cosmetically sensitive areas, autografts are not meshed, or, if necessary, meshed at a narrow ratio (≤2:1). Grafts are secured with absorbable sutures or staples. For very large wounds, split-thickness skin grafts can be meshed up to 4:1 and may be overlaid with meshed allograft tissue. However, cosmesis is poor, and graft take rates may be compromised. 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 extensions in the neck, knee, elbow, wrist, and interphalangeal joints, with 15-degree flexion at metacarpophalangeal joints and abduction at the shoulder (*Clin Plast Surg.* 1992;19:721).

c. Vacuum-assisted closure devices have gained popularity as a means of securing skin grafts with improved take rates compared with standard bolster dressing (*Arch Surg.* 2002;137:930; *J Burn Care Res.* 2014;35:338; *Burns.* 2011;37:925).

III. 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. Gases containing

substances that have undergone incomplete combustion (particularly aldehydes), toxic fumes (hydrogen cyanide), and carbon monoxide can cause tracheobronchitis, pneumonitis, and edema. Mortality may be increased by as much as 20% in these patients. 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 (a 250-minute halflife 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. If it is elevated (>5% in nonsmokers or >10% in smokers), oxygen therapy should continue until normal levels are achieved. Consideration for adjunctive hyperbaric oxygen treatment in CO poisonings and burns should be according to guidelines as set forth by the Undersea & Hyperbaric Medical Society Indications Report. The increased ventilation Ñperfusion gradient and the reduction in peak airway flow in distal airways and alveoli can be evaluated using a xenon-133 ventilationÑperfusion lung scan. Management of minor inhalation injury is by delivery of humidified oxygen. Major injuries require endotracheal intubation for airway protection, preferably with a large-bore tube (7.5 to 8 mm) to facilitate pulmonary toilet of viscous secretions. As discussed earlier, decreased pulmonary compliance is often seen after inhalation injury and can lead to iatrogenic ventilator-associated lung injury. Inhaled bronchodilators can be given to treat bronchospasm whereas nebulized heparin and *N*-acetylcysteine can limit cast formation. It should be mentioned that inadequate fluid resuscitation actually worsens pulmonary injury, likely due to concentration of neutrophils, whose reactive mediators cause lung injury. Prophylactic antibiotic usage is not indicated. Extubation is performed as soon as possible to prevent pneumonia because coughing clears pulmonary secretions more effectively than suctioning.

B. Electrical

1. Factors influencing severity include the voltage (high is >1,000 V), resistance, type of current, current pathway through the body, and duration of contact with an electrical source (*Annu Rev Biomed Eng.* 2000;2:477). Electrical current passes in a straight line between points of body contact with the 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 (*Ann Intern Med.* 2006;145:531). 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 that have a higher resistance to electricity, such as skin, bone, and fat, tend to increase in temperature and coagulate, causing deep thermal burns. Nerves and blood vessels have low resistance and readily conduct electricity (*Crit Care Clin.* 1999;15:319). 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 (i.e., from household power lines) can lead to repetitive, tetanic muscle contraction. In fact, when contact occurs between the palm and an electrical source, alternating current can cause a hand to grip the source of electricity because of a stronger flexor than extensor tone and lead to longer electrical exposure (*J Forensic Sci.* 1980;25:514). 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 is passed through the body or during a fall.

(2) Direct current emanates from batteries and lightning and causes a single muscle contraction, often throwing the person receiving the electrical shock away from the source of electricity. With a voltage of at least 100,000,000 V and a current of 200,000 A, lightning kills 150 to 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 Òsplashed-onÓ pattern of skin burn.

2. Complications include cardiopulmonary arrest (more common with alternating current) (*Br Heart J.* 1987;57:279), thrombosis, associated fractures related to fall or severe muscle contraction (*Am J Surg.* 1977;134:95), spinal cord injury (*Neurology.* 2003;60:182), and cataracts (*J Burn Care Rehabil.* 1991;12:458). **Rhabdomyolysis** may occur and result in myoglobin release from injured cells of deep tissues. Precipitation of protein in the renal tubules can cause acute renal failure (*Burns.* 2004;30:680). Dark urine is the first clinical indication of myoglobinuria, and intravenous lactated Ringer solution should be administered to maintain a urine output greater than 2 mL/kg/hour. Although somewhat controversial, concomitant administration of intravenous sodium bicarbonate and mannitol to solubilize hemochromogens can potentially minimize nephrotoxicity from 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 to 30 minutes. Alkali burns penetrate more deeply than acid burns and require longer periods of irrigation. Irrigation has a threefold effect: It dilutes the chemicals already in contact

with the skin, washes unreacted agent from the skin, and helps to correct the hygroscopic effects that some agents have on tissues (*ANZ J Surg.* 2003;73:45). Neutralizing the chemicals is not recommended because the resulting reaction generates

heat, which can exacerbate the injury. All chemical injuries to the eye are potentially blinding and require copious irrigation with several liters of water and prompt referral to an ophthalmologist (*BMJ.* 2004;328:36). Tar can cause ongoing burns which can be quite deep if not removed promptly. Treat them by cooling the tar with cold water followed by removing any remaining tar with adhesive remover.

D. Cold Injury

1. Hypothermia is defined as a core body temperature less than 35¡C. Mild hypothermia is classified as a core body temperature of 32¡C to 35¡C; moderate hypothermia is 30¡C; and severe hypothermia is less than 30¡C (*CMAJ.* 2003;168:305). The elderly and children are particularly susceptible. Signs of hypothermia include reduced levels of consciousness, dysrhythmias, and skin that appears cold, gray, or cyanotic. Moderate to severe hypothermia is a medical emergency and necessitates maintenance of airway, breathing, and circulation. Core body temperature should be monitored by means of an esophageal or rectal probe. The heart becomes increasingly irritable at core temperatures below 34¡C, and cardiac monitoring should be routine in all hypothermic patients (*Ann Emerg Med.* 1989;18:72). Asystole may occur below 28¡C, and cardiopulmonary resuscitation should be started and maintained until the patient is rewarmed to at least 36¡C. Rewarming can be passive or active. Passive rewarming involves using blankets to cover the body and head. The warming rate ranges between 0.5¡C and 2¡C per hour. Active external warming includes the use of heating blankets or a heated forced-air system, which can increase rewarming rates by 1¡C per hour as compared with simple cotton blankets (*Ann Emerg Med.* 1996;27:479). Active internal rewarming can be started immediately in the case of severe hypothermia and includes the use of warmed intravenous fluids and oxygen, together warming at a rate of 1¡C to 2¡C per hour (*Resuscitation.* 1998;36:101). Although rarely used, active invasive rewarming methods can warm faster, at a rate 1¡C to 4¡C per hour. Examples of this approach include warmed peritoneal lavage, thoracostomy lavage, and bladder lavage. Extracorporeal rewarming of blood via a continuous veno-venous bypass circuit or heated hemodialysis can rewarm at a rate of 1¡C to 2¡C every 5 minutes (*N Engl J Med.* 1997;337:1500).

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 consists of rapid rewarming in a warm water bath between 40¡C and 42¡C until the tissue perfusion returns, which also may help to minimize tissue loss (*Surg Clin North Am.* 1991;71:345). Splinting and elevation of the frostbitten extremity may reduce edema and promote tissue perfusion. 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. Any ruptured blisters should be debrided and covered with a topical antimicrobial and gauze. 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.

CHAPTER 8: BURNS

Multiple Choice Questions

1. A 31-year-old otherwise healthy male is brought to the hospital from a house fire. He answers questions with a hoarse voice and has burns to his face, chest, and arms. His heart rate is 120, with a blood pressure of 80/43, a respiratory rate of 35, and an oxygen saturation of 98% on 2 L nasal cannula. What is the next step in management?

a. Chest x-ray

b. Aggressive fluid resuscitation

c. Intubation

d. Blood transfusion

e. Escharotomies

View Answer

2. A patient receives a scald burn to his arm after spilling hot tea. The burn is red and blistered, and is painful to touch. What is the depth of this burn?

a. First degree

b. Second degree

c. Third degree

d. Fourth degree

View Answer

3. A 42-year-old patient presents with second-degree burns to the anterior surface of both legs and anterior torso. What is his total percentage body surface area burn?

a. 18%

b. 36%

c. 45%

d. 54%

e. 63%

View Answer

4. What is the most common organism to cause burn sepsis?

a. *E. Coli*

b. Group A streptococci

c. *Staphylococcus epidermidis*

d. Enterococcus

e. Pseudomonas

View Answer

5. What is a side effect of mafenide acetate?

a. Metabolic acidosis

b. Neutropenia

c. Hyponatremia

d. Thrombocytopenia

e. Gastrointestinal upset

View Answer

6. A 42-year-old man city worker presents after sustaining an electrical burn. He has contact burns to his hands and feet. His EKG shows normal sinus rhythm. What is this patient at risk for?

a. Respiratory distress

b. Renal failure

c. Hyperthermia

d. Hypothermia

e. Infection

View Answer

7. Which of these is critical for management of significant burns?

a. Prophylactic antibiotics

b. Early irrigation and debridement

c. Adequate nutrition

d. a, b, and c

e. b and c

View Answer

9
Wound Care

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John P. Kirby

Acute wound healing is the normal orderly process that occurs after injury and often requires minimal practitioner intervention. *Chronic* wound healing *does not* follow that orderly progression of healing and often necessitates a variety of interventions to facilitate complete healing.

ACUTE WOUND HEALING

I. PHYSIOLOGY OF THE ACUTE WOUND.

Disruption of tissue integrity initiates a sequence of events directed at restoring the injured tissue to a healed, normal state. Normal wound healing occurs in an orderly fashion and is a balance of repair and regeneration of tissue.

A. Early Wound Healing

1. Stages of hemostasis. Tissue trauma commonly causes bleeding. Vasoconstriction immediately follows, and the coagulation cascade is initiated. This process contains hemorrhage and stimulates fibrin. The fibrin matrix further activates **platelets** and also serves as the initial scaffold for wound healing. In later phases of wound healing, the fibrin matrix facilitates cell attachment and serves as a reservoir for cytokines.

2. Inflammatory phase (days 1 to 4). Injury immediately activates three plasma-based systems: The coagulation cascade, the complement cascade, and the kinin cascade. Proinflammatory factors attract leukocytes and facilitate their migration out of the intravascular space and into the wound. **Polymorphonuclear leukocytes (PMNs)** are the dominant inflammatory cells in the wound for the first 24 to 48 hours, which phagocytize bacteria and damaged tissue, and also release cytokines such as TNF-alpha and interleukin-1 that further stimulate the inflammatory response and local vasodilation. The inflammatory phase progresses with the infiltration of circulating **monocytes** into the wound. Monocytes migrate into the extravascular space through capillaries and differentiate into **macrophages.** Macrophages are activated by the locally produced cytokines and are essential for normal healing because of their important role in the coordination of the healing process. They phagocytize bacteria and damaged tissue, secrete enzymes for the degradation of tissue and extracellular matrix, and release cytokines for inflammatory cell recruitment and fibroblast proliferation. The inflammatory phase lasts a well-defined period of time in primarily closed wounds (÷4 days), but it continues

indefinitely to the end point of complete epithelialization in wounds that close by secondary or tertiary intention. Foreign material, bacteria, or other imbalances that are not

overcome can change a normal healing wound into one with chronic inflammation and chronic nonhealing.

B. Intermediate wound-healing events involve mesenchymal cell migration and proliferation, angiogenesis, and epithelialization.

1. Fibroblast migration occurs 2 to 4 days after wounding. Chemotactic cytokines influence fibroblasts to migrate into the wound from undamaged tissue.

2. While the wound is infiltrated by mesenchymal cells, **angiogenesis** takes place to restore the vasculature that has been disrupted by the wound.

3. Epithelialization 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 of epithelial cells occurs at the rate of 1 mm/day in clean, open wounds. Primarily closed wounds have a contiguous epithelial layer at 24 to 48 hours.

C. Late wound healing involves the deposition of collagen and other matrix proteins and wound contraction. The primary function of the fibroblast at this stage becomes protein synthesis.

1. Collagen is the main protein secreted by fibroblasts. It provides strength and structure in the wound. Collagen is synthesized at an accelerated rate for 2 to 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.

2. Wound contraction is a decrease in the size of the 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 through the contraction of myofibroblasts. Wound contraction begins 4 to 5 days after wounding and continues for 12 to 15 days or longer if the wound remains open.

3. The final wound-healing event is **scar formation and 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. 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 to 18 months, but it may last indefinitely.

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 due to a disruption of the normal process of acute wound healing. Most chronic wounds are slowed or arrested in the inflammatory or proliferative phases of healing and have increased levels of matrix metalloproteinases, which bind up or degrade the

various cytokines and growth factors at the wound surface. Treatment of these causes, along with maximal medical management of underlying medical problems, restores more normal healing processes.

A. Intrinsic or local factors are abnormalities within the wound that prevent normal wound healing. These factors include (1) foreign body, (2) necrotic tissue, (3) repetitive trauma, (4) hypoxia/ischemia, (5) venous insufficiency, (6) infection, (7) growth factor deficiency, (8) excessive matrix protein degradation, and (9) radiation.

B. Extrinsic or systemic factors also contribute to abnormal wound healing. Optimization of these factors is critical to healing a chronic wound: (1) Diabetes mellitus, (2) use of steroids and antineoplastic drugs, (3) smoking, (4) collagen vascular disease, (5) repetitive trauma, and (6) chronic disease states in the kidney and liver.

II. SPECIAL CATEGORIES OF CHRONIC WOUNDS

A. Diabetic Foot Ulcers

1. Evaluation and treatment (Fig. 9-1)

a. Examination. The quality of the peripheral circulation, the extent of the wound, and the degree of sensory loss should be recorded. Web spaces and nails should be examined for evidence of mycotic infection, which may lead to fissuring of the skin and subsequent infection. Neuropathic, arthropathic, and vasculopathic ulcers occur on the plantar surface of the metatarsals and extend to the metatarsal head, leaving exposed cartilage. Evaluation of diabetic foot ulcers should include plain x-rays of the foot to evaluate for osteomyelitis, ankleÑbrachial index measurements for vascular insufficiency, and the SemmesÑWeinstein monofilament test for neuropathy.

b. Treatment. Critical to treatment of any diabetic foot wound is complete offloading of the ulcer with an appropriate diabetic shoe or other orthotic device as well as documentation that the patient is neuropathically insensate.

(1) Clean wounds are treated with minimal debridement and damp gauze or hydrogel-based dressing changes. Hydrogel dressings may be more effective than damp gauze (*Cochrane Database Syst Rev.* 2010;20:CD003556). Exudative wounds may benefit from alginate, hydrocolloid, or Negative Pressure Wound Therapy (NPWT) that removes excess wound exudate, which inhibits wound healing. Close follow-up is essential.

(2) Infected wounds are diagnosed based on clinical signs of infection. Plain x-rays may show

osteomyelitis or gas in the soft tissues. Patients with a suspected infected diabetic foot ulcer should be admitted for inpatient wound care and broad-spectrum antibiotic therapy. Infected wounds require a thorough exploration with drainage of all abscess cavities and debridement of infected, necrotic, or devitalized tissues. The clean wound can then be managed with local wound care as described above.

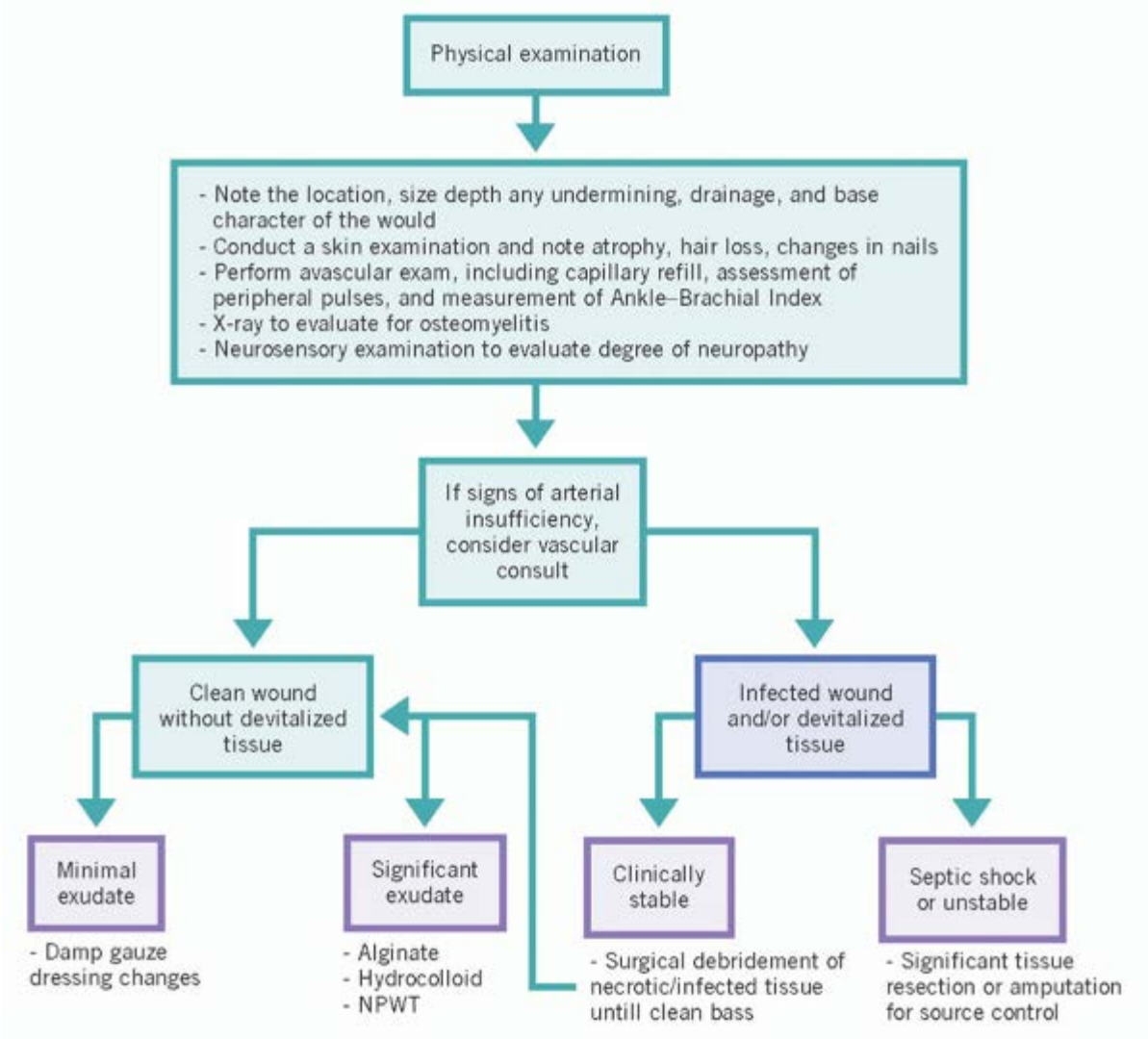

Figure 9-1 Algorithm for the management of diabetic foot wounds.

(3) Antibiotic therapy. For infected wounds, initial antibiotic therapy should be broad spectrum directed at both Gram-positive and Gram-negative organisms. In the acute phase parenteral treatment is indicated. Wound cultures should be obtained prior to initiation of antibiosis. Duration of antibiosis depends on severity of infection. For mild infections limited to the soft tissue 1 to 2 weeks of therapy is sufficient, whereas moderate or severe infections require 2 to 4 weeks of total antibiotic therapy. For osteomyelitis involving viable bone, 4 to 6 weeks of IV

therapy may be indicated. Consultation with an infectious disease specialist is helpful in guiding therapy (*Clin Infect Dis.* 2012;54:e134).

(4) Prevention remains one of the 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. Arterial insufficiency ulcers tend to occur distally on the tips of the patient's toes or near the lateral malleolus. The surrounding skin is thin, shiny, and hairless. Patients frequently complain of claudication or rest pain; however, some patients may have sufficient neuropathy that they lack any pain symptoms even in critical limb ischemia. Peripheral pulses are diminished or absent. When arterial ulcers are suspected, a vascular evaluation should be obtained, including a peripheral and central pulse examination and segmental Doppler limb pressures and flow waveforms with calculation of ankleÑbrachial indices and toe-pressures.

Neglected chronic arterial insufficiency can result in dry or wet gangrene. Wet gangrene can lead to an ascending necrotizing infection while dry gangrene can convert to wet at any time. Critical to treatment of these wounds is restoration of arterial inflow (see Chapter 28). After optimization of arterial inflow, devitalized tissue can be resected to facilitate healing. Arterial insufficiency wounds and dry gangrene must be carefully assessed for signs of infection. If infection is suspected, obtain wound cultures, debride infected tissue, and institute appropriate antibiosis.

2. Venous stasis ulcers are among the most common types of leg ulcers and typically occur on the medial leg in the supramedial malleolar location. A patient with a venous stasis ulcer usually has a history of ulceration and associated leg swelling or of deep venous thrombosis. See Chapter 29 for a complete description of venous stasis ulcers and their treatment.

C. Pressure Ulcers

1. Pathophysiology. Prolonged pressure applied to soft tissue over bony prominences, usually caused by paralysis or the immobility associated with severe illness, predictably leads to ischemic ulceration and tissue breakdown. Muscle tissue seems to be the most susceptible. The prevalence of pressure ulcers is 10% of all hospitalized patients, 28% of nursing home patients, and 39% of spinal cord injury patients (*JAMA.* 2006;296:974). Pressure ulcers increase in-hospital mortality rates more than twofold as well as increase the risk of hospital readmissions (*J Am Geriatr Soc.* 2012;60:1603). 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 9-1). 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 go backward through the stages 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. The examiner must also look for underlying bony breakdown, osteomyelitis, or an overall physiologic decline as the root cause of a ÒpressureÓ ulcer whose actual etiology may be multifactorial in nature, and any successful healing regimen must be equally multifactorial.

TABLE 9-1 National Pressure Ulcer Advisory Panel Classification Scheme

Stage	Description
I	Nonblanchable erythema of intact skin; wounds generally reversible at this stage with intervention
II	Partial-thickness skin loss involving epidermis or dermis; may present as an abrasion, blister, or shallow crater
III	Full-thickness skin loss involving damage or necrosis of subcutaneous tissue but not extending through underlying structures or fascia
IV	Full-thickness skin loss with damage to underlying support structures (i.e., fascia, tendon, or joint capsule)
Unstageable	Full-thickness tissue loss with actual depth of ulcer unknown due to slough and/or eschar in wound bed
Suspected Deep Tissue Injury	Localized area of discolored skin or blood-filled blister due to damage of underlying tissue

2. Prevention

a. Skin care. Skin should be kept well moisturized but protected from excessive contact with extraneous fluids. Barrier products may reduce the risk of pressure ulcers by protecting skin against excessive moisture. Skin should also be cleaned promptly following episodes of incontinence.

b. Frequent repositioning. High-risk patients should be repositioned at a minimum every 2 hours, either while seated or in bed.

c. Appropriate support surfaces. Adequate support surfaces redistribute pressure from the bony prominences that cause pressure ulcers.

d. Static support surfaces. Foam, air, gel, and water-overlay support surfaces are appropriate for low-risk patients.

e. Dynamic support surfaces. These are support modalities that are powered and actively redistribute pressure. These include alternating and low air-loss mattresses. These surfaces are appropriate for highrisk patients.

f. Nutrition. High-risk patients should also undergo nutritional screening to ensure that caloric and protein goals are met.

3. Treatment

a. Debridement. Eschar and necrotic tissue should be debrided unless contraindicated. Sharp debridement of small wounds can be done at the bedside. Larger wounds require operative debridement. Once the bulk of eschar and devitalized tissue is removed, debridement can be continued with wet-to-damp gauze dressings or with enzymatic debridement with topical agents such as collagenase.

b. Wound cleansing. The base of uninfected ulcers should be cleaned with saline irrigation or a commercially available wound cleanser at each dressing change. Antiseptic solutions such as hydrogen peroxide, povidoneÑiodine, or Dakin solution should not be routinely used as they are toxic to tissues and impede healing. For actively infected wounds, a short course (3 to 5 days) of damp-to-dry dressing changes with one-fourth strength Dakins solution may facilitate local bacterial control. However, topical antiseptic solutions cannot take the place of appropriate debridement and systemic antibiotic therapy.

c. Dressing. Dressings should be selected to ensure the wound base remains moist while keeping the surrounding skin dry. Wet-todamp gauze and hydrocolloid dressings are appropriate. NPWT is also useful for pressure ulcers and may facilitate closure as compared to traditional dressings (*Br J Nurs.* 2004;13:135). See the section on negative pressure dressing for indications/contraindications to NPWT.

d. Control of infection and bacterial colonization. All open ulcers are colonized with bacteria. Surface colonization is best controlled with topical wound cleansing. Superficial colonization does not require antibiotic therapy. Evidence of active infection (purulence, surrounding cellulitis, or foul odor) should prompt reexploration of the wound with debridement of any necrotic or infected tissue. Bacterial infection $>10^5$ organisms per gram of tissue can impair wound healing. Quantitative tissue cultures should be obtained from wounds that fail to heal. The underlying bone should be evaluated for osteomyelitis with appropriate imaging.

e. Nutrition. Successful treatment of pressure ulcers requires adequate nutrition. Patients should be provided with 30 to 35 kcal/kg body weight and 1.25 to 1.5 g protein/kg body weight (*National Pressure Ulcer Advisory Panel Quick Reference Guide;* 2014). These estimates should be adjusted for factors such as recent weight changes, BMI, and renal failure or other comorbid conditions.

4. Surgical 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 creation of a musculocutaneous flap; but these measures should be reserved for well-motivated patients in whom a real reduction in risk factors for recurrence is possible.

WOUND CLOSURE AND CARE

I. TYPES OF WOUND CLOSURE

A. Primary intention occurs when the wound is closed by direct approximation of the wound margins. Direct approximation of the edges of a wound provides the optimal treatment on the condition 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 less likely to develop into chronic wounds. At times, release of local tissues is required to achieve tension-free closure. Directly approximated wounds typically heal as outlined earlier, 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. Epithelialization of surgical incisions occurs within 24 to 48 hours of closure. CDC guidelines dictate that a sterile dressing should be left in place during this susceptible period to prevent bacterial contamination.

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-mediated process that aids in wound closure by decreasing the circumference of the wound (myofibroblasts are modified fibroblasts that have smooth muscle cell-like contractile properties). This method is commonly used in the management of wounds that are treated beyond the initial 6-hour window

or for contaminated or infected wounds with a bacterial count of $>10^5$/g of tissue. These wounds are characterized by prolonged inflammatory and proliferative phases of healing that continue until the wound has either 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 to 5 days of open observation so that the cutaneous edges can be approximated at that time. During this period, the normally low arterial partial pressure of oxygen (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.

II. OPEN WOUND CARE OPTIONS.

This brief review is not meant to be comprehensive or an endorsement of any product or product category (Table 9-2). It remains an area of intense research, clinical, and commercial interest in which availability and indications of both established and new products can be expected to change during the publication cycle of this manual. The clinician would do well to weigh each patient's response to treatment, the indications and risks of any particular product, and need for further treatment.

A. Topical Ointments. Petroleum-based ointments that contain one or several antibiotics prevent adherence of dressings to wounds and, by maintaining moisture of the wound environment, they accelerate epithelialization and healing of primarily approximated wounds.

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TABLE 9-2 Wound and Skin Care Products

Product/Trade Name	Advantages	Limitations	Applications
Gauze			

Kerlix (roll gauze)	Debride mechanically	May disrupt viable tissue during change	Moderately/heavily exudating wounds
	Manages exudates by capillarity Permeable to gases	May cause bleeding on removal	Partial- and full-thickness chronic wounds (stages II, III, & IV)
Gauze sponges	Fills dead space Conformable Adaptable	May cause pain on removal Particulate matter may be left in wound Permeable to fluids and bacteria Limited thermal insulation May dehydrate wound bed (if allowed to dry) Damp to dry dressings contraindicatedÑ wound ostomy Continence nurses (WOCN) Society Standards of Care, 1992	Acute wounds Secondary dressing
Transparent Adhesive Dressings			
Tegaderm (3M)	Manages exudates by moisture vapor	Manage light exudates only	IV entry sites
Opsite (Smith & Nephew)	Impermeable to fluids and bacteria	May disrupt fragile skin	Minor burns or lacerations

	Permeable to gases	Application may be difficult	Reduces surface friction in high-risk areas (stage I)
	Visualization of wound		Lightly exudating partial-thickness chronic wounds (stage II)
	Conformable		Over eschar to promote autolytic debridement
	Low profile		Cover dressing
Hydrocolloids			
Restore Hydrocolloid (Hollister)	Forms moist gel in wound bed	Manages moderate exudates	Reduces surface friction in high-risk areas
DuoDerm (ConvaTec)	Impermeable to fluids and bacteria	Impermeable to gases	
Comfeel Ulcer Care Dressing (Coloplast)	Manages exudates by particle swelling	May traumatize fragile skin	Partial- and full-thickness wounds
Tegasorb (3M)		Do not use over eschar or puncture wounds	Moderately exudating wounds
	Thermal insulation good	Use with extreme caution on diabetic ulcers	Venous stasis ulcers in conjunction with Unna boot

	Conformable	Contraindicated in third-degree burns	
Wound Fillers			
AcryDerm strands	Wound filler	Not recommended in dry wounds or wounds with sinus tracts or tunnels	Absorbs moderate to minimal exudate
Absorbent Wound Dressing (AcryMed)	Absorbs exudate Forms moist wound bed		May be used in combination with other wound dressing to increase absorption or fill shallow areas
Hydrogels			
Amorphous	Forms moist wound bed	May dehydrate	Partial- and full-thickness chronic wound (stages II, III)
Restore Hydrogel (Hollister)	Conformable	Minimal absorption	Partial- and full-thickness burns
IntraSite Gel (Smith & Nephew)	Manages exudates by swelling	Requires secondary dressing	Diabetic ulcers Lightly exudating wounds
Enzymatic Debriding Agents			
Collagenase (Santyl, Smith &	Liquefies necrotic tissue	Conditions with pH higher or lower	Debridement of chronic dermal

Nephew)		than 6-8 decrease enzyme activity	ulcers and severely burned areas
Accuzyme (Healthpoint)	Contributes toward formation of granulation tissue and epithelialization of wounds Does not attack healthy tissue or newly formed granulation tissue		
Absorbent Dressings			
	Manages exudates by osmotic action	Permeable to fluids and bacteria	Heavily exudating wounds
Bard Absorption Dressing (Bard Medical)	Cleans debris	May increase pH beyond physiologic levels	Full-thickness chronic wounds (stages III, IV)
	Reduces odor	May sting on application	Malodorous wounds
	Maintains moist wound bed Permeable to gases Molds to wound contour Fills dead space Extends life of secondary dressing Daily dressing change	Requires secondary dressing	

	Inexpensive		
Alginate			
Restore CalciCare (Hollister)	Forms moist gel in wound bed	Permeable to fluids and bacteria	Moderately/heavily exudating wounds
Sorbsan (Dow Hickman Pharmaceuticals)	Manages exudates by capillarity Permeable to gases	May produce burning sensation on application	Partial- and full-thickness wounds (stages III, IV)
Kaltostat (ConvaTec)	Molds to wound contour Fills dead space Irrigates easily from wound bed Reduces wound pain Fibers left in wound are absorbed May be used on clinically infected wounds Nonirritating	Requires irrigation before removal if allowed to dry out	Partial-thickness burns Skin donor sites
Solutions			
Normal saline (0.9%)	Noncytotoxic solution for wound care	Wound dehydrates if allowed to dry out	Partial- and full-thickness wounds
		If dressing saturated, may macerate periwound skin	Dressing changes two to three times daily

Hydrogen peroxide	Chemical debridement of necrotic tissue when used as an irrigating solution	Cytotoxic to fibroblasts Has been documented to result in air embolus if instilled into wound cavities under pressure	Wound irrigationÑ use only half-strength and always rinse wound with normal saline
Povidone-iodine (Betadine)	FDA has not approved for use in wounds	Cytotoxic to fibroblasts until diluted to 1:1,000 May cause acidosis in burn patients Lasting systemic effects include cardiovascular toxicity, renal toxicity, hepatotoxicity, and neuropathy Impairs wound's ability to fight infection and increases potential for wound infection	None for wound care
Antibacterial Cream			
Silver sulfadiazine (Silvadene)	Broad-spectrum antibacterial (*S. aureus, E. coli, P. aeruginosa, P. mirabilis,* β-	Never approved by FDA for wound management Should not be used in presence of	Apply one-eighth in to clean, debrided wound daily or twice daily

	hemolytic streptococci)	hepatic or renal impairment	
Platelet-derived Growth Factor			
Becaplermin (Regranex, Ortho-McNeil Pharmaceuticals)	May promote wound healing in otherwise recalcitrant neuropathic ulcer	Dressing protocol may be confusing	Calculate dose by multiplying length by width of wound in cm and divide by 4
	Very few side effects	Wound must have adequate blood supply Wound must be free of infection No osteomyelitis Wound must be free of necrotic tissue Complex dosing	Wound is irrigated with NS Apply precise amount of drug to wound, cover with NS dressing Leave in place for 12 hrs. Then irrigate wound with NS Pack wound with NS dressing Leave in place for 12 hrs

FDA, Food and Drug Administration; *E. coli, Escherichia coli;* NS, normal saline; *P. aeruginosa, Pseudomonas aeruginosa; P. mirabilis, Proteus mirabilis; S. aureus, Staphylococcus aureus;* IV, intravenous.
Adapted with permission from Rolstad BS, Ovington LG, Harris A. Wound care product formulary. In: Bryand RA, ed. Acute and Chronic Wounds: Nursing Management, 2nd ed. St. Louis: Mosby; 2000.

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 debridement. The maximum amount of debridement 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 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 debridement usually benefit most from sharp debridement 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. Wet-to-moist dressings provide a much gentler debridement, are less painful, and can include sterile normal saline or various additives. Dakin solution, [in full (0.5% sodium hypochlorite), half, or quarter strength] can be used to pack infected open wounds for a brief period when antimicrobial action is desirable. Because of toxic effects upon keratinocytes, the use of Dakin solution is not indicated except in infected wounds for a short period (*Adv Skin Wound Care.* 2005;18:373). Improvement in the foul odor that often emanates from drained abscesses and other infected open wounds is an added benefit of using this additive.

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 debridement, absorbing exudate produced by the wounds, and maintaining a moist wound environment. A nonadherent, nonabsorbent secondary dressing is applied over the gel, and 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 to moderate exudate. They can remain in place for 3 to 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 are 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 chronic wounds may

respond better to collagen than to other dressing materials (*J Am Col Certif Wound Spec.* 2010;2:50).

I. Hydrofibers represent a newer dressing category of strands; they are some of the most absorptive materials available for packing in a heavily draining wound.

J. Growth Factors. Human recombinant platelet-derived growth factor (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, the drug is often used on other wound types. Epidermal growth factor (EGF) is in clinical trials for the treatment of venous stasis ulcers.

K. Skin Substitutes. There are many different types of biologically active materials and skin substitutes and a comprehensive review of their properties and use is beyond the scope of this chapter. The indication and usage of these products is guided by their biologic and material properties. Skin substitutes can be used to facilitate healing of chronic open wounds; provide temporary or permanent wound coverage; and bridge skin, soft tissue, or fascial defects. The usage of individual products is guided by the manufacturer's recommendations and the nature of the wound.

1. Xenograft products (Permacol, EZ derm, Matriderm, Oasis) are derived from animal tissues and consist of a collagen and/or proteoglycan matrix designed to promote influx of fibroblasts.

2. Allogeneic products are acellular tissue substitutes derived from cadaveric sources (AlloDerm, Strattice, Graftjacket, GammaGraft) that can be used to provide wound coverage. Each of these products is differently processed and material properties guide usages including wound coverage and hernia repair.

3. Bioengineered living tissues are composites of a structural mesh and cultured keratinocytes. Cells can be derived from neonatal sources (Dermagraft, TransCyte, Apligraf, OrCel) or autologous skin (Epicel, Laserskin, Epidex, Hyalograft). These advanced products bring living, biologically active cells into the wound bed.

L. Negative-pressure Wound Therapy. Negative pressure created by vacuum-assisted closure devices (Wound VAC or Blue Sky or institutionally created dressings) appears to stimulate capillary ingrowth and the formation of granulation tissue in open wounds while keeping a relatively clean wound environment. VAC therapy is effective in the management of wounds as diverse as diabetic foot wounds, sacral ulcers, mediastinal dehiscence, perineum wounds, and wounds including prosthetic mesh (*Plast Reconstr Surg.* 2006;117:127S). Recently, VAC therapy has been reported to be successful in managing enterocutaneous fistulae (*J Wound Care.* 2003;12:343) and wounds with areas of exposed bone (*Wounds.* 2005;17:137) or tendon (*J Burn*

Care Rehabil. 2002;23:167). VAC therapy is contraindicated when there are exposed major blood vessels, untreated osteomyelitis, or cancer within the wound, and it is relatively contraindicated in anticoagulated patients.

M. Metallic Silver-impregnated Dressings. The broad antimicrobial properties of silver have long been recognized. Silver-impregnated dressings are used extensively for burns, chronic leg ulcers, diabetic, and traumatic injuries. A variety of silver-based dressings are available with specific indications determined by the manufacturer.

III. HYPERBARIC OXYGEN TREATMENT.

Local hypoxia in wound tissue may contribute to delayed healing. Hyperbaric oxygen treatment (HBOT) has been found to increase healing of diabetic foot ulcers as well as reduce the risk of diabetic amputations (*PMR.* 2009;1:471). Standard treatment protocols are based on appropriate debridement and wound care in conjunction with 90 minutes/day at 2 ATA (atmosphere absolute) of oxygen. The Undersea and Hyperbaric Medical Society (UHMS) has recognized the use of HBOT for chronic wounds, diabetic ulcers, and burns (Hyperbaric Oxygen Therapy Indications Manual 2014, http://www.UHMS.org).

CHAPTER 9: WOUND CARE

Multiple Choice Questions

1. When does a well-healed wound reach the original strength of uninjured issue?

a. 5 days

b. 2 weeks

c. 1 month

d. 6 months

e. Never

View Answer

2. A 58-year-old diabetic man presents with a 2×2 cm ulcer on the lateral aspect of big toe. He complains of chronic pain in his foot at rest and when walking. There are no signs of erythema or drainage. What is the best initial step in management?

a. Noninvasive vascular studies

b. Antibiotics

c. Debridement

d. Amputation

View Answer

3. You are called to evaluate a sacral wound on an 85-year-old female patient. She has had multiple strokes and is bed bound. On her sacrum, she has a 5×4 cm area of black eschar that has no drainage. What is the stage of this pressure ulcer?

a. Stage I

b. Stage II

c. Stage III

d. Stage IV

e. Stage is unknown

View Answer

4. After debridement of the sacral decubitus ulcer in Question 3, the wound bed appears clean without any signs of infection. Once all apparent devitalized tissue is removed, what is the next step in treatment?

a. Nothing further is required

b. Normal saline wet to moist dressing changes

c. Dakin's wet to moist dressing changes

d. Hydrogen peroxide damp to dry dressing changes

e. Musculocutaneous flap

View Answer

5. After taking a 24-year-old male with a gunshot wound to the abdomen, it is discovered that he has a significant sigmoid colon injury with gross spillage. He is given a Hartman colostomy, and his fascia is closed while the skin is left open. How will this wound heal?

a. It will not heal

b. Primary intention

c. Secondary intention

d. Tertiary intension

e. Quaternary intention

View Answer

6. What are the benefits of Negative Pressure Wound Therapy (NPWT)?

a. Keeps wound clean

b. Increases angiogenesis

c. Decreases edema

d. Increases granulation tissue growth

e. All of the above

View Answer

7. Which patient is an appropriate candidate for NPWT?

a. A 23-year-old female with necrotizing fasciitis of perineum right after initial wound debridement.

b. A 68-year-old man with wound breakdown after below-the-knee amputation with osteomyelitis.

c. A 58-year-old man with wound breakdown after a femoral popliteal bypass with exposed femoral artery.

d. A 63-year-old female with a noninfected diabetic foot ulcer after debridement.

e. A 54-year-old female with a right breast nonhealing wound after mastectomy with local recurrence in the wound.

View Answer

10

Head, Neck, and Spinal Trauma

Bola Aladegbami

Bradley D. Freeman

The initial evaluation and management of common injuries of the head, neck, and spine is the focus of this chapter.

I. TRAUMA EVALUATION FOR THE HEAD, NECK, AND SPINE.

Evaluation of the head, neck, and spine should be included as part of the primary and secondary surveys and proceed as follows.

A. Head

1. The **Glasgow Coma Score** (GCS) score provides a reliable and reproducible measure of the level of consciousness and should be performed both as part of the initial assessment and serially with changes in clinical condition. The patient is assessed by criteria of eye opening, verbal response, and motor response (Table 10-1), resulting in a score between 3 (deep unconsciousness) and 15. Patients with a score less than 8 are unable to protect their airway and should be intubated.

2. Assessment for laceration and fractures. In order to assess for lacerations and fractures, a systematic visual inspection and palpation of the head and face is performed starting from the cranial vault down to the mandible. The cranial vault and mandible should be palpated for depressed skull fractures and obvious bone deformities, respectively. The presence of a mid-facial fracture is sought by grasping the maxilla and attempting to move it. The conscious patient should be made to bite down to assess for malocclusion (suggestive of either a mandibular or maxillary fracture) and loss of dentition.

3. Eye examination. The surgeon should assess the pupillary size and response, extraocular movement (EOM) and vision and note the presence of periorbital hematomas (raccoon eyes), foreign bodies, and any other obvious signs of eye trauma. Unilateral pupillary dilatation may herald the onset of early brain herniation. Therefore, pharmacologic dilatation of the pupils of a head-injured patient should be avoided. An abnormal EOM examination may signify extraocular entrapment, and raccoon eyes may signify a basal skull fracture.

4. Nasal examination. The nares should be assessed for septal deviation, bleeding, CSF rhinorrhea (a sign of a basal skull fracture), and septal hematoma, which may require evacuation

to prevent septal necrosis.

5. Ear examination. The ears should be assessed for ruptured tympanic membranes, hemotympanum, and otorrhea. The latter two are both signs of a basal skull fracture.

TABLE 10-1 Glasgow Coma Scale[a]

Component	Points
Eye Opening	**Ñ**
Spontaneous	4
To voice	3
To stimulation	2
None	1
Motor Response	Ñ
To command	6
Localizes	5
Withdraws	4
Abnormal flexion	3
Extension	2
None	1

Verbal Response	Ñ
Oriented	5
Confused but comprehensible	4
Inappropriate or incoherent	3
Incomprehensible (no words)	2
None	1

[a]Glasgow Coma Score = Best eye opening + best motor response + best verbal response. If the patient is intubated, the verbal score is omitted and an addendum of ÒTÓ is given to the best eye opening + best motor response score.

6. Oral cavity examination. The oral cavity should be assessed for missing dentition, mucosal or glossal violations, and foreign bodies.

B. Neck and Spine

1. Cervical spine. Patients with potential cervical spine injuries require cervical spine immobilization. To examine the cervical spine, the cervical collar should be removed and the neck must be maintained in a neutral position. The cervical spine should be assessed for posterior midline tenderness and range of motion (flexion, extension, and rotation to the left and right) and palpated for obvious deformities.

2. The **neck** should also be inspected for signs of bleeding and airway injury, such as an expanding hematoma, a pulsatile mass, dysphonia and

crepitus, and to ensure that the trachea is midline. The carotid arteries should be palpated for pulselessness and thrills.

3. Zones of injury. Penetrating injuries to the neck are historically classified according to location and depth. There are three zones of the neck: **Zone I** denotes the thoracic inlet (from the manubrium to the cricoid cartilage); **Zone II** encompasses the mid-neck (from the cricoid cartilage to the angle of the mandible); and **Zone III** spans the upper neck (from the angle of mandible to the base of the skull). Injuries that do not violate the platysma are considered superficial.

4. Spine. The remainder of the spine should be examined for obvious deformity, step-off, and point tenderness. Weakness, paralysis, asymmetry, and loss of sensation in the extremities should be noted. Assessing reflexes and rectal sphincter tone completes the examination.

II. RADIOGRAPHIC EVALUATION

A. Brain Injury

1. Blunt trauma. A noncontrast CT scan (NCCT) is recommended for the assessment of patients at risk for moderate (GCS 9 to 12) to severe (GCS 3 to 8) Traumatic Brain Injury (TBI). Evidence of hematoma, midline shift, and signs of increased intracranial pressure (ICP) may all be identified on an NCCT. Selected patients sustaining mild TBI (GCS 13 to 15) should also be examined using this modality (Table 10-2) (*J Trauma Acute Care Surg.* 2012;73(5 Suppl 4):S307-314).

2. Penetrating brain injury (PBI). The initial imaging for patients with PBI is also an NCCT. In addition, computed tomographic angiography (CTA) is recommended for patients with potential vascular injuries. This encompasses injuries with trajectories through or proximal to vascular structures/sinuses or the Sylvian fissure, as well as the presence of significant subarachnoid hemorrhage or delayed hemorrhage.

B. Facial Fracture. Patients with significant craniofacial soft-tissue injury or clinical signs of facial fractures require radiographic evaluation to determine bony integrity. Facial CT has supplanted most facial plain films for this purpose.

C. Neck. Regardless of the mechanism of injury, patients with neck trauma who are either hemodynamically unstable or who display evidence of major aerodigestive or vascular injury should be intubated. The decision as to whether such patients should undergo diagnostic studies or be taken for operative exploration must be individualized.

1. Blunt neck injury. For stable patients, flexible laryngoscopy or bronchoscopy is typically performed when patients have signs or symptoms concerning for airway injury (stridor, hoarseness, hemoptysis, aphonia, subcutaneous emphysema, or hematoma). A gastrografin study is typically performed on patients with signs or symptoms concerning for an esophageal injury (hematemesis, dysphagia, subcutaneous emphysema, or odynophagia). When a persistent concern exists despite a negative gastrograffin study, a thin barium swallow or esophagoscopy should be performed. CT scans are a useful adjunct study in this patient population.

TABLE 10-2 Criteria for Use of Noncontrasted Head CT (NCCT) in Patients Sustaining Blunt Injury

New Orleans Criteria (Initial GCS 15)[a]	Canadian CT Head Rule (Initial GCS 13-15)[b]
Age >60	Age ³65
Headache	GCS <15 after 2 hours
Intoxication	Open/depressed skull fracture
Posttraumatic seizure	Signs of basal skull fracture
Emesis	>Two episodes of emesis
Persistent anterograde amnesia	Amnesia ³30 min
Supraclavicular trauma	Significant mechanism (peds vs. auto, ejected passenger, fall ³3 ft or 5 stairs)

[a] The New Orleans criteria for use of NCCT applies to patients sustaining blunt head trauma with an initial GCS of 15 who have one of the listed manifestations.

[b] The Canadian CT rule applies to patients who present with a GCS of 13-15, loss of consciousness, amnesia or confusion, and any of the manifestations in the table (*JAMA.* 2005;294(12):1511-1518).

2. Blunt cerebrovascular injury (BCVI). Injury to the carotid or vertebral arteries carries significant morbidity, including the possibility of cerebral hemorrhage or stroke (CVA). Such injuries are commonly a result of hyperextension or hyperflexion with rotation of the neck or a direct blow to the cervical region. The Denver Criteria listed in Table 10-3 are typically used to screen patients for BCVI. It is recommended that patients that meet these criteria undergo CTA.

3. Cervical spine/collar management. Classic radiographic evaluation of the cervical spine includes antero-posterior, lateral, and open-mouth (odontoid) views. However, CT scans with coronal and sagittal reconstructions are replacing plain x-rays as the primary imaging modality (*Trauma.* 2009;67(3):651-659; *Neurosurgery.* 2013;60 (Suppl 1):82-91).

4. Plain radiographs are now primarily recommended when a CT scan is not easily accessible, when CT results are poor due to motion artifacts, to further assess spinal instability (with flexion/extension films), and to differentiate rotatory subluxation from positional rotation of the atlanto-axial joint. The algorithm to adequately rule out the possibility of a cervical spine injury differs based on whether or not a patient is cognitively intact (Fig. 10-1). No imaging is required for asymptomatic patients that are neurologically and cognitively intact, and c-collars that were placed in the field may be removed from these patients. Patients

that continue to have symptoms despite a negative CT require a more detailed assessment with either an MRI performed within 48 hours of the injury or flexion/extension x-rays before the c-collar can be removed. If this additional workup is negative or the patient's symptoms resolve, the c-collar can then be removed. Patients with transiently altered mental status should be re-evaluated when they become cognitively intact.

TABLE 10-3 Updated Denver Screening Criteria for BCVI (Burlew et al., 2011)

Signs and Symptoms

- Arterial hemorrhage from neck/nose/mouth
- Cervical bruit in patient <50 years old
- Expanding cervical hematoma
- Focal neurologic defect: TIA, hemiparesis, vertebrobasilar symptoms, Horner syndrome
- Neurologic deficit inconsistent with head CT
- Stroke on CT or MRI

Risk Factors

High-energy transfer mechanism with:

- LeForte II or III fracture
- Mandible fracture
- Complex skull fracture/basilar skull fracture/occipital condyle fracture
- Traumatic brain injury consistent with diffuse axonal injury and GCS <6, cervical subluxation or ligamentous injury, transverse foramen fracture, any vertebral body

fracture, or any fracture of C1-3

- Near hanging with anoxic brain injury
- Clothesline type injury or seat belt abrasion with significant swelling, pain, or altered MS
- Traumatic brain injury with thoracic injuries
- Scalp degloving
- Thoracic vascular injuries
- Blunt cardiac rupture

5. Penetrating neck injury. For the stable patient with a penetrating neck injury, a CTA is the initial recommended radiographic study. It delineates the location of potential vascular or aerodigestive injuries and the tract of the injury, and it aids in operative planning. For patients with CTAs that are equivocal for esophageal injury, esophagoscopy and esophagography should be performed. Bronchoscopy should be performed if tracheal injury is suspected (Fig. 10-2).

D. Thoracolumbar Spine. Patients with back pain, thoracolumbar spine tenderness, neurologic deficits, or known or suspected high-energy mechanisms in which spinal trauma might be sustained should be screened with a CT scan of the axial spine to include the thoracic, lumbar, and sacral regions. A CT scan should be considered for patients with a known or suspected injury to the cervical spine, or any other region of the spine, due

to the high incidence of concurrent spinal injuries. For patients with penetrating spinal injuries, an x-ray or a CT scan can be performed to localize retained foreign bodies.

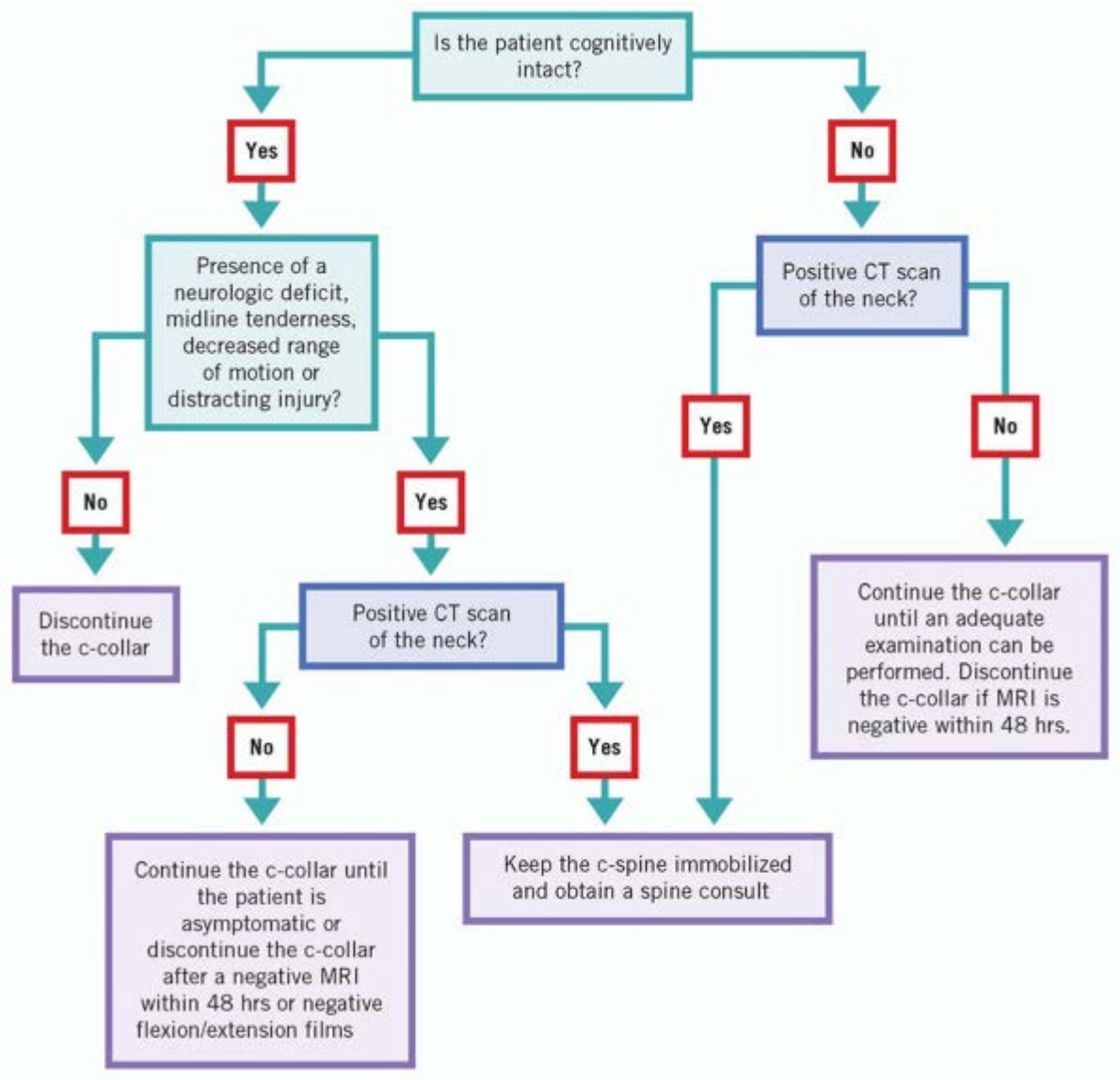

Figure 10-1 Algorithm for Clearing the C-spine.

III. TYPES OF HEAD INJURIES.

This section will provide a brief overview of various head injuries. For a further discussion of the management of these various injuries, please refer to the guidelines for the surgical management of TBI (*Neurosurgery.* 2006;58(Supplement):S2-1-S2-3). Neurosurgical consultation should be sought immediately when these injuries are identified.

A. Epidural hematomas (EDHs) classically present with a Òlucid intervalÓ after injury, which precedes rapid deterioration. This sign is inconsistent and nonspecific, however, and may also be seen with other forms of severe brain injury. EDHs typically result from laceration of the middle meningeal artery due to fracture of the squamosal portion of the temporal bone. Other vessels that are frequently involved include the middle meningeal vein, venous sinuses, and diploic vein.

They appear on head CT scan as biconvex hyperdensities that typically respect the suture lines (Fig. 10-3A,B).

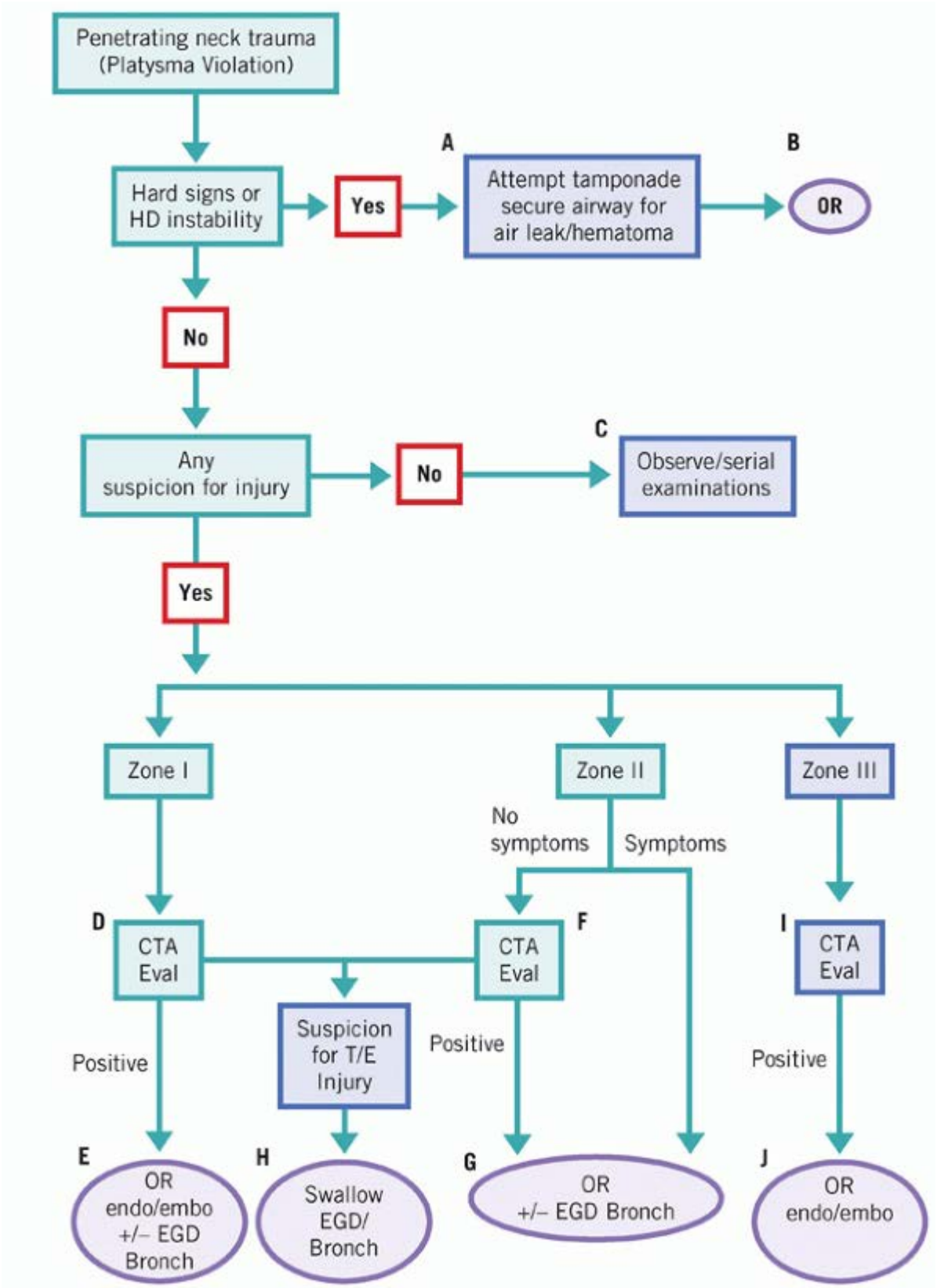

Figure 10-2 Western Trauma Association management algorithm for penetrating neck trauma. (Adapted from Sperry JL, Moore EE, Coimbra R, et al. Western Trauma Association critical decisions in trauma: penetrating neck trauma. *J Trauma Acute Care Surg.* 2013;75(6):936-940, with permission.)

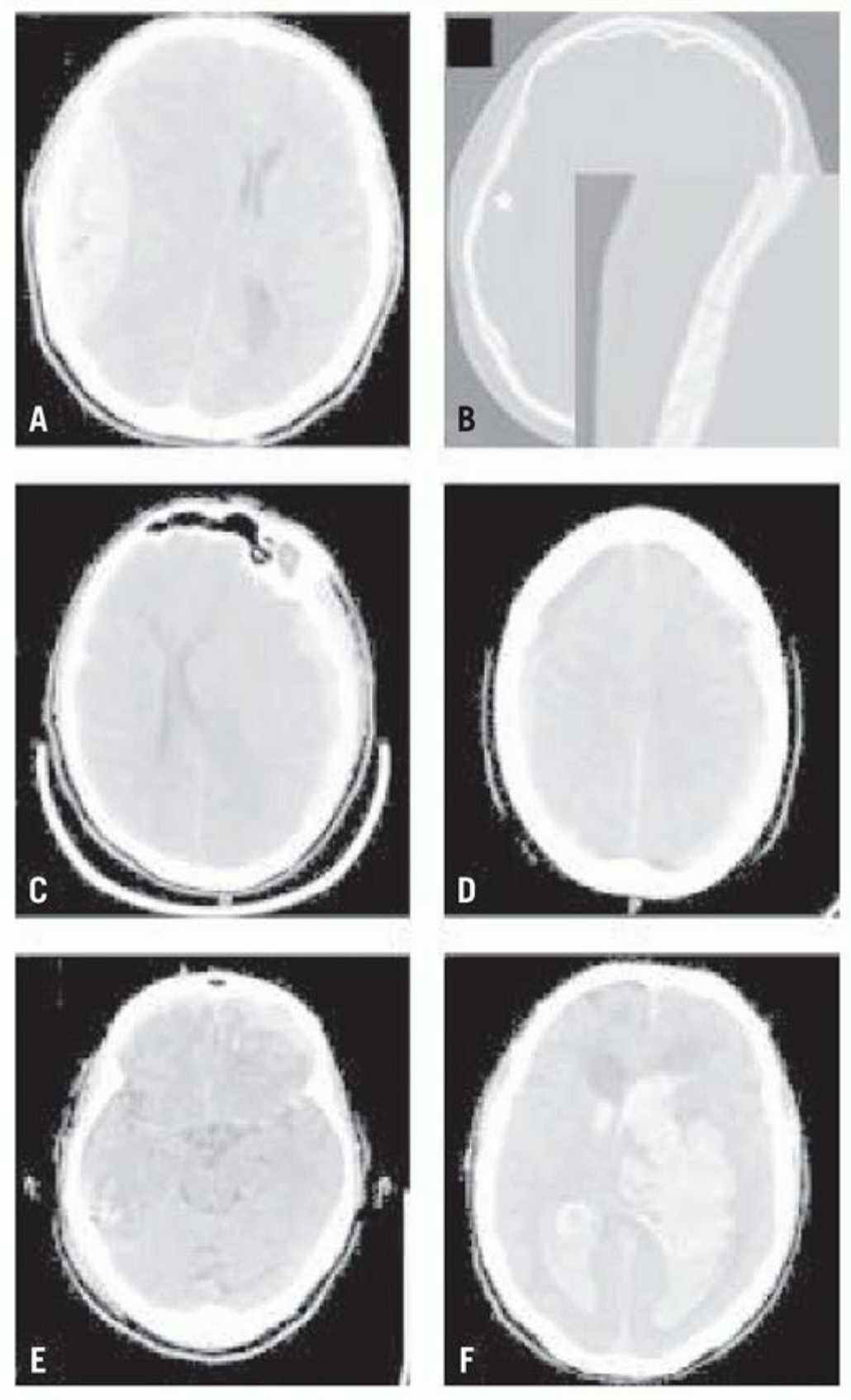

Figure 10-3 Noncontrast head CTs showing **(A)** large right-sided epidural hematoma with mass effect and midline shift, **(B)** bone windows from panel ÒAÓ demonstrating associated linear temporal bone fracture (asterisk, see inset), **(C)** left-sided acute subdural hematoma

with significant midline shift, **(D)** bilateral mixed-density subdural hematomas with both acute (hyperdense) and chronic (hypodense) components, **(E)** bilateral frontal and right-sided temporal hemorrhagic contusions with surrounding edema (hypodense), and (**F**) large left-sided basal ganglia intraparenchymal hemorrhage (nontraumatic, likely related to hypertension) with intraventricular extension resulting in acute hydrocephalus.

B. Acute subdural hematomas (aSDHs) typically appear on head CT scan as hyperdense crescents where the blood spreads around the surface of the brain (Fig. 10-3C). Often, aSDHs result from high-speed acceleration or deceleration trauma and portend severe underlying intracranial injury. These injuries typically result from shearing/tearing forces applied to small bridging (emissary) veins that drain the underlying neural tissue into the dural sinuses.

C. Chronic SDHs (cSDHs) can present days to weeks after the initial head injury, especially in the elderly and alcoholic populations. cSDHs may cause focal neurologic deficits, mental status changes, metabolic abnormalities, and/or seizures. Noncontrast head CT typically shows a hypodense crescentic collection tracking between the dura and the brain (Fig. 10-3D).

D. Cerebral contusions manifest on noncontrast head CT scan as small, punctuate hyperdensities that are commonly located in the basal frontal and temporal lobes (Fig. 10-3E). They may occur during blunt trauma to the head or with acceleration/deceleration injuries. In many cases, damage occurs when the brain comes into contact with the sharp bony ridges on the interior skull base. Contusions may be observed in a ÒcoupÓ pattern, whereby injury to the cerebral cortex occurs in the region immediately underlying the site of impact as the brain collides with the inert table of the skull. Alternatively, a ÒcountercoupÓ pattern occurs when the brain comes into contact with the opposite side of the skull following the initial impact.

E. Intraparenchymal hemorrhages (IPHs) are identified on noncontrast head CT as focal areas of hyperdensity, typically with hypodense surrounding areas of edema (Fig. 10-3F). They may be caused by hypertension, coagulopathy, hemorrhagic transformation of ischemic stroke or tumor, venous outflow obstruction, ruptured aneurysms, or vascular malformations and trauma. Typically, laceration of larger cerebral vessels is the inciting event. Mechanical complications of mass effect may quickly progress to brain herniation in severe cases. Extension of bleeding into the ventricular system may result in **intraventricular hemorrhage** with increased risk of communicating or noncommunicating hydrocephalus due to impaired cerebrospinal fluid (CSF) reabsorption by the arachnoid granulations or focal blockade of CSF flow, respectively.

F. Skull Fractures. There are multiple types of skull fractures. Closed, nondepressed, linear skull fractures are usually managed conservatively. Simple depressed skull fractures which have no skin or galeal disruption are also usually managed conservatively; however, they may require surgical treatment of the fracture if its depression is greater than the width of the skull table. Patients with open, depressed skull fractures may require elevation and debridement of

depressed bony fragments as well as devitalized tissue, followed by a course of antibiotics.

G. Missile injuries from gunshot wounds present several associated problems. Shock waves can result in widespread destruction of brain tissue and vasculature. In cases in which operative management is indicated, the operation may include the removal of accessible foreign bodies and bone fragments, evacuation of intracranial hematomas (ICHs), debridement, and decompression.

IV. OTHER CONSIDERATIONS WHEN MANAGING BRAIN INJURY

A. Monitoring and Treatment of Elevated ICP. ICP monitoring is recommended if serial neurologic examinations cannot be used as a reliable indicator

of progressive intracranial pathology. The goal of monitoring is to maintain ICP <20 mm Hg and cerebral perfusion pressure (CPP = MAP Ð ICP) between 50 and 70 mm Hg. The multiple treatment modalities for elevated ICP are listed in Table 10-4. Initial steps in the management of elevated ICP include sedation, pain control, osmotic agents, and hyperventilation (Fig. 10-4).

TABLE 10-4 Therapies for Intracranial Hypertension

Therapeutic Modality	Usage
Mannitol	• Dose: 0.25-1 g/kg IV every 2-6 hrs in hemodynamically stable patients with adequate renal function • Monitor electrolytes • Keep serum osmolarity <320 • Strict I and O
Hypertonic saline 3%	• Dose: 250 mL bolus IV every 6 hours (30-60 mL IVq6h if 23.4% hypertonic saline is used) or continuous infusion 1 mL/kg/hr • Place central line • Keep Na <160
Hyperventilation ($PaCO_2$ 30-35 mm Hg)	• Limit use to acute and emergent situations, until other interventions take effect • This may worsen cerebral ischemia

Sedation	• To prevent agitation, pain, and patient-ventilator dyssynchrony
Metabolic suppression (Barbiturate coma)	• Usually reserved for refractory ICP • Adequate hemodynamic monitoring is needed to prevent hypotension
Surgical decompression	Consider for patients with: • mass lesions • uncontrollable ICPs • deteriorating neurologic examination
Temperature control	• Avoid fevers (antipyretics, cooling blankets etc.) • Hypothermia (32-34¡C) can be considered for patients with refractory ICP
Other	• Elevate the head of the bed to 30 degrees • CSF drainage by ventriculostomy

B. 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 poor outcomes in severely head-injured patients.

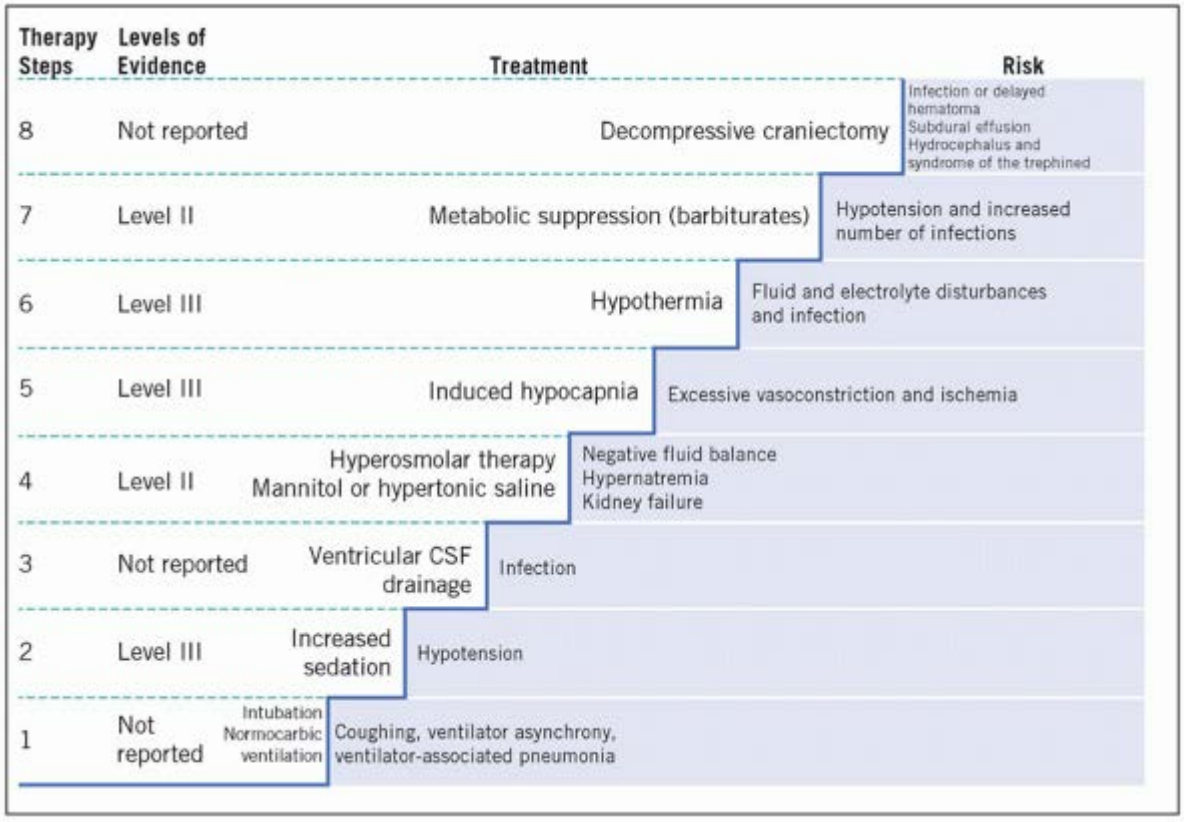

Figure 10-4 Staircase approach for managing Intracranial hypertension. (Adapted from Stocchetti N, Maas A. Traumatic intracranial hypertension. *N Engl J Med.* 2014;370:2121-2130, with permission.)

C. Coagulopathy, if present, should be corrected as expeditiously as possible. The goal of therapy is minimizing hematoma expansion. Anticoagulants should be discontinued. For patients with warfarin-associated ICH, patients should be given 10 mg of IV vitamin K and transfused with FFP. An alternative is to transfuse prothrombin complex concentrate (PCC), which contains factors II, VI, IX, and X. PCC should also be considered in patients taking Factor II (i.e., Dabigatran) or Xa (i.e., Apixaban, Rivaroxaban) inhibitors. International normalized ratio (INR) and partial thromboplastin time (PTT) should be maintained at ²1.4 and <40 seconds, respectively, and platelets should be kept ³100,000. Table 10-5 contains steps for the reversal of anticoagulation in ICH patients.

D. Deep Venous Thrombosis Prophylaxis. Patients with severe head injury are at high risk for deep venous thrombosis and subsequent pulmonary embolism. Early use of intermittent pneumatic compression devices is recommended. Recent studies also suggest no increased risk of intracranial hemorrhage or expansion of hemorrhage in head-injured patients who receive chemical prophylaxis (*J Am Coll Surg.* 2011;213(1):148-153; discussion 153-154).

E. Seizures. TBI patients with ICHs, depressed skull fractures, penetrating brain injuries, cortical

contusion, seizures within 24 hours of injury, and a GCS <10 are at an increased risk for seizures. Antiepileptic medication for a duration of 7 days is recommended to prevent early posttraumatic seizures. Levetiracetam (Keppra) may be loaded orally or intravenously at 1,000 mg and then continued at 500 to 1,000 mg twice daily. Phenytoin may also be used, but the former agent is preferred because it does not require serum drug level monitoring, has an acceptable side-effect profile, and is superior for patients with hepatic disease. Levetiracetam appears to be as effective as phenytoin in the prevention of early posttraumatic seizures.

TABLE 10-5 Reversal of Anticoagulation

Anticoagulant	Lab Investigation	Reversal
VKA (warfarin)	• PT/INR STAT • PT/INR 15-30 min after 4-factor PCC administration	1. Hold VKA 2. Vitamin K 10 mg slow IV infusion 3. If the INR is: 1. 2-4: give 25 U/kg of 4F-PCC 2. 4-6: give 35 U/kg of 4F-PCC 3. >6: give 50 U/kg of 4F-PCC
Dabigatran	• Dabigatran TT and APTT STAT • Drug concentration by anti-IIa or ECT, if available • TT and APTT 15-30 min after 4-factor PCC administration	1. Hold dabigatran 2. If acute presentation consider orally activated charcoal 3. If possible, initiate dialysis 4. 50 U/kg of 4F-PCC
Rivaroxaban	• PT/INR STAT	1. Hold rivaroxaban 2. If possible, initiate plasma exchange

	Drug concentration by anti-Xa assay, if available • PT/INR 15-30 min after 4-factor PCC administration	3. Give 50 U/kg of 4F-PCC
Apixaban	• PT/INR STAT • Drug concentration by anti-Xa assay, if available • PT/INR 15-30 min after 4-factor PCC administration	1. Hold apixaban 2. Give 50 U/kg of 4F-PCC
UFH	• APTT STAT • APTT 15 min following protamine administration	1. Discontinue UFH Infusion 2. Administer protamine. To calculate the dose, determine the amount of heparin units administered in the previous 2 hours, target neutralization of 80%; 1 mg of protamine neutralizes 100 units of heparin.[a]
LMWH	• Anti-Xa assay STAT	1. Hold LMWH 2. Administer protamine: When the last enoxaparin dose is <8 hrs ago, give a slow intravenous infusion: 1 mg protamine for every 1 mg LMWH[a]. When the last enoxaparin dose is 8-24 hrs ago, give a slow intravenous infusion: 0.5 mg protamine for every 1 mg LMWH[a]

PT, Prothrombin Time; INR, International Normalized Ratio; PCC, Prothrombin Complex

Concentrate; TT, Thrombin Time; APTT, Activated Partial Thromboplastin Time; VKA, Vitamin K Antagonist; UFH, Unfractionated Heparin; LMWH, Low-Molecular-Weight Heparin; ECT, Ecarin Clotting Time.

[a] Protamine has a maximum dose of 50 mg.

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F. Close monitoring of **fluid and electrolytes** is essential because alterations in sodium and water balance are common in TBI patients. This may also worsen the neurologic status of the patient. Head-injured patients are at risk for development of diabetes insipidus, syndrome of inappropriate antidiuretic hormone (SIADH), and cerebral salt wasting syndrome (CSWS).

G. Diabetes insipidus can develop rapidly and is characterized by large amounts of urine output (>200 mL/hour) with a low specific gravity (<1.005) and low urine osmolarity (<200 mOsm/kg). The patients are typically hypernatremic and are usually treated with fluid hydration. Persistent DI may necessitate ADH supplementation with vasopressin 5 to 10 units IM/SC q8-12hours or desmopressin (DDAVP) 1 to 2 µg SC/IV q12hours. Both SIADH and CSWS can cause hyponatremia and are characterized by increased urinary sodium. They both can be treated with infusions of hypertonic fluid, salt tablets, and fludrocortisone. SIADH alone can be treated with ADH receptor inhibitors like Conivaptan due to increased ADH activity.

V. SPINAL INJURIES.

The spinal column is divided into the anterior, middle, and posterior columns. The **anterior column** contains the anterior longitudinal ligament, anterior half of the annulus fibrosus and vertebral body. The **middle column** consists of the posterior ligament, posterior half of the annulus fibrosus and vertebral body, and the **posterior column** consists of the ligamentum flavum, articulating facets, lamina, and spinous processes. As a generalized rule, an injury affecting one column is stable; however an injury affecting two or more columns is unstable.

A. Compression Fractures. These injuries usually occur as a result of axial loading forces on the spinal column, resulting in height loss of the anterior portion of the vertebral body. These are usually stable injuries due to the fact that only one column is usually affected. Hence, they can be treated with a cervical collar or thoracic braces. In some patients, there is a greater than 50% loss in height of the anterior vertebral body, which places the posterior column under significant strain. These patients may be considered for possible fixation procedures.

B. Burst Fractures. These fractures involve both the anterior and middle columns and occur as

a result of axial loading. Burst fractures are potentially unstable and carry a high incidence of associated of neurologic injuries. Management of these injuries is individualized.

C. Penetrating injuries to the neck and torso may result in fractures of the spine or penetration of the spinal canal. While these injuries are often stable, indications for surgery include deterioration of neurologic status, spinal compression or cauda equina syndrome, as well as complications related to presence of a foreign body.

VI. OTHER RELEVANT TOPICS IN SPINAL INJURY MANAGEMENT

A. Hemodynamic Lability. Patients sustaining spinal cord injury may be prone to hypotension due to disruption of sympathetic tone. They should be managed in a suitably resourced environment (i.e., ICU or stepdown unit) for purposes of blood pressure monitoring.

B. Steroids. The practice of early use of high-dose corticosteroids in the setting of acute spinal cord injury is not supported by current evidence (*Neurosurgery.* 2013;60 (Suppl 1):82-91).

C. DVT Prophylaxis. Acute spinal cord injury patients are at an increased risk for DVT. These patients should have mechanical and chemical prophylaxis initiated as early as possible within the first 72 hours. They continue to be at an increased risk of developing a DVT during the 3 months following their injury, so it is recommended that they are treated with chemical DVT for that time. Patients who fail chemical anticoagulation or who are not candidates for chemical anticoagulation should be considered for inferior vena cava filters.

CHAPTER 10: HEAD, NECK, AND SPINAL TRAUMA

Multiple Choice Questions

1. A 27-year-old male helmeted motorcycle driver is involved in a collision with a motor vehicle. EMS reports emesis at the scene. The patient has a GCS of 14 on arrival. His vital signs are a heart rate of 75 with a systolic blood pressure of 135. The only significant finding on examination is an area of significant redness behind his ears bilaterally. What is the next best course in management?

a. Observe in ED for 4 hours and discharge if stable

b. Admit for observation

c. Perform a noncontrast CT scan of the head

d. Perform a CTA of the head

e. Perform a CTA of the neck

View Answer

2. A 60-year-old female presents after a motor vehicle crash. The patient was unresponsive at the scene and was intubated by the EMS team. Her vital signs include a heart rate of 89 and a systolic blood pressure of 150. Initial primary and secondary assessments were unremarkable and a FAST examination was negative. The patient is imaged and upon return from the scanner is observed to have a new finding of a blown right pupil. What is the next most appropriate course in management?

a. Observe the patient for further changes

b. Consult neurosurgery for an ICP bolt

c. Call radiology for the results of the head CT scan

d. Elevate the head of the bed, hyperventilate, and order a bolus of Mannitol

e. Give steroids

View Answer

3. An outside hospital transfer presents to your emergency room. The patient is a 35-year-old male who fell off the roof of a moving vehicle. The patient is intubated and sedated with a GCS = 3T and a c-collar in place. Examination shows a scalp degloving injury and clear discharge from his nostrils. A FAST examination was negative. His vital signs include a HR of 65 and an SBP of 120. The outside hospital head and c-spine CT scans show a basilar skull fracture, facial fractures, and a C2 burst fracture. The chest x-ray shows a miniscule right-sided pneumothorax. Prior to transfer to the ICU, what other procedure is indicated?

a. X-ray of the T and L spine

b. Irrigation and closure of his wounds

c. CTA of the neck

d. Insert chest tube

e. Repeat NCCT of the head and c-spine

View Answer

4. A 66-year-old intoxicated male presents to your emergency department after a motor vehicle crash. The patient is not cooperative and vehemently denies any cervical spine tenderness. What is the appropriate next step to minimize the potential for cervical spine

injury?

a. Reassess the patient when he is sober

b. Place a cervical collar on the patient

c. Obtain a plain x-ray of the c-spine

d. Obtain a c-spine CT

e. Obtain a c-spine MRI

View Answer

5. A 54-year-old male presents with a gunshot wound to his neck, which occurred during an attempted robbery. The patient's voice is inaudible and he is noted to have a significant hematoma of his neck. His blood pressure is 60/palp and his HR is 125. What is the appropriate next course of action?

a. CTA of the neck

b. Bronchoscopy

c. Endoscopy

d. Secure airway and attempt tamponade

e. Transfuse blood products

View Answer

11
Chest Trauma

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Although approximately 75% of patients with thoracic traumatic injuries can be managed expectantly, significant injury to the heart, lungs, great vessels, and other mediastinal structures may result from both penetrating and blunt trauma. Injury to the thoracic cavity results in approximately 12 injured persons per one million population per day, with one-third of these patients requiring inpatient management (http://www.cdc.gov/nchs/fastats/accidental-injury.html). Thoracic injury is a contributing factor in as many as 75% of all trauma-related deaths.

Every hemodynamically stable patient with suspected thoracic trauma should undergo plain chest radiography following the ATLS primary survey. Based on the results of the initial imaging and laboratory studies, as well as the overall ongoing stability of the patient, further workup and management can be directed from an algorithmic approach (Fig. 11-1). Knowledge of the mechanism of injury, as well as the patterns of injury that typically accompany both penetrating and blunt chest trauma, is useful in directing further diagnostics and management.

I. PENETRATING THORACIC INJURIES.

As many as 40% of all penetrating injuries involve the thorax. These occur most frequently from gunshot wounds and stabbings, and represent 10% of all major traumas in the United States (*J Trauma.* 1990;30(11):1356-1365). Although less common than blunt thoracic trauma, penetrating trauma to the chest is more deadly, with 15% to 30% of all penetrating injuries to the chest requiring thoracotomy.

A. Chest Wall Injuries. Penetrating injuries to the chest wall are often less significant than those seen in blunt thoracic trauma. Low-velocity injuries, such as those suffered after a stab wound to the chest, may include intercostal artery laceration requiring ligation, or fracture of a single rib. High-velocity penetrating trauma, such as that seen with a shotgun blast to the chest, may include more significant chest wall injury, such as soft tissue loss and multiple rib fractures affecting subsequent ventilation and oxygenation. In these patients, pain control, local wound care, and aggressive support of pulmonary mechanics comprise the majority of treatment.

B. Open Pneumothorax. An open pneumothorax (Òsucking chest woundÓ) may develop with more significant chest wall trauma, in which a soft tissue defect ³two-thirds the circumference of

the trachea is present. In this condition, air is entrained into the pleural space preferentially through the chest wall when negative intrathoracic pressure is generated during inspiration. This occurs because the chest wall defect provides less resistance to flow than the trachea itself, creating a wound that appears to be Òsucking airÓ into the chest, and may be visibly bubbling. Management includes supplemental oxygen and intubation when oxygenation or ventilation is inadequate; however, definitive treatment via chest tube placement and closure of the wound should not be delayed. If definitive operative repair is not an immediate option, an occlusive dressing taped on three sides provides a temporizing flap-valve effect in which air escapes from the pleural space during expiration, but does not enter during inspiration.

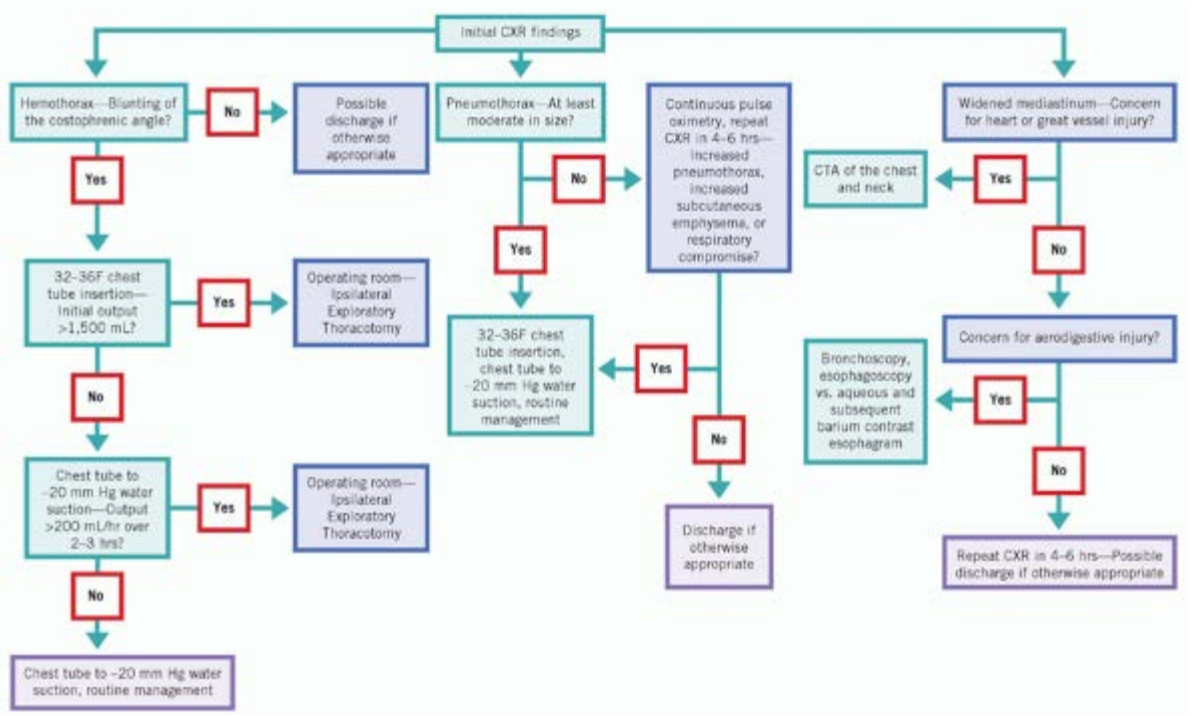


Figure 11-1 An algorithmic approach to a stable patient with chest trauma based on initial chest x-ray findings.

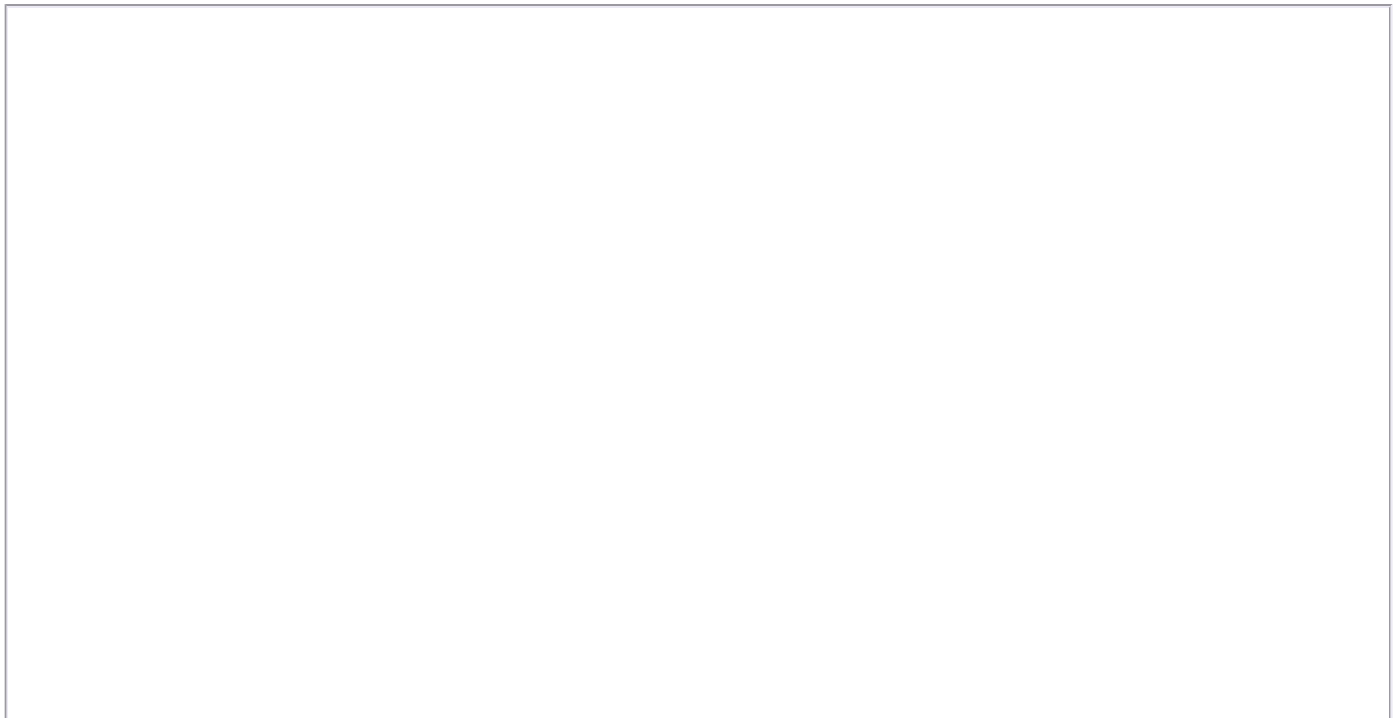
C. Lung Injuries. Injury to the lungs occurs in 65% to 90% of all penetrating trauma to the chest. As a result of pulmonary laceration, pneumothorax, hemothorax, and pulmonary contusion may develop in the injured patient. Concomitant injury to other thoracic structures may also occur at the time of penetrating lung injury, and this demands a high level of suspicion. Although rare, penetrating tracheobronchial wounds are associated with concurrent esophageal and major vascular injuries in approximately 30% of cases.

1. Pneumothorax and hemothorax. It should be assumed that a traumatic pneumothorax has

a component of hemothorax. The initial management of a hemopneumothorax diagnosed by chest x-ray in the stable patient includes drainage via a 32- to 36-French tube thoracostomy. Subsequent need for thoracotomy is determined via an algorithmic approach (Fig. 11-1). Pulmonary contusions occur secondary to early parenchymal hemorrhage and subsequent tissue edema along an injury tract. Physiologic complications typically peak 24 hours postinjury and frequently involve difficulty with oxygenation and ventilation requiring aggressive maintenance of pulmonary mechanics and, at times, mechanical ventilation.

2. Tension pneumothorax. In the clinically unstable patient with chest trauma, a high degree of suspicion must be maintained for tension physiology. Following pulmonary laceration, a progressive accumulation of air in the pleural cavity may pressurize the space such that the mediastinum deviates to the opposite hemithorax, thereby obstructing venous return to the heart. Although easily recognizable on chest x-ray (Fig. 11-2), tension pneumothorax is a clinical diagnosis marked by several clinical signs that may be easily missed in the trauma bay (Table 11-1). Without rapid diagnosis and treatment, these early signs are followed by obstructive shock, marked by precipitous circulatory collapse and subsequent traumatic arrest with pulseless electrical activity. As such, obvious chest trauma accompanied by an absence of breath sounds on examination of a hemithorax, tachycardia, and hypotension refractory to fluid resuscitation merits immediate decompression. In the field, this may be accomplished with a large-bore angiocatheter inserted in the second intercostal space at the midclavicular line. Subsequent ipsilateral tube thoracostomy provides definitive decompression and directs further management (Fig. 11-1).

3. Late complications. Late complications of penetrating injury to the lung may include retained hemothorax and empyema, intrapulmonary abscess, and bronchopleural fistula, and may be amenable to treatment with Video-Assisted Thoracoscopic Surgery (VATS) as discussed below.



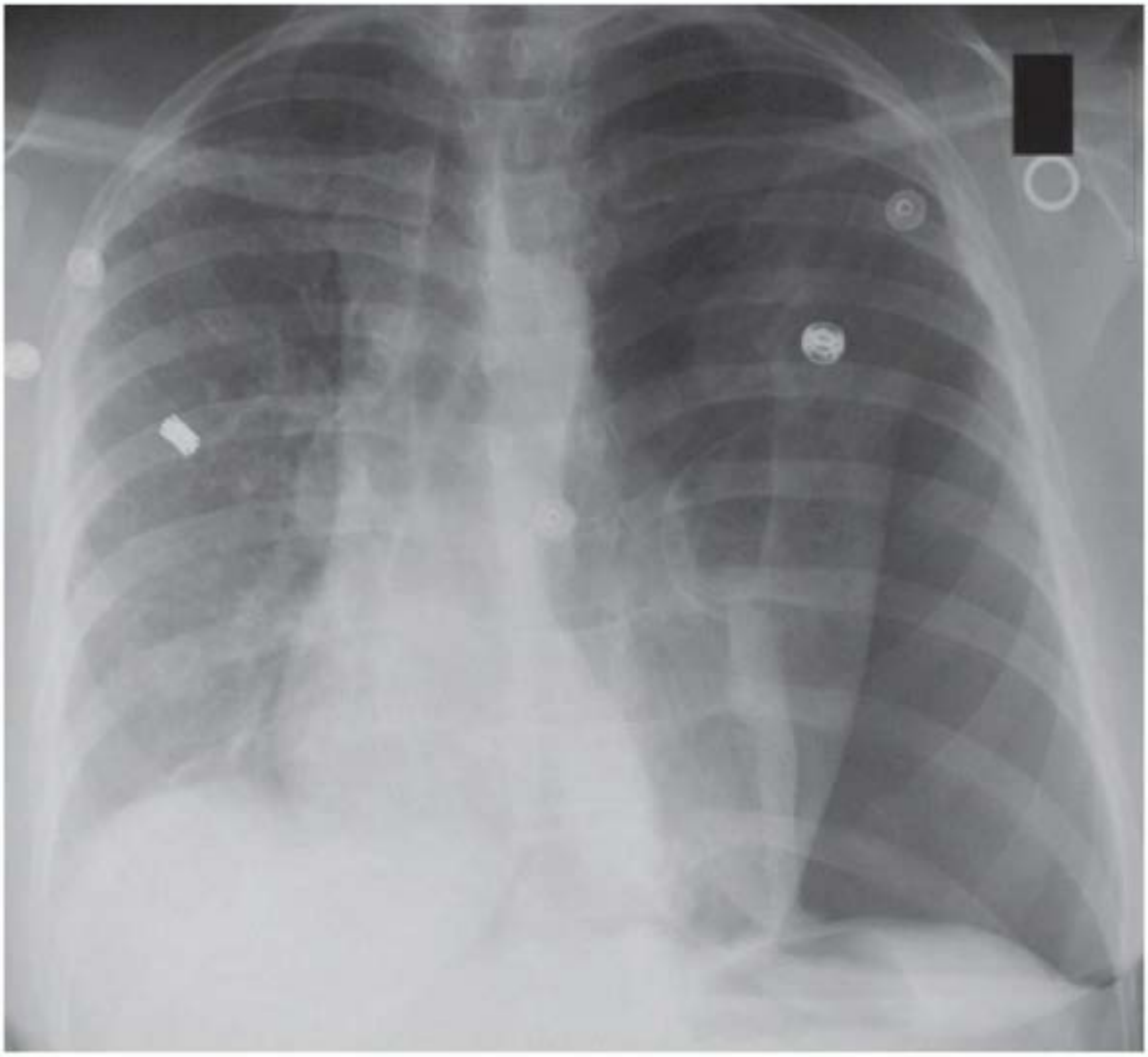


Figure 11-2 Plain chest x-ray of a patient with tension pneumothorax demonstrates deviation of the trachea and shift of the mediastinum away from the side of the tension. Depression of the ipsilateral hemi-diaphragm is also noted. (Photo courtesy of open access clinicalcases.org CC-BY-SA-2.5)

TABLE 11-1 Clinical Signs of Tension Pneumothorax

Tachycardia

Tachypnea

Hypoxia

Hypotension refractory to fluid resuscitation

Hyper-expanded chest

Increased percussion note

Decreased chest excursion with respiration

Increased CVP[a]

Increased airway pressure

[a] May be normal or low in hypovolemic states.

D. Heart and Great Vessels. Major injuries to the heart and great vessels occur in approximately 4% of patients with penetrating chest injuries, and are associated with high mortality. These injuries are rarely encountered as part of a trauma activation, as over 80% of these patients expire in the field. However, of those that survive transport to a Level I trauma center, one large series has demonstrated better than 25% survival (*Arch Surg.* 2011;146(9):1061-1066). Overall, gunshot wounds to the heart are more universally fatal, with an overall mortality of greater than 90%. In contrast, stab wounds to the heart are said to carry an overall 67% mortality.

1. Pathophysiology of cardiac trauma. Although cardiac injury is usually associated with penetrating anterior chest trauma between the midclavicular lines, it can occur in the setting of penetrating trauma outside of these anatomical landmarks as well. Due to its anterior position in the chest cavity, the right ventricle is the most commonly injured chamber in penetrating trauma, followed by the left ventricle. Atrial injuries are both less common and less severe, although they cause higher overall mortality when involved in a multi-chamber injury. These injuries can cause both obstructive and hemorrhagic shock, depending on the integrity of the pericardial sac. In the patient with distended neck veins and diminished heart sounds presenting in shock (Beck's triad), pericardial tamponade should be suspected. Due to the poor compliance of the pericardium, the acute accumulation of as little as 50 mL of blood can cause tamponade physiology. In the case of coronary artery disruption, cardiogenic shock can further complicate management.

2. Diagnosis. In the hemodynamically stable patient with suspicion for an occult penetrating cardiac injury, transesophageal echocardiography is the diagnostic modality of choice. However, many of these injuries can also be identified with transthoracic echocardiography as part of the Focused Assessment with Sonography for Trauma (FAST). The presence of pericardial fluid on echocardiography warrants emergent operative exploration. Another diagnostic and potentially therapeutic 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 diaphragm is exposed via a subxiphoid approach, and a longitudinal incision is made to expose the pericardium. A 1-cm longitudinal pericardial incision is then made under direct vision. The presence of straw-colored fluid within the pericardium constitutes a negative examination. Blood within the pericardium mandates definitive exploration and cardiorrhaphy.

3. Treatment. In hemodynamically unstable patients with suspected penetrating injury to the mediastinum, the preferred operative approach is via median sternotomy. This approach allows access to the proximal aorta; superior vena cava; right subclavian, innominate, and carotid arteries; and heart. If injury to the left subclavian artery is identified or suspected, a supraclavicular (ÒtrapdoorÓ) extension off the median sternotomy is often required. A left anterolateral thoracotomy is another option for management of an injury to the left subclavian artery. Atrial

and ventricular cardiac wounds are repaired primarily using interrupted or running monofilament sutures. Either skin staples or a Foley catheter inserted into the wound with subsequent inflation of the balloon may be used as temporizing measures until definitive management can be performed. Care must be taken to avoid injury to coronary arteries during the repair. Wounds adjacent to major branches of the coronary circulation require horizontal mattress sutures placed beneath the artery (Fig. 11-3). Distal coronary artery branches may be ligated. Early consultation with a cardiothoracic surgeon is essential, especially in cases involving complex repairs or cardiopulmonary bypass. Patients in extremis may require resuscitative thoracotomy in the emergency department as a life-saving diagnostic and therapeutic measure, as is discussed below.

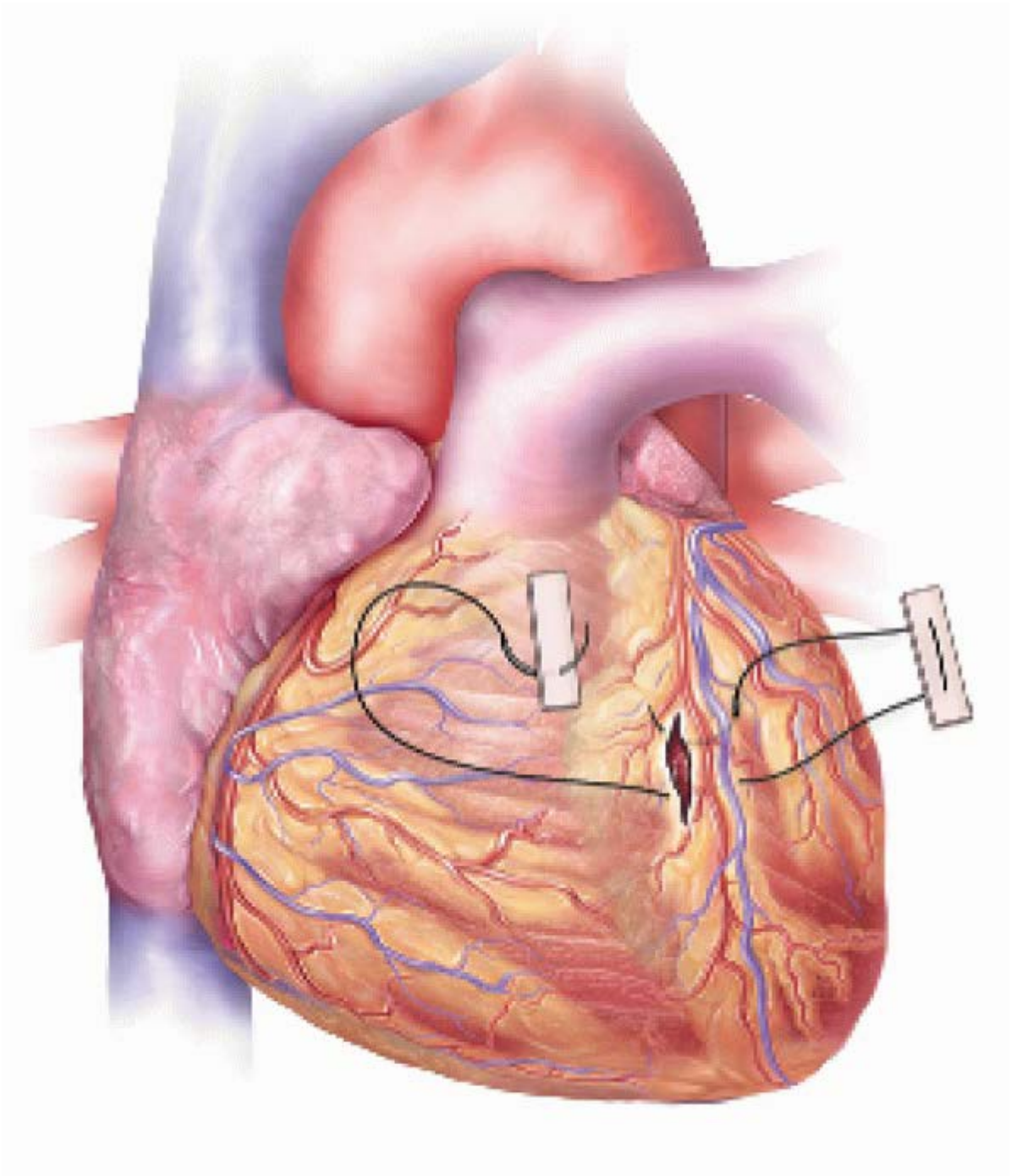


Figure 11-3 Horizontal mattress repair of a penetrating cardiac injury. Monofilament suture closure of the wound passing underneath the coronary artery avoids subsequent myocardial ischemia.

E. Diaphragm. Injuries to the diaphragm may be difficult to diagnose, as up to 31% of patients may demonstrate no abdominal tenderness and 40% may have normal chest radiographs (*J Trauma.* 2003;55(4):646-650). Among all asymptomatic patients with penetrating chest injuries, the risk of occult diaphragm injury is reported to be 7%. When undiagnosed, diaphragmatic injury is associated with a high risk of bowel herniation. Please see the chapter on abdominal trauma in

this volume for further details regarding diaphragmatic injury.

F. Aerodigestive System. Although the tracheobronchial tree and the esophagus are occasionally injured when the chest sustains blunt trauma, they are more commonly injured in the case of penetrating trauma. Aerodigestive injuries are often accompanied by subcutaneous emphysema along with visible air and fluid on chest x-ray. As with many other mediastinal injuries, a high index of suspicion is necessary to ensure the diagnosis.

1. If tracheobronchial injury is suspected, control of an unstable airway is first priority. Once the airway is stabilized, fiberoptic bronchoscopy may be necessary in order to evaluate the tracheobronchial tree for

injury. Consultation with a thoracic surgeon may be required if injury exists. 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 debrided and repaired primarily. Transections resulting from blunt injuries usually require debridement 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.

2. Suspicion of **esophageal injury** requires prompt attention, as well, as secondary mediastinitis is wrought with very high mortality. Esophagoscopy can be performed to evaluate for mucosal defect. In addition, esophagography with an aqueous contrast followed by barium can often rule out esophageal injury. As in the case of tracheobronchial injuries, the operative approach for esophageal injuries 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, and complex flap closure. Esophageal resection is reserved for the injured esophagus with underlying pathology and is associated with a high rate of morbidity and mortality.

II. BLUNT THORACIC INJURIES.

Blunt thoracic injury directly accounts for 20% to 25% of deaths resulting from trauma, representing over 16,000 deaths annually in the United States. Over 70% of these injuries are the result of motor vehicle collisions (MVCs). Blunt thoracic injuries are identified in 40% to 50% of all unrestrained drivers following MVC, and greater than 25% of drivers who die as a result of MVC have sustained blunt thoracic trauma.

A. Trauma Evaluation. Patients who enter the trauma bay following a blunt trauma with suspected thoracic injury should undergo ATLS with specific focus on the ABC's. Blunt trauma to the thoracic cage and its inhabitants can quickly lead to hemodynamic and respiratory compromise and can quickly lead to a spiraling decline in patient status. Chest x-ray is the first diagnostic tool utilized to evaluate the chest wall and thoracic cavity in the trauma bay. Computed tomography has become a valuable imaging modality, as well, as it can often be obtained quickly and easily. The use of computed tomography should be based on patient stability and mechanism and severity of injury. FAST examination is a helpful tool in the quick workup of the trauma patient, and the subxiphoid view can help diagnose traumatic pericardial effusion and tamponade. Also, as ultrasound is being

utilized more and more in the emergency setting, some series have shown that it can be used to help diagnose pneumothorax with even greater sensitivity than the traditional upright chest x-ray (http://www.trauma.org/archive/thoracic/CHESTflail.html).

B. Chest Wall Injuries. The chest wall is injured in 70% of blunt trauma to the chest. Injuries may include chest wall contusion and rib contusion or fracture. Occasionally, chest wall hematoma may develop secondary to disruption of subcutaneous and intramuscular blood vessels. **Flail chest** is one significant chest wall injury that occurs when a segment of the rib cage fractures under extreme stress and becomes detached from the rest of the chest wall. The mechanism of flail chest requires fracture of multiple adjacent ribs in multiple places, separating a segment so that a part of the chest wall moves independently. Underlying pulmonary contusion is extremely common in patients sustaining flail chest and can lead to significant pulmonary compromise. If a patient sustains multiple **rib fractures** with or without underlying **pulmonary contusion,** respiratory compromise can become an issue. Early placement of epidural catheters often help with early aggressive pulmonary hygiene and can help prevent subsequent complication such as respiratory insufficiency/failure and pneumonia. This especially holds true in the elderly and debilitated patient. Most trauma surgeons advocate for aggressive early placement of epidural catheters in patients with significant chest wall trauma without any contraindications. Occasionally, flail chest can result in significant pulmonary compromise and may require surgical intervention. Recent development of a technique for surgical internal fixation of broken ribs is showing promising results and helps patients liberate from the ventilator and recover more quickly from significant chest wall trauma. The exact indications for rib fixation are still unclear, but it seems that some patients with significant chest wall and respiratory compromise certainly benefit from the procedure (*Scand J Surg.* 2014;103(2):120-125). Occasionally, other bony structures of the thoracic cage are injured or fractured during blunt trauma. These include the sternum, clavicles, and scapulae and may require surgical repair if damage is significant enough.

C. Lung Injuries. As in penetrating injuries to the chest, pneumothorax and hemothorax may be seen in blunt thoracic trauma. In the case of blunt trauma, however, damage to the lung with

subsequent hemopneumothorax is often the result of broken ribs causing laceration to the lung parenchyma or intercostal arteries. Rarely, acutely increased intrathoracic pressure can lead to bleb rupture and pneumothorax in patients with pre-existing emphysematous disease. The lung is injured in just over 20% of blunt trauma and the principles remain the same as in penetrating trauma to the chest. The treatment for hemopneumothorax caused by blunt trauma is the same as that caused by penetrating trauma and the potential later complications are the same as well (see Section I.C.1).

D. Blunt Cardiac Injury (BCI). The heart is injured in roughly 7% of blunt thoracic traumas. BCI accounts for up to 20% of deaths from MVC. BCI may be seen in as many as 75% of trauma patients who sustain multiple

injuries or severe thoracic injuries. Findings that would lead a clinician to be concerned for BCI include midanterior chest pain and/or tenderness, fractured sternum, mechanism of major blunt force to the chest, and ongoing signs or symptoms of cardiac disease.

1. Diagnosis. Due to its anterior position in the mediastinum deep to the sternum, the right side of the heart (right ventricle and atrium) is injured more frequently in blunt trauma. A complete understanding of injury mechanism along with a high index of suspicion is necessary for the diagnosis of BCI. Following routine chest x-ray, the first diagnostic test that should be performed to evaluate a patient for BCI is an electrocardiogram. Although there is not a pathognomonic rhythm associated with BCI, electrical abnormalities including arrhythmia, bundle branch block, ST changes, and even unexplained tachycardia should prompt further investigation including cardiac monitoring and echocardiography. While previous recommendations did not include the measurement of cardiac enzymes to aid in the evaluation of BCI, recent additions to the Eastern Association for the Surgery of Trauma (EAST) guidelines suggest their utility (see below and Table 11-2).

2. Classification. The American Association for the Surgery of Trauma (AAST) grades cardiac injuries I to VI with Grade VI injuries being

the most severe. They also break down BCI into more comprehensive categories. These include pericardial injury which results from high pressure rupture of the pericardium and leads to cardiac evisceration and torsion of the great vessels. This rupture usually occurs parallel to the phrenic nerves. Valvular injuries (most commonly the aortic and mitral) are rare, but can lead to left ventricular dysfunction and cardiogenic shock or pulmonary edema. New murmurs in the setting of blunt chest trauma should prompt investigation for an injured valve. Very rarely, direct impact to a coronary artery may lead to thrombus formation with subsequent myocardial ischemia. BCI may lead to cardiac chamber rupture which is a rare, but often fatal, phenomenon. Survivors in the field often present to the ED in obstructive shock from cardiac tamponade, or hypovolemic shock from significant bleeding. Myocardial contusion is likely the most common manifestation of BCI and includes direct injury to the cardiac muscle itself. This injury to the heart wall may be

accompanied by elevated cardiac enzymes and/or EKG changes.

TABLE 11-2 East Guidelines for the Evaluation of Blunt Cardiac Injury (BCI)

Level I

Admission EKG should be obtained in all patients where there is suspected BCI

Level II

1. If admission EKG is abnormal, the patient should be admitted for continuous EKG monitoring for 24-48 hrs. If admission EKG and troponin I value are normal, BCI is ruled out and further pursuit of diagnosis should be abandoned. However, the optimal timing of obtaining cardiac enzyme levels is yet to be determined, and a patient with normal EKG but elevated troponin should be kept for observation/monitoring.

2. If the patient is hemodynamically unstable, an imaging study such as TTE or TEE should be obtained.

3. Nuclear medicine scans add little compared with echocardiography and are not useful if echocardiography has been performed.

Level III

1. Elderly patients with known cardiac disease, unstable patients, and those with abnormal admission EKGs can be safely operated on provided that they are closely monitored.

2. The presence of a sternal fracture does not predict the presence of BCI and does not necessarily indicate that monitoring should be performed.

3. Neither CPK analysis nor measurement of circulating cardiac troponin T are useful in predicting which patients have or will have complication related to BCI.

3. Treatment. Due to the significant morbidity and relative frequency associated with this injury, the EAST has developed evidence-based guidelines for the evaluation and treatment of myocardial contusion in BCI (Table 11-2).

E. Aortic Injury. Injury to the thoracic aorta occurs in roughly 5% of blunt chest trauma. While relatively uncommon, injury to the aorta carries a tremendous mortality. Roughly 80% to 90% of patients who sustain a ruptured thoracic aorta die before reaching the hospital and up to 15% of deaths following MVC can be attributed to aortic injury. Anatomically, the area of injury is most commonly at the level of the takeoff of the left subclavian artery, where the aorta is fixed at the ligamentum arteriosum.

1. Diagnosis and management. Widened mediastinum on CXR, bright red blood return from the thoracostomy tube, and hemodynamic instability should heighten one's suspicion for aortic injury. The EAST guidelines state that blunt aortic injury is often lethal and should be considered in severe trauma with acceleration/deceleration forces (*J Trauma.* 2000;48(6):1128-1143). Radiographic findings suggestive of mediastinal injury warrant further investigation with CT or angiography. CT angiography, using modern technology, carries a 97% to 100% sensitivity for diagnosing aortic injury, and approaches the diagnostic capabilities of the traditional gold standard of formal angiography. Prompt repair should be the goal along with maintenance of distal perfusion to minimize spinal cord and renal ischemia. Strict blood pressure control is imperative when prompt repair is not feasible.

Patients who survive long enough to make it to the trauma bay are likely suffering from a partial tear which is best served by aggressive blood pressure control and avoidance of over-resuscitation. Quick evaluation and identification of other injuries and other sources of bleeding, followed by rapid transport to the operating room for definitive repair is crucial in the treatment of patients with aortic injury. It is important to note, however, that patients who manage to survive

their injury long enough to present to the trauma bay are likely stable enough from the aortic injury standpoint to allow time for exploration and repair of other injuries. In other words, if a patient presents with intraabdominal hemorrhage and an aortic injury, the abdominal injury should be addressed prior to definitive aortic repair. Often, in centers with particular expertise, endovascular repair of aortic injury can follow exploratory laparotomy to definitively address intra-abdominal injuries.

III. PROCEDURES

A. VATS is a minimally invasive surgical technique similar to laparoscopy of the abdomen. One or more port sites are created in the chest wall and operations in the thoracic cavity are conducted with the aid of a camera. VATS is used by trauma surgeons to surgically manage retained hemothorax, persistent air leak,

and empyema. Recent data suggests that the early use of VATS (3 to 7 days postinjury) to address these problems in both penetrating and blunt trauma leads to fewer complications and decreased length of hospital stay (*J Trauma.* 2011;70(2):510-518).

B. Resuscitative Thoracotomy. Resuscitative thoracotomy is performed in 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 resuscitative thoracotomy have been refined over time. 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. In addition, it can be used in certain cases of penetrating abdominal trauma fulfilling the same criteria.

2. Technique. Resuscitative thoracotomy is performed via a left anterolateral 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 vertically 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. 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 atrium can be performed. In severely hypovolemic patients, the descending thoracic aorta may be exposed and crossclamped 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. Complications of resuscitative thoracotomy are many, and may include lung injury while gaining access to the heart, transection of the

phrenic nerve while performing pericardotomy, injury to the coronary vessels during cardiorrhaphy, and esophageal trauma while clamping the descending thoracic aorta. Therefore, care must be taken during each step of the procedure to avoid causing additional injuries. In addition, a member of the trauma team sustains a needle-stick or other sharp injury in roughly 10% of resuscitative thoracotomies performed in the emergency department. As this procedure is most commonly performed in a patient population at high risk of carrying blood-borne disease, the risk to the trauma team is not insubstantial and must be considered.

IV. CONCLUSION.

Injury to the chest and its contents represents a significant portion of the trauma spectrum. Aggressive attention to mechanism and a high index of suspicion for injury are crucial in the evaluation and treatment of the traumatically injured patient. The majority of thoracic trauma can be managed expectantly or with minimal therapy including thoracostomy tube placement, but certain substantial life-threatening injuries require early and accurate diagnosis and treatment.

CHAPTER 11: CHEST TRAUMA

Multiple Choice Questions

1. A 34-year-old male is brought to the emergency department after sustaining a GSW to the right chest. Upon arrival, his HR is 125 and SBP is 80. His trachea is deviated to the left and breath sounds on the right are absent. He is awake and agitated. Which of the following is the first step in management?

a. Left-sided thoracotomy

b. Right-sided thoracotomy

c. CT scan of the chest and abdomen

d. Needle decompression of the right chest

e. Endotracheal intubation via direct laryngoscopy

View Answer

2. In the above patient, a right-sided thoracostomy tube is inserted. Which of the following resultant findings would prompt an immediate trip to the operating room?

a. 500 mL initial output of blood or 100 mL output of blood over the

following 4 hours

b. 1,500 mL initial output of blood or 200 mL/hour output over the following 2 to 3 hours

c. Bubbling in the water seal chamber consistent with an air leak

d. 2 L total output over the following 24 hours

e. A rush of air upon making the thoracostomy tube incision in the chest

View Answer

3. A 25-year-old female is brought to the emergency department after suffering a stab wound just to the left of the sternum. Upon arrival, she becomes pulseless. Which of the following is a component of a resuscitative thoracotomy?

a. Right thoracotomy in the fifth intercostal space

b. Ultrasound of the left chest

c. Incision in the pericardium anterior to the phrenic nerves

d. Cross-clamp of the abdominal aorta

e. Pericardial window with incision inferior to the xyphoid process

View Answer

4. A 50-year-old woman is involved in an MVC and strikes her chest on the steering wheel. Which of the following should be ordered to evaluate for blunt cardiac injury (BCI)?

a. Electrocardiogram

b. CT angiogram of the aortic arch

c. Sestamibi scan of the heart

d. Chest x-ray

e. Dobutamine stress test

View Answer

5. On primary survey, the patient above is noted to have crepitus of her neck and upper chest. Which of the following will help best determine whether she has an aerodigestive injury?

a. Abdominal CT

b. Chest x-ray

c. Esophagram with water-soluble contrast

d. FAST examination

e. Plain x-ray of the neck and C-spine

View Answer

6. Tension pneumothorax and cardiac tamponade are examples of which of the following?

a. Cardiogenic shock

b. Distributive shock

c. Hemorrhagic shock

d. Obstructive shock

e. Neurogenic shock

View Answer

12

Abdominal Trauma

Paul M. Evans

Douglas J. Schuerer

I. GENERAL APPROACH TO ABDOMINAL TRAUMA IN THE TRAUMA BAY.

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. The mechanism of injury often provides important clues to the potential organs injured and dictates further workup.

A. Stab Wounds. Only one-third of stab wounds to the anterior abdomen penetrate the peritoneal cavity and cause significant injury. Options to evaluate for injuries include local wound exploration, laparoscopy or celiotomy, Computed Tomography (CT), Focused Abdominal Sonography for Trauma (FAST), diagnostic peritoneal lavage (DPL), and admission with observation.

B. Gunshot Wounds (GSWs). GSWs within the surface markings of the abdomen have a high probability of causing a significant intra-abdominal injury and therefore traditionally require immediate celiotomy, but this imperative has been challenged for those patients with stable hemodynamics and no peritoneal signs on physical examination. In a large retrospective study of patients with abdominal GSWs, selective nonoperative management was reported to result in a significant decrease in the percentage of unnecessary laparotomies (*Ann Surg.* 2001;234:395). Current recommendations for nonoperative management of penetrating trauma include the use of triple-contrast CT (which accurately predicts the need for laparotomy) and serial examination. The majority of these patients can be discharged after 24 hours of observation (EAST guidelines 2007).

C. 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 intra-abdominal injury.

1. In the awake, unimpaired patient without abdominal complaints, combining hospital admission and serial abdominal examinations is a costeffective strategy for excluding 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. An immediate celiotomy is required for an **unstable patient with injuries confined to the**

abdomen.

3. If an **unstable patient has multiple injuries** and there is uncertainty about whether the abdomen is the source of shock, a FAST examination may be useful. If a patient is fairly stable and access to CT is readily

available, head and abdomen/pelvis CT scans can be obtained. DPL may be useful in patients with head injuries requiring immediate operative therapy. In many large centers, a CT scan can be obtained as readily as the performance of a DPL.

4. If a **stable patient has multiple injuries** and the abdomen may harbor occult organ involvement that is not immediately life threatening, a CT evaluation is necessary. In addition to identifying the presence of intra-abdominal injury, CT scanning can provide information helpful for determining the probability that a celiotomy will be therapeutic. Laparoscopy has also been proposed as an adjunct in this situation.

II. IMAGING MODALITIES

A. Trauma Ultrasonography. Many trauma centers now use FAST as an initial radiographic screening evaluation for all traumas following the primary survey. 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) Morrison pouch, (3) pericardium, (4) perisplenic region, (5) left paracolic gutter, and (6) suprapubic region. 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. However, if a FAST examination is negative, it does not exclude major intra-abdominal injury.

B. CT Scanning. The care of injured patients has been significantly changed by the use of CT scanning. Unnecessary laparotomy is associated with significant morbidity and cost. Because of CT, an increasing amount of both blunt and penetrating trauma has been safely managed nonoperatively. Although triple-contrast CT (oral, IV, rectal) has been traditionally used, more recent evidence suggests that single-contrast CT scanning with a high-resolution, multi-slice scanner may obviate the need for oral and rectal contrast.

III. TRAUMA RESUSCITATION AND OPERATIVE MANAGEMENT.

Trauma patients should immediately have two large-bore IV lines placed (14 or 16 gauge), with the antecubital veins being the preferred sites. Laboratory samples can be efficiently sent at the time of initial venous access, the most important of which is a Type and Screen. All hypotensive trauma patients should be assumed to be in hemorrhagic shock until proven otherwise (Table 12-1), and should be resuscitated with blood products as soon as possible, preferably in a 1:1:1 ratio

of pRBC:FFP:Platelets. A Massive Transfusion Protocol (MTP) should be in place at each institution and activated in this setting. Crystalloid, colloid, and hypertonic saline should be avoided. Permissive hypotension and balanced resuscitation prior to definitive control of bleeding reduces overall transfusion requirements and coagulopathy (*J Trauma.* 2011;70:652-663). However, the target blood pressure should be tailored to the patient's age and medical history, as elderly patients may not tolerate the same degree of hypotension as younger patients.

TABLE 12-1 Estimated Blood Loss by Initial Hemodynamic Variables

	Class I	Class II	Class III	Class IV
Blood loss (mL)	Up to 750	750-1,500	1,500-2,000	>2,000
Blood loss (% blood volume)	Up to 15%	15-30%	30-40%	>40%
Pulse rate	<100	>100	>120	>140
Blood pressure (mm Hg)	Normal	Normal	Decreased	Decreased
Pulse pressure (mm Hg)	Normal or increased	Decreased	Decreased	Decreased
Urinary output (mL/hr)	>30	20-30	5-15	Negligible

Successful operative management of trauma in a hemodynamically unstable patient requires a team approach, with close communication between the attending surgeon, surgical assistants, anesthesiologist, circulating nurse, and surgical technologist. The surgeon should clearly communicate the plan to rest of the team. Close communication with the anesthesiologist about the hemodynamic status of the patient is important to determine if the operation should be

temporarily interrupted (damage control laparotomy).

Once in the operating room, the patient should be placed in a supine position with both arms outstretched at 90 degrees. A Foley catheter and gastric tube should be placed, and the patient should be prepped and draped widely from the chin to both knees. A single dose of a broad spectrum antibiotic with aerobic and anaerobic coverage (such as cefoxitin) should be given, keeping in mind that the initial dosage may need to be increased and repeated after transfusion of 10 units of blood products (EAST guidelines 2012).

Initial access to the intra-abdominal cavity should begin with a generous midline incision. The small bowel should be eviscerated and all four quadrants should be rapidly packed off with laparotomy pads. Management of individual injuries will be discussed in the next section.

IV. MANAGEMENT OF SPECIFIC INJURIES

A. 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 intra-abdominal pressure. Frequently, diagnosis is made during celiotomy, but injury can occasionally be recognized on radiographic studies (e.g., chest x-ray or CT). Therapy entails primary repair using permanent sutures in a horizontal mattress fashion. Immediate

repair prevents the long-term complications associated with diaphragmatic hernias.

B. Abdominal esophageal injuries are managed much like thoracic esophageal injuries (see Chapter 11). In addition to primary repair and drain placement, the fundus of the stomach can be used to buttress the site via a partial wrap (e.g., a Dor fundoplication).

C. Gastric Injuries. Injuries to the stomach occur most often in the setting of penetrating trauma. Bloody drainage from a gastric tube should raise the possibility of gastric injury. Diagnosis is usually made at laparotomy. Simple lacerations can be repaired in one layer using a synthetic absorbable suture. Massive devitalization may require formal resection with restoration of GI continuity via gastroenterostomy. In such cases, vagotomy is helpful in reducing the risk of a marginal ulcer.

D. Hepatic Injuries. The use of CT in blunt trauma has increased the diagnosis of occult liver injuries, making the liver the most commonly injured abdominal solid organ.

1. Penetrating trauma. Although some centers advocate nonoperative management for penetrating trauma isolated to the liver in a hemodynamically normal patient (*J Trauma.* 68:721-733), the diagnosis of penetrating hepatic injury is usually made at exploratory laparotomy. 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. Rapid mobilization of the injured lobe with perihepatic compression can often provide initial hemostasis. Complex injuries that are controlled with packing may be best managed with a damage control approach, including ICU admission and resuscitation, followed by return to the operating room in 24 to 48 hours.

Other methods to control hemorrhage include a Pringle maneuver, total vascular isolation, and atriocaval shunt. Definitive hemostasis can then obtained with a combination of cautery, chromic suture, topical hemostatic agents, finger fracture and ligation, and omental packing. 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 to identify and control biliary leaks.

2. Blunt trauma. CT with IV contrast is the recommended diagnostic modality for evaluation of the stable patient suspected of having blunt hepatic trauma. The unstable patient requires operative exploration and control of hemorrhage as described. The stable patient without an alternate indication for celiotomy should be admitted for close hemodynamic monitoring 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 evaluation and embolization of the bleeding source.

E. 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.

F. Common bile duct injuries are most often a result of penetrating trauma. 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.

G. 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.

1. 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 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.

2. Duodenal perforation can be difficult to diagnose. Patients often complain only of vague back or flank pain, and symptoms can evolve slowly. Plain 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 may also show evidence of a leak. The diagnostic modality of choice, however, is CT using oral and IV contrast, with the oral

contrast administered in the trauma room. 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.* 1979;19:334).

3. Complex duodenal injuries are an operative challenge, and management remains controversial, especially in the presence of tissue devitalization. Whenever possible, debridement with primary repair should be performed. The repair should be protected via triple-tube drainage or pyloric exclusion with diverting gastrojejunostomy. 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.

H. Pancreatic Injuries. Injuries to the pancreas often occur as a result of penetrating trauma. Isolated pancreatic trauma is rare and typically, the liver or stomach is also involved. CT is the best diagnostic imaging modality available, and, importantly, pancreatic enzymes are not helpful in the diagnosis. Treatment focuses on determining the presence and location of major ductal involvement. Adequate exploration entails performing a Kocher maneuver and transecting the gastrohepatic and gastrocolic ligaments to inspect the body and tail of the pancreas. Injuries in which the pancreatic duct is intact are treated with closed suction drainage. Transection of the pancreatic duct requires more extensive procedures involving debridement and/or resection combined with closed suction drainage of the pancreatic bed. Severe injury to the head of the pancreas, especially in conjunction with duodenal and biliary trauma, may require pancreaticoduodenectomy but usually not during the initial operation.

I. Splenic Injuries. The spleen is the second-most-common solid organ injured in abdominal trauma. An algorithm for management of splenic trauma is presented in Figure 12-1.

1. Penetrating trauma. Penetrating splenic injuries are usually diagnosed at laparotomy. 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 debrided and the wound closed with absorbable horizontal mattress sutures (usually 2-0 chromic). Alternatively, the spleen can be wrapped in an absorbable mesh. Partial resection is indicated for isolated superior or inferior pole injuries. In unstable patients or in patients for whom splenic salvage fails, splenectomy should be performed in an expeditious manner. All patients undergoing emergent splenectomy require postoperative immunization against *Streptococcus pneumoniae, Haemophilus influenzae,* and *Neisseria meningitidis.* It is recommended that vaccines be given 14 days following trauma splenectomy; however, vaccines should be given prior to discharge from the hospital if concerns exist that the patient will be lost to follow up.

2. Blunt trauma. 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 CT evidence of a contrast ÒblushÓ or evidence of continuing blood loss who remain stable should undergo embolization. Patients who are hemodynamically unstable or are failing nonoperative management (e.g., require continuing transfusion) should undergo operative exploration.

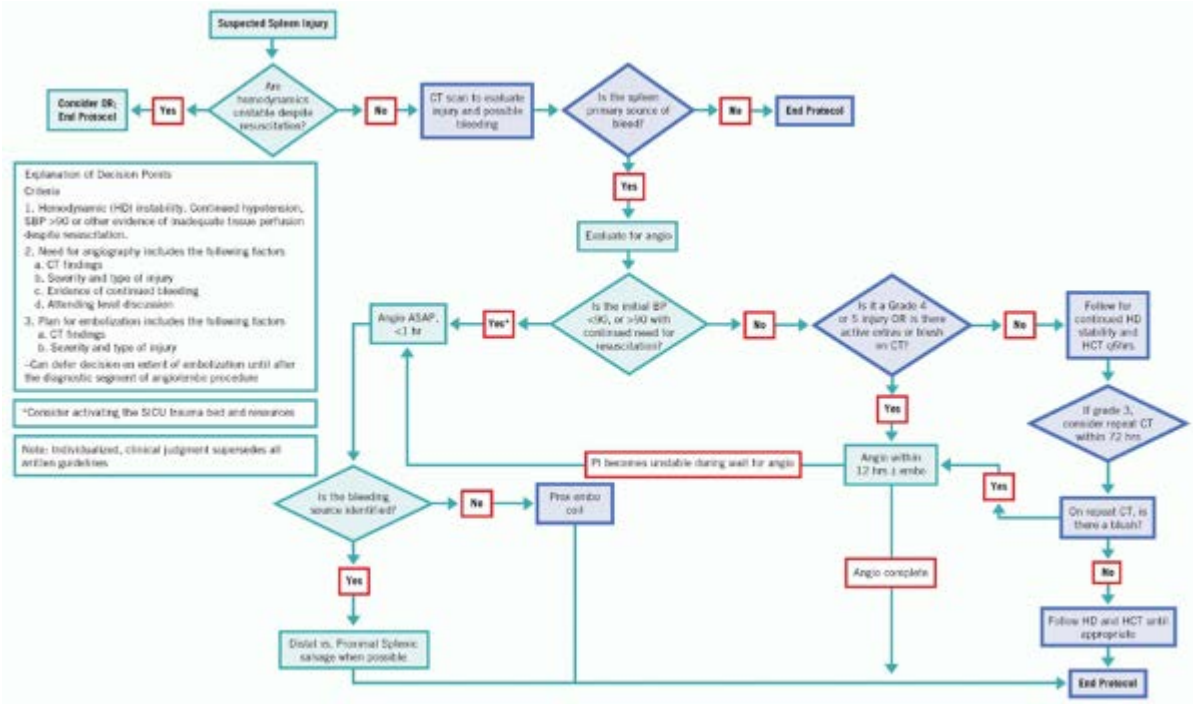


Figure 12-1 Splenic trauma algorithm.

J. Small-Bowel Injuries. The small bowel is prone to both penetrating and blunt trauma. Treatment consists of primary repair or segmental resection with anastomosis.

K. Large-Bowel Injuries. Most colonic injuries occur due to penetrating trauma and are diagnosed at the time of laparotomy. Primary repair, therefore, should be considered in all penetrating colonic injuries unless the patient experiences prolonged intraoperative hypotension (*J Trauma.* 2001;50:765).

L. Rectal Injuries. Penetrating trauma is also responsible for most rectal injuries and often occurs in association with genitourinary or pelvic vascular trauma. Primary repair of intraperitoneal rectal and accessible extraperitoneal injuries is indicated. Selective use of a diverting colostomy should be considered based on the hemodynamic status of the patient or in the setting of inaccessible injuries (*J Trauma.* 2006;60:508-514).

M. Retroperitoneal Vascular Injuries. Injuries to the major retroperitoneal vessels or their abdominal branches can be life-threatening. These wounds usually present with frank intra-abdominal hemorrhage or retroperitoneal hematoma formation. Management is based on both mechanism of trauma and location of injury.

1. Penetrating trauma. The majority of retroperitoneal vascular injuries are the result of penetrating trauma and all merit operative intervention. Options for initial control include occluding the supraceliac aorta at the level of the diaphragmatic hiatus using a vascular clamp, a T bar, or direct pressure. Division of the gastrohepatic ligament and 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 vascular injuries 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 (Cattell-Braasch maneuver) readily exposes the vena cava, right renal vessels, and iliac veins. The infrarenal aorta may also be approached via a transperitoneal incision at the base of the mesocolon. Most injuries can be repaired with simple suture repair or ligation.

2. Blunt trauma can cause retroperitoneal vascular injury with resultant hematoma. The location of the hematoma determines management.

a. Central abdominal hematomas (Zone I). All central abdominal hematomas caused by blunt trauma require operative exploration

as they are usually due to injury to the aorta, IVC, or their major branches.

b. 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 they are discovered at the time of celiotomy.

c. Pelvic hematomas (Zone III). Central pelvic hematomas in the setting of blunt trauma are

usually due to pelvic fractures. If they are discovered at celiotomy, they should not be explored unless iliac arterial injury is suspected (loss of ipsilateral groin pulse, rapidly expanding hematoma, or pulsatile hematoma) or rupture has occurred. Unstable pelvic fractures in association with hypotension should undergo some form of external stabilization (e.g., bed sheet, pelvic binder, or C-clamp). Formal external fixation should follow as soon as possible. If patient continues to be hemodynamically unstable, the patient should go to the operating room emergently for preperitoneal pelvic packing. If the patient responds to resuscitation, scanner CT scan should be performed. 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.

V. ABDOMINAL TRAUMA IN PREGNANCY.

Trauma is responsible for approximately 50% of all deaths in pregnant women, most commonly due to motor vehicle collisions, although both falls and abuse are major causes as well. A gravid uterus displaces the majority of the intra-abdominal organs, and thus relatively protects the mother from penetrating abdominal injury. In general, pregnant patients should be managed similarly to nonpregnant patients, following the dictum that the best way to take care of the fetus is to take care of the mother. Thus, concerns for fetal well-being should not preclude any urgent operative or radiologic investigations. For all pregnancies of 20 weeks of gestation or greater, or any unknown gestation, fetal heart monitoring and an urgent obstetrical consultation should be obtained. If possible, the mother should be placed in left lateral decubitus position to off-load pressure on the IVC and be given supplemental oxygen. Placental abruption is the most common cause of fetal demise and typically presents with vaginal bleeding. If the patient expires in the trauma bay or operating room and the fetus is at least 26 weeks of gestation, postmortem caesarean section should be performed expediently.

CHAPTER 12: ABDOMINAL TRAUMA

Multiple Choice Questions

1. A 22-year-old male suffers a GSW to his right buttock. He is hemodynamically normal with a benign abdominal examination. CT scanning is suggestive of an extraperitoneal rectal injury. Proctoscopy finds blood in the rectal vault, but you cannot clearly identify the level of injury. At laparotomy, you find no evidence of intraperitoneal injury. What is the best next step?

a. Diversion with end colostomy, distal rectal washout, and presacral drainage

b. Diversion with end colostomy alone

c. Mobilization of distal rectum with identification and suture repair of injury

d. Abdominoperineal resection

e. Nothing further and close the abdomen

View Answer

2. A 14-year-old female was riding her bicycle and accidentally rode into a ditch, resulting in her abdomen hitting her handlebars. In the trauma bay, she complains of nausea, vomiting, and abdominal pain radiating to her mid-back. She is hemodynamically normal and only has mild epigastric tenderness. CT scan demonstrates a 3-cm duodenal hematoma, nearly occluding the lumen of the first portion of the duodenum. What is the next best step?

a. Exploratory laparotomy with drainage of hematoma

b. Whipple procedure (pancreaticoduodenectomy)

c. Nasogastric drainage and TPN

d. Pyloric exclusion with gastrojejunostomy

e. Primary resection and anastomosis

View Answer

3. A 19-year-old male arrives to the trauma bay. EMS states that he was the unrestrained driver in a rollover MVC and was ejected from the vehicle. The patient was intubated by them at the scene for combativeness. He has bilateral breath sounds, HR is 150, BP 80/palp, and SaO_2 100%. He has bony crepitus over his chest and multiple bruises over his abdomen. His pelvis is stable and he has no obvious deformities in his extremities. His CXR and pelvic x-rays are normal. You send off laboratory samples and start infusing uncrossed matched Type O positive blood. What is the next best step to try to identify the source of his hypotension?

a. FAST

b. CT Chest/Abdomen/Pelvis

c. Immediate laparotomy

d. Empiric bilateral chest tubes

e. DPL

View Answer

4. A morbidly obese 40-year-old male suffered a right flank GSW. He is hemodynamically normal and has a benign abdominal examination. On physical examination, you find a bullet hole on his mid-right flank and another hole more posteriorly on his right mid-back. What is the next best step?

a. Immediate laparotomy

b. FAST

c. DPL

d. CT Abdomen/Pelvis

e. Admission for serial abdominal examinations

View Answer

5. A 64-year-old female was a restrained passenger in a high-speed MVC. She complains of RUQ pain but does not have peritonitis. Her HR is 100, BP 140/70. CT scan demonstrates a Grade IV liver injury with no evidence of contrast extravasation. Hgb is 13.5. What is the next best step?

a. Admit to the floor, daily CBCs and serial abdominal examinations

b. Admit to the ICU, Foley, q6h CBCs and serial abdominal examinations

c. Immediate laparotomy and liver packing

d. Angiography and embolization

e. Repeat CT in 24 hours

View Answer

6. A 64-year-old female was a restrained passenger in a high-speed MVC. She complains of RUQ pain and has peritonitis. Her HR is 120, BP 90/50. On laparotomy, you find a liter of blood in her abdomen and a deep laceration involving the right hemiliver that is actively bleeding. You are able to control the bleeding with laparotomy packs; however, the anesthesiologist now tells you that the patient's pH is 7.1, her temperature is 34°C, and her INR is 2.5. What is your next best step?

a. Right hepatectomy

b. Argon beam coagulation of the raw liver surfaces

c. Blunt-tipped chromic mattress sutures to the bleeding liver edges

d. Damage control laparotomy with plans for returning to the OR in 24 to 48 hours

e. Electocautery

View Answer

7. A 22-year-old female was just involved in a motorcycle accident and now arrives in the trauma bay hypotensive and in shock. You have adequate IV access and a type and screen is sent. The nurse asks you what kind of fluids you want to give. You answer:

a. Type O negative uncrossmatched blood

b. Type O positive uncrossmatched blood

c. Lactated Ringer's

d. Normal saline

e. 5% albumin

View Answer

8. A 36-year-old male was intoxicated and fell off his ATV, landing on his left side. In the trauma bay, his HR is 130, BP 70/30. On laparotomy, you find a shattered spleen, which you remove. You find no other injury and close his abdomen. It is now postop day 3 and the patient is ready to go home. It is important to give him vaccines against which of the following organisms prior to discharge?

a. *Streptococcus pneumoniae, Haemophilus influenzae,* and *Neisseria gonorrhoeae*

b. *Streptococcus pneumoniae, Haemophilus influenzae,* and *Neisseria meningitidis*

c. *Staphylococcus aureus, Haemophilus influenzae,* and *Neisseria meningitidis*

d. *Staphylococcus aureus,* Influenza, and *Neisseria meningitidis*

e. *Klebsiella pneumoniae, Haemophilus influenzae,* and *Neisseria meningitides*

View Answer

9. A 70-year-old female living in a nursing home wandered off and fell down a flight of stairs. She complains of pelvic pain. Her HR is 110, BP 90/50. Her pelvic x-ray shows an open-book pelvic fracture. What is your next step?

a. OR for preperitoneal pelvic packing

b. IR for angioembolization

c. Placement of a pelvic binder

d. CT Abdomen/Pelvis

e. OR for ORIF of the pelvis

View Answer

10. You are in the operating room with a patient who suffered a GSW to his anterior abdomen. You find a small defect of the sigmoid colon (<50% of diameter) along the antimesenteric border, with scant stool within the abdominal cavity. You find no other injuries and the anesthesiologist tells you the patient is hemodynamically stable. What is your next step?

a. End colostomy and drain placement

b. Resection of involved colon and primary anastomosis

c. Resection of involved colon and damage control laparotomy

d. Primary repair with diverting loop ileostomy

e. Primary repair alone

View Answer

13
Extremity Trauma

Lauren T. Steward

Christopher M. McAndrew

TREATMENT OF ORTHOPEDIC INJURIES

I. INITIAL ASSESSMENT

A. Priorities of Management. Assessment and management of ABCs (*a*irway, *b*reathing, and *c*irculation) take precedence over extremity injuries. Polytrauma patients benefit from early treatment of extremity and pelvic trauma.

B. History. In addition to medical history, the mechanism of injury, especially the relative energy associated with the injury (e.g., low-energy fall vs. high-energy motor vehicle crash), is important. An orthopedic history should also include preinjury functional level, especially previous occupation and ambulatory status, and hand-dominance in the case of upper-extremity injuries.

C. Examination

1. An orthopedic examination includes inspection, palpation, range of motion, strength, stability, and body region specific tests. Severe or multiple injuries often result in missed extremity injuries, especially in the foot and hand (*J Trauma.* 2003;54:38). In this setting, a complete primary evaluation where each joint and bone is inspected, and then a repeat secondary evaluation will decrease missed injuries. Inspect the extremities for bruising, swelling, lacerations, abrasions, deformity, and asymmetry. Systematically palpate all extremities, noting tenderness, crepitus, and deformity of the underlying bone. In suspected cervical spine (C-spine) injury, maintain immobilization in a cervical collar. Logroll the patient to examine and palpate the spine.

2. Assess extremity vascular status by checking pulses, capillary refill, temperature, color, and comparing to the opposite side.

3. Sensorimotor evaluation (Table 13-1). Muscle strength evaluation in the setting of acute spinal cord injury or peripheral nerve injury is critical, and serial examinations are often required. A sensory examination includes light touch in dermatomal and peripheral nerve distributions. In upper-extremity or C-spine trauma, two-point discrimination of the fingers should be assessed.

II. RADIOLOGIC EXAMINATION.

Radiologic assessment should include a minimum of x-rays with two orthogonal (90-degree) views, usually AP and lateral views (Table 13-2). A complete evaluation includes the joints above

and below the injury. Certain injuries (articular injuries and pelvic injuries) often necessitate further advanced imaging with computed tomography.

TABLE 13-1 Peripheral Nerve Examination

Nerve	Sensory	Motor	Muscle
Deep peroneal (DP)	Web space between great and second toe	Ankle and great toe dorsiflexion	Tibialis anterior (TA), Extensor hallucis longus (EHL)
Superficial peroneal (SP)	Lateral dorsum of foot	Eversion of hindfoot	Peroneus brevis and longus
Tibial (T)	Plantar surface of foot	Ankle and great toe plantarflexion	Gastrocnemius and soleus (GS), flexor hallucis longus (FHL)
Axillary (A)	Lateral deltoid	Shoulder abduction	Deltoid
Radial (R)	Dorsal web space between thumb and index	Extension of thumb IP joint	Extensor pollicis longus (EPL)
Median (M)	Two-point discrimination of thumb, index, long	Abduct thumb perpendicular to palm, flex index DIP joint	Abductor pollicis brevis (APB), flexor digitorum profundus to index (FDP2)
Ulnar (U)	Two-point discrimination of ring, small	Spread fingers apart, flex small finger DIP joint	Interossei (IO), flexor digitorum profundus to small (FDP5)

IP, interphalangeal; DIP, distal interphalangeal.

III. FRACTURES AND DISLOCATIONS

A. General Management Principles

1. Dislocation. All dislocated joints, especially in the setting of neurovascular compromise, should be reduced emergently. Successful reduction reduces the risk and degree of soft-tissue injury (e.g., pressure necrosis) and neurovascular compromise. Postreduction radiographs are essential to confirm reduction and to evaluate for associated fractures previously not visualized because of deformity. Persistently diminished or absent pulses require further evaluation.

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TABLE 13-2 Imaging Examinations for Orthopedic Injuries

Conditions	X-ray	Advanced Imaging
Clavicle fracture	Two views of the clavicle + CXR to evaluate the clavicle length of the contralateral side	
Proximal humerus fracture	AP glenohumeral joint, scapular Y, axillary view	
Scapula fracture	AP, scapular Y, and axillary lateral view	CT
Shoulder dislocation	See proximal humerus	
AC dislocation	See proximal humerus and clavicle	

Sternoclavicular dislocation		CT to evaluate displacement and visualize adjacent neurovascular structures
Humeral shaft fracture	Two orthogonal views of the humerus, including the shoulder and elbow joints	
Distal humerus fracture	Orthogonal views of the elbow	
Radial head fracture	Three views of the elbow joint	
Distal phalanx fracture	Two views of the hand	
Olecranon fracture	Three views of the elbow	
Elbow dislocation	AP and lateral radiographs of the elbow	
Radius and ulna fracture	AP and lateral radiographs for the forearm, elbow, and wrist	
Distal radius fracture	Three views of the wrist	CT to evaluate comminuted intraarticular fractures
Scaphoid fracture	Four views of the wrist	
Pelvic fracture	AP pelvis, with inlet and outlet views	CT to evaluate pelvic, sacral, and lumbar fractures

Pubic rami fracture	AP pelvis, inlet and outlet views	
Acetabular fracture	Oblique views of the pelvis	CT
Femoral neck and intertrochanteric	AP pelvis, AP and lateral hip	MRI or bone scan if history suggests fracture, but none seen on x-ray
Femoral shaft fracture	AP and lateral of the femur, including thigh and knee joints	
Hip dislocation	Pelvic films to evaluate for acetabular, femoral head, or hip fractures; check for component positioning, loosening, or periprosthetic fractures in hip replacements	
Knee and tibia	Four views of the knee	
Patella fracture	Four views of the knee	
Tibial plateau fracture	AP and lateral, and views of the knee and ankle	CT scan
Tibial shaft fracture	AP and lateral views of the tibia, including both ankle and knee joints	
Knee dislocation	Four views of the knee	Angio/CT angio to evaluate for vascular injury; MRI to evaluate for ligamentous injury
Pilon fracture	Three views of the ankle and foot films to evaluate for associated food fractures	

Ankle fracture/dislocation	Three views of the ankle	
Calcaneus fracture	Three views of the ankle and foot	Lumbar spine films to evaluate for associated lumbar fractures
Talus fracture/dislocation	Three views of the ankle and foot	CT scan
Metatarsal fracture	Three views of the foot	MRI or bone scan if concern for stress fracture
Lisfranc fracture	Three views of the foot	

2. Fractures: Pediatric versus adult

a. Pediatric: Children, especially those with open growth plates, have a greater potential for bony remodeling than adults, and therefore a greater amount of malalignment is acceptable. In children, at least limited reduction of deformity is often necessary to decrease the risk of permanent deformity.

b. Adults: In the adult, the inability to achieve and obtain an acceptable reduction is a relative indication for surgical treatment. Throughout this chapter, we will focus primarily on management of injuries in adult patients unless otherwise specified.

c. Physeal plate injuries (Ògrowth plateÓ) are common because this is the weakest part of the bone (*Skeletal Radiol.* 1981;6:237). The Salter-Harris classification categorizes these fractures into five types of increasing severity and likelihood of future growth disturbance (Table 13-3 and Fig. 13-1).

TABLE 13-3 Salter-Harris Classification of Growth Plate Injuries

Type 1	Fracture through the growth plate without any metaphyseal or epiphyseal involvement
Type 2	Fracture through the growth plate is associated with a metaphyseal fracture
Type 3	Fracture through the growth plate is associated with an epiphyseal fracture
Type 4	Fracture through the metaphysis, across the growth plate, and exiting the epiphysis
Type 5	Severe crush injury to the growth plate

IV. SOFT-TISSUE INJURY

A. Principles of Management. In general, isolated soft-tissue injuries, such as ligament sprains and muscle strains, are treated with *r*est, *i*ce, *c*ompression bandage, and *e*levation (RICE therapy) with or without immobilization.

1. Skin lacerations/defects. All devitalized tissue should be debrided. If the wound cannot be closed due to excessive tension, it should be covered with a moist saline dressing, and a delayed primary closure or skin grafting should be planned.

2. Muscle

a. Mechanism. Strains of the musculotendinous unit are usually secondary to violent contraction or excessive stretch. Injury spans the range from stretch of the fibers to a complete tear with loss of function.

b. Physical examination. Swelling, tenderness, and pain with movement occur. RICE-type treatment of the involved muscle is adequate for most such injuries.

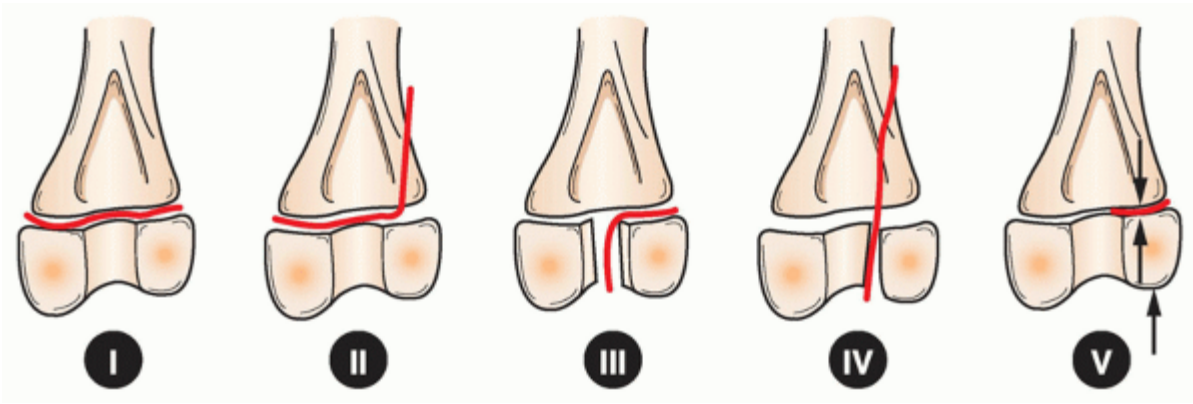


Figure 13-1 The five types of Salter-Harris growth plate injuries.

3. Tendon. Lacerated, ruptured, or avulsed tendons, especially those of the upper extremity, should be surgically repaired because such injuries result in loss of function. Examination reveals loss of motion or weakness. Open wounds with a tendon laceration are debrided, irrigated, and closed primarily with early planned repair of the tendon in the operating room (OR). In grossly contaminated wounds, debridement in the OR is needed. Splints are applied with the extremity in a functional position.

4. Ligament. Ligament sprains range from mild stretch to complete tear and are commonly sports related. Pain, localized tenderness, and joint 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 based on the RICE principle is used, and early range of motion is encouraged.

V. SPECIFIC INJURIES BY ANATOMIC LOCATION

A. Shoulder

1. Fractures

a. Clavicle

(1) Typical mechanism. A fall or direct blow to the shoulder.

(2) Typical physical signs. A visible or palpable deformity is often present at the fracture site.

(3) Typical management. Most clavicle fractures heal with nonoperative treatment, managed with a sling. Severe displacement/deformity, particularly if associated with soft-tissue compromise, is a surgical indication.

b. Proximal humerus fractures

(1) Typical mechanism. A low-energy fall in the elderly.

(2) Typical physical signs include decreased range of motion, swelling, ecchymosis, and pain. The neurovascular examination is critical to evaluate possible associated injury to the brachial plexus.

(3) Typical management. If nondisplaced and stable, these fractures can be treated with a sling and early, controlled mobilization. Comminution and displacement increases the risk of humeral head avascular necrosis and may be surgical indications. In the elderly, a primary shoulder arthroplasty may be considered if stabile internal fixation cannot be achieved.

(4) When associated with dislocation. Fracture dislocations of the shoulder are difficult to reduce closed. If there is an associated neurovascular compromise, these should be taken emergently to the OR for open reduction.

c. Scapula fractures

(1) Typical mechanism. High-energy chest trauma.

(2) Typical physical signs include tenderness to palpation over the scapula. Patients should be observed for signs of pneumothorax or other chest trauma when scapula fractures are present.

(3) Typical management. Treatment is typically with a sling unless intraarticular glenoid displacement mandates surgical treatment.

2. Dislocations

a. Shoulder dislocations (glenohumeral dislocation)

(1) Typical mechanism. Anterior shoulder dislocations (most common, ÷85%) occur with forced shoulder abduction and/or external rotation. Posterior shoulder dislocations are associated with seizure and electrical shock.

(2) Typical physical signs. Shoulder dislocation presents with decreased and painful range of motion and the humeral head may be palpable anteriorly or posteriorly.

(3) Typical management. Reduction is performed under sedation with axial traction and bringing the arm into full abduction above the head. Care should be taken with the elderly to avoid iatrogenic fracture. The arm is then immobilized in the position of greatest stability: Internal rotation for anterior dislocations and external rotation for posterior dislocations.

b. Acromioclavicular (AC) dislocations (Òa separated shoulderÓ)

(1) Typical mechanism. Fall onto or a direct blow to the shoulder.

(2) Typical physical signs. Variable deformity can be seen. Sideto-side asymmetry should be assessed. Pain with cross-body adduction and tenderness to palpation is common.

(3) Typical management. AC joint dislocations can be treated with a sling and early motion in most cases. Significant displacement and deformity may require surgical treatment, especially if associated with soft-tissue compromise.

c. Sternoclavicular dislocations

(1) Typical mechanism. High energy direct loads through the shoulder or upper chest.

(2) Typical physical signs. Localized pain, swelling, and tenderness are seen. Hoarseness, dyspnea, dysphagia, or engorged neck veins are red flags for posterior sternoclavicular joint dislocations with neurovascular compromise and should prompt emergent evaluation and treatment.

(3) Typical management. Anterior sternoclavicular dislocation can be treated with a sling or shoulder immobilizer, whereas posterior dislocations commonly require reduction because of potential neurovascular and airway compromise. This should be done in the OR under general anesthesia with general or thoracic surgery backup in case of injury to the lung or great vessels.

B. Arm and Elbow

1. Fractures

a. Humeral shaft fractures

(1) Typical mechanism. Fall onto an outstretched arm.

(2) Typical physical signs include deformity of the upper arm, pain, and ecchymosis. A careful neurovascular examination should be performed because the radial nerve is especially

vulnerable to injury due to its close apposition with the humeral shaft.

(3) Typical management. Closed fractures are placed in a coaptation splint or Sarmiento brace. Indications for surgical fixation include open fractures, injuries to multiple extremities, concurrent injury below the elbow (Òfloating elbowÓ), or a body habitus that is not amenable to bracing. Radial nerve palsy is not an indication for operative treatment of humerus fractures in isolation, but exploration of the nerve is recommended if associated with open fractures, stab/lacerating injuries, or changes in examination following manipulation (*J Hand Surg Am.* 2004;29:144).

b. Distal humerus fractures

(1) Typical mechanism. Fall onto an outstretched hand or directly onto the elbow. Supracondylar humerus fractures are the most common fractures seen in children.

(2) Typical physical signs include swelling, pain, ecchymosis, and decreased elbow range of motion. In children, displaced supracondylar fractures are frequently associated with peripheral nerve injuries. Patients should undergo serial examinations to rule out the development of

compartment syndrome.

(3) Typical management. Supracondylar fractures in children can be treated in a splint acutely if they are nondisplaced but require percutaneous pinning and casting if they are displaced. In adults, displacement of or neurovascular compromise with supracondylar fractures are indications for surgical treatment.

c. Radial head fractures

(1) Typical mechanism. Fall onto an outstretched arm.

(2) Typical physical signs. These present with tenderness to palpation and pain with forearm rotation.

(3) Typical management. Radial head fractures with minimal involvement of the articular surface (<30%) can be treated nonoperatively with early range-of-motion exercises. Compromise of elbow or forearm stability is a surgical indication.

2. Elbow dislocations (ulnohumeral)

a. Typical mechanism. Fall onto an outstretched hand.

b. Typical physical signs. Examination reveals pain, swelling, bruising, and deformity with loss of elbow range of motion. Posterior dislocations are most common.

c. Typical management. Initial treatment consists of prompt reduction and assessment of stability through gentle passive range of motion. Stable dislocations benefit from early, controlled motion, whereas unstable elbows may require surgical stabilization.

C. Forearm, Wrist, and Hand

1. Fractures

a. Radius and ulna fractures

(1) Typical mechanism. Fall onto the outstretched arm. A direct blow can cause a Ònight-stick fracture,Ó which is a mid-shaft

fracture of the ulna caused by a forceful blow to the forearm positioned for protection, often during an assault.

(2) Typical physical signs. Examination reveals deformity, pain, and focal tenderness. Variable amounts of swelling can be seen, and compartment syndrome occurs in 2% to 3% of cases. Acute carpal tunnel syndrome can occur with distal fractures due to swelling and hematoma. To evaluate for carpal tunnel syndrome, test two-point discrimination of the fingers (normally <5 to 7 mm), and test motor strength of the thumb abductors.

(3) Typical management. In children, most diaphyseal and wrist fractures can be managed with closed reduction and splinting. In adults, shaft fractures that involve both bones are almost

always treated with open reduction/internal fixation (ORIF) after initial closed reduction and splinting. Associated acute carpal tunnel syndrome or forearm compartment syndrome is a surgical emergency.

b. Distal radius fractures

(1) Typical mechanism. Fall on an outstretched hand.

(2) Typical physical signs include pain, deformity, swelling, ecchymosis, and focal tenderness. Similar to diaphyseal fractures, there can be an associated compartment syndrome or acute carpal tunnel syndrome.

(3) Typical management. Many can be treated with splinting and cast immobilization for 4 to 6 weeks. Displacement and shortening are indications for surgical treatment.

c. Scaphoid fractures

(1) Typical presentation. A fall onto an outstretched hand.

(2) Typical physical signs include local swelling, pain with motion, and tenderness in the Òanatomic snuffbox.Ó

(3) Typical management. Nondisplaced scaphoid fractures are treated in a thumb spica splint. Suspected scaphoid fractures, with pain in the anatomic snuffbox but no fracture seen on x-ray, should be treated as nondisplaced fractures and immobilized in the ER. Displaced fractures are at risk of nonunion and avascular necrosis and benefit from surgical treatment.

d. Metacarpal fractures

(1) Typical mechanism. Crush injury or axial load onto a closed fist.

(2) Typical physical signs include swelling and bruising, often with less knuckle prominence; the most common fracture is of the distal fifth metacarpal, otherwise known as a Òboxer's fracture.Ó

(3) Typical management involves reduction and splinting with fingers in the intrinsic plus position, followed by reexamination for rotational malalignment. If unstable, significantly angulated, or rotationally malaligned, these may require surgical treatment.

e. Distal phalanx fractures

(1) Typical mechanism. Crush injury.

(2) Typical physical signs. These are typically associated with lacerations of the fingertip or nail-bed injuries.

(3) Typical management. While these are technically open fractures, they can be adequately irrigated and debrided in the ER and do not require a formal I&D in the OR. If there is any question of a nail-bed injury, the nail should be removed. Preformed finger splints are used to

immobilize the fracture.

D. Pelvic Fractures

1. Disruptions of the pelvic ring

a. Typical mechanism. Pelvic ring injuries typically result from high-energy mechanisms, such as motor vehicle collisions or a fall from height.

b. Typical physical signs. Crepitus, pelvic instability, or pain with iliac wing compression or distraction should alert the examiner to possible pelvic ring injury. The patient should be inspected for soft-tissue injury, including a degloving injury. Rectal and vaginal examinations are performed to check for blood, open communication with a fracture, or a high-riding prostate. Blood at the urethral meatus at time of catheterization is a sign of lower urogenital injury. Retrograde urethrogram should be obtained to identify these injuries and define their location. Pelvic bleeding may result in a loss of 2 to 3 L of blood or more, and signs of hypovolemic shock should be treated with aggressive fluid and blood product replacement. High-energy pelvic fractures rarely occur in isolation, and associated injuries are likely.

c. Typical management. The initial treatment consists of adherence to ATLS protocols. Maintenance of adequate intravascular volume and systolic blood pressure is essential in the hemodynamically unstable patient. In the persistently unstable patient, sources of bleeding other than the pelvis should be ruled out followed by emergent stabilization of the pelvis with a linen sheet or pelvic binder in patients with volume expanding pelvic ring injuries (AP compression Òopen bookÓ and vertical shear). Angiogram and embolization of bleeding pelvic vessels should be considered for those patients who remain hemodynamically unstable after volume control and stabilization of the pelvis with a sheet/binder (*Clin Orthop Relat Res.* 1995;318:61; *J Trauma.* 2005;58:778).

2. Pubic rami fractures

a. Typical mechanism. Same level falls in an elderly patient.

b. Typical physical signs include groin pain and pain with weightbearing.

c. Typical management. These patients are allowed to bear weight as tolerated, but they often initially have significant pain with weightbearing. Physical therapy and mobilization is important to prevent secondary morbidity.

3. Acetabular fractures

a. Typical mechanism. High-energy trauma such as a motor vehicle collision or fall from height.

b. Typical physical signs include hip pain and pain with logroll. In cases with an associated hip dislocation, the limb may be shortened and internally rotated. A sciatic palsy is also possible with a posterior hip dislocation.

c. Typical management. Skeletal traction may be indicated for fractures of the acetabulum, depending on the size and location of the fracture and an associated dislocation. Fractures involving the weightbearing portion of the acetabulum are usually treated with surgical reduction and fixation.

E. Hip and Femur

1. Fractures of the hip and femur

a. Hip fractures (femoral neck and intertrochanteric fractures)

(1) Typical mechanism. Low-energy falls onto the hip in the elderly.

(2) Typical physical signs. Shortening of the limb may be seen in addition to pain with motion and the inability to bear weight. Displaced hip fractures are associated with a typical presentation: A shortened, externally rotated lower extremity. A high index of suspicion must be maintained in the elderly after a low-energy fall presenting with complaints of groin or medial thigh pain (site of referred pain from the hip joint) because these may be the only signs of a nondisplaced hip fracture.

(3) Typical management. Displaced femoral neck fractures in the young require urgent anatomic reduction and internal fixation to reduce the risk of avascular necrosis. In the elderly, surgical treatment is generally the rule for hip fractures. Stable femoral neck fractures are usually treated with internal fixation (most commonly, percutaneous screws) and unstable femoral neck fractures with hip arthroplasty (hemi- or total hip arthroplasty). Pertrochanteric fractures are treated with various internal fixation methods, including the use of compression screws and plates or an intramedullary nail.

b. Femoral shaft fractures

(1) Typical mechanism. High-energy mechanisms, most commonly motor vehicle accidents.

(2) Typical physical signs. Patients usually have gross deformity and instability.

(3) Typical management. Initial management involves long-leg splinting or skeletal traction, based on institutional or consultant preference, to increase comfort and stability while maintaining length and protecting the soft tissues. Most are treated with intramedullary nailing soon after the injury to allow early mobilization and decrease the risk of additional complications (*J Bone Joint Surg Am.* 1989;71:336). In the unstable, multiply injured patient, external fixation may be the initial treatment to minimize adverse systemic effects caused by instrumentation of the medullary canal (*J Trauma.* 2002;53:452; *J Trauma.* 2009;67:1013).

2. Hip dislocations

a. Typical mechanism. High-energy motor vehicle crash, often associated with acetabular fracture. In patients with a previous hip replacement, dislocation is typically atraumatic.

b. Typical physical signs. Posterior dislocations (most common) cause limb shortening with an

adducted and internally rotated posture.

Sciatic nerve function should be assessed for palsy with posterior dislocations (the peroneal division is most commonly affected).

c. Typical management. Once a hip dislocation is identified in a native hip, immediate closed reduction followed by additional imaging should be performed to reduce the risk of avascular necrosis. Adequate sedation and muscle relaxation are essential for successful reduction without iatrogenic fracture. Assessment of stability and postreduction neurologic examination are necessary. Skeletal traction is indicated when the hip remains unstable after a reduction. Postreduction radiographs, including AP, lateral, and Judet views, are needed to confirm reduction and assess for associated fractures. Patients with dislocated hip arthroplasties can usually be reduced, closed, and placed in an abduction brace.

F. Knee and Tibia

1. Fractures

a. Supracondylar femur fractures

(1) Typical mechanism. Can be low energy in the elderly, generally higher energy in younger patients.

(2) Typical physical signs are deformity and swelling around the knee.

(3) Typical management. These fractures are initially reduced and splinted in the ER. Almost all of these will require operative treatment.

b. Patellar fractures

(1) Typical mechanism. Patella fractures are commonly caused by falling directly onto the knee or striking a dashboard.

(2) Typical physical signs. Patella fractures often have a palpable defect, and patients have an associated inability to perform a straight-leg raise.

(3) Typical management. Patella fractures with displacement, joint incongruity, or loss of active knee extension require surgical treatment. Nondisplaced fractures can be treated with a knee immobilizer and weightbearing as tolerated.

c. Tibial plateau fractures

(1) Typical mechanism. Motor vehicle collision, fall from a height, direct blow, and pedestrian versus car are all common.

(2) Typical physical signs include knee effusions, swelling in the lower leg, ecchymosis, and deformity. Tibial plateau fractures should be carefully monitored for compartment syndrome.

(3) Typical management. Tibial plateau fractures are treated with splinting and early motion if

they are nondisplaced and stable, but they require surgical treatment for articular incongruity, significant displacement, deformity, or instability. When significant soft-tissue swelling is present, plateau fractures are treated with a temporary, spanning external fixator across the knee until swelling decreases to the point that ORIF is appropriate (*Skeletal Trauma: Basic Science, Management, and Reconstruction.* 2015;1937).

d. Tibial shaft fractures

(1) Typical mechanism. Motor vehicle and motorcycle collisions, falls from height, and gunshot wounds are all common mechanisms.

(2) Typical physical signs. Gross deformity and instability is usually present. The subcutaneous location of the tibia predisposes to open fractures. Thorough evaluation of lacerations is necessary, as they commonly communicate with the fracture. Nerve function, pulses and foot perfusion should be compared with the contralateral side. The patients should be carefully monitored for compartment syndrome.

(3) Typical management. Stable tibial shaft fractures can be treated with casting; however, most are treated with intramedullary nailing to allow early weightbearing and joint motion. Open tibial shaft fractures may require multiple surgical debridements and soft-tissue coverage.

2. Knee dislocations

a. Typical mechanism. Knee dislocations are the result of very high-energy injuries and require multiple ligamentous disruptions to occur. In extremely obese persons, these can occur with a same level fall or even walking on uneven surfaces. These are very different from patella dislocations, which usually occur with a twisting force while in extension, often during sports, and usually reduce spontaneously.

b. Typical physical examination. Knee dislocations present with deformity, shortening, ligamentous instability, and often signs of significant neurovascular compromise. Check side-to-side differences in pulse examination serially. Knee dislocations that have reduced spontaneously are easy to miss in the acute setting, and the knees should be examined for ligamentous instability in the setting of high-energy trauma.

c. Typical management. Knee dislocations require immediate, emergent reduction. These should be reduced even before radiographs are taken if possible. The incidence of concomitant vascular injury is approximately 30%, and pedal pulse examination has a low sensitivity (79%) for detecting significant vascular injury. Ankle-Brachial Index (ABI) testing and, possibly, arteriography can be used for further evaluation (*J Trauma.* 2004;56:1261). A vascular surgery consultation may be required. If vascular repair is necessary, a spanning external fixator can be placed to stabilize the knee. After any vascular repair, prophylactic fasciotomy should be considered. Delayed ligamentous reconstruction may be necessary to restore knee stability (*J Trauma.* 2007;63:855).

G. Distal Tibia and Ankle

1. Fractures

a. Pilon fractures

(1) Typical mechanism. Distal tibial intraarticular fractures (pilon fractures) are associated with an axial loading mechanism such

as falls from a height or floor board injury from a motor vehicle accident.

(2) Typical physical signs include deformity, instability, swelling, and ecchymosis about the ankle. Note soft-tissue injury, which is often significant with pilon injuries, including location of fracture blisters and whether the blisters are blood filled (marker of deeper injury).

(3) Typical management. Pilon fractures with significant shortening, comminution, or soft-tissue injury are best managed initially with closed reduction and placement of a spanning external fixator. External fixation is then maintained until soft-tissue swelling resolves and the leg can tolerate an open procedure. Soft-tissue management is critical in the presence of these injuries (*J Orthop Trauma.* 1999;13:85; *J Orthop Trauma.* 1999;13:78).

b. Ankle fractures

(1) Typical mechanism. Ankle fractures are commonly caused by a twisting mechanism.

(2) Typical physical signs include deformity and instability of the lower leg and ankle joint. A neurovascular examination should be performed and documented.

(3) Typical management. Stable, nondisplaced fractures of the ankle can be treated with immobilization and protected weightbearing. Unstable fractures (one with both medial and lateral injuries) and fractures with joint subluxation benefit from operative treatment. Ankle fractures associated with dislocation represent injuries from higher-energy mechanisms that are associated with more severe soft-tissue injury. All fractures should be reduced in the ER with postreduction radiographs demonstrating adequate joint and fracture reduction. If adequate joint reduction cannot be achieved or maintained, early surgical treatment is indicated.

2. Soft-tissue injuries

a. Ankle sprains are commonly caused by inversion or eversion of the foot. Patients present with swelling, ecchymosis, and maximal tenderness along the injured ligaments. Radiographs are normal or reveal cortical avulsions. Initial treatment with RICE is usually adequate, followed by physical therapy for proprioceptive training to reduce the risk of reinjury. Immobilization is generally not indicated.

b. A ruptured Achilles tendon usually occurs during running, jumping, or vigorous activity, with sudden pain and difficulty in walking. Examination can reveal a palpable defect, weak plantar flexion, and, in cases of complete rupture, no passive ankle plantar flexion on squeezing the

patient's calf (positive Thompson sign). Treatment consists of either nonoperative management in a splint with the ankle plantar flexed or direct surgical repair. Previous literature has supported operative treatment to decrease the risk of rerupture, but this has not been found in more recent literature with modern nonoperative treatment courses (*J Bone Joint Surg Am.* 2012;94:2136).

H. Foot

1. Fractures

a. Calcaneus fractures

(1) Typical mechanism. They are usually the result of an axial load such as a fall from height.

(2) Typical physical signs. Calcaneal fractures are associated with swelling, heel widening, tenderness, and ecchymosis. Associated fractures are common, especially in the thoracolumbar spine, due to axial loading.

(3) Typical management. Calcaneal fractures should be placed in a well-padded splint. Significant subtalar joint depression and comminution may require ORIF once soft-tissue swelling allows.

b. Talus fractures

(1) Typical mechanism. Talus fractures (the second most common) are also generally higher energy (motor vehicle collision or falls) and are usually caused by forced dorsiflexion (e.g., slamming on the brake at the time of impact).

(2) Typical physical signs. Talus fractures can also present with swelling, and when they are associated with a dislocation of the tibiotalar joint and/or the subtalar joint, a significant deformity can be present.

(3) Typical management. Talus fractures can be treated with cast immobilization if they are nondisplaced, but most talus fractures are treated with ORIF to decrease the risk of nonunion and avascular necrosis.

c. Metatarsal fractures

(1) Typical mechanism. Metatarsal fractures can be seen with lower energy trauma. Stress fractures can occur in runners or others who have recently increased their distance or activity.

(2) Typical physical signs. Stress fractures may present only with tenderness to palpation at the level of the injury.

(3) Typical management. Most metatarsal fractures can be treated nonoperatively with splinting. Transverse fractures of the proximal fifth metatarsal diaphysis (Jones fracture), due to being in a vascular watershed region, are prone to healing complications and require more aggressive treatment than other metatarsal fractures, including either strict non-weightbearing

with cast immobilization or surgery. An avulsion of the base of the fifth metatarsal, in contrast, is treated with a controlled ankle motion boot and early weightbearing.

d. Toe fractures. Toe injuries are best treated by Òbuddy tapingÓ to the adjacent digit and giving the patient a hard-soled shoe for more comfortable ambulation. Distal phalanx fractures with nail-bed injuries or soft-tissue lacerations are treated the same as similar injuries in the fingers.

e. Fractures in diabetic feet. Diabetics with peripheral neuropathy and injuries to the foot or ankle require special attention and care. Because of neuropathic changes, casts and splints must be well

padded and adapted to any deformity of the foot. Typically, foot and ankle fractures in the diabetic require twice the normal period of immobilization. A hot, swollen foot in a diabetic patient should be examined radiographically for neuropathic fractures (the Charcot foot) and immobilized. This should be differentiated from cellulitis and infection with laboratory tests, although they can occur simultaneously.

2. Dislocations

a. Talar dislocations

(1) Typical mechanism. The level of energy is similar to that for calcaneal and talar fractures. Talar dislocation occurs with forced foot inversion.

(2) Typical physical signs. With talar dislocations, there is often significant deformity. Dislocation of the talar body can commonly impinge on adjacent neurovascular structures and can be entrapped by tendons.

(3) Typical management. Talar dislocations are treated with emergent reduction to decrease the risk of avascular necrosis, neurovascular injury, and skin compromise. Soft-tissue interposition can prevent closed reduction, in which case open reduction is required. Associated fractures must be anatomically reduced and stabilized as described previously.

b. Lisfranc dislocations

(1) Typical mechanism. Lisfranc injuries are disruptions of the tarsal-metatarsal joints by either dislocation or fracture dislocation and are caused by a bending or twisting force through the mid-foot.

(2) Typical physical signs. Lisfranc injuries are associated with significant swelling and mid-foot tenderness. Compartment syndrome of the foot may be present.

(3) Typical management. Lisfranc injuries are splinted, iced, and elevated in preparation for eventual operative treatment. An attempt at closed reduction should be made to help decrease soft-tissue injury.

VI. OTHER ORTHOPEDIC CONDITIONS

A. Compartment syndrome is characterized by an increase in tissue pressure within a closed osteofascial space sufficient to compromise microcirculation, leading to irreversible damage to tissues within that compartment, including death of muscle and nerves. This occurs in association with prolonged limb ischemia/reperfusion, external pressure, fractures, and burns (*Skeletal Trauma: Basic Science, Management, and Reconstruction.* 2015;437).

1. Location. Although it occurs most frequently in the four muscle compartments of the leg or the muscle compartments of the forearm, it can also occur about the elbow or in the thigh, hand, or foot.

2. Examination. Signs and symptoms of compartment syndrome include pain (especially with passive motion), pressure, paralysis, paresthesia,

pulselessness, and pallor (the so-called six *P*s). The earliest and most important sign is pain out of proportion to the injury, particularly an increasing and disproportionate narcotic demand and unexpectedly poor response to appropriate pain medication, and/or pain with passive motion of involved muscles or tendons traversing the involved compartment. A high index of suspicion is necessary for early diagnosis because Òhard signsÓ like nerve dysfunction or pulselessness occur late in the process. Serial compartment pressure measurements should be considered in obtunded patients with risk factors. In the awake patient, compartment syndrome is a clinical diagnosis. However, when the clinical picture and physical examination are sufficiently uncertain or in the unresponsive patient, compartment pressures should be measured. A difference between diastolic blood pressure and compartment pressure of <30 mm Hg is diagnostic of compartment syndrome (*J Bone Joint Surg Br.* 1996;78:99).

3. Treatment. Fasciotomy of all involved compartments is necessary when compartment syndrome is diagnosed. Prophylactic fasciotomy should also be performed after repair of traumatic vascular injuries (particularly those presenting with ischemia). Fasciotomy techniques are detailed below.

a. Forearm fasciotomy technique (*Clin Orthop Relat Res.* 1981;161:252). A single incision should start 1 cm proximal and 2 cm lateral to the medial epicondyle, extend medially and obliquely across the antecubital fossa over the volar aspect of the brachioradialis muscle, and then turn distally and medially into the midline, where it is extended longitudinally just ulnar to the palmaris longus tendon. The incision should be curved across the wrist crease to allow for carpal tunnel release. An adequate fasciotomy of the forearm in the setting of trauma includes careful release of the lacertus fibrosus in the medial portion of the antecubital fossa, the fascia over the brachioradialis/mobile wad muscles laterally, the fascia of the superficial and deep extrinsic muscles of the forearm, and carpal tunnel release. Many times, the pressure in the dorsal compartment of the forearm is decreased following volar fasciotomy; however, if pressure remains elevated in the dorsal compartment, a second incision should be made from 2 cm distal

to the lateral epicondyle extending distally toward the midline of the wrist (10 cm in length) to visualize and then release the fascia overlying the dorsal forearm muscles.

b. Leg fasciotomy techniques

(1) Two-incision technique (*J Bone Joint Surg Am.* 1977;59:184). The lateral incision should be made 1 cm anterior to and in line with the fibula, extending from the fibular head to 4 cm proximal to the lateral malleolus. The incision is taken through the skin and subcutaneous tissue to the level of the fascia. The subcutaneous flap is raised off the fascia medially in order to identify the intermuscular septum between the anterior and lateral compartments, through which perforating vessels to the skin are seen. The fascia of the anterior compartment should be incised

over the entire length of the wound, exposing the muscular origins proximally and beyond the myotendinous junctions distally. Care must be taken to identify and protect the superficial peroneal nerve in the subcutaneous space crossing from lateral to medial at the distal portion of this fascial incision. Next, subcutaneous flaps are raised as needed to expose the fascia of the lateral compartment 1 cm lateral/posterior to the fibula. The fascia over the entire length of the wound must be incised to expose the muscular origins proximally and beyond the myotendinous junctions distally. The superficial peroneal nerve, located deep to the fascia of the lateral compartment at the mid-leg and superficial to the fascial just distal to the midleg and proximal to where it was identified over the anterior compartment, needs to be found and protected. The medial incision should be 1 to 2 cm posterior to the medial border of the tibia and extended from the level of the knee to the ankle. The greater saphenous vein and saphenous nerve in the subcutaneous tissue ought to be identified and retracted anteriorly for protection. Fascia should be incised from the gastrocnemius-soleus complex over the entire course of the wound. To visualize the fascia of the deep compartment adequately, it is necessary that a portion of the soleus origin on the posterior and proximal tibia be released. Care must be taken not to incise too far medially and risk injury to the posterior tibial artery and vein. Once the soleus origin has been elevated, the fascia over the flexor digitorum longus can be visualized and incised over the entire course of the wound. There should be a skin bridge between the two incisions of *at least* 8 cm.

(2) Single incision technique (*J Orthop Trauma.* 2008;22:723). A single, straight incision should be made through the skin and subcutaneous tissue in line with the fibula from the fibular head to 4 cm proximal to the lateral malleolus. These landmarks are easily palpable. The subcutaneous flap is elevated medially and the intermuscular septum and perforating skin vessels are identified. The superficial peroneal nerve can be found in the subcutaneous space distally and should be protected. The fascia of the anterior compartment must be incised over the entire length of the wound, exposing the muscular origins proximally and beyond the myotendinous junctions distally. The fascia over the lateral compartment is identifiable just anterior to the lateral intermuscular septum, and it should be incised from its muscular origin to beyond the myotendinous junctions. The peroneal muscles may then be elevated off of the intermuscular septum to the fibula. Along the lateral border of the fibula, the intermuscular septum should be

incised over the entire length of the wound, taking care to identify and coagulate communicating blood vessels from the peroneal trunk to the deep posterior compartment. Exposure of the flexor hallucis longus may be verified

by extending and flexing the interphalangeal joint of the great toe and seeing the muscle move in the wound. This confirms decompression of the deep posterior compartment. Finally, the fascia overlying the gastrocnemius-soleus complex should be incised from the proximal wound to beyond the myotendinous junction distally.

B. Open Fractures and Joints. Lacerations or wounds near fractures or joints can communicate and should be carefully evaluated. If exposed bone is not evident, wounds should be carefully probed to determine whether communication with the fracture is present. Air in the joint on x-ray and fat droplets in blood from the wound also confirm communication with a joint or fracture, respectively.

1. Treatment consists of assessing the wounds, removing any obvious gross contamination, applying moist saline dressings, reducing the fracture or joint, and splinting the extremity. Tetanus prophylaxis and IV antibiotics should be administered. Open fractures are classified according to the amount of soft-tissue injury, which provide prognosis for development of infection (*J Bone Joint Surg Am.* 1976;58:453). Type I and Type II open fractures are those associated with low-energy mechanisms, little evidence of deep soft-tissue stripping, and lacerations measure less than (Type I) or more than (Type II) 1 cm. Type III open fractures are the result of high-energy mechanisms. These include open fractures associated with vascular injury requiring repair, severe contamination, severe deep tissue stripping (segmental injuries), or injury from high velocity ballistics, regardless of the size of the skin laceration. First-generation cephalosporins are given for all open fractures, unless allergy requires an alternate antibiotic. Some centers give an aminoglycoside for Type III open fractures, but the risk/benefit of its use has come under recent scrutiny, and increasing numbers of trauma centers do not add an aminoglycoside. Debridement of the open fracture and stabilization (either temporary or permanent) should be done urgently, but previously taught time frames (6 to 8 hours) have not been shown to affect the outcomes following open fracture. While there is no proven time frame to operative debridement shown to decrease the risk of infection, decreasing the time to the definitive treating trauma center and decreasing the time from injury to first IV antibiotic dose has been shown to decrease the risk of infection (*J Bone Joint Surg Am.* 2010;92:7). Operative debridement should be performed as soon as possible after the patient is resuscitated and appropriate OR personnel are available for a thorough wound debridement and skeletal stabilization.

C. Upper-extremity Traumatic Amputation. A team approach is needed to evaluate for possible reimplantation, and all necessary consultants should be contacted early.

1. Management. The proximal stump should be cleaned, and a compression dressing should be applied. Tourniquets are not used. Amputated parts should be wrapped in moist gauze, placed in

a bag, and cooled by placing on ice (taking care to avoid freezing damage), and sent to the

OR before the patient for preparation. Reimplantation is most likely to be successful with a sharp amputation and is typically not likely possible with crush injuries or other injuries with a wide zone of injury. Timing is of the essence, and a rapid and efficient evaluation is critical. Indication for reimplantation of fingers includes injury in a child, involvement of the thumb, multiple involved digits, and injury distal to the middle phalanx.

D. The Mangled Extremity. The mangled extremity is one that has sustained significant injury to the vascular, bony, soft tissue, and/or nerve structures. Management of these injuries has to take into account the patient's clinical status on presentation, the ability to revascularize the limb within a timely fashion, and the overall presumed functional status of the limb if it is able to be salvaged. Many attempts have been made to establish severity scores to aid decisions about limb salvage versus amputation (*J Bone Joint Surg Am.* 2005;87:2601; *J Trauma.* 2002;52:641). While these scores provide guidelines in the initial evaluation of these injuries, they have failed to provide prognostic information to direct treatment when deciding between limb salvage and amputation. Outcomes following these severe injuries have been associated with multiple psychosocial factors and may not be independently related to the decision to salvage the limb or proceed with amputation. Figure 13-2 is one proposed algorithm for the management of mangled extremity (*J Trauma Acute Care Surg.* 2012;72:86).

VII. EXTREMITY VASCULAR INJURIES.

A wounded extremity can tolerate approximately 6 hours of ischemia before the onset of irreversible loss of function. Therefore, quickly identifying and repairing vascular injuries is essential to reduce morbidity.

A. Examination. 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. Patients with possible (soft) signs of vascular injury (nerve deficit, nonexpanding hematoma, associated fracture, significant soft-tissue injury, history of bleeding, or hypotension) require evaluation of vascular integrity. A useful algorithm is to check the ABI in the trauma bay. If the ABI for the affected limb is greater than 0.9, no further radiographic evaluation is necessary. If it is less than 0.9, CT angiogram of the affected extremity or operative exploration should be considered.

B. Treatment

1. Arterial injuries should be repaired within 6 hours to maximize limb salvage rates. The operative approach is similar to elective vascular procedures, and endovascular therapy may be feasible if available. 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 polytetrafluoroethylene (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.

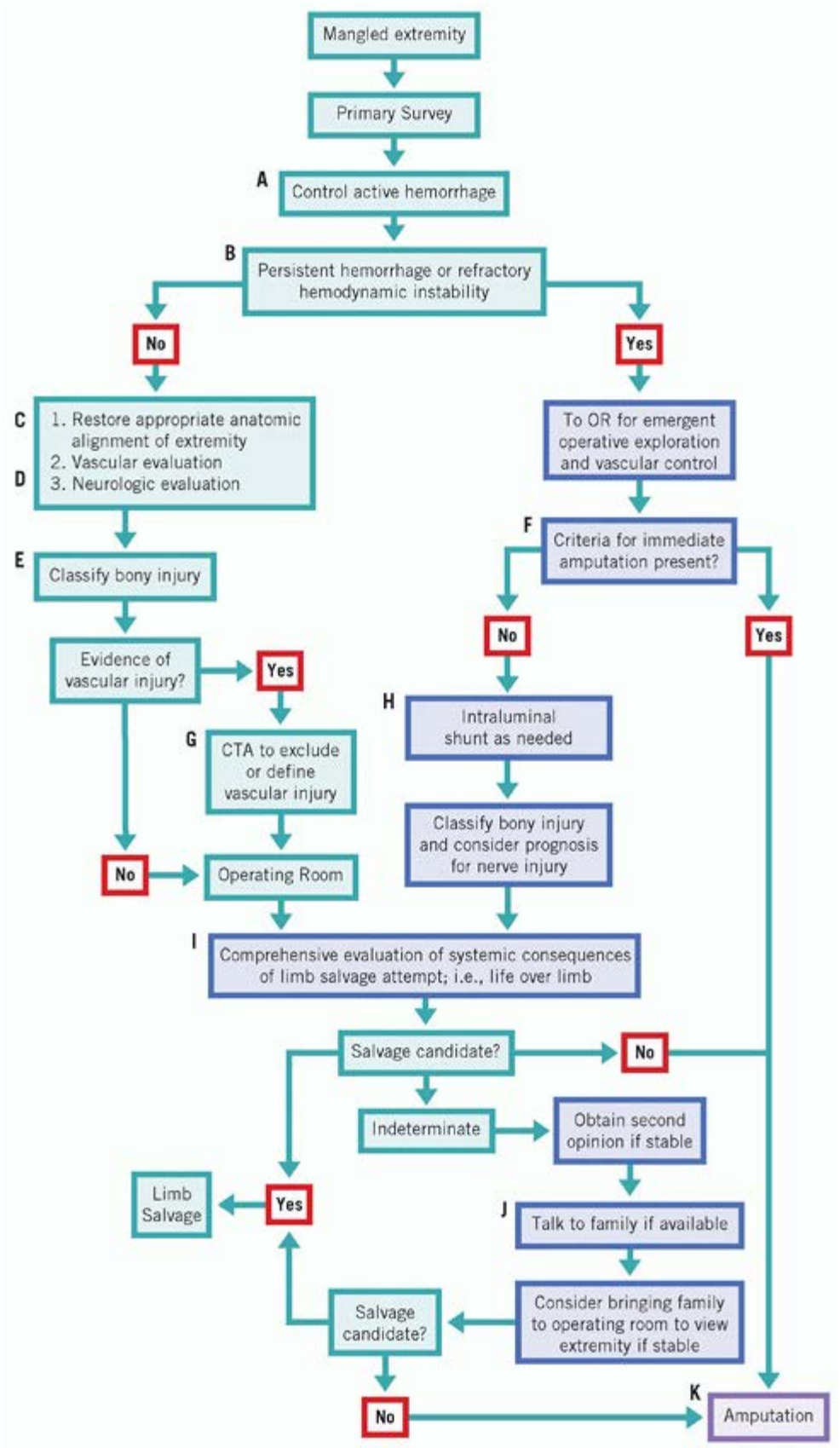


Figure 13-2 Algorithm for management of patients with mangled extremities. (From: Scalea TM, DuBose J, Moore EE, et al. Western Trauma Association critical decisions in trauma: management of the mangled extremity. *J Trauma Acute Care Surg.* 2011;72(1):86-

93, with permission.)

2. 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.

VIII. COMPLICATIONS

A. Surgical site infection is more commonly associated with treatment of open fractures and treatment of certain fracture types (e.g., tibial pilon fractures). Initial treatment of open fractures with IV antibiotics and patient resuscitation, along with timely debridement and stabilization, has decreased historically high rates of infection (25% to 40%) for open fractures overall (*J Bone Joint Surg Am.* 1976;58:453). However, for the most severely injured lower extremities with open fractures, infection rates still remain high (27%) (*J Bone Joint Surg Am.* 2010;92:7). Staged management of fractures associated with soft-tissue swelling has led to a decrease in the incidence of surgical site infection associated with tibial pilon fractures. Initial stabilization with an external fixator and delayed open surgery has led to a decrease in surgical site infection from 30% to 40% to less than 10% (*J Trauma.* 1999;47:937; *J Orthop Trauma.* 1999;13:85; *J Orthop.* 1999;13:78).

B. Fracture healing problems include failure of healing and healing with deformity.

1. Nonunion is the failure of skeletal healing, which can result from a lack of biologic activity (caused by injury or iatrogenically with surgical intervention) or from inadequate mechanical stability to promote bone healing (fibrous tissue forms instead). Nonunions resulting from loss of biologic activity appear atrophic on radiographs, with little bone/callous formation. Nonunions resulting from a lack of sufficient stability appear hypertrophic on radiographs and demonstrate excess bone/callous formation, often expanding widely outside the previous shape of the fractured bone (*Skeletal Trauma: Basic Science, Management, and Reconstruction*. 2015;637, *Injury*. 2014;45 Suppl 2:S3).

2. Malunion is healing of bone with deformity. This is much less common with modern surgical intervention, but can still result from nonoperative treatment, inadequate alignment at the time of surgical intervention, or failure to maintain alignment with implants. Malunions affect the patient to varying degrees depending on location and severity. Malunions near joints affect the function of

those joints and are usually the least well tolerated, leading to corrective surgical intervention. When implant failure is the cause following surgical treatment of fractures, surgical site infection should be considered a potential etiology for development of the deformity following delayed healing.

CHAPTER 13: EXTREMITY TRAUMA

Multiple Choice Questions

1. In the medial approach for dual incision fasciotomy for compartment syndrome of the lower leg, what structure must be released to allow access to the deep posterior compartment?

a. Plantaris

b. Sartorial expansion

c. Soleus

d. Flexor digitorum longus

e. Flexor hallucis longus

View Answer

2. Diagnosis of compartment syndrome of the leg in an obtunded patient is made by measuring a difference of less than 30 mm Hg between the compartment pressure and:

a. Systolic blood pressure

b. Diastolic blood pressure

c. Mean arterial pressure

d. Cerebral perfusion pressure

View Answer

3. The potential for associated vascular injury is highest in which of the following tibial plateau fracture patterns?

a. Split lateral plateau fracture

b. Split depression lateral plateau fracture

c. Medial plateau fracture-dislocation

d. Bicondylar plateau fracture

e. Bicondylar fracture with metaphyseal dissociation

View Answer

4. A 22-year-old man injured in a motor vehicle accident is hypotensive

and tachycardic. Anteroposterior pelvis radiograph reveals an anteroposterior compression-type pelvic ring injury. In addition to resuscitation, what is the next most appropriate step in management?

a. Pelvic angiography

b. CT of the pelvis

c. Application of a pelvic sheet or binder

d. Emergent open reduction and internal fixation

e. Focused assessment with sonography for trauma (FAST examination)

View Answer

5. A 27-year-old male presents with thoracic contusions, a left femur shaft fracture, and initial lactate of 5.1. Following temporary stabilization with skeletal traction, the patient is resuscitated overnight in the ICU and is intubated for pulmonary failure. The following morning the patient has a lactate of 4.7. What is the recommended treatment for the patient's femur fracture on postinjury day 1?

a. Reamed intramedullary nail fixation

b. Unreamed intramedullary nail fixation

c. Open reduction and internal fixation with plates and screws

d. External fixation

e. Conversion of skeletal traction to a hip spica cast

View Answer

6. Following admission to the trauma ward following extubation after 3 days in the ICU for pulmonary failure, the patient reports pain in his foot. Swelling is noted, and radiographs demonstrate multiple metatarsal fractures. Missed injuries are associated with _____, most are located in the _____, and rates can be decreased with _____.

a. intoxication, abdomen, primary survey

b. intubation, spine, secondary survey

c. intubation, distal extremities, tertiary survey

d. intoxication, thorax, tertiary survey

e. combativeness, distal extremities, secondary survey

View Answer

7. Tibia pilon fractures differ from other fractures of the ankle in that they are more likely the result of a(n) _____ force and are _____ energy injuries.

a. rotational, low

b. axial, high

c. rotational, high

d. axial, low

e. shear, high

View Answer

8. Treatment of tibia pilon fractures is done in stages using initial spanning external fixation followed by delayed open reduction and internal fixation to decrease the risk of what complication?

a. Posttraumatic arthritis

b. Skeletal deformity

c. Surgical site infection

d. Postoperative knee pain

e. Low patient satisfaction scores

View Answer

9. Intramedullary nail fixation of femur fractures in under-resuscitated multitrauma patients is associated with which complication?

a. Malunion

b. Fat embolism syndrome

c. Acute respiratory distress syndrome

d. Acute blood loss anemia

e. Nonunion

View Answer

10. A 23-year-old female presents following MVC with an open humerus shaft fracture. On examination, she is unable to extend her wrist, fingers, and thumb. Definitive treatment of her extremity injury should include:

a. Cleansing in the ER with saline, wet-to-dry dressings, coaptation splint converted to functional brace

b. External fixation of the humerus

c. Debridement and irrigation of the wound with exploration of the radial nerve, followed by internal fixation of the fracture

d. Debridement and irrigation of the wound with exploration of the ulnar nerve, followed by internal fixation of the fracture

e. Debridement and irrigation of the wound with exploration of the median nerve, followed by internal fixation of the fracture

View Answer

14
Common Surgical Procedures

Lauren M. Barron

John E. Mazuski

This chapter reviews concepts, indications, and technical aspects of bedside procedures commonly performed in hospitalized surgical patients, focusing on central venous catheterization, thoracic and peritoneal drainage procedures, and airway access.

I. CENTRAL VENOUS CATHETERIZATION.

Central venous catheterization is frequently used in surgical patients both for diagnosis and treatment. Before placement of a central venous access device (CVAD), the patient should be evaluated for the presence of an indwelling central venous device, such as a transvenous pacemaker, and for signs of central venous obstruction, such as distended collateral veins about the shoulder and neck.

Contraindications: Venous thrombosis is an absolute contraindication to catheter placement at the affected site. Relative contraindications include coagulopathy (international normalized ratio [INR] >2 or partial prothrombin time [PTT] >two times control) and thrombocytopenia (platelet count <50,000/μL). For an elective procedure, administration of blood products prior to the procedure to partially correct the coagulopathy or thrombocytopenia should be considered. If that is not feasible, the internal jugular is the preferred approach.

A. Types of Catheters. Prior to insertion of a CVAD, one must consider the expected duration of treatment and number of lumens necessary to achieve the treatment goals. Multilumen catheters are associated with slightly higher rates of infection than single-lumen catheters. Therefore, in the setting of a single therapy, such as total parenteral nutrition (TPN), a single lumen is the optimal choice. Peripherally inserted central catheters (PICCs) and Hohn catheters are considered intermediate-term CVADs and may be ideal for home administration of intravenous antibiotics. For long-term use, a tunneled line, such as a Hickman or a Broviac (Bard Access Systems, Murray Hill, NJ), should be considered to decrease the risk of bacterial migration along the catheter from the skin.

B. Internal Jugular Approach

1. Indications. The internal jugular vein is easily and rapidly accessible in most patients. Advantages of this site include decreased risk of pneumothorax and ready compressibility of the vessels in case of bleeding. Disadvantages of this approach consist of patient discomfort during

the procedure and difficulty maintaining a sterile dressing on the insertion site in the presence of a tracheostomy. This site is commonly used for the placement of tunneled catheters and ports where the catheter's exit site is on the chest.

2. Technique. Ultrasound guidance has been shown to increase first attempt success rate and decrease complication rate in internal jugular central venous cannulation in both prospective randomized trials and meta-analyses. Therefore, the American College of Surgeons recommends use of ultrasound guidance when available (*Bulletin of the American College of Surgeons.* 2011;96:36-37). In emergent cases and when equipment is unavailable, one must use anatomic landmarks for insertion (Fig. 14-1) and a small 21- to 25-gauge ÒseekerÓ needle should precede any attempt at cannulation with a large needle or catheter. The physician begins by standing at the head of the bed with the patient in Trendelenburg position at an angle of 10 to 15 degrees. The patient's head should be flat on the bed and turned away from the side of the procedure. The skin is prepped and draped prior to anesthetizing the subcutaneous tissue over the belly and border of the SCM. Two equally effective approaches to the internal jugular vein are described: The central and posterior approaches. For the central approach, a 21-gauge ÒseekerÓ needle is introduced approximately 1 cm lateral to the carotid pulse into the belly of the SCM and advanced toward the ipsilateral nipple at a 45-degree angle. 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. Constant negative pressure is exerted on the syringe, and entry into the vein is confirmed by the return of venous blood. The vein should be entered within 5 to 7 cm with both approaches. If the vein is not entered, the needle should be withdrawn just below the surface of the skin and redirected for another attempt. Following venous access with the seeker needle, an 18-gauge needle or needle/catheter is then introduced just inferior to the seeker needle and advanced along the same path until venous blood is aspirated. Under ultrasound guidance, the carotid artery and internal jugular vein are visualized and an 18-gauge needle or needle/catheter can be introduced under direct visualization. If a needle/catheter system is used, the needle is withdrawn. Confirmation of venous cannulation can be obtained by sonography or by attaching tubing to the needle or catheter and demonstrating venous, and not arterial pressure. The Seldinger technique is employed, whereby a flexible guidewire is passed into the vein through the 18-gauge needle or catheter, and the needle or catheter is removed over the wire. It is important to maintain control of the guidewire at all times. A nick is then made in the skin at the puncture site with a no. 11 blade to allow passage of the dilator. The dilator is threaded over the wire and into the subcutaneous tissue 1 to 2 cm in order to provide a tract through the subcutaneous tissue. The dilator is removed over the guidewire. The catheter is introduced and advanced to 12 to 20 cm so that its tip is at the junction of the superior vena cava (SVC) and the right atrium. The guidewire is then removed. Aspiration of blood from all ports and subsequent flushing with saline confirm that the catheter is positioned in the vein and that all 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 radiograph is obtained to confirm the location of the catheter tip and to rule out the presence of a pneumothorax.

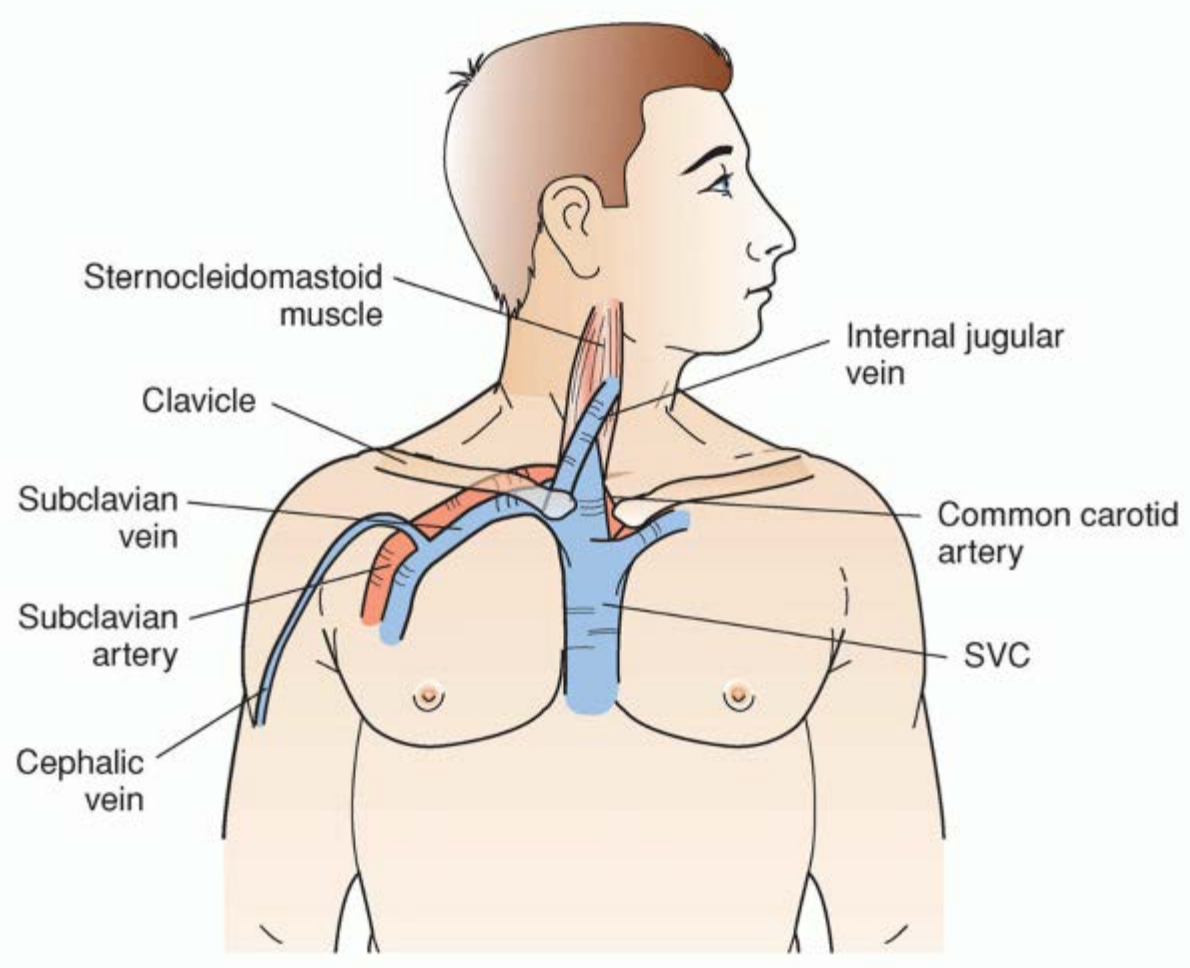


Figure 14-1 Anatomy of the upper chest and neck, including the vasculature and important landmarks. SVC, superior vena cava.

3. Complications

a. Pneumothorax. All percutaneously placed neck or chest catheters carry a risk of pneumothorax. Every attempt at placement of a central venous catheter should be followed by an erect chest radiograph before the catheter is used or catheter placement is attempted at another site. A small pneumothorax may be observed with serial chest radiographs. Unstable hemodynamics, worsening respiratory status, or expanding pneumothorax mandates tube thoracostomy placement.

b. Carotid artery injury. Carotid artery puncture complicates internal jugular cannulation in as many as 10% of cases, representing 80% to 90% of all insertion-related complications. Inadvertent carotid artery puncture is usually tolerated in the noncoagulopathic patient and

treated by direct pressure over the carotid artery. Although carotid artery puncture is usually benign, it can be life-threatening when it results in inadvertent intraarterial cannulation, stroke, hemothorax, or carotid artery-internal jugular vein fistula. If the dilator or catheter is 7 French (F) or smaller, it can usually be

removed and direct pressure held over the carotid puncture site without further detrimental sequelae. Catheters larger than 7F should be removed in a setting in which operative repair of the arteriotomy can be performed.

c. Venous stenosis. 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 collateralization, stenosis or thrombosis is usually well tolerated, but consideration must be given to the possible need for future dialysis access.

d. Other. Air embolus, perforation of the right atrium or ventricle with resultant hemopericardium and cardiac tamponade, and injury to the trachea, esophagus, thoracic duct, vagus nerve, phrenic nerve, or brachial plexus can all complicate the placement of central venous catheters.

C. Subclavian Vein Approach

1. Indications. The subclavian approach is generally most comfortable for the patient and easiest to maintain. A meta-analysis in 2012 found no difference in catheter-related bloodstream infection (CRBSI) between internal jugular, subclavian, and femoral sites (*Crit Care Med.* 2012;40:2479-2485). However, the Centers for Disease Control and Prevention's (CDC) guidelines continue to recommend subclavian access as the preferred site in patients at risk for CRBSI. In the presence of an open wound, tracheostomy, and tumors of the head and neck, CVAD should be placed in the subclavian position to minimize infection risk. In patients with renal failure or at risk for renal failure, subclavian access should be avoided to minimize the risk of central stenosis.

2. Technique. The patient is placed in the Trendelenburg position with a rolled towel between the scapulas, allowing the shoulders to fall posteriorly. The skin is prepped, draped, and anesthetized in the infraclavicular space near the middle and lateral third of the clavicle down into the deep soft tissue and to the periosteum of the clavicle. An 18-gauge needle is introduced below the middle third of the clavicle. The needle is advanced deep to the clavicle and parallel to the plane of the floor toward the sternal notch. Constant negative pressure is applied to the syringe. Once the needle enters the subclavian vein, the guidewire, the dilator, and the catheter are introduced by the Seldinger technique. All catheter ports are aspirated and flushed to ensure functionality. A chest radiograph is obtained to confirm the location of the catheter tip and to evaluate for pneumothorax.

3. Complications. The complications of subclavian access include those described in the previous section. The risk of pneumothorax is higher with this approach than with the internal jugular approach. Because the clavicle prevents the application of direct pressure to achieve hemostasis, puncture of the subclavian artery can be troublesome. Therefore, this approach

should be avoided in the patient with uncorrectable coagulopathy. If the artery is punctured, the patient should have close hemodynamic monitoring for the next 30 to 45 minutes. Inadvertent

cannulation of the subclavian artery with the dilator or catheter is a potentially fatal complication. The device should be left in place and angiography performed. Removal of the catheter should be done in the operating room so that open arteriotomy repair may be performed if necessary. Left-sided subclavian catheter placement poses the risk of injury to the thoracic duct, brachiocephalic vein, and SVC with the needle or dilator.

D. Femoral Vein Approach

1. Indications. The femoral vein can be used for obtaining central access in an emergent situation, and does not interfere with the other procedures of cardiopulmonary resuscitation. Therefore, it is the preferred approach during trauma or cardiopulmonary resuscitation as long as there is no injury to the inferior vena cava or iliac veins. A femoral vein catheter does not reach the central circulation and therefore may not be ideal for the administration of vasoactive drugs. The femoral vein catheter inhibits patient mobility, and maintaining sterility at the site of entry in the groin can be difficult. For these reasons it should not be used in elective situations except when upper-extremity and neck sites are not available. Catheters placed at any site during a medical emergency or code when sterile technique cannot be assured should be replaced within 48 hours of insertion to minimize the risk of CRBSI.

2. Technique. The skin is prepped and draped. The subcutaneous tissue medial to the femoral artery and inferior to the inguinal ligament is anesthetized. The pulse of the femoral artery is palpated below the inguinal ligament (Fig. 14-2), and an 18-gauge needle or needle/catheter is introduced medial to the pulse at a 30-degree angle. It is directed cephalad with constant negative pressure until the vein is entered. Once the needle enters the femoral vein, the guidewire, the dilator, and ultimately the catheter are inserted using the Seldinger technique. 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, with the vein lying 1 to 2 cm medial to this point. Once the catheter is successfully placed, all ports are aspirated and flushed to ensure that they are functional.

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 also be damaged. Injury to the inguinal lymphatic system can result in a lymphocele. The possibility of injuring peritoneal structures also exists 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 femoral vein thrombosis.

E. Prevention and Treatment of CRBSIs. CSRBs occur in 1 to 5 patients per 1,000 CVC days, cause an increase in total hospital days, and have an attributable cost ranging from $4,000 to

instituted together, hand hygiene, chlorhexidine-based skin preparation, maximal barrier precautions, avoidance of the femoral vein for insertion, and daily review of the necessity for and removal of all unnecessary central venous catheters have been demonstrated to result in an up to 66% reduction in CRBSI (*NEJM.* 2006;355:2725-2732). Other options for prevention include antiseptic/antibiotic impregnated catheters and chlorhexidine impregnated sponge dressings, but replacement (either changing position to a new site or rewiring an existing catheter after an arbitrary length of time) has not been demonstrated to decrease the incidence of catheterrelated infections.

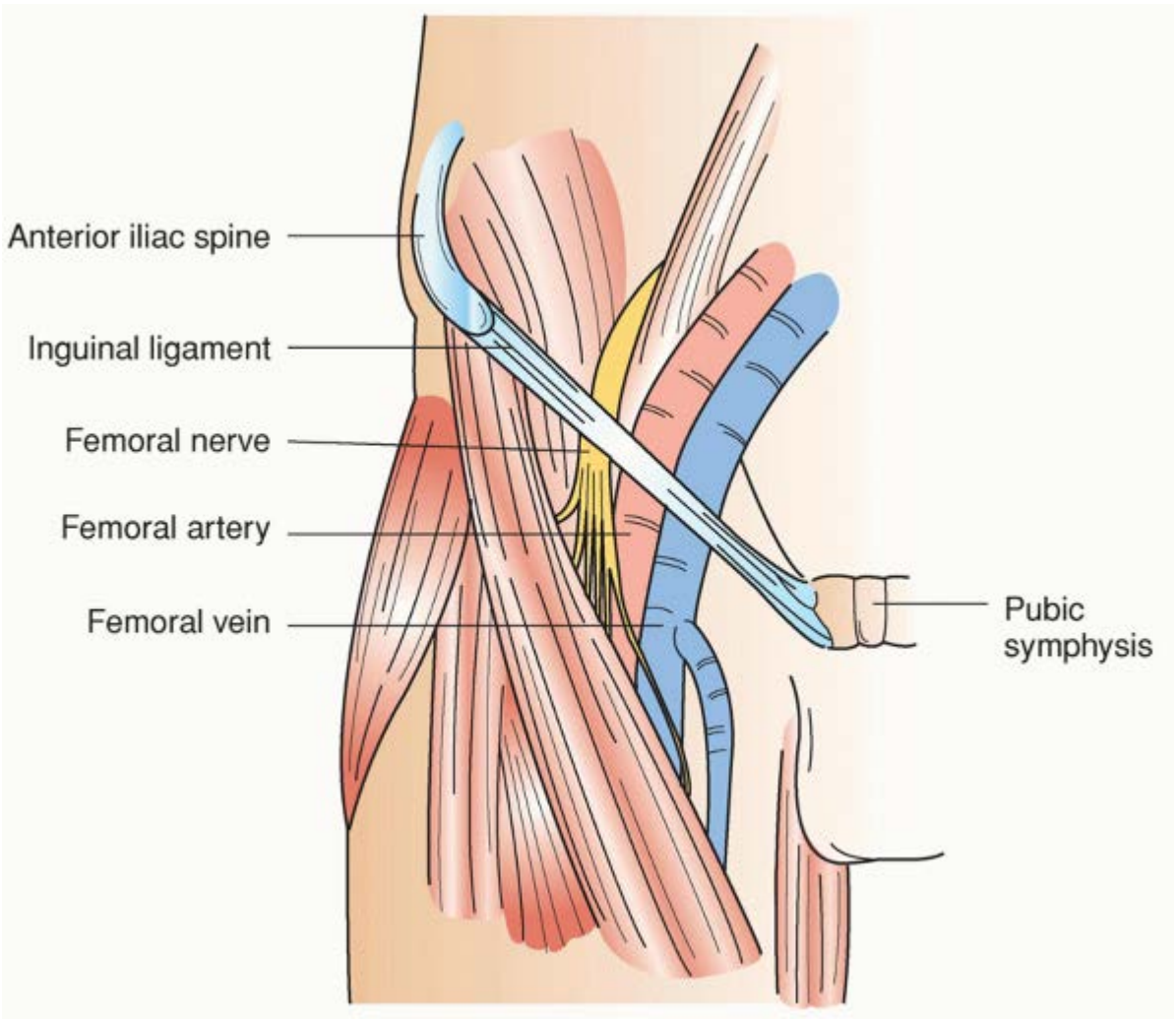


Figure 14-2 Anatomy of the femoral vessels.

CRBSIs are generally monomicrobial. The most common microorganisms isolated are coagulase-negative Staphylococci (31%), *Staphylococcus aureus* (20%), Enterococci (9%), and *Candida albicans* (9%). Catheter infections may manifest with local, regional, or systemic signs. Treatment

is based on the severity of infection, causative organism, type of catheter, and remaining options for vascular access (Fig. 14-3). In the setting of bacteremia, indwelling catheters generally should be removed and the patient should be treated with a course of systemic antibiotics. Empiric antibiotic treatment while awaiting culture results should consist of coverage against Gram-positive cocci, including methicillin-resistant staphylococci. For lowerextremity CVADs, coverage against Gram-negative bacilli and Candida should be included as well. In patients with a single culture demonstrating coagulase-negative Staphylococcus species with limited vascular access and other potential sources for infection, salvage of a short-term catheter can be attempted with broad-spectrum antibiotics and antibiotic lock therapy for 10 to 14 days. Persistent bacteremia after 72 hours necessitates catheter removal. In patients with long-term catheters for whom survival is catheter dependent and access options are limited (i.e., hemodialysis and short gut syndrome patients), catheter salvage should be attempted with systemic and antibiotic lock therapy except in the setting of severe sepsis; suppurative thrombophlebitis; endocarditis; >72 hours of bacteremia in the setting of antibiotic therapy; or infections due to *S. aureus, P. aeruginosa,* fungi, or mycobacteria.

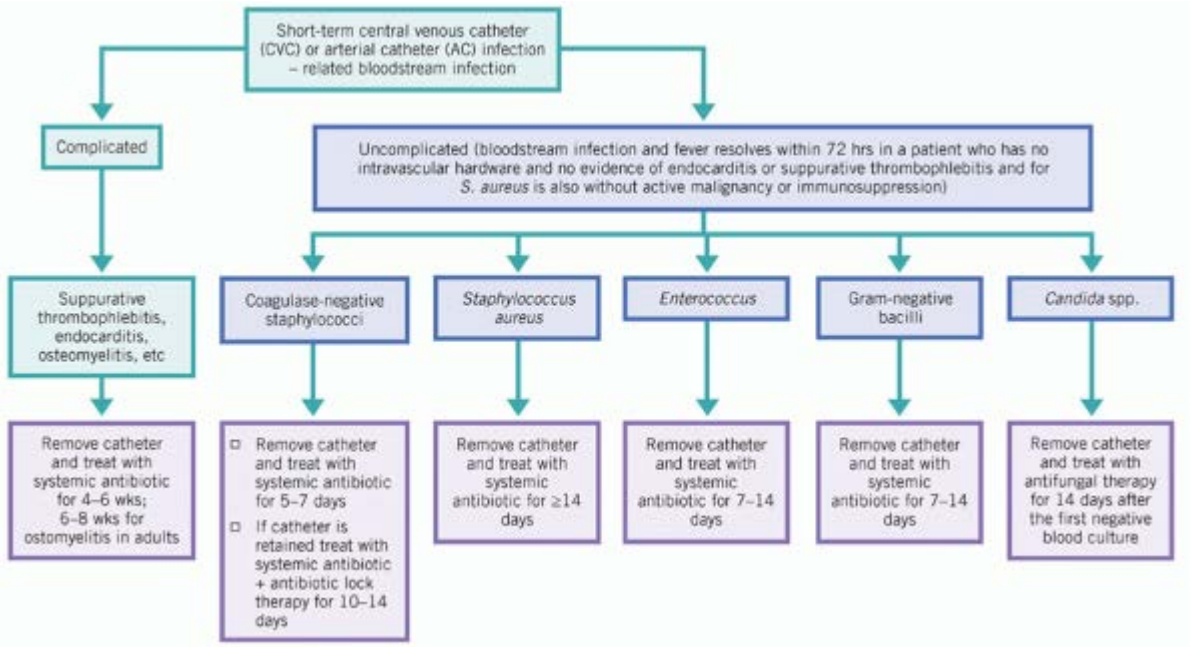


Figure 14-3 Management algorithm for treating CRBSI. (From Mermel LA, Allon M, Bouza E, et al. Clinical practice guidelines for the diagnosis and management of intravascular catheter-related infection: 2009 Update by the Infectious Diseases Society of America. *Clin Infect Dis.* 2009;49(1):1-45, with permission.)

F. Thrombosis. CVAD thrombosis may occur within the lumen, at the tip, or within the vessel beyond the catheter. For occlusions within the lumen or at the tip, recombinant tissue plasminogen activator (rt-PA) may be instilled with a dwell time of 2 hours to clear a thrombotic occlusion. Success rates are 74% after one attempt and 90% after two attempts. If this procedure fails or if the patient is symptomatic, imaging with Doppler ultrasonography or venography should be performed. In the presence of a venous thrombosis, the catheter should be removed and systemic anticoagulation should be initiated.

G. Catheter Removal. Catheters should be removed as soon as they are no longer clinically indicated given the increased risk of CRBSI with increased catheter duration. For removal of internal jugular or subclavian catheters, the patient must be placed in Trendelenburg position. To prevent an air embolus, the patient is instructed to perform the Valsalva maneuver; taking a deep breath in, holding the breath, and bearing down (to create a high intrathoracic pressure) during removal. The catheter tip should be inspected to verify that it is intact and manual pressure applied for a minimum of 5 minutes. Once hemostasis is obtained, an occlusive dressing is applied.

II. THORACIC DRAINAGE PROCEDURES

A. Thoracentesis

1. Indications. Thoracentesis can provide both diagnostic and therapeutic benefit for patients with pleural effusions. Diagnostic thoracentesis is indicated for an effusion of unknown etiology. Pleural fluid lactate dehydrogenase (LDH), protein, pH, glucose, amylase, lipid, Gram stain, culture, and cytology should be performed. Therapeutic thoracentesis is indicated to relieve respiratory compromise resulting from large pleural effusions. For recurrent pleural effusions, when repeated therapeutic thoracentesis is needed, chest tube drainage and pleural sclerosis should be considered.

2. Technique. Erect and lateral decubitus chest radiographs or equivalent imaging studies should be obtained to assess the size, location, and characteristics (free flowing or loculated) of the effusion. For free-flowing effusions, the patient is seated upright and slightly forward. The thorax

should be entered posteriorly, 4 to 6 cm lateral to the spinal column and one to two interspaces below where percussion becomes dull. Loculated effusions should be localized by ultrasonography, and the site for thoracentesis is marked on the skin. Once the site is prepped and draped, the subcutaneous tissue covering the rib below the interspace to be entered is anesthetized. The infiltration is carried deep to the periosteum of the rib. With negative pressure placed on the syringe, the needle is advanced over the top of the rib to avoid injury to the neurovascular bundle until pleural fluid is returned. It is then

withdrawn a fraction to allow for injection of lidocaine to anesthetize the pleura and lidocaine is infiltrated into the intercostal muscles as the needle is withdrawn. Most thoracentesis kits contain a long needle inserted into a plastic catheter with an attached syringe and stopcock. This apparatus is introduced along the previous tract with negative pressure applied to the syringe until fluid is returned. Aspiration of air bubbles indicates puncture of the lung parenchyma, in which case the needle should be promptly pulled back 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 container is attached to the stopcock to remove the pleural fluid. A diagnostic thoracentesis requires 20 to 30 mL of fluid for the appropriate tests; a therapeutic thoracentesis can drain 1 to 2 L of fluid at one time. Care should be taken when draining large volumes of effusions, as fluid shifts can occur and cause hemodynamic instability. A chest radiograph 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. Small pneumothoraces (i.e., <10%) are generally well tolerated and can be followed with serial radiographs. Tube thoracotomy is indicated for large pneumothoraces. Reexpansion pulmonary edema can occur in situations when a large amount of fluid is removed. Hemothorax, empyema, injury to the neurovascular bundle, laceration of the lung parenchyma, and subcutaneous hematoma are other potential complications.

B. Tube Thoracostomy

1. Indications and contraindications. Tube thoracostomy is indicated for a pneumothorax, hemothorax, recurrent pleural effusion, chylothorax, and empyema. In an emergent situation such as a tension pneumothorax, a needle thoracostomy using a 14- or 16-gauge needle inserted in the second intercostal space, midclavicular line, can allow for air decompression while awaiting tube thoracostomy.

2. Tubes. The size of the thoracostomy tube needed depends on the material to be drained. A 32F to 36F tube is used for the evacuation of a hemothorax or pleural effusion, while a smaller 24F to 28F tube is used for treatment of a pneumothorax.

3. Technique. The patient is positioned at a 30- to 45-degree angle in a semi-decubitus position by placing a bump under the affected side.

The patient's ipsilateral arm is extended and secured above the head in order to expose the axillary area. The tube should typically be placed in the “safe

triangle" delineated by the lateral border of the pectoralis major muscle, the anterior border of the latissimus dorsi muscle, and an imaginary line at the level of the nipple. With the skin prepped and draped, lidocaine is infiltrated into the fourth or fifth intercostal space at the anterior axillary line at the location of the intended incision. With negative pressure placed on the syringe, the needle is advanced slowly over the top of the rib until a rush of air or fluid is returned. The needle is then withdrawn a fraction to allow for injection of lidocaine to anesthetize the pleura, and lidocaine is infiltrated into the intercostal muscles as the needle is retracted. Posterior rib blocks should also be performed at the level of insertion and one level above and below to increase comfort in an awake patient. A 2- to 3-cm transverse incision is then made through the skin and subcutaneous tissue and a curved clamp is used bluntly to dissect an oblique tract to the rib (Fig. 14-4A). The clamp is advanced over the top of the rib to puncture and spread the parietal pleura with care taken not to stab the clamp into the lung parenchyma. 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. 14-4B). A clamp is placed at the free end of the thoracostomy tube to prevent drainage from the chest until it can be connected to a closed suction or water-seal system. The thoracostomy tube is then introduced into the pleural space (Fig. 14-4C) and is directed posteriorly or basally for a dependent effusion and apically for a pneumothorax. The tube is advanced until the last hole on the tube is clearly inside the thoracic cavity. When the tube is positioned properly and functioning adequately, it is secured to the skin and covered with an occlusive dressing to prevent air leaks. A U-stitch around the tube may be placed for use as a purse-string suture to close the tract once the tube is removed. A chest radiograph is obtained to assess for lung reexpansion and tube position. Under certain circumstances, such as the presence of loculated pleural effusions or prior thoracic surgery, radiographic guidance is required for tube placement.

4. Complications. Placement of a thoracostomy tube in the inferior aspect of the chest may result in intraperitoneal placement and inadvertent injury to abdominal organs, such as the spleen or liver. Failure to guide the tube into the pleural space can result in the tube remaining in the subcutaneous plane and failure to treat the pleural pathology. If this is suspected, anteroposterior and lateral chest radiographs should be obtained and the extrapleurally placed tube should be removed. Parenchymal, hilar injuries, or cardiac injuries 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 infectious complications

include empyema, infection along the thoracostomy tube tract, and abscess.

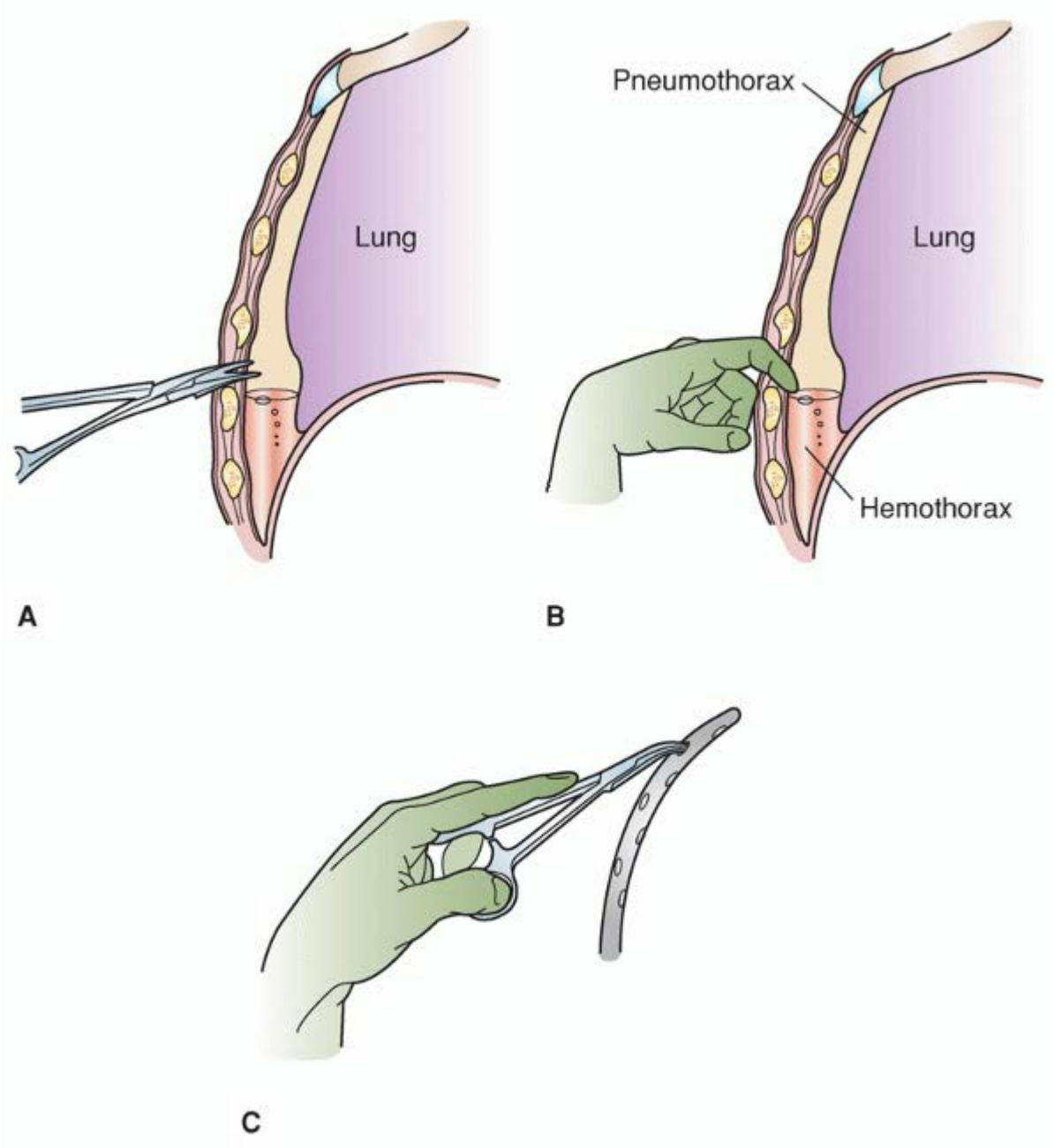


Figure 14-4 Tube thoracostomy placement. **A:** Pleural space entered by blunt spreading of the clamp over the top of the adjacent rib. **B:** A finger is introduced to ensure position within the pleural space and to lyse adhesions. **C:** The thoracostomy tube is placed into the tunnel and directed with the help of a Kelly clamp. The tube is directed posterior and caudal for an effusion or hemothorax and cephalad for a pneumothorax.

III. PERITONEAL DRAINAGE PROCEDURES

A. Paracentesis

1. Indications. Diagnostic paracentesis is most commonly indicated in the surgical patient to determine if ascites is infected. Ascites should be submitted for cell count, Gram stain, microscopy, and culture. 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, dilated bowel due to obstruction or ileus, and coagulopathy.

2. Technique. Patients should be in a supine position. The level of the ascites should be confirmed using ultrasound guidance to avoid visceral injury. Depending on the height of the ascites, a midline or lateral approach can be used. Care must be taken with the midline approach because an air-filled bowel tends to float on top of ascites. The skin at the site of entry should be prepped, draped, and anesthetized to the level of the peritoneum. For the midline approach, a needle is introduced at a point midway between the umbilicus and the pubic symphysis. For the lateral approach, the point of entry can be in the right or left lower quadrant in the area bounded 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 pubic symphysis. A diagnostic tap consists of inserting a needle or needle/catheter combination into the peritoneal cavity and aspirating 20 to 30 mL of fluid. For a therapeutic paracentesis, a needle fitted with a catheter, similar to that used for thoracentesis, allows for efficient drainage of larger volumes of ascites. With either approach, once ascites is withdrawn, the catheter is advanced over the needle and directed toward the pelvis.

3. Complications. Injuries to the bowel or bladder can occur with paracentesis and may be prevented by emptying the bladder prior to the procedure, avoiding the insertion of the needle near surgical scars, and maintaining control of the needle once inside the peritoneum. 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. It can also result in peritonitis or abdominal wall abscesses. Removal of a large amount of ascites can result in fluid shifts and hemodynamic instability.

IV. SURGICAL AIRWAY ACCESS

A. Cricothyroidotomy

1. Indications. Cricothyroidotomy is indicated when attempts at establishing translaryngeal intubation fail.

2. Technique. Most cricothyroidotomies are done in emergent situations. Therefore, an understanding of the anatomy in the region of the trachea is necessary to minimize complications (Fig. 14-5). The thyroid cartilage is easily palpated in the midline of the neck. The cricoid is the first ring inferior to the thyroid cartilage and the cricothyroid membrane joins these two cartilages. This area should be prepped, draped, and anesthetized, if possible. A vertical skin incision is made. The cricoid cartilage is identified and held firmly and circumferentially in the physician's nondominant hand until the end of the procedure. With a no. 11 or no. 15 blade, a small, 3- to 5-mm transverse incision is made over the cricothyroid membrane and carried deep until the airway is entered through the cricothyroid membrane. The tract is widened using a clamp, a tracheal dilator, or the end of the scalpel handle. A

tracheostomy tube or small (6 mm) endotracheal tube is inserted along its curve into the trachea, and the cuff is inflated. Proper position should be confirmed with end-tidal capnography.

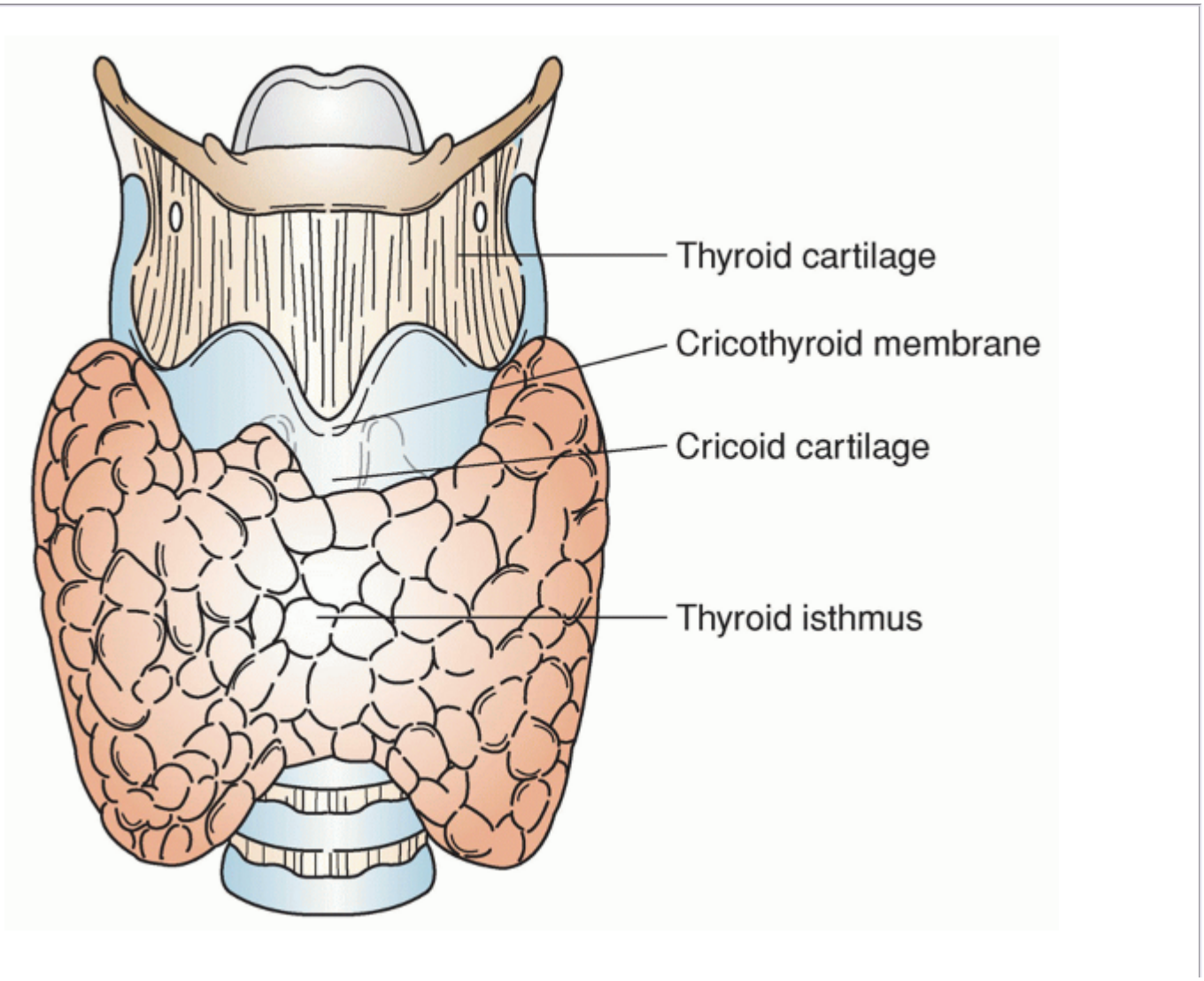


Figure 14-5 Anatomy of the larynx.

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, lack of end-tidal carbon dioxide, and the development of subcutaneous emphysema. Pneumothorax can also occur. Injury to surrounding structures can also occur in situations of urgency. Subglottic stenosis and granuloma formation are potential long-term complications.

B. Percutaneous Tracheostomy

1. Indications. Percutaneous tracheostomy (PT) has become increasingly utilized for the establishment of a nonemergent, surgical airway. The advantages of PT over surgical tracheostomy (ST) are primarily related to reduced tissue trauma and the increased likelihood of being able to perform the procedure in the ICU rather than in the operating room, thereby avoiding the need to transport the critically ill patient. Contraindications include an unstable cervical spine, inability to identify anatomic landmarks, refractory coagulopathy, and difficult oropharyngeal anatomy such that reestablishing a translaryngeal airway would be difficult in the event of airway loss. PT is an elective, not an emergency procedure.

2. Technique. PT is generally performed under bronchoscopic guidance. The patient should be adequately sedated and positioned in a moderate

degree of neck extension. An initial 1.5-cm skin incision over the first tracheal ring is made and the tissues are bluntly dissected down to the level of the pretracheal fascia using a hemostat. The existing endotracheal tube is withdrawn into the subglottic position, permitting a needle/catheter to be introduced between the first and second or second and third tracheal rings midline. Once the airway has been accessed, the needle is withdrawn, and a guidewire is inserted through the catheter and directed caudally into the trachea. The catheter is then removed. Progressive dilation of the tracheal stoma is achieved using Seldinger technique. The tracheostomy tube is introduced into the trachea over the guidewire, using a dilator as an obturator. The tracheostomy is then secured to the skin, using heavy, nonabsorbable, monofilament sutures.

3. Complications. There are no significant differences in the rate of complications between PT and ST. However, postoperative stoma inflammation and infection may be reduced with PT as compared to ST (*Crit Care.* 2014;18(6):544). The complications associated with cricothyroidotomy, including creation of a false passage, pneumothorax, injury to surrounding structures, and long-term subglottic stenosis and granuloma formation can also occur with PT.

CHAPTER 14: COMMON SURGICAL PROCEDURES

Multiple Choice Questions

1. Where would you place a central venous access in the following patient? A 38 y/o morbidly obese male in need of TPN due to prolonged ileus after small bowel resection for an incarcerated ventral hernia. His workup reveals the following laboratory results: Hemoglobin A1c, 12 mg/dL; creatinine, 2.1 mg/dL; hemoglobin, 11 g/dL; platelets 350,000/µL.

a. Right subclavian vein

b. Left subclavian vein

c. Right internal jugular vein

d. Right femoral vein

View Answer

2. Where would you place a central venous access in the following patient? A 25 y/o female who presents to the trauma bay after an MVC with multiple extremity fractures, a GCS of 2, and an unmeasurable systolic blood pressure?

a. Right subclavian vein

b. Left subclavian vein

c. Right internal jugular vein

d. Right femoral vein

View Answer

3. The patient in the above scenario has a return of spontaneous circulation after 20 minutes of CPR. She is intubated and transferred to the ICU. How should the ICU team maintain her central venous access?

a. Remove the current CVAD and replace at a new site within 48 hours of insertion.

b. Use the current CVAD until it no longer functions or the patient no longer requires central venous access.

c. Replace the dressing over the femoral CVAD every 7 days or as needed.

d. Replace the current dressing with a chlorhexidine impregnated dressing to prevent CRBSI.

View Answer

4. You have been consulted to perform a diagnostic abdominal paracentesis for a patient with colorectal cancer and liver metastases who presents with fever, leukocytosis, new onset ascites, and ileus.

Where would you insert the needle?

a. A point midway between the umbilicus and the pubic symphysis

b. The right upper quadrant 3 fingerbreadths below the costal margin in the mid-clavicular line.

c. The left lower quadrant in the area bounded by the lateral border of the rectus abdominis muscle, a line between the umbilicus and the anterior iliac spine, and a line between the anterior iliac spine and the pubic symphysis.

d. The left upper quadrant 3 fingerbreadths below the costal margin in the mid-clavicular line.

View Answer

5. You are part of the code team in the trauma bay and are placing a femoral CVAD during CPR in a 27 y/o male without a pulse. Which of these accurately describes how you will locate the correct insertion site?

a. You will rely on real time ultrasound guidance to locate the femoral vein.

b. You identify the midpoint between the anterosuperior iliac spine and the pubic tubercle, and puncture 1 to 2 cm medial to this point.

c. You identify the midpoint between the anterosuperior iliac spine and the pubic tubercle, and puncture 1 to 2 cm lateral to this point.

d. Wait until the patient has a return of spontaneous circulation. Then palpate the pulse below the inguinal ligament, and insert a 14-gauge needle lateral to the pulse at a 30-degree angle.

View Answer

6. You are placing an 8F Swan-Ganz catheter in an 82 y/o male with BP of 80/40 and have just dilated the tract prior to placement of the catheter when your attending shows you on ultrasound that you have cannulated the carotid artery. What is your next step?

a. Remove the dilator and apply pressure to the site for 10 minutes.

b. Consult vascular surgery for removal in the OR.

c. Remove the dilator and consult vascular surgery.

d. Consult vascular surgery for bedside removal and followup.

View Answer

7. In which of these patients would a RIGHT subclavian central venous device (CVAD) be absolutely contraindicated during this admission?

a. A 62 y/o female on an IV heparin drip with a decreasing cardiac output after an acute MI where cardiology has recommended pressor support.

b. A 70 y/o male with chronic renal failure on hemodialysis who presents with pulmonary edema and an infected right AV fistula.

c. A 55 y/o male with colorectal cancer currently undergoing his first round of chemotherapy via left subclavian port-a-cath who presents with osteomyelitis and requires 6 weeks of IV antibiotics.

d. A 58 y/o female with pancreatic cancer who is transferred to your hospital with fever and edema of the right arm.

View Answer

15
Acute Abdomen

Haniee Chung

Grant Bochicchio

Acute abdomen is defined as the recent or sudden onset of severe abdominal pain. It can be new pain or an exacerbation of chronic pain. A thorough history and physical examination in conjunction with selective diagnostic testing are of paramount importance in the evaluation of the patient with acute abdominal pain. Acute abdominal pain is the most common emergent general surgical problem and has a vast differential diagnosis, including both intra- and extraperitoneal processes. While the acute abdomen does not always require surgical intervention, surgical evaluation is warranted.

I. PATHOPHYSIOLOGY.

This chapter focuses on intra-abdominal causes of abdominal pain; however, it is important to be cognizant of other sources of pain arising from such sites as the abdominal wall (e.g., rectus sheath hematoma) or extra-abdominal organs (e.g., testicular torsion). Irritation of the peritoneum is responsible for the origin of pain arising from an intra-abdominal process. **Visceral pain** is poorly localized and triggered by inflammation; ischemia; and geometric changes such as distention, traction, and pressure, creating deep, dull, and vague pain. The general location of pain can correlate with the anatomic location of disease (Fig. 15-1). In contrast, **parietal pain** is in a distinct abdominal quadrant, causing sharp and severe pain that is well localized and occurs due to peritoneal irritation by localized inflammation of an organ in contact with the parietal peritoneum, chemical peritonitis from a perforated viscus, or mechanical stimulation as from a surgical incision or trauma (Fig. 15-2). Parietal pain can correlate with local or diffuse peritonitis and usually signifies the need for surgical treatment. **Referred pain** arises from a deep structure but is superficial at the painful site; examples include biliary tract pain which refers to the right inferior scapular area, renal colic referring down to the ipsilateral groin, or a ruptured aortic aneurysm or pancreatitis radiating to the back. **Epigastric:** Foregut-derived structures (stomach to second portion of duodenum, liver, biliary tract, pancreas, spleen). **Periumbilical:** Midgut-derived structures (second portion of duodenum to proximal two-thirds of transverse colon). **Suprapubic:** Hindgut-derived structures (distal transverse colon to anal verge).

II. EVALUATION.

A thorough history and physical examination with ancillary imaging and laboratory tests can guide

the diagnostic and treatment process (Fig. 15-3).

A. History of Present Illness

1. Pain characterization (Table 15-1).

a. Onset and duration

b. Character

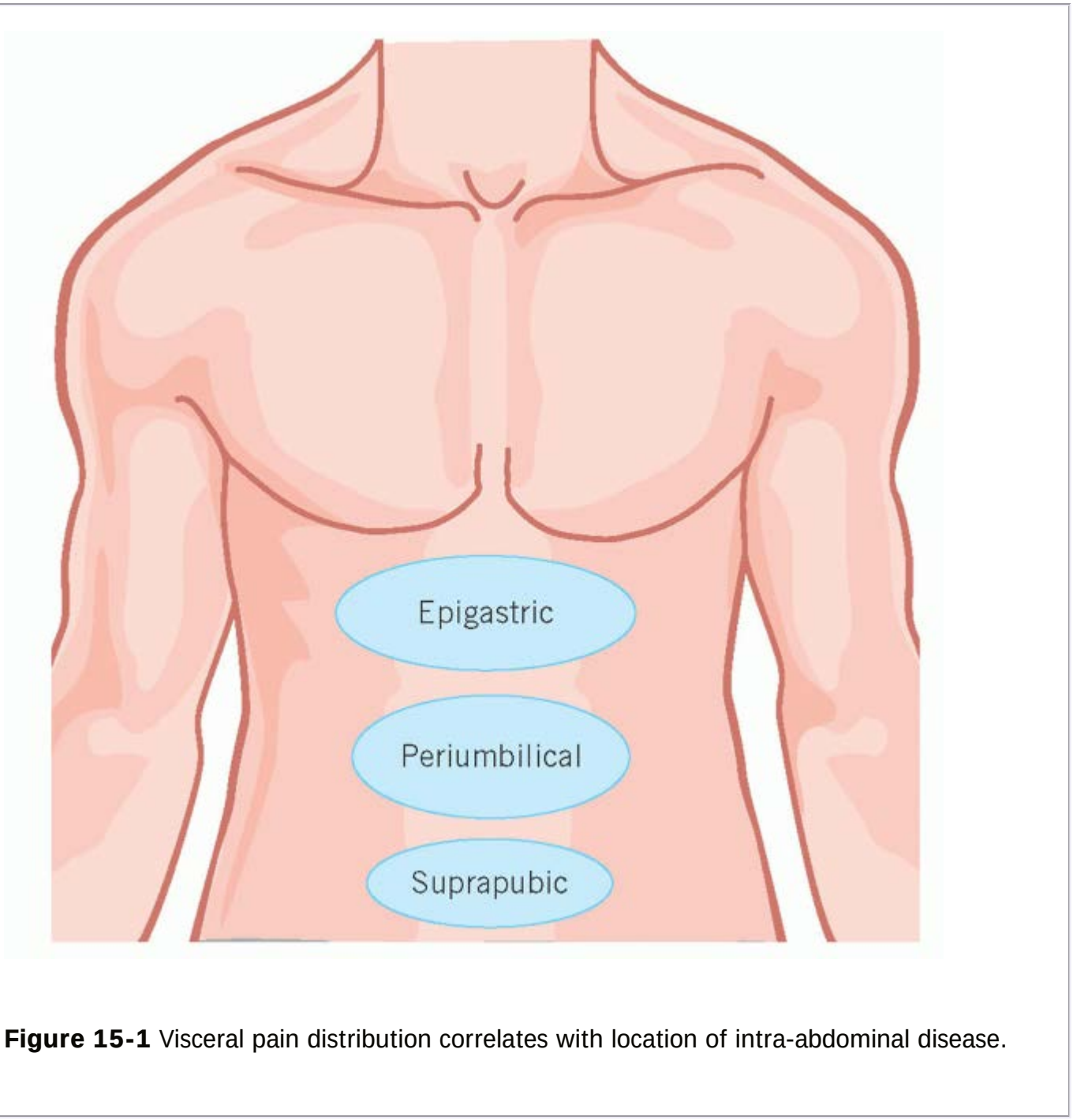


Figure 15-1 Visceral pain distribution correlates with location of intra-abdominal disease.

c. Location

d. Alleviating and aggravating factors

e. Associated symptoms

B. Past Medical/Surgical History, Review of Systems

1. Medical conditions precipitating intra-abdominal pathology

a. Peripheral **vascular disease** or coronary artery disease may predispose patients to abdominal vascular disease, such as AAA or mesenteric ischemia.

b. Cancer history should raise suspicion for bowel obstruction of perforation from progression or recurrence.

c. Major medical problems should be noted early, especially when surgical exploration is likely.

2. Organ-system review

a. History of DM, CAD, or PVD presenting with vague abdominal symptoms may have myocardial ischemia.

b. Pneumonia may present with upper abdominal pain and be associated with cough and fevers.

c. In women, a thorough **gynecologic history** is important to rule out ruptured ovarian cysts, ectopic pregnancy, and pelvic inflammatory disease.

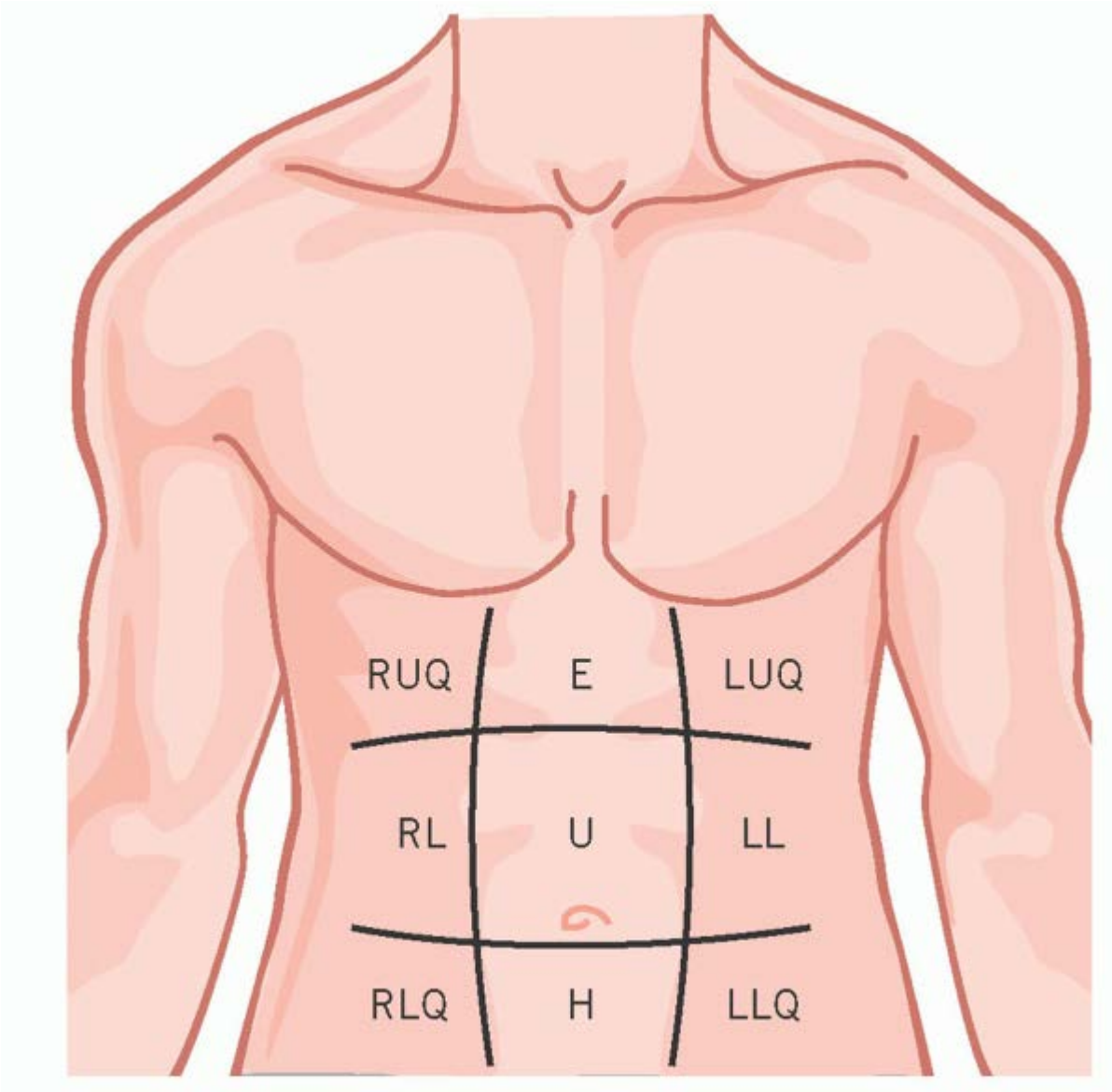


Figure 15-2 Parietal pain distribution, when localized, may correlate with inflammatory processes involving the underlying intra-abdominal structures. **RUQ,** right upper quadrant (biliary, gastric, pancreatic); **E,** epigastric (gastric, pancreatic, biliary, hernia); **LUQ,** left upper quadrant (splenic, gastric, duodenal, biliary, pancreatic); **RL,** right lumbar (renal, colonic, hernia); **U,** umbilical (pancreatic, appendiceal, gastric, small bowel, hernia); **LL,** left lumbar (renal, colonic); **RLQ,** right lower quadrant (appendiceal, colonic, pelvic, hernia); **H,** hypogastric (bladder, appendiceal, colonic, pelvic); **LLQ,** left lower quadrant (colonic, pelvic, hernia).

C. Medications

1. Nonsteroidal anti-inflammatory medications, such as aspirin or ibuprofen increase the risk of complicated peptic ulcer disease, namely, bleeding, obstruction, and perforation.

2. Corticosteroids often mask classic signs of inflammation such as fever and peritoneal signs.

3. Antibiotics may either attenuate abdominal symptoms due to treatment of the underlying disease process, or cause diarrhea/abdominal pain from antibiotic-induced pseudomembranous colitis.

D. Physical Examination

1. Overall appearance

a. Diffuse **peritonitis.** Acutely ill patients tend to lie quietly on their sides in fetal position to minimize stimulation to the abdomen

b. Colic tends to cause patients to be restless or writhing in pain, as they are unable to find a comfortable position

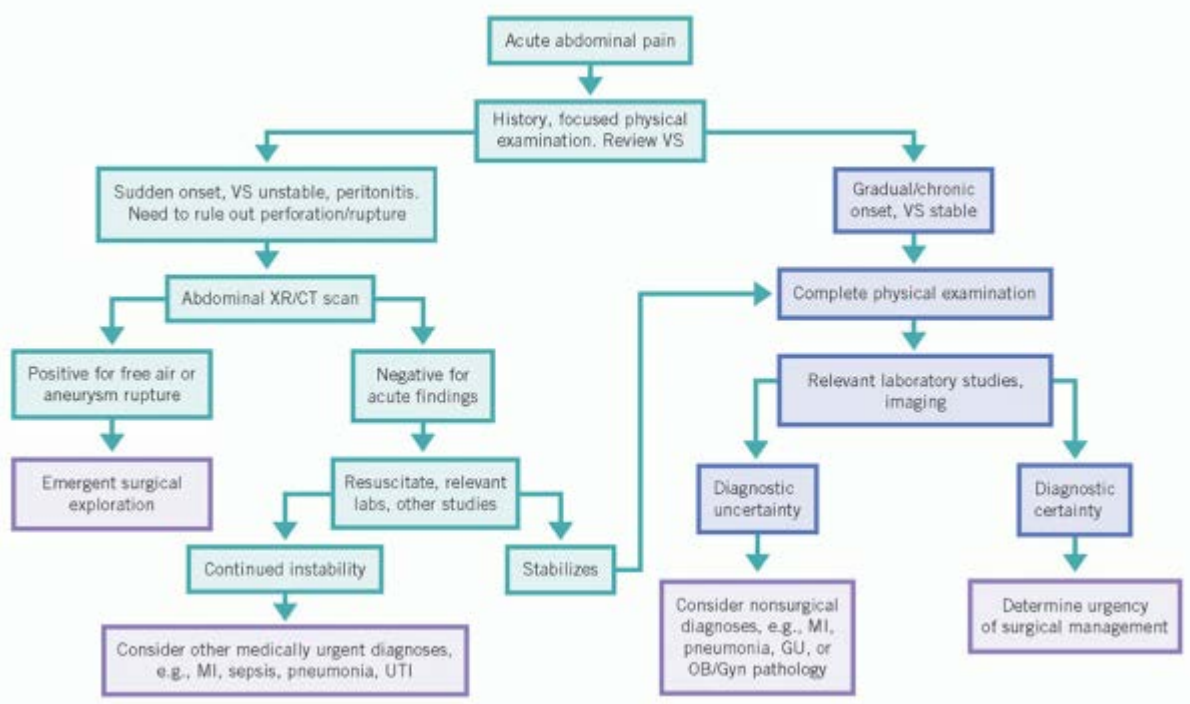


Figure 15-3 Diagnostic algorithm for the evaluation of acute abdominal pain.

TABLE 15-1 Findings to Elicit during History Taking, and Differential Diagnoses to Consider

Findings on History	Differential Diagnosis
Onset/Duration	
• Sudden (within seconds)	Perforated viscus, ruptured aneurysm, myocardial infarction, acute mesenteric occlusion
• Rapid acceleration (within minutes)	• Colic syndromes: biliary, ureteral, bowel obstruction • Inflammatory: appendicitis, pancreatitis, diverticulitis • Ischemic: mesenteric ischemia, bowel strangulation, volvulus
• Gradual (over hours)	• Inflammatory: appendicitis, cholecystitis • Obstructive: nonstrangulated bowel obstruction, urinary retention • Other mechanical: ectopic pregnancy, tumors
Character	
• Colicky, waxing, and waning	Hyperperistalsis of smooth muscle against mechanical obstruction (SBO, renal stone) Exception is biliary colicÑconstant, intense, lasting 30 min to hrs.
• Severe, persistent, steadily increasing	Infectious or inflammatory process
Location	Specific organs localizing to their respective quadrants, refer to Figure 15-2
Alleviating/Aggravating Factors	Diffuse peritonitisÑworse with movement ColicÑunable to find a comfortable position ObstructionÑtransient relief from vomiting Peptic ulcerÑtransient relief from food intake
Associated Symptoms	

• Nausea/vomiting	Vomiting after painÑappendicitis Vomiting before painÑ gastroenteritis/food poisoning BiliousÑdistal to duodenum HematemesisÑpeptic ulcer, gastritis
• Fevers/chills	Inflammation/infection
• Anorexia	Common symptom in acute abdominal pain

2. Vital signs

a. Fever suggests inflammatory or infectious process; marked fevers >39¡C suggests the presence of abscess, cholangitis, or pneumonia.

b. Hypotension and/or tachycardia signal hypovolemia or sepsis.

3. Abdominal examination should be done systematically. Analgesia administered prior to examination may alter findings, but does not decrease diagnostic accuracy (*Ann Emerg Med.* 2006;48:150).

a. Inspection should be carried out for distention, scars, masses, or skin changes.

b. Auscultation may reveal high-pitched bowel sounds of obstruction of the absence of sounds from ileus or diffuse peritonitis.

c. Percussion may reveal tympanitic sounds from bowel distention or fluid wave of ascites; it is also useful for localizing tenderness and peritoneal irritation when it is clearly present so as to expose the patient to deep palpation.

d. Palpation should be performed with the patient supine.

(1) Begin at a site remote from the reported site of pain.

(2) Note areas of tenderness and guarding.

(3) Peritonitis can be evoked by rocking the patient's pelvis or shaking the bed and assessing for pain.

(4) Pain out of proportion to examination is classic for mesenteric ischemia.

(5) Search for hernias and palpable masses.

(6) Consider referred pain patterns.

e. Rectal examination should be done routinely in all patients with suspected GI bleeding,

obstruction, or lower abdominal/pelvic pathology.

(1) Rectal mass may be an obstructing cancer; note fraction of circumference involved, mobility, and distance from anal verge.

(2) Occult blood in stool specimen indicates GI bleeding.

f. Pelvic examination must be performed in all women of child-bearing age with lower abdominal pain.

(1) Note appearance of the cervix and any discharge.

(2) Bimanual examination should be performed for cervical motion tenderness, adnexal tenderness, or masses.

g. Testicular/scrotal examination must be performed in all males with abdominal pain.

(1) Testicular torsion produces painful, swollen, and tender testicles that retract upward in the scrotum.

(2) Epididymitis may coexist with urinary tract infection (UTI), with the epididymis and vas deferens becoming swollen and tender.

E. Laboratory Evaluation

1. Complete blood count with differential

a. Leukocytosis indicates the likelihood of an infectious source.

b. Left shift on the white count differential points to an inflammatory process in the setting of a normal WBC count.

c. Hematocrit can be elevated from volume contraction due to dehydration; conversely, it may be low from occult blood loss.

2. Electrolyte profile

a. Hypokalemic, hypochloremic metabolic alkalosis classically appears in patients with prolonged vomiting and volume depletion.

b. Metabolic acidosis with a low serum bicarbonate level suggests general tissue hypoperfusion, and may suggest an underlying ischemic process.

c. Elevated BUN or creatinine suggests volume depletion.

3. Liver function panel

a. Mild transaminitis (<two times normal), elevation of alkaline phosphatase and total bilirubin are seen in acute cholecystitis.

b. Moderate transaminitis (>three times normal) in the setting of acute right upper quadrant

(RUQ) pain is most likely an obstructing stone in the common bile duct. Transaminitis precedes elevation of total bilirubin or alkaline phosphatase in the acute setting.

c. Marked transaminitis (>1,000 IU/L) is likely due to acute hepatitis or ischemia.

4. Pancreatic enzymes **amylase and lipase** are measured when pancreatitis is suspected. The degree of elevation does not correlate with the severity of pancreatitis.

a. Mild hyperamylasemia can be nonspecific, also being elevated in sialadenitis, perforated ulcer, cholecystitis, or bowel obstruction.

b. Elevation of **lipase** is more specific for pancreatic parenchymal disease.

5. Lactic acid level is measured when intestinal ischemia is suspected.

a. Serum lactate is a general indicator of tissue hypoxia.

b. Mild lactic acidosis is seen in patients with arterial hypotension.

c. Ongoing elevation despite resuscitation is concerning for progressive tissue ischemia.

6. Urinalysis assesses urologic causes of abdominal pain.

a. Bacteriuria, pyuria, and presence of leukocyte esterase suggest the presence of UTI. Recurrent UTI in males is unusual and warrants further evaluation.

b. Hematuria is seen with nephrolithiasis and renal or urothelial cancer.

7. β-Human chorionic gonadotropin must be obtained in all women of child-bearing age. A positive urine test should be followed by quantitated serum levels.

a. Low level (<4,000 mIU) usually accompanies ectopic pregnancy.

b. Higher levels (>4,000 mIU) indicate intrauterine pregnancy, usually detectable on ultrasound (US).

F. Radiographic evaluation, while an important component of a diagnostic workup, should be used selectively to minimize cost and potential morbidity to the patient.

1. Plain abdominal x-rays are often the initial radiologic evaluation.

a. Acute abdominal series consists of three x-rays: Upright chest, upright abdominal, and supine abdominal x-rays.

b. Free intraperitoneal air is best visualized on **upright chest x-rays which include both hemi-diaphragms.**

(1) If the patient is unable to assume an upright position, a left lateral decubitus x-ray should be performed.

(2) Free air may not be detected in up to 50% of cases of perforated viscus (*Am J Surg.*

1983;146(6):830-833).

c. Bowel gas pattern

(1) In SBO, small bowel dilation and air-fluid levels proximal to the obstruction can be seen, along with a paucity of gas in distal bowel. Absence of air in the rectum suggests complete obstruction.

(2) A sentinel loop may be seen adjacent to an inflamed organ representing localized ileus.

(3) The **Òbent inner tubeÓ** or **ÒomegaÓ** signs are classic for sigmoid and cecal volvulus, respectively.

d. Calcifications

(1) The majority of urinary stones (90%) contain calcium and therefore are visible on plain films. Only 15% of gallstones are calcified.

(2) Pancreatic calcifications suggest chronic pancreatitis.

(3) Aortic calcifications suggest vascular disease.

(4) Phleboliths are benign calcifications of the pelvic veins and are the most common calcifications in the abdomen. These are distinguished from renal stones by their central lucency.

e. Pneumatosis (intramural gas in the GI tract) or **pneumobilia** (gas in the biliary tree) in the absence of surgical anastomosis or sphincterotomy suggests bowel ischemia. Gas in the portal venous or mesenteric venous systems is ominous when associated with intestinal ischemia and carries a mortality rate of >75%. Portal venous gas can also be associated with benign infection that can be managed nonoperatively (*Arch Surg.* 2009;144(6):575-581).

2. US is an important diagnostic adjunct in evaluating the biliary tract and ovaries. US is portable, inexpensive, and has little associated morbidity. Body habitus, bowel gas patterns, and subcutaneous air can all limit US visibility. It is also the modality that is most operator-dependent.

a. Up to 95% of gallstones are detectable by US. Findings suggestive of acute cholecystitis include gallbladder wall thickening >4 mm, pericholecystic fluid, impacted stone, or sonographic Murphy sign. Murphy sign is inspiratory arrest due to pain while an examiner's hand or the US probe is maintained in the RUQ, and suggests the presence of inflammation. Dilation of CBD (>8 mm or >10 mm after cholecystectomy) indicates biliary obstruction.

b. Pelvic or transvaginal US should be performed in women in whom ovarian pathology or ectopic pregnancy is suspected.

c. Testicular US is an adjunct to physical examination for the diagnosis of testicular pathology such as testicular torsion, epididymitis, or orchitis.

3. Contrast studies are rarely indicated in the acute setting, but can be useful in some

situations.

a. Contrast enema can differentiate an ileus from distal colonic obstruction.

b. Water-soluble contrast agents such as Hypaque should be used to avoid barium peritonitis if there is any risk of bowel perforation.

4. Computed tomography (CT) provides a thorough evaluation of the abdomen and pelvis relatively quickly. Oral and IV contrast should be administered unless contraindicated by allergy, renal insufficiency, or hemodynamic instability. CT is the best study in patients with an unclear etiology for abdominal pain:

a. When accurate history cannot be obtained because of patient factors such as mental status changes or an atypical course.

b. When the abdominal examination findings are worrisome but not definitive for peritonitis.

c. In patients with chronic illnesses such as Crohn disease, who present with acute exacerbation.

d. When evaluating retroperitoneal structures, such as the kidneys and aorta.

e. When evaluating a patient with a history of intra-abdominal malignancy.

f. When CT angiography is necessary for the evaluation of acute mesenteric ischemia.

g. In differentiating sources of pelvic and lower abdominal pain in women.

5. Magnetic resonance imaging (MRI) provides cross-sectional imaging without exposure to ionizing radiation.

a. Acquisition takes longer and the patient must be able to lie supine for a prolonged period of time.

b. Greatest application is in pregnant women with acute abdominal or pelvic pain.

6. Radionuclide imaging studies have few indications in the acute setting.

a. Biliary radiopharmaceuticals, such as hydroxy iminodiacetic acid (HIDA) scanning can evaluate for filling and emptying of the gallbladder. Nonfilling verifies cystic duct obstruction and suggests acute cholecystitis, while also able to diagnose acalculous cholecystitis and biliary dyskinesia.

b. Radioisotope-labeled RBC or WBC scans can aid in localizing sites of bleeding or inflammation.

c. Technetium-99m pertechnetate can be used to detect a Meckel diverticulum as the tracer concentrates in the ectopic gastric mucosa lining the diverticulum. Meckel scan is most frequently performed in the pediatric population with lower GI bleed.

7. Invasive radiologic techniques may be necessary in certain clinical situations, including angiographic diagnosis and therapy for mesenteric arterial occlusion and acute GI bleeding.

III. DIFFERENTIAL DIAGNOSIS.

This section will explore the various diagnoses associated with the acute abdomen, with a special focus on the management of acute appendicitis. Other etiologies will be briefly discussed for presentation and diagnostic considerations. For details on management, readers are referred to the respective chapters.

A. Appendicitis

1. Epidemiology. Appendectomy is the most common urgent surgical procedure performed. Lifetime risk of undergoing appendectomy is 7% to 12% in the general population, with the highest incidence in the second and third decades of life.

2. Presentation. Progressive, persistent midabdominal discomfort from appendiceal obstruction and distention, stimulating visceral pain response. Anorexia and low-grade fevers ensue, and as increased appendiceal distention and venous congestion stimulate peristalsis, cramping sensation is followed by nausea and vomiting. Inflammation extends transmurally to the parietal peritoneum, stimulating somatic pain fibers, leading to localized right lower quadrant (RLQ) pain which is worsened with movement. Mild fever and tachycardia follow. Onset of symptoms to time of presentation is classically less than 24 hours for acute appendicitis.

3. Unusual presentations. Retrocecal or retroileal location of the appendix may create a separation of the appendix from the anterior abdominal peritoneum, making localizing abdominal signs absent. Irritation of adjacent structures can cause diarrhea, urinary frequency, pyuria, or microscopic hematuria. Pelvic location of the appendix may mimic gastroenteritis with diffuse pain, nausea, vomiting, and diarrhea.

4. Physical examination. Assess the abdomen away from areas of suspected tenderness. While the location of the appendix is variable, the base is usually at the level of S1 vertebral body, lateral to the right midclavicular line at **McBurney point,** two-thirds the distance from the umbilicus to the anterosuperior iliac spine. Rectal examination is performed to rule out inflammatory disease in the perirectal area and to examine for atypical presentations suggestive of pelvic or retrocecal appendix. A pelvic examination in female patients is necessary to assess for cervical motion tenderness or masses. A palpable mass is uncommon, but its presence may indicate a periappendiceal abscess or phlegmon.

a. Obturator sign. With the patient supine, hips and knees flexed, rotate the hip internally and externally. A positive test is if the patient experiences hypogastric pain during the maneuver. Its presence may indicate acute appendicitis or an obturator hernia.

b. Iliopsoas sign. With the patient in left lateral decubitus position, knees flexed, hyperextend the thigh. A positive finding is reproducible pain on the right side. It occurs in retrocecal appendicitis that irritates the iliopsoas muscle.

c. Rovsing sign. With the patient supine, palpation of the left lower quadrant (LLQ) produces pain in the RLQ, indicating an inflammatory process in the RLQ.

5. Laboratory evaluation

a. Complete blood cell count. A leukocyte count >10,000 cells/µL, with polymorphonuclear cell predominance (>75%), carries a *77%* sensitivity and 63% specificity for appendicitis (*Radiology.* 2004;230(2):472-478). The total WBC count and proportion of immature forms increase with appendiceal perforation.

b. Urinalysis is frequently abnormal in patients with appendicitis. Pyuria, albuminuria, and hematuria are common. Large quantities of bacteria, >20 WBCs or >30 RBCs per high power field suggest UTI as the source of abdominal pain. Gross hematuria should prompt a consideration of urolithiasis.

c. Serum electrolytes, BUN, creatinine. Abnormalities may indicate dehydration due to vomiting or poor oral intake, and require identification and correction.

d. Serum pregnancy test must be performed in all ovulating females.

6. Radiologic evaluation. Usually, the diagnosis of appendicitis can be made clinically without the aid of radiologic examinations, especially in young thin males. Complex cases may require diagnostic imaging.

a. US is most useful in women of child-bearing age and children, as it avoids ionizing radiation. It allows simultaneous evaluation for gynecologic pathology. Findings consistent with acute appendicitis include an appendiceal diameter >6 mm, lack of luminal compressibility, and the presence of an appendicolith. An enlarged appendix has a sensitivity of 86% and specificity of 81% (*Radiology.* 2004;230(2):472-478). Appendiceal perforation is more difficult to diagnose and is characterized by the loss of echogenic submucosa and the presence of loculated periappendiceal or pelvic fluid collection. The quality and accuracy of US are highly operator dependent.

b. CT scan has become the most commonly ordered radiographic diagnostic test. It is superior to US in diagnosing appendicitis, with a sensitivity of 94% and specificity of 95% (*Ann Intern Med.* 2004;141(7):537-546). Positive CT findings include a distended, thick-walled appendix with inflammatory streaking of periappendiceal fat, a pericecal phlegmon or abscess or appendicolith, or RLQ intraabdominal free air consistent with perforation. CT scan is especially useful for distinguishing between periappendiceal abscesses and phlegmon.

c. MRI is an alternative cross-sectional imaging that is useful in working up the pregnant patient whose appendix is not visualized on US (*Radiology.* 2006;238(3):891-899).

7. Treatment

a. Preoperative preparation involves intravenous **fluid resuscitation** to achieve adequate urine

output and correct any electrolyte abnormalities. Nasogastric suction can be helpful in patients with peritonitis. Antipyretics may be used.

b. Antibiotic therapy is generally effective in the prevention of postoperative infectious complications (e.g., wound infection, intraabdominal abscess). Preoperative initiation is preferred and coverage consists of a second generation cephalosporin. In the absence of perforation,

a single preoperative dose is adequate. In cases of perforation or gangrenous appendicitis, therapy should continue for 3 to 5 days or until normalization of leukocytosis.

c. Appendectomy is generally the required treatment for appendicitis. Surgeon preference generally drives the decision to perform a laparoscopic or open appendectomy.

(1) Laparoscopic appendectomy leads to a slightly shorter length of hospital stay, reduced postoperative pain, quicker return to function and a lower risk of wound infection (*Surg Endosc.* 2006;20(3):495-499). It is, however, balanced by a higher cost and longer operative time. A laparoscopic approach is most useful when there is diagnostic uncertainty or when patient's body habitus would require a large incision.

(a) Positioning the patient generally requires placement of a urinary catheter and tucking the left arm. Three ports are placed: 10 mm at the umbilicus, 5 mm at the LLQ, and 5 mm at the suprapubic midline. The patient is placed in Trendelenburg with the right side up to improve visualization of the RLQ.

(b) The steps of the operation may vary between surgeons, but in general involves identification of the mesoappendix, with the creation of a window in it at the base of the appendix. The mesoappendix is then divided using a vascular stapler or a sealing device. The appendix itself is then divided using an endoscopic stapler. If the appendix appears normal on diagnostic laparoscopy, another etiology is sought, such as a Meckel diverticulum, tubo-ovarian pathology, or inflammatory bowel disease. While some advocate not removing a normal appearing appendix (*Br J Surg.* 2001;88(2):251-254), most will remove the appendix if no other etiology for the abdominal pain can be identified.

(2) Open appendectomy begins with a transverse incision lateral to the rectus muscle at McBurney point. A preoperative CT scan can be helpful in adjusting the incision directly over the expected location of the base of the appendix. The external and internal oblique and transversus abdominis muscle layers are split in the direction of their fibers. The abdominal cavity is entered; the cecum is identified and the anterior taenia is tracked to the base of the appendix. The appendix is delivered into the wound and surrounding adhesions are carefully disrupted. If the appendix is normal on inspection, it is removed and alternative diagnoses are considered. If purulent fluid is encountered in the abdomen, it is sent for Gram stain and culture.

d. Drainage of **periappendiceal abscess** involves clinical judgment on timing and assessing the status of the patient. Patients with known RLQ abscesses treated with immediate appendectomy

have higher complication rates and longer hospital stays that those treated nonoperatively (*Am Surg.* 2003;69(10):829-832). Patients

with well-localized periappendiceal abscesses can be treated with antibiotics and considered for image-guided catheter drainage followed by elective appendectomy 6 to 12 weeks later (*Radiology.* 1987;163(1):23-26). This approach is successful in >80% of patients. It has also been argued that an interval appendectomy is unnecessary (*Ann Surg.* 2007;246(5):741-748).

e. Incidental appendectomy performed at laparotomy for another condition may be performed, but must only be done in a clinically stable patient able to tolerate the additional operative time. The benefit of incidental appendectomy diminishes in patients over the age of 30, as the incidence of appendicitis decreases substantially with age. Contraindications to incidental appendectomy include cecal Crohn disease, radiation treatment to the cecum, immunosuppression, and vascular grafts of bioprosthesis, as there is an increased risk of infectious complications or appendiceal stump leak.

8. Appendicitis in pregnancy

a. The incidence of appendicitis in pregnancy is 1/1,500. It is the most common nongynecologic surgical emergency during pregnancy (*Can Fam Physician.* 2004;50:355-357).

b. Evaluation of a pregnant woman with abdominal pain begins with a high suspicion, as many of the classic signs and symptoms may be attributed to pregnancy.

(1) Nausea and vomiting may be incorrectly presumed to be hyperemesis gravidarum, especially in the first trimester.

(2) Tachycardia is a normal physiologic change in pregnancy.

(3) Fever is often absent during pregnancy.

(4) Leukocytosis is common, with a WBC of 12,000 being a normal response to pregnancy. A left shift, however, is always abnormal, and necessitates further evaluation.

(5) Despite the theoretical shifting of the appendix by the gravid uterus, the most common location of pain is still in the RLQ.

c. Appendectomy during pregnancy is indicated as soon as the diagnosis is suspected. A negative laparotomy carries a risk of fetal loss of up to 3%, but in setting of a perforation and diffuse peritonitis, the risk of fetal demise reaches 35% (*Southern Med J.* 1976;69(9):1161-1163). The choice of approach for appendectomy during pregnancy has long been a debated issue. A systematic review on the topic did not reveal strong preference for either modality, and showed only low-grade evidence for possibly higher rates of fetal loss during laparoscopy (*Int J Surg.* 2014;12(11):1235-1241). Generally, a laparoscopic approach is considered as safe as open, with some alteration in technique: Insufflation pressure is set lower, between 8 and 12 mm Hg; the umbilical port is placed higher, around 6 cm above the fundus. Prophylactic intraoperative use of

tocolytics has not been shown to be effective (*J Am Coll Surg.* 2007;205(1):37-42).

9. Appendicitis in children

a. The annual incidence of appendicitis in the pediatric population increases with age until a peak in the second decade of life (*Pediatr*

Emerg Care. 1992;8:126-128). Appendicitis is the most common indication for emergent abdominal surgery in childhood. Delay in diagnosis is common, especially in younger children.

b. Appendicitis in young children can be difficult to diagnose due to the constraints in obtaining an accurate history and physical examination, and their propensity to present with **atypical symptoms.**

(1) Physical examination findings may be negative or unusual. As many as half of children lack the classic migration of pain to the RLQ, 40% do not have anorexia, and 52% do not display rebound tenderness (*Acad Emerg Med.* 2007;14(2):124-129). While the classic signs may be absent in many cases, they may still be elicited and a complete examination needs to be performed.

(2) Laboratory findings may demonstrate an elevated WBC or the percentage of neutrophils. Pyuria may be present in 7% to 25% of patients with appendicitis (*Ann Emerg Med.* 1991;20(1):45-50).

(3) Imaging. The shift toward using CT as the initial diagnostic modality has not yielded a corresponding decline in the rates of negative appendectomy, while perforation rate remains as high as 33% (*J Pedatr Surg.* 2004;39(6):886-890). This in addition to the growing concern over ionizing radiation and its negative health effects, greater emphasis is being placed on the role of US as the initial modality of choice. The absence of nausea, lack of maximal tenderness in the RLQ and the absolute neutrophil count <6.7 had a negative predictive value of 98% for identifying children who could be observed or discharged without imaging studies (*Pediatrics.* 2005;116(3):709-716).

10. Complications of appendicitis

a. Perforation is usually accompanied by pain and fever. It is unusual during the first 12 hours, but is often present in patients younger than 10 years and older than 50 years. Acute consequences of perforation included fever, tachycardia, and generalized peritonitis. Treatment is appendectomy, peritoneal irrigation, and broad-spectrum antibiotics for 3 to 5 days or until resolution of fever and leukocytosis.

b. Postoperative wound infection rate can be decreased by the administration of IV antibiotics before skin incision. The incidence of **wound infection** increases from 3% in nonperforated appendicitis to 4.7% in perforated or gangrenous appendix. Wound infections are managed by opening, draining, and packing the wound to allow healing by secondary intention. IV antibiotics are indicated for cellulitis or systemic sepsis.

c. Intra-abdominal or pelvic abscesses occur most commonly in the setting of perforation, and are best managed by percutaneous imageguided drainage. If the abscess is inaccessible or resistant to drainage, operative drainage may be necessary. Antibiotic therapy can mask but does not treat or prevent a significant abscess. Patients with persistent fever or leukocytosis beyond postoperative day 7 should

have a CT scan to evaluate for an abscess (*Cochrane Database Syst Rev.* 2005;20(3):CD001439).

d. Other complications

(1) Small bowel obstruction is four times more common after surgery in cases of perforated appendicitis than in uncomplicated cases.

(2) Enterocutaneous fistulae may result from an appendiceal stump leak. Occasionally, these require surgical closure but most close spontaneously.

(3) Pylephlebitis, or septic portal vein thrombosis, is caused by *Escherichia coli* and presents with high fevers, jaundice, and can lead to hepatic abscesses. CT demonstrates thrombus and gas in the portal vein. Prompt operative or percutaneous treatment of the primary infection is critical, along with broad-spectrum IV antibiotics.

B. Acute Cholecystitis

1. Presentation

a. Classically, an antecedent history of **biliary colic** presenting as epigastric or RUQ pain after a fatty meal is endorsed.

b. The typical presentation is epigastric or RUQ pain, nausea/vomiting 4 to 6 hours postprandially.

c. Physical examination is characterized by RUQ tenderness and positive **Murphy sign.**

2. Ancillary tests

a. Laboratory tests may reveal leukocytosis and slight elevation in liver enzymes. An elevated bilirubin should alert to the possibility of an obstructing process in the common bile duct.

b. Radiologic studies include **US** looking for gallstones, gall bladder wall thickening, pericholecystic fluid, sonographic Murphy sign, bile duct size, and diisopropyliminodiacetic acid scan showing nonfilling of the gallbladder.

C. Acute Pancreatitis

1. The most common etiology is **alcohol** consumption, with **gallstones** accounting for the majority of remaining cases. Others include **post-ERCP** complication, **medications,** and **hypertriglyceridemia.**

2. Presentation

a. Severe epigastric pain radiating to the back

b. Examination findings are characterized by epigastric tenderness and varying degrees of tachycardia, fever, and hypotension, depending on the severity of the episode.

c. The spectrum of severity ranges from mild peripancreatic edema to pancreatic necrosis with infection.

d. Severity of the episode and prognosis may be estimated using grading schemes such as **Ranson criteria** and the APACHE II score.

3. Ancillary studies

a. Laboratory studies demonstrate elevation of amylase, lipase, and serum transaminases; the degree of elevation do not correlate with severity.

b. Plain x-rays may reveal a sentinel loop or pancreatic calcifications consistent with chronic pancreatitis.

c. CT scan with IV contrast is necessary in severe cases in order to identify **pancreatic necrosis or fluid collection.** These are optimally obtained 3 days after acute onset.

D. Perforated Peptic Ulcer

1. Duodenal ulcers are more common than gastric ulcers.

2. Presentation

a. Perforated ulcers are associated with the chronic use of nonsteroidal anti-inflammatory medications.

b. Most patients report a history consistent with peptic ulcer disease.

c. Perforated ulcers typically present as sudden onset, severe epigastric pain that progresses to peritonitis.

d. Physical examination is remarkable for diffuse abdominal tenderness, rigidity, and peritonitis.

3. Plain x-rays usually reveal free intraperitoneal air.

E. Intestinal Obstruction

1. Small bowel obstruction

a. The most common cause is **adhesions** from prior surgery; others include incarcerated **hernias, cancer, intussusceptions,** and **volvulus.**

b. Usually present as sharp, crampy periumbilical pain with intervening pain-free periods; often associated with nausea, vomiting, and obstipation.

c. Examination is notable for abdominal distention, high-pitched or tinkling bowel sounds, and a variable degree of abdominal tenderness.

d. Plain x-rays show **dilated loops of small bowel,** air-fluid levels, and **paucity of gas distally** in the colon and rectum. Proximal obstruction may not result in much dilated bowel loops on plain films, and may require a contrast study for diagnosis.

2. Large bowel obstruction

a. Etiologies include cancer, diverticulitis, volvulus, stool impaction, and pseudo-obstruction.

b. Presenting symptoms include constipation, **abdominal distention,** and varying degrees of abdominal pain.

c. Examination may reveal abdominal distention or a mass on rectal examination.

d. Plain x-rays reveal colonic distention. A contrast enema is necessary to rule out an obstruction mass in the colon or rectum.

e. The risk of colonic perforation increases with a cecal diameter exceeding 12 cm.

F. Mesenteric Ischemia

1. Etiologies include superior mesenteric artery **thrombosis,** severe vascular disease, or an embolic process from a cardiac source such as **atrial fibrillation.**

2. Presents as sudden onset of severe, constant abdominal pain associated with vomiting and diarrhea.

3. Physical examination often reveals pain out of proportion to examination.

4. Laboratory findings show leukocytosis and lactic acidosis.

5. Angiography may confirm the diagnosis; however, radiologic studies are not indicated if peritonitis is present on physical examination.

G. Ruptured AAA

1. Presents as sudden onset of abdominal pain with varying manifestations of radiation to the flank or back.

2. Free intra-abdominal rupture has a high mortality rate prior to presentation; contained ruptures or leaks may present with shock.

3. Physical examination is notable for a tender, pulsatile abdominal mass.

4. Plain x-rays may show calcification in the aortic wall. **CT scan** is the gold standard for diagnosis, but only performed in hemodynamically stable patients.

5. Patients with **hypotension from a known aneurysm** should be taken emergently to the

operating room without further workup. Induction of anesthesia and ensuing hypotension should be delayed until the patient is prepped and draped for quick access to the abdomen.

CHAPTER 15: ACUTE ABDOMEN

Multiple Choice questions

1. A 16-year-old male has a 10-hour history of periumbilical pain and anorexia that is now localized to the right lower quadrant. On examination, he has tenderness medial and superior to the anterior superior iliac spine. Which of the following explains the localized nature of his pain?

a. Localized ileus from appendiceal inflammation

b. Inflammation of the visceral peritoneum

c. Localized pain is unequivocal for perforation

d. Appendiceal luminal distention

e. Irritation of the parietal peritoneum

View Answer

2. An 84-year-old male presents after becoming unresponsive approximately 2 hours after complaining of abdominal pain. What is the first necessary step in management?

a. Transfuse 2 units of uncross-matched blood

b. Obtain IV access, laboratory values, and plain abdominal x-ray

c. Intubate after induction

d. Obtain CT scan with IV contrast

e. Emergent laparotomy

View Answer

3. A 30-year-old woman is 24 weeks pregnant and presents with a 5-hour history of right lower quadrant pain. Temperature is 37.4°C, HR 120, BP 100/65. On laboratory examination, her WBC is 14, with a left shift. What is the next best diagnostic step?

a. Urinalysis

b. MRI of the abdomen, pelvis

c. US of the abdomen, pelvis

d. Transvaginal US to monitor the fetus

e. CT scan without IV contrast

View Answer

4. The above patient undergoes the appropriate diagnostic workup and is felt to have acute appendicitis. What is the next step in treatment?

a. IV Zosyn, bowel rest, and observation in a monitored obstetrics unit

b. Urgent laparoscopic appendectomy

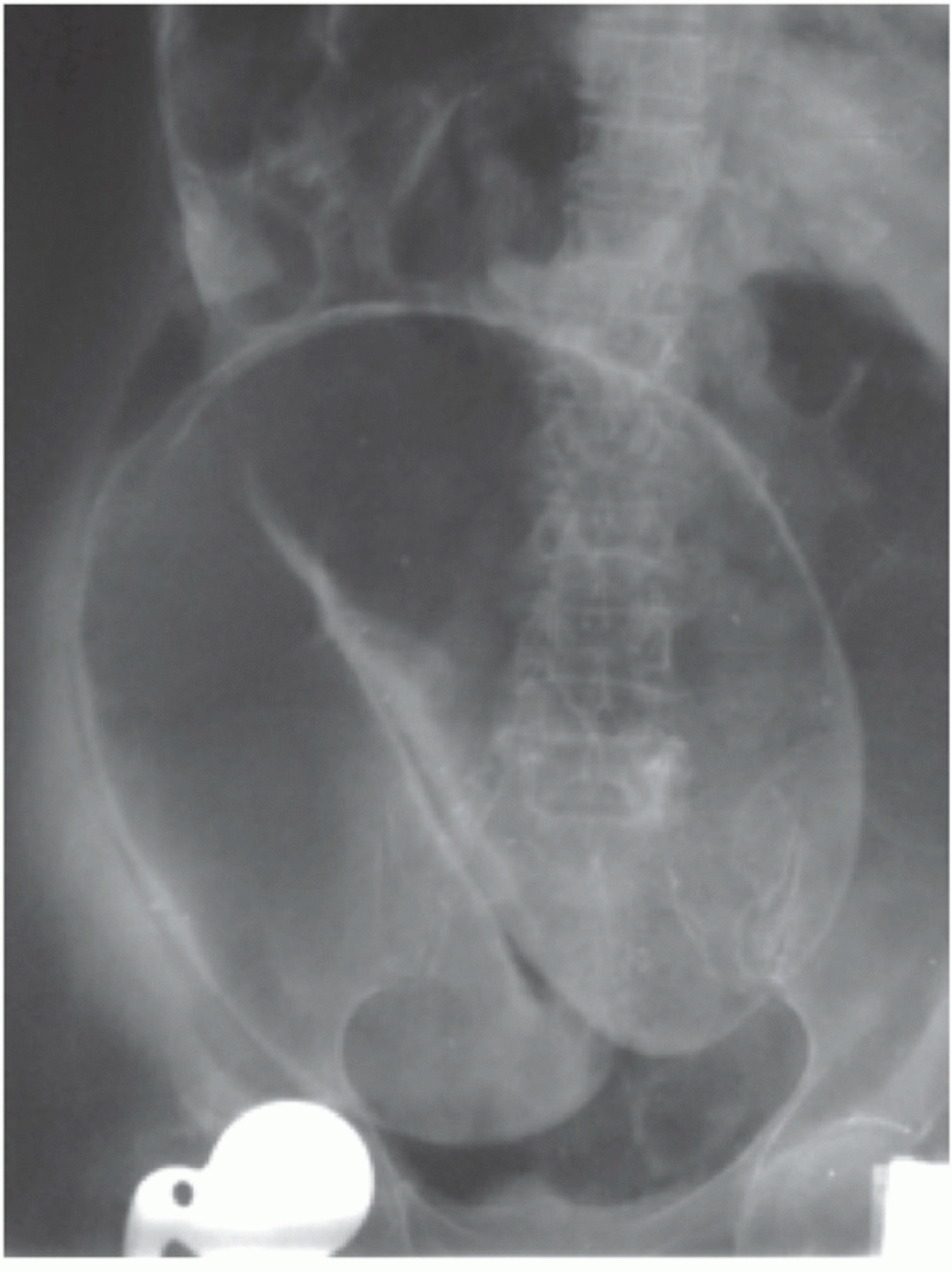
c. Tocolytic therapy prior to further treatment to protect the fetus

d. Image-guided placement of a drainage for presumed perforation

e. Fluid resuscitation and observation on a surgical floor

View Answer

5. A 74-year-old demented female nursing home resident presents after 12 hours of abdominal pain and longstanding history of constipation. Temperature is 37.8°C, HR 101, BP 140/86. A plain radiograph is obtained, which shows a dilated lucency in the midabdomen. What is the appropriate next step?



a. IV fluid resuscitation, bowel prep, colonoscopy

b. Admit to surgical floor for serial abdominal examinations, minimize narcotics

c. Exploratory laparotomy for extended right hemicolectomy

d. Barium enema

e. Immediate sigmoidoscopic reduction

View Answer

6. The above patient undergoes the appropriate initial therapeutic intervention, which is successful. What is the next step in management?

a. Admit for bowel prep and formal colonoscopy

b. Transfer to ICU with open abdomen and resuscitate

c. Admit for bowel prep and surgical resection

d. CT scan of the abdomen and pelvis to confirm successful reduction

e. Discharge on home antibiotics

View Answer

7. A 35-year-old man presents with abdominal pain and vomiting. Temperature is 38.3°C, HR 120, BP 100/54. He is uncooperative with abdominal examination, and is lying still in the fetal position. Which of the following ancillary test is most urgent in determining the cause of this patient's pain?

a. Plain chest and abdominal x-rays

b. CT of the abdomen

c. Serum electrolytes and liver function tests

d. Upper endoscopy

e. Drug screen and alcohol level

View Answer

8. A 65-year-old male with a known large abdominal aortic aneurysm who has regular interval followup presents with acute abdominal pain and altered mental status. Temperature is 37.7°C, HR 115, BP 88/56. Hemoglobin is 7.6, serum bicarbonate 18, and creatinine 1.3. What is the most appropriate next step?

a. CT scan with IV contrast to delineate aneurysm anatomy

b. Admission to ICU for resuscitation and intubation under anesthesia

c. Urgent aneurysm repair

d. US evaluation of aneurysm

e. Placement of central lines and aggressive IV fluid administration

View Answer

16
Esophagus

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STRUCTURAL AND FUNCTIONAL DISORDERS OF THE ESOPHAGUS

I. HIATAL HERNIA

A. Epidemiology. Hiatal hernias occur in roughly 10% of the population, most commonly in women 50 to 70 years old. Most hiatal hernias are asymptomatic; however, 5% of patients with a hiatal hernia have symptoms related to persistent gastroesophageal reflux disease (GERD).

B. The **type of hiatal hernia** is defined by the location of the gastroesophageal (GE) junction and the relationship of the stomach to the distal esophagus.

1. Type I (sliding) is the most common type of hiatal hernia, though usually asymptomatic. The distal esophagus and gastric cardia herniate up through the hiatus.

2. Type II (paraesophageal) is a rare manifestation; the peritoneum and greater curvature of the stomach herniate along the distal esophagus. However, the GE junction remains anchored within the abdomen.

3. Type III (combination of types I and II) is more common than pure type II and involves the herniation of both the greater curvature of the stomach and the GE junction into the chest.

4. Type IV hiatal hernias occur when abdominal organs (e.g., colon or spleen) other than or in addition to the stomach herniate through the hiatus into the chest.

C. Symptoms and complications in patients with **type I** hiatal hernias are related to reflux. **Type II, III, and IV** hiatal hernias frequently produce postprandial pain or bloating, early satiety, breathlessness with meals, and mild dysphagia. The herniated stomach is susceptible to volvulus and can develop ischemic longitudinal ulcers.

D. Diagnosis and Evaluation

1. A **chest x-ray** with an air-fluid level in the posterior mediastinum suggests a hiatal hernia. Differential diagnosis includes mediastinal cyst, abscess, or a dilated obstructed esophagus.

2. A **barium swallow** confirms the diagnosis, defines the type of hiatal hernia, and identifies coexisting abnormalities (strictures or ulcers).

3. Esophagogastroduodenoscopy (EGD) is indicated in patients with symptoms of reflux or dysphagia to assess for the presence of esophagitis, stricture, or Barrett esophagus. EGD also establishes the location of the GE junction in relation to the hiatus. A sliding hiatal hernia is defined as existing when greater than 2 cm of gastric mucosa is present

between the diaphragmatic hiatus and the mucosal squamocolumnar junction.

4. Esophageal manometry evaluates esophageal motility in patients who are being considered for operative repair to rule out an esophageal motility disorder.

5. CT **scan** can define the anatomy and guide preoperative planning for large type III or IV hernias.

E. Management

1. Asymptomatic type I hernias: No treatment.

2. Symptomatic type I hernias (GERD) should undergo a trial of medical therapy. Patients who should be evaluated for an **anti-reflux procedure** (see Section II) **and hiatal hernia repair** include patients who have **failed medical therapy,** have ongoing regurgitation or respiratory symptoms, have Barrett esophagus, and young patients who would require lifelong PPIs. Patients with atypical symptoms (chest pain or dysphagia) require further testing and continued medical therapy.

3. Patients with a **type II, III, or IV hiatal hernia** should be **considered for repair** via a thoracic or abdominal approach. Operative principles include reduction of the hernia, resection of the sac, and closure of the hiatal defect. In type III hiatal hernias, the esophagus frequently is shortened, and a lengthening procedure (Collis gastroplasty) must be considered. **Type II** repair should include a fundoplication due to a 60% incidence of associated GERD and the potential to develop GERD symptoms postoperatively secondary to intraoperative dissection.

II. GASTROESOPHAGEAL REFLUX

A. **Prevalence.** Symptoms of heartburn and excessive regurgitation are relatively common in the United States, occurring daily in 7% of the population, and monthly in 33%.

B. Pathophysiology in GERD relates to abnormal exposure of the distal esophagus to refluxed stomach contents. In 60% of patients, it is due to a mechanically defective lower esophageal sphincter (LES).

C. The classic **symptom** of GERD is posturally aggravated epigastric burning pain that is readily relieved by antacids. Other symptoms include regurgitation and dysphagia. Atypical symptoms may mimic laryngeal, respiratory, cardiac, biliary, pancreatic, or gastric disease.

D. Diagnosis and Evaluation

1. Upper GI radiography demonstrates spontaneous reflux in only approximately 40% of patients with GERD. However, it may be useful for identifying hiatal hernia or complications of reflux.

2. EGD is indicated in patients with symptoms of GERD to evaluate for esophagitis and Barrett changes. **Esophagitis** is a clinical and pathologic diagnosis.

a. Grade I: Reddened mucosa

b. Grade II: Superficial mucosal erosions and some ulcerations

c. Grade III: Extensive ulceration with luminal narrowing

d. Grade IV: Fibrotic peptic stricture

3. Esophageal manometry guides the selection of the best antireflux procedure by defining the location and function of the LES. It helps exclude achalasia, scleroderma, and diffuse esophageal spasm. Characteristics of a manometrically abnormal LES are (1) a resting pressure less than 6 mm Hg, (2) an overall length less than 2 cm, and (3) an abdominal length less than 1 cm. A patient with one or more of these abnormal values has a 90% probability of having reflux.

4. Esophageal pH testing over a 24-hour period is the gold standard in the diagnosis of GERD. A DeMeester score less than 15.7 is normal.

5. A **gastric emptying study** can be useful in evaluating redo patients (vagus nerve injury) or symptoms of gastroparesis. Patients with gastroparesis may benefit additionally from a pyloric drainage procedure (i.e., pyloroplasty or pyloromyotomy).

E. Complications. Approximately 20% of patients with GERD have complications, including esophagitis, stricture, or Barrett esophagus.

F. Treatment

1. Medical treatment aims to reduce the duration and amount of esophageal exposure to gastric contents.

a. Behavioral recommendations include remaining upright after meals for at least 1 hour, sleeping with an elevated head of the bed, and avoiding bending or straining.

b. Dietary alterations are aimed at maximizing LES pressure and decreasing stomach acidity. Patients are instructed to lose weight, eat small, frequent meals, and to stop smoking. Fatty foods, alcohol, caffeine, chocolate, peppermint, and certain medications may exacerbate reflux.

c. Pharmacologic therapy is indicated in patients who do not improve with postural or dietary measures and include **antacids, H2-receptor antagonists, and proton-pump inhibitors.**

2. Surgical treatment should be considered in patients who have symptomatic reflux despite optimal medical management and manometric evidence of a defective LES. Also, patients who have achieved relief with medical therapy but want to avoid a lifetime of medication may be candidates. However, they should be counseled that use of acid-reducing medications following surgery is not uncommon. Surgery consists of either a transabdominal or a transthoracic antireflux operation to replicate a competent LES and to keep the GE junction in the abdomen.

a. A **laparoscopic, transabdominal approach** is preferred, although the transthoracic approach may be beneficial in redo cases with a shortened esophagus.

(1) Nissen fundoplication (360-degree fundic wrap) is the most commonly performed procedure for GERD. It is very effective at preventing reflux but is associated with a higher incidence of inability to vomit, gas bloating, and dysphagia. During surgery,

care must be taken to ensure that the wrap is short, loose, and placed appropriately around the distal esophagus.

(2) The **Toupet fundoplication** is a partial 270-degree posterior wrap, with the wrapped segment sutured to the crural margins and to the anterolateral esophageal wall. It is the preferred posterior fundoplication for GERD patients, and may be considered for patients with abnormal esophageal motility due to a lower incidence of postoperative dysphagia (*Br J Surg.* 2010;97(9):1318-1330).

b. A **transthoracic approach** is a reasonable alternative in patients with esophageal shortening or stricture, coexistent motor disorder, morbid obesity, coexistent pulmonary lesion, or prior antireflux repair.

(1) Nissen fundoplication can be done via a transthoracic approach with similar results.

(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 with a 360-degree wrap. The ability to belch is preserved, thereby avoiding gas-bloat syndrome (*Surg Endosc.* 2003;17:1212).

(3) Collis gastroplasty is a technique used to lengthen a shortened esophagus (i.e., <3 cm of intraabdominal esophagus after mobilization) 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 Òneo-esophagus.Ó A gastroplasty should be considered preoperatively in patients with gross ulcerative esophagitis or stricture, failed prior antireflux procedure, or total intrathoracic stomach (*Surg Clin N Am.*

2005;85:433).

c. Complications of antireflux repairs may result from overly tight wraps or excessive tension on the repair. Most will require operative revision.

(1) Postoperative dysphagia can result from a fundoplication that is too long or tight, a misplaced or slipped fundoplication, or a complete fundoplication in the setting of poor esophageal contractile function.

(2) Recurrent reflux after surgery is increasingly common as time progresses, due to natural loosening of the wrap and may require medical therapy. However, reflux immediately after surgical repair may suggest an inadequate or disrupted repair.

(3) Gas bloating can occur if the fundoplication is too tight or if there is unrecognized gastric outlet obstruction.

III. FUNCTIONAL ESOPHAGEAL DISORDERS

A. Motor disorders of esophageal skeletal muscle result in defective swallowing and aspiration.

B. Motor Disorders of Esophageal Smooth Muscle and LES

1. Primary dysmotility

a. Achalasia is rare but is the most common primary esophageal motility disorder. Achalasia is characterized by loss of effective esophageal body peristalsis and failure of the LES to relax with swallowing, resulting in esophageal dilatation. The characteristic pathology is alteration in the ganglia of Auerbach plexus.

(1) Symptoms include progressive dysphagia (÷100%); regurgitation immediately after meals (>70%); odynophagia (30%); and aspiration with resultant bronchitis and pneumonia (10%). Some patients experience chest pain due to esophageal spasms.

(2) Diagnostic chest x-ray often shows a fluid-filled, dilated esophagus, and absence of a gastric air bubble. A **barium esophagogram** demonstrates tapering (Òbird's beakÓ) of the distal esophagus and a dilated proximal esophagus. **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** rules out benign strictures or malignancy. When these symptoms are caused by malignancy, the syndrome is referred to as pseudoachalasia.

(3) Medical treatment decreases the LES tone and includes nitrates and calcium channel blockers.

(4) Surgical treatment with a **modified Heller esophagomyotomy** has been shown to produce excellent results in >90% of patients (*J Thorac Cardiovasc Surg.* 2010;140:962). A concomitant antireflux procedure (270-degree or 180-degree fundoplication) with the esophagomyotomy helps avoid late stricture due to GERD caused by the incompetent LES (*J Clin Gastroenterol.* 2008;42:603-609).

(5) Peroral endoscopic myotomy (POEM) consists of endoscopically creating a submucosal tunnel; then dividing the circular muscle layer of distal esophagus, LES, and proximal stomach, keeping the mucosa intact. Dysphagia outcomes are equivalent to laparoscopic Heller/fundoplication, though it is associated with a higher rate of mild GERD. Postoperative complications include pneumoperitoneum, pneumomediastinum, pneumothorax, and perforation (*Adv Surg.* 2014;48:27-41).

b. Diffuse esophageal spasm is characterized by loss of the normal peristaltic coordination of the esophageal smooth muscle.

(1) The primary **symptoms** are severe spastic pain, dysphagia, regurgitation, and weight loss.

(2) The **diagnosis** is confirmed with esophageal manometry, which demonstrates spontaneous activity, repetitive waves, and prolonged, high-amplitude contractions.

(3) Treatment with calcium channel blockers and nitrates, but these may not be beneficial. Surgical treatment is very rare and may consist of a long esophagomyotomy and often a concomitant antireflux procedure.

c. Nutcracker esophagus is 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.

2. Secondary dysmotility represents the esophageal response to inflammatory injury or systemic disorders. Inflammation can produce fibrosis, which can lead to loss of peristalsis and esophageal contractility.

a. The most common cause of secondary dysfunction is **GERD,** resulting in erosive esophagitis and stricture formation. Intensive medical treatment of the reflux is essential before operation. Most surgeons prefer a Collis gastroplasty and a Toupet or Belsey antireflux procedure for these patients because of the presence of esophageal shortening and impaired peristalsis.

b. Progressive systemic sclerosis produces esophageal manifestations in 60% to 80% of patients, and often the esophagus is the earliest site of GI involvement. Smooth muscle atrophy and fibrosis results in absent contractions in the mid-distal esophagus. However, contractility is preserved within the striated muscle of the proximal esophagus.

IV. ESOPHAGEAL STRICTURES.

Esophageal strictures are either benign or malignant. **Benign strictures** are either congenital or acquired.

A. Congenital webs represent a failure of appropriate canalization of the esophagus during development and can occur at any level.

B. Acquired Strictures

1. Esophageal rings or **webs** occur at all levels. An example is **Schatzki ring,** which occurs in the lower esophagus at the junction of the squamous and columnar epitheliums due to GERD. A hiatal hernia is always present though 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 result from any esophageal injury, including chronic reflux, previous perforation, infection, or inflammation.

C. Symptoms associated with a stricture begin when the lumen narrows beyond 12 mm and consist of progressive dysphagia to solid food.

D. Evaluation and treatment of a stricture begins with the categorical **exclusion of malignancy.** The diagnosis of stricture usually is based on a **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. Most benign strictures are amenable to **dilation** to relieve symptoms, then focus is directed to correcting the underlying etiology. **Resection** can

be required for recurrent or persistent strictures or if malignancy cannot be ruled out.

V. ESOPHAGEAL DIVERTICULA.

Esophageal diverticula are acquired conditions of the esophagus found primarily in adults.

A. A **pharyngoesophageal (or Zenker) diverticulum** is a **pulsion diverticulum.** It is the most common type of symptomatic diverticulum. A hypertensive upper esophageal sphincter (UES) or uncoordinated pharyngeal contraction and opening of the UES results in increased pharyngeal intraluminal pressure. Herniation of only the mucosa and submucosa results in this false diverticulum. **Symptoms** include progressive cervical dysphagia, halitosis, cough on assuming a recumbent position, and spontaneous regurgitation of undigested food. **Diagnosis** with a barium swallow should prompt **surgical correction** with cricopharyngeal myotomy and diverticulectomy or diverticulopexy.

B. A **traction** (midesophageal or parabronchial) **diverticulum** occurs rarely in the middle third of the esophagus and is a true (full thickness) diverticulum. It occurs secondary to mediastinal inflammatory diseases (histoplasmosis or tuberculosis). Symptoms are rare, but when present, they may prompt operative excision of the diverticulum and adjacent inflammatory mass.

C. An **epiphrenic diverticulum** is associated with underlying esophageal motility disorder and can be located at almost every level but typically occurs in the **distal 10 cm** of the thoracic esophagus. Many patients are asymptomatic and the **diagnosis** is made with a contrast esophagogram, though endoscopy and esophageal function studies are needed to define the underlying pathophysiology. **Operative treatment** is indicated for patients with progressive or incapacitating symptoms and consists of diverticulectomy or diverticulopexy, along with an extramucosal esophagomyotomy.

TRAUMATIC INJURY TO THE ESOPHAGUS

I. ESOPHAGEAL PERFORATION

A. Overall, Perforation is Associated with a 20% Mortality Rate

1. Intraluminal causes

a. Instrumentation injuries represent 75% of esophageal perforations and most commonly occur at anatomical narrowings of the esophagus.

b. Foreign bodies can cause acute perforation, or more commonly follow an indolent course with late abscess formation in the mediastinum or development of empyema.

c. Ingested caustic substances, such as alkali chemicals, can produce coagulation necrosis of the esophagus.

d. Cancer of the esophagus may lead to perforation.

e. Barotrauma induced by external compression, forceful vomiting (Boerhaave syndrome), seizures, childbirth, or lifting can produce

esophageal perforation. Almost all of these injuries occur in the distal esophagus on the left side.

2. Extraluminal causes

a. Penetrating injuries to the esophagus can occur from stab wounds or, more commonly, gunshot wounds.

b. 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.

c. Operative injury to the esophagus during an unrelated procedure occurs infrequently, but may occur during spine surgery, aortic surgery, or mediastinoscopy.

B. Signs and symptoms include dysphagia, pain, and fever and quickly progress to sepsis if left undiagnosed or untreated. **Cervical perforations** may present with neck stiffness and subcutaneous emphysema. **Intrathoracic perforation** present with chest pain, subcutaneous emphysema, dyspnea, and a pleural effusion (right-sided in proximal perforations, left in distal perforations). **Intra-abdominal perforations** present with peritonitis.

C. Diagnosis of perforation is suggested by pneumomediastinum, pleural effusion, pneumothorax, atelectasis, and soft-tissue emphysema on **chest x-ray** or mediastinal air and fluid on **computed tomography (CT) scan.** Rapid evaluation with **water-soluble contrast (Gastrografin) or dilute barium contrast esophagography** (10% false-negative rate) is **mandatory.** Intramural perforation after endoscopic procedures appears to have a thin collection of contrast material parallel to the esophageal lumen without spillage into the mediastinum. **Esophagoscopy** is used primarily as an adjunctive study and can miss sizable perforations.

D. Initial management includes (1) adequate **drainage** of the leak, (2) intravenous **antibiotics,** (3) aggressive 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. Patients are kept NPO, a nasogastric tube is carefully placed, 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. Esophageal stent placement and appropriate drainage has been effective for spontaneous perforations and anastomotic leaks (*Ann Surg.* 2014;259(5):852-860).

1. Cervical and upper thoracic perforations usually are treated by cervical drainage alone or in combination with esophageal repair.

2. Thoracic perforations should be closed primarily and buttressed with healthy tissue, and the mediastinum should be drained widely. If primary closure is not possible, options include wide drainage alone or in conjunction with resection, or with exclusion and diversion in cases of severe traumatic injury to the esophagus.

3. Abdominal esophageal perforations typically require an upper abdominal midline incision to correct.

4. Perforations associated with intrinsic esophageal disease (e.g., carcinoma, hiatal hernia, or achalasia) require addressing the perforation and surgically correcting the associated esophageal disease.

II. CAUSTIC INGESTION.

Liquid alkali solutions (e.g., **drain cleaners, lye**) are responsible for most of the serious caustic esophageal and gastric injuries, producing coagulation necrosis in both organs. Acid ingestion is more likely to cause isolated gastric injury.

A. Initial management is directed at hemodynamic stabilization and evaluation of the airway and extent of injury.

1. Evaluate for airway compromise, burns may require tracheostomy.

2. Fluid resuscitation and broad-spectrum **antibiotics.**

3. Do not induce vomiting, place on NPO, and give patients an oral suction device.

B. Evaluation with **water-soluble contrast esophagography** and gentle **esophagoscopy** should be done early to assess the severity and extent of injury and to rule out esophageal perforation or gastric necrosis.

C. Management

1. Without perforation, **management is supportive,** with acute symptoms generally resolving 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.

3. Late complications include the development of **strictures** and an increased risk (×1,000) of **esophageal carcinoma.**

ESOPHAGEAL TUMORS

I. BENIGN ESOPHAGEAL NEOPLASMS.

Benign esophageal neoplasms are rare, the most common lesions are mesenchymal tumors (leiomyomas and some GI stromal tumors) and polyps.

A. Clinical features depend primarily on the location of the tumor within the esophagus. **Intraluminal** tumors, like polyps, cause esophageal obstruction, and patients present with dysphagia, vomiting, and aspiration. **Intramural** tumors, like leiomyomas, are typically asymptomatic, but can produce dysphagia or chest pain if large enough.

B. Diagnosis usually involves a combination of barium swallow, esophagoscopy, and perhaps CT scanning or magnetic resonance (MR) scan studies.

C. Treatment of all symptomatic or enlarging tumors is **surgical removal.** Intraluminal tumors can usually be removed endoscopically. Intramural tumors usually can be enucleated from the esophageal muscular wall without entering the mucosa.

II. BARRETT ESOPHAGUS.

Barrett esophagus is a complication of chronic GERD; histologically demonstrates intestinal-type metaplasia. The columnar epithelium may replace the normal squamous epithelium circumferentially, or it may be asymmetric and irregular.

A. Prevalence. Barrett esophagus is diagnosed in approximately 2% of all patients undergoing esophagoscopy and in 10% to 15% of patients with esophagitis. Most patients diagnosed with Barrett esophagus are middle-aged White men.

B. Symptoms of Barrett esophagus arise from chronic GERD including heartburn (50%), dysphagia (75%), and bleeding (25%) (*Ann Surg.* 1983;198:554).

C. Diagnosis requires endoscopy and correlation between endoscopic and histologic appearances.

D. Complications

1. Esophageal ulceration and stricture are more likely to occur in patients with Barrett esophagus than in those with GERD alone.

a. Barrett ulcers, like gastric ulcers, penetrate the metaplastic columnar epithelium. They occur in up to 50% of patients with Barrett esophagus.

b. A benign stricture occurs in 30% to 50% of patients with Barrett esophagus. The stricture is located at the squamocolumnar junction, which may be found proximal to the GE junction.

2. The metaplastic columnar epithelium of Barrett esophagus is prone to development of **dysplasia,** detected by biopsy. Low-grade dysplasia is present in 5% to 10% of patients with Barrett esophagus. **Malignant degeneration** from benign to dysplastic to malignant epithelium occurs in Barrett esophagus.

3. Adenocarcinomas above the normal GE junction are characteristic of malignant degeneration in Barrett esophagus. The risk of development of adenocarcinoma in Barrett esophagus is 50 to 100 times that of the general population; yet adenocarcinoma is still a rare event in a Barrett patient. Approximately 0.12% to 0.43% per year will progress from Barrett's to adenocarcinoma (*Best Pract Clin Gastroenterol.* 2015;29(1):125-138).

E. Treatment (Fig. 16-1)

1. Uncomplicated Barrett esophagus in **asymptomatic** patients requires endoscopic surveillance and biopsy annually or even less frequently in the absence of dysplasia.

2. Uncomplicated Barrett esophagus in **symptomatic** patients should be managed like GERD patients and should have periodic endoscopic surveillance with four-quadrant biopsies. Elimination of reflux with an **antireflux procedure** may halt progression of the disease, heal ulceration, and prevent stricture formation but will not reverse the columnar metaplasia of Barrett's.

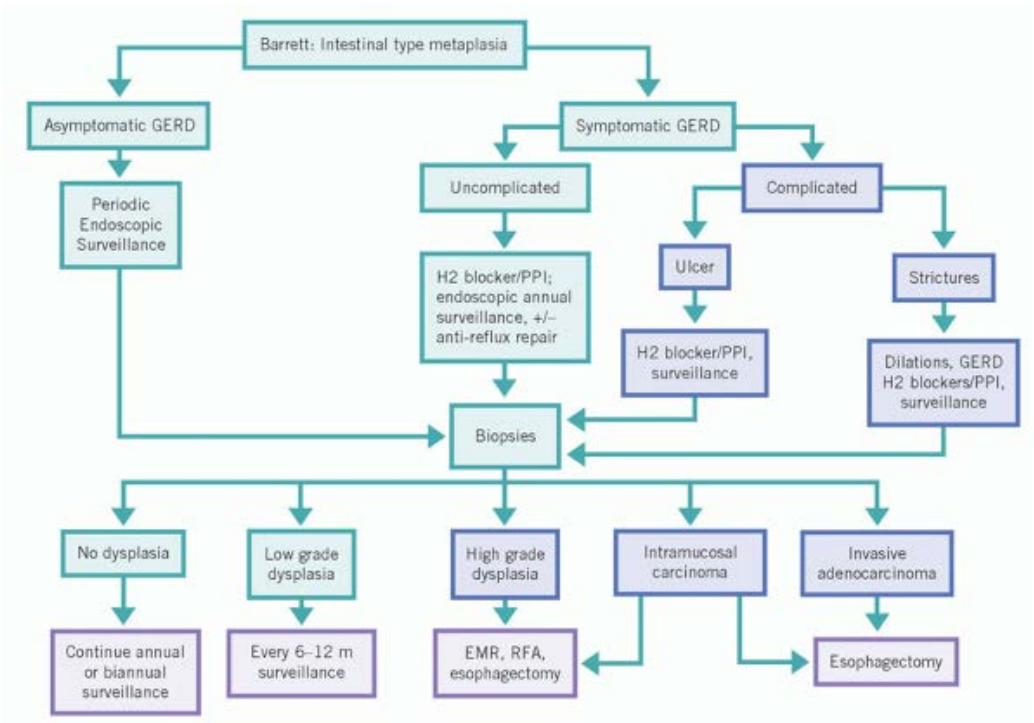


Figure 16-1 Algorithm for surveillance and management of Barrett esophagus.

3. Barrett ulcers usually heal with medical therapy. Frequently, 8 weeks of treatment with a PPI are necessary to achieve complete healing. Ulcers that fail to heal or recur despite 4 months of medical therapy are an indication for rebiopsy and antireflux surgery.

4. Strictures associated with Barrett esophagus are managed with periodic esophageal dilation combined with medical management. Recurrent or persistent strictures warrant an antireflux operation combined. Rarely, undilatable strictures require resection.

5. Dysplasia

a. Low-grade dysplasia requires frequent (every 3 to 6 months) surveillance esophagoscopy and biopsy. Medical therapy for GERD is recommended in these patients, even when asymptomatic.

b. High-grade dysplasia is pathologically indistinguishable from carcinoma *in situ* and was recently an indication for esophagectomy. Nonresective options are increasingly utilized such as endoscopic mucosal resection (EMR) and radiofrequency ablation. Resection via esophagectomy is reserved for failure of these less-invasive approaches.

6. Adenocarcinoma in patients with Barrett esophagus is an indication for esophagogastrectomy. Early detection offers the best opportunity to improve survival after resection, which is 20% at 5 years for all patients with cancer but far higher in those detected by surveillance and screening.

III. ESOPHAGEAL CARCINOMA

A. Epidemiology. Adenocarcinoma and squamous cell carcinoma of the esophagus represents 1% of all cancers in the United States.

1. Risk factors for **squamous cell carcinoma** include African American race; alcohol and cigarette use; achalasia; caustic esophageal injury; and geographic locations of China, South Africa, France, and Japan.

2. Risk factors for **adenocarcinoma** include White race, GERD, Barrett esophagus, obesity, and cigarette smoking.

B. Pathology

1. Squamous cell carcinoma is multicentric and most frequently involves the middle third of the esophagus.

2. Adenocarcinoma constitutes the majority of malignant esophageal tumors in the United States. It typically exhibits extensive proximal and distal submucosal invasion, is not multicentric, and commonly involves the distal esophagus.

3. Less common malignant esophageal tumors include small-cell carcinoma, melanoma, leiomyosarcoma, lymphoma, and esophageal involvement by metastatic cancer.

C. Most patients with early-stage disease are asymptomatic or may have **symptoms** of reflux, dysphagia, odynophagia, and weight loss. Hoarseness, abdominal pain, persistent bone pain, hiccups, and respiratory symptoms may indicate a more advanced stage. Approximately 50% of presenting

patients have unresectable primary tumors or distant metastasis at the time of diagnosis.

D. Diagnosis is suggested by a barium swallow and confirmed with esophagoscopy and biopsy.

E. Staging. Esophageal adenocarcinoma and squamous cell carcinoma are staged differently; squamous cell carcinoma has the additional variable of anatomical location (*Cancer.* 2010;116:3763) (Table 16-1). Evaluation for lymph node and distant-organ metastatic disease is performed by PET-CT. Endoscopic ultrasonography is most accurate for determining the depth of wall invasion and the involvement of peritumoral lymph nodes. Upper esophageal and midesophageal lesions require bronchoscopy to evaluate the airway for involvement by tumor.

F. Treatment

1. Surgical resection remains a mainstay of curative treatment of patients with localized disease. 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. Options for **esophageal replacement** include the stomach, colon, and jejunum.

a. Complications of esophagectomy patients include aspiration pneumonia, anastomotic leak, and atrial

fibrillation.

(1) Respiratory complications, including pneumonia, can be reduced by using retrograde drainage of the conduit (retrograde tube gastrostomy), instead of a nasogastric tube (*Ann Thorac Surg.* 2011;92(2):499-503).

(2) Management of an **anastomotic leak** is based on the size of the leak, the location of the anastomosis, and the clinical status of the patient.

(a) Cervical anastomotic leaks can usually be managed by opening the incision to allow drainage. Occasionally, the leak tracks below the thoracic inlet into the mediastinum, necessitating evaluation of ischemic injury to the stomach and wider debridement and drainage.

(b) Intrathoracic anastomotic leaks are associated with a high mortality rate and large or poorly drained leaks require operative exploration.

2. Neoadjuvant therapy with preoperative chemotherapy or chemoradiotherapy may enhance local control and resectability. Patients with dysphagia may require feeding tube placement for nutrition during neoadjuvant therapy.

3. Radiotherapy is used worldwide for attempted cure and palliation of patients with squamous cell esophageal cancer deemed unsuitable for resection. The 5-year survival rate is 5% to 10%.

4. Palliative treatment is used to relieve obstruction and mitigate dysphagia.

TABLE 16-1 TNM (Tumor, Node, Metastasis) Staging System for Esophageal Cancer

Definition of TNM

T: Primary Tumor

Tis	Carcinoma *in situ*/high-grade dysplasia
T1	Tumor invades the submucosa
T2	Tumor invades the muscularis propria
T3	Tumor invades adventitia
T4	Tumor invades adjacent structures
T4a	Pleura, pericardium, diaphragm, or adjacent peritoneum
T4b	Other adjacent structures, e.g., aorta, cerebral body, trachea

N: Regional Lymph Nodes

N0	No regional node metastasis
N1	1-2 regional lymph nodes
N2	3-6 regional lymph nodes
N3	>6 regional lymph nodes

M: Distant Metastasis

M0	No distant metastasis
M1	Distant metastasis

G: Histologic Grade

GX:	Grade cannot be assessedÑstage grouping as G1
G1:	Well differentiated
G2:	Moderately differentiated
G3:	Poorly differentiated
G4:	UndifferentiatedÑstage grouping as G3 squamous

Adenocarcinoma Stage Grouping

Stage	T	N	M	G
0	Tis	0	0	1
IA	1	0	0	1-2
IB	1	0	0	3
	2	0	0	1-2

IIA	2	0	0	3
IIB	3	0	0	Any
	1-2	1	0	Any
IIIA	1-2	2	0	Any
	3	1	0	Any
	4a	0	0	Any
IIIB	3	2	0	Any
IIIC	4a	1-s2	0	Any
	4b	Any	0	Any
	Any	3	0	Any
IV	Any	Any	M1	Any

Squamous Cell Carcinoma Stage Grouping

Stage	T	N	M	G	Location
0	Tis	0	0	1	Any
IA	1	0	0	1	Any
IB	1	0	0	2-3	Any
IIA	2-3	0	0	1	Lower
IIB	2-3	0	0	1	Upper, middle
IIIA	2-3	0	0	2-3	Lower
IIIB	2-3	0	0	2-3	Upper, middle

IIIC	1-2	1	0	Any	Any
IV	1-2	2	0	Any	Any
	3	1	0	Any	Any
	4a	0	0	Any	Any
	3	2	0	Any	Any
	4a	1-2	0	Any	Any
	4b	Any	0	Any	Any
	Any	3	0	Any	Any
	Any	Any	1	Any	Any

a. Radiotherapy and chemotherapy work best in patients with squamous cell carcinoma above the carina. Adenocarcinoma is less responsive to radiation.

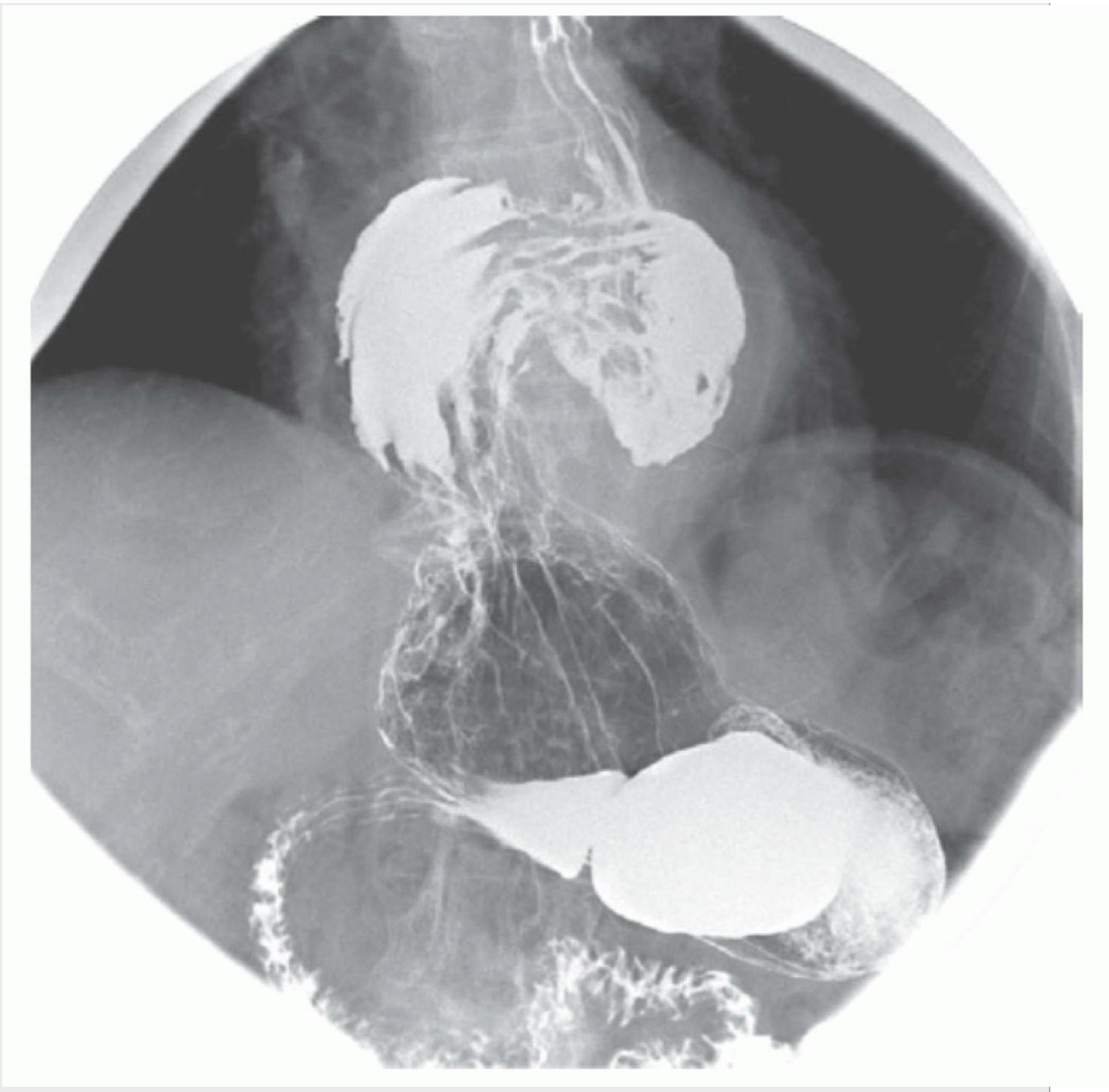
b. Intraluminal prostheses intubate the esophagus and stent the obstruction. Potential complications include perforation, erosion or migration of the stent, and obstruction of the tube.

c. Endoscopic laser techniques can restore an esophageal lumen successfully 90% of the time, with only a 4% to 5% perforation rate.

CHAPTER 16: ESOPHAGUS

Multiple Choice Questions

1. A 57-year-old female presents to her primary care physician after a recent chest x-ray showed an air-fluid level in the posterior mediastinum. She undergoes a barium swallow to further evaluate the abnormality, which shows the following image. What is her most likely diagnosis?



a. A mediastinal cyst

b. A sliding (type I) hiatal hernia

c. A paraesophageal (type II) hiatal hernia

d. A combination (type III) hiatal hernia

e. A bird's beak achalasia esophagus

View Answer

2. A 62-year-old female has a newly diagnosed type III hiatal hernia with 40% of the stomach noted to be in the chest. She presents with dysphagia, postprandial epigastric pain, and breathlessness when eating. She is evaluated for treatment.

Which of the following is the most appropriate option?

a. PPI

b. Watch and wait

c. Hiatal hernia repair with a Collis gastroplasty (lengthening procedure)

d. Hiatal hernia repair with a Dor procedure

e. Toupet fundoplication

View Answer

3. A 45-year-old male with a 5-year history of progressively worsening epigastric pain and occasional regurgitation presents to his primary care physician for further evaluation. The PMD orders an esophageal manometry study. Which of the following would indicate that the patient has an abnormal LES?

a. Intraabdominal length 0.5 cm

b. Resting pressure of LES 12 mm Hg

c. Length of LES 3 cm

d. Intraabdominal length 2 cm

e. Resting pressure of LES 20 mm Hg

View Answer

4. A 40-year-old women presents to her primary care physician complaining of sharp epigastric pain more than 3 days/week. She smokes 1ppd and her BMI is 32. Which of the following would be the most appropriate initial therapy?

a. Recommend a 3-month trial of: Stop smoking, losing weight, reducing fat consumption, and elevating the head of her bed.

b. Recommend a 3-month trial of: Stop smoking, eating 3 meals per day, drinking peppermint tea at the end of meals, resting in bed after meals.

c. Prescribe a PPI and recommend Tums for occasional flares.

d. Schedule her for a laparoscopic Nissen fundoplication

e. Prescribe a PPI and order an EGD, manometry study, and gastric emptying study.

View Answer

5. Which of the following procedures reduces the incidence of postoperative dysphagia in patients with GERD that also have abnormal esophageal motility?

a. Laparoscopic Nissen fundoplication

b. Transthoracic Nissen fundoplication

c. Extensive mobilization of the esophagus

d. Collis gastroplasty

e. Toupet fundoplication

View Answer

6. A young woman with esophageal neuromotor dysfunction undergoes an uncomplicated laparoscopic Nissen fundoplication. However, on POD 3 she notes

that she cannot belch and begins to feel as though she is retaining air in her stomach. Which of the following antireflux procedures is known to reduce the incidence of gas-bloat syndrome?

a. Laparoscopic Nissen fundoplication

b. Transthoracic Nissen fundoplication

c. Belsey Mark IV repair

d. Collis gastroplasty

e. Collis/Nissen fundoplication

View Answer

7. Which of the following is the most common symptom of achalasia?

a. Aspiration events

b. Pneumonia

c. Postprandial regurgitation

d. Odynophagia

e. Dysphagia

View Answer

8. A 70-year-old male with achalasia, COPD, diabetes, and a history of bowel obstruction and resection, reports that his regurgitation and dysphagia have not improved with the calcium channel blocker recently prescribed for him. Given his persistent symptoms what would be the best next step?

a. Peroral endoscopic myotomy

b. Addition of nitrates to his calcium channel blockers

c. Laparoscopic Toupet fundoplication

d. Vigorous dilation

e. Gastrostomy tube placement

View Answer

9. A 65-years-old female presents with atypical chest pain; she has been ruled out for an MI three times in the past year and is otherwise healthy. To rule out esophageal causes she undergoes a manometry study, which shows prolonged high amplitude peristaltic waves. Which of the following is the most likely diagnosis?

a. Variant achalasia

b. Achalasia

c. Nutcracker esophagus

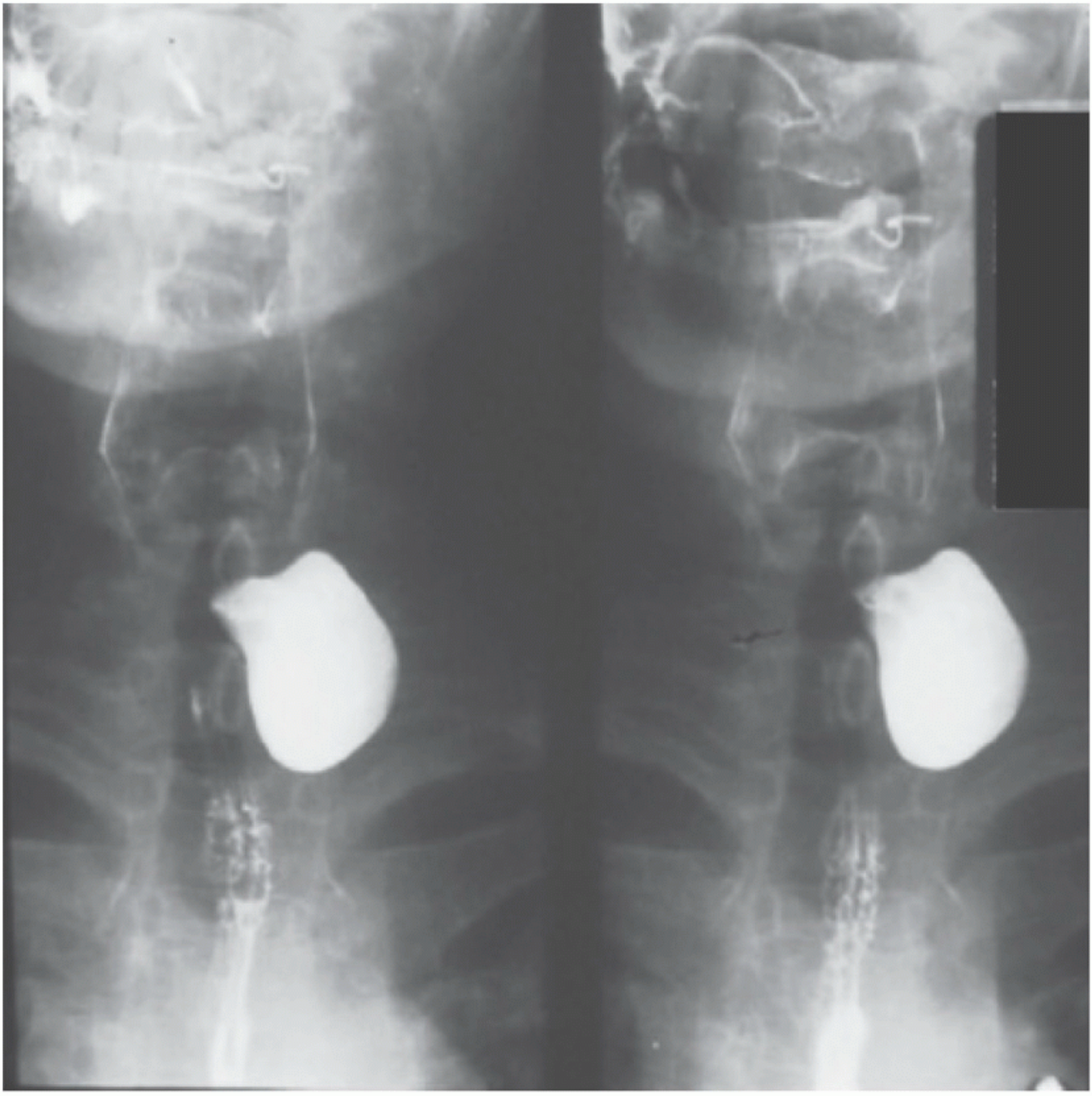
d. Esophageal spasm

e. Hiatal hernia

View Answer

10. A 50-year-old male presents to his primary care after his wife complained about

his chronic cough and constant foul-smelling breath. Upon questioning he admits that he frequently regurgitates undigested food. He undergoes a barium swallow, which shows the following. What is the most likely diagnosis?



a. Traction diverticulum

b. Achalasia

c. Epiphrenic diverticulum

d. Zenker diverticulum

e. Esophageal abscess

View Answer

11. Which of the following is the most common cause of esophageal perforation?

a. Lye ingestion

b. Cancer

c. Boerhaave syndrome

d. Iatrogenic injury

e. Trauma

View Answer

12. A 60-year-old male with a long history of poorly controlled GERD undergoes an upper endoscopy with biopsies. The biopsies show lowgrade dysplasia. What is the next step in therapy?

a. Continue medical therapy, endoscopic surveillance in 6 months

b. Add a second proton pump inhibitor medical therapy

c. Endoscopic mucosal resection

d. Radiofrequency ablation

e. Esophagectomy

View Answer

13. What is the overall 5-year survival for patients with esophageal adenocarcinoma?

a. 5%

b. 20%

c. 30%

d. 50%

e. 80%

View Answer

14. Which of the following is a risk factor for esophageal adenocarcinoma?

a. Intestinal metaplasia of the esophagus

b. Achalasia

c. African American race

d. Location: South Africa

e. Location: China

View Answer

15. Which of the following conditions can be treated by enucleation from the esophageal muscular wall?

a. Esophageal polyps

b. Esophageal leiomyomas

c. Esophageal squamous cell carcinoma

d. Esophageal adenocarcinoma

e. High-grade esophageal dysplasia

View Answer

17
Stomach

Kerri A. Serecky

William G. Hawkins

ANATOMY AND PHYSIOLOGY

The stomach is divided into five regions based on gross anatomy: The **cardia,** the portion just distal to the gastroesophageal (GE) junction; the **fundus,** the portion above and to the left of the GE junction; the **body** (or **corpus**), the largest portion and between the fundus and antrum; the **antrum,** the distal portion located between the incisura angularis and the pylorus; and the **pylorus,** a thickened ring of smooth muscle forming the boundary between the stomach and duodenum. The stomach is very well vascularized with extensive collaterals, maintaining viability if a vessel is ligated. The lesser curvature is supplied by the left gastric artery, a branch of the celiac axis, and the right gastric artery, a branch from the common hepatic artery. The greater curvature is supplied by the short gastric and left gastroepiploic arteries, branches from the splenic artery, as well as from the right gastroepiploic artery, a branch from the gastroduodenal artery. The left gastric (coronary) and right gastric veins empty into the portal vein, the left gastroepiploic vein drains into the splenic vein, and the right gastroepiploic vein drains into the superior mesenteric vein. The right and left vagal trunks provide principal innervation. The stomach serves to store and prepare ingested food for digestion and absorption by mechanical digestion and biologically active peptides.

DISORDERS OF THE STOMACH

I. PEPTIC ULCER DISEASE.

Peptic ulcer disease (PUD) is characterized by ulceration of the stomach or proximal duodenum due to an imbalance between mucosal defense and aggravating factors such as acid secretion, alcohol, and NSAIDs.

A. Epidemiology. In the United States there are approximately 300,000 new cases of PUD each year with an annual incidence of 1% to 2% and lifetime prevalence of 8% to 14%.

B. Location. Duodenal ulcers are typically located at the antralÑpylorus junction and gastric ulcers usually fall within one of five categories (Modified Johnson Classification).

1. Type 1: Body of stomach, usually lesser curvature, associated with low mucosal protection (60% to 70%).

2. Type 2: Lesser curvature and duodenal, associated with high acid secretion (15%).

3. Type 3: Prepyloric, associated with high acid secretion (20%).

4. Type 4: Proximal stomach/cardia, associated with low mucosal protection.

5. Type 5: Anywhere in stomach, medication-induced.

C. Pathogenesis

1. *Helicobacter pylori* (*H. pylori*) **infection** is associated with 90% to 95% of duodenal ulcers and 70% to 90% of gastric ulcers and produces chronic antral gastritis, increased acid and gastrin secretion, and decreased mucosal resistance to acid.

2. Nonsteroidal anti-inflammatory drug (NSAID) use increases risk of duodenal ulcers eight-fold and gastric ulcers 40-fold due to suppression of prostaglandin production.

3. Cigarette smoking.

4. Acid hypersecretion.

D. Presentation in uncomplicated disease is characterized by burning, gnawing, intermittent epigastric pain relieved by food or antacid ingestion for duodenal ulcers but exacerbated for gastric ulcers. Associated symptoms include nausea, vomiting, and mild weight loss.

E. Diagnosis is made by **esophagogastroduodenoscopy (EGD)** or barium contrast radiography. **EGD** is more sensitive and specific and offers both therapeutic and diagnostic options. Once PUD is confirmed, further testing is performed to determine its etiology.

1. *H. pylori* **infection** can be detected noninvasively by radiolabeled **urea breath test** or **serologic antibody testing.** Antral tissue can be assessed by direct **histologic examination** or rapid urease testing using the **cod liver oil (CLO) test.**

2. Fasting serum gastrin levels are obtained if the patient has no history of NSAID use, is *H. pylori* negative, or has recurrent ulcers despite adequate treatment, multiple ulcers, ulcers in unusual locations, or complicated PUD. **Zollinger-Ellison syndrome** is a rare condition that causes PUD in 0.1% to 1% of patients.

3. Endoscopic biopsy of gastric ulcers is indicated to rule out malignancy if the patient has atypical signs or symptoms (weight loss, malaise, anemia, obstruction) or if the ulcer has an atypical appearance (associated mass, folds around ulcer).

F. Treatment has shifted from primarily surgical to medical, as *H. pylori* eradication has become the cornerstone in PUD treatment.

1. Medical therapy

a. *H. pylori* eradication includes an acid-reducing medication (PPI, H2 blocker, bismuth salicylates) with two antibiotics administered for 10 to 14 days (triple therapy). These regimens

are 85% to 90% effective in eradicating *H. pylori.* Antisecretory therapy is then continued until the ulcer has healed.

b. NSAID-associated PUD is treated by discontinuing all NSAID use and starting antisecretory therapy. If the NSAID must be continued, PPIs are most effective for facilitating ulcer healing.

c. Smoking cessation promotes ulcer healing but compliance rates are low.

d. Follow-up endoscopy for gastric ulcers given risk of malignancy.

2. Surgical therapy for uncomplicated PUD is nearly obsolete given the advent of PPIs. Indications include bleeding (acute/chronic), perforation, obstruction, failure of medical therapy (intractability), and inability to exclude malignancy.

a. Duodenal ulcers

(1) Truncal vagotomy with pyloroplasty.

(2) Truncal vagotomy with antrectomy and Billroth I (gastroduodenostomy) or Billroth II (gastrojejunostomy) reconstruction: Maximal acid suppression with lowest ulcer recurrence rates (1% to 2%) but highest postoperative morbidity (15% to 30%) and mortality (1% to 2% rates).

(3) Highly selective vagotomy (HSV): Lowest postoperative morbidity (3% to 8%) and mortality rates but technically demanding and has higher recurrence rates (5% to 15%).

b. Gastric ulcers are treated with wedge excision or antrectomy with inclusion of the ulcer. Concurrent acid-reducing operation is reserved for acid hypersecreting patients (type II and III) or patients with refractory ulcer disease despite maximal medical management.

II. COMPLICATED PEPTIC ULCER DISEASE.

Complicated peptic ulcer disease refers to hemorrhage, perforation, or obstruction in the setting of PUD. While the global prevalence of PUD has declined, complicated disease and the rate of emergency surgery have remained stable.

A. Hemorrhage is the leading cause of death with estimated 5% to 10% mortality. After resuscitation, EGD is performed. Although bleeding stops spontaneously in 70% of patients, recurrent bleeding may occur in highrisk individuals or if clot, visible vessel, or active bleeding are observed during EGD. **Indications for surgery** include repeated episodes of bleeding, continued hemodynamic instability, ongoing transfusion requirement of more than 4 to 6 units of packed red blood cells over 24 hours, and more than one unsuccessful endoscopic intervention.

1. Bleeding duodenal ulcers usually are located on the posterior duodenal wall within 2 cm of the pylorus and erode into the gastroduodenal artery. Bleeding is controlled by duodenotomy and three-point ligation of the bleeding vessels. Consider concomitant acid-reducing procedure in hemodynamically stable patients who have failed or are noncompliant with medical therapy.

Postoperative *H. pylori* eradication is required.

2. Bleeding gastric ulcers are managed by biopsy followed by oversewing or wedge excision of the ulcer in unstable patients or by acid-reducing procedures in stable patients.

B. Perforated peptic ulcers present with sudden onset of severe abdominal pain but may be less dramatic in hospitalized, elderly, or immunocompromised patients. Examination reveals fever, tachycardia, and abdominal wall rigidity. Peritonitis may be localized if the perforation is walled off by adjacent viscera. Laboratory evaluation demonstrates leukocytosis, and abdominal x-ray may demonstrate free subdiaphragmatic gas. Treatment is aggressive fluid resuscitation and broad-spectrum antibiotics followed by

prompt operative, but nonoperative treatment can be considered in poor operative candidates in whom the perforation has been present for more than 24 hours, the pain is well localized, and there is no evidence of extravasation on upper GI water-soluble contrast study (*Dig Surg.* 2010;27:161). Laparoscopic repair is increasing but it is inconclusive whether it offers a benefit compared to open repair (*BJS.* 2014;101:e51).

1. Perforated duodenal ulcers require simple omental patching, peritoneal debridement, and *H. pylori* eradication. Vagotomy is seldom performed, thus long-term antacid treatment is required. If the patient is stable, an acid-reducing procedure (preferably truncal vagotomy and pyloroplasty) may be added if the patient is *H. pylori*-negative or has failed medical therapy.

2. Perforated gastric ulcers are treated by simple wedge resection to eliminate the perforation and exclude malignancy. If resection cannot be performed due to a juxtapyloric location, multiple biopsies are taken and omental patching is performed.

C. Gastric outlet obstruction (GOO) may result from fibrosis and scarring in chronic PUD or from edema, spasm, and pyloric dysmotility in acute disease. Patients present with recurrent vomiting of poorly digested food, dehydration, and hypochloremic hypokalemic metabolic alkalosis. Management consists of correction of volume and electrolyte abnormalities, nasogastric decompression, and intravenous antisecretory agents. EGD is performed to evaluate the nature of the obstruction to rule out malignancy, and **endoscopic hydrostatic balloon dilation** can be performed at the same time. The overall success rate is 76% but the rate of recurrence of ulcer complications is 36% at 2 years (*Gastrointest Endosc.* 2014;60:229). **Indications for surgical therapy** include persistent obstruction after 7 days of nonoperative management and recurrent obstruction. Antrectomy to include the ulcer and truncal vagotomy is the ideal operation for most patients. Truncal vagotomy with gastrojejunostomy may be considered in patients whose pyloroduodenal inflammation precludes safe management with Billroth I or II reconstructions.

III. GASTRIC ADENOCARCINOMA.

Gastric adenocarcinoma is the fifth most common cancer worldwide but only the fourteenth most common in the United States. The incidence has decreased dramatically, thought to be secondary

to improvements in refrigeration and diet and *H. pylori* treatment. However, gastric cancer is often diagnosed at an advanced stage; only 24% of cancers are localized to the stomach, resulting in an overall 5-year survival rate of 29%.

A. The etiology of gastric cancer is complex and multifactorial. Risk factors include male gender; family history; low socioeconomic status; polyposis syndromes; diets high in nitrates, salts, or pickled foods; adenomatous gastric polyps; previous gastric resection; MŽnŽtrier disease; smoking; *H. pylori* infection; and chronic gastritis. Aspirin, fresh fruits and vegetables, selenium, and vitamin C may be protective.

B. Classification. Ninety-five percent of gastric cancers are adenocarcinomas arising from mucus-producing cells in the gastric mucosa. The

Lauren classification system is most widely used and divides gastric cancers into two subtypes:

1. Intestinal-type cancers (30%) are glandular and arise from the gastric mucosa. They occur more commonly in elderly men and in the distal stomach and are associated with *H. pylori* and other environmental exposures that lead to chronic gastritis, intestinal metaplasia, and dysplasia. Hematogenous metastatic spread to distant organs is seen.

2. Diffuse-type cancers (70%) arise from the lamina propria and are associated with an invasive growth pattern with rapid submucosal spread. They occur more commonly in younger patients, females, and in the proximal stomach. Transmural and lymphatic spread with early metastases are more common, and diffuse-type cancers have worse overall prognosis.

C. Presentation involves nonspecific signs and symptoms such as epigastric abdominal pain, unexplained weight loss, nausea, vomiting, anorexia, early satiety, and fatigue. Dysphagia is associated with proximal gastric cancers, whereas GOO is more typical of distal cancers. Classic physical findings such as enlarged supraclavicular nodes (Virchow node) and infiltration of the umbilicus (Sister Mary Joseph's node) represent metastatic and incurable disease. Perforation and hemorrhage present in the minority and portend advanced disease.

D. Diagnosis is made by double-contrast upper GI barium contrast studies or EGD. EGD permits direct visualization and biopsies of suspicious lesions. **Screening examination** is not cost-effective for the general US population but may be warranted in high-risk individuals such as those more than 20 years post partial gastrectomy, with pernicious anemia or atrophic gastritis, from endemic areas, and with familial or hereditary gastric cancer. Mass screening is performed in Japan, a country with a high incidence of gastric cancer, and resulted in an increase in detection of early-stage gastric cancer and improved 5-year survival rates.

E. Staging primarily involves **computed tomography (CT) and endoscopic**

ultrasonography (EUS) and the American Joint Committee on Cancer and International Union against Cancer (AJCC/UICC) jointly developed a staging system that is most widely used worldwide (Table 17-1).

1. CT scan of the abdomen and pelvis is the best noninvasive modality for detecting metastatic disease in the form of malignant ascites or hematogenous spread to distant organs. Overall accuracy for tumor staging is 43% to 82%. **Positron emission tomography (PET)/CT** combines spatial resolution of CT with the contrast resolution of PET, and can detect nodal and distant metastatic disease not apparent on CT alone.

2. EUS delineates depth of tumor invasion in the gastric wall and adjacent structures and identifying perigastric lymphadenopathy but is suboptimal for distant nodes.

3. Laparoscopic staging can detect occult metastases may distant metastatic disease in 31% of patients (*Am J Surg.* 2006;191:134). Limitations include difficult identification of hepatic metastases and perigastric lymph nodes. Peritoneal washings with cytology should be performed in patients receiving preoperative therapy as well as in those with advanced (T3 or N1) disease.

TABLE 17-1 TNM (Tumor, Node, Metastasis) Staging of Gastric Carcinoma

T: Primary Tumor

T0	No evidence of primary tumor
Tis	Carcinoma *in situ*
T1a	Invasion of lamina propria, muscularis mucosae
T1b	Invades submucosa
T2	Invasion of muscularis propria
T3	Invades subserosa
T4a	Perforates serosa (visceral peritoneum)
T4b	Invasion of adjacent structures

N: Regional Lymph Nodes

N0	No regional node metastasis
N1	Metastasis in 1 to 2 regional lymph nodes
N2	Metastasis in 3 to 6 regional lymph nodes
N3a	Metastasis in 7 to 15 regional lymph nodes
N3b	Metastasis in 16 or more regional lymph nodes

M: Distant Metastasis

M0	No distant metastases
M1	Distant metastases

G: Histologic Grade

GX	Cannot be assessed
G1	Well differentiated
G2	Moderately differentiated
G3	Poorly differentiated
G4	Undifferentiated

Stage Grouping

Stage 0	Tis	N0	M0
Stage IA	T1	N0	M0

Stage IB	T1	N1	M0
	T2	N0	M0
Stage IIA	T1	N2	M0
	T2	N1	M0
	T3	N0	M0
Stage IIB	T1	N3	M0
	T2	N2	M0
	T3	N1	M0
	T4a	N0	M0
Stage IIIA	T2	N3	M0
	T3	N2	M0
	T4a	N1	M0
Stage IIIB	T3	N3	M0
	T4a	N2	M0
	T4b	N0, N1	M0
Stage IIIC	T4a	N3	M0
	T4b	N2, N3	M0

Stage IV	Any T	Any N	Any M1

Used with the permission of the American Joint Committee on Cancer (AJCC), Chicago, Illinois. The original source for this material is the AJCC Cancer Staging Manual, Seventh Edition (2010) published by Springer Science and Business Media LLC, www.springer.com.

F. Treatment. Surgery is the primary treatment in the absence of disseminated disease.

1. Surgical therapy necessitates complete resection with negative microscopic margins (4 cm or greater) for T1b-T3 tumors and en bloc resection for T4 tumors (*J Am Coll Surg.* 2004;199:880).

a. Proximal tumors comprise nearly half of all cancers and require total gastrectomy or proximal subtotal gastrectomy and are associated with nutritional impairment. Total gastrectomy with Roux-en-Y esophagojejunostomy is preferred to avoid reflux esophagitis and impaired gastric emptying. Tumors of the GE junction may require esophagogastrectomy.

b. Midbody tumors comprise 15% to 30% of tumors and generally require total gastrectomy.

c. Distal tumors are approached by subtotal gastrectomy, which has a similar surgical outcome but decreased complications as compared to total gastrectomy (*Ann Surg.* 1999;230:170).

d. Early gastric cancers are confined to the mucosa and have limited propensity for lymph node metastasis and may be treated by limited gastric resections or **endoscopic mucosal resection.**

e. Laparoscopic gastric resections have advantages of reduced pain, shorter hospitalization, and improved quality of life. Long-term outcome is comparable to open gastrectomy and may offer some advantages (*J Clin Oncol.* 2014;32:627*; World J Gastroenterol.* 2013;28:8114).

2. Lymphadenectomy entails removal of 15 nodes generally along the left gastric, hepatic, splenic, and celiac vessels.

3. Adjuvant therapy is important because the majority of patients with locoregional disease (all patients except those with T1Ñ2N0M0 disease) are at high risk for local or systemic recurrence following curative surgery.

a. Adjuvant combined modality therapy improves overall and disease-free survival rates in patients with resected gastric cancer treated postoperatively with 5-fluorouracil (5-FU)/leucovorin chemotherapy coupled with radiation therapy (*N Engl J Med.* 2001;345:725; *J Clin Oncol.* 2010;28:2430).

b. Neoadjuvant chemotherapy has the potential for improving patient tolerance, resectability rates, and overall survival. A recent meta-analysis demonstrated improvement in 3-year disease free survival, tumor down-staging rate, and R0 resection rate in locally advanced gastric cancer (*Surg Oncol.* 2014;14:97).

4. Palliative therapy is important due to overall low cure rates. Patients with peritoneal disease, hepatic or nodal metastases, or other poor prognostic factors benefit most from endoscopic palliation. Palliative surgical resection may be considered in patients with better prognosis and performance status. Palliative chemoradiation therapy also prolongs survival and improves symptoms and quality of life.

IV. PRIMARY GASTRIC LYMPHOMA.

Primary gastric lymphoma (PGL) accounts for fewer than 5% of gastric neoplasms but two-thirds of all primary GI lymphomas. PGLs are usually B-cell, non-Hodgkin lymphomas and most occur in the distal stomach.

A. Presentation is typically in the sixth decade with epigastric pain, weight loss, anorexia, nausea, and vomiting. Evaluation includes EGD, EUS, CT of chest/abdomen/pelvis, bone marrow biopsy, and biopsy of enlarged peripheral lymph nodes.

B. Low-grade PGLs have features resembling mucosa-associated lymphoid tissue (MALT). **The majority of low-grade MALT lymphomas are associated with** *H. pylori* **infection and first-line therapy is** *H. pylori* **eradication.** Surgical resection is reserved for failure of medical therapy or complications of therapy (bleeding or perforation).

V. BENIGN GASTRIC TUMORS.

Benign gastric tumors represent fewer than 2% of all gastric tumors and are usually located in the antrum or corpus.

A. Gastric polyps are classified by histologic findings. Endoscopic removal is appropriate if the polyp can be completely excised.

1. Hyperplastic polyps are regenerative and constitute 75% of gastric polyps. Minimal risk of malignant transformation.

2. Adenomatous polyps are neoplastic, and the incidence of carcinoma is proportional 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 be performed laparoscopically and if endoscopic excision is not possible.

VI. GASTROINTESTINAL STROMAL TUMORS (GISTs).

Gastrointestinal stromal tumors (GISTs) comprise 3% of gastric malignancies and arise from mesenchymal components of the gastric wall. The median age at diagnosis is 60 years, with a slight male predominance. GISTs frequently display prominent extraluminal growth and can attain large sizes before becoming symptomatic.

A. Presentation includes asymptomatic masses found incidentally, vague abdominal pain secondary to mass effect, and hemorrhage. Diagnosis is made by endoscopy (with or without EUS) and FNA biopsy. GISTs are graded according to tumor size and histologic frequency of mitoses. Staging is accomplished by CT of abdomen/pelvis and chest x-ray.

B. Treatment is open or laparoscopic surgical resection with 2 cm margins of grossly normal gastric wall. En bloc resection of structures involved by local invasion should be attempted, but lymphadenectomy is not indicated because lymph node metastases are rare. Local recurrence after resection as well as metastases via hematogenous spread is common. GISTs are not radiosensitive or responsive to traditional chemotherapy. However, most GISTs express the **c-kit** receptor, a tyrosine kinase that acts as a growth factor receptor. **Imatinib mesylate (Gleevec)** is a small-molecule inhibitor of the c-kit receptor that is first-line therapy for metastatic or recurrent GIST. Approximately 60% of patients experience a partial response, and surgical therapy should be considered for patients in whom all gross disease can be removed.

VII. GASTRIC CARCINOIDS.

Gastric carcinoids are rare neuroendocrine tumors comprising less than 1% of all gastric neoplasms. Carcinoid tumors arise from enterochromaffin-like cells and can be secondary to hypergastrinemia (types 1 and 2) or occur sporadically independent of gastrin (Type 3). Tumors are small, multiple, and asymptomatic, although larger solitary tumors may cause ulceration and symptoms similar to PUD. EGD with biopsy generally is diagnostic. Treatment of large (>2 cm), solitary tumors is gastrectomy because of high invasive potential. Treatment of smaller, multifocal tumors is less clear, with options ranging from observation, gastrectomy to include the tumors, and antrectomy without inclusion of tumors to reduce gastrin levels and induce tumor regression.

VIII. POSTGASTRECTOMY SYNDROMES.

Postgrastrectomy syndromes are caused by changes in gastric emptying and 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. Most are treated nonoperatively and resolve with time.

A. Nutritional disturbances occur in 30% of patients, either as a result of functional changes or postgastrectomy syndromes. Prolonged **iron, folate, vitamin B_{12}, calcium, and vitamin D deficiencies** can result in anemia, neuropathy, dementia, and osteomalacia but can be

prevented with supplementation.

B. Dumping syndrome results from rapid emptying of a high-osmolar carbohydrate load into the small intestine and is most common after Billroth II reconstruction due to loss of reservoir capacity and pylorus function.

1. Early dumping occurs within 30 minutes of eating and is characterized by nausea, epigastric distress, explosive diarrhea, and vasomotor symptoms. It is caused by a rapid shift of extracellular fluid into the bowel lumen in response to a hyperosmolar load entering the small intestine from the stomach.

2. Late dumping is primarily vasomotor and occurs 1 to 4 hours after eating. The hormonal response to high simple carbohydrate loads results in hyperinsulinemia and reactive hypoglycemia. Symptoms are relieved by carbohydrate ingestion.

3. Treatment is primarily nonsurgical and results in improvement in nearly all patients over time. Meals are decreased in volume but increased in frequency, liquids should be ingested 30 minutes after eating solids, and simple carbohydrates should be avoided. If reoperation is necessary, conversion to Roux-en-Y gastrojejunostomy or an isoperistaltic/antiperistaltic jejunal loop is usually successful.

C. Alkaline reflux gastritis is most commonly associated with Billroth II gastrojejunostomy and is characterized by epigastric pain, nausea, and bilious emesis. Pain is not relieved by vomiting or associated with meals. Endoscopy reveals inflamed, beefy red, friable gastric mucosa and may demonstrate bile reflux into the stomach, which can be confirmed by hydroxy iminodiacetic acid (HIDA) scan. **Nonoperative therapy** consists of frequent meals, antacids, and cholestyramine but is usually ineffective. **Surgery** to divert bile flow from the gastric mucosa is the only proven treatment. The creation of a long-limb (45-cm) Roux-en-Y gastrojejunostomy is the preferred option for most patients (*Gastroenterol Clin North Am.* 1994;23:281).

D. Roux stasis syndrome may occur in up to 30% of patients after Roux-en-Y gastroenterostomy and results from functional obstruction due to disruption of the normal propagation of pacesetter potentials in the Roux limb from the proximal duodenum as well as altered motility in the gastric remnant. It is characterized by chronic abdominal pain, nausea, and vomiting that is aggravated with eating. Near-total gastrectomy to remove the atonic stomach can improve gastric emptying and is occasionally useful in patients with refractory Roux stasis.

E. Loop syndromes result from mechanical obstruction of either the **afferent** or **efferent** limbs of the Billroth II gastrojejunostomy. Evaluation includes plain abdominal x-rays, CT scan, upper GI contrast studies, and endoscopy. Relief of the obstruction may require adhesiolysis, revision of the anastomosis, bowel resection, or conversion of Billroth II to Roux-en-Y gastrojejunostomy.

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 suggests this complication. Examination may reveal a fluid-filled abdominal mass, and laboratory findings may include elevated bilirubin or amylase. **Duodenal stump blowout** results from progressive afferent limb dilation, leading to peritonitis, abscess, or fistula formation. In the urgent setting, jejunojejunostomy can effectively decompress the afferent limb. A **chronic form** of afferent loop syndrome results from partial mechanical obstruction of the afferent limb. Patients present with 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 and vitamin B_{12}, folate, and iron deficiency) by interfering with fat and vitamin B_{12} absorption.

2. Efferent loop syndrome results from intermittent obstruction of the efferent limb of the gastrojejunostomy. Patients complain of abdominal pain and bilious emesis months to years after surgery, similar to the situation with regard to a proximal small bowel obstruction.

F. Postvagotomy diarrhea occurs in 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 is rarely indicated.

CHAPTER 17: STOMACH

Multiple Choice Questions

1. A patient with gastric outlet obstruction and prolonged emesis has which electrolyte disturbance?

a. Hyperchloremic, hyperkalemic metabolic acidosis

b. Hyperchloremic, hypokalemic metabolic acidosis

c. Hypochloremic, hyperkalemic metabolic alkalosis

d. Hypochloremic, hypokalemic metabolic alkalosis

e. Hyponatremic, hypokalemic metabolic acidosis

View Answer

2. What is the typical first-line therapy for low-grade MALT lymphoma of the stomach?

a. Chemotherapy

b. Radiation

c. Total gastrectomy

d. Wedge resection of lesion without reconstruction

e. *H. pylori* eradication

View Answer

3. What is the preferred surgical therapy for hemodynamically unstable patients with bleeding duodenal ulcers?

a. Graham patch

b. Duodenotomy and three-point ligation of the bleeding vessel

c. Duodenotomy, three-point ligation of the bleeding vessels, highly selective vagotomy

d. Duodenotomy, three-point ligation of the bleeding vessels, truncal vagotomy, pyloroplasty

e. Duodenal resection with reconstruction

View Answer

4. What is true regarding gastrointestinal stromal tumors?

a. Local recurrence is uncommon after resection

b. Lymphadenectomy should be attempted given high propensity of lymph node metastasis

c. Gleevec is first-line therapy for metastatic or recurrent disease

d. En bloc resection of involved structures should not be attempted

e. Tumors are highly radiosensitive

View Answer

5. What is true among the treatment principles for gastric cancer?

a. Distal tumors comprise the majority of gastric cancers

b. There is a low risk of recurrence of disease

c. Lymphadenectomy is not required in early-stage disease

d. A minimum of 15 lymph nodes should be resected during lymphadenectomy

e. Early gastric cancers always require total gastrectomy

View Answer

18
The Surgical Management of Obesity

Iheoma Nwaogu

J. Christopher Eagon

Obesity is a disease process that has reached epidemic proportions worldwide, with the highest prevalence in the United States, where 5% of the adult population is morbidly obese. Obesity is also becoming increasingly prevalent in the pediatric population. Severe obesity is a condition characterized by the pathologic accumulation of excess body fat. In adults, it is **defined** as a body mass index [BMI = weight (kg)/height (m^2)] equal to or greater than 40, which generally correlates with an actual body weight 100 lb greater than ideal body weight. In children, severe obesity is defined as a BMI that is equal to or greater than 120% of the 95^{th} percentile or equal to or greater than 35 kg/m^2 (whichever is lower).

The **etiology** of morbid obesity is poorly understood and thought to result from an imbalance in biologic, psychosocial, and environmental factors governing caloric intake and caloric expenditure. Risk factors for the development of morbid obesity include **genetic predisposition,** diet, and culture.

Most patients with morbid obesity present with one or more of a number of weight-related comorbidities. Patients with **central** obesity (android or ÒappleÓ fat distribution) are at higher risk for development of obesity-related complications than those with **peripheral** obesity (gynecoid or ÒpearÓ fat distribution). This is due to increased visceral fat distribution, producing increased intra-abdominal pressure and increasing fat metabolism (with subsequent hyperglycemia, hyperinsulinemia, and peripheral insulin resistance). Table 18-1 lists some of the medical complications associated with morbid obesity. In addition to the aforementioned comorbidities, obesity also increases mortality. One study showed an increase in mortality among morbidly obese individuals (*NEJM.* 2006;355:8).

Treatment of morbid obesity is of paramount importance because of the many medical sequelae associated with obesity, nearly all of which are reversible on resolution the obese state.

A. Lifestyle changes in diet, exercise habits, and behavior modification are first-line therapy for all obese patients. In combination, such changes can achieve 8% to 10% weight loss over a 6-month period, but losses are sustained at 1 year in only 60% of patients. However, certain comorbidities, such as diabetes, benefit from as little as 3% weight loss, and lifestyle changes alone may be sufficient in patients with BMI less than 27.

B. Pharmacotherapy is second-tier therapy used in patients with BMI greater than 27 and in combination with lifestyle changes. Currently, sibutramine, an appetite suppressant, and orlistat, a lipase inhibitor that reduces lipid absorption, are the only approved drugs for weight loss treatment. Weight

loss with these agents is 6% to 10% at 1 year, but relapse rates after discontinuation of the drugs are high.

TABLE 18-1 Complications of Morbid Obesity

Cardiac

- Hypertension
- Coronary artery disease
- Heart failure
- Arrhythmias

Pulmonary

- Obesity hypoventilation syndrome
- Obstructive sleep apnea
- Respiratory insufficiency of obesity (pickwickian syndrome)
- Pulmonary embolism

Metabolic

- Type II diabetes
- Hyperlipidemia
- Hypercholesterolemia
- Nonalcoholic steatohepatitis

Musculoskeletal

- Degenerative joint disease
- Lumbar disc disease
- Osteoarthritis

Gastrointestinal

- Cholelithiasis
- Gastroesophageal reflux disease
- Hernias

Vascular

- Deep venous thrombosis
- Venous stasis ulceration

Infectious

- Fungal infections
- Necrotizing soft tissue infections

Genitourinary

- Nephrotic syndrome
- Stress urinary incontinence

Gynecologic

- Polycystic ovary syndrome

Neurologic/psychiatric

- Pseudotumor cerebri
- Depression
- Stroke
- Low self-esteem

Oncologic

- Cancers of uterus, breast, colon/rectum, and prostate

C. Bariatric surgery is the most effective approach for achieving durable weight loss in the morbidly obese. Multiple studies have confirmed the superiority of surgery to nonsurgical approaches in achieving and maintaining weight reduction in the morbidly obese (*N Engl J Med.* 2004;351:2683; *Surg Obes Relat Disord.* 2010;6:347). A National Institutes of Health Consensus Development Conference on morbid obesity established guidelines for the

evaluation and treatment of morbidly obese patients with bariatric surgical procedures (*Ann Surg.* 2010;250:399).

1. Indications. Patients who have failed intensive efforts at weight control using medical means are candidates for bariatric surgery if they have a BMI index greater than 40 or greater than 35 with weight-related comorbidities. In addition, patients who have a BMI index greater than 30 with poorly controlled diabetes or metabolic syndrome may be offered bariatric surgery although long-term data demonstrating benefit is still lacking. Proposed **contraindications** include untreated or uncontrolled severe psychiatric illness, binge-eating disorders, active alcohol or drug abuse, prohibitive operative risks secondary to severe cardiac disease such as severe congestive heart failure or unstable angina, as well as the inability to comprehend the nature of the surgical intervention or comply with required postoperative nutritional and lifestyle changes. Further, patients actively pregnant or intending to get pregnant in 12 to 18 months postoperatively should not undergo bariatric surgery.

2. Preoperative evaluation. A bariatric multidisciplinary team including primary care physicians, dietitians, physical therapists, anesthesiologists, nurses, and psychiatrists or psychologists evaluates a patient's weight history, dietary habits, motivation, social history, and comorbid medical conditions prior to surgery.

3. Benefits of surgery are related to reversal of the disease processes associated with severe obesity. Hypertension completely resolves in 62% of patients and resolves or improves in 79%. Diabetes is completely resolved in *77%* of patients and resolves or improves in 86%. Obstructive sleep apnea resolves or improves in 85% of patients and hyperlipidemia improves in 70%. The quality of life is markedly better. Most importantly, recent studies demonstrate reduced mortality rates in morbidly obese patients undergoing bariatric surgery compared to matched controls (*NEJM.* 2004;351:2683; *Ann Surg.* 2010;250:399).

Bariatric surgical procedures can generally be divided into two types: **Restrictive procedures,** which limit the amount of food that can be ingested, and **malabsorptive procedures,** which limit the absorption of nutrients and calories from ingested food by bypassing predetermined lengths of small intestine. The four standard operations used to produce weight loss in the morbidly obese include adjustable gastric banding (AGB) and vertical banded gastroplasty (restrictive procedures), biliopancreatic diversion (BPD) with and without duodenal switch (DS) (malabsorptive procedures), and Rouxen-Y gastric bypass (RYGBP) (combination).

Sleeve gastrectomy, the first component of a DS operation, increasingly is being performed alone as a restrictive procedure.

A. Adjustable gastric banding (**AGB**) involves open or laparoscopic placement of a silicone band with an inflatable balloon around the proximal stomach at the angle of His. The band is connected to a reservoir that is implanted over the rectus sheath. The patient undergoes serial adjustments to inflate the band and create a small proximal gastric pouch. Excess weight

loss is approximately 50%. Perioperative mortality is exceedingly low (0.05%), and overall complication rate is near 11%. Most complications are related to band slippage, which presents with obstructive symptoms or problems with the port (kinking or leaking of access tubing). Band erosion can occur but is far less frequent than the aforementioned complications. Advantages include safety, adjustability, and reversibility, whereas disadvantages include need for frequent postoperative visits.

B. Roux-en-Y gastric bypass (RYGBP) is the most popular bariatric surgical procedure performed in the United States. A 30-mL proximal gastric pouch is created by either transection or occlusion using a stapling device. A 1-cm-diameter anastomosis is then performed between the pouch and a Roux limb of small bowel. This results in a small reservoir, a small passage for pouch emptying, and bypass of the distal stomach, duodenum, and proximal jejunum. The length of the Roux limb directly correlates with the degree of postoperative weight loss, with a 75-cm limb used for standard gastric bypasses and a 150-cm limb used for the superobese. Gastric bypass results in weight loss superior to that achieved with restrictive procedures, with mean excess weight loss of 70%. Perioperative mortality is 1%, and despite aggressive prophylaxis, **pulmonary embolism** (PE) remains the most common cause of death after bariatric surgery. Anastomotic leak at the gastrojejunostomy is another serious early complication, occurring in approximately 2% of cases. Unexplained **tachycardia** is often the only presenting sign of either complication in the perioperative period and warrants prompt investigation. Other early complications include wound infection (4% to 10%), gastric remnant dilation, and Roux limb obstruction. Late complications include incisional hernia (15% to 25%), stomal stenosis (2% to 14%), marginal ulcer (2% to 10%), bowel obstruction (2%), and internal hernia (1%). Early or late **bowel obstruction** after RYGBP can be a life-threatening complication and generally requires prompt reoperation because of its association with internal hernia and potential for bowel strangulation. **CT scan with oral contrast** is the best diagnostic test to evaluate for leak or obstruction after RYGBP. Nutritional complications include folate, vitamin B_{12}, iron, and calcium deficiency. Dumping syndrome occurs in many patients and may reinforce dietary behavior modification to avoid sweets and high-calorie foods. Laparoscopic RYGBP is a technically challenging but safe procedure when performed by surgeons with advanced laparoscopic skills. Laparoscopic RYGBP produces equal excess weight loss and has similar mortality and leak rates as the open procedure. Its main advantages are reduced postoperative pain, reduced length of stay, and significantly reduced wound-related complications, such as wound infections,

dehiscence, and incisional hernias (*NEJM.* 2009;361:445).

C. Biliopancreatic diversion (BPD) and **biliopancreatic diversion with duodenal switch (BPD-DS)** are two additional procedures for morbidly obese patients. BPD requires antrectomy with formation of a 200-cm alimentary channel and a 50- to 75-cm common channel. BPD-DS includes a sleeve gastrectomy, preservation of the pylorus, a 150-cm alimentary

channel, and a 75- to 100-cm common channel. These procedures are done at select centers for the superobese and those who have failed to maintain weight loss following gastric bypass or restrictive procedures. Long-term outcomes indicate excess weight loss of 75% at 1 year, but nutritional deficiencies are more common than for RYGBP. Postoperative complications include anemia (30%), protein-calorie malnutrition (20%), dumping syndrome, and marginal ulceration (10%). These procedures are technically demanding and the applicability of these procedures to the obese population remains to be determined.

D. Sleeve gastrectomy, the first component of a DS operation, can be used alone as a purely restrictive procedure for the treatment of morbid obesity. It does not produce malabsorption and is technically easier to perform than BPD-DS or RYGBP. Preliminary reports have demonstrated 70% to 80% excess body weight loss at 1 year, but long-term outcomes and durability of this procedure remain unknown. It may be indicated as an initial procedure in the superobese population to induce enough weight loss to make BPD-DS or RYGBP technically more feasible (*Surg Obes Relat Disord.* 2010;6:1; *Ann Surg.* 2010;252:319).

Typical **postoperative management** includes postoperative analgesia, frequent measurements of intake and output with monitoring of the drain output to evaluate for potential anastomotic leak, as well as gradual advancement of diet from nil per os (NPO) to a high-protein liquid diet. Aggressive pulmonary management with early institution of continuous positive airway pressure (when indicated) is necessary to prevent hypoxemia. Early ambulation is highly encouraged and mechanical and pharmacologic venous thromboembolism prophylaxis is recommended for all patients. Upper gastrointestinal series with Gastrografin are routinely performed by most bariatric surgeons before further diet progression in order to detect any subclinical leaks.

In the long-term care of bariatric surgery patients, the follow-up plan depends on the type of bariatric procedure performed and the severity of comorbidities. Any severe or persistent gastrointestinal complaints warrant further examination, typically employing radiographic imaging studies to ensure prompt diagnosis of potential complications. Nonsteroidal anti-inflammatory drugs should be avoided following bariatric surgery due to its association with marginal ulcers or perforations. Close followup for adequate weight loss, improvement or resolution of comorbidities, in addition to close metabolic and nutritional monitoring is crucial and all patients should be encouraged to engage in physical activity for at least 30 minutes daily, take smaller more frequent meals chewed thoroughly, and avoid high-fat or high-sugar liquids which could precipitate dumping syndrome and impede weight loss. Of note, inadequate weight loss following bariatric surgery should warrant further evaluation to determine the etiology (including surgical

failure potentially requiring revision or poor compliance with nutritional or lifestyle requirements). Lifelong nutritional supplementation with multivitamins, iron, calcium, vitamin D, and vitamin B_{12} is indicated (*Endocr Pract.* 2013;19(2):337-372).

CHAPTER 18: THE SURGICAL MANAGEMENT OF OBESITY

Multiple Choice Questions

1. A 50-year-old woman with a history of poorly controlled diabetes presents for evaluation for bariatric surgery. Her BMI is 33 kg/m^2 and has fluctuated from 31 to 34.3 with physician-supervised diet and exercise over the past year. She has unsuccessfully tried multiple weight loss programs in the previous 7 years and now seeks surgical management. What treatment plan is appropriate for this patient?

a. Continue physician-supervised diet and exercise program as she has seen some benefit and followup in 1 year.

b. Recommend bariatric surgery after appropriate multidisciplinary preoperative evaluation as patient meets the indication for surgery.

c. Bariatric surgery is not recommended at this time as patient's BMI index is not considered “severely obese” and no followup needed.

d. Recommend multidisciplinary evaluation now with bariatric surgery offered when patient's BMI goes above 35 kg/m^2.

e. None of the above.

View Answer

2. A 29-year-old woman reports severe abdominal pain along with persistent nausea and vomiting 4 days after Roux-en-Y gastric bypass. On evaluation, she is tachycardic with a blood pressure of 100/65. Examination reveals severe upper abdominal tenderness to palpation and CT scan reveals distended small bowel loops. What is the most appropriate next step in management?

a. Intravenous fluid resuscitation and prompt surgical exploration

b. Intravenous fluid resuscitation and serial abdominal examinations

c. Change analgesic and antiemetic medications in an effort to improve symptoms

d. Obtain upper gastrointestinal study with Gastrografin in an effort to further localize the area of obstruction

e. None of the above

View Answer

3. A 37-year-old woman presents 10 weeks after her laparoscopic adjustable gastric banding with severe heartburn, nausea, and persistent vomiting for the past week. She reports compliance with the postoperative diet and exercise regimen recommended and notes that her band was tightened at her last office visit 2 weeks prior to her presentation. On examination, she is tachycardic and has mild epigastric tenderness to palpation. What is the most appropriate next step?

a. Obtain a CT abdomen with oral and IV contrast

b. Make patient NPO and place nasogastric tube

c. Start Esomeprazole today and reassess in 2 weeks

d. Advise patient to eat smaller portions at each meal

e. Immediate removal of all the fluid from the adjustable band

View Answer

4. A 52-year-old woman presents, 3 months after her sleeve gastrectomy, with a 5-cm painless and easily reducible periumbilical bulge that is exacerbated by Valsalva maneuvers. She notes that it does not bother her although it has been increasing in size and is cosmetically unappealing. She remains compliant with her postsurgical diet and exercise and reports adequate weight loss. What is the best management plan at this time?

a. Recommend surgical repair now given the risk of incarceration or strangulation of hernia

b. Decrease frequency of exercise to avoid worsening the problem and defer surgical management at this time until weight loss has stabilized and nutritional status is optimized

c. Recommend surgical repair immediately after admission to optimize patient's nutritional status

d. Defer surgical management at this time until weight loss has stabilized and nutritional status is optimized

e. None of the above

View Answer

5. A 45-year-old woman presents with a 3-week history of epigastric pain and occasional nausea 1 year after undergoing her Roux-en-Y

gastric bypass. During workup, upper endoscopy reveals a 1.5-cm ulceration near the gastrojejunostomy. Which of the following is(are) associated with this condition?

a. Nonsteroidal anti-inflammatory drugs

b. *Helicobacter pylori* infection

c. Smoking

d. Poor tissue perfusion due to ischemia at the anastomosis

e. All of the above

View Answer

19
Small Intestine

Jennifer A. Leinicke

Paul E. Wise

SMALL-BOWEL OBSTRUCTION (SBO)

Mechanical obstruction of the small intestine (SI) can be partial, allowing some distal passage of gas or fluid, or complete, with total occlusion of the lumen. In a strangulated obstruction, the involved bowel has vascular compromise leading to infarction and eventual perforation of the intestinal wall. No clinical or laboratory values are pathognomonic for strangulated obstructions, although characteristic findings include constant, as opposed to only crampy, abdominal pain, fever, leukocytosis, and acidosis. **Ileus** implies failure of peristalsis without mechanical obstruction. Recent abdominal operations, electrolyte disturbances, trauma, peritonitis, systemic infections, bowel ischemia, and medications can cause ileus.

A. Etiology

1. Adhesions due to previous abdominal operations (or rarely isolated congenital adhesions/bands) are the *most common* cause of SBO in US adults. Intra-abdominal adhesions account for about 60% to 70% of SBOs.

2. Incarcerated hernias are the second most common cause of SBOs in industrialized nations and the most common cause of SBO worldwide. In children and patients without prior abdominal surgery, hernias are the most common cause of SBO in developed nations.

3. Intussusception occurs when one portion of bowel (the intussusceptum) telescopes into another (the intussuscipiens). Tumors, polyps, enlarged mesenteric lymph nodes, or a Meckel diverticulum may serve as lead points of the telescoped segment. As opposed to intussusception in children, adults with intussusception require workup for bowel pathology.

4. Volvulus, or the rotation of a segment of bowel around its vascular pedicle, is often caused by adhesions or congenital anomalies such as intestinal malrotation.

5. Strictures secondary to ischemia, inflammation (Crohn disease, CD), radiation, or prior surgery may cause SBO.

6. Gallstone ileus occurs as a complication of cholecystitis. Fistulization between the biliary tree and the small bowel (cholecystoduodenal or choledochoduodenal fistula) allows one or more gallstones to travel distally and become lodged, typically at the ileocecal valve.

7. External compression from tumors, abscesses, hematomas, or other masses can cause SBO.

8. Foreign bodies typically pass without incident. Items presenting with obstruction may require operation if they cannot be retrieved endoscopically.

B. Diagnosis

1. Signs and symptoms. Proximal SBOs present with early bilious **emesis.** Distal obstructions present later with thicker, more feculent emesis. Early in the disease course, **nausea** may be observed in the absence of vomiting. **Abdominal distention** typically increases the more distal the obstruction. **Abdominal pain** is poorly localized and often colicky in nature. **Obstipation** is observed once the distal bowel (beyond a complete obstruction) is evacuated. With a persistent obstruction, **hypovolemia** progresses due to impaired intestinal absorption, increased secretion, and fluid losses from emesis.

2. Physical examination. Abnormal **vital signs** are generally indicative of hypovolemia (e.g., tachycardia and hypotension). **Abdominal examination** may reveal distension, prior surgical scars, masses, or hernias. Peritonitis mandates prompt surgical treatment due to the risk of bowel strangulation.

3. Laboratory evaluation. In the early stages of SBO, laboratory values may be normal. As the process progresses, laboratory values commonly reflect dehydration demonstrating hypochloremic, hypokalemic contraction alkalosis. Elevated white blood cell (WBC) count and serum lactate level are concerning for possible strangulation.

4. Radiologic evaluation. Abdominal plain films may demonstrate dilated loops of SI, airÑ fluid levels, and paucity of colorectal gas. These findings may be absent in early, proximal, and/or closed-loop obstructions. Pneumatosis intestinalis or portal venous gas suggests strangulated obstruction and necrosis. Free intra-abdominal air indicates hollow viscus perforation. Air in the biliary tree and a radiopaque gallstone in the right lower quadrant are pathognomonic of gallstone ileus. Paralytic ileus appears as gaseous distention uniformly distributed throughout the stomach, SI, and colon. **Computed tomography (CT)** can localize and characterize the obstruction and provides information regarding etiology of SBO and presence of other intra-abdominal pathology.

5. Differential diagnosis. Mesenteric vascular ischemia 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. **Colonic obstruction** can easily be confused with a distal SBO. A CT or water-soluble contrast enema can aid in diagnosis. Radiography of primary **hypomotility** disorders reveals gas throughout the entire GI tract with particular distention of the small bowel.

C. Treatment of SBO is evolving and includes prevention at initial laparotomy.

1. Prevention. The highest risk of adhesive SBO occurs after ileal pouchanal anastomosis, open colectomy, and open gynecologic surgeries. Excluding acute appendicitis, laparoscopic as opposed to open techniques result in fewer adhesions. Available bioabsorbable antiadhesion barriers, such as hyaluronic acid/carboxymethyl-cellulose (Seprafilm) and icodextrin 4% solution (Adept), have been shown to reduce adhesions, but whether they reduce the incidence and severity of later SBOs

is unclear. A multicenter trial comparing Seprafilm to no treatment found no difference in overall rate of SBO but a reduction in risk of SBO *requiring operation* was observed (1.8% vs. 3.4%; $P < 0.05$) at a mean followup of 3.5 years (*Dis Colon Rectum.* 2006;49(1):1-11).

2. Strangulated obstruction or peritonitis requires prompt operative intervention. Mortality associated with gangrenous bowel approaches 30% if operation is delayed beyond 36 hours but is improved when surgical intervention is prompt. Fluid/electrolyte resuscitation and nasogastric (NG) tube decompression are crucial in the preoperative preparation of the patient.

3. Nonstrangulated obstructions can be treated nonoperatively if the patient is clinically stable. Fluid resuscitation and NG decompression are the primary therapy for any SBO. A trial of nonoperative management requires close observation with serial abdominal examinations every 4 to 6 hours, preferably by the same person. If the patient develops signs of shock or peritonitis, or fails to improve within a few days, laparotomy is indicated. Early evaluation of patients with a water-soluble oral contrast agent may be useful in differentiating patients who will spontaneously resolve their obstruction from patients who require operative intervention. A sample treatment algorithm is given in Figure 19-1.

In patients with SBO secondary to incarcerated hernia, attempts to reduce the hernia with mild sedation and manual pressure is warranted if the symptoms were present less than 24 to 48 hours. If successful, the patient requires close monitoring for evidence of bowel infarction or perforation. Severe initial tenderness, erythema or ecchymosis at the hernia site, or symptoms >48 hours, increases suspicion for strangulation. That, or inability to reduce the hernia, requires urgent operation. A trial of nonoperative therapy is also indicated for SBO in the early postoperative state and for patients with multiple prior SBOs, frozen/hostile abdomen, abdominal irradiation, CD, or carcinomatosis.

4. Operative intervention is generally performed via midline incision, though a standard groin incision can be used for incarcerated inguinal or femoral hernias. The goal of operation is to identify and treat the origin of obstruction. Extensive adhesiolysis and bowel resection may be necessary. If adjacent bowel viability is questionable, a second-look operation within 24 to 48 hours may be required. Enteroenteric or enterocolic anastomosis can bypass an unresectable obstructing lesion. Placement of a gastrostomy tube for postoperative decompression should be considered in select cases, such as carcinomatosis or unresectable obstructing cancer.

D. Prognosis is largely related to the presence of intestinal ischemia. The postoperative

mortality from a nonstrangulating obstruction is very low. Obstructions associated with strangulated bowel carry a mortality of less than 10% if operation is performed shortly after presentation. Patients with SBO admitted to surgical services have shorter hospital stays, earlier operative intervention, and reduced direct health-care costs when compared to patients with SBO admitted to a medical service.

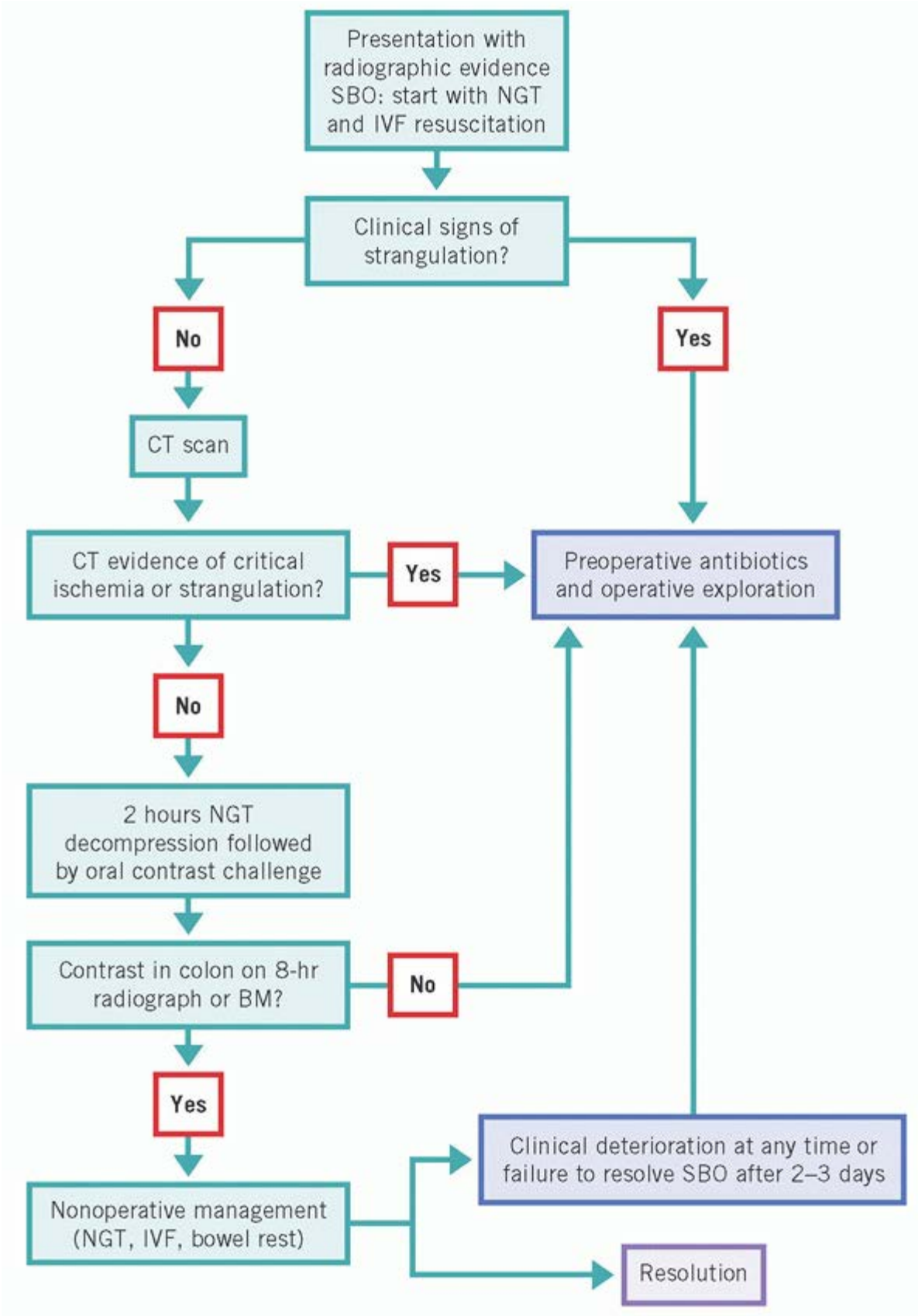


Figure 19-1 Algorithm for management of small bowel obstruction. SBO, small bowel obstruction; NGT, nasogastric tube; IVF, intravenous fluid; oral contrast challenge, water soluble oral contrast administration; BM, bowel movement.

MECKEL DIVERTICULUM

Meckel diverticulum is the most common congenital anomaly of the GI tract and occurs from failure of the vitelline or omphalomesenteric duct to obliterate by the sixth week of fetal development. A Meckel's is a true diverticulum containing all layers of the bowel wall and located on the antimesenteric border of the ileum, usually 2 feet from the ileocecal valve. This is part of the Ò*rule of twos*Ó for Meckel's, including a 2% incidence, a 2:1 male:female ratio, patients usually present before 2 years of age, and Meckel often contain two types of mucosa: Intestinal and heterotopic, gastric or pancreatic.

A. Presentation. The vast majority of Meckel diverticula are *asymptomatic.* Painless, episodic **bleeding** is the most common presenting sign. The source is typically a peptic ulcer of adjacent normal ileum caused by acid secretion from gastric mucosa within the diverticulum. Intussusception or incarcerated hernia (LittrŽ hernia) causing **intestinal obstruction** is the second most common presentation. Obstruction can also occur due to volvulus of small bowel around a fibrous band connecting the diverticulum to the anterior abdominal wall. **Meckel diverticulitis** occurs in 20% of symptomatic patients and is often mistaken for acute appendicitis.

B. Diagnosis. In adults, clinical diagnosis of a Meckel diverticulum is extremely difficult except in the presence of bleeding. A **Meckel scan** is a radionucleotide study based on the uptake of Tc-99m pertechnetate by ectopic gastric mucosa. In children, this test is the most accurate (90%) for diagnosing a Meckel's but is less accurate (46%) in adults because of reduced prevalence of ectopic gastric mucosa within the diverticulum. In the presence of bleeding, a **tagged red blood cell scan** can also be useful. **Contrast studies,** such as SBFT and enteroclysis are diagnostic in up to 75% patients. **CT** and **sonography** are typically of little value because distinguishing between a diverticulum and intestinal loops can be very difficult unless Meckel diverticulitis is present.

C. Treatment. Resection is indicated in symptomatic patients. For patients who present with obstruction, simple diverticulectomy can be performed. Segmental small-bowel resections should be performed for acute diverticulitis, a wide-based diverticulum, volvulus with necrotic bowel, or bleeding from a mesenteric ulcer. **Incidental diverticulectomy** during surgery for other abdominal pathology is *not indicated.* Lifelong morbidity associated with the presence of a Meckel diverticulum is extremely low.

ENTERIC FISTULAS

A fistula is defined as an abnormal communication between two epithelialized surfaces. Fistulas are categorized according to anatomy, output, and etiology.

A. Anatomic Considerations. *External* fistulas are most common and connect an internal organ system with the skin or atmosphere, for example, an enterocutaneous fistula (ECF) or an enteroatmospheric (EAF) fistula. *Internal* fistulas connect two hollow structures of the same or different organ system. Examples include colovesicular and enteroenteric fistulas.

Proximal fistulas located in the stomach, duodenum, or jejunum and are usually associated with high outputs of 3 or more liters per day. Profound dehydration, malnutrition, and electrolyte disturbances are common with these fistulas. *Distal* fistulas of the ileum or colon tend to be lower in output and associated with fewer complications than proximal fistulas, and they more often close with nonoperative treatment. Physiologic categorization of fistulas is centered on the *output* and is divided into *high* (>500 mL/day), *moderate* (200 to 500 mL/day), and *low* (<200 mL/day) output fistulas.

B. Pathophysiology. The overall mortality for all enteric fistulas is 5% to 20%. Loss of GI contents leads to **hypovolemia,** as well as **acid/base** and **electrolyte abnormalities.** High-output fistulas release large volumes of fluid that cannot be adequately replaced by enteral means, leading to dehydration and intravascular volume depletion. Malnutrition is often due to both insufficient caloric intake and functional exclusion of portions of the GI tract limiting absorptive capacity.

C. Etiology

1. Abdominal operations are the leading cause of fistula formation. The risk is greatest for operations performed for inflammatory bowel disease (IBD), ischemia, malignancy, or extensive intestinal adhesions. Malnutrition and immunosuppression significantly increase the risk of fistula formation as well.

2. CD is a common cause of ECF and enteroenteric fistulas.

3. Diverticular disease results in fistula formation 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 or recurrent urinary tract infections or sepsis.

4. Malignant fistulas form when tumor perforates or invades adjacent structures. Healing does not occur if cancer is present, and resection is the only means of cure.

5. Radiation enteritis predisposes to fistula formation after operation, regardless of the temporal proximity of exposure.

6. Trauma to the abdomen or pelvis may also cause SI fistulas. Missed enteric injuries, or those repaired in a contaminated field, are prone to leak and subsequent fistula formation.

7. Other causes of SI fistulas include a foreign body (mesh, suture), vascular compromise, and infectious diseases (amebiasis, tuberculosis, or actinomyces).

D. Diagnosis

1. Imaging. Contrast radiography aids in determining prognosis and assists with the planning of operative repair. Fistulography is the preferred test for mature external fistula tracts and typically provides good visualization of all tracts and sites of enteral communication. Oral contrast studies, such as an upper GI with small bowel follow through (SBFT), can demonstrate contrast extravasation through the fistula, but are less sensitive than fistulogram. A contrast enema is the study

of choice for rectal or colonic fistulas. CT scanning may be helpful for identifying the etiology of an internal fistula.

2. Endoscopy is useful to assess the bowel for underlying pathology, such as peptic ulceration, IBD, or cancer.

E. Spontaneous Closure. Common conditions under which fistulas fail to close can be remembered with the aid of the mnemonic ÒFRIENDSÓ: Foreign body, Radiation, Inflammation or Infection, Epithelialization, Neoplasm or lack of Nutrition, Distal obstruction, and/or Steroids (immunosuppression). Approximately 40% of ECFs will close spontaneously in 4 to 6 weeks with adequate nutritional support and control of sepsis. Increased rates of closure are seen in fistulas with low-output, long tracts (>2 cm), small orifices (<1 cm^2), and absence of malnutrition, abscess, sepsis, or active IBD. Delaying reoperation allows adhesions to attenuate and the patient to recover nutritional status and general health. For small fistulas, reoperation should be delayed at least 4 to 6 months from the time of last laparotomy. Improved home intravenous (IV) therapy, parenteral nutrition (TPN), wound care, and somatostatin analogs (see below) have allowed longer periods of waiting for fistula closure to be possible.

F. Nonoperative Treatment

1. Fluid resuscitation and electrolyte correction. The initial phase of ECF management focuses on correction of hypovolemia and electrolyte imbalance and accurate measurement of fistula output. IV fluid administration is typically necessary because adequate enteral replacement of fistula output is difficult.

2. Sepsis control is critical as sepsis remains the primary determinant of fistula mortality. Sepsis accompanies a large percentage of fistulas and is caused by undrained enteric leaks or abscesses. Percutaneous abscess drainage should be performed if present. **IV antibiotics** directed against bowel flora are indicated when infection is present. Reoperation for *source control* may be required to manage continuous bacterial seeding from the GI tract. **Infected wounds** are opened and packed to allow complete drainage, debridement, and healing by secondary intention.

3. Nutritional support. No level I evidence exists to support a nutritional route, although enteral feeding is widely preferred.

a. Complete bowel rest. Initial NPO status reduces fistula drainage and simplifies the evaluation and stabilization of the patient.

b. Enteral feeding is preferred as long as fistula output does not increase. Patients with low-output colonic or distal SI fistulas are often safely fed with standard enteral formulas. However, if the available bowel is short, elemental feeding may maximize absorption. In proximal fistula patients, feeding distal to the fistula is typically effective (e.g., feeding jejunostomy tube for a gastric fistula).

c. TPN provides adequate nourishment when enteral feeding is not possible. Indications include intolerance to enteral nutrition, high-output fistulas, and proximal fistulas where distal enteral access is not possible. Complications of TPN include biliary stasis, hepatic

dysfunction, trace element (zinc, copper, chromium) and essential fatty acid deficiencies, and venous catheter-related complications.

4. Control of fistula drainage. H_2-receptor antagonists or proton-pump inhibitors are used to reduce gastric and duodenal fistula output and provide stress ulceration prophylaxis. Use of somatostatin analogs has revealed mixed results.

5. Skin protection. Fistula effluent is corrosive to the skin and must be controlled. For low-output fistulas, dressings may be used to simply absorb effluent, but may impede healing and cause skin breakdown if prolonged contact occurs. Barrier/ostomy devices are useful as they isolate the effluent away from the skin and allow for quantification of output. Vacuum-assisted wound closure devices may help to control skin irritation and speed fistula closure. Early involvement of an enterostomal therapist is critical in the management of fistula patients.

G. Operative treatment is indicated when a fistula fails to heal with nonoperative management, or when sepsis cannot be controlled. The goals of surgery are to eradicate the fistula tract and to restore the epithelial continuity of the associated organ systems.

1. Gastric fistulas can arise from anastomotic breakdown or ulcer perforation. Most low-output gastric fistulas close spontaneously, such as that occurring after removal of a gastrostomy tube. In cases where surgery is needed, primary repair or serosal patch placement is usually successful.

2. Duodenal fistulas typically close spontaneously with nonoperative management. When operative intervention is required, primary closure of small duodenal wall disruptions may be performed, but a duodenal stricture may result with primary closure of large defects. In these cases, duodenal wall integrity may be restored by a serosal patch using another segment of bowel. Alternatively, a Roux-en-Y duodenoenterostomy may be performed.

3. Small-bowel fistulas typically require bowel resection and primary reanastomosis. For enteroenteric or other internal fistulas, openings that are in close proximity to the involved region are resected en bloc.

4. Large-bowel fistulas are associated with high spontaneous closure rates. If operative closure is required, resection with primary reanastomosis is preferred. A proximal, diverting loop ileostomy should be considered in the setting of malnutrition or suboptimal anastomosis.

5. Enteral feeding tubes placed at the time of definitive repair may facilitate postoperative management.

SHORT BOWEL SYNDROME (SBS)

SBS is a malabsorptive state and symptom complex following massive small-bowel resection. In adults, the normal length of the SI varies from 300 to 600 cm and correlates directly with body surface area. Adults with less than 200 cm of functional bowel or less than 30% of the initial SI length are at high risk of developing SBS. With an end stoma, resection resulting in <100 cm of intact SI generally leads to SBS. However, in patients with an intact ileocecal valve and one-third of the colon, SBS may

not develop until <75 cm SI remains. Children tend to develop SBS when <30% of normal SI length for age remains. Infants may survive resection of up to 85% of their bowel because of enhanced adaptation and growth. SBS may be seen with greater lengths of SI if an underlying disease, such as CD or radiation enteritis, is present. Because the ileum has specialized absorptive function, complete resection is not well tolerated. On the contrary, the entire jejunum can usually be resected without serious adverse nutritional sequela.

A. Etiology. In children, the most common etiologies for SBS include necrotizing enterocolitis, congenital intestinal atresia, midgut volvulus, and gastroschisis. The leading causes of massive intestinal resection in adults and elderly patients are mesenteric ischemia, trauma, IBD, strangulated hernia, SI or mesenteric neoplasms, volvulus, and portal vein thrombosis.

B. Pathophysiology. SBS is characterized by diarrhea, dehydration, electrolyte disturbances, steatorrhea, malnutrition, and weight loss.

1. Adaptation. The SI undergoes several adaptive changes in response to massive SI resection in an attempt to counteract the development of SBS. Slower transit and increased nutrient absorption occurs through functional adaptations. If colon is present, adaptation manifests as increased colonic absorption and colonocyte degradation of carbohydrates into short-chain fatty acids (SCFA), which increases caloric uptake up to 50%. With resection of the jejunum, the distal SI has the greatest adaptive potential and can assume nearly all of the absorptive properties of the proximal gut.

2. Fluid and electrolyte response. Of the 7 to 10 L of fluid presented daily to the SI, only 1 to 2 L are delivered into the colon. Significant quantities of electrolytes are absorbed in this process. The colon, if present, can absorb a significant amount of the increased fluid it encounters with SBS.

3. Malabsorption and malnutrition. Gastric hypersecretion causes increased acid load,

injures distal bowel mucosa, and leads to hypermotility and impaired absorption. Altered bilirubin metabolism after ileal resection increases the risk of **cholelithiasis** secondary to a decreased bile salt. Delivery of bile acids into the colon also produces a reactive, often severe watery diarrhea. **Hyperoxaluria** results from excessive fatty acids in the colonic lumen binding intraluminal calcium and leads to calcium oxalate **nephrolithiasis.** Loss of the ileocecal valve permits reflux of colonic bacteria into the SI leading to **bacterial overgrowth** and colonization that impairs digestion and absorption of nutrients. Rapid intestinal transit, hyperosmolar contents in the distal SI, disruption of the enterohepatic bile acid circulation, and bacterial overgrowth all promote **steatorrhea** and **diarrhea.** Unabsorbed fats in the colon further inhibit absorption of water and electrolytes and stimulate secretion.

C. Acute Phase Treatment. The primary goal in the acute phase (initial 4 weeks) is stabilization as metabolic, respiratory, and cardiovascular derangements frequently accompany massive small-bowel resection. Close monitoring of fluid balance and serum electrolytes are critical. **Prolonged ileus** is common. TPN should be provided until GI function resumes.

Early initiation of nutritional support promotes a positive nitrogen balance, wound healing, and adaptation of remnant bowel. Enteral nutrition has positive trophic effects on bowel mucosa and should be initiated. Feeding tubes placed at laparotomy are often necessary. Initial feeds should be gradual, continuous, low-volume, low-fat, and isosmotic.

D. Maintenance Phase Treatment. Maintenance therapy in SBS focuses on long-term nutritional goals, support of adaptation that takes place over the first 1 to 2 years, and addressing various clinical issues that arise.

1. Nutritional support with supplemental electrolytes (potassium, magnesium), vitamins (A, D, E, K, B_{12}), trace elements and minerals (zinc, selenium, and iron), and essential fatty acids (linoleic acid) should be given parenterally until adequate enteral absorption is achieved. Teduglutide, a glucagon-like peptide (GLP-2) analogue, is a novel therapy shown to increase adaptation and decrease TPN dependence. Growth hormone and glutamine have also been used with some success.

2. Diarrhea is often multifactorial and dietary modifications can improve symptoms. Medications such as H_2-receptor blockers, chelating resins (cholestyramine), antisecretory medications (loperamide, somatostatin analogs), and low-dose narcotics (diphenoxylate hydrochloride and atropine [Lomotil], codeine, or tincture of opium) are useful for decreasing output.

3. Late complications are common and include nephrolithiasis, cholelithiasis, nutritional deficiencies (anemia, bone disease, and coagulopathy), liver dysfunction, as well as TPN and central access-related complications. Anastomotic leaks, fistulas, strictures, and late bowel obstructions can also occur well beyond the early postoperative period and commonly require reoperation.

E. Surgical Therapy. Various surgical procedures have been described for the management of SBS but have not been widely adopted. Intestinal lengthening procedures may decrease TPN dependence, increase oral caloric intake, and reverse liver disease. The most common procedures are Serial Transverse Enteroplasty (STEP) and the Bianchi procedure, both with similar efficacy. Isolated small-bowel transplants or multivisceral transplantations are additional options for SBS.

NEOPLASMS

Small-bowel neoplasms are relatively uncommon. Benign neoplasms are often discovered incidentally, and malignant tumors account for <2% of all GI cancers. Most malignant tumors eventually become symptomatic with weight loss, abdominal pain, obstruction, perforation, or hemorrhage. Small-bowel neoplasms can also be a lead point for intussusception.

A. Benign Tumors. Benign small-bowel masses are more common than malignant.

1. Leiomyoma is the most common benign neoplasm of the SI and arises from mesenchymal cells. These tumors grow submucosally and project into the bowel lumen. On contrast studies, they appear as smooth,

eccentric filling defects with normal-appearing mucosa. Histopathologic examination is needed to distinguish benign from malignant stromal tumors. Treatment consists of segmental bowel resection.

2. Adenomas can occur sporadically as solitary lesions, or in association with familial adenomatous polyposis syndrome. Adenomas can cause intermittent pain secondary to obstruction, intussusception, or bleeding. Three subtypes include: Simple tubular, Brunner gland, and villous adenomas. The duodenum is the most common site for all three types of adenomas. Tubular and Brunner gland adenomas have low malignant potential and may be treated with endoscopic polypectomy. Villous adenomas have significant malignant potential. If complete endoscopic resection is not possible, transduodenal excision with adequate margins is appropriate. Villous adenomas of the jejunum or ileum require smallbowel resection.

3. Hamartomas may be spontaneous, but most arise in patients with PeutzÑJeghers syndrome, an autosomal-dominant syndrome characterized by mucocutaneous hyperpigmentation and multiple GI polyps. Operative intervention is indicated only for symptoms, and all polyps larger than 1 cm should be resected. These patients are at increased risk for *de novo* SI and colonic adenocarcinoma (arising separately from the hamartomas) and require frequent endoscopic screening. Multiple resections can lead to risk of SBS, so local excision or endoscopic treatment of noncancerous polyps is preferred.

4. Other benign tumors. Lipomas occur most often in the ileum and have no malignant potential. **Hemangiomas** are associated with OslerÑ WeberÑRendu disease and present with bleeding. **Neurofibromas and fibromas** are less common tumors that can cause intussusception. **Endometriosis** implants appear as puckered, bluish-red, serosal-based nodules

that can cause GI bleeding or obstruction.

B. Malignant Tumors

1. Adenocarcinoma is the most common malignant SI tumor, with 40% in the duodenum and then with decreasing frequency distally through the SI. Risk factors for development of adenocarcinoma include villous adenomas, polyposis syndromes, CD, and hereditary nonpolyposis colorectal cancer (HNPCC). Presenting symptoms depend on the location of the primary tumor. Periampullary tumors present with painless jaundice, duodenal obstruction, or bleeding. Distal tumors tend to present with abdominal pain and weight loss from progressive obstruction. *Diagnosis* is made via CT and endoscopy with or without ERCP for biopsy. *Treatment* consists of en bloc resection with the associated mesenteric nodal basin. Tumors of the terminal ileum are resected with the right colon as well. Carcinomas of the duodenum usually require pancreaticoduodenectomy. The 5-year survival rate for duodenal adenocarcinoma is 56% for node-positive and 83% for node-negative disease. Patients with metastatic disease at the time of diagnosis rarely survive past 6 months. 5-FluorouracilÑbased chemotherapy regimens are often used, but data on their efficacy are lacking.

2. Gastrointestinal stromal tumors (GISTs) arise from mesodermal-derived components of the bowel and are equally distributed along the length of the intestine. These tumors grow extraluminally and cause symptoms late in their course. Hemorrhage into either peritoneum or bowel lumen may result when these tumors outgrow their blood supply and necrose. Mutations of **c-kit** (CD117, a tyrosine kinase) allow diagnosis by immunohistochemistry. Histologic grade and tumor size are predictors of survival. Curative *treatment* for GIST requires wide en bloc resection with tumor-free margins. Extensive lymphadenectomy is unnecessary as these tumors have low potential for lymphatic spread. Traditional chemo/radiation therapy is not the effective treatment for GISTs. However, the tyrosine kinase inhibitor *imatinib mesylate (Gleevec)* effectively inhibits the overactive tyrosine receptor c-kit found on all GIST cells. Adjuvant imatinib therapy improves recurrence-free survival (*Lancet.* 2009;373(9669):1097-1104), and has also been shown to cause radiographic and histologic regression of metastatic lesions. Neoadjuvant imatinib is indicated for tumors deemed unresectable at diagnosis in order to downstage disease with a goal of curative resection. Sunitinib malate (Sutent) is second-line therapy for metastatic GIST resistant to imatinib (*Lancet.* 2006;368:1329-1338). Selected patients with metastatic GIST appear to benefit from elective surgical resection. After complete resection, overall 5-year survival rate is 50%. In low-grade tumors, survival rate is 60% to 80%, whereas in high-grade tumors, survival rate is less than 20%. Median length of survival is 9 to 12 months with locally recurrent disease, and 20 months with metastatic disease.

3. Primary small-bowel lymphomas are most common in the ileum due to relatively large amounts of gut-associated lymphoid tissue. Virtually all small-bowel lymphomas are non-Hodgkin, B-cell lymphomas (NHL) that arise either de novo or in association with a preexisting systemic condition such as celiac disease, CD, or immunosuppression (iatrogenic, HIV, etc.). The

presentation of these patients is highly variable. Imaging can help make a diagnosis, but operation is frequently required for histologic confirmation. For stage I and II intestinal NHL, treatment includes wide segmental bowel resection and adjuvant chemotherapy (CHOP regimen). Early stage NHL treated with combined surgical and chemotherapy results in improved overall 3-year survival rates as compared to treatment with chemotherapy alone (91% vs. 62%). Treatment of advanced or systemic intestinal NHL (stage III/IV) remains controversial. Resection of the affected intestine may be performed to prevent complications such as obstruction or bleeding.

4. Neuroendocrine tumors (NET) arise from enterochromaffin cells of intestinal crypts. Most intestinal NETs occur within 2 feet of the ileocecal valve. Small-bowel NETs tend to be more aggressive than their appendiceal or rectal counterparts. Patients typically remain asymptomatic until advanced disease causes local complications of GI obstruction, pain, or bleeding, or the systemic carcinoid syndrome. Metastases are rare in tumors <1 cm in size, while half of tumors between 1 and 2 cm metastasize, and almost all tumors >2 cm spread.

a. *Carcinoid syndrome* implies hepatic metastatic spread. Hormones released by carcinoid tumors are metabolized by the liver and produce no symptoms; however, hepatic metastases drain into the systemic circulation causing *diarrhea* and *flushing* of the face, neck, and upper chest. Tachycardia, hypotension, bronchospasm, and coma may be observed. In long-standing carcinoid syndrome, patients develop right heart endocardial and valvular fibrosis. Diagnosis of NET is made by measuring a 24-hour urinary 5-hydroxyindoleacetic acid (5-HIAA), the breakdown product of serotonin secreted by the tumor. Serum chromogranin A measurement is another diagnostic test for GI NET with high sensitivity (80% to 100%), but lower specificity than urine 5-HIAA levels.

b. The **treatment** of NET is operative. The entire bowel should be inspected as 30% of cases have synchronous lesions. Jejunal and ileal tumors are treated with segmental resection including adjacent mesentery. Small tumors (<1 cm) of the third or fourth portions of the duodenum can be either locally excised or included in a segmental resection. Large duodenal tumors and periampullary tumors require pancreaticoduodenectomy. Locally advanced disease with involvement of adjacent organs or peritoneum requires aggressive resection to delay occurrence of mesenteric desmoplastic reaction, hepatic metastases, and carcinoid syndrome. Solitary and accessible liver lesions should be resected. Adjuvant cytotoxic chemotherapy and radiotherapy are of little benefit. The somatostatin analog **octreotide** offers excellent palliation of carcinoid syndrome symptoms in patients with unresectable disease.

c. NETs are slow-growing tumors, and **prognosis** depends on stage of the tumor. Overall 5-year survival rate is 60%, and patients with local disease that is completely resected have a normal life expectancy. For patients with resectable node-positive disease, median length of survival is 15 years. With unresectable intra-abdominal disease median length of survival drops to 5 years, and

is 3 years for those with hepatic metastases.

5. Metastases can spread to the small bowel and palliative resection may be appropriate if required for symptom relief. Several primary cancers are known to metastasize to the small bowel including melanoma, colorectal, gynecologic, breast, stomach, lung, prostate, and renal cancers. Median survival is poor. Cases should be considered individually. Palliative gastrostomy with or without TPN may be appropriate in advanced cases where nonoperative management is chosen.

CROHN DISEASE

CD is an idiopathic, chronic, granulomatous IBD that can affect any part of the GI tract from mouth to anus. CD is incurable, slowly progressive, and characterized by episodes of exacerbation and remission. The incidence is 4/100,000 with a bimodal age distribution at 15 to 29 and 55 to 70 years old.

A. Etiology. The cause of CD is unknown, but is believed to involve both genetic and environmental factors. CD is 25 times more common among

patients with a family history and has a concordance rate of 60% in monozygotic twins. Environmental aspects, such as smoking, also increase the risk of developing CD. Pathogenesis likely relates to a defective mucosal barrier and/or dysregulated intestinal immunity leading to chronic inflammation within the intestinal wall.

B. Bowel Involvement. The *terminal ileum* is the most common site of disease and is involved in 75% of all cases of CD. **Ileocolic** disease affecting the terminal ileum and/or cecum is the most common form, affecting 40% of patients. **Small-bowel-only** disease (30% of patients) is confined to the more-proximal bowel. **Colonic** disease (30% of patients) affects only the large intestine. **Perianal involvemen**t commonly coexists with more proximal forms, especially when the colon is affected. Isolated **anorectal** disease is rare (5%).

C. Histology. CD is characterized by *transmural inflammation.* Grossly, the bowel is thickened with creeping fat, corkscrew vessels, and a shortened fibrotic mesentery with lymphadenopathy. Mucosal changes include pinpoint hemorrhages, aphthous ulcers, deep linear fissures, crypt abscesses, and *cobblestoning.* These findings commonly occur segmentally, causing *skip lesions* along the intestine rather than being continuous. *Granulomas* are found in the bowel wall in 40% to 60% of patients.

D. Clinical Presentation. CD has a highly variable presentation. Physical examination is performed with special attention to the abdominal and anorectal areas. No physical signs are pathognomonic for CD, although the appearance of the perineum may be highly suggestive. **Diarrhea** occurs in almost all patients. Patients with ileal disease may have steatorrhea secondary to bile salt deficiency. **Abdominal pain** typically is colicky, worse after meals, relieved by defecation, and poorly localized. **Weight loss** occurs as a result of decreased oral intake, malabsorption, protein-losing enteropathy, and/or steatorrhea. Children with CD may develop

vitamin and mineral deficiencies and growth retardation. **Constitutional symptoms** such as malaise and fever are common. **Anorectal** disease is common and may precede intestinal symptoms. **Extraintestinal manifestations** can be numerous, including conjunctivitis, iritis, uveitis, pyoderma gangrenosum, erythema nodosum multiforme, arthritis, ankylosing spondylitis, and even sclerosing cholangitis.

E. Endoscopy. Lower, and sometimes upper, endoscopy is crucial for determination of location and severity of disease as well as diagnostic biopsies. Those with long-standing (>7 to 10 years) Crohn colitis are at increased risk for adenocarcinoma, and surveillance colonoscopy for cancer is important.

F. Imaging. Barium studies are widely available and produce quality images; however, they only assess intraluminal mucosal pathology and may have limited diagnostic accuracy. These techniques have been all but supplanted by modern cross-sectional imaging, such as CT- and MR-enterography (CTE and MRE, respectively).

G. Differential Diagnosis. Other IBD as well as common infectious abdominal conditions can mimic CD and include ulcerative colitis, appendicitis,

infectious ileitis, intestinal lymphoma, intestinal tuberculosis, ischemic enteritis, diverticulitis, pseudomembranous colitis, and irritable bowel syndrome.

H. Treatment

1. Medical management is important to palliate symptoms, correct nutritional disturbances, and reduce inflammation. Disease location, severity, and complications dictate therapeutic recommendations. Mild-to-moderate disease can be treated as an outpatient with oral aminosalicylates. Initiation of antibiotics (ciprofloxacin, metronidazole) is indicated in patients who do not tolerate aminosalicylates or do not improve with aminosalicylate therapy. Oral prednisone may be used for patients who are unresponsive to the above measures, or for those presenting with more severe initial symptoms (but not requiring hospitalization). Budesonide is a glucocorticoid with a high first-pass hepatic metabolism that is an alternative to prednisone for patients with active ileitis or right-sided Crohn colitis. Hospitalization is required for patients who present with severe or fulminant disease. Inpatient treatment includes bowel rest, TPN, and IV glucocorticoids. Patients who are steroid dependent or steroid resistant may require treatment with immunomodulator or biologic therapies. Immunomodulators include azathioprine, 6-mercaptopurine, and methotrexate. Biologic therapies include anti-tumor necrosis factor-alpha (anti-TNF) antagonists and anti-integrin antibodies. Anti-TNFs are effective in treatment of moderate to severe luminal CD, including infliximab, adalimumab, and certolizumab. Anti-integrin antibodies act by blocking leukocyte migration to sites of inflammation, and include natalizumab and vedolizumab.

2. Surgical therapy is indicated when medical therapy has failed or to address complications such as high-output fistulas, perforation, intraabdominal abscess, severe colitis, bleeding, or

obstruction from fibrotic strictures.

a. At the time of operation, the most important principle is to correct the complication while *preserving bowel length* to prevent SBS. Resection to histologically negative margins does not reduce likelihood of disease recurrence; therefore, grossly normal margins are accepted. In the absence of free perforation, large abscesses, massively dilated bowel, severe malnutrition, or high-dose immunosuppression, primary anastomosis is safe. Laparoscopic resections are safe alternatives to open procedures. Strictures can be treated with stricturoplasty.

b. *Appendectomy.* Patients who are being explored for presumed acute appendicitis and are found to have Crohn ileitis should have appendectomy if the cecum is not inflamed. Conventional teaching has been that the terminal ileum should not be removed.

I. Prognosis. CD is a chronic, pan-intestinal disease that currently has no cure and requires chronic, lifelong treatment, with operation reserved for severe complications. Specific Òsusceptibility genesÓ (e.g., *NOD2/CARD15*) have been identified in patients with CD. Further study of the pathways involved may shed light on pathogenesis and lead to more effective medical treatments.

CHAPTER 19: SMALL INTESTINE

Multiple Choice Questions

1. A 29-year-old male presents to the emergency department with complaints of abdominal pain, nausea, and bilious vomiting for 2 days. He has no significant past medical history, and his past surgical history is significant for an open appendectomy for perforated appendicitis. CT scan demonstrates dilated loops of small bowel with a transition point in the right lower quadrant and is negative for free air or fluid. The most likely etiology for this patient's condition is:

a. Intussusception

b. Malignancy

c. Adhesions

d. Crohn disease

e. Gallstone ileus

View Answer

2. A 45-year-old male presents to the emergency department with complaints of abdominal pain, nausea, and bilious vomiting for 2 days. His last bowel movement was 3 days ago. His past medical history is significant for hypertension and diabetes, and his past surgical history is significant for an open appendectomy for perforated appendicitis and

open ventral hernia repair. His initial vitals in the emergency room are: Temperature 39°C, heart rate 115, blood pressure 90/54, respirations 22, O_2 saturation 92% on room air. On physical examination, he has a well-healed lower midline incision, is firm, moderately distended, and has a diffuse tenderness to palpation of his abdomen with rebound and guarding. CT scan demonstrates dilated loops of small bowel with a transition point in the right lower quadrant and a moderate amount of free fluid in the pelvis. You place an NGT and begin fluid resuscitation with LR. The next best step in the treatment of this patient would be:

a. Admission for close monitoring

b. Urgent laparotomy

c. Obtain an upper GI study with small bowel follow through using water-soluble contrast

d. Reassurance and discharge with followup in 1 week

e. Urgent colonoscopy

View Answer

3. A 70-year-old male presents to the emergency department with abdominal pain, nausea, and bilious vomiting. He also complains of a painful bulge in his left groin that has been present for 4 hours. His past medical history is significant for hypertension, chronic obstructive pulmonary disease, and hypothyroidism. He has no past surgical history. His initial vitals in the emergency room are: Temperature 37.5°C, heart rate 80, blood pressure 165/90, respirations 14, O_2 saturation 93% on room air. On physical examination, he is moderately distended, and has mild tenderness to palpation throughout his abdomen, and a firm, painful bulge in his left groin above the inguinal ligament. There are no overlying skin changes in his groin. You start fluid resuscitation with LR. The next best step in the treatment of this patient would be:

a. Attempt manual reduction in the ED with light sedation

b. Obtain a CT scan of the abdomen

c. Discharge the patient with a follow-up appointment in 1 week

d. Urgent laparotomy

e. Elective inguinal hernia repair

View Answer

4. A 56-year-old female on whom you performed an ileocolic resection for Crohn disease presents to your office for routine postoperative followup 2 weeks after her operation. She reports that she has been feeling well, but that stool began draining from the inferior aspect of her midline incision a few days ago. Vital signs are: Temperature 37°C, heart rate 75, blood pressure 130/75, respirations 15, O_2 saturation 100% on room air. On physical examination, her abdomen is soft, nontender, and compressible. Her upper midline incision has a dressing in place with a minimal amount of brown drainage. Which of the following treatments would NOT be indicated for the patient at this time?

a. Consultation with enterostomal therapist for skin protection regimen

b. Quantification of fistula output

c. Bowel rest and parenteral nutrition

d. Urgent laparotomy

e. CT scan with oral and IV contrast

View Answer

5. A 60-year-old female presents to the emergency room with signs and symptoms of bowel obstruction. On CT scan, a mass is visualized in the jejunum with dilation of the small bowel proximal to the mass and several lesions in the liver concerning for metastasis. You perform a laparotomy and perform a segmental bowel resection and core biopsy of a liver lesion. Final pathology reveals cells that are c-kit positive. Which of the following is the best chemotherapeutic regimen for this patient?

a. Imatinib mesylate (Gleevec)

b. 5-FU and oxaliplatin

c. Cyclophosphamide + doxorubicin + vincristine + prednisone (CHOP)

d. Trastuzumab (Herceptin)

e. Octreotide

View Answer

6. A 45-year-old male with widely metastatic small-bowel neuroendocrine tumor has symptoms of flushing and diarrhea. Which of the following drugs would be useful to control his symptoms?

a. 5-FU and oxaliplatin

b. Sunitinib malate (Sutent)

c. Octreotide

d. Tincture of opium

e. Diphenoxylate hydrochloride and atropine

View Answer

7. A 10-year-old girl with short bowel syndrome secondary to congenital malrotation and volvulus presents to the emergency department with fevers of 39.5°C, tachycardia, and hypotension. She receives TPN via a tunneled central venous catheter. Her physical examination is unremarkable. WBC is 12. Chest x-ray demonstrates her central line, and abdominal plain films demonstrate a normal gas pattern, no free air. The most likely acute diagnosis for this patient is:

a. Hyperthyroidism

b. Central venous catheter associated blood stream infection

c. Intra-abdominal abscess

d. Pneumonia

e. Small-bowel perforation

View Answer

20
Surgical Diseases of the Liver

Chun-Cheng (Richard) Chen

William C. Chapman

I. CAVERNOUS HEMANGIOMAS

A. Presentation. Hemangiomas are the most common benign liver tumor. The pathogenesis of hemangiomas is poorly understood. They are thought to represent hamartomatous outgrowths of endothelium rather than true neoplasms. Accelerated growth has been associated with high-estrogen states, such as puberty, pregnancy, and when oral contraceptives (OCPs) and androgens are used. Hemangiomas are usually less than 5 cm in diameter, but they can reach 20 cm or larger. Their blood supply is derived from the hepatic artery. On gross inspection, they are flat, redÑblue, well circumscribed, soft, and easily compressible. Malignant degeneration does not occur, and spontaneous rupture is exceedingly rare. Most hemangiomas are asymptomatic and are identified incidentally during imaging examinations for unrelated reasons. Patients with large lesions occasionally complain of nonspecific abdominal symptoms such as upper abdominal fullness or vague pain. Intermittent symptoms may occur with necrosis, infarction, or thrombosis of the tumor. Life-threatening hemorrhage can be precipitated by needle biopsy. *KasabachÑ Merritt syndrome* is a rare consumptive coagulopathy resulting from sequestration of platelets and clotting factors in a giant hemangioma, and this is usually treated with urgent resection.

B. Diagnosis. Laboratory abnormalities are rare. Because of the possibility of severe hemorrhage from a biopsy, diagnosis relies on imaging. On ultrasound, hemangiomas appear as well-demarcated, lobulated, homogeneous, hyperechoic masses, with hypoechoic regions representing hemorrhage, fibrosis, and/or calcification. Compressibility of the lesion is pathognomonic. Ultrasound is highly sensitive but not specific, with an estimated overall accuracy of 70% to 80%. Contrast-enhanced tomographic imaging typically show *centripetal enhancement over time.* On computed tomography (CT) scans, a low-density area with characteristic peripheral enhancement is seen in the early phase. Delayed images show contrast enhancement progresses toward the center of the lesion until the tumor appears uniformly enhanced. The best imaging study is gadolinium-enhanced magnetic resonance imaging (MRI), with specificity and sensitivity approximately 90% and 95%, respectively. These tumors appear bright on T2-weighted images, with initial hyperintense nodules that enhance centripetally with delayed images as in CT.

C. Treatment. Most hemangiomas can be safely observed. Indications for intervention include severe symptoms, complications, and inability to

exclude malignancy. In these select patients, the preferred treatment is typically enucleation under vascular control with intermittent Pringle maneuver. Formal anatomic resection is reserved for tumors that have 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 poor operative candidates. In the very rare case of spontaneous hemorrhage, control with vascular embolization provides temporary help until a definitive operative approach can be safely implemented.

II. FOCAL NODULAR HYPERPLASIA

A. Presentation. Focal nodular hyperplasia (FNH) is the second most common benign hepatic tumor, constituting about 8% of cases. The pathogenesis of FNH is thought to represent a nonneoplastic, hyperplastic response to hyperperfusion from a congenital arterial malformation. The lesions usually are solitary, well circumscribed, lobulated, and unencapsulated. FNH is found predominantly in women of child-bearing age. Although an association with OCPs has been suggested, the correlations are much lower than are those for hepatic adenomas (HAs). Only a minority of patients are symptomatic with epigastric or right upper quadrant pain and a palpable mass. Spontaneous rupture with hemorrhage is extremely rare. Malignant degeneration has not been reported, but it is critical to distinguish FNH from the *fibrolamellar variant of HCC,* a malignant lesion that may have a similar central scar. It is important to note that the latter's scar is usually large and eccentric, with broad fibrous bands and calcifications.

B. Diagnosis. Ultrasound does not discriminate FNH from other pathology well. FNH lesions are often isoechoic to surrounding normal liver, although the scar may be slightly hyperechoic. Distinguishing characteristics on multiphasic CT can be readily identified. Because of its vascular supply, FNH has a bright homogeneous *enhancement in the arterial phase* and a *hypodense central scar.* Delayed images may show hyperattenuation of the central scar. When MRI is employed, the central scar appears hyperintense on T2-weighted images, and when contrast is used, the enhancement pattern is similar to that seen on CT. Superparamagnetic iron oxide (SPIO) is an MR contrast agent that undergoes phagocytosis by the reticuloendothelial system. On SPIO-enhanced T2-weighted images, FNH is hypointense but with a bright central scar. On hepatic scintigraphy with Tc-99m sulfur colloid, FNH has variable colloid uptake compared with the normal liver. However, intense colloid uptake (10% of cases, related to the number of Kupffer cells present) is a very specific finding for FNH. In combination, use of different imaging modalities, especially MRI, yields a precise diagnosis of FNH in 70% to 90% of cases. When the diagnosis remains in doubt, biopsy and rarely, resection, may be indicated.

C. Treatment. There is no role for resection in asymptomatic patients when studies can differentiate FNH from adenoma or malignant lesions. If the lesion is symptomatic and unresectable, transarterial embolization has been

reported to be effective, but this need is exceedingly rare. There is no contraindication to

pregnancy with this lesion, but close observation for tumor growth during pregnancy and the postpartum period is prudent.

III. HEPATIC ADENOMA

A. Presentation. HA is a benign proliferation of hepatocytes that is found predominantly in young women, with a strong association to synthetic estrogen and progesterone use; however, the incidence has stabilized due to the decreased estrogen content found in OCPs. Anabolic steroids may drive the growth of these lesions. Adenomas are usually solitary (70% to 80%), round, well-circumscribed lesions. Although unencapsulated, the tumor often has a pseudocapsule formed by compressed normal surrounding tissue. Microscopically, they are made up of monotonous sheets of hepatocytes separated by dilated sinusoids but does not contain bile ductules, a key histologic finding distinguishing it from FNH where these are present. HA is of clinical importance because of its tendency to *spontaneous rupture and hemorrhage.* The rate of rupture has been estimated between 25% and 35% with nearly 100% of all spontaneous ruptures occurring in lesions greater than 5. About one-third of patients with symptoms present with intraperitoneal bleeding, and others present with abdominal pain without rupture. More often, these lesions present with vague symptoms such as fullness or discomfort in the right upper quadrant or are detected incidentally. These lesions are potentially premalignant, with large or multiple tumors and those in men carrying a greater risk of malignant degeneration. Spontaneous rupture also occurs more often in men, especially in steroid users. It may also occur during pregnancy due to rapid growth under the influence of estrogens.

B. Diagnosis. Ultrasonography can identify lesions but cannot differentiate an adenoma from a malignant lesion. Unlike FNH, HA frequently appears heterogeneous on CT and MRI due to intratumor hemorrhage, necrosis, and fat. On multiphasic CT, HA demonstrates *arterial-phase enhancement* with smooth surfaces and evidence of tumor capsule or hemorrhage. MRI is the best imaging modality because of its sensitivity for fat, thus making the lesion hyperintense on T1, but the tumor heterogeneity is considered a hallmark feature of HA.

C. Treatment. Operative intervention after HA rupture is mandatory, but usually arterial embolization is utilized for control of the initial hemorrhage and surgical resection considered 6 to 8 weeks later. Small (<3 cm) and sometimes larger, asymptomatic lesions occasionally regress with cessation of OCPs. Tumors greater than 5 cm should be considered for surgical resection. Anatomic or segmental resections are recommended, but a wide margin is not necessary. Although radio frequency ablation (RFA) may be an option when there are multiple adenomas, resection of HA remains the standard therapy.

IV. BILE DUCT HAMARTOMAS.

Bile duct hamartomas are the most common liver lesions seen at laparotomy. They are usually peripherally located and firm,

smooth, and white in appearance. Typically, lesions are 1 to 5 mm in diameter, but they may be larger. Distinguishing them from military metastatic lesions (especially those from colorectal cancer or cholangiocarcinoma) may be difficult. Where there is uncertainty, biopsy should be performed.

V. HEPATOCELLULAR CARCINOMA (HEPATOMA)

A. Presentation. The annual incidence in the United States of hepatocellular carcinoma (HCC) is approximately 2.4 per 100,000, with around 30,000 new cases and around 20,000 deaths annually from this disease. The incidence is rising due, in large part, to the hepatitis C virus (HCV) epidemic. There is a 2 to 3:1 male-to-female predominance. The incidence in African-American men is almost twice that in White men. HCC is diagnosed mainly in the fifth and sixth decades. *Major risk factors* for cirrhosis in the United States include hepatitis C, alcohol abuse, autoimmune phenomena such as primary biliary cirrhosis (PBC), primary sclerosing cholangitis (PSC), and autoimmune hepatitis, and hereditary metabolic disorders (e.g., hemochromatosis). Seventy to eighty-five percent of HCC arises in the setting of cirrhosis. Malignant tumors of the liver occur in 4.5% of cirrhotic patients and in up to 10% when hemochromatosis is the inciting factor. Initial presenting symptoms are vague and include weight loss, weakness, and dull, persistent, epigastric, or right upper quadrant pain. Acute severe abdominal pain infrequently has been associated with intraperitoneal hemorrhage due to rupture of a necrotic nodule or erosion of a blood vessel.

B. Diagnosis. Initial workup includes obtaining imaging studies as well as liver function tests to determine prognosis and the best treatment strategy.

1. Biochemical. In cirrhotic patients, HCC may be associated with abnormal liver function tests due to hepatitis. Elevated *serum α-fetoprotein* (AFP) occurs in 75% of affected African patients but in only 30% of patients in the United States. AFP is also often elevated in chronic hepatitis and cirrhosis, in the absence of malignancy. An AFP level greater than 200 ng/mL (normal <20 ng/mL) is suggestive of HCC, even in the cirrhotic patient.

2. Imaging. *Ultrasonography* can be accurate in the detection of HCC, especially when coupled with concomitant AFP elevations. *MRI* can be useful in differentiating other small nodular masses from HCC. This is the most accurate imaging modality for distinguishing HCC from dysplastic or regenerative nodules in the cirrhotic patient. *Multiphasic CT scan* with arterial and portal venous phase contrast imaging can distinguish among different types of liver masses. *HCC enhances in the arterial and not usually in the portal venous phase.* Washout of contrast in the delayed (portal venous) phases of enhancement is an additional characteristic of HCC, with sensitivity of about 80% with specificity of 95% to 100%. Washout is defined as hypointensity of a nodule in the delayed phase compared with surrounding liver parenchyma.

3. Tissue biopsy. When required, laparoscopic or image-guided percutaneous biopsies may be used to obtain a tissue diagnosis. However,

a tissue diagnosis is not required before therapeutic intervention if other diagnostic modalities favor HCC as the diagnosis. Unresectable tumors likewise usually do not require biopsy to confirm the diagnosis because imaging and laboratory studies allow a definitive diagnosis in the majority of cases.

C. Treatment. Without treatment, HCC has a poor prognosis, with a median length of survival of 3 to 6 months after the diagnosis. Treatment options depend on availability of donor livers for transplantation and, for patients not eligible for transplantation, the resectability of the tumor.

1. Orthotopic liver transplantation (OLT) is the best treatment option for HCC because it removes the tumor together with the entire diseased liver, thus reducing the risk of *de novo* or recurrent disease. Mazzaferro and colleagues *(N Engl J Med.* 1996;334:693) demonstrated that when OLT was restricted to patients with a single tumor 5 cm or less or patients with up to three tumors with the largest less than 3 cm in size with no vascular invasion on imaging and no nodal or distant metastases, the 4-year actuarial survival rate was 75%, with recurrence-free survival of 83%. These so-called ÒMilan criteriaÓ have subsequently been adopted by the United Network for Organ Sharing (UNOS). Recent reports suggest that even more advanced patients who are downstaged with pretransplant therapy have results similar to those with early stage HCC.

2. Surgical resection with anatomic resection is the treatment of choice for patients who have normal background liver and sometimes for those with fibrosis or well-compensated early cirrhosis and no evidence of portal hypertension (PH) and have a normal bilirubin (ChildÑPugh class A). However, this constitutes only 5% of HCC patients in the United States and up to 40% of those in Asian countries. A macroscopic margin of 1 cm generally is regarded as adequate. Overall 5-year survival rates for patients with HCC treated with resection is 40% to 50%, with recurrence rates of around 40% to 50%. The most important predictors of recurrence are microvascular invasion and multinodular tumors. Repeat hepatic resection for recurrence has been demonstrated to be safe and effective in selected patients. Safety of hepatic resection depends on ensuring a sufficient *future liver remnant* (FLR) to prevent postoperative liver failure. FLR can be estimated directly using preoperative tomographic imaging with computer software. The recommended FLR (minimum) is about 25% for healthy livers and 40% for diseased livers.

3. Local ablation is the best treatment option for patients who have early-stage HCC and are not suitable for resection or OLT. In addition, these therapies may serve as bridges to OLT for those on the transplant waiting list. Indeed, downstaging tumors improve survival of HCC patients who subsequently undergo OLT. Today ablation is most frequently accomplished by transarterial approaches (chemo or radioembolization) or sometimes with thermal ablation with radiofrequency, microwave, or cryoablation for smaller tumors. *Radiofrequency*

ablation has emerged as the most common modality in most centers and operates by heating lesions to temperatures of greater than 60¡C using electrical currents. RFA may be performed intraoperatively or percutaneously under imaging guidance. Extendable electrodes within the

needle allow larger tumors upward of 4 cm to be ablated, but there is a higher recurrence rate in ablative approaches than resection. *Transarterial chemoembolization (TACE)* involves selective intraarterial administration of chemotherapeutic agents followed by embolization of the major tumor artery. HCC preferentially derives its blood supply from the hepatic artery rather than from a combination of the hepatic artery and portal vein as for normal hepatic parenchyma. TACE has a survival benefit for select patients with unresectable tumors, Child class A cirrhosis, and tumors less than 5 cm. The procedure rarely may be complicated by hepatic failure due to infarction of adjacent normal liver. For this reason, it should not be used in decompensated (Child class C) cirrhosis.

4. Systemic chemotherapy and external beam radiation have had poor results. Chemotherapy with conventional cytotoxic agents is ineffective and does not seem to modify the natural history of disease. Recent identification of signaling pathways in HCC has resulted in the development of drugs directed at specific therapeutic targets. One such drug is sorafenib, a tyrosine kinase inhibitor with antiangiogenic and antiproliferative properties that has shown modest efficacy in patients with advanced HCC. Toxicity may lead to decompensation of liver disease. Combining chemotherapy with surgical resection preoperatively or postoperatively has no benefit in terms of patient survival.

VI. FIBROLAMELLAR HEPATOCELLULAR CARCINOMA.

Fibrolamellar hepatocellular carcinoma (FLC) is a rare histologic variant of HCC. However, there is considerable evidence that FLC is distinct from HCC in its epidemiology, biology, and prognosis. Males and females are equally affected, commonly at a younger age (20 to 40 years old). It is uncommon for FLC to be associated with underlying liver disease such as cirrhosis. The histology of FLC strongly resembles that of FNH, but any etiologic association between them remains unproven. FLC appears as a hypoattenuated, well-defined, solitary mass on nonenhanced CT scan. On contrast-enhanced CT, the cellular portion enhances homogeneously; the central scar usually does not enhance, unlike the scar of FNH. FLC is best treated with complete surgical resection, which is possible in 80% of patients. Resectable FLC is associated with a better prognosis than HCC, with a 5-year survival rate greater than 70%. Late recurrence occurs in more than two-thirds of cases, and repeat resection of local disease should be considered. Liver transplantation is an option for unresectable but nonmetastatic lesions.

VII. METASTATIC DISEASE TO THE LIVER.

This represents the most common malignancy of the liver in the United States. The liver is a common site of metastasis from gastrointestinal (GI) cancers because it is the first organ

drainage site of venous blood from the GI tract. Prognosis typically depends on the resectability of the existing disease which in turn is dependent on the total tumor burden in the liver. Without treatment, hepatic metastases herald a dismal prognosis, with a median survival of 5 to 10

months.

A. Colorectal cancer metastases to the liver afflicts about 50% of all patients with colorectal cancer, and of these, about one-third have disease limited to the liver. Numerous studies have shown that resection of hepatic metastases is associated with a 25% to 45% 5-year survival rate and a 20% 10-year survival rate. As a result, operative resection has been established as the most effective therapy for patients with isolated colorectal liver metastases.

1. Staging. The purpose of preoperative evaluation is to exclude the presence of extrahepatic disease and to determine the extent of lesions in the liver that require treatment. An abdominal/pelvic CT scan with oral and intravenous contrast is performed, along with chest x-ray. There should be a colonoscopy within the last 6 months to document absence of anastomotic recurrence or a metachronous colorectal cancer. Whole-body positron emission tomography (PET) after administration of ^{18}F-fluorodeoxyglucose (FDG) is valuable for the detection of occult metastases, both intra- and extrahepatic. On MRI, hepatic metastases appear as low-intensity lesions on T1-weighted images and intermediate intensity on T2-weighted images. MRI also provides greater visualization of vascular structures such as the hepatic veins and the IVC.

2. Treatment. The main objective in the resection of colorectal metastasis is removal of all disease with gross negative margins. Formal anatomic resection has not been demonstrated to be superior to nonanatomic resection as long as R-0 resection is achieved. Resection type should be based on the number and location of tumors, rather than on segmental anatomy. In the case of synchronous liver metastasis, the primary colonic tumor and the secondary liver tumor may be resected simultaneously or sequentially. Combined resection avoids a second laparotomy and reduces the overall complication rate without changing operative mortality, but this should only be done in highly selected cases. When the colorectal and liver resections are both extensive (e.g., extended hepatic lobectomy and low anterior resection), then a staged approach is usually preferable. Factors predictive of a worse prognosis after resection of hepatic colorectal metastases include node positivity of the primary tumor, multiple metastases, disease-free interval less than 12 months, carcinoembryonic antigen (CEA) levels greater than 200 ng/mL, and size of the largest metastasis greater than 5 cm. An emphasis on the preservation of hepatic parenchyma may be of increasing importance in the setting of chemotherapy-associated steatohepatitis, and the growing number of patients undergoing repeated metastasectomy. *Postoperative followup* consists of serial physical examination, serum CEA level, and abdominal/pelvic CT scans every 3 to 4 months for the first 2 years, then every 6 months for the subsequent 3 years. Unfortunately, disease recurrence is common, but when cancer is isolated to the liver, repeat resection can provide additional survival benefit. *Local ablation with RFA*

should be considered in patients unfit for operative resection or who have unresectable disease. In those patients with multiple scattered tumors, a combined approach of resection of the dominant or larger tumors with RFA of the remaining lesions may be feasible.

B. Other Metastatic Disease to the Liver

1. GI neuroendocrine tumors. Reasons to resect neuroendocrine hepatic metastases include their relatively long tumor doubling time, a lack of effective chemotherapy, and the ability of metastasectomy to provide symptom palliation and long-term survival. For those patients with unresectable disease, hepatic artery embolization may provide symptom relief.

2. There is limited experience with liver resection for **noncolorectal and nonneuroendocrine metastasis.** Liver resection may provide the only chance for long-term survival. In a study at Memorial Sloan-Kettering Cancer Center of 96 patients with noncolorectal, nonneuroendocrine metastatic tumors of the liver, liver resection was associated with an overall actuarial survival of 37% at 5 years, with a median survival of 32 months *(Surgery.* 1997;121:625). The presence of liver metastasis from melanoma or cancer of the breast or stomach should be viewed as a marker of disseminated disease, and liver resection in these contexts is generally not recommended.

VIII. 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 occur secondary to other sources of bacterial sepsis. Up to 60% of cases arise from direct spread of bacteria from biliary infections such as empyema of the gallbladder or cholangitis. Ruptured appendicitis or diverticulitis is other potential sources for bacterial seeding to the liver. For liver abscesses arising from an intra-abdominal infection, it is important to note that the mechanism of spread of infection to the liver is along channels within the peritoneal cavity as opposed to direct hematogenous spread. Liver abscesses are usually found in the right lobe of the liver. The bacteria cultured from pyogenic liver abscesses reflect the origin of the infectious process. Most commonly, **mixed species** are isolated, with one-third of cultures containing anaerobes. When the biliary tree is the source, enteric Gram-negative bacilli and enterococci are common isolates. When the abscess develops from hematogenous seeding, there is most likely a single organism responsible, such as *Staphylococcus aureus* or *Streptococcus milleri.* Fungal abscesses have been associated with patients who are recovering from chemotherapy. There should be suspicion of amebic abscesses in patients who are from or have recently traveled to an endemic area.

1. Presentation. *Fever and abdominal pain* are the most common symptoms, whereas nonspecific symptoms such as anorexia, weight loss, chills, and malaise may also be present.

2. Diagnosis. *Laboratory findings* are usually nonspecific, such as leukocytosis and elevated serum alkaline phosphatase. A chest x-ray may demonstrate new elevation of the right hemidiaphragm, an infiltrate at the right lung base, or a right-sided pleural effusion. Definitive diagnosis is by CT scanning.

3. Treatment consists in identifying the infectious source as well as managing the liver abscess. *Pyogenic liver abscesses usually require drainage and systemic antibiotic therapy.* Drainage can be performed percutaneously in most cases, but occasionally an operative procedure is recommended when there are multiple, large, loculated abscesses and in patients who otherwise require laparotomy for the underlying cause of the abscess. Drains are usually left in place until drainage becomes minimal. Empirical antibiotic treatment should include coverage for bowel flora, and then antibiotic therapy should be modified to reflect the sensitivities of the cultured fluids. Therapy should continue for at least 1 week beyond clinical recovery and resolution of the abscess on followup imaging.

B. Amebic abscess should be considered in *every* case of solitary hepatic abscess. Amebiasis is caused by the protozoan *Entamoeba histolytica,* and liver abscess is the most common extraintestinal manifestation as the infection spreads hematogenously from the gut via the portal venous system. Amebic liver abscesses are 7 to 10 times more frequent in adult men, despite an equal sex distribution of intestinal amebic disease.

1. Presentation. Clinical symptoms are classically *persistent fever and right upper quadrant pain.* The presence of diarrhea reflecting concurrent intestinal amebiasis is more variable. Presentation usually occurs with 4 months after return from endemic areas. On examination, patients have hepatomegaly and point tenderness over the liver. Rupture of the abscess may cause peritonitis.

2. Diagnosis. Serologic tests for amebic infestation are positive in nearly 100% of affected patients. Ultrasound and CT are the most useful imaging modalities.

3. Treatment requires systemic *metronidazole* 750 mg orally three times a day or 500 mg intravenously every 6 hours, for 7 to 10 days, and has rendered operative intervention nearly obsolete. Needle aspiration should be considered if there is no response to initial therapy or if there is doubt about the diagnosis. The material aspirated contains proteinaceous debris and an Òanchovy pasteÓ fluid of necrotic hepatocytes. After completion of the course of metronidazole, the patient should be treated with an intraluminal agent, even if stools are negative for amebae. Intraluminal agents include paromomycin, iodoquinol, and diloxanide furoate. Complications can include bacterial superinfection, erosion into surrounding structures, or free rupture into the peritoneal cavity. Although mortality is infrequent in uncomplicated cases, complicated cases may carry a considerable mortality as high as 20%.

IX. HEPATIC CYSTS.

Hepatic cysts can be divided into nonparasitic and echinococcal cysts.

A. Nonparasitic cysts generally are benign. They can be solitary or multiple and often are identified incidentally on imaging for other symptoms. *Asymptomatic simple cysts* require no treatment regardless of size. Large cysts may be symptomatic because of increased abdominal

girth or compression of adjacent structures. Bleeding, infection, or obstructive jaundice can occur but are exceedingly rare. *Symptomatic simple 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, enucleated, or drained, with closure of the biliary communication. Polycystic kidney disease sometimes is accompanied by *polycystic liver disease,* which usually is asymptomatic. 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 cysts into the abdominal cavity and fenestration of deeper cysts into the superficial cyst cavities. Liver resection and retention of the least-cystic areas of hepatic parenchyma may be more effective. Rarely, liver transplantation is required for patients with marked symptomatic hepatomegaly in the setting of diffuse cysts not amenable to safe unroofing or resection. Neoplastic cystic lesions such as cystadenoma or cystadenocarcinoma 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 or enucleation (in the case of cystadenoma) to completely remove cyst epithelium.

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 liver. The most common presenting symptoms and signs are right upper quadrant abdominal pain and palpable hepatomegaly. Imaging by nuclear medicine scan, ultrasonography, CT scan, or MR scan can demonstrate the abnormality. The *cyst should not be aspirated* as an initial test because aspiration can cause spillage of the organisms and spread the disease throughout the abdominal compartment. A peripheral *eosinophilia* is often detected. Serologic tests include indirect hemagglutination and Casoni skin test, each of which is 85% sensitive. *Treatment* is primarily operative consisting of cyst aspiration, scolicidal treatment (hypertonic saline, 80% alcohol, or 0.5% cetrimide), and pericystectomy. Formal hepatectomy is rarely necessary except for large and/or multiple cysts. Scolicidal therapy with mebendazole or albendazole has been advocated to prevent recurrence. Percutaneous treatment after antihelminthic treatment is increasingly utilized for treatment with acceptable results.

X. PORTAL HYPERTENSION

A. Etiology. The most common cause of PH in the United States is *intrahepatic* obstruction of portal venous flow from *cirrhosis* most commonly from alcohol abuse or hepatitis C. Intrahepatic portal venous obstruction can also be due to hepatic fibrosis from hemochromatosis, Wilson disease, and

congenital fibrosis. *Prehepatic* portal venous obstruction due to congenital atresia or portal vein thrombosis is far less common. *Posthepatic* obstruction may occur at any level between the liver and the right heart. This includes thrombosis of the hepatic veins (BuddÑChiari syndrome), congenital IVC malformations (web, diaphragm), IVC thrombosis, congestive heart failure, and

constrictive pericarditis.

B. Diagnosis. Formal measurement of portal pressure by catheterization of the portal vein is seldom performed. Indirect evaluation by measurement of the hepatic wedge pressure after hepatic vein catheterization is considered the gold standard for monitoring PH. The hepatic venous pressure gradient (HVPG) is the difference between the wedged and free hepatic venous pressures, and *PH is considered present with HPVG 8 mm Hg or greater.* Varices do not develop until the HVPG reaches 10 to 12 mm Hg. Reduction in the HVPG below 12 mm Hg is accepted as the therapeutic target for treating PH.

C. Ascites and edema are caused by salt and water retention in the kidneys, decreased plasma oncotic pressure, and increased lymphatic flow from increased portal venous hydrostatic pressure. Although the ascites can be massive, it is rarely life-threatening unless complications occur, such as erosion or incarceration of an umbilical hernia, respiratory compromise, and spontaneous bacterial peritonitis (SBP). The diagnosis of SBP is made by paracentesis and is likely when ascitic fluid contains more than 250 polymorphonuclear leukocytes per microliter and if a single organism is cultured. The most common organisms are *Escherichia coli,* pneumococci, and streptococci. Frequently, however, it is not possible to obtain a positive culture, and so the diagnosis relies on ascitic fluid cell count and differential. Treatment is directed at reducing salt intake and retention, with diet modifications as well as use of diuretics such as spironolactone and furosemide. Paracentesis may also provide acute relief as well as diagnostic information. TIPS can be used for refractory ascites, and complete resolution of ascites has been reported in 57% to 74% of patients and partial response in another 9% to 22%. In patients with significant ascites undergoing abdominal surgery, in addition to the treatments mentioned above, an intraperitoneal drain is sometimes utilized for postoperative drainage of fluid to minimize ascites leak, prevent fascial dehiscence, promote wound healing, and reduce risk of sepsis.

D. Portosystemic shunting is caused by increased blood flow through the portal vein leading to increased flow through collateral venous beds that bypass the liver directly into the systemic circulation. The most clinically significant sites are those at the gastroesophageal junction connecting the *left gastric vein* (portal circulation) to the esophageal veins (systemic circulation). 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. Left-sided, *sinistral,* PH can be caused by isolated splenic vein thrombosis, 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. This segmental area of PH typically causes gastric varices without esophageal varices.

E. Variceal bleeding is a significant cause of morbidity and mortality in cirrhotics. *Prophylaxis* includes both the prevention of variceal hemorrhage in patients who have never bled (primary

prophylaxis) and preventing rebleeding in patients who have survived a bleeding episode (secondary prophylaxis). Every cirrhotic patient should be screened endoscopically for varices at time of diagnosis. Those without varices at this time should have endoscopy repeated after 2 to 3 years, whereas monitoring every 1 to 2 years is recommended when varices are present. *Propranolol* or nadolol therapy has been shown to markedly reduce risk of variceal bleeding, as well as slow the progression of small varices into larger ones. The dose should be titrated to the maximal tolerable dose and maintained indefinitely. For prevention of recurrent bleeding, endoscopic band ligation versus combination pharmacologic therapy (β-blocker plus isosorbide mononitrate) have equivalent results. *Transjugular intrahepatic portosystemic shunting (TIPS)* has been shown to be superior to either endoscopic or pharmacologic therapies at reducing the rate of rebleeding. However, its use does not improve mortality, has been associated with a greater risk of hepatic encephalopathy (HE), and is more costly than endoscopic procedures. Thus, it is limited to situations in which endoscopic therapy has failed or in patients who would not tolerate a rebleed such as those with Child class C cirrhosis. *Management of acute variceal hemorrhage* requires timely interventions. Up to one-third of patients with hemorrhage from gastroesophageal varices die during the initial hospitalization for GI bleeding. All patients with known or suspected esophageal varices and active GI bleeding should be admitted immediately to an 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 via short, large-bore peripheral lines should be secured. Recombinant activated factor VII (rFVIIa) may be useful for correcting the prothrombin time in cirrhotics. Infection is a strong prognostic indicator in acute variceal hemorrhage, and use of antibiotics has been shown to reduce both the risk of rebleeding and mortality. Once stabilized, the patient 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. Recommendations for specific therapy are (1) early administration of vasoactive drugs, even if active bleeding is only suspected and (2) endoscopic band ligation after initial resuscitation. The pharmacologic treatment of choice for active variceal bleeding in the United States is *octreotide* given as an initial intravenous bolus followed by infusion for 5 days. It has been shown to be more effective for controlling bleeding than placebo or vasopressin. *Endoscopic therapy* is the definitive therapy for active variceal hemorrhage. Two forms of treatment are available:

Sclerotherapy and variceal band ligation (EBL). A meta-analysis found that EBL is superior to sclerotherapy in the initial control of bleeding and is associated with fewer adverse events and improved mortality. Emergent endoscopic therapy fails to control bleeding in 10% to 20% of patients. If a second attempt at endoscopic hemostasis fails, then more definitive therapy must be enacted immediately. *Balloon tamponade* is useful as a temporary remedy for severe variceal bleeding while more definitive therapy is planned. Specially designed balloon catheters such as

the SengstakenÑBlakemore tube and the Minnesota tube have a gastric and an esophageal balloon. The position of the gastric balloon in the stomach must always be confirmed radiographically before inflation because inflation of the larger gastric balloon in the esophagus can be disastrous. The pressure of the esophageal balloon must be maintained as directed by the manufacturer to avoid the complications of mucosal ulceration and necrosis. Balloon tamponade achieves bleeding control in 60% to 90% of cases, but should be used only when there is massive bleeding and for up to 24 hours until definitive therapy is instituted. *TIPS* can be used in the acute management of patients with variceal bleeding. It involves the intrahepatic placement of a 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. The TIPS procedure can provide acute decompression of portal pressure and thus control refractory variceal bleeding. TIPS stenosis requires careful followup and revision procedures in a significant percentage of patients. Use of polytetrafluoroethylene (PTFE) stents rather than bare metal stents has dramatically decreased the rate of TIPS dysfunction, clinical relapses, and the need for interventions. *Emergency portocaval shunt* generally is reserved for patients in whom other measures have failed and is almost never performed today. This operation carries significant in-hospital mortality and risk of HE, particularly because the patients undergoing the operation typically have failed other measures and have advanced liver disease. Only the technically simpler central portocaval shunts should be used in the emergency setting because other shunts require more dissection and operative time.

CHAPTER 20: SURGICAL DISEASES OF THE LIVER

Multiple Choice Questions

1. A 50-year-old female with alcoholic cirrhosis presents to the emergency room with abdominal pain, vomiting, and CT scan showing an incarcerated loop of bowel in an umbilical hernia. She has a distended abdomen with skin changes over her umbilicus. Her WBC is 11,000 cells/mm^3, INR is 1.2, and a platelet count of 90,000/uL. She consents to operative reduction of the hernia and repair. What steps should be taken to minimize postoperative complications?

a. Medical optimization of her cirrhosis prior to operative repair

b. Transfusion of platelets preoperatively to minimize bleeding risks

c. Placement of an intraperitoneal drain after hernia repair to minimize postoperative ascites leak

d. Paracentesis prior to operative repair

e. Antibiotics and observation

View Answer

2. A 60-year-old male with hepatitis C undergoes laparotomy for small bowel perforation. Postoperatively, he has an episode of moderate hematemesis. He is normotensive, alert, and his hemoglobin level is 9.8 g/dL. After rendering him NPO and starting fluids through large-bore IVs, he is transferred to the intensive care unit. What is the next best step in management?

a. Blood transfusion to maintain hemoglobin level >10 g/dL

b. Placement of nasogastric tube, gastric lavage, and serial abdominal examinations

c. Initiating propranolol therapy

d. Endoscopy with banding of varices

e. Transjugular intrahepatic portosystemic shunt

View Answer

3. A 54-year-old woman with chronic hepatitis B infection presents to clinic with a 4-cm mass in the right lobe of her liver. She denies jaundice or history of variceal bleeding. On review of her MRI, her tumor is consistent with a solitary hepatocellular carcinoma in segment 6 without evidence of extrahepatic disease. Laboratory studies show normal liver function tests, and her alpha-fetoprotein level is elevated to 76. Her INR is 1.4 and her creatinine is 1.0 mg/dL. What is the next best step in her management?

a. Biopsy of the mass to obtain tissue diagnosis

b. Place the patient on liver transplantation list

c. Perform a formal right hepatectomy

d. Radiofrequency ablation of the lesion

e. Perform a nonanatomic resection of the mass with 2 cm margins

View Answer

4. The same patient returns to clinic 3 years later now with two hepatic lesions, each approximately 2 cm in size, located adjacent to the prior resection bed. Which of the following treatments is contraindicated?

a. Transarterial chemoembolization

b. Resection of each of the lesions

c. Radiofrequency ablation

d. Liver transplantation

e. Sorafenib therapy

View Answer

5. Which of the following liver lesions mandate operative excision?

a. An asymptomatic 6 cm hepatic adenoma

b. An asymptomatic 3 cm FNH

c. An incidentally discovered 5 cm hepatic cyst

d. An asymptomatic 4 cm hemangioma

e. Bile duct hamartoma seen intraoperatively

View Answer

21

Surgical Diseases of the Biliary Tree

Chun-Cheng (Richard) Chen

William C. Chapman

I. CHOLELITHIASIS

A. Asymptomatic Gallstones

1. Presentation. Asymptomatic gallstones are usually discovered on routine imaging studies or incidentally at laparotomy for unrelated problems.

2. Management. There is no role for prophylactic cholecystectomy in most patients with asymptomatic gallstones. Prophylactic cholecystectomy may be warranted in patients with asymptomatic gallstones who have other risk factors for gallbladder cancer as outlined later. *Children with gallstones* have a relative indication for cholecystectomy due to the general difficulty of declaring and interpreting symptoms in this population. Management of *gallstones discovered at laparotomy* remains controversial because the literature is conflicting with regard to the incidence of biliary symptoms after surgery in patients in whom the gallbladder is not removed.

B. Symptomatic Gallstones

1. Presentation. *Biliary colic* is the main symptom and is initiated by impaction of a gallstone in the outlet of the gallbladder. An attack has characteristic *periodicity, location, and timing.* The pain comes in waves lasting 30 minutes to several hours and is typically situated in the epigastrium or right upper quadrant, occasionally with concomitant back or left upper quadrant symptoms. The pain is commonly severe after a meal with such intensity as to wake the patient from sleep. Other symptoms include nausea and vomiting.

2. Diagnosis. *Physical signs* include mild right upper quadrant tenderness, although there may be few abdominal findings during an attack. If jaundice is present, another cause should be sought. Ultrasound diagnosis is based on the presence of echogenic structures having posterior acoustic shadows. There is usually little or no associated gallbladder wall thickening or other evidence of cholecystitis. The bile ducts must be assessed for evidence of dilation or choledocholithiasis.

3. Treatment. *Laparoscopic cholecystectomy (LC)* is the appropriate treatment of the vast majority of patients with symptomatic gallstones, as described below.

C. Acute calculous cholecystitis is initiated by obstruction of the cystic duct by an impacted

gallstone. Persistence of stone impaction leads to inflammation of the gallbladder.

1. Diagnosis can be made according to the *Tokyo Guidelines* by a combination of local and systemic signs of inflammation, correlated with imaging findings. Local inflammatory signs include right upper quadrant pain and tenderness as well as *Murphy sign,* which is inspiratory arrest during deep palpation of the right upper quadrant. Systemic signs include fever, leukocytosis, and an elevated C-reactive protein level. Mild jaundice may be present, but severe jaundice is rare and suggests the presence of CBD stones, cholangitis, or obstruction of the CBD caused by external compression from *Mirizzi syndrome.* Complications, such as gangrene, perforation, or cholangitis, are suggested by moderate leukocytosis (>20,000 cells/μL). Liver function tests (LFTs), including serum bilirubin, alkaline phosphatase, alanine transaminase (ALT), aspartate transaminase (AST), and serum amylase, also may be abnormal. *Ultrasonography* may reveal gallbladder wall thickening, pericholecystic fluid, and a *sonographic Murphy sign,* which is tenderness over the gallbladder when compressed by the ultrasound probe. *Computed tomographic (CT) scanning* is now frequently performed to evaluate the patient with acute abdominal pain. CT can demonstrate gallstones, although it is less sensitive for these than ultrasonography. Other signs of acute cholecystitis on CT include gallbladder wall thickening, pericholecystic fluid, edema, and emphysematous changes. *Radionuclide cholescintigraphy* can be useful as an adjunct in the diagnosis of acute cholecystitis. Scintigraphic scanning with hepatic 2,6-dimethyliminodiacetic acid (HIDA) enables visualization of the biliary system. The radionuclide is concentrated and secreted by the liver, allowing visualization of the bile ducts and the gallbladder normally within 30 minutes. Since the test depends on hepatic excretion of bile, it may not be useful in jaundiced patients. Nonfilling of the gallbladder after 4 hours is diagnostic of acute cholecystitis. Administration of morphine may enhance the test by causing spasm of the sphincter of Oddi and thereby stimulating gallbladder filling. Although its sensitivity and specificity are higher than ultrasound, its expense and total study duration limit it from being the first imaging choice.

2. Management. Initial steps for patients with acute cholecystitis include hospitalization, intravenous (IV) fluid resuscitation, and parenteral antibiotics. The *Tokyo guidelines* provide further recommendations depending on the severity of acute cholecystitis (Table 21-1). For mild acute cholecystitis, early LC is recommended. For moderate acute cholecystitis, either early or delayed cholecystectomy may be performed. In the small minority of patients with severe acute cholecystitis or with severe concomitant medical illness, *percutaneous cholecystostomy* can be performed. Subsequently, the patient can undergo either cholecystectomy or percutaneous stone extraction and removal of the cholecystostomy tube. Such nonoperative stone removal as definitive treatment is reasonable in very elderly or debilitated patients who cannot tolerate general anesthetic. *Several prospective, randomized trials* have compared early versus delayed (6 weeks) LC for acute cholecystitis. Recent meta-analyses of the existing literature showed no significant differences

in early versus delayed procedures with regard to mortality, conversion rate, bile duct injury, and perioperative complications. However, these studies almost universally showed in the early group significantly fewer readmissions for interval complications and significantly reduced hospital length of stay. Controversy still exists about the relationship between operation in the acute phase of inflammation and bile duct injury. A large registry series reported that when LC is performed for acute cholecystitis, the incidence of injury is three times higher than for elective LC and twice as high as for open cholecystectomy.

TABLE 21-1 Severity Grading for Acute Cholecystitis

Grade	Criteria
Mild (grade 1)	Acute cholecystitis that does not meet the criteria for a more severe grade
Moderate (grade 2)	The presence of one or more of the following: ¥ Leukocytosis >18,000 cells/mm^3 ¥ Palpable tender mass in the right upper quadrant ¥ Duration >72 hr ¥ Marked local inflammation including biliary peritonitis, pericholecystitis abscess, hepatic abscess, gangrenous cholecystitis, and emphysematous cholecystitis
Severe (grade 3)	Presence of any organ dysfunction (e.g., hypotension, mental status changes, respiratory failure, and acute renal failure)

From Yokoe M, Takada T, Strasberg SM, et al. TG13 diagnostic criteria and severity grading of acute cholecystitis (with videos). Modified from *J Hepatobiliary Pancreat Sci.* 2013;20(1):35-46.

D. Choledocholithiasis generally is due to gallstones that originate in the gallbladder and pass through the cystic duct into the common bile duct (CBD). Stones rarely originate in the hepatic or common ducts in Western countries, but intrahepatic stones are more common in Asia.

1. Diagnosis. The most common manifestation of uncomplicated choledocholithiasis is *jaundice,*

with bilirubin levels typically between 3 and 10 mg/dL. Cholangitis is often caused by choledocholithiasis and its management is discussed below. Biliary colic is common. Ultrasonography usually demonstrates gallbladder stones and bile duct dilation. Because of obscuring gas in the duodenum, ductal stones are visible in only about 50% of cases. The diagnosis may be confirmed by endoscopic retrograde cholangiopancreatography (ERCP) or percutaneous transhepatic cholangiography (PTC), which can opacify the biliary tree and demonstrate the intraductal stones. Occasionally, the diagnosis of choledocholithiasis is confirmed by intraoperative cholangiography (IOC) at the time of cholecystectomy.

2. Management depends on available expertise and clinical situation. Patients with low risk of choledocholithiasis have no evidence of LFT derangements, jaundice, cholangitis, pancreatitis, or imaging evidence of CBD stones. In these patients, standard management consists of LC without IOC, as long as anatomy is normal. Patients with high risk of choledocholithiasis have ultrasound evidence of CBD stone, bilirubin levels >4 mg/dL, clinical ascending cholangitis, or dilated CBD on ultrasound with a bilirubin >1.8 mg/dL. In these patients, ERCP is recommended prior to interval LC with IOC. For patients that are of intermediate risk, namely who are neither low- nor high-risk patients, management is controversial. Recent studies favor early LC with IOC, followed by laparoscopic CBD exploration if stones are seen, as opposed to ERCP. Intraoperative measures to clear the CBD of stones include administration of IV glucagon, use of irrigation, blind passage of balloon catheters or stone baskets, or passage of these devices via choledochoscope. If the bile duct cannot be cleared of stones by laparoscopic exploration, open bile duct exploration or postoperative ERCP or PTC may be required, but this is uncommon. ERCP with sphincterotomy and stone removal is used in patients who are not surgical candidates, who have had prior cholecystectomy, who are jaundiced or who have acute cholangitis. Patients with intrahepatic stones and those with many CBD stones are also usually treated with ERCP. Appropriate testing is dictated by clinical suspicion of these entities. ERCP with sphincterotomy carries a less than 1% risk of mortality and a 5% to 10% risk of morbidity, principally acute pancreatitis. Rectal indomethacin has been advocated as prophylaxis to post-ERCP pancreatitis. An intraoperative cholangiogram should be performed at the time of surgery even when preoperative ERCP has been done because residual stones may be present in a small percentage of patients.

E. Biliary pancreatitis is caused by blockage of pancreatic secretions by passage of a gallstone into the common biliary pancreatic channel. The greatest risk is carried by small (÷2 mm) stones. The management of severe pancreatitis is discussed elsewhere in this manual. Once the acute episode of pancreatitis has resolved, the gallbladder should be removed during the same admission to avoid recurrent pancreatitis. A longer delay may be justified in patients who have had severe pancreatitis and in whom local inflammation or systemic illness contraindicates surgery. An IOC should *always* be done at the time of the cholecystectomy to confirm that the bile duct is free of stones. In patients in whom cholecystectomy is contraindicated, endoscopic sphincterotomy (ES) may be protective against further attacks of pancreatitis.

F. Gallstone ileus is a small bowel obstruction caused by a gallstone, an uncommon complication resulting from a gallstone eroding through the gallbladder into the adjacent bowel (usually duodenum). The stone migrates until it lodges in the narrowest portion of the small bowel, just proximal to the ileocecal valve. Patients present with symptoms of bowel obstruction and ascending cholangitis from the cholecystoenteric fistula.

Treatment is exploratory laparotomy and removal of the obstructing gallstone by milking it back to an enterotomy made in healthy intestine. The entire bowel should be searched diligently for other stones, and cholecystectomy with closure of the fistula should be performed if the patient is stable and the inflammation is not too severe.

G. Laparoscopic Cholecystectomy

1. Indications. LC has a low complication rate and available for 95% of patients, with excellent recovery and return-to-work times. Contraindications include generalized peritonitis, cholangitis, concomitant diseases that prevent use of a general anesthetic, and the patient's refusal of open cholecystectomy should urgent conversion be required.

2. Technique. Because misidentification of the cystic duct is the commonest cause of biliary injury, the surgeon must use a technique to provide conclusive identification of the cystic duct and artery. In the *Critical View of Safety Technique* pioneered at our institution, the triangle of Calot is dissected free of fat, fibrous, and areolar tissue. Importantly, the lower end of the gallbladder must be dissected off the liver bed. A complete dissection demonstrates *only* two structures *(i.e.,* the cystic duct and artery) entering the gallbladder, constituting the Òcritical view of safety.Ó *IOC* may be used to assist with anatomical definition, especially whenever the critical view is not achieved. IOC is also indicated in patients with recent choledocholithiasis, jaundice, pancreatitis, a large cystic duct and small gallstones, any abnormality in preoperative LFTs, or dilated biliary ducts on ultrasonography. *Laparoscopic ultrasound* is an alternative method for the detection of CBD stones that is highly accurate and has decreased operative time and cost in experienced hands.

3. Complications. LC appears to be associated with a higher incidence (÷2.5/1,000) of major bile duct injury than open cholecystectomy. This serious problem is further discussed below. In addition, there are also risks to other structures, including the hepatic artery and the bowel. Spilled and retained gallstones can be the source of infrequent, but serious long-term complications such as abscess and fistula formation. Factors associated with an increased rate of conversion to an open procedure include emergent cholecystectomy, male sex, age greater than 60 years, obesity, severe gallbladder inflammation, choledocholithiasis, and prior upper abdominal surgery.

H. Open cholecystectomy is performed in patients who have contraindications to LC, who require conversion from LC because of inability to complete the laparoscopic procedure, or who are undergoing laparotomy for another operation (e.g., pancreaticoduodenectomy). Conversion to

an open operation in the face of a difficult laparoscopic procedure should never be viewed as a surgical failure or complication but rather as a way to avoid potential injury to the patient. A *partial cholecystectomy* is advocated when the ductal and vascular structures in the triangle of Calot cannot be safely identified in the setting of severe acute inflammation. In this situation, the fundus is opened and stones are extracted. The anterior wall of

the gallbladder is excised, leaving the posterior wall with the liver bed. The mucosa is then fulgurated, and a drain is left near the infundibulum.

II. ACALCULOUS CHOLECYSTITIS

A. Presentation. *Acalculous cholecystitis* typically occurs in severely ill hospitalized patients, especially those with a history of hypotension. It is also associated with prolonged nothing-by-mouth (NPO) status, dependence on parenteral nutrition, episodes of systemic sepsis, or multiorgan system failure. Mortality rate is at around 30%. Presentation depends largely on the patient's concurrent medical conditions and often is suspected during an infectious workup. Alert patients can complain of right upper quadrant or diffuse upper abdominal pain and tenderness. However, because many of these patients may not be alert, pain and tenderness are absent in up to 75% of patients.

B. Diagnosis. In sedated patients, *leukocytosis and abnormal LFTs,* although variable, may be the only indications of acalculous cholecystitis. Imaging is essential for establishing the diagnosis because a false-positive result may lead to an unnecessary intervention in a critically ill patient. The same imaging modalities used to diagnose calculous cholecystitis may be used to diagnose acalculous cholecystitis. Because of its portability and low cost, ultrasound is almost universally the first test of choice, but if the diagnosis is in doubt, then scintigraphy can be added significantly to improve the diagnostic index. In difficult cases, *percutaneous cholecystostomy* may be both diagnostic and therapeutic because an infected gallbladder can be decompressed and inciting stones extracted via the tube. CT can be used to evaluate other potential sources of abdominal pathology, whereas percutaneous cholecystostomy may avoid a trip to the operating room for patients who are unable to tolerate surgery.

C. Management of acalculous cholecystitis involves systemic antibiotics, NPO status, and treatment of any comorbidities. Primary treatment involves decompression of the gallbladder, typically with a percutaneously placed tube. The definitive treatment is interval cholecystectomy.

III. BENIGN STRICTURES AND BILE DUCT INJURIES.

They occur in association with a number of conditions, including pancreatitis, choledocholithiasis, primary sclerosing cholangitis (PSC), prior hepatic transplantation, trauma, or iatrogenic injury after instrumentation or surgery. LC is the leading cause of iatrogenic bile duct injuries and subsequent benign strictures. Biliary malignancy may masquerade as a benign stricture. Attempts to differentiate between the two etiologies should be made prior to surgery

because the patient may not be a candidate for a curative resection if an advanced stage of cancer is found.

A. Risk factors for intraoperative bile duct injury include *patient-related factors* such as acute and chronic inflammation, presence of a large impacted stone, and congenital anomalies. These factors cause the structures in the triangle of Calot to be difficult to be identified. *Technical factors* include such issues as inadvertent injury to the bile ducts with electrocautery, failure

to develop the critical view of safety as described above, failure to close the cystic stump adequately, lack of operator experience, a sense of infallibility during the operation, and inadequate maintenance of laparoscopic instruments.

B. Classification. A widely accepted classification scheme has been developed at this institution. 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 a transected duct. Type D injuries are *lateral* injuries to the extrahepatic bile ducts. Type E injuries (subtypes 1 to 5) are derived from the Bismuth classification and represent transections or occlusions at various levels of the CBD.

C. Presentation depends on the type of injury. Approximately 25% of major bile duct injuries are recognized at the time of the initial procedure. Intraoperative signs of a major ductal injury include unexpected bile leakage, abnormal IOC, and delayed recognition of the anatomy after transection of important structures. If an injury is not recognized intraoperatively, the patient usually presents with symptoms within 1 week and almost always within 3 to 4 weeks after the initial procedure. Patients with a bile leak often present with right upper quadrant pain, fever, and sepsis secondary to biloma and may have bile drainage from a surgical incision. Patients with occlusion of the CBD without a bile leak present with jaundice. Occasionally, a delayed presentation of months or years is seen.

D. Diagnosis. Axial imaging with CT scan or MRI is useful for detecting abdominal bile collections that require percutaneous drainage. MRCP with angiography is now often the initial imaging test of choice because of its ability to define the biliary anatomy as well as any associated vascular injuries. Ongoing bile leaks can also be diagnosed by HIDA scan. ERCP is for diagnostic and therapeutic purposes, such as biliary stent deployment. In the case of occlusion of the CBD, PTC can demonstrate the biliary anatomy distal to the occlusion and be used for decompression of the biliary tree.

E. Management depends on the presentation. If the injury is identified at the time of the initial procedure, the surgeon should proceed directly to open exploration and repair only if trained with complex techniques in hepatobiliary surgery or to control life-threatening hemorrhage. Otherwise, the patient should be resuscitated, a drain should be placed in the right upper quadrant, and an immediate referral must be placed to a hepatobiliary specialist. Some simpler injuries can be successfully managed with ERCP and sphincterotomy and stenting. Occlusive lesions, usually with

clip occlusion, require decompression of the proximal system via PTC. Ideally, if a reparable injury is identified early, the procedure should be done within the first few days after the initial operation when inflammation is at a minimum. In delayed diagnosis, temporization for at least 8 weeks can allow the acute inflammation to resolve. In addition, if there is a concern about a concomitant vascular injury, definitive repair should be delayed to identify areas of

ductal ischemia more easily, which should not be incorporated in the repair. Control of sepsis, percutaneous drainage, and adequate nutrition should be optimized before definitive repair.

F. Operative repair is best achieved by means of a Roux-en-Y hepaticojejunostomy after debridement of the bile duct to viable tissue. All bile ducts must be accounted for, and an adequate blood supply must be apparent for each. A tension-free mucosa-to-mucosa anastomosis constructed with fine absorbable suture is desired. Excellent long-term outcomes have been described, with anastomotic stricture the most common, yet infrequent, complication.

IV. ACUTE CHOLANGITIS

A. Presentation. *Acute cholangitis* is a potentially life-threatening bacterial infection of the biliary tree typically associated with obstruction of the ductal system. Although acute cholangitis is often associated with choledocholithiasis, other causes include benign and malignant strictures of the bile ducts or at biliary-enteric anastomoses, parasites, and indwelling tubes or stents, but almost always this occurs with incomplete biliary decompression (e.g., stent occlusion). ERCP without concomitant stenting in the presence of a stricture may lead to cholangitis above the stricture. Therefore, patients should routinely be pretreated with antibiotics in case a stent cannot be placed.

B. Diagnosis. Patients present with a spectrum of disease severity, ranging from subclinical illness to acute toxic cholangitis. Fever is present in >90% of patients. *Charcot triad* (fever, jaundice, and right upper quadrant pain) is present in only 50% to 70% of patients, and *Reynold pentad* (Charcot triad with hemodynamic instability and mental status changes) in less than 10% of patients, mostly in the elderly and those with a septic course. *Laboratory data* may demonstrate leukocytosis and abnormalities in LFTs. Ultrasonography or CT scan can reveal gallstones and biliary dilatation, but *definitive diagnosis is made by ERCP or PTC.* These studies are both diagnostic and therapeutic because they demonstrate the level of obstruction and allow culture of bile, removal of stones or indwelling foreign bodies, and placement of drainage catheters if necessary.

C. Treatment. Initial management of cholangitis includes *IV antibiotics* appropriate for the coverage of the most common Gram-negative aerobic and anaerobic organisms. In patients with acute toxic cholangitis or in patients who fail to respond to antibiotic therapy, *emergent decompression of the biliary tree* via ERCP or PTC is required. If these means are not available, operative intervention to decompress the biliary tree is indicated, though it should usually be limited to extraction of obvious stones and insertion of a T-tube in the CBD. Cholangitis in

patients with indwelling tubes or stents generally requires stent removal and replacement. Definitive operative therapy for benign or malignant biliary tract strictures should be deferred until a later date.

V. BILIARY DYSKINESIA.

Biliary dyskinesia is seen in patients with *typical* symptoms of biliary colic but without evidence of gallstones. These patients require extensive workup to exclude other causes of right upper quadrant pain. In a cholecystokininÑtechnetiumÑHIDA scan the gallbladder fills with the labeled radionuclide, and a gallbladder ejection fraction is calculated 20 minutes after cholecystokinin injection. An ejection fraction of less than 35% is suggestive of biliary dyskinesia. The definitive treatment is cholecystectomy, and greater than 85% of patients report postoperative improvement or relief of symptoms.

VI. PRIMARY SCLEROSING CHOLANGITIS

A. Presentation. PSC is an autoimmune cholestatic disorder characterized by a progressive fibrous stricturing in the bile ducts. PSC is present in 1% to 5% of those with inflammatory bowel disease (IBD), and approximately 75% of patients with PSC have or will ultimately develop IBD. PSC is a risk factor for cholangiocarcinoma, which may occur in approximately 10% to 20% of patients. Prolonged disease ultimately leads to progressive hepatic failure. The condition is characterized by relapses and remissions, with quiescent periods. Jaundice with pale, acholic 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 may ultimately occur. Many patients have a course that progresses to cirrhosis and liver failure despite early palliative interventions. Overall, the median length of survival from diagnosis to death or liver transplantation is 10 to 12 years.

B. Diagnosis. Physical examination commonly reveals jaundice and hepatosplenomegaly. Alkaline phosphatase level is almost always elevated, usually out of proportion to the bilirubin. Serum transaminases may be mildly elevated. Screening for hepatitis viruses is negative. Perinuclear antineutrophil cytoplasmic antibodies (pANCA) are present in the serum of 80% of patients who have PSC, and are highly suggestive but not specific. The procedure of choice is ERCP; PTC may be complementary if the intrahepatic biliary tree is not well visualized. The commonest finding is diffuse and irregular narrowing of the entire biliary tree, with short, annular strictures giving a beaded appearance. In progressive disease, the strictures cause diverticula of the ducts to appear. Although cholangiography is the gold standard, sonography and tomographic imaging including CT scan, magnetic resonance imaging (MRI), and magnetic resonance cholangiopancreatography (MRCP) also are useful in making the diagnosis.

C. Treatment. Symptomatic improvements have been reported with the use of various drugs aimed at reversing the presumed autoimmune etiology, including corticosteroids, azathioprine,

cyclosporine, and methotrexate. However, none of these alters the natural history of the disease. Because of risk for cholangiocarcinoma, close surveillance of patients is needed. The diagnosis is difficult because cholangiocarcinomas also masquerade as

strictures. A dominant biliary stricture or elevated tumor markers (e.g., CA 19-9) should raise the suspicion of cholangiocarcinoma in a PSC patient. PSC has been effectively palliated with endoscopic or percutaneous dilation of strictures. In cases where cholangiocarcinoma is not suspected, the role of resection is limited to situations when the disease is located around the extrahepatic bile ducts that can be excised or bypassed by hepaticojejunostomy (i.e., a dominant extrahepatic duct stricture with relatively spared intrahepatic ducts). Extensive, diffuse stricture disease with endstage cirrhosis is an indication for orthotopic liver transplantation (OLT). OLT likely improves survival and quality of life, and early referral for liver transplantation is indicated to decrease the risk of developing cholangiocarcinoma, but does not prevent the recurrence of PSC. If the patient has undergone a previous decompressive operation, transplantation is technically more challenging but not contraindicated.

VII. CHOLEDOCHAL CYSTS.

Choledochal cysts are congenital dilations of the biliary tree that may occur in any bile duct but characteristically involve the common hepatic and CBDs. Diagnosis and treatment are essential because the cysts predispose to choledocholithiasis, cholangitis, portal hypertension, and cholangiocarcinoma, which develop in up to 30% of cysts. *An anatomic classification scheme* has identified five distinct types. Type I cysts are fusiform dilations of the CBD and are the most common (65% to 90%). Type II cysts are rare, isolated saccular diverticula of the CBD. Type III cysts, also termed *choledochoceles,* are localized dilations within the intraduodenal part of the CBD. Most lesions thought to be choledochoceles are in fact duodenal duplications. Type IV cysts are characterized by multiple cystic areas of the intrahepatic and extrahepatic biliary tract. Type V cysts are single or multiple lesions based only in the intrahepatic portion of the tract (Caroli disease).

A. Diagnosis. The classic triad of jaundice, a palpable abdominal mass, and right upper quadrant pain mimicking biliary colic is present only a minority of the time. Neonates frequently present with biliary obstruction, whereas older children suffer from jaundice and abdominal pain. Rarely, there is pancreatitis or duodenal obstruction. Initial diagnosis is often made with ultrasonography and/or CT. Further evaluation of the cyst should be obtained with specific biliary imaging such as ERCP or MRCP.

B. Treatment is primarily surgical. Cyst excision with a Roux-en-Y hepaticojejunostomy is the treatment of choice for types I and IV. Simple excision of the rare type II cyst has been performed. Local endoscopic cyst unroofing plus sphincteroplasty is usually effective for type III disease. Caroli disease can be treated with hemihepatectomy when it is confined to one side of the liver. More often, bilateral disease is present and mandates OLT.

VIII. BENIGN BILE DUCT TUMORS.

Benign bile duct 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 CBD being the next most common site. The malignant

potential of these uncommon lesions is unclear. Most patients present with intermittent obstructive jaundice, often accompanied by right upper quadrant pain. Treatment should involve complete resection of the tumor with a margin of duct wall. Lesions situated at the ampulla can usually be managed by transduodenal papillotomy or wide local excision, but cases concerning for malignancy usually require Whipple resection.

IX. CHOLANGIOCARCINOMA

A. Presentation. Jaundice, followed by weight loss and pain, is the most frequently encountered clinical feature at presentation. Cholangiocarcinomas arise from the bile duct epithelium and can occur anywhere along the course of the biliary tree. Tumors tend to be locally invasive, and when they metastasize, they usually involve regional lymph nodes, the liver, and the peritoneum. They characteristically spread along the bile ducts microscopically for long distances beyond the palpable end of the tumor. Predisposing conditions include male gender, PSC, choledochal cysts, intrahepatic stones, and parasitic infestations such as *Clonorchis* species. Cholangiocarcinoma has been classified according to its anatomic location: Intrahepatic (20%), extrahepatic upper duct (also called *hilar* or *Klatskin tumor,* 40%), and extrahepatic lower duct (40%). The specific anatomic location and growth pattern of perihilar tumors are further described by the Bismuth classification scheme (Table 21-2).

B. Diagnosis. *Carbohydrate antigen 19-9 (CA19-9)* is the most commonly used marker in the diagnosis of cholangiocarcinoma. Sensitivity and specificity vary depending on the threshold used and coexisting conditions such as inflammation and cholestasis, which may elevate the levels. *Carcinoembryonic antigen (CEA)* is another tumor marker, most commonly measured in colorectal cancer. CEA has demonstrated some elevation in patients with malignancies of biliary origin. *MRCP* can be used for investigation of cholangiocarcinoma, giving cholangiography, the relationship of the lesion to key vessels, and intrahepatic metastases. *Ultrasonography can* demonstrate bile duct masses and dilation and provide rudimentary

information on the extent of tumor involvement within the liver and predicting portal vein involvement. *CT* may be helpful when MRI is contraindicated or cannot be tolerated. *ERCP* is the most valuable diagnostic tool for cholangiography of lower duct tumors. Distal lesions may be indistinguishable from small pancreatic carcinomas 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. ERCP is increasingly being used for preoperative therapeutic decompression of the biliary tree, which has the

advantage of improving liver function prior to resection but has the risk of cholangitis and increased postoperative infection. Only the less-affected hemiliver should be decompressed so that it will hypertrophy while the undrained side atrophies. The side to be resected should be drained only if cholangitis is present or with significant bilirubin elevation (>10 mg/dL). ERCP carries the potential added benefit of obtaining cellular material for cytologic analysis, either via ductal brushing, fine needle aspiration (FNA), or forceps biopsy. If a tumor is resectable, however, there is no role for tissue diagnosis before resection. *PTC* has been used when ERCP and MRI cannot precisely delineate the upper limit of a tumor. *Endoscopic ultrasound (EUS)* with FNA represents an important development in the investigation of *lower* bile duct strictures and masses. It is useful as an alternative to ERCP for obtaining tissue to establish a cytologic diagnosis, especially if a primary pancreatic mass is suspected. The potential value of such a biopsy needs to be weighed against risks such as bleeding and potentially seeding the traversed peritoneal cavity with malignant cells. Transperitoneal biopsy should not be undertaken if the patient is a potential liver transplant candidate (see below) since it will preclude OLT at some centers.

TABLE 21-2 Bismuth-Corlette Classification of Hilar Cholangiocarcinoma

Type I	Tumor remains below the confluence of the right and left hepatic ducts
Type II	Tumor involves the confluence of the right and left hepatic ducts
Type III	Tumor involves *either* the right *or* the left hepatic duct and extends to secondary radicals
Type IV	Tumor involves secondary radicals of *both* the right *and* left hepatic ducts

C. Treatment. Resection remains the primary treatment of cholangiocarcinoma, although only 15% to 20% are resectable at presentation. *Intrahepatic tumors* are best treated with hepatic resection, similar to any other mass lesion in the liver. Resectability is assessed as for other types of intrahepatic tumors, with a goal of 1-cm tumor-free margins and maintenance of an adequate future liver remnant (FLR, discussed in section on liver diseases). If there is risk of insufficient FLR, preoperative portal vein embolization can be used to atrophy the affected hemiliver and

induce hypertrophy of the unaffected liver segments. For *extrahepatic upper duct (hilar) tumors,* resection of hilar tumors includes the bile duct bifurcation and the caudate lobe; ipsilateral hemihepatectomy is often required to obtain an R0 resection. Biliary reconstruction is performed as a Roux-en-Y hepaticojejunostomy. Pancreaticoduodenectomy may be necessary in some cases to obtain negative lower margins of the CBD. Vascular involvement is not an absolute contraindication to resection because portal venous resection and reconstruction may be possible. Contraindications to resection include bilateral intrahepatic ductal spread, extensive involvement of the main trunk of the portal vein, bilateral involvement of hepatic arterial and/or portal venous branches, a combination of vascular involvement with evidence of contralateral

ductal spread, and significant lymph node involvement or distant spread. Orthotopic liver transplant with neoadjuvant chemoradiation is currently considered for carefully selected patients in selected US centers for unresectable perihilar cholangiocarcinomas with excellent results in this group. Tumors in the *middle of the extrahepatic bile duct* may be approached with an excision of the supraduodenal extrahepatic bile duct, cholecystectomy, and portal lymphadenectomy. However, most malignant strictures in the mid-CBD are due to local invasion of a gallbladder cancer rather than cholangiocarcinoma. Considerations for extrahepatic lower duct tumors are the same as for carcinoma of the head of the pancreas. Approximately 80% of lower duct tumors are resectable by pancreaticoduodenectomy. 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.

D. Palliation for patients with unresectable disease involves surgical, radiologic, or endoscopic biliary decompression. Biliary decompression via endoscopic or percutaneous internal stenting is often the first choice. When encountered at laparotomy, internal biliary drainage is best achieved by choledochojejunostomy for lower duct lesions.

X. GALLBLADDER CANCER

A. Presentation. Gallbladder cancer is the most common malignancy of the biliary tract. It is more aggressive than cholangiocarcinoma and has a poor prognosis with median survival of 5 to 8 months. There is a strong correlation with gallstones (95%). Histologically, nearly all gallbladder cancers are adenocarcinomas, and concomitant cholecystitis is frequently present. Tumors spread primarily by direct extension into liver segments IV and V adjacent to the gallbladder fossa but also via lymphatics along the cystic duct to the CBD. Because of its generally advanced stage at presentation, only a small percentage of patients with a preoperative diagnosis of gallbladder cancer are resectable for potential cure. Polyps 1.5 cm or greater in diameter have a 46% to 70% prevalence of cancer, whereas in those smaller than 1 cm, the risk of malignancy is <5%. Malignant polyps also tend to be sessile in nature and echogenic on ultrasound. Prophylactic cholecystectomy should be considered for polyps >1 cm in size or meeting morphologic criteria. Other risk factors include porcelain gallbladder, PSC, and anomalous junction of the pancreatobiliary duct.

B. Diagnosis. Approximately one-third of these tumors are diagnosed incidentally during cholecystectomy, *found in 0.3% to 1%* of all cholecystectomy specimens. Symptoms of stage I and II gallbladder cancer are often directly caused by gallstones rather than the cancer, whereas stage III and IV cancers present with weight loss and symptoms typical of CBD obstruction. Suggestive ultrasound findings include thickening or irregularity of the gallbladder, a polypoid mass, or diffuse wall calcification indicative of porcelain gallbladder.

C. Treatment. Mucosal disease confined to the gallbladder wall (Tis and T1a tumors) is often identified after routine LC. Because the overall 5-year survival rate is as high as 80%, *cholecystectomy alone with negative resection margins,* including the cystic duct margin, is adequate therapy. Patients with a preoperative suspicion of gallbladder cancer should undergo open cholecystectomy because port site recurrences and late peritoneal metastases (associated with bile spillage) have been reported even with in situ disease. T2 tumors have invaded the muscularis and may be treated by radical cholecystectomy that includes the gallbladder, the gallbladder bed of the liver, as well as the hepatoduodenal ligament, paraduodenal, peripancreatic, hepatic artery, and celiac lymph nodes. Presence of lymph node metastases or extension of disease beyond the gallbladder wall into local organs (T3Ñ T4 disease) 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. Improvement in survival has been demonstrated after radical resection. Because of the aggressive nature of this malignancy, adjuvant chemoradiation is often recommended, but little proof of efficacy is available. Most gallbladder cancers have invaded adjacent organs, extended into the porta hepatis, or distantly metastasized before clinical diagnosis. Extensive liver involvement or discontiguous metastases preclude surgical resection. Jaundice may be palliated by percutaneous or endoscopically placed biliary stents. Duodenal obstruction can be surgically bypassed if present.

CHAPTER 21: SURGICAL DISEASES OF THE BILIARY TREE

Multiple Choice Questions

1. A 70-year-old male with no significant medical problems presents to the emergency room at midnight with right subcostal pain, nausea, and vomiting for the last 48 hours. He is afebrile and normotensive, but tender to palpation in the right upper quadrant of the abdomen without peritonitis. His WBC is 15, and he has no other laboratory abnormalities. A CT of the abdomen shows a distended gallbladder with minimal fat stranding around it and no other significant findings. What is the next best step?

a. Order an abdominal sonogram to confirm acute cholecystitis

b. Order a HIDA scan to confirm acute cholecystitis

c. Initiate antibiotics that cover the usual gut flora and start intravenous fluids

d. Take the patient emergently to the operating room for laparoscopic cholecystectomy

e. Consult interventional radiology for a percutaneous cholecystostomy tube

View Answer

2. An inpatient consultation was placed for a 30-year-old obese woman who recently gave birth via C-section presents to the hospital with acute abdominal pain, nausea, and vomiting. She is nontoxic at presentation with tenderness to her epigastrium and has laboratory data showing no leukocytosis, a bilirubin of 1.5 mg/dL, mild elevations of amylase and lipase, and an abdominal ultrasound showing numerous small gallstones without gallbladder wall thickening, pericholecystic fluid, CBD stone, or dilation. After pain control, initiating NPO status, and fluid resuscitation, what is the next best step?

a. Consultation to gastroenterology for an ERCP to evaluate the common bile duct and perform a sphincterotomy

b. Obtain an MRCP to evaluate the biliary tree

c. Schedule the patient for open cholecystectomy after resolution of biochemical evidence of jaundice and pancreatitis

d. Schedule the patient for laparoscopic cholecystectomy after resolution of abdominal pain with a diet

e. Discharge the patient after resolution of symptoms and laboratory abnormalities

View Answer

3. A 60-year-old male with coronary artery disease and congestive heart failure with an ejection fraction of 25% is in the medical intensive care unit recovering from an ST-elevated myocardial infarction complicated by hospital-acquired pneumonia. Recently, the patient became febrile with respiratory distress requiring mechanical ventilation. He has a leukocytosis of 25,000 cells/mm^3, a bilirubin level of 11 mg/dL, and an abdominal ultrasound showing a dilated

gallbladder with no stones and no CBD dilation, but also dilated intrahepatic ducts. After initiation with antibiotics and placement of a percutaneous cholecystostomy tube, what is the next best step in managing this patient?

a. Perform a percutaneous transhepatic cholangiogram to decompress the biliary tree

b. Perform a laparoscopic cholecystectomy with possible cholangiography

c. Ask the gastroenterology consultants for endoscopic ultrasound with brushings of the biliary tract

d. Obtain an MRCP to delineate the biliary anatomy and level of obstruction

e. Obtain CEA, CA19-9 and AFP levels

View Answer

4. During workup for symptomatic cholelithiasis in a 50-year-old male, an ultrasound showed an incidental mass in the gallbladder. After laparoscopic cholecystectomy, pathology reports adenocarcinoma that resides in the lamina propria. Which of the following is the best management for this patient?

a. Counseling for extended resection of the gallbladder fossa as well as periportal lymph node dissection

b. Serial annual ultrasound examination for 5 years

c. MRCP to evaluate the biliary system for additional pathology

d. Obtain serial CA19-9 and CEA levels

e. Examination of the cystic duct margin, and if negative, no further intervention is required

View Answer

5. A surgeon at a community hospital reports that during laparoscopic cholecystectomy for acute cholecystitis he thinks the common bile duct was transected. The procedure is not yet completed and he calls a hepatobiliary surgeon asking from the operating room for advice regarding further management. What should be the next recommendation?

a. Place a percutaneous drain near the dissection, close the incisions, and transfer the patient to the specialist

b. Perform an intraoperative cholangiogram to evaluate the level of injury

c. Complete the cholecystectomy and schedule the patient to be

evaluated by the hepatobiliary surgeon in clinic

d. Convert to an open procedure and attempt a choledochojejunostomy

e. Convert to an open procedure and attempt a primary repair of the injury

View Answer

22

Surgical Diseases of the Pancreas

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The pancreas has four sections moving from right to left: The head/uncinate, neck, body, and tail. It is a **retroperitoneal** organ, lying obliquely across the upper abdomen with the tail higher than the head. The head or proximal pancreas is cradled by the C loop of the duodenum. The neck, lies anterior to the mesenteric vessels and portal vein. The body, which is generally accepted to begin at the left border of the superior mesenteric vein (SMV), lies posterior to the stomach and anterior to the splenic vein. The distal pancreas ends in the tail which sits close to the splenic hilum, anterior to the left adrenal gland. The pancreas receives its blood supply from both the celiac trunk and the superior mesenteric artery (SMA). The arterial supply of the pancreatic head is provided by the superior pancreaticoduodenal arteries (from the gastroduodenal artery) and the inferior pancreaticoduodenal arteries (from the SMA). The distal pancreas receives its arterial supply from branches of the splenic artery. Venous drainage is primarily by the pancreaticoduodenal and splenic veins, which drain into the portal vein. The pancreas forms from ventral and dorsal outpouchings of the duodenum, each with its own duct entering the duodenum. In the commonest final pattern, the ventral duct (Wirsung) joins with the dorsal duct at the neck of the pancreas with the dorsal duct in the head regressing. Two completely separate ductal systems occasionally persist (pancreas divisum).

I. ACUTE PANCREATITIS.

Acute pancreatitis is an inflammatory illness of variable severity. Approximately 80% of cases are **interstitial edematous acute pancreatitis** characterized by acute inflammation of the pancreatic parenchyma and peripancreatic tissues, which usually is self-limited and associated with ÒmildÓ transitory clinical manifestations. By contrast, 20% of patients develop **necrotizing acute pancreatitis** characterized by inflammation and pancreatic parenchymal necrosis, which is associated with a much higher morbidity and a substantial mortality rate *(Gut.* 2013;62:102). Occasionally, inflammation and necrosis are accompanied by pancreatic parenchymal hemorrhage **(acute hemorrhagic pancreatitis).** The exact mechanism by which various factors induce acute pancreatitis is unclear. However, it seems that the initial insult is **unregulated activation of trypsin within pancreatic acinar cells,** leading to autodigestion and an inflammatory cascade that may progress to SIRS *(Lancet.* 2008;371(9607):143-152). In severe cases, autodigestion extends beyond the pancreas into the retroperitoneum digesting peripancreatic tissues, causing fat necrosis and erosion of blood vessels with hemorrhage. Entry of enzymes into

the blood stream may cause respiratory and renal injury and other effects.

A. Etiology. The two most common causes of acute pancreatitis in the United States are **gallstones** and **alcoholism,** collectively accounting for nearly

80% of cases. Endoscopic retrograde cholangiopancreatography (ERCP) accounts for another 2% to 5% of cases. Other causes include **metabolic** (hypercalcemia and hypertriglyceridemia), **drugs** (azathioprine, sulfamethoxazoleÑtrimethoprim, furosemide, opiates, and valproic acid), **toxins** (scorpion stings and organophosphates), **infections** (mumps, Coxsackie virus B, EpsteinÑ Barr virus, cytomegalovirus, rubella, and *Ascaris* species), **neoplasms** (benign and malignant), **trauma, autoimmune** (Sjšgren syndrome, systemic lupus erythematosus [SLE], primary biliary cirrhosis [PBC], and autoimmune pancreatitis), and **idiopathic.**

B. Diagnosis

1. Patients typically present with **epigastric pain,** often radiating to the back. Occasionally, irritation from intraperitoneal pancreatic enzymes results in impressive peritoneal signs, simulating other causes of an acute abdomen. Nausea, vomiting, and low-grade fever are common, as are tachycardia and hypotension secondary to hypovolemia. Hypoxemia, renal failure, hypocalcemia, hyperglycemia and respiratory failure are evidence of severe systemic effects. Flank ecchymosis **(Gray-Turner sign)** or periumbilical ecchymosis **(Cullen sign)** are due to tracking of retroperitoneal hemorrhage and are always a manifestation of severe pancreatitis. However, these signs are present in only 1% to 3% of cases and do not usually develop until 48 hours after the onset of symptoms.

2. Laboratory studies

a. Serum amylase levels rise within a few hours of the onset of symptoms and may return to normal over the following 3 to 5 days. Persistent elevations of levels for longer than 10 days indicate complications, such as pseudocyst formation. However, there is no correlation between amylase level and severity. In addition, hyperamylasemia can be found in a variety of other clinical conditions including renal failure, intestinal obstruction, sialadenitis, and malignancy.

b. Serum lipase generally is considered more sensitive for pancreatic disease (95%), and remains elevated for a longer period of time, which can be useful in patients with a delayed presentation.

c. Acute phase proteins such as C-reactive protein (CRP), tumor necrosis factor-alpha (TNF-a), and interleukin-6 (IL-6) may be measured as a marker of severity.

d. Serum calcium levels may fall as a result of complexing with fatty acids (saponification or fat necrosis) produced by activated lipases as well as from hypoalbuminemia.

e. Hepatic function panel (aspartate aminotransferase [AST], alanine aminotransferase [ALT], bilirubin, alkaline phosphatase) should be checked to assess for concomitant biliary disease or as

an etiology of pancreatitis (gallstone disease) although normal values do not rule out biliary etiologies.

3. Radiologic imaging complements clinical history and examination because no single modality provides a perfect diagnostic index of severity.

a. Ultrasonography (US) is quite user dependant, and the pancreas is not visualized in up to 40% of patients due to overlying bowel gas

and body habitus. The primary utility of US in acute pancreatitis is to evaluate for biliary etiology.

b. Computed tomography (CT) has a sensitivity and specificity of 90% and 100%, respectively and is the gold standard for the disease. Iodinated contrast enhancement is essential to detect the presence of pancreatic necrosis. CT findings include parenchymal enlargement and edema, necrosis, blurring of fat planes, peripancreatic fluid collections, bowel distention, and mesenteric edema. Pleural effusion and atelectasis especially on the left side are also common. CT imaging may be useful in predicting the severity and course of disease using the modified CT severity index (CTSI, see below, *Am J Roent.* 2004;183:1261-1265). In general, CT imaging is indicated in patients in whom the diagnosis is not highly probable, in severely ill patients in whom necrosis is more likely, and in any patient who exhibits clinical deterioration or fails to improve with medical management.

c. Magnetic resonance imaging (MRI) is a useful substitute for CT scan in patients allergic to iodinated contrast or in acute renal failure with sensitivity 83% and specificity 91%. In addition, MRI/MR cholangiopancreatography (MRCP) is better than CT at visualizing cholelithiasis, choledocholithiasis, and anomalies of the pancreatic and common bile ducts.

4. ERCP is not routinely indicated for the evaluation of patients during an attack of acute pancreatitis, and is a subject of some controversy.

Indications for ERCP are as follows:

a. Patients with **jaundice, suspected biliary pancreatitis, and possible cholangitis** who are not clinically improving by 24 hours after admission should undergo endoscopic sphincterotomy and stone extraction. However, the literature is clear that routine, early endoscopic intervention for gallstone pancreatitis does not beneficially influence morbidity or mortality *(Ann Surg.* 2008;247(2):250-257).

b. Patients with no identifiable cause to rule out occult common bile duct stones, strictures, or neoplasms.

c. Suspected pancreatic ductal disruption, such as with traumatic pancreatitis.

C. Prognosis. Because the associated mortality of fulminant acute pancreatitis approaches 40% and randomized studies have shown that early aggressive supportive care improves outcomes, attempts have been made to identify clinical parameters that predict patients at higher risk of

developing severe outcomes.

1. Ranson criteria (Table 22-1) constitute the most frequently utilized predictor of mortality associated with acute pancreatitis. The limitation of this assessment tool is that a score cannot be calculated until 48 hours after admission.

2. CTSI is a prognostic scale based on CT findings, including peripancreatic fluid collections and fat inflammation, and extent of pancreatic necrosis was originally described by Balthazar et al. *(Radiology.* 1994;174:331-336) and then modified to a simpler model *(Am J Roent.* 2004;183:1261-1265) (Tables 22-2 and 22-3).

TABLE 22-1 Ranson Criteria

Admission	
Age	>55 yrs
White blood cell count	>16,000/μL
Blood glucose	>200 mg/dL
Serum lactate dehydrogenase	>350 IU/L
Aspartate aminotransferase	>250 IU/L
Initial 48 hrs	
Hematocrit decrease	>10%
Blood urea nitrogen elevation	>5 mg/dL
Serum calcium	<8 mg/dL
Arterial Po_2	<60 mm Hg

Base deficit	>4 mEq/L
Estimated fluid sequestration	>6 L

Mortality

Number of Ranson Signs	Approximate Mortality (%)
0-2	0
3-4	15
5-6	50
>6	70-90

3. The **Acute Physiology and Chronic Health Evaluation (APACHE) II** can be calculated at admission and updated daily to allow continual reassessment. However, the APACHE II score is somewhat cumbersome and difficult to calculate that limits its everyday use.

4. Multiple Organ Dysfunction Score (MODS) and Sequential Organ Failure Assessment (SOFA) have been shown to be important predictors of disease severity in critically ill patients and have been extended to patients with severe acute pancreatitis and are predictive of mortality and development of complications *(Br J Surg.* 2009;96(2):137-150).

D. Complications

1. Necrotizing pancreatitis occurs in about 10% to 20% of acute pancreatitis cases, and its presence correlates with prognosis (see CTSI above). It may be present at initial presentation or develop later in the clinical course. Necrosis is diagnosed on CT as failure to enhance with intravenous contrast.

2. Infected pancreatic necrosis occurs in 5% to 10% of cases and is the cause of most late deaths (>14 days). Whether pancreatic necrosis

visualized on CT scan is infected cannot be determined by imaging; however, gas in the areas of necrosis is suggestive. The gold standard for diagnosing infected pancreatic necrosis is fine needle aspiration (FNA), but this is rarely necessary needed as treatment is based on clinical status and blood cultures.

TABLE 22-2 CT Severity Grading Index (CTSI) Scoring Based on Imaging Characteristics

Scoring for Pancreatic Necrosis	
0 Points	No pancreatic necrosis
2 Points	²30% pancreatic necrosis
4 Points	>30% necrosis
Evaluation of Pancreatic Morphology, Not Including Necrosis	
0 Points (grade A)	Normal pancreas
2 Points (grade B/C)	Focal or diffuse enlargement of the gland, including contour irregularities and inhomogeneous attenuation with or without peripancreatic inflammation
4 Points (grade D/E)	Pancreatic or peripancreatic fluid collection or peripancreatic fat necrosis
Additional 2 points	Extra pancreatic complications including one or more of the following: pleural effusion, ascites, vascular complications, parenchymal complications, or gastrointestinal tract involvement

3. Acute pseudocyst (see Section V).

4. Visceral pseudoaneurysm is a rare complication, and is most common in patients with necrotizing pancreatitis. The most common arteries involved are the splenic, gastroduodenal, and left gastric arteries. Rupture is a life-threatening emergency and usually presents with signs and symptoms of upper gastrointestinal bleeding. Angiography is the first step in management and can be both diagnostic and therapeutic *(Am J Surg.* 2005;190(3):489-495).

TABLE 22-3 Prognosis Based on CTSI Score

Index	Predicted Morbidity	Predicted Mortality
0-3	8%	3%
4-6	35%	6%
7-10	92%	17%

5. Because of the proximity of the splenic, superior mesenteric, and portal veins, **venous thrombosis** is not uncommon in patients with acute pancreatitis.

E. Treatment. End-organ failure is associated with poorer outcomes. Therefore, the initial approach to managing acute pancreatitis focuses on supporting patients with aggressive fluid resuscitation and close monitoring.

1. Supportive care

a. Volume resuscitation with isotonic fluids is crucial; urinary output is monitored with a Foley catheter targeting greater than 0.5 mL/kg/hour. During the course of resuscitation, patients should be maintained on continuous pulse oximetry as patients often require large volume fluid resuscitation and frequent monitoring of electrolytes.

b. Gastric rest with nutritional support. Nasogastric decompression is performed to decrease neurohormonal stimulation of pancreatic secretion. Acute pancreatitis is a hypercatabolic state, and nutritional support has been shown to have a significant impact on outcomes in critically ill patients. Enteral feeding is generally preferred to parenteral nutrition. Early enteral feeding in patients with severe acute pancreatitis is associated with lower rates of infection, surgical intervention, and length of stay *(BMJ.* 2004;328:1407).

c. Analgesics are required for pain relief.

d. Respiratory monitoring and arterial blood gases are usually necessary in severe pancreatitis to assess oxygenation and acidÑbase status. Hypoxemia is common, even in mild cases of acute pancreatitis given the volume of fluid resuscitation and the potential for development of sympathetic effusions. Pulmonary complications occur in up to 50% of patients.

e. Antibiotics. The routine use of antibiotic prophylaxis in acute pancreatitis, especially in mild-to-moderate cases, is not supported in the literature. Conflicting data exist regarding antibiotics in severe cases, as there are small prospective, randomized trials demonstrating significantly lower rates of septic complications in patients receiving antibiotics *(Ann Surg.* 2006;243:154) and subsequent data from an RCT that found differences in infection or surgical intervention *(Ann Surg.* 2007;245:674). However, a meta-analysis demonstrated no difference in mortality, infected necrosis, or overall infections with antibiotic therapy *(Cochrane Database Syst Rev.* 2010;5:CD002941). When infection is confirmed or suspected, patients should be treated with broad-spectrum systemic antibiotics that cover Gram-negative bacteria and, depending on length of hospitalization, common hospital-acquired pathogens, including fungal organisms, as super infection can be seen commonly.

2. Interventional and surgical treatment is necessary in a small percent of cases, fluid collections and/or pancreatic necrosis needs to be treated. The indications are clinical deterioration, sepsis, hypotension,

and evidence of gastrointestinal obstruction due to the collections. Debridement of necrotic pancreas should be delayed to at least 18 to 20 days after onset of attack to allow sequestration of the necrosis. In severely ill patients percutaneous or endoscopic drainage using multiple and/or large drains is often used as a first step to stabilize patients. Resection of necrosis by endoscopic or minimally invasive or open surgery is performed as a second procedure several weeks later if necessary *(NEJM.* 2010;362(16):1491-1502; *Br J Surg.* 2011;98(1):18-27). Open surgery was the mainstay of treatment but is needed uncommonly today. Rarely surgery is needed for bleeding or organ perforation.

3. Treatment of gallstone pancreatitis. In mild cases of acute pancreatitis, laparoscopic cholecystectomy with operative cholangiogram is indicated on the index admission or soon thereafter in healthy patients. Delay has resulted in the occurrence of a second attack, which may be more severe. In patients with severe gallstone pancreatitis, cholecystectomy should be performed when general and local conditions permit. Operative cholangiography is needed to rule out persistent choledocholithiasis, although since acute pancreatitis is caused by small stones only 10% are found to have residual stones at the time of surgery. In patients not fit for surgery endoscopic sphincterotomy may protect against further attacks of pancreatitis.

II. CHRONIC PANCREATITIS

A. Etiology. Alcohol (EtOH) abuse is the most common cause (70%); however, other etiologies include idiopathic, metabolic (hypercalcemia, hypertriglyceridemia, hypercholesterolemia, hyperparathyroidism), drugs, trauma, genetic (SPINK1, cystic fibrosis), and congenital abnormalities (sphincter of Oddi dysfunction or pancreas divisum). It also appears that tobacco abuse plays an important role in the development of chronic pancreatitis and particularly in patients with EtOH-related disease *(Arch Intern Med.* 2009;169:1035-1045). A history of

recurrent acute pancreatitis is present in some but not all patients with chronic pancreatitis.

B. Pathophysiology. Chronic pancreatitis is characterized by diffuse scarring and strictures in the pancreatic duct and commonly leads to endocrine or exocrine insufficiency, although substantial glandular destruction must occur before secretory function is lost. Most patients who develop diabetes already have pancreatic exocrine insufficiency and steatorrhea. Reduced food intake, due to pain, and malabsorption lead to malnutrition.

C. Diagnosis is based on history and examination, complemented by appropriate investigative studies. **Upper midepigastric pain radiating to the back** is the cardinal symptom and is present in 85% to 90% of cases, and becomes progressively worse over time. Changes in bowel habits and bloating are other common early symptoms, followed later by steatorrhea

and diabetes as the disease progresses. Weight loss is common and food fear may be present. Upper abdominal tenderness may be present. Less common findings include jaundice secondary to stricture of the common bile duct, enlarged spleen secondary to thrombosis of the splenic vein, or ascites secondary to a pancreatic peritoneal fistula.

1. Laboratory tests

a. Amylase and lipase levels are elevated in acute pancreatitis but rarely are useful in chronic pancreatitis and are **commonly normal** due to progressive loss of pancreatic function.

b. Pancreatic secretin stimulation tests have proven to be highly sensitive (90% to 100%) and specific (>90%) test for the diagnosis of chronic pancreatitis.

c. Pancreatic endocrine function. Fasting and 2-hour postprandial blood glucose levels or glucose tolerance tests may be abnormal in 14% to 65% of patients with early chronic pancreatitis and in up to 90% of patients when calcifications are present.

d. A 72-hour fecal collection for estimation of daily fecal fat is relatively simple and cheap, but plays a limited role in the definitive diagnosis of chronic pancreatitis as patients must have a high degree of pancreatic insufficiency to have a positive test.

2. Radiologic studies

a. Plain films of the abdomen may show diffuse calcification of the pancreas in 30% to 40% of patients.

b. Ultrasound. Transabdominal ultrasound has low sensitivity and is subject to limitations related to user dependency, body habitus, and overlying bowel gas, and plays a limited role in the diagnosis of chronic pancreatitis.

c. CT is 80% sensitive and 75% to 90% specific for the diagnosis of parenchymal or ductal disease. Common findings include ductal dilatation, calcifications, atrophy, and cystic lesions. CT is also useful to evaluate for mass lesions and sequelae of chronic pancreatitis.

d. MRI is less sensitive than CT for detection of calcification. MR pancreatography is more sensitive in visualizing a dilated duct and strictures but loses sensitivity relative to ERCP in evaluating sidebranch disease (i.e., small duct disease).

e. ERCP provides the greatest detail of pancreatic duct anatomy, demonstrating strictures and areas of dilation. The presence of both may give the characteristic Òchain of lakesÓ picture. ERCP may also be beneficial for evaluation of pancreatic mass lesions, cytology, and can be therapeutic. There are drawbacks, however, in that images must be interpreted by specialized individuals, and there is a 3% to 7% risk of causing acute pancreatitis.

f. Endoscopic ultrasound (EUS). EUS has come to play a more important role in the diagnosis of biliary obstruction. Criteria for the diagnosis of chronic pancreatitis is based on EUS characteristics, such as lithiasis within the main pancreatic duct and parenchymal honeycombing, referred to as the Rosemont criteria *(Gastrointest Endosc.* 2009;69(7):1251-1261).

D. Complications

1. Common bile duct obstruction may result from transient obstruction from pancreatic inflammation and edema or from stricture of the intrapancreatic common bile duct. When present, **strictures** are often **long and smooth** (2 to 4 cm in length) and must be distinguished from malignancy.

2. Intestinal obstruction. Duodenal obstruction can occur due to acute pancreatic inflammation, chronic fibrotic reaction, pancreatic pseudocyst, or neoplasm. Rarely the colon may become obstructed.

3. Pancreaticoenteric fistulas result from spontaneous drainage 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.

4. Pancreaticopleural fistulas often have communication from the distal duct traversing the esophageal hiatus.

5. Pseudocyst (see Section V.A).

6. Splenic vein thrombosis (see Section I.D.5).

7. Pancreatic carcinoma. Chronic pancreatitis has been suggested to increase the risk of pancreatic carcinoma by two- to threefold.

E. Treatment

1. Medical management

a. Malabsorption or steatorrhea. Most patients will experience improvement in steatorrhea and fat absorption with pancreatic enzyme supplementation. In addition, there is some evidence that adequate enzyme supplementation improves pain control.

b. Diabetes initially is responsive to careful attention to overall good nutrition and dietary control; however, use of oral hypoglycemic agents or insulin therapy often is required.

c. Narcotics are often required for pain relief. In selected patients, tricyclic antidepressants and gabapentin may be effective.

d. Abstinence from alcohol results in improved pain control in approximately 50% of patients.

e. Cholecystokinin antagonists and somatostatin analogs have been considered for treatment of chronic pancreatitis, but have yet to show improvements in pain control.

f. Tube thoracostomy or repeated paracentesis may be required for pancreatic pleural effusions or pancreatic ascites. Approximately 40% to 65% of patients respond to nonsurgical management within 2 to 3 weeks.

2. Endoscopic therapy. Endoscopic sphincterotomy, stenting, stone retrieval, and lithotripsy have all been used with moderate success in the management of patients with ductal complications from chronic pancreatitis. Endoscopic celiac plexus block may improve symptoms in patients with severe pain. See Section V.A for the discussion of pancreatic pseudocysts. Surgical drainage of the pancreatic duct has been demonstrated to be effective than endoscopic treatment in patients with obstruction of the pancreatic duct due to chronic

pancreatitis in a small randomized clinical trial *(N Engl J Med.* 2007; 15:356(7):676-84).

3. Surgical principles

a. Indications for surgery. By far the most common indication is unremitting pain, but others include the inability to rule out neoplasm and management of complications (pseudocyst, aneurysm, and fistula).

b. Choice of procedure. The goals of surgical therapy are drainage and/or resection of the diseased pancreas to alleviate pain and complications associated with chronic pancreatitis. Most modern procedures combine drainage with some resection of the pancreas.

c. Drainage/resection procedures

(1) The Frey procedure is a major modification of earlier operations which removed duct stones and opened ductal strictures in the body of the gland and then provided new drainage of the duct by lateral pancreatojejunostomy (Puestow, PartingtonÑ Rochelle). These operations and the Frey procedure are best suited for patients with dilated ducts. The earlier operations often failed because the pancreatic duct in the head of the gland was not drained adequately. In the Frey procedure, the proximal pancreatic duct is also cleared by extensive coring of the head of the gland. This is the most common procedure performed at our institution and throughout North America. The Frey procedure has shown to provide excellent pain control and patient satisfaction in chronic pancreatitis.

(2) The Beger procedure is a duodenum-preserving resection of most 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. The pancreas is then transected at the pancreatic neck. This procedure has also shown excellent long-term results *(Ann Surg.* 1999;230(4):512-519); however, the procedure is more difficult because it requires dissection along the SMV. It is rarely performed in North America.

d. Pancreatectomy

(1) PD (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 cancer cannot be ruled out in the head of the pancreas. The Whipple has been shown to be inferior to both the Beger *(Int J Pancreatol.* 2000;27(2):131-142) and Frey *(Ann Surg.* 1998;228:771) procedures for this indication.

(2) Distal subtotal pancreatectomy is used for disease in the tail of the gland and in patients with previous ductal injury from blunt abdominal trauma with fracture of the pancreas and stenosis of the duct at the midbody level.

(3) Total pancreatectomy is performed only as a last resort in patients whose previous operations have failed and who appear to

be capable of managing an apancreatic state. Some centers have combined this procedure with islet cell transplantation. The latter seems particularly applicable in cases of juvenile pancreatitis.

e. Celiac plexus block can be achieved surgically by either ganglionectomy or direct injection of sclerosing agents. However, today endoscopic injection is used most commonly. Its effect is temporary.

III. PANCREATIC DUCTAL ADENOCARCINOMA

A. Incidence and Epidemiology. Pancreatic cancer is the fourth-leading cause of cancer-related mortality in the United States. Most patients have incurable disease at the time of diagnosis, and the overall 5-year survival is approximately 6%. The median age at diagnosis is 65 years. The survival or resected patients is about 20%.

B. Risk Factors. An increased risk of pancreatic ductal adenocarcinoma (PDAC) has been associated with smoking, alcoholism, family history, hereditary disorders (hereditary nonpolyposis colon cancer [HNPCC], von HippelÑLindau disease [VHL], PeutzÑJeghers syndrome, familial breast cancer [BRCA2], familial atypical multiple mole melanoma [FAMMM]), and chronic pancreatitis.

C. Pathology. PDAC accounts for the majority of pancreatic malignancies (90%). Seventy percent of PDAC occur at the head, 20% in the body, and 10% in the tail.

D. Diagnosis. Symptoms associated with pancreatic cancer are almost always gradual in onset

and are nonspecific.

1. History and examination. In cancer of the head of the pancreas bile duct obstruction, which is frequent, leads to the classical presentation of painless jaundice, pruritus, dark urine, and pale stools. Malaise, nausea, fatigue, and weight loss are common and some patients do have epigastric or back pain. Epigastric abdominal pain improved with leaning forward (Ingelfinger sign) is also sometimes present. In cancer of the distal pancreas pain and weight loss predominate. Some patients present just with steatorrhea when the pancreatic duct alone is obstructed. New-onset diabetes within the year prior to diagnosis is found in 15% of patients with pancreatic cancer. *Trousseau sign* (migratory thrombophlebitis) has been associated with pancreas cancer.

2. Laboratory tests

a. Elevated serum bilirubin with >50% direct reacting bilirubin.

b. Elevated alkaline phosphatase.

c. Prolonged obstruction may lead to mild increase in AST and ALT.

a, b, and c are seen with biliary obstruction.

d. Tumor markers. Serum CA19-9 is often elevated. It is a useful marker to follow in patients with elevated levels prior to initiation of therapy; however, it is often low in patients with resectable disease and can be elevated in nonmalignant biliary obstructive disease. CA19-9 levels pretreatment may also have some role in determining

prognosis *(Cancer.* 2009;115(12):2630-2639). Carcinoembryonic antigen (**CEA**) is elevated in 40% to 50% of patients with pancreas cancer.

3. Radiologic studies

a. CT imaging should be a fine-cut, Òpancreatic protocol CTÓ including three phases (arterial, venous, and portal venous) and thin slices (²3 mm) to allow for assessment of the relationship of the mass to vascular structures as this is crucial to determine resectability. Pancreatic cancer on CT usually appears as a hypoattenuating indistinct mass that distorts the normal architecture of the gland, often paired with findings of a dilated pancreatic and biliary ductal system (the so-called Òdouble-ductÓ sign). The CT criteria used to define resectability have been outlined in an expert consensus statement *(Ann Surg Oncol.* 2009;16(7):1727-1733):

(1) Locally resectable disease: No distant metastases; no radiographic evidence of SMV and portal vein abutment, distortion, tumor thrombus, or venous encasement; clear fat planes around the celiac axis, hepatic artery, and SMA.

(2) Borderline resectable: No distant metastases; venous involvement of SMV/portal vein demonstrating tumor abutment with or without impingement and narrowing of the lumen, encasement of the SMV/portal vein but without encasement of the nearby arteries, or short

segment venous occlusion resulting from either tumor thrombus or encasement but with adequate vessels above and below site of malignancy to allow for safe resection and reconstruction; gastroduodenal artery encasement up to the hepatic artery with either short segment encasement or direct abutment of the hepatic artery, without extension to the celiac axis; tumor abutment of the SMA not to exceed greater than 180 degree of the vessel circumference.

(3) Unresectable: Distant metastases; major venous thrombosis of the SMV or portal vein for several centimeters; encasement of SMA, celiac axis, or hepatic artery.

b. EUS and **ERCP,** especially the former, play an important role in patients in whom a mass is not seen on CT, obtaining tissue diagnosis when necessary (e.g., to determine candidacy for neoadjuvant therapy or when the diagnosis is in doubt). In addition, ERCP can be performed for drainage of biliary obstruction. Preoperative stenting is controversial as it has been associated with an increase in postoperative complications *(NEJM.* 2010;362(2):129-137). However, it is advisable in patients whose bilirubin is very high and in those whose surgery will be delayed due to neoadjuvant therapy or treatment of comorbidities.

c. MRI and MRCP can provide information similar to that in conventional CT.

d. Staging laparoscopy is used sparingly in cancer of the head of the pancreas where palliative operations are useful. A high suspicion for metastatic disease would be an indication (e.g., high CA19-9). It is

advisable for cancers of the distal pancreas where peritoneal metastases are common and surgical palliation is not performed

E. Treatment

1. Resection

a. PD (Whipple procedure) consists of en bloc resection of the head of the pancreas, distal common bile duct, duodenum, jejunum, and gastric antrum. Pylorus-sparing PD has been advocated by some, but there are no data demonstrating improved survival or lower morbidity *(Cochrane Database Syst Rev.* 2011;2:CD006053). There has been a sharp decline in morbidity and mortality in specialized centers, with a 30-day mortality of less than 3%.

b. Distal pancreatectomy. The procedure of choice for lesions of the body and tail of the pancreas is distal pancreatectomy. Distal pancreatectomy consists of resection of the pancreas, generally at the SMV laterally to include the spleen. We have described a technique that provides a more radical resection with improved R0 resection rates, the radical antegrade modular pancreatosplenectomy (RAMPS), when compared to traditional series which is the procedure of choice for malignant tumors of the distal pancreas at our institution *(J Am Coll Surg.* 2012;214(1):46-52).

2. Postoperative considerations. Delayed gastric emptying, pancreatic fistula, and wound infection are the three most common complications of PD. Delayed gastric emptying almost always subsides with conservative treatment. The rate of pancreatic fistula may be reduced by meticulous attention to the blood supply of the pancreaticoenteric duct-to-mucosa anastomosis *(J Am Coll Surg.* 2002;194:746). Most surgeons routinely place abdominal drains, and this supported by a recent distal pancreatectomy has a higher morbidity and leak rates than PD with an approximately 20% pancreatic leak rate in most series; however, this is usually amenable to percutaneous treatment, and distal pancreatectomy has a similar mortality to PD.

3. Radiotherapy and chemotherapy

a. Neoadjuvant therapy. Some groups routinely use preoperative chemotherapy with or without radiation, while others use this selectively.

b. Adjuvant therapy. There is a clear benefit to adjuvant therapy in pancreatic cancer *(J Gastrointest Surg.* 2008;12(4):657-661); however, the choice between chemoradiation and chemotherapy is less clear. The role of radiation therapy in pancreatic cancer and what role clinic-pathologic factors may play in selecting patients for radiation therapy has yet to be fully elucidated.

4. Prognosis. Surgical resection increases survival over patients with similar stage disease that do not undergo resection. Overall 5-year survival rates are approximately 20% for patients after resection. In patients with small tumors, negative resection margins, and no evidence of nodal metastases, the 5-year survival rate is as high as 40%. Median survival for unresectable locally advanced disease is 12 months, and for hepatic metastatic disease it is 6 months.

F. Pseudotumors of the Pancreas

1. Inflammatory and fibrosing conditions of the pancreas may form dense, fibrotic masses, and segmental fibrosis that are difficult to differentiate from carcinoma preoperatively. **Lymphoplasmacytic sclerosing pancreatitis** is often misdiagnosed as pancreatic cancer. Patients are typically young (30s to 50s) and may be associated with other autoimmune disorders (Sjšgren, ulcerative colitis, sclerosing cholangitis). When compared to patients with pancreatic cancer of all stages, these patients may have increased levels of serum IgG4, which can aid in making this diagnosis *(Ann Surg Oncol.* 2008;15(4):1147-1154).

IV. NEUROENDOCRINE NEOPLASMS OF THE PANCREAS

(See Chapter 39)

V. RARE NEOPLASMS OF THE PANCREAS

A. Acinar cell carcinoma is more common in men, and treatment is resection. Prognosis is slightly better than with pancreatic adenocarcinoma, but recurrence is common.

B. Solid pseudopapillary tumor is most commonly seen in young females, especially African-American. These tumors are typically large at presentation and are less frequently metastatic. Treatment is resection, and prognosis is generally favorable.

C. Metastatic tumors to the pancreas are most commonly renal cell carcinomas (RCC). Less common primaries include ovarian, colon, and melanoma. When isolated to the pancreas, resection in the setting of RCC has been associated with 60% 5-year survival *(J Am Coll Surg.* 2010;211(6):749-53).

D. Lymphoma can be primary or metastatic to the pancreas. Treatment is combined multimodality therapy with chemotherapy and radiation, without surgical resection.

VI. CONGENITAL ABNORMALITIES

A. Failure of the ventral and dorsal pancreatic buds to fuse during the sixth week of development results in **pancreatic divisum.** In this condition, the dorsal duct of Santorini becomes the means of pancreatic drainage from the bulk of pancreatic tissue (body, tail, and superior portion of head). The condition is present in about 10% of the population. Pancreas divisum is associated with an increased risk of pancreatitis. Minor papilla endotherapy may improve outcomes in patients with recurrent pancreatitis. Patients with severe symptomatic pancreas divisum may require surgical therapy.

B. Malrotation of the ventral primordium during the fifth week results in **annular pancreas:** A thin, flat band of normal pancreatic tissue surrounding the second part of the duodenum. The annular pancreas usually contains a duct that connects to the main pancreatic duct. Annular pancreas may cause duodenal obstruction usually early in life but sometimes later in life. The treatment of choice is duodenoduodenostomy or duodenojejunostomy.

VII. CYSTIC DISEASES

A. Pancreatic Pseudocysts. It is important to distinguish pseudocysts from tumors, cystic pancreatic neoplasms, and other fluid collections. 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, and many resolve spontaneously. Pseudocysts differ from true cysts in that the wall is reactive inflammatory tissue as opposed to an epithelial-lined sac that secretes fluid. By definition, a fluid collection appearing in the first 4 weeks after the onset of pancreatitis is an *acute fluid collection;* after 4 weeks, it becomes an *acute pseudocyst.* Pseudocysts become chronic and may require treatment months after the acute attack has subsided.

1. Causes. Pseudocysts develop after disruption of the pancreatic duct with or without proximal obstruction, usually occurring after an episode of acute pancreatitis.

2. Diagnosis

a. Clinical presentation. The most common complaint is recurrent or persistent upper abdominal pain. Other symptoms include nausea, vomiting, early satiety, anorexia, weight loss, back pain, and jaundice. Physical examination may reveal upper abdominal tenderness, a mass.

b. Laboratory tests

(1) Amylase. Serum concentrations are elevated in approximately one-half of cases.

(2) Liver function tests occasionally are elevated and may be useful if biliary obstruction is suspected.

(3) Cystic fluid analysis is discussed in Section V.B.2.

c. Radiologic studies

(1) CT is the radiographic study of choice for initial evaluation of pancreatic pseudocysts. CT scan findings that determine prognosis include the following:

(a) Pseudocysts **smaller than** 4 **cm** usually resolve spontaneously.

(b) Pseudocysts with **wall calcifications** generally do not resolve.

(c) Pseudocysts with **thick walls** are resistant to spontaneous resolution.

(2) MRI and MRCP can be useful to delineate ductal anatomy and are not associated with the risks of pancreatitis and infection with ERCP. MRCP is not as sensitive for small duct involvement as ERCP.

d. 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. ERCP itself risks infection of a communicating pseudocyst.

3. Complications

a. Infection is reported in 5% to 20% of pseudocysts and requires external drainage.

b. Hemorrhage results from erosion into surrounding visceral vessels. The most common arteries are the splenic (45%), gastroduodenal (18%), and pancreaticoduodenal (18%) arteries. Immediate angiographic embolization has emerged as the initial treatment of choice.

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 onehalf of patients can be treated nonsurgically, with total parenteral nutrition and symptomatic paracentesis or thoracentesis.

However, rupture is occasionally a surgical emergency.

e. Enteric fistula can occur spontaneously and usually results in resolution of the cyst.

4. Treatment depends on symptoms, age, pseudocyst size, and the presence of complications. **Pseudocysts smaller than 6 cm and present for less than 6 weeks have low complication rates.** The chance of spontaneous resolution after 6 weeks is low, and the risk of complications rises significantly after 6 weeks.

a. Nonoperative. If the pseudocyst is new, asymptomatic, and without complications, the patient can be followed with serial CT scans or US to evaluate size and maturation.

b. Percutaneous drainage can be considered for patients in whom the pseudocyst does not communicate with the pancreatic duct and for those who cannot tolerate surgery or endoscopy. External drainage is indicated when the pseudocyst is infected and without a mature wall.

c. Excision, including resection is only performed in unusual settings including bleeding, systemic sepsis, and concern for malignancy.

d. Internal drainage. Cystoenteric drainage is the procedure of choice in uncomplicated pseudocysts requiring intervention. Drainage can be undertaken by either surgical or endoscopic means. Endoscopic cystogastrostomy or cystoduodenostomy has a 60% to 90% success rate, and is the initial treatment of choice at our center. Endoscopic therapy also allows transsphincteric stenting in the case of duct-cyst communication. In the event drainage cannot be accomplished by endoscopic methods, surgical methods include Roux-en-Y cystojejunostomy, loop cystojejunostomy, cystogastrostomy, and rarely cystoduodenostomy. A biopsy of the cyst wall should be obtained to rule out neoplasia in the cyst.

B. True pancreatic cysts are most commonly serous cystadenomas (SCAs), mucinous cystic neoplasms (MCNs), and intraductal papillary mucinous neoplasms (IPMNs). The latter two are mucin-secreting premalignant cysts and depending on circumstances may require resection in asymptomatic patients. SCAs become malignant very rarely and are resected only if symptomatic. Cyst fluid, usually obtained by EUS is analyzed to determine whether the cyst is an SCA or an MCN/IPMN. Cyst fluid CEA >192 is

diagnostic of the latter two while CEA of <5 IU is indicative of an SCA. Intermediate values are indeterminate and diagnosis may be aided by presence of mucin or dysplasia in the sample.

1. SCAs are benign lesions that are usually asymptomatic. Symptoms correlate with size (>4 cm). They are more common in women, are most commonly located in the head of the pancreas, and account for 30% of all cystic pancreatic neoplasms. Lesions are characterized by an epithelial lining, nonviscous fluid, and low CEA and amylase on cyst fluid analysis. They are usually multicystic and in some a honeycomb of cystic cells with a calcified center. These may be mistaken for solid tumors. Asymptomatic SCAs do not require treatment.

2. MCNs are considered premalignant lesions and account for approximately 50% of all cystic

pancreatic neoplasms. At presentation they are usually asymptomatic, twice as likely to present in women, and more commonly located in the body or tail. These cystic lesions do not communicate with the pancreatic ductal system. Invasive cancer is present in 17.5% of resected MCN, and malignancy is associated with larger size (>4 cm) and advanced age (>55). Five-year survival was 100% for noninvasive MCN and 57% for patients with malignant lesions *(Ann Surg.* 2008;247(4):571-579). As there is a clear survival advantage for those patients who undergo resection prior to the development of invasive cancer, and it is felt that there is an adenomaÑ adenocarcinoma sequence, it is recommended that all patients with MCN undergo resection.

3. IPMNs account for 25% of all cystic pancreatic neoplasms and have a slight male predominance. IPMNs communicate with the pancreatic ductal system. Characteristics of IPMN on ERCP include diffuse gland involvement; ductal dilation; and thick, viscous fluid within the cyst. **IPMNs are separated into three subgroups based on ductal involvement: Main duct, side branch, and mixed;** and therapy is different depending on subgroup. Main duct IPMN carries a malignant potential, up to 50% in some series *(Ann Surg.* 2004;239(6):788-799) and requires resection. Usually the dilation of the main duct is diffuse, but resection is limited to the head of the pancreas unless carcinoma in situ is identified at the resection margin. SideÑbranch IPMN is a more controversial topic, and consensus guidelines are evolving *(Pancreatology.* 2012;12(3):183-197). Generally accepted criteria for resection of side-branch IPMN include, size greater than 3 cm, symptomatic patient, and mural nodules. Patients with side-branch IPMN requiring resection should undergo a standard oncologic resection (Whipple or distal pancreatectomy). The current recommendation is to extend resection based on the invasive component or high-grade dysplasia only, rather than obtaining margins free of IPMN *(World J Gastrointest Surg.* 2010;2(10):352-358).

4. Other rare cystic pancreatic neoplasms (remaining 10%) include acinar cell cystadenocarcinoma, cystic choriocarcinoma, cystic teratoma, and angiomatous neoplasms. All lesions with carcinoma noted on preoperative biopsy or with a concern for malignancy should undergo resection if tolerated.

CHAPTER 22: SURGICAL DISEASES OF THE PANCREAS

Multiple Choice Questions

1. A 60-year-old male alcoholic with diabetes presents with 12 hours of abdominal pain and an elevated amylase and lipase. At admission, his white blood cell count is 11,000, AST is 100, and total bilirubin is 2. Which of the following findings in his history is associated with increased mortality according to Ranson criteria?

a. Age

b. White blood cell count

c. AST

d. Total bilirubin

e. Diabetes

View Answer

2. A 45-year-old female presents with abdominal pain and an elevated amylase and lipase. The rest of her laboratory values are remarkable for a mildly elevated AST of 100 and elevated white blood cell count of 15,000. On hospital day 3, her pain is resolved and she is tolerating a regular diet. Which of the following should be performed prior to discharge?

a. ERCP

b. RUQ ultrasound

c. Amylase and/or lipase

d. CT scan of abdomen

e. Serum ethanol level

View Answer

3. A 73-year-old male is referred for evaluation of an incidentally discovered 2-cm cyst in the tail of his pancreas. On examination the patient has no abdominal pain, and his laboratory values are unremarkable. He undergoes an endoscopic ultrasound which shows a cyst lesion that appears to communicate with the pancreatic duct, originating from a side branch. Which of the following is the next step in management?

a. Distal pancreatectomy

b. Total pancreatectomy

c. Observation

d. Enucleation

e. Biopsy

View Answer

4. A 35-year-old female is found to have an incidentally discovered 3-cm cystic lesion in the tail of her pancreas on a CT scan. She undergoes an endoscopic ultrasound which reveals a 3.5-cm cyst without communication with the pancreatic duct. Analysis of cyst fluid reveals high levels of mucin. What is the next step in the management of this

patient?

a. Distal pancreatectomy

b. Repeat CT scan in 1 year

c. MRCP

d. Total pancreatectomy

e. Endoscopic drainage

View Answer

5. A 59-year-old male is 2 weeks out from a pancreaticoduodenectomy for pancreatic adenocarcinoma complicated by a pancreatic fistula. He presents to the ED with new onset of bloody output in his drain. He is tachycardic to the 110s, but otherwise looks well. His Hgb is 10. What is the best course of management for this patient?

a. CT scan

b. ERCP

c. Angiogram

d. Exploratory laparotomy

e. Remove the drain

View Answer

6. A 60-year-old physically fit female with painless jaundice and a 20-lb weight loss presents for evaluation of a 2-cm hypodense mass in the head of the pancreas on CT scan. The patient was referred to you from a gastrointestinal medicine colleague, who performed an endoscopic ultrasound with biopsy and ERCP with stent. The biopsy is suspicious for malignancy. By imaging, the lesion appears to be clearly resectable without evidence of malignancy. Which of the following is the most appropriate management of this patient?

a. Repeat endoscopic ultrasound with biopsy

b. Pancreaticoduodenectomy

c. Total pancreatectomy

d. MRI pancreatogram

e. Neoadjuvant chemoradiation

View Answer

23
Spleen

Timothy M. Nywening

Maria B. Doyle

A. Anatomy. The spleen is derived from the mesoderm and resides in the left upper quadrant of the abdomen, where it is protected by the ninth to eleventh ribs. The average adult spleen is 12 cm long × 7 cm wide × 4 cm thick and weighs between 1,000 and 1,500 g. The spleen is highly vascularized, receiving up to 5% of cardiac output. The splenic artery, a branch of the celiac axis, runs posterior to the pancreas and most commonly arborizes into multiple small arteries to enter the hilum of the spleen. The inferior mesenteric vein drains into the splenic vein, which ultimately joins with the superior mesenteric vein to form the portal vein. Accessory spleens are found in 10% to 20% of the population and can be located anywhere in the abdomen but are most commonly found in the splenic hilum (Fig. 23-1).

B. Function. Histology of the spleen reveals highly vascularized red pulp interspersed with areas of white pulp. Red pulp consists of branching, thin walled sinuses and splenic cords filled with red blood cells (erythrocytes) and phagocytic cells. White pulp consists of T-cell rich periarteriolar sheaths, B-cell containing lymphoid nodules, and the marginal zone that serves as an interface between the lymphoid-dominant white pulp and erythrocyte-rich red pulp. These two histologies constitute the two major functions of the spleen:

1. Reticuloendothelial system: The red pulp serves to cull senescent erythrocytes and remodel healthy red cells. The spleen also serves as a reservoir for platelets. While extramedullary hematopoiesis uncommon in adults, the spleen may be a site of erythrocyte production in some disease states (i.e., myelofibrosis).

2. Immune system: The spleen is involved in both the innate (opsonization) and adaptive (antigen presentation) immune system. Opsonization of pathogens by the complement system results in enhanced phagocytosis and clearance in the spleen. The white pulp also acts as a site of antigen presentation to lymphocytes that, along with an appropriate cytokine milieu, leads to effective T-cell mediated cytotoxic activity and B-cell antibody responses.

C. Indications for Splenectomy (Table 23-1)

1. Hematologic conditions

a. Thrombocytopenias

(1) Idiopathic Immune Thrombocytopenic Purpura (ITP) is the most common indication

for elective splenectomy. It is an acquired disease that results from autoantibodies to platelet glycoprotein and results in immune mediated thrombocytopenia. The spleen

is both the major site of production of these antibodies as well as the principal site of platelet destruction.

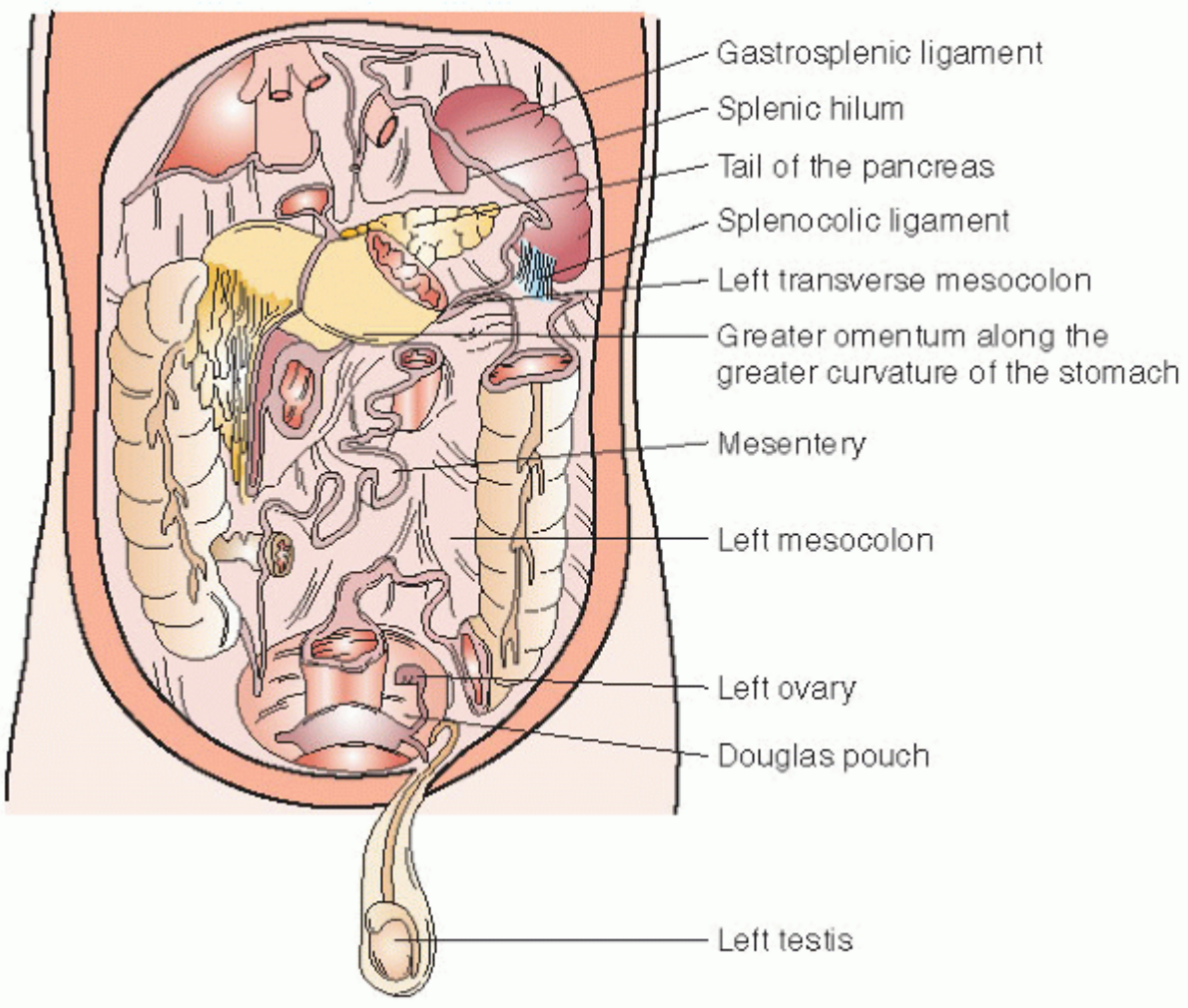

Figure 23-1 Location of accessory spleens. Usual location of accessory spleens: (1) Gastrosplenic ligament, (2) Splenic hilum, (3) Tail of the pancreas, (4) Splenocolic ligament, (5) Left transverse mesocolon, (6) Greater omentum along the greater curvature of the stomach, (7) Mesentery, (8) Left mesocolon, (9) Left ovary, (10) Douglas pouch, and (11) Left testis.

(a) Children: Most commonly present with acute ITP, in 70% to 90% of cases symptoms will remit regardless of therapy *(NEJM.* 2002;346:995). In refractory cases a waiting period of 12 months is recommended, especially in children below 5 years of age where risk of post-splenectomy sepsis is increased *(Blood.* 1996.88:871-875).

(b) Adults: Usually present with chronic ITP. First-line treatment with steroids results in a 50% to 75% response rate and may be combined with other modalities such as intravenous immune globulin (IVIG) and/or anti-Rh(D) infusions. However, 80% will have recurrence after cessation of therapy. Splenectomy results in 65% long-term remission (>5 years) and remains the treatment of choice in patients with platelets less the 30,000/mm^3 or with a high risk of bleeding. Most patients will achieve a response to splenectomy within

10 days postoperatively *(Am J Surg.* 2004;187:720-723). Alternatives to splenectomy include Rituximab (anti-CD20 monoclonal antibody) and thrombopoietin receptor antagonists which have shown efficacy as second-line agents *(Blood.* 2012;120:960-969). Rituximab has also been shown to have some efficacy in patient failing to respond to splenectomy *(Am J Hematology.* 2005;78:275-280) (Fig. 23-2).

TABLE 23-1 Clinical Conditions Requiring Splenectomy

Category	Common	Uncommon
Thrombocytopenias	Immune thrombocytopenic purpura	Thrombotic thrombocytopenic purpura
Anemias	Hereditary spherocytosis Autoimmune hemolytic anemias Sickle cell anemia	Thalassemias Hereditary elliptocytosis
Myeloproliferative and myelodysplastic disorders	Ñ	Chronic myelogenous leukemia Polycythemia vera Myelofibrosis Myeloid metaplasia Essential thrombocytosis
Lymphoproliferative disorders	Ñ	Chronic lymphocytic

		leukemia Hairy-cell leukemia Non-Hodgkin lymphoma Hodgkin lymphoma
Neutropenias	Ñ	Felty syndrome
Nonhematologic Etiologies	Trauma Incidental/iatrogenic splenectomy Splenic artery aneurysm	Splenic abscess Splenic cyst/pseudocyst Glycogen storage diseases

(2) Thrombotic Thrombocytopenic Purpura (TTP) is a systemic disease of resulting in the pentad of thrombocytopenia, microangiopathic hemolytic anemia (MAHA), altered mental status, renal failure, and fever. It is a result of decreased ADAMT13, a protease responsible for cleaving von Willebrand factor, leading to platelet aggregation and thrombosis of the microvasculature. It is most common in adults and usually idiopathic or drug (cyclosporine, gemcitabine, clopidogrel, quinine) related.

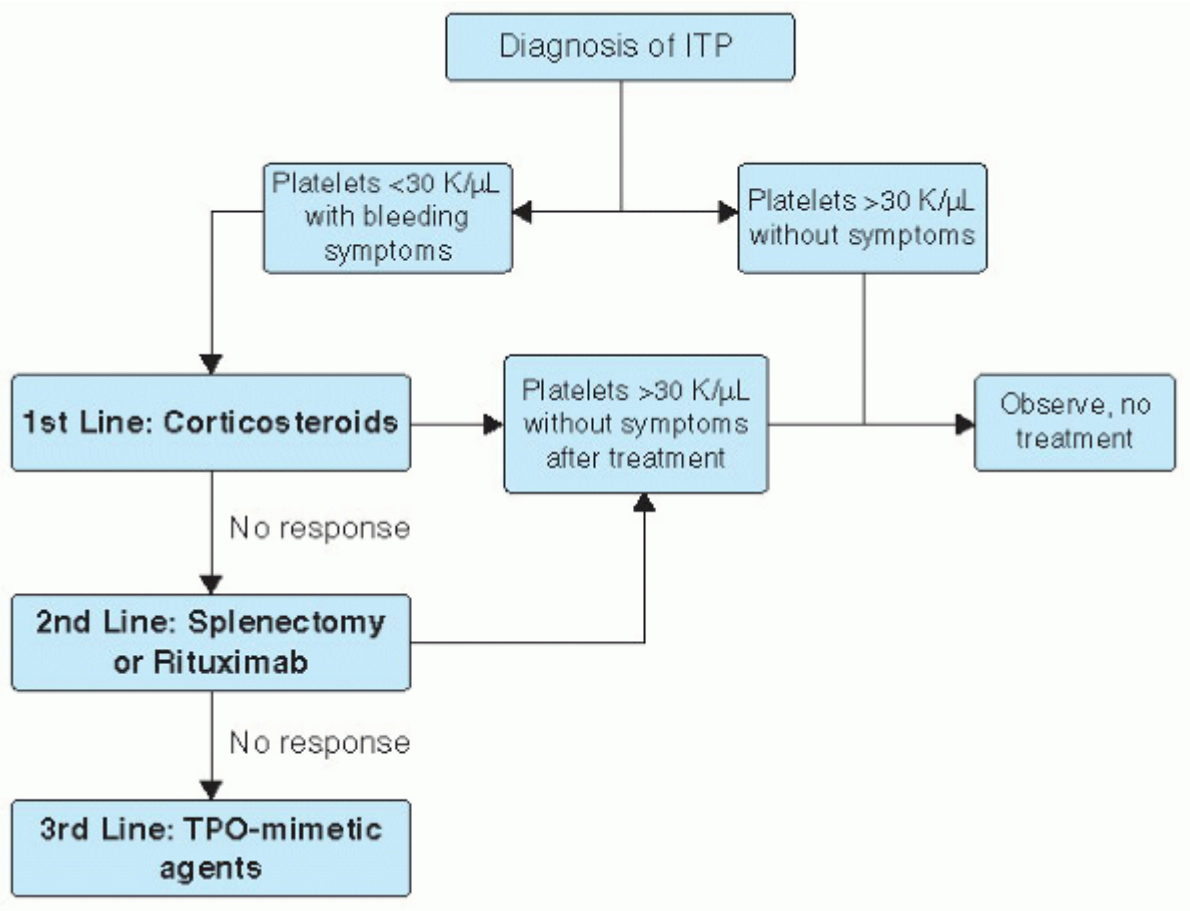

Figure 23-2 Treatment approach in ITP in adults. This diagram represents a simplified approach to the treatment of patients with ITP. A threshold platelet count of 30,000/µL for clinical decisions, rather than a range of platelet counts, is presented, but clinical symptoms and patients' concerns are more important for treatment decisions. (Adapted from George J, Leung LLP. *Treatment and prognosis of immune (idiopathic) thrombocytopenic purpura in adults.* UpToDate, 2011.)

(a) First-line treatment: Medical management with plasmapheresis, which had improved initial response and 6-month survival compared with plasma infusion *(NEJM.* 1991;325:393-397). Steroid therapy in addition to plasmapheresis is used in the treatment of relapse. Second-line agents include rituximab, cyclosporin, and increased frequency of plasmapheresis *(Br J Haematol.* 2012;158:323-335).

(b) Splenectomy: Reserved for those who do not respond to medical therapy or with chronically relapsing disease. Furthermore, splenectomy has only shown benefit when used in conjunction with plasmapheresis in order to achieve durable remission *(Br J Haematol.* 2005;130:768-776).

b. Anemias

(1) Hemolytic anemias constitute a group of diseases for which splenectomy is almost

universally curative.

(a) Hereditary spherocytosis is an autosomal dominant disorder characterized by a defect in an RBC membrane protein. The most common mutation is in the protein spectrin, but

other mutations in ankyrin, band 3, and palladin have been found. This defect results in small, spherical, rigid erythrocytes that fail to deform adequately to transverse the splenic microcirculation. This ultimately leads to the sequestration and destruction of erythrocytes in the spleen. Symptoms include anemia, jaundice (indirect bilirubinemia), and pigmented gallstones. Diagnosis is confirmed by the presence of spherocytes on peripheral blood smear, + osmotic fragility test, and decreased eosin-5-maleimide (EMA) binding *(Blood Rev.* 2013;27:167-178). Treatment includes folate supplementation and splenectomy for moderate to severe cases.

(b) Hereditary elliptocytosis is an autosomal dominant disorder in which an RBC cytoskeletal protein defect results in elliptical shaped erythrocytes. Most patients are asymptomatic with a mild anemia and do not require additional treatment. For select patients with symptomatic anemia splenectomy is usually curative.

(2) Acquired autoimmune hemolytic anemias

(a) Warm autoimmune hemolytic anemia occurs when IgG autoantibodies interact optimally with antigens at 37¡C. Diagnosis is confirmed with a positive direct Coombs test (incubation with anti-IgG serum results in RBC agglutination). Etiology is most often idiopathic but may also include chronic lymphocytic leukemia (CLL), non-Hodgkin lymphoma, collagen vascular disease, and drugs. Splenectomy is reserved for nonresponders or those requiring high steroid doses and is 60% to 70% effective in achieving remission. Rituximab has also shown efficacy and is suitable second-line treatment for those patients who do not desire to undergo splenectomy *(Blood.* 2010;116:1831-1838).

(b) Cold autoimmune hemolytic anemias are mediated by C3 complement fixation to IgM autoantibodies resulting in hemolysis at temperatures approaching 0¡C. Features include Reynaud like symptoms along with anemia. Most cases respond to protective clothing; however severe episodes may require cyclophosphamide, rituximab, or interferon. Splenectomy does not play a role in the treatment of cold autoimmune hemolytic anemias.

c. Congenital hemoglobinopathies

(1) Sickle cell anemia is a result of homozygous inheritance of the S variant of the hemoglobin beta chain. Autosplenectomy usually occurs secondary to repeated vaso-occlusive events and splenectomy is rarely required. However, splenectomy may be reasonable for selected patients with splenic abscess, symptomatic splenomegaly, hypersplenism, or acute splenic sequestration crisis.

(2) Thalassemias are hereditary anemias that result from a defect in hemoglobin synthesis. β-thalassemia major is typically treated

with iron chelation therapy as most patients will succumb to hemosiderosis at an early age. Splenectomy is reserved for palliation of symptomatic splenomegaly or splenic infarcts.

d. Myeloproliferative and myelodysplastic disorders

(1) Chronic myelogenous leukemia is a myelodysplastic disorder characterized by the *bcr-abl* fusion oncogene, known as the *Philadelphia chromosome.* This oncogene results in a constitutively active tyrosine kinase.

(a) Treatment: First-line therapy utilizes the tyrosine kinase inhibitor (TKI) imatinib mesylate (Gleevec). Alternative TKI treatments *(dasatinib* and *nilotinib)* are used in cases of intolerance or suboptimal response. Stem cell transplantation is used for cases of treatment failure in eligible patients *(Blood.* 2006;108:1809-1820).

(b) Splenectomy: A large prospectively randomized trial compared splenectomy plus chemotherapy or chemotherapy alone in the treatment of early phase of CML. Splenectomy had no effect on survival or disease progression, but it did increase the rate of thrombosis and vascular accidents *(Cancer.* 1984;54:333-338). Splenectomy is indicated only for palliation of symptomatic splenomegaly or hypersplenism that significantly limits therapy.

(2) Polycythemia vera and essential thrombocytosis are chronic diseases of uncontrolled RBC and platelet production, respectively. These diseases are treated medically, but splenectomy can be required to treat symptomatic splenomegaly or pain from splenic infarcts. Splenectomy can result in severe thrombocytosis, causing thrombosis or hemorrhage, which requires perioperative antiplatelet, anticoagulation, and myelosuppressive treatment.

(3) Myelofibrosis and myeloid metaplasia are incurable myeloproliferative disorders that usually present in patients older than 60 years. The condition is characterized by bone marrow fibrosis, leukoerythroblastosis, and extramedullary hematopoiesis, which can result in massive splenomegaly. Indications for splenectomy include symptomatic splenomegaly and transfusion-dependent anemias. Although the compressive symptoms are effectively palliated with splenectomy, the cytopenias frequently recur. In addition, these patients are at increased risk for postoperative hemorrhage and thrombotic complications after splenectomy.

e. Lymphoproliferative disorders

(1) CLL, a B-cell leukemia, is the most common of the chronic leukemias and is characterized by the accumulation of mature but nonfunctional lymphocytes. Primary therapy is medical, with splenectomy reserved for those patients with symptomatic splenomegaly and severe hypersplenism.

(2) Non-Hodgkin lymphoma is a diverse group of disorders with a wide range of clinical behaviors, ranging from indolent to highly aggressive. As with other malignant processes, splenectomy is

indicated for palliation of hypersplenism and cytopenias or for diagnosis in patients with suspected persistent or recurrent disease after systemic therapy. Splenectomy plays an important role in the diagnosis and staging of patients with isolated splenic lymphoma (known as malignant lymphoma with prominent splenic involvement). In these cases, improved survival has been shown in patients undergoing splenectomy *(Cancer.* 1993;71: 207-215).

(3) Hodgkin lymphoma historically had utilized splenectomy for diagnostic staging. However, due to refinements in imaging techniques and progress in the methods of treatment splenectomy for Hodgkin lymphoma is rare. Indications for surgery are similar to those for non-Hodgkin lymphoma.

(4) Hairy cell leukemia is a rare disease of elderly men that is characterized by B lymphocytes with membrane ruffling. Splenectomy was previously regarded as the primary therapy for this disease, but improvements in systemic chemotherapy have reduced the role of splenectomy, which is now reserved for patients with massive splenomegaly or refractory disease.

f. Neutropenias

(1) Felty syndrome is characterized by rheumatoid arthritis, splenomegaly, and neutropenia. The primary treatment is steroids, but refractory cases may require splenectomy to reverse the neutropenia. Patients with recurrent infections and significant anemia may benefit from splenectomy. Granulocytopenia is improved in approximately 80% of patients *(Arch Intern Med.* 1978;138:597-602). The clinical course of the arthritis is not affected.

2. Nonhematologic conditions

a. Trauma is the most common indication for splenectomy. In the unstable trauma patient the procedure is traditionally performed via laparotomy. With current imaging modalities grading of splenic injuries (Table 23-2) allows for conservative management in selected patients.

b. Incidental splenectomy occurs when the spleen is iatrogenically injured during an intra-abdominal procedure. Injury may result from a retractor placed in the left upper quadrant or during mobilization of the splenic flexure. Small injuries such as capsular tears may be controlled with hemostatic agents or electrocautery, but injuries resulting in significant blood loss may require splenectomy to achieve rapid hemostasis.

c. Vascular

(1) Splenic artery aneurysm is the most common visceral artery aneurysm and is typically an incidental finding. It occurs more commonly in females and associated with a high incidence of rupture during pregnancy with significant maternal and fetal mortality. Asymptomatic aneurysms in a patient whom pregnancy is not anticipated may be observed. Indications for intervention

include size ³2 cm, females of child-bearing age who may become pregnant and inflammatory pseudoaneurysms. Management depends on the location of the aneurysm during the course of

the splenic artery. Proximal and middle third aneurysms may be excluded by proximal and distal ligation of the artery. Splenic perfusion persists via collateralization from the short gastric vessels. For more distal lesions proximal ligation with splenectomy is required. Alternatives treatments include endovascular approaches with transcatheter embolization.

TABLE 23-2 The American Association for the Surgery of Trauma (AAST) Spleen Injury Scale (2008 Edition)

Grade	Injury Type	Injury Description
I	Hematoma Laceration	Subcapsular: <10% surface area Capsular: <1 cm parenchymal depth
II	Hematoma	Subcapsular: 10-50% surface area Intraparenchymal: <5 cm in diameter
	Laceration	Parenchymal depth: 1-3 cm *and* No trabecular vessel involvement
III	Hematoma	Subcapsular: >50% surface area *or* expanding/ruptured Intraparenchymal hematoma: >5 cm *or* expanding/ruptured
	Laceration	Parenchymal depth: >3 cm *or* Involving trabecular vessel
IV	Laceration	Laceration involving segmental or hilar vessels producing major devascularization (>25% of spleen)
V	Laceration Vascular	Shattered spleen Hilar vascular injury

[a]Advance one grade for multiple injuries *up to grade III.*
Adapted from Tinkoff G, Esposito TJ, Reed J, et al. American Association for the Surgery of Trauma Organ Injury Scale 1: Spleen, liver and kidney. *J Trauma* 2008;207(5):646-655.

d. Infectious

(1) Parasitic infections account for 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. The primary treatment is splenectomy, with careful attention not to spill the cyst contents. The cyst may be aspirated and injected with hypertonic saline prior to mobilization if concern about rupture exists.

(2) Splenic abscesses are rare, but potentially lethal if not accurately diagnosed and timely treatment instituted. Two-thirds arise from seeding of the spleen by a distant site, most commonly endocarditis and urinary tract infections. Abdominal CT and/or ultrasound imaging are the diagnostic modalities of choice. CT images reveal a low intensity lesion that does not enhance with contrast. Staphylococcus and streptococcus account for the most commonly identified organisms, accounting for >50% of cases. Fungal infections are rare, and may resolve with anti-fungal treatment alone. Percutaneous drainage may be used in select cases; however, splenectomy and appropriate antibiotic therapy is definitive treatment.

e. Cystic lesions of the spleen may be either true cysts or pseudocysts, but this differentiation is difficult to make preoperatively.

(1) True cysts (or primary cysts) have an epithelial lining and are most often congenital. Other rare true cysts include epidermoid and dermoid cysts.

(2) Pseudocysts (or secondary cysts) lack an epithelial lining and make up more than two-thirds of nonparasitic cysts. They typically result from traumatic hematoma formation and subsequently resorb.

(3) Treatment of splenic cysts depends on the size of the lesion and associated symptoms. Most are typically asymptomatic, but they may present with left upper abdominal or shoulder pain. Those smaller than 5 cm can be followed with ultrasonography and often resolve spontaneously. Larger cysts risk rupture and require cyst unroofing or splenectomy. Percutaneous aspiration is associated with infection and reaccumulation and is not indicated. Laparoscopic management of splenic cysts yields shorter hospital length of stay and fewer complications with no adverse effects *(Surg Endosc.* 2007;21:206-208).

D. Preoperative Preparation

1. Imaging with CT or MRI may be required in patients with malignancy or splenomegaly to accurately estimate splenic size and evaluate for hilar adenopathy that may complicate a laparoscopic approach. Right upper quadrant ultrasound is indicated for preoperative assessment of gallstone disease in patients with hemolytic or sickle cell anemias for planning of concomitant cholecystectomy.

2. Vaccination for encapsulated organisms is an important aspect of managing patients undergoing splenectomy. Pneumococcal vaccine should be administered 2 to 3 weeks prior or 2 weeks after splenectomy *(J Traum.* 2002;53:1037-1042) to allow for adequate immune response. If patient has not had H influenza type B vaccine or meningococcal

vaccine this should also be administered (if older than 2 years of age). Influenza vaccine is recommended annually for asplenic patients as it increases susceptibility to bacterial infections.

3. Transfusions*

a. Patients with hematologic disease, particularly those with autoimmune disorders, often have autoantibodies and are difficult to crossmatch. Thus, blood should be typed and screened at least 24 hours prior to the scheduled operative time. Patients with splenomegaly should have 2 to 4 units of packed RBCs cross-matched and available for surgery.

b. Patients with severe thrombocytopenia (particularly those with counts <10,000/µL) should have platelets available for transfusion, but these should be withheld until the splenic artery is ligated so they will not be quickly consumed by the spleen. Most patients with thrombocytopenia from ITP can undergo splenectomy safely without platelet transfusion even in the setting of very low platelet counts.

4. Other considerations

a. Perioperative stressÑdose steroids treatment should be considered for patients receiving steroids preoperatively and should be continued orally postoperatively and tapered gradually once a hematologic response to splenectomy has occurred.

b. Patients who are to undergo a laparoscopic splenectomy should be counseled preoperatively about the possibility of conversion to open splenectomy or a hand-assisted approach and should be prepared identically to those patients for whom an open procedure is planned.

E. Open and Laparoscopic Splenectomy

1. Open splenectomy

a. The incision used is either an upper midline or a left subcostal incision. When significant splenomegaly is present, a midline incision is usually preferred. A drain is not routinely required unless it is suspected that the pancreatic tail may have been injured during the hilar dissection.

2. Laparoscopic splenectomy has been shown to be safe and effective under most conditions and is the preferred method for elective splenectomy. Contraindications for a laparoscopic approach

are listed in Table 23-3.

a. Splenomegaly increases the complexity of the laparoscopic approach because of the difficulty of manipulating the organ atraumatically and achieving adequate exposure of the ligaments and hilum. Large spleens are also more difficult to place in an entrapment bag using a strictly laparoscopic approach. Although the size limits for attempting laparoscopic or laparoscopic-assisted splenectomy are evolving, most moderately enlarged spleens (<1,000 g weight or 15 to 20 cm in length) can be removed in a minimally invasive fashion, often without a hand-port device. For spleens larger than 20 cm in longitudinal length or those that weigh between 1,000 and 3,000 g, the use of a hand port should be considered. The use of a hand port

in this setting has been associated with reduced operative times, less blood loss, and lower rates of conversion to open operation *(Arch Surg.* 2006;141:755-761). In general, massive splenomegaly (spleens greater than 30 cm in craniocaudal length and weighing >3,000 g) should be approached in an open fashion because of the reduced working space and increased difficultly in manipulating the spleen. A search for accessory splenic tissue should always be conducted, particularly if the patient has a hematologic indication for splenectomy.

TABLE 23-3 Contraindications for Laparoscopic Splenectomy

Absolute Contraindications	Relative Contraindication
Massive splenomegaly (>30 cm)	Moderate splenomegaly (20-25 cm)
Portal hypertension	Severe, uncorrectable cytopenia
Splenic trauma (unstable patient)	Splenic vein thrombosis
Ñ	Splenic trauma (stable patient)
Ñ	Bulky hilar adenopathy
Ñ	Morbid obesity

b. Outcomes of laparoscopic splenectomy. Several large series of laparoscopic splenectomy have

been published with excellent results. In a meta-analysis of 51 reports including 2,940 patients, laparoscopic splenectomy was associated with significantly fewer complications overall, primarily as a result of fewer wound and pulmonary complications *(Surgery.* 2003;134:647-653).

F. Complications

1. Intraoperative

a. Hemorrhage is the most common intraoperative complication of splenectomy, which can occur during the hilar dissection or from a capsular tear during retraction. The incidence of this complication is 2% to 3% during open splenectomy but is nearly 5% using the laparoscopic approach. Bleeding during laparoscopic splenectomy may necessitate conversion to a hand-assisted or open procedure.

b. Pancreatic injury occurs in 0% to 6% of splenectomies, whether done open or laparoscopically. A retrospective review of one center's experience with laparoscopic splenectomy found pancreatic injury in 16% of patients; half of these were isolated instances of hyperamylasemia *(J Surg.* 1996;172(5):596-599). If one suspects that the pancreatic parenchyma has been violated during laparoscopic splenectomy, a closed suction drain should be placed adjacent to the

pancreas, and a drain amylase obtained prior to removal after the patient is eating a regular diet.

c. Bowel injury

(1) Colonic injuries are rare but because of the close proximity of the splenic flexure to the lower pole of the spleen, it is possible to injure the colon during mobilization. Mechanical bowel preparation is not indicated preoperatively.

(2) Gastric injuries can occur by direct trauma or can result from thermal injury during division of the short gastric vessels. Use of energy devices too close to the greater curvature of the stomach can result in a delayed gastric necrosis and perforation.

(3) Diaphragmatic injury has been described during the mobilization of the superior pole, especially with perisplenitis, and is of no consequence if recognized and repaired. In laparoscopic splenectomies, it may be more difficult to recognize the injury given the pneumoperitoneum, but careful dissection of the splenophrenic ligament can minimize its occurrence. The pleural space should be evacuated under positive-pressure ventilation prior to closure to minimize the pneumothorax.

2. Postoperative complications

a. Early

(1) Pulmonary complications develop in nearly 10% of patients after open splenectomy, and these range from atelectasis to pneumonia and pleural effusion. Pulmonary complications are significantly less common with the laparoscopic approach *(Surgery.* 2003;134:647-653).

(2) Subphrenic abscess occurs in 2% to 3% of patients after open splenectomy but is uncommon after laparoscopic splenectomy (0.7%). Treatment usually consists of percutaneous drainage and the intravenous antibiotics.

(3) Wound problems such as hematomas, seromas, and wound infections are common after open splenectomy (4% to 5%). Splenectomy utilizing minimally invasive techniques is associated with wound complications that are usually minor (hematoma, seroma) and less frequent (1% to 2%).

(4) Thrombocytosis and thrombotic complications can occur after either open or laparoscopic splenectomy. The presumed causes of thrombosis after splenectomy may relate to the occurrence of thrombocytosis, alterations in platelet function, and a low-flow stasis phenomenon in the ligated splenic vein. As a result, splenomegaly is a major risk factor for splenic/portal vein thrombosis. Symptomatic portal vein thrombosis occurs more commonly than expected (8% to 12.5%) and can result in extensive mesenteric thrombosis if not recognized promptly and treated expeditiously *(Surg Endosc.* 2004;18:1140-1143). Symptoms of portal vein thrombosis may be subtle and include abdominal pain and low-grade fever. Massive splenomegaly and myelofibrosis are the two main risk factors for portal vein thrombosis *(Ann Surg.*

2005;5:745-746). All patients undergoing splenectomy should be considered for thrombolytic prophylaxis with low-molecularweight heparin or suitable alternative.

(5) Ileus can occur after open splenectomy, but a prolonged postoperative ileus should prompt the surgeon to search for concomitant problems such as a subphrenic abscess or portal vein thrombosis.

b. Late

(1) Overwhelming postsplenectomy infection (OPSI) is an uncommon complication of splenectomy that may occur at any point in an asplenic or hyposplenic patient's lifetime. The risk of overwhelming infection is very small with an estimated mortality of 0.73 per 1,000 patient years *(Ann Intern Med.* 1995;122:187-188). Patients present with nonspecific flu-like symptoms rapidly progressing to fulminant sepsis, consumptive coagulopathy, bacteremia, and ultimately death within 12 to 48 hours. Encapsulated bacteria, especially *Streptococcus pneumoniae, Haemophilus influenzae* type B, and *Neisseria meningitidis,* are the most commonly involved organisms. Successful treatment of OPSI requires early supportive care and high-dose third-generation cephalosporins. OPSI appears to have a higher incidence in children, particularly below the age of 5. Daily prophylactic antibiotics (oral penicillin) have been recommended after operation in all children younger than 5 years and in immunocompromised patients because these patients are unlikely to produce adequate antibody in response to pneumococcal vaccination. 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.

(2) Splenosis is the presence of disseminated intraabdominal splenic tissue, which usually occurs

after splenic rupture. Splenosis does not appear to be more common after laparoscopic splenectomy, but care should be taken during splenic morcellation to avoid bag rupture and spillage of splenic tissue.

CHAPTER 23: SPLEEN

Multiple Choice Questions

1. Which of the following concerning thrombotic thrombocytopenic purpura (TTP) is true?

a. Rituximab is standard first-line treatment.

b. Splenectomy is limited to patients who do not respond to medical management.

c. Plasmapheresis improves survival compared with plasma infusions.

d. It is associated with severe deficiency of ADAMTS-13.

e. Results in a hemolytic anemia with a positive Coombs test.

View Answer

2. Splenic abscesses:

a. Abdominal CT reveals a hyperechoic lesion that intensifies with contrast

b. Fungal abscesses mandate operative intervention

c. Percutaneous drainage is contraindicated

d. Are most commonly due to seeding from distant site of infection

e. Are predominately caused by Gram-negative rods

View Answer

3. Which of the following is true regarding overwhelming postsplenectomy sepsis?

a. It is highest in patients who have undergone splenectomy for trauma.

b. It is most commonly due to *H. influenzae.*

c. Treatment should include the empiric use of an anti-fungal agent.

d. May be prevented with the use of prophylactic antibiotics in selected patients.

e. Most commonly occurs several years after splenectomy.

View Answer

4. The most common cause of elective splenectomy is:

a. Hodgkin lymphoma

b. Thrombotic thrombocytopenic purpura

c. Sickle cell anemia

d. Idiopathic thrombocytopenic purpura

e. Hereditary spherocytosis

View Answer

5. A 25-year-old female presents with incidental finding of a proximal 2 cm splenic artery aneurysm. Which of the following therapies would be most appropriate?

a. Conservative management with routine surveillance

b. Aneurysm exclusion and in situ reconstruction with vein graft

c. Aneurysm exclusion and in situ reconstruction with PTFE

d. Resection with splenectomy

e. Proximal and distal ligation of the splenic artery

View Answer

6. A 55-year-old female who underwent splenectomy 7 days ago for myelofibrosis and massive splenomegaly presents with abdominal pain, fever, and WBC of 17,000. CT of the abdomen reveals a small amount of pneumatosis in the small bowel and ascites.

The most likely etiology is:

a. Nonocclusive mesenteric ischemia

b. Portal vein thrombus

c. Perforated viscus

d. SMA occlusion

e. *Clostridium difficile* colitis

View Answer

7. The most common site of an accessory spleen is:

a. Splenorenal ligament

b. Mesentery of the small bowel

c. Bifurcation of the aorta

d. Gastrohepatic ligament

e. Splenic hilum

View Answer

8. Optimal timing of vaccination for pneumococcal vaccination in adult undergoing elective splenectomy is:

- **a.** 14 days before surgery
- **b.** 7 days before surgery
- **c.** At time of surgery
- **d.** 7 days postoperatively
- **e.** 14 days postoperatively

View Answer

9. Laparoscopy compared with open splenectomy has increased risk for which of the following?

- **a.** Hemorrhage
- **b.** Bowel injury
- **c.** Pneumonia
- **d.** Wound infection
- **e.** Subphrenic abscess

View Answer

10. Vaccination of a patient undergoing splenectomy should include:

- **a.** Pneumococcal vaccine
- **b.** *H. influenzae* vaccine
- **c.** Meningococcal vaccine
- **d.** Influenza vaccine
- **e.** All of the above

View Answer

24
Colon and Rectum

Jonathan B. Mitchem

Steven R. Hunt

I. DISORDERS OF COLONIC PHYSIOLOGY

A. Normal Colonic Physiology. The primary function of the colon is to act as the final arbiter of bowel fluid and sodium resorption, as well as to provide a means for moving stool and coordinate defecation. The colon normally resorbs ÷1.5 L of fluid per day, but can reabsorb up to 5 to 6 L if necessary, primarily via passive means. Sodium and chloride are also conserved by active transport in exchange for potassium and bicarbonate. The colon does participate in digestion via fermentation of complex carbohydrates, producing short chain fatty acids **(SCFA)** which are primarily used locally to provide nutrition for colonic epithelial cells. Normal colon motility is characterized by **segmental contractions** that act to mix stool and **mass movements** that occur three to four times per day and act to move stool through the colon.

B. The diagnosis of **constipation** is made using the Rome criteria. It must include two of the following, and not meet criteria for irritable bowel syndrome (IBS):

- Straining during at least 25% of defecations.
- Lumpy or hard stools in at least 25% of defecations.
- Sensation of incomplete evacuation for at least 25% of defecations.
- Sensation of anorectal obstruction/blockage for at least 25% of defecations.
- Manual maneuvers to facilitate at least 25% of defecations (e.g., digital evacuation, support of the pelvic floor).
- Fewer than three defecations per week.

1. Etiologies of constipation include medications (narcotics, anticholinergics, antidepressants, and calcium channel blockers), chronic laxative abuse, hypothyroidism, hypercalcemia, dietary factors (low fluid or fiber intake), inactivity, and neurologic disorders (e.g., Parkinson disease and multiple sclerosis). Symptoms of constipation may also be caused by obstruction secondary to disorders such as stricture (Crohn disease [CD], diverticulitis, rectal cancer), pelvic floor dysfunction, and rectal prolapse, as well as intrinsic disorders of the colonic myenteric plexus (colonic inertia, Chagas disease, Hirschsprung disease).

2. Evaluation. The initial evaluation of constipation should include a complete history and physical, including a digital rectal examination (DRE). The initial diagnostic workup includes laboratory evaluation to look for metabolic or endocrine causes, and either a **contrast enema** or a **full colonoscopic examination** to rule out structural causes.

Provided these tests are negative, patients are given a trial of high-fiber (25 to 30 g/day) diet and increased fluid intake; if this is not sufficient to resolve the problem, the next step is a **colonic transit study.** Patients continue high-fiber diet and ingest a capsule containing 24 radiopaque markers and abdominal x-rays are obtained on days 3 and 5 after ingestion. Normal transit results in 80% of the rings in the left colon by day 3 and 80% of all the rings expelled by day 5. The persistence of >5 rings throughout the colon on day 5 indicates colonic inertia. When the rings stall in the rectosigmoid region, functional anorectal obstruction (obstructed defecation) may be present and warrants further evaluation.

3. Treatment of colonic inertia initially includes increased water intake, osmotic laxatives, fiber, exercise, and avoidance of predisposing factors. In patients with debilitating symptoms refractory to nonoperative measures, **total abdominal colectomy (TAC) with ileorectal anastomosis (IRA)** may prove curative. The risk of total intestinal inertia after surgery is significant, and the patient should understand this.

C. Colonic pseudo-obstruction (Ogilvie syndrome) is a profound colonic ileus without mechanical obstruction. This most

commonly occurs in critically ill or institutionalized patients, and lack of mechanical obstruction must be confirmed via imaging studies or colonoscopy. Initial management in patients without evidence of peritonitis or perforation consists of nasogastric decompression, bowel rest, correction of systemic contributing factors (i.e., shock, heart failure, metabolic derangements), and discontinuation of medications that decrease colonic motility (including narcotics). If these conservative measures are not sufficient after 24 to 48 hours, neostigmine should be considered. Neostigmine is not a benign medication and should only be given in a monitored setting as it may cause significant bradyarrhythmia. If patients are not candidates for or have failed neostigmine, colonoscopic decompression should be considered. Patients with evidence of perforation, peritonitis, or prolonged distension unresponsive to therapy should undergo **total colectomy with end ileostomy (EI)** unless the patient's comorbid conditions preclude operative intervention.

D. Volvulus accounts for nearly 10% to 15% of colonic obstruction in the United States.

1. Sigmoid volvulus accounts for ÷60% of all cases and is most common in the elderly or institutionalized, as well as patients with 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. **Abdominal x-ray** may show a characteristic **inverted-U,** or **Òbent inner tube sign.Ó** If the diagnosis is still in question, **water soluble contrast enema** or **computed tomography (CT)** may be obtained. Contrast enema may show a **bird's beak deformity** at the obstructed rectosigmoid junction and CT may show a characteristic **Òswirl sign.Ó**

b. Treatment involves decompression via flexible or rigid **sigmoidoscopy** and placement of a rectal tube for decompression. After

decompressive sigmoidoscopy, elective sigmoid colectomy should be undertaken as the risk of recurrence is as high as 40% and emergent surgery is associated with higher mortality than elective surgery. If peritonitis is present, the patient should undergo exploration and **Hartmann procedure** (sigmoid colectomy, end-descending colostomy, blind rectal stump).

2. Cecal volvulus accounts for ÷30% of colonic volvulus, occurs in a younger population than sigmoid volvulus, and is likely due to congenital failure of appropriate cecal tethering. Cecal volvulus occurs as either a true axially rotated volvulus (90%) or antero-superior folding in Òcecal basculeÓ (10%).

a. Diagnosis. Presentation is similar to that of distal small-bowel obstruction, with nausea, vomiting, abdominal pain, and distention. **Abdominal x-ray** may show a **coffee bean-shaped,** air-filled cecum extending into the left upper quadrant. Water soluble enema may be performed, but CT scan is a more commonly utilized imaging modality and generally more useful in the undifferentiated patient with abdominal pain.

b. Management involves urgent laparotomy and ileocolectomy with either primary anastomosis or ileostomy. Cecopexy alone has an unacceptably high rate of recurrence and colonoscopic decompression has limited utility.

3. Transverse and splenic flexure volvulus are extremely rare with clinical presentation similar to that of sigmoid volvulus. Diagnosis is made based on the results of abdominal x-ray and contrast enema or CT. Operative resection is usually required.

E. Diverticular Disease

1. General considerations. Colonic diverticula are an outpouching of the colonic mucosa and submucosa through interruptions in the muscular layer associated with the small arteries supplying the mucosa. Formation is related to high colonic intraluminal pressures and associated with a low-fiber diet. The **incidence increases with age** to a 75% prevalence after the age of 80 years.

2. Complications

a. Diverticulitis develops in 10% to 20% of patients with diverticulosis.

(1) Patients most commonly present with abdominal pain. There is the potential for constipation or diarrhea, fevers, and dysuria. Pneumaturia or fecaluria may indicate a colovesicular fistula. Colovaginal fistula may be indicated by expulsion of gas or feces from the vagina.

(2) Evaluation and staging in the acute setting is done using CT scan. Colonoscopy and barium or water-soluble enemas are not recommended in the acute setting.

(3) Treatment is tailored to severity.

(a) Simple diverticulitis may involve fever and/or leukocytosis, but is **localized** and nonperforated. This can often be treated as an outpatient with oral antibiotics, clear liquids, and followup.

TABLE 24-1 Hinchey Classification

Grade	Description	Treatment
I	Localized pericolonic abscess	Conservative management with antibiotics, bowel rest, and monitoring. Can be treated as outpatient in stable, reliable patients.
II	Pelvic abscess	Bowel rest, IV antibiotics, monitoring, imageguided drainage, possible surgical intervention
III	Purulent peritonitis	Bowel rest, IV antibiotics, surgery
IV	Fecal peritonitis	Bowel rest, IV antibiotics, surgery

(b) Complicated diverticulitis involves evidence of perforation and is generally classified using the Hinchey classification which helps to guide treatment (Table 24-1).

(4) Radiologic guided **percutaneous drainage** may be indicated in patients with localized abscess and lack of diffuse peritonitis.

(5) Surgical intervention for complicated diverticulitis can often be avoided in patients with localized abscess using percutaneous drainage. In patients with diffuse peritonitis, surgical intervention is generally required and usually involves Hartmann procedure. In selected circumstances (stable patients with minimal contamination), resection and primary anastomosis can be considered.

(6) Elective resection for diverticulitis usually consists of a sigmoid colectomy. The proximal resection margin is through uninflamed, nonthickened bowel, but there is no need to resect all diverticula in the colon. The distal margin extends to normal, pliable rectum, even if this means dissection beyond the anterior peritoneal reflection. It is important that patients undergo a complete colonoscopic evaluation of the colon prior to elective resection to rule out malignancy.

b. Fistulization secondary to diverticulitis may occur between the colon and other organs, including the bladder, vagina, small intestine, and skin. Diverticulitis is the most common etiology of colovesical fistulas. Colovaginal and colovesical fistulas usually occur in women who have 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. Fistula takedown is usually undertaken at the index operation with resection and primary closure of the bladder or vagina, but may require flap closure depending on complexity.

F. Lower Gastrointestinal Bleeding (LGIB). LGIB is generally self-limited; however, up to 25% of patients may require

surgical intervention. The most common causes of LGIB are diverticulosis (30% to 35%), hemorrhoids (20%), colorectal polyps (13%), colorectal cancer (9%), intestinal ischemia (6.6%), and angiodysplasia (6%).

1. The **management** of LGIB in the acute setting varies by the volume of bleeding. Patients with a small amount of bleeding can be worked up as an outpatient. Patients may, however, present with hemodynamic instability to the emergency department. **Massive LGIB** is defined as any patient who requires >2 U of red blood cells in a 24-hour period. In the unstable patient, principles of resuscitation should be followed including the ABCs and ensuring the patient has adequate access for resuscitation (see **Chapter 7**, **Critical Care)**.

2. Once hemodynamic stability has been assured or resuscitation has been initiated, it is important to discern the cause of bleeding. The **workup** of LGIB involves the use of multiple different imaging and diagnostic modalities.

a. As always, the history and physical is key to discerning the source of bleeding. **Hematochezia** is more likely to come from an LGI source whereas **melena** may originate from an UGI or small bowel (SB) source. Recent weight loss or history of anemia may point to a chronic process, such as cancer or inflammatory bowel disease (IBD). **Stigmata of cirrhosis** may be evident. Rectal examination should be performed in all patients as this may point out an obvious source such as hemorrhoids, rectal mass, or fissure. An NGT should be placed to determine an obvious UGI source.

b. Laboratory studies include a coagulation profile, basic metabolic profile, hepatic function panel, and complete blood count. This will indicate the degree of anemia and coagulopathy. Hepatic function may point toward liver dysfunction and the serum creatinine whether the patient has renal failure.

c. Diagnosing the source of hemorrhage is key, as this will help to tailor therapy and is important in the event the patient may require surgical intervention.

(1) Endoscopy: EGD should be considered in any patient with massive LGIB or melena if an UGI source has not already been ruled out. **Colonoscopy** can be both diagnostic and therapeutic. Actively bleeding lesions may be injected with dilute epinephrine solution for vasoconstriction, cauterized or clipped. In stable patients who have no evidence of bleeding on EGD or colonoscopy with persistent transfusion requirement, **capsule endoscopy** or **SB ÒpushÓ enteroscopy** should be considered.

(2) Nuclear scan using technetium-99m sulfur colloid or tagged RBCs can identify bleeding sources with rates as low as 0.1 to 0.5 mL/minute. Tagged RBC scan can identify bleeding up to 24 hours after isotope injection, but does not definitively identify the anatomic source of bleeding.

(3) Mesenteric angiography should be performed in the patient with a positive nuclear medicine bleeding scan to identify the anatomic source of bleeding. This may be diagnostic and therapeutic. Angiography can localize bleeding exceeding 1 mL/minute and allows therapeutic vasopressin infusion (0.2 unit/minute) or embolization, which together are successful in 85% of cases.

(4) In the rare **patient who continues to bleed with an unidentifiable source,** diagnostic laparoscopy or laparotomy with intraoperative endoscopy can be considered.

II. COLITIDES

A. IBD is an umbrella term that traditionally covers ulcerative colitis (UC), CD, and Òindeterminate colitis.Ó The exact etiology of IBD is as yet unclear, but there is clearly both an environmental and genetic component. Extraintestinal manifestations can be associated with both UC and CD and include primary sclerosing cholangitis (÷3%), pyoderma gangrenosum, erythema nodosum, iritis/uveitis (2% to 8%), and stomatitis. In addition, patients with IBD have an increased risk of thrombosis including portal and mesenteric venous thrombosis, as well as deep venous thrombosis (DVT) and pulmonary embolus (PE).

1. UC is an inflammatory process of the colonic mucosa. There is a slight male predominance. The disease **always involves the rectum and extends continuously for a variable distance proximally.** Patients can present with bloody diarrhea, tenesmus, abdominal pain, fever, and weight loss. 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.

Cancer must be considered in any colonic stricture in a patient with UC. The risk of colon cancer is increased in patients with UC, but is related to length of disease with risk increasing significantly after 20 years, approaching almost 10% after that duration.

a. Diagnosis is made primarily by colonoscopy with biopsy and by the constellation of symptoms. Imaging studies can help to determine if the patient has SB disease or fistulae indicative of CD.

b. Medical management revolves around therapy which decreases colonic inflammation. Patients with distal disease (proctitis) often respond to topical 5-aminosalicylic acid derivatives (5-ASA) in the form of enemas or suppositories. For those with more proximal disease, oral 5-ASA or sulfasalazine (SSZ) will induce remission in the majority of patients with mild or moderate disease. Patients unresponsive to topical and/or oral 5-ASA and SSZ can then be treated with oral corticosteroids and transitioned back to 5-ASA or SSZ. Intravenous corticosteroids are given to those that are unresponsive oral corticosteroids or are systemically ill with severe

colitis. Azathioprine (AZA) and 6-mercaptopurine (6-MP) have been shown to help wean patients off steroids and can be used as maintenance therapy. Biologic therapy with TNF-α inhibitors has been shown to decrease colectomy rates in studies with short-term followup. Long-term data will be forthcoming as experience with these medications increases.

c. Surgery is indicated in patients who have a high risk of malignancy; disease refractory to medical therapy; and cannot be weaned from steroids, toxic colitis, or intractable bleeding. In the acutely ill patient the operation of choice is **TAC with EI.** These patients, once stabilized and healthy, can be considered for restorative proctocolectomy with ileal pouch anal anastomosis (IPAA) and diverting loop ileostomy (DLI). This is considered a **three-stage approach.** In patients who are subacutely ill or stable, a **two-stage approach** can be considered, consisting of total proctocolectomy (TPC) and IPAA with DLI at the index operation followed by takedown of DLI at a later date. Anticipated function after restorative proctocolectomy with IPAA is approximately six to eight bowel movements a day often with the aid of bulking agents (>50%). Additional complications to consider are impaired continence, sexual dysfunction/infertility, pouchitis, and bowel obstruction. Despite the risks, 95% of patients are satisfied with the procedure and have a good quality of life after IPAA *(Dis Colon Rectum.* 2003;46(11):1489-1491). The S-pouch or W-pouch are other options for restoration of continuity that have utility in specific situations and are done in some specialized centers. Restoration is contraindicated in patients with poor precolectomy continence. In addition, older patients and the obese have worse outcomes with restoration. IPAA should be approached with caution in patients where CD is a concern.

2. CD is a transmural inflammatory process that can affect any area of the GI tract, from the mouth to the anus. It has a female predominance. The disease has a segmental distribution, with **normal mucosa interspersed between areas of diseased bowel.** Common symptoms include diarrhea, abdominal pain, nausea and vomiting, weight loss, and fever. There can be an abdominal mass or perianal fistulas on physical examination. The **terminal ileum** is involved in up to 45% of patients at presentation. Common pathologic changes include fissures, fistulae, 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 the disease progresses, the bowel lumen narrows, and obstruction or perforation may result. SB CD is discussed in **Chapter 19** and perianal CD is discussed in **Chapter 25**.

a. Diagnosis is made using colonoscopy, imaging, and the clinical picture. Unfortunately, patients with Crohn colitis (CC) will often present similarly to patients with UC and up to one-third of patients with CC or UC will be diagnosed incorrectly prior to operative intervention. Based on the clinical picture, CC can be discerned from UC by the presence of perianal disease, Òskip lesions,Ó ileal

inflammation on colonoscopy, and the presence of SB involvement on imaging (SBFT, CT, or MRI/MRE).

b. Medical management revolves around the use of immune suppression. **In the acute setting, sepsis should be controlled by drainage of abscesses and immune suppression.** After the initial control of patients with oral or IV steroids, patients are weaned using immunomodulators as listed above for UC. In addition, **budesonide,** a topical corticosteroid administered orally without systemic absorption, can be administered. Biologic therapy using **TNF-α inhibitors infliximab, certolizumab, and adalimumab** has been shown to decrease steroids **and prolong surgical intervention** in **CD.**

c. Surgical intervention is indicated in patients with medically refractory disease, acute systemic sepsis/perforation, uncontrolled hemorrhage, failure to thrive/malnutrition, and dysplasia/malignancy. Patients with CD with segmental disease should be considered for limited resection. Colectomy with IRA can be entertained for patients with colitis and rectal sparing with limited perianal disease. In the setting of total proctocolitis, patients will likely require TPC with EI. In some centers, TPC with IPAA is considered for isolated CC with no perianal disease. While stricturoplasty has a role in the treatment of SB CD, stricturoplasty plays no role in the treatment of CC, as there is a 7% risk of malignancy over 20 years.

3. ÒIndeterminate colitisÓ is a term used for cases in which the pathologic pattern does not fall clearly into one or the other of the aforementioned patterns (10% to 15% of patients with IBD). The indeterminacy can be due either to inadequate tissue biopsy or to a truly indeterminate form of disease. Typically, surgical therapy for these patients is approached similarly to UC, although they may have a slightly higher rate of pouch complications than patients with UC.

B. Ischemic colitis may result from many low-flow states, including venous or arterial thrombosis, embolization, iatrogenic inferior mesenteric artery (IMA) ligation after abdominal aortic aneurysm repair, and vasculopathy. It is **idiopathic** in the majority of patients. Patients are usually elderly and present with lower abdominal pain localizing to the left and melena or hematochezia. Contrast enema may show **thumbprinting** that corresponds to submucosal hemorrhage and edema. Diagnosis depends on the appearance of the mucosa on colonoscopy. This disease is present most frequently at the watershed areas of the splenic flexure and sigmoid colon. In the presence of full-thickness necrosis or peritonitis, emergent resection with diversion is recommended. Patients without peritonitis or free air but with fever or an elevated white blood cell (WBC) count may be treated with bowel rest, close observation, and intravenous antibiotics. Up to 50% of patients develop focal colonic strictures eventually.

C. Radiation proctocolitis results from pelvic irradiation for the treatment of various malignancies. Risk factors include a dose of greater than 6,000 cGy, vascular disease, diabetes mellitus, hypertension, prior low anterior resection, 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, and is associated with tenesmus and hematochezia with bowel thickening and fibrosis. Ulceration with bleeding, stricture, and fistula formation may occur. Medical treatment may be successful in mild cases, with the use of stool softeners, steroid enemas, and topical 5-aminosalicylic acid products. If these measures fail, transanal application of formalin 4% to affected mucosa may be efficacious in patients with transfusion-dependent rectal bleeding. Patients with stricture or fistula require proctoscopy and biopsy to rule out locally recurrent disease or primary neoplasm. 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 fistulae.

D. Infectious Colitis

1. Pseudomembranous colitis is an acute diarrheal illness resulting from toxins produced by overgrowth of *Clostridium difficile* after antibiotic treatment (especially the use of clindamycin, ampicillin, or cephalosporins). Antibiotics already have been discontinued in one-fourth of cases, and symptoms can occur up to 6 weeks after even a single dose. **Diagnosis** is made by detection of **toxin A** in one of at least three stool samples or stool culture if toxin A is not found but symptoms are present. Proctoscopy demonstrates sloughing colonic mucosa or pseudomembranes, and CT often shows transmural colonic thickening. **Treatment** begins with stopping unnecessary antibiotics and starting oral or intravenous metronidazole. Oral **(not intravenous)** vancomycin is an alternative expensive therapy. For severe cases in patients unable to take oral medications, vancomycin enemas (500 mg in 250 mL saline) may be useful. Rarely, pseudomembranous colitis presents with severe sepsis and colonic distention with **toxic megacolon** or **perforation.** Emergency laparotomy with total colectomy and end-ileostomy is required.

2. Other causes of colitis include bacteria *(E. coli, Shigella),* **amoebic colitis, CMV colitis, and actinomycosis;** however, these conditions are rarely encountered. Typically they are diagnosed by fecal testing or culture and treatment is dictated based on these results. Actinomycosis is treated with appropriate antibiotic therapy, CMV colitis is treated with ganciclovir, and amoebic colitis is treated with oral flagyl.

3. Neutropenic 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.

Initial treatment includes bowel rest, total parenteral nutrition, granulocyte colony-stimulating factor (G-CSF), and broad-spectrum intravenous antibiotics. Laparotomy with total colectomy and ileostomy is required only if peritonitis develops.

III. NEOPLASTIC DISEASE

A. Colorectal neoplasms are typically diagnosed either by **screening** or symptomatic presentation: Hematochezia, melena, anemia, abdominal

pain, and constipation. Initiation and frequency of screening is recommended by most major societies and is outlined in Table 24-2 *(CA Cancer J Clin.* 2008; 58(3):130-160). Colonoscopy is the gold standard screening test and has been shown to prevent cancer. The US Preventive Services Task Force does not recommend screening patients over 75 years of age and recommends against screening patients over 85 years of age based on risk-benefit analysis. While complications are rare, there are risks associated with colonoscopy including perforation (0.04%), bleeding (0.1%), and mortality (0.2%).

B. Polyps

1. Nonadenomatous polyps

a. Hamartomatous polyps make up less than 1% of all polyps diagnosed in adults and may be associated with several rare diseases including **Peutz-Jeghers syndrome, PTEN hamartoma tumor syndrome (PHTS), multiple endocrine neoplasia 2B, familial juvenile polyposis syndrome (JPS),** and **neurofibromatosis type 1 (NF1).** Hamartomatous polyps of the colon are typically either juvenile type or Peutz-Jeghers type, have only rare malignant potential, are pedunculated and >1 cm in size. Isolated colonic hamartomas typically present in the sigmoid colon or rectum with bleeding and/or polyp prolapse, but can present with anemia, diarrhea, obstruction, or mucoid stools. Treatment of hamartomas is via endoscopic resection, but if they are too large, segmental colectomy is considered.

b. Hyperplastic polyps are the most common colorectal neoplasm (10 times more common than adenomas) and have an extremely limited malignant potential. Most are less than 0.5 cm in diameter, are found in the distal colon, and rarely need treatment. Right-sided lesions or lesions >1 cm should be removed and may be a marker of increased risk of adenoma.

2. Adenomas are dysplastic lesions with the ability to progress to malignancy and are thought to be the precursor of most colorectal cancers. Risk of invasive malignancy is higher in villous adenomas than tubular; however all adenomas are treated with endoscopic removal. The risk of malignancy increases with size. Sessile polyps have a higher malignant risk than pedunculated polyps. If a polyp is too large for endoscopic removal, segmental colectomy should be considered.

a. Tubular adenomas are usually pedunculated and account for roughly 85% of adenomas and can contain up to 25% villous elements.

b. Tubulovillous adenomas account for 10% to 15% of adenomas and contain 25% to 50% villous features.

c. Villous adenomas are usually sessile and account for 5% to 10% of adenomas. They contain predominantly villous architecture.

3. Malignant polyps are those polyps that contain foci of malignancy and are considered T1 colorectal cancers. The most important factor in the treatment of malignant polyps is the level of invasion typically classified using the Haggitt (Table 24-3) and Kudo classifications (Table 24-4).

TABLE 24-2 Screening Recommendations Based on Patient Risk

Risk	Description	Modality	Age at Initiation
Average (75% of	Sporadic	1. Colonoscopy every 10 yrs 2. Flexible sigmoidoscopy every 5	50

newly diagnosed colorectal cancer)		yrs 3. Double contrast enema every 5 yrs 4. High sensitivity fecal occult blood test yearly 5. Fecal immunochemical test yearly 6. Stool DNA unclear interval	
Family History (15-20%)	One first-degree relative with adenomatous polyps (AP) or CRC or two second-degree relatives with CRC	Colonoscopy every 5 yrs	40 or 10 yrs prior to youngest relative's diagnosis
Hereditary Nonpolyposis colorectal cancer (HNPCC, 3-8%)	Genetic or clinical diagnosis of HNPCC or individuals with high risk of HNPCC	Colonoscopy every 1-2 yrs and consideration of genetic counseling	20 to 25 or 10 yrs prior to youngest relative's diagnosis
Familial Adenomatous Polyposis (FAP, 1%)	Genetic diagnosis of FAP or suspected FAP without diagnosis	Flexible sigmoidoscopy every year and counseling regarding genetic testing. If genetic testing positive, strong consideration for surgery	10 to 12
Inflammatory Bowel Disease (1%)	Chronic Crohn colitis or ulcerative colitis	Colonoscopy with random biopsies for dysplasia every 1-2 yrs	Risk of cancer is significant 8 yrs after the diagnosis of pancolitis and 12-15 yrs after diagnosis of left-sided colitis

TABLE 24-3 Haggitt Classification of Malignant Polyps of the Colon and Rectum

Level	Description	Risk of Lymph Node Metastasis	Treatment
0	Noninvasive, high-grade dysplasia	<1%	Endoscopic removal with ³2 mm margin
I	Focus of invasive cancer in head of pedunculated polyp	<1%	Endoscopic removal with ³2 mm margin

II	Focus of invasive cancer in neck of pedunculated polyp	<1%	Endoscopic removal with ³2 mm margin
III	Focus of invasive cancer in stalk of pedunculated polyp	<1%	Endoscopic removal with ³2 mm margin
IV	Focus of invasive cancer in base of pedunculated polyp; **all sessile polyps**	up to 25%	**See Kudo classification**

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These classification systems importantly help to stratify the risk of lymph node metastasis, therefore, the patient's need for segmental colectomy instead of simple endoscopic removal. The Haggitt classification system classifies the level of invasion related to the polyp stalk. By definition, this makes all sessile polyps Haggitt level 4. The Kudo

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classification separates the submucosa into three levels of depth (SM1 to 3) as it relates to the muscularis propria. In addition, lymphovascular invasion (LVI) and poor differentiation have been shown to increase the likelihood of lymph node metastases. Patients with an inadequate endoscopic resection margin (<2 mm), LVI, SM3 invasion, or poor differentiation should undergo segmental colectomy. Followup for polyps with foci of invasive cancer that do not undergo colectomy involves repeat colonoscopy at 3 months, 6 months, and 1 year to evaluate the site of lesion removal.

TABLE 24-4 Kudo Classification of Submucosal Invasion of Malignant Polyps of the Colon and Rectum

Level	Description	Treatment
SM1	Invasion of the superficial one-third of submucosa	Endoscopic removal with ³2 mm margin; in distal rectum transanal full thickness removal
SM2	Invasion of the middle one-third of submucosa	Endoscopic removal with ³2 mm margin; in distal rectum transanal full thickness removal
SM3	Invasion of the deep onethird of submucosa	Segmental colectomy

TABLE 24-5 Hereditary Colorectal Cancer (CRC) Syndromes

Syndrome	Percentage of Total CRC Burden	Genetic Basis	Phenotype	Extracolonic Manifestations	Treatment	Notes
Familial	<1%	Mutations in	<100	CHRPE,	TPC with	Variants

adenomatous polyposis (FAP)		tumor suppressor gene *APC* (5q21)	adenomatous polyps; near 100% with CRC by age 40 yrs	osteomas, epidermal cysts, periampullary neoplasms	end-ileostomy or IPAA or TAC with IRA and lifelong surveillance	include Turcot (CNS tumors) and Gardener (desmoids) syndromes
Hereditary nonpolyposis colorectal cancer (HNPCC)	5-7%	Defective mismatch repair: *MSH2* and *MLH1* (90%), *MSH6* (10%)	Few polyps, predominantly rightsided CRC, 80% lifetime risk of CRC	At risk for uterine, ovarian, small intestinal, pancreatic malignancies	Genetic counseling; consider prophylactic resections, including TAH/BSO	High microsatellite instability (MSI-H) tumors, better prognosis than sporadic CRC
Peutz-Jeghers (PJS)	<1%	Loss of tumor suppressor gene *LKB1*/STK11 (19p13)	Hamartomas throughout GI tract	Mucocutaneous pigmentation, risk for pancreatic cancer	Surveillance EGD and colonoscopy q3yr; resect polyps >1.5 cm	Majority present with SBO due to intussuscepting polyp
Familial juvenile polyposis (FJP)	<1%	Mutated *SMAD4/DPC* (18q21)	Hamartomas throughout GI tract; >3 juvenile polyps; 15% with CRC by age 35 yrs	Gastric, duodenal, and pancreatic neoplasms; pulmonary AVMs	Genetic counseling; consider prophylactic TAC with IRA for diffuse disease	Presents with rectal bleeding or diarrhea

AVM, arteriovenous malformation; CHRPE, congenital hypertrophy of retinal pigmented epithelium; CNS, central nervous system; EGD, esophagogastroduodenoscopy; GI, gastrointestinal; IPAA, ileal pouch-anal anastomosis; IRA, ileal-rectal anastomosis; TAC, total abdominal colectomy; TAH/BSO, total abdominal hysterectomy and bilateral salpingo-oophorectomy; TPC, total proctocolectomy.

a. Malignant polyps of the proximal two-thirds of the rectum can be treated as colon polyps; however, there is some controversy regarding the treatment of malignant polyps of the distal one-third of the rectum as these lesions may have an increased risk of lymph node metastasis. **All T1 lesions of the distal rectum should be approached with at**

least transanal full thickness excision using traditional transanal excision, Transanal Endoscopic Microsurgery (TEM) or Transanal Minimally Invasive Surgery (TAMIS) techniques.

C. Colon Cancer

1. There are approximately 150,000 new diagnoses of colorectal cancer each year, of which 70% to 75% are colon cancer. Colorectal cancer is the fourth leading cause of cancer death worldwide and about one-third of patients diagnosed with colorectal cancer will eventually die of their disease. See Table 24-5 for hereditary colorectal cancer syndromes.

2. The **clinical presentation** of colon cancer is most commonly asymptomatic and diagnosed during screening, highlighting the importance of appropriate screening. The most common presenting symptoms are abdominal pain, hematochezia, change in bowel habits, or anemia.

Right-sided lesions more commonly present with asymptomatic anemia and abdominal pain, whereas l**eft-sided** lesions more often cause changes in bowel habits, rectal bleeding, and crampy abdominal pain associated with defecation. Obstruction, weight loss, and perforation, which are often markers of advanced disease are less frequently encountered today due to effective screening programs.

3. Diagnosis and staging

a. As indicated above, the majority of patients are **diagnosed** after the biopsy of a mass or polyp removed on colonoscopy. After the diagnosis is made, every effort should be made to ensure that the remainder of the colon is free of lesions. In the acute setting in patients who are severely ill from obstruction or perforation, a complete colonoscopy can be undertaken after patients have recovered within 3 to 6 months and prior to initiating adjuvant treatment if warranted.

b. Standard staging studies include chest x-ray and abdominal CT scan to evaluate the lung and liver, the most common sites of metastasis. **Routine PET/CT** has no proven benefit at this time. **MRI** may be useful if there are concerning hepatic lesions on CT. **CEA** should be drawn prior to initiating therapy as this can be used in followup, but does not play a role in diagnosis or staging.

4. Surgical treatment

a. Preoperative preparation is coordinated using a team approach. Most centers use **preoperative oral antibiotic bowel preparation** as this has been shown to significantly decrease wound infections. We routinely administer both mechanical and antibiotic preparation to patients. As a part of our postsurgical recovery, we employ multimodal pain management techniques including preoperative Tylenol and routine epidural placement. Patients who are not taking opioid pain medications also receive alvimopan as this has been shown to decrease length of stay and speeds return of bowel function *(Ann Surg.* 2007;245(3):355-363). If patients do not receive an epidural preoperatively, they receive 40 mg subcutaneous enoxaparin.

b. Colectomy may be approached laparoscopically, open, or robotically. For colonic lesions, this means ensuring an adequate proximal and distal margin, high ligation of the arterial pedicle for lymph node clearance, tension-free anastomosis, and good blood supply to the ensuing anastomosis or stoma. Adequate lymph node retrieval has been established as at least 12 nodes to ensure appropriate staging. The laparoscopic approach to right, left, and sigmoid colon lesions has been established as oncologically equal to open surgery with the benefit of shorter recovery by multiple studies *(Lancet Oncol.* 2009;10(1):44-52). Lesions of the cecum and ascending colon should be resected via right colectomy. Lesions of the descending and sigmoid colon are removed via left colectomy. Transverse colon lesions are typically approached using an extended right colectomy.

c. In the **emergent** setting intraoperative decisions may be necessary regarding appropriate therapy. This may include tumor resection with or without anastomosis or proximal diversion if the tumor is

unresectable. In the case of obstruction, the distal obstructed limb should be vented via loop ostomy or mucus fistula.

5. Colon cancer is staged using the American Joint Committee on Cancer (AJCC) TNM staging which is based on the depth of invasion (T), lymph node status (N), and presence of distant metastases (M) (Table 24-6). Stage I tumors have a 90% 5-year survival. Stage II tumors have a 60% to 80% 5-year survival. Stage III tumors have a 60% 5-year survival. Stage IV tumors have a 5-year survival of 10%. Unfavorable characteristics include poor differentiation, pericolonic tumor deposits, multiple lymph node involvement, mucinous or signet-ring pathology,

venous or perineural invasion, bowel perforation, aneuploid nuclei, and elevated CEA.

TABLE 24-6 TNM Categories for Colorectal Cancer

T	Local tumor spread
T0	No tumor
Tis	Tumor only involves mucosa and has not grown beyond muscularis mucosa
T1	Tumor extends into the submucosa
T2	Tumor extends into muscularis propria
T3	Tumor extends through muscularis propria but not beyond outermost layer of colon
T4	Tumor extends through other organs or structures or penetrates the visceral peritoneum
N	Nodal involvement
N0	No lymph node involvement
N1	Cancer cells in 1-3 nearby lymph nodes
N2	Cancer cells in 4 or more nearby lymph nodes
M	Distant spread
M0	No distant organ spread
M1	Spread to a distant organ or distant set of lymph nodes

6. Adjuvant chemotherapy is currently recommended in patients with stage III and IV colon cancer. Adjuvant therapy is also recommended for patients with stage II who have inadequate lymph node retrieval (<12) or with unfavorable characteristics. Current therapy involves the combination of 5-fluorouracil/leucovorin with either irinotecan (FOLFIRI) or oxaliplatin (FOLFOX). The role of targeted therapy using vascular endothelial growth factor (VEGF) inhibitors (bevacizumab) or epidermal growth factor receptor (EGFR) inhibitors (cetuximab) has not been proven to be of benefit, but is used in stage IV disease.

7. Followup is crucial in the first 2 years after surgery, when 90% of recurrences occur. Surveillance colonoscopy is recommended the first year after resection and then every 3 years until negative, at which time every 5 years is recommended. CEA can be followed, and rising levels should prompt a CT scan of the chest, abdomen, and pelvis with possible colonoscopy if the patient has not had recently.

D. Rectal Cancer

1. The **pathophysiology** of rectal cancer differs from that of colon cancer because of several anatomic factors: (1) Confinement of pelvis and sphincters; (2) proximity to urogenital structures and nerves; (3) dual blood supply and lymphatic drainage; and (4) transanal accessibility. The rectum is defined by the NCI as 12 cm above the anal verge on rigid proctoscopy.

2. Diagnosis and staging of the rectum is done using the AJCC staging as outlined above for colon cancer with additional considerations regarding local staging. DRE can give information on the size, height, fixation, ulceration, local invasion, and lymph node status. Rigid sigmoidoscopy and biopsy are important for precisely measuring the distance to the anal verge and dentate line. **Transrectal ultrasonography** or **rectal protocol magnetic resonance imaging (MRI)** is an integral part of staging rectal tumors to evaluate depth of invasion, the circumferential resection margin (**CRM**), and lymph node status as this will help determine the need for preoperative chemoradiation therapy.

Distant spread is evaluated (as with colon cancer) with abdominal CT and chest x-ray or CT. It is helpful to have a preoperative CEA for patient followup.

3. Neoadjuvant chemoradiation, typically consisting of 5-FU, leucovorin with concomitant radiation therapy (XRT, 54 cGy) is currently standard for all patients with T3 or T4 lesions or node positive disease on imaging (TRUS or MRI) (Fig. 24-1). Radiation therapy improves local control, but does not prolong survival; and preoperative therapy is associated with similar results with significantly less toxicity than postoperative therapy *(Lancet Oncol.* 2011;12(6):575-582).

4. The **goal of surgical therapy** is to remove the cancer with adequate margins, **total mesorectal excision** (TME), lymph node clearance with high ligation of the arterial pedicle (IMA), and consideration of future

continence and urogenital function. Patients with clinical or imaging evidence of sphincter involvement, incontinence, or concern for distal margin should undergo abdominoperineal resection (APR). Bowel preparation is considered similar to colon resection. Possible stoma sites including colostomy and proximal DLI should be marked preoperatively. **Preoperative ureteral stents** should be considered in patients who are at high risk of ureteral injury.

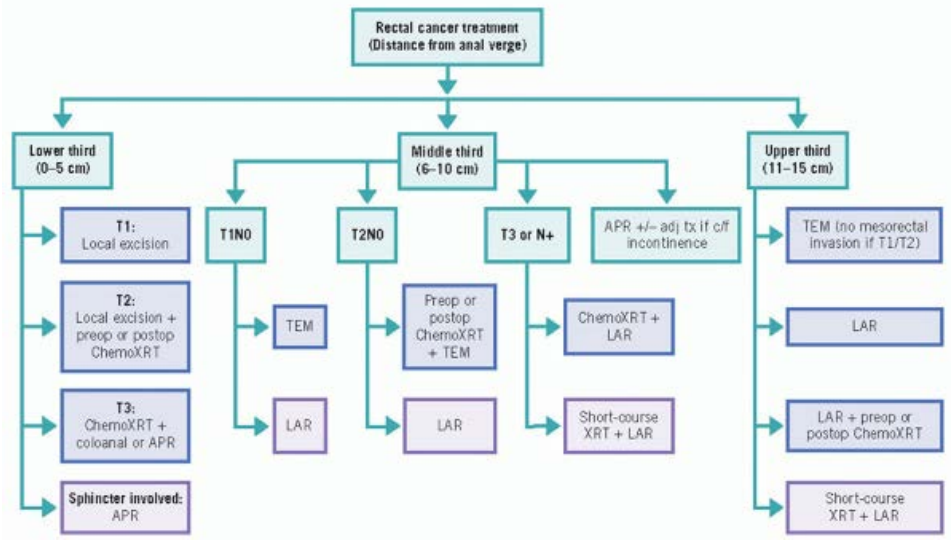

Figure 24-1 Rectal cancer treatment based on location relative to anal verge and stage.

a. As with colectomy, proctectomy can be approached open, laparoscopically, or robotically. Regardless of the approach undertaken, the principles of surgery are the same. The distal margin can be <2 cm in patients with distal tumors to preserve continence; however, it must be ensured that there is a negative margin. Based on current data, any patient with a T2 rectal cancer should undergo radical excision with LAR or APR. Appropriate surgical treatment for T1 rectal cancers is an

area of considerable study at present and includes transanal excision techniques. Rectal cancer with extension into the bladder, sacrum, vagina, or other local pelvic structures can be reliably resected for cure. It is important to remember the anatomic confines and anatomy of the pelvis as infertility, sexual dysfunction, and continence are affected by the parasympathetic and sympathetic nerves and are common complications. In addition, leak is more common for coloproctostomy than colocolostomy, and leak testing in the operating room is recommended. Proximal diversion is recommended for any low or tenuous anastomosis, as leaks can have devastating consequences.

5. Obstructing rectal cancers should be evaluated by hypaque enema and/or colonoscopy in patients without clear signs of peritonitis. Endoluminal stents can be used as a short-term bridge to operative therapy, but should not be used to get patients through preoperative chemoradiation therapy as they have been shown to have a high risk or perforation and complications in this setting. In addition, stents should not be used in patients with mid to low rectal cancers as this can lead to considerable pain and urgency issues.

6. Rectal cancer recurrence typically presents with pain, rectal bleeding, or on followup testing. Diagnosis is confirmed by examination and biopsy. Patients should then be worked up for systemic recurrence including CT and PET-CT. If there is no evidence of systemic recurrence, resection can be considered if patients are fit. Pelvic MRI is useful to evaluate the relationship to other pelvic structures. Preoperative therapy can be considered if patients have not received XRT previously. Curative resection of recurrent rectal cancer can lead to significant long-term survival *(Ann Surg.* 1994;220(4):586-595).

E. Other Colorectal Tumors

1. Lymphoma is most often metastatic to the colorectum, but primary non-Hodgkin colonic lymphoma accounts for 10% of all GI lymphomas. The GI tract is also a common site of non-Hodgkin lymphoma associated with human immunodeficiency virus. The most common

presenting symptoms include abdominal pain, altered bowel habits, weight loss, and hematochezia. Biopsies are often not diagnostic because the lesion is submucosal. Treatment is resection with postoperative chemotherapy. Intestinal bypass, biopsy, and postoperative chemotherapy should be considered for locally advanced tumors.

2. Retrorectal tumors usually present with postural pain and a posterior rectal mass on physical examination and CT scan.

a. The **differential diagnosis** includes congenital, neurogenic, osseous, and inflammatory masses. Chordomas are the most common malignant retrorectal tumor; they typically are slow growing but difficult to resect for cure.

b. Diagnosis is based on CT scan and physical findings. Biopsy should not be performed. Formal resection should be undertaken if there is significant concern for malignancy or symptoms.

3. Carcinoid tumor

a. Colonic carcinoids account 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 length of survival of less than 12 months. These lesions are treated with local excision if small and with formal resection if greater than 2 cm.

b. Rectal carcinoid accounts for 15% of GI carcinoids. As with colonic carcinoids, lesions less than 2 cm in diameter have low malignant potential and can be 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 APR is probably warranted.

IV. INTESTINAL STOMAS

A. Ileostomy creation and care was revolutionized with the description of the eversion technique by **Brooke** in 1952. The small intestine adapts to ileostomy formation within 10 days postoperatively. Average output is 500 mL/day, but may be up to 1,500 mL/day. Volumes above this may be pathologic and/or cause dehydration and electrolyte abnormalities. Stoma construction of either a loop ileostomy or end-ileostomy should be ÒBrookedÓ or everted 2 to 2.5 cm to create an easier stoma to pouch. Stoma creation should be within the rectus abdominis to decrease the risk of peristomal herniation. Preoperative marking of the planned site prevents improper placement near bony prominences, belt/pant lines, abdominal creases, and scars. Reversal of a loop ileostomy is relatively straightforward and rarely requires laparotomy. A side-to-side,

functional end-to-end technique with a GIA stapler is often utilized.

B. Colostomy construction is typically associated with fewer electrolyte and physiologic derangements than ileostomy. Left-sided or sigmoid colostomies are preferred to right-sided or transverse colostomies. Colostomies can be created in either a loop or end-loop configuration. Common

colostomy complications include obstipation, prolapse, and parastomal hernia. Obstipation/constipation can be treated with stoma irrigation and/or hypaque enema, which is diagnostic and therapeutic. Parastomal hernia repair is indicated for the same reasons as other abdominal wall hernias. Colostomy prolapse does not require revision unless there is an inability to reduce the mucosa or obstruction results. End colostomy takedown can be difficult and all patients should undergo full colonoscopic evaluation including the distal defunctionalized colon/rectum to rule out stricture or mass prior to takedown.

CHAPTER 24: COLON AND RECTUM

Multiple Choice Questions

1. Which of the following is part of the Rome criteria for the diagnosis of constipation?

- **a.** Three or fewer bowel movements per week
- **b.** Manual maneuvers to assist with 50% of bowel movement
- **c.** Fulfilling criteria of irritable bowel syndrome
- **d.** Sensation of incomplete evacuation with 100% of bowel movements

View Answer

2. When administering neostigmine to a patient with Ogilvie syndrome, why is it important to ensure the patient is in a monitored setting?

- **a.** Often there is a rapid response causing a large evacuation which can be difficult to manage
- **b.** There is a high risk of hypotension due to vasovagal stimulation related to having a large bowel movement
- **c.** Neostigmine can cause significant bradyarrhythmias potentially requiring cardioversion
- **d.** There is a significant risk of perforation with the administration of neostigmine

View Answer

3. The most important aspect in the care of a patient with LGIB is which of the following?

- **a.** Obtaining early tagged red blood cell scan
- **b.** Ensuring appropriate resuscitation and stabilizing patient
- **c.** Using fecal occult blood test to test for bleeding
- **d.** Placing an NGT to rule out an upper GI source

View Answer

4. Definitive treatment of sigmoid volvulus is accomplished by:

- **a.** Endoscopic decompression
- **b.** Endoscopic decompression and placement of a long rectal tube
- **c.** Sigmoidopexy
- **d.** Sigmoidectomy

View Answer

5. The most common cause of LGIB is:

- **a.** Diverticulosis

b. Colorectal cancer

c. Ischemic colitis

d. Ulcerative colitis

View Answer

6. Hinchey IV diverticulitis demands operative therapy and is characterized by:

a. Pericolonic abscess

b. Fecal peritonitis

c. Generalized peritonitis

d. Pelvic abscess

View Answer

7. An important distinguishing feature of Crohn disease when compared to ulcerative colitis is:

a. The lack of “skip” lesions

b. Response to biologic therapy

c. Perianal disease

d. The presence of pyoderma gangrenosum

View Answer

8. Surgical treatment of medically refractory ulcerative colitis includes:

a. Abdominoperineal resection with end colostomy

b. Total proctocolectomy with ileal-anal anastomosis

c. Segmental colectomy involving the diseased area and colo- or ileo-colostomy

d. Total abdominal colectomy with end ileostomy

View Answer

9. A patient presents to the ED with abdominal pain and hematochezia after endovascular aortic aneurysm repair (EVAAR), how would you confirm your clinical suspicion?

a. Flexible sigmoidoscopy

b. CT scan of the abdomen and pelvis

c. Barium enema

d. Acute abdominal series

View Answer

10. The most common type of colonic polyps diagnosed on endoscopy are:

a. Adenomatous polyps

b. Malignant polyps

c. Hamartomatous polyps

d. Hyperplastic polyps

View Answer

11. The Kudo classification of polyp invasion is important to the treatment of malignant colon and rectal polyps because:

a. The Kudo classification is more sensitive than the Haggitt classification for the diagnosis of

malignancy

b. The Kudo classification accurately predicts who needs adjuvant therapy after resection

c. The Kudo classification predicts the risk of lymph node metastasis and the need for surgical resection

d. The Kudo classification accurately predicts which polyps are technically amenable to endoscopic retrieval

View Answer

12. An asymptomatic patient presents to your office for consultation regarding screening colonoscopy due to the fact that the patient's father was diagnosed with colon cancer. What is the most important factor when considering initiating screening colonoscopy?

a. Recent weight loss

b. Smoking history

c. The age of the patient's father at diagnosis

d. The patient's mother had breast cancer

View Answer

13. On pathologic examination after right colectomy a patient is diagnosed with a T3 tumor with 0 or 9 lymph nodes negative. What do you tell this patient about his or her need for adjuvant therapy?

a. The patient does not need adjuvant therapy because there is only marginal benefit in patients with stage II disease.

b. Adjuvant therapy should be considered because although the patient is stage II, there was inadequate lymph node harvest.

c. The patient should consider not receiving adjuvant therapy because although the patient has stage III disease, they have low-risk stage III disease.

d. The patient should receive adjuvant therapy because there is clearly a benefit for patients with stage III disease.

View Answer

14. To appropriately stage rectal cancer, patients need what imaging studies?

a. Chest x-ray, abdomen CT, pelvic MRI

b. Chest x-ray, abdomen and pelvis CT, PET/CT

c. Abdomen CT, pelvic MRI, PET/CT

d. Chest x-ray, abdomen and pelvis CT, pelvic MRI, PET/CT

View Answer

15. The principles of surgical resection for the treatment of rectal cancer include which of the following?

a. Resection of Denonvilliers fascia to ensure an adequate anterior margin

b. Ensuring an intact and complete total mesorectal excision

c. Resection of the hypogastric nerves along the pelvic sidewall as this is a common site of recurrence

d. Performing an abdominoperineal resection for any patient with a tumor <5 cm from the dentate line due to the dual blood supply of the distal rectum

View Answer

25

Anorectal Disease

Ashley M. Holder

Matthew G. Mutch

I. ANORECTAL PHYSIOLOGY

A. Normal Anorectal Function

1. The **rectum functions as a capacitance organ,** with a reservoir of 650 to 1,200 mL compared to an average daily stool output of 250 to 750 mL.

2. The **anal sphincter mechanism** allows defecation and maintains 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 external anal sphincter contracts in response to sensed rectal contents and relaxes during defecation.

3. Defecation has four components: (1) Mass movement of feces into the rectal vault; (2) rectal Ñanal inhibitory reflex, by which distal rectal distention causes involuntary relaxation of the internal sphincter and the external sphincter contracts (this process is known as sampling and allows for determination of contents as gas, liquid, or solid); (3) voluntary relaxation of the external sphincter mechanism and puborectalis muscle; and (4) increased intraabdominal pressure.

4. Continence requires normal capacitance, normal sensation at the anorectal transition zone, puborectalis function for solid stool, external sphincter function for fine control, and internal sphincter function and hemorrhoidal pillars for resting pressure.

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

1. Etiologies include (1) **mechanical defects,** such as sphincter damage from obstetric trauma, fistulotomy, and scleroderma affecting the external sphincter; (2) **neurogenic defects,** including spinal cord injuries, pudendal nerve injury due to birth trauma or lifelong straining, and systemic neuropathies such as multiple sclerosis; and (3) **stool content-related causes,** such as diarrhea and radiation proctitis.

2. Evaluation includes visual and digital examination observing for gross tone or squeeze abnormalities and determining muscle bulk. **Anal manometry** quantitatively measures parameters of anal function, including resting and squeeze pressure (normal mean >40 and >80 mm Hg, respectively), sphincter length (4 cm in men, 3 cm in women), and minimal sensory volume of the rectum. Pudendal nerve terminal motor latency **(PNTML)** testing and endoanal

ultrasound provide neural and anatomic information.

3. Treatment depends upon the underlying cause. Neurogenic and minor mechanical anal sphincter defects are initially treated using dietary fiber

to increase stool bulk and **biofeedback** to strengthen muscle and improve early sensation. Major defects require **anal sphincter reconstruction,** in which the anatomic sphincter defect is repaired. Sacral nerve stimulation, used in patients with an intact sphincter complex or even if there is less than a 30-degree defect, is emerging as the most durable treatment for fecal incontinence: (1) Patients maintain a journal of their bowel and continence function for 2 weeks; (2) temporary leads are then imbedded in the S2 to S4 nerve roots and the journal maintained for another 2 weeks; and (3) if there is >50% improvement in incontinence episodes, patients are eligible for implantation of the permanent device. Artificial anal sphincters may be used in patients without a reconstructible native anal sphincter or with neurogenic incontinence. However, long-term success rate is complicated by a 60% explantation rate. A palliative diverting colostomy is indicated when all other treatment modalities fail.

C. Obstructed defecation (pelvic floor outlet obstruction) presents with symptoms of chronic constipation, straining with bowel movements, incomplete evacuation of the rectum, pelvic pressure, and the need for perineal pressure to evacuate. Evaluation includes: (1) **Video defecography** to evaluate fixation of the posterior rectum to the sacrum and relaxation of the puborectalis; (2) **anal manometry and surface EMG testing** to assess rectal sensation, ability to expel a balloon, and paradoxical contraction of the external sphincter with straining; and (3) **colonic transit study** to assess colonic motility. Problems associated with obstructive defecation may include **fecal impaction** and **stercoral ulcer** (mucosal ulceration due to pressure necrosis from impacted stool); both are treated with enemas, increased dietary fiber, and stool softeners. Attempts at surgical correction of any of the following conditions without addressing the underlying pathology are doomed to failure.

1. 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, inflammation, and trauma. Initial treatment is anal dilation, although advanced cases are treated with advancement flaps of normal perianal skin.

2. Nonrelaxation of puborectalis results in straining and incomplete evacuation. Colonic transit time reveals outlet obstruction. Persistent puborectalis distortion is seen on defecography. Biofeedback is the treatment of choice.

3. 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 due to failure of the pelvic floor to relax during defecation. Treatment includes bowel regimens with high fiber, suppositories, enemas, and biofeedback.

D. Abnormal rectal fixation leads to internal or external prolapse of the full thickness of the rectum.

1. Internal intussusception (internal rectal prolapse) causes outlet obstruction with mucus discharge, hematochezia, tenesmus, and constipation. The underlying pathophysiology is a nonrelaxing puborectalis and resulting chronic straining. Proctoscopy demonstrates an inflamed, irritated rectal mucosa and a **solitary rectal ulcer** may develop at the lead point of the internal prolapse. **Treatment** consists of a bowel regimen of increased fiber, stool softeners, enemas, glycerin suppositories, and biofeedback to retrain the function of the puborectalis muscle. **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 prominent among the patient's complaints. Chronic ischemia of the solitary rectal ulcer causes entrapment of mucin-producing cells, eventually resulting in **colitis cystica profunda.** Treatment is low anterior resection and rectopexy.

2. External rectal prolapse is protrusion of full-thickness rectum through the anus. Symptoms include pain, bleeding, mucous discharge, and incontinence. Physical examination can distinguish rectal prolapse (concentric mucosal rings) from prolapsing internal hemorrhoids (deep radial grooves with a rosebud appearance). Acute prolapse needs urgent reduction and may be facilitated by applying table sugar to the mucosa to reduce edema; if unsuccessful, the patient will need to be brought to the operating room. **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. In general, abdominal procedures trade higher operative morbidity with lower recurrence rates relative to perineal-only operations. Continence improves in almost all patients, regardless of procedure.

a. Sigmoid resection and rectopexy (FrykmanÑGoldberg procedure) shortens the redundant rectosigmoid colon with posterior sacral fixation. Prolapse recurs in less than 10% of patients following rectopexy with or without resection.

b. Ventral rectopexy is a newer option in which the anterior plane is mobilized, a permanent mesh is secured to the anterior rectal wall at the level the pelvic floor, and then the mesh is anchored to the sacral promontory. Proponents cite lower complication rates, similar recurrence rates, and improved functional outcomes *(Dis Colon Rectum.* 2014;57:1442).

c. Perineal proctectomy (modified Altemeier procedure) is an alternative for patients with severe anal incontinence due to complete eversion and stretch of the anal canal. Recurrence rate is generally around 20%, although lower rates have been reported in retrospective, single-institution studies *(Dis Colon Rectum.* 2006;49:1052).

II. HEMORRHOIDS.

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 of the pelvic floor contribute to the abnormal enlargement of hemorrhoidal tissue. Treatments are based on grading and patient symptoms (Table 25-1); options include the following:

TABLE 25-1 Classification and Treatment of Symptomatic Internal Hemorrhoids

Grade	Description	Treatments
I	Palpable, nonprolapsing enlarged venous cushions	Dietary fiber, stool softeners
II	Prolapse with straining and defecation, spontaneously reduce	Dietary fiber, stool softeners, elastic ligation
III	Protrude spontaneously or with straining, require manual reduction	Dietary fiber, stool softeners, elastic ligation, excisional hemorrhoidectomy, stapled hemorrhoidectomy
IV	Chronically prolapsed and cannot be reduced, often with dentate line released from internal position	Dietary fiber, stool softeners, excisional hemorrhoidectomy, stapled hemorrhoidectomy

A. Medical treatment of first-degree and most second-degree hemorrhoids includes increased dietary fiber and water to increase stool bulk, 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 to 2 cm above the dentate line 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 to 10 days with bleeding occurring at that time. Patients on anticoagulation should have their anticoagulation stopped for a full 7 to 10 days after banding. 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. Patients with this life-threatening disorder should undergo examination under anesthesia, immediate removal of rubber bands, and debridement of any necrotic tissue, accompanied by broad-spectrum intravenous antibiotics. Patients who undergo banding still have a 30% recurrence rate *(Dis Colon Rectum.* 2004;47:1364).

B. 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, often with monitored anesthesia care/sedation and local anesthetic or spinal anesthesia,** and the resulting elliptical defects are completely closed with chromic suture (Ferguson hemorrhoidectomy). Complications include a 10% to 50% incidence of urinary retention, bleeding, infection, sphincter injury, and anal stenosis from taking too much anoderm. Urinary retention, the most common complication, can be minimized by the judicious use of intravenous fluids perioperatively.

C. Stapled hemorrhoidectomy is an alternative to traditional excisional hemorrhoidectomy for large prolapsing, bleeding third-degree hemorrhoids with minimal external disease. 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, ensuring avoidance of vaginal tissue in female patients *(Dis Colon Rectum.* 2004;47:1824). Stapled hemorrhoidectomy results in significantly less perioperative discomfort, but there is a higher recurrence rate following stapled hemorrhoidectomy *(Cochrane Database Syst Rev.* 2006;4:5393).

D. Acutely thrombosed external hemorrhoids are treated by excision of the thrombosed vein 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. The recurrence rate of thrombosed external hemorrhoids was significantly higher with expectant management (25%) than excision (6%) *(Dis Colon Rectum.* 2004;47:1493).

III. ANAL FISSURE.

Anal fissure is a split in the anoderm. Ninety percent of anal fissures occur posteriorly and 10% occur anteriorly; location elsewhere should prompt examination under anesthesia and biopsy. Symptoms include tearing pain with defecation and severe anal spasm that lasts for hours afterward and blood (usually on the toilet paper). Manometry and digital rectal examination demonstrate increased sphincter tone, muscular hypertrophy in the distal one-third of the internal

sphincter, and exaggerated constriction of the internal sphincter muscle associated with the anorectal inhibitory reflex. An external skin tag or Òsentinel pileÓ may also be present. Atypical fissures are those that occur laterally and the differential diagnosis includes Crohn disease, tuberculosis, anal cancer, abscess or fistula, cytomegalovirus, herpes simplex virus, chlamydia, and syphilis. **Ninety percent of patients heal with medical treatment** that includes increased fiber, sitz baths, and topical nifedipine ointment (0.2%) TID. If surgery is required, options include BOTOX therapy or **lateral internal sphincterotomy.** Botox provides a temporary chemical sphincterotomy that lasts for 3 months and offers 60% success versus lateral internal sphincterotomy which is 95% successful. Recurrence and minor incontinence occur in fewer than 10% of patients.

IV. INFECTION OF THE ANORECTUM

A. Anorectal Abscess

1. Cryptoglandular abscess results from infection of the anal glands in the crypts at the dentate line. The initial abscess occurs in the intersphincteric space. Infection then can spread (1) superficial to the external sphincter into the **perianal** space; (2) cephalad in the **intersphincteric plane;** (3) through the external sphincter into the **ischiorectal** space (which in turn may connect posteriorly via the deep postanal space, resulting in a horseshoe abscess); or (4) deep to the external sphincter into the **supralevator** space.

a. Diagnosis usually is obvious, with severe anal pain and a palpable, tender, fluctuant mass. An intersphincteric abscess yields only a painful bulge in the rectal wall and no external manifestations.

b. Treatment is surgical drainage, with the skin incision kept close to the anal verge to avoid the possible creation of a long fistula tract. Intersphincteric abscesses are drained by an internal sphincterotomy over the entire length of the abscess. Draining an intersphincteric abscess externally will result in a supralevator fistula, which are technically challenging to repair. Perianal and ischiorectal abscesses are drained through the perianal skin with a small mushroom-shaped catheter placed to keep the abscess unroofed. Supralevator abscesses, originating from intersphincteric abscesses, should be drained into the rectum. 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 40% of patients develop a chronic fistula. We do not advocate fistulotomy at the initial operation because the abscess will not recur in 60% of cases, the internal opening may not be evident, and a complicated fistulotomy may result in sphincter injury.

2. Fistula-in-ano represents the chronic stage of cryptoglandular abscess but also may be due to trauma, Crohn disease, tuberculosis, cancer, or radiation.

a. Patients present with persistent fecopurulent **perianal drainage** from the external opening of the fistula. The location of the internal opening along the dentate line is approximated by using **Goodsall 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 (Fig. 25-1), and may involve the sphincters in one of four configurations (Fig. 25-2).

b. Treatment depends on the level that the fistula traverses the external sphincter and preexisting sphincter function. Placement of a soft, noncutting seton permits resolution of surrounding inflammation

while preserving sphincter musculature, often acting as a first-stage operation. **Fistulotomy,** dividing the overlying internal sphincter, may be performed for intersphincteric and low transsphincteric fistulas. For, posterior midline fistulas, up to 50% of the sphincter can safely be divided. In women, anterior fistulas should never be treated with a fistulotomy because the risk of incontinence is too high. Therefore, anterior and high transsphincteric fistulas should be treated with sphincter-sparing techniques. **Fibrin glue injection** and **anal fistula plug** initially provided encouraging results, but, over time, the success of these techniques has ranged from 25% to 40%. The **Ligation of Intersphincteric Fistula Tract (LIFT)** procedure has gained acceptance with success rates of 60% to 75%. This procedure requires dissection in the intersphincteric plane, isolation of the fistula tract, and ligation of both sides of the tract. Endorectal advancement flaps for anterior fistulas remain the gold standard with success rates ranging from 70% to 90%.

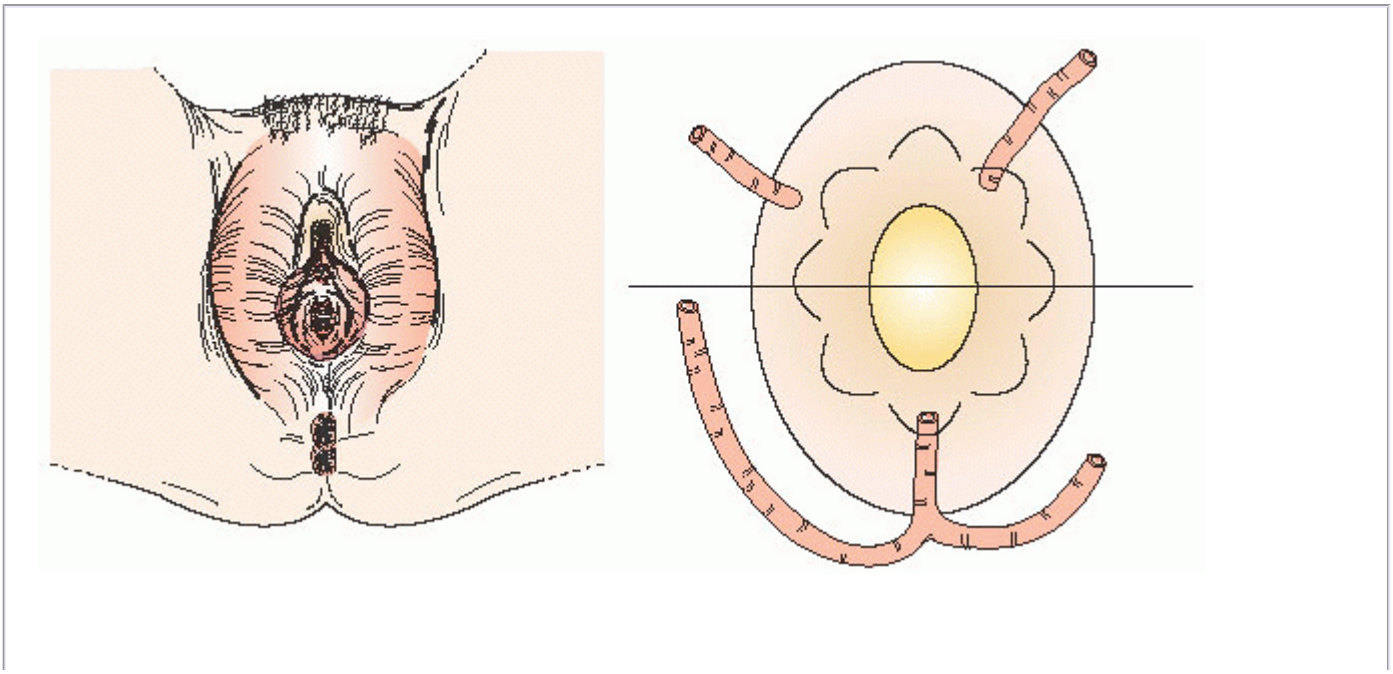

Figure 25-1 Goodsall rule. The anteriorÑposterior location of the external opening of the fistula helps to identify the internal opening of the fistula.

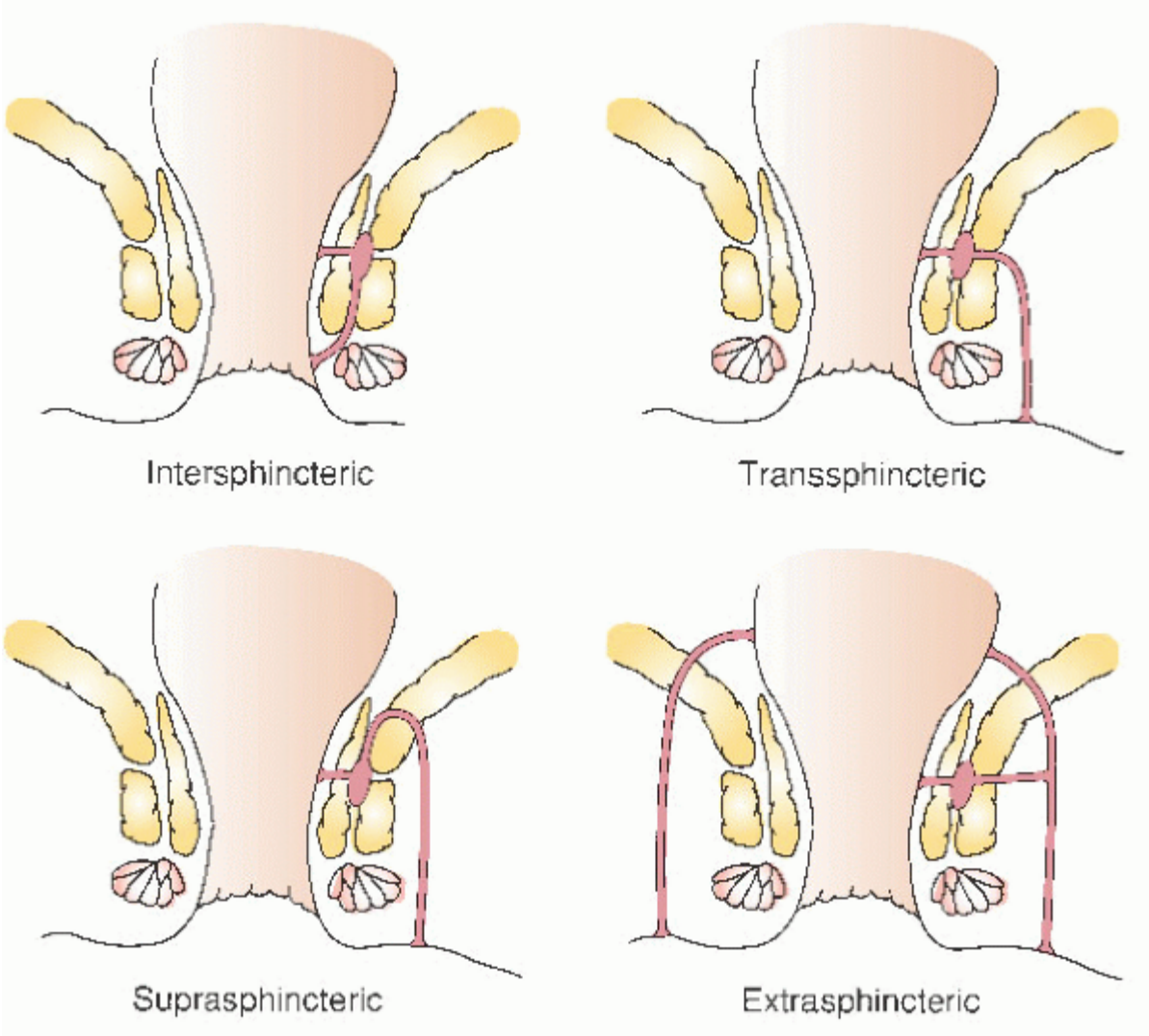

Figure 25-2 The four main anatomic types of fistula. (From Mulholland MW, Lillemoe KD, Doherty GM, et al. *Greenfield's surgery: scientific principles and practice,* 4th ed. New York, NY: Lippincott Williams and Wilkins, 2005, with permission.)

B. Necrotizing anorectal infection (Fournier gangrene) can result in massive, life-threatening tissue destruction. Patients present with systemic toxicity and perianal pain. There may be crepitance 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 debridement of all nonviable tissue and intravenous antibiotics are mandatory. Early treatment is critical, but mortality still approximates 50%. Fecal diversion is

rarely needed as most patients can be managed with a medical colostomy (constipation) and TPN.

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, with allowance for secondary closure when the sinus is 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.

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 and has superficial tracts. The treatment of choice is wide incision of the involved skin.

E. Pruritus ani is a common symptom of hemorrhoids, fissure, rectal prolapse, rectal polyp, anal warts, and intraepithelial dysplasia of squamous or apocrine gland origin. Treatment is directed toward resolution of the underlying cause. Failure to find an underlying cause should prompt investigation of dietary factors (e.g., coffee and alcohol). Children should be

evaluated for pinworms, which, if found, are treated with piperazine citrate. However, as the most common cause is over cleaning, therapy includes a high-fiber diet, minimizing wiping, and avoiding the use of soaps and alcohol-containing products.

F. Condyloma acuminatum is an anorectal and urogenital wart caused by infection with human papilloma virus (HPV). The virus is sexually transmitted and presents with visible perianal growth, often accompanied by pruritus, anal discharge, bleeding, and pain. Common treatments include topical trichloroacetic acid, Aldara (imiquimod), or excision with electrocoagulation under local anesthesia. The role of HPV vaccination is as yet unknown, but vaccination is not effective if administered after infection. Smoke generated by coagulation contains viable organisms and must be completely evacuated. Anal canal warts must be destroyed at the same time as external warts. Biopsies should be obtained looking for highgrade squamous intraepithelial lesions as malignant transformation can occur resulting in squamous carcinoma, requiring treatment according to Nigro protocol.

V. ANAL NEOPLASMS

A. Tumors of the Anal Margin. Any atypical anal lesions should be biopsied to attempt to diagnose these lesions at the earliest stage possible.

1. Squamous cell carcinoma behaves like cutaneous squamous cell carcinoma, is well differentiated and keratinizing, and is treated with wide local excision and chemoradiation if large

or involving the sphincter complex.

2. Basal cell carcinoma is a rare, male-predominant cancer that is treated with local excision.

3. High-grade squamous intraepithelial lesions (also known as Bowen disease) are becoming common in HIV-positive patients and also are more frequently seen in other immunosuppressed patients, such as solid organ transplant recipients. Local excision or destruction of identified lesions during high resolution anal mapping with 9% acetic acid can prevent progression to cancer. Long-term surveillance is required.

4. Paget disease is an intraepithelial adenocarcinoma, most commonly discovered in elderly patients. Paget disease begins as a benign in situ neoplasm, thought to originate from apocrine cells, which presents as a pruritic, erythematous rash that mimics eczema or psoriasis. Biopsy provides diagnosis; however, any patient with suspected Paget disease should also undergo colonoscopic evaluation to ensure that the biopsy results are not due to the inferior spread of a rectal signet-ring cell cancer (up to 50% of Paget disease have a coexisting visceral carcinoma requiring an abdominal perineal resection [APR]). Treatment of noninvasive disease is wide local excision of all lesions.

B. Anal Canal Tumors

1. Epidermoid carcinoma is nonkeratinizing and derives from the anal canal 6 to 12 mm above the dentate line.

a. Epidermoid cancer 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% to 40% are metastatic at the time of diagnosis. Preoperative staging requires CT of the abdomen and pelvis and PET scan to rule out metastatic disease.

b. Treatment involves chemoradiation according to the **Nigro protocol:** 3,000-cGy external-beam radiation, mitomycin C, and 5-fluorouracil. Surgical treatment is reserved for locally persistent or recurrent disease only. The procedure of choice is abdominoperineal resection; perineal wound complications are frequent.

2. Adenocarcinoma is usually an extension of a low rectal cancer but may arise from anal glands and has a poor prognosis.

3. Melanoma accounts for 1% to 3% of anal cancers and is more common 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, although the 5-year survival rate is less than 20%.

CHAPTER 25: ANORECTAL DISEASE

Multiple Choice Questions

1. A 41-year-old woman has a feeling of fullness after defecation and bright red blood on the toilet paper, especially after straining. Physical examination reveals two large, pink nonreducible columns of mucosa protruding from her anal canal. Which is the most likely complication following surgical treatment of her anorectal disease?

a. Sphincter injury

b. Bleeding

c. Infection

d. Urinary retention

e. Anal stenosis

View Answer

2. A 68-year-old man has perianal mucus and pain. Physical examination reveals a fistula. On examination under anesthesia, you discover the fistula crosses the internal and external anal sphincters. Which is the most appropriate treatment at this time?

a. Fistulotomy using electrocautery over entire fistula tract

b. Division of the internal sphincter using electrocautery and placement of seton encircling the external sphincter

c. Diverting colostomy

d. Antibiotics only

e. Anal advancement flap

View Answer

3. The goal of distal transection beyond gross tumor in rectal cancer is:

a. 2 cm because there is no correlation between local recurrence and the extent of distal margin when it is greater than 2 cm.

b. 5 cm because 30% of patients have microscopic evidence of intramural spread beyond 3 cm from the palpable tumor.

c. 4 cm as local recurrence rates correlate with distal resection margins less than 4 cm.

d. Just beyond gross tumor (<5 mm).

e. 3 cm because only 30% of patients have microscopic evidence of intramural spread beyond that.

View Answer

4. Which of the following is correct regarding the anal sphincter mechanism?

a. The internal sphincter is innervated by the autonomic nervous system; thus it is subject to voluntary control.

b. The increase in pressure during voluntary contraction (squeeze pressure) is due to the external sphincter.

c. The external sphincter accounts for the majority of resting pressure.

d. Distal rectal distention causes involuntary relaxation of the external sphincter.

e. The rectal-anal inhibitory reflex refers to the external sphincter.

View Answer

5. A 54-year-old woman has anal pain and blood on the toilet tissue after defecation. Physical examination reveals a 2 × 2 cm ulcer within the anal canal. Biopsy of the ulcer returns as squamous cell carcinoma. Which of the following is the appropriate treatment?

a. Low anterior resection with diverting ileostomy

b. Abdominoperineal resection

c. Primary chemoradiation therapy

d. Wide local excision, skin grafting, permanent colostomy

e. Wide local excision and primary closure

View Answer

6. A 75-year-old man has a perianal pruritic, erythematous rash. Biopsy reveals Paget disease. Which of the following is correct regarding Paget disease?

a. Regardless of level of invasion, all patients require abdominoperineal resection.

b. Since Paget disease is an intraepithelial neoplasm, patients do not require colonoscopy.

c. Extramammary Paget disease is most common in 20- to 30-year-old women.

d. Fulguration with laser or electrocautery is the most effective treatment.

e. Wide local excision is the most appropriate treatment in noninvasive disease.

View Answer

7. A 26-year-old man has severe anal pain during and after bowel movements for the past 6 weeks. Physical examination reveals a split in the anoderm in the posterior midline. Which of the following is correct regarding anal fissures?

a. Lateral sphincterotomy is only effective in 30% to 40% of cases.

b. Fissurectomy is the mainstay of treatment.

c. Ninety percent of patients heal with medical treatment, including fiber, sitz baths, and topical nifedipine ointment.

d. An equal number of fissures occur posteriorly as anteriorly.

e. The anal fissure triad consists of internal hemorrhoid, sentinel skin tag, and fissure.

View Answer

26
Cerebrovascular Disease

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CEREBROVASCULAR DISEASE

Atherosclerotic occlusive disease of the extracranial carotid artery is a major risk factor for stroke, the primary cause of disability, and the fifth most common cause of death in the United States. Nearly 800,000 new strokes occur annually, with an estimated total cost of more than $40 billion. The initial mortality from stroke is approximately 30%. Among those who survive the initial stroke event, 60% suffer long-term disability, and 40% recover with mild or no deficits.

PRESENTATION

The clinical presentation of patients with symptomatic occlusive disease is a **neurologic deficit.** However, many patients have asymptomatic stenosis that is identified by a healthcare provider based on auscultation of carotid bruits or screening duplex ultrasound. **Lateralizing ischemic events** can result in aphasia (expressive 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 [ICA] and its branches).

Transient ischemic attacks (TIAs) are transient hemispheric neurologic deficits that may last from several seconds to hours, but no longer than 24 hours. TIAs that occur in rapid succession, interspersed with complete recovery, but with progressively smaller intervals between attacks, are termed **crescendo TIAs,** and carry a high risk of progression to a permanent neurologic deficit; emergent evaluation is mandatory. **Amaurosis fugax** (temporary monocular blindness)Ñoften described as a shade coming down over one eyeÑresults from atheroemboli lodging in the ophthalmic artery. Fundoscopic examination demonstrates Hollenhorst plaques.

If the neurologic deficit persists beyond 24 hours, it is considered a **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 similar to crescendo TIAs, demands immediate attention. Unlike crescendo TIAs, symptoms of stroke in evolution do not return to baseline. **Global ischemic events** are manifested by symptoms such as vertigo, dizziness, perioral numbness, ataxia, or drop attacks. These are usually associated with interruption of the posterior circulation supplying the brain stem

(i.e., the vertebrobasilar system).

PATHOPHYSIOLOGY

Atherosclerotic disease of the carotid artery can cause stroke or TIA by three mechanisms:

A. Atheroembolization of debris originating from the carotid artery plaque and traveling to the brain

B. Thrombotic Occlusion of a Severe Stenosis

C. Global Cerebral Hypoperfusion (Rare). The major site of formation of atherosclerotic plaque is the carotid bulb, which can be attributed to dynamic flow changes and wall stress. A “vulnerable” plaque refers to an unstable plaque that is prone to embolization, thrombosis, and subsequent stroke. **Carotid duplex ultrasound features predictive of a vulnerable plaque include hypoechoic and heterogeneous plaques, and those with increased plaque area.** These plaque features confer a three- to fourfold increased risk of stroke.

DIAGNOSTIC EVALUATION

In the evaluation of a patient suspected of having cerebrovascular disease, a careful history and physical examination is performed before obtaining any diagnostic studies. The patient or patient advocate is asked to describe the acuity and duration of symptoms, as well as the resolution of those symptoms if they are no longer present. Risk factors for TIA and stroke should be sought, including age greater than 55 years; male gender, African-American or Hispanic race, presence of hypertension, diabetes, atrial fibrillation, hypercholesterolemia, morbid obesity, and renal insufficiency; family history of stroke, and tobacco or alcohol consumption.

Physical examination of the patient with cerebrovascular disease should begin with a review of the patient's vital signs, with particular attention paid to the blood pressure, heart rate, and rhythm (atrial arrhythmias). A focused neurologic assessment should be conducted, with observation of the patient's alertness and orientation, analysis of any speech deficits (i.e., dysarthria or aphasia), detection of facial asymmetry, and assessment of cranial nerves. Motor and sensory deficits should be tested for, as should any visual field deficits. Palpation and auscultation of carotid pulses is essential, as the presence of a carotid bruit or absence of pulsation warrants diagnostic evaluation. **The absence of a bruit does not exclude the presence of carotid stenosis.**

Any patient with neurologic symptoms suggestive of acute stroke or TIA requires immediate brain imaging. Noncontrast brain computed tomography is often adequate to rule out cerebral hemorrhage. Further imaging of the carotid arterial system is useful for determining prognosis,

and can be employed to classify the degree of stenosis. Because of methodologic differences in calculating the percentage of stenosis encountered in different studies, there is some disagreement about exact cutoff percentages. **However, four levels of stenosis are typically described: Mild (<50%), moderate (50% to** *79%),* **severe (80% to 99%), and occluded (100%).** For consideration of optimal treatment strategies, a variety of noninvasive and invasive diagnostic studies are available:

A. Color-flow duplex scanning uses real-time B-mode ultrasound and color-enhanced pulsed Doppler flow measurements to determine the extent of the carotid stenosis. This is the initial screening test for carotid disease. When using an accredited vascular laboratory, carotid endarterectomy (CEA) can be undertaken on the basis of ultrasound duplex scanning alone in up to 95% of cases. However, the vascular laboratory must have clear and validated criteria for measuring carotid stenosis, well-trained technologists, and must provide information about plaque morphology and the extent of distal ICA involvement.

B. Conventional catheter angiography is the gold standard for imaging the extracranial and intracranial circulation in cerebrovascular disease. Unlike duplex scanning, angiography is an invasive procedure with inherent risks, such as contrast allergy, renal toxicity, and stroke (less than 1% of patients). Because of these risks, and improvements in duplex ultrasonography, carotid angiography is generally limited to patients with technically inadequate duplex ultrasonography, patients felt to be candidates for carotid artery stenting (CAS), patients in whom carotid occlusion must be verified, or patients with discordant noninvasive study findings.

C. Magnetic resonance angiography (MRA) and **computed tomographic angiography (CTA)** are reliable means of supplementary imaging, but remain inferior to conventional angiography. Both have advantages of being noninvasive, with the former allowing for the avoidance of radiation exposure. Limitations of MRA include poor sensitivity and specificity for diagnosing 50% to 69% stenoses, the presence of flow-related artifacts, and a tendency to overestimate stenosis severity. However, image quality can be improved after administration of gadolinium. CTA can be performed with excellent three-dimensional reconstructions, but carries the risk of contrast-induced nephropathy. For patients with complete occlusion and contralateral high-grade occlusion, results of duplex ultrasound are often confirmed by CTA, or occasionally by angiography. CTA has the advantage of being fast, inexpensive compared to MRA, and offering exceptional spatial resolution and visualization of soft tissues, bone, and vessels. It also is useful to define the extracranial and intracranial vascular anatomy, and the extent of vessel calcification, particularly in the aortic arch.

MANAGEMENT

A. Best Medical Therapy. It is important to make every effort to modify risk factors to prevent progression of carotid occlusive disease. Control of hypertension and hyperglycemia, cessation of smoking, management of lipid disorders, attainment of ideal body weight, and regular exercise

should be undertaken, and are the cornerstones of medical therapy. The components of best medical therapy are summarized in Table 26-1. 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. In a meta-analysis of more than 8,000 patients, the risk of major vascular events was reduced by 22% in patients receiving aspirin *(Br Med J.* 1988;296:320). Low doses (81 mg/day) are as efficacious as higher doses (325 mg/day). Clopidogrel (Plavix) is a potent antiplatelet agent, but it has not been evaluated as part of medical therapy compared to CEA. Anticoagulation with heparin is beneficial in patients who have cardiac emboli. In addition, heparin may be useful in preventing progression of thrombus in evolving nonhemorrhagic strokes. The major contraindication to heparinization is a recent hemorrhagic brain infarct; therefore, a CT scan of the brain should be obtained before heparin is given.

TABLE 26-1 Summary of Effect of Best Medical Therapy

Best Medical Therapy for Prevention of Stroke and Cardiovascular Events

Treatment	Target	Evidence
Antiplatelet therapy	1. Either single or dual antiplatelet therapy acceptable 2. Aspirin 81 to 325 mg/day is recommended 3. Plavix 75 mg equivalent to aspirin 4. Ticlopidine 250 mg twice daily similar to aspirin in effectiveness	Reduces both stroke risk and overall cardiovascular morbidity
Antihypertensive therapy	1. Decrease systolic blood pressure by 10 mm Hg	Reduces stroke recurrence Reduce blood pressure 24 hrs after acute stroke

	2. Decrease diastolic blood pressure by 5 mm Hg 3. Target blood pressure of 120/80 mm Hg in hypertensive patients	No definitive benefit of one class of antihypertensive agents over another
Diabetes mellitus	Target HgbA1c < 7	Reduce overall stroke rate No definitive benefit of tight control (i.e., HgbA1c < 6)
Smoking cessation	Total abstinence	Reduces risk of stroke and major adverse cardiovascular events
Statin therapy	1. Reduce LDL by 50% 2. Target LD < 70 mg/dL	Reduces risk of stroke and major adverse cardiovascular events, particularly among patients with a history of cardiovascular disease
Alcohol	Avoid excessive consumption	

B. Carotid Endarterectomy. Surgical management is the treatment of choice for extracranial carotid artery disease, and has been documented to reduce stroke rates. Indications for CEA have been extensively studied in both asymptomatic and symptomatic patients, comparing surgical treatment to best medical therapy. Table 26-2 provides results of four

randomized controlled trials comparing CEA and best medical therapy. Among these trials, the two US trials include the Asymptomatic Carotid Atherosclerosis Study (ACAS) and the North American Symptomatic Carotid Endarterectomy Trial (NASCET). In the ACAS trial, the ipsilateral *5-year* stroke rate in asymptomatic patients with at least 60% stenosis was 5.1% in patients undergoing a CEA versus 11% receiving best medical therapy. For symptomatic patients with at

least 70% stenosis in the NASCET trial, the **2-year** ipsilateral stroke rate was 9% versus 26% in the CEA and best medical therapy groups, respectively. Based largely on these two definitive trials, current indications for CEA include the following:

TABLE 26-2 Summary of Randomized Trials Comparing CEA and Best Medical Therapy

Results of Randomized Trials of CEA versus BMT

Study	Population	Stroke Rate for BMT	Stroke Rate for CEA+BMT	Conclusions
NASCET[a]	Symptomatic patients with carotid stenosis ³70%	26% (2-yr)	9% (2-yr)	CEA is beneficial for symptomatic patients with ³70% stenosis ($p < 0.001$)
NASCET[b]	Symptomatic patients with carotid stenosis 50-69%	22% (2-yr)	15 (5-yr)	CEA is beneficial for symptomatic patients with ³50% stenosis ($p = 0.045$)
ECST[c]	Symptomatic patients with carotid stenosis 80-99% (60-99% by NASCET criteria)	20% (3-yr)	7% (3-yr)	CEA is beneficial for symptomatic patients with ³60% stenosis by NASCET criteria ($p < .001$)
ACAS[d]	Asymptomatic patients with carotid stenosis ³60%	11% (5-yr)	5% (5-yr)	CEA is beneficial for asymptomatic patients with ³ 60% carotid

				stenosis (p = .004)
ACST[e]	Asymptomatic patients with carotid stenosis ³60%	12% (5-yr)	6% (5-yr)	CEA is beneficial for asymptomatic patients with ³60% carotid stenosis (p < 0.001)

[a] NASCET, North American Symptomatic Carotid Endarterectomy Trial, *N Engl J Med.* 1991;325:445-453.

[b] NASCET, North American Symptomatic Carotid Endarterectomy Trial, *N Engl J Med.* 1998;339:1415-1425.

[c] ECST, European Carotid Surgery Trial, *Lancet.* 1998;351:1379-1387.

[d] ACAS, Asymptomatic Carotid Atherosclerosis Study, *JAMA.* 1995;273:1421-1428.

[e] ACST, Asymptomatic Carotid Surgery Trial, *Lancet.* 2004;363:1491-1502.

1. Asymptomatic patients with greater than 60% stenosis.

2. Symptomatic patients with greater than 50% stenosis.

3. Symptomatic patients with greater than 50% stenosis who have an **ulcerated lesion** or whose **symptoms persist** while they are on **aspirin** or other antiplatelet therapy.

4. Selected patients with stroke in evolution. Surgery 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, though most experts favor intervention within 2 weeks.

5. Selected patients with completed strokes. Interventions in these patients are performed in the hope of reducing stroke recurrence, which is 7% to 8% per year with nonsurgical therapy. Candidates for surgery include patients with a mild deficit and (1) greater than 70% stenosis or (2) greater than 50% stenosis and an ulcerated plaque, and patients with a moderate deficit and a lesion greater than 70% with an occluded contralateral carotid artery. The timing of surgery in these cases is debatable; however, surgery traditionally has been delayed to reduce the risk of perioperative hemorrhagic stroke. A prudent approach is to wait 2 weeks postinfarction to minimize the risk of intracranial hemorrhage.

6. Rarely, surgery is performed on patients with **completely occluded carotid arteries.** Candidates for surgery include those who have had: (1) Recent endarterectomy with immediate postoperative thrombosis; (2) recent occlusion with fluctuating or progressive symptoms; and (3) new internal carotid occlusion that can be operated on within 2 to 4 hours of the onset of symptoms. **There is no role for surgical revascularization in patients with** asymptomatic **ICA occlusion.**

Best practice guidelines merit that CEA should be performed with perioperative adverse event rates (stroke and death) below 3% for asymptomatic patients (6% for symptomatic patients). Any specialist or general surgeon performing this procedure must achieve similar (or better) perioperative outcomes. Anesthesia for CEA consists of 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 demonstrated superiority.

The technique of CEA relies on meticulous technique and a thorough understanding of the cervical anatomy. Prophylactic antibiotics should be administered. An arterial line is placed for continuous blood pressure monitoring; vasoactive agents should be readily available to control hypertensive and hypotensive states.

After induction, the patient is placed in the semi-Fowler position, with the head of the table slightly elevated in the reverse Trendelenburg position and the knees slightly flexed. A rolled up towel or sandbag is placed between the shoulders to extend the neck, and the arms are tucked at the sides of the patient. The head is turned away from the side that is being operated, and the endotracheal tube is taped to the corner of the mouth opposite the surgical field. The neck and ipsilateral earlobe are prepped, as well as the upper thorax, to allow for a sternotomy if exposure and control of the supra-aortic vessels is warranted, either under planned or emergent circumstances.

An oblique longitudinal incision centered over the carotid bifurcation is preferred, and is made along the anterior border of the sternocleidomastoid muscle. Alternatively, a transverse incision in the skin crease can be used. The skin, subcutaneous tissues, and platysma muscle are divided. The deep cervical fascia is then divided in the plane of the incision, and a self-retaining retractor is placed in the incision to reflect the sternocleidomastoid muscle posteriorly. The common facial vein is identified, ligated, and divided to expose the underlying carotid sheath. The carotid sheath is opened longitudinally, and the internal jugular vein is mobilized. The vein is then reflected laterally by repositioning the retractor. The common carotid artery and both the internal and external carotid arteries are exposed using a combination of sharp and blunt dissection. Care must be taken to identify and preserve the hypoglossal and vagus nerves. The ansa hypoglossi can usually be found anterior to the internal jugular vein. If necessary, it can be transected to facilitate exposure of the carotid artery or it can be followed cephalad to identify the hypoglossal nerve. During exposure 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. The carotid sinus area can be infiltrated with 1% lidocaine to prevent hypotension and bradycardia. Vessel loops are carefully passed around the common, internal, and external carotid arteries. Systemic heparinization of the patient is established with an intravenous bolus of 5,000 units of heparin (or approximately 80 units per patient kilogram). After 3 minutes, the internal, common, and external carotid arteries are clamped sequentially in that order to avoid embolization to the intracranial circulation.

A longitudinal arteriotomy is made on the anterior wall of the common carotid artery from a segment just proximal to the plaque, and extended into the ICA to a point a few millimeters distal to the plaque with right-angle Potts scissors. The use of a shunt is controversial, and is described later. If employed, the shunt is first inserted into the ICA, and blood is allowed to backflow to fill the shunt. The proximal end of

the shunt is then placed into the common carotid artery, and cerebral blood flow restored.

Endarterectomy begins with the plaque being separated from the media in an appropriate plane with a spatula. The proximal extent of the plaque is transected with fine scissors, and the endarterectomy is continued distally. The plaque is first dissected from the external carotid artery using an eversion technique and the use of a fine hemostat clamp. In the ICA, the plaque typically feathers to the end. If intimal flaps are present, they are carefully tacked down with U-shaped 6-0 polypropylene monofilament sutures. The artery is then flushed with heparinized saline, and the artery is typically closed with a patch using a running 5-0 or 6-0 suture. Common patch materials include Dacron, polytetrafluoroethylene (Gore-Tex), bovine pericardium, or autogenous vein. Just before the suture line is completed, the shunt is removed, and the clamps are removed sequentially, allowing for flushing of debris and air from the carotid artery. The clamps are first removed from the external, then the common, and finally the internal carotid arteries. Blood flow is confirmed with a Doppler probe, although other groups have published on the role of intraoperative angiography and duplex as completion studies. Hemostasis from the suture line can be achieved with placement of hemostatic agents; areas of vigorous bleeding are controlled with figure-of-eight sutures using 6-0 polypropylene. Protamine sulfate can be administered to reverse heparinization if needed (typically 0.5Ñ1 mg of protamine per 100 units of heparin).

Closure of the incision begins by reapproximating the sternocleidomastoid muscle and cervical fascia with interrupted 3-0 absorbable sutures. Through a separate stab incision, a 7-mm Jackson ÑPratt drain is placed below the platysma.

A long-standing debate has been the use of intravascular shunts. Several large series have demonstrated excellent results of CEA without shunts, with low rates of stroke presumably due to cerebral ischemia during carotid artery clamping. Similarly, some surgeons routinely use shunts and also report a small incidence of stroke, attributed to technical problems related to the use of the shunt. A third option is to use shunts selectively in patients who are at high risk for ischemic

stroke. Intraoperative techniques to identify patients who need a shunt include intraoperative measurement of carotid stump pressure after the common and external carotid arteries have been clamped, intraoperative neurologic monitoring with EEG or somatosensory evoked potentials, measurement of middle cerebral artery flow by transcranial Doppler ultrasound, and monitoring with cerebral oximetry.

Placement of an intravascular shunt from the common carotid artery to the ICA during endarterectomy is a controversial practice. Adequate cerebral perfusion without shunting occurs in 85% to 90% of patients. Awake assessment is the most sensitive and specific method of determining the need for shunt placement. Intraoperative neurologic assessment of the awake patient under local anesthesia can be as simple as having the patient squeeze a noise toy in the contralateral hand and

answering a few simple questions after carotid occlusion. Patients who develop weakness or changes in mental status should be shunted. For patients under general anesthesia, stump pressures lower than 50 mm Hg have been found to correlate with cerebral ischemia by EEG criteria, and thus should be shunted.

C. Postoperative Care. Immediately after endarterectomy, neurologic function and blood pressure alterations should be monitored. Hypertension and hypotension are common after endarterectomy and may cause neurologic complications. The extremes of blood pressure should be treated with either sodium nitroprusside or phenylephrine to keep the systolic blood pressure within 20 mm Hg of preoperative levels (slightly higher in chronically hypertensive patients). The wound should be examined for hematoma formation. Aspirin is resumed in the immediate postoperative period. Some advocate the use of dextran-40 (up to 20 mL/kg/day for up to 72 hours) as an additional antithrombotic agent, which can be started intraoperatively and continued into the early postoperative period. Dextran-40 is an intravascular volume expander and should therefore be used with caution in patients with preexisting congestive heart failure.

D. Patient Followup. A baseline duplex scan is obtained 1 month after the procedure, and again at 12 months. Patients can then be followed annually. Patients who can tolerate aspirin are given 81 or 325 mg/day.

E. Complications

1. Stroke rates must be low (3% for asymptomatic patients; 6% for symptomatic) to make operative management of cerebrovascular disease reasonable, especially in asymptomatic patients.

2. 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. The timing of coronary intervention relative to CEA is under debate.

3. Cranial nerve injuries, the majority of which are transient, occur in 5% to 10% of patients who

undergo CEA. The most commonly injured nerve is the hypoglossal nerve, followed by the recurrent laryngeal, superior laryngeal, marginal mandibular, and glossopharyngeal nerves.

4. Recurrent carotid stenosis has been reported to occur in 5% to 10% of cases, although symptoms are present in fewer than 3%. Recurrent stenoses are more common among women, patients who continue to smoke, and in those who have hyperlipidemia, diabetes, or hypertension. Early recurrent stenosis typically develops within 2 years of CEA, and results from neointimal hyperplasia. Lesions that develop more than 2 to 3 years after CEA generally result from progressive or new atherosclerotic disease. 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 CAS.

5. Cerebral hyperperfusion syndrome is a feared complication that usually occurs several days after CEA. The incidence is 0.5% to 5% in published reports. **It is often associated with severe hypertension,**

and patients initially complain of a severe headache. Intracranial hemorrhage is the most devastating consequence of this phenomenon. Patients suspected of having cerebral hyperperfusion syndrome should undergo noncontrast brain computed tomography to rule out cerebral hemorrhage, and should be admitted, preferably to an intensive care unit. Strict control of systemic blood pressure and administration of antiseizure medications is paramount.

F. Carotid Artery Stenting. The indications for CAS are the same as those for a CEA; however, this technique remains under investigation. Several studies have been completed or are under way to examine the efficacy of CAS compared to CEA, particularly in high-risk patients. Outcomes have varied, especially as device technology and operator experience have improved. The Carotid Revascularization Endarterectomy versus Stenting Trial (CREST) study randomly assigned patients with symptomatic or asymptomatic carotid stenosis to undergo CAS or CEA. This trial showed that the risk of composite stroke, MI, or death did not differ significantly in the group undergoing CAS and the group undergoing CEA. During the periprocedural period, there was a higher risk of stroke with stenting and a higher risk of MI, defined as an elevation in troponin, with endarterectomy. However, the patients who suffered a stroke were more affected than those who had an MI. Because CEA is well tolerated and has a very low risk of complications, CAS is commonly reserved for high-risk patients, including (but not limited to) patients with the following conditions:

1. Severe cardiac disease, including NYHA class III or IV congestive heart failure, left ventricular ejection fraction <30%, recent unstable angina or myocardial infarction

2. Prior ipsilateral neck surgery

3. Prior neck radiation

4. Contralateral vocal cord paralysis or recurrent laryngeal nerve injury

5. Surgically inaccessible lesion (i.e., C2 or higher based on appropriate imaging)

Relative contraindications to CAS include severe tortuosity of the common and internal carotid arteries; complex aortic arch anatomy (increasing difficulty as great vessels arise from ascending rather than transverse aortic arch); severe calcification or extensive thrombus formation; near-complete or complete ICA occlusion.

Carotid stenting has evolved since its introduction. Meticulous technique is critical for reducing the incidence of stroke. Special attention must be taken to avoid catheter and wire manipulation of the lesion prior to cerebral protection device deployment, and to ensure removal of all air bubbles within the angiography tubing. Retrograde femoral access, followed by a 6F introducer access sheath, is the most commonly used technique. Other approaches include transcervical (via a 2- to 3-cm neck incision and surgical exposure of the common carotid artery) and transbrachial (using a 6F introducer access sheath) access. When the common carotid artery has been reached, an arteriogram is obtained with anteroposterior and lateral projections to obtain minimal overlap

of the internal and external carotid arteries. After angiography, the stenosis is crossed and an embolic protection device is deployed into the ICA. The typical device used today is a filter-like device that is advanced across the lesion and then opened in the distal ICA prior to angioplasty and stent deployment. After placement of the embolic protection device, predilatation of the stenosed artery is performed if necessary. Self-expanding stents are deployed across the carotid bifurcation and dilated as necessary. These come in a variety of diameters (6 to 10 mm), lengths (3 to 4 cm), and shapes (cylindrical or tapered). The diameter is typically sized to the diameter of the distal common carotid artery.

G. Patient Followup. Followup using duplex ultrasound is important to identify patients with restenosis and is usually performed at baseline following CAS, then at 1 month, and at least every year thereafter. Significant elevation of peak systolic velocity from baseline should prompt further evaluation with angiography. Patients should receive antiplatelet therapy with Clopidogrel for a minimum of 3 months.

H. Complications

1. Embolic stroke is the most common complication of CAS. Risk factors include lack of a cerebral protection device, long or multiple lesions, and age above 80 years. Thrombolysis may be a successful treatment option, especially if the source of emboli is an acute thrombus. When an embolus is composed of atheroma or chronic thrombus, however, mechanical removal of emboli may become necessary to restore flow.

2. Hemodynamic instability may occur during manipulation and angioplasty of the carotid bifurcation. Bradycardia should be anticipated and pretreated with atropine or glycopyrrolate prior to dilation of the carotid bifurcation. Postoperatively, as with CEA, patients should be monitored to avoid extremes of blood pressure.

3. Restenosis occurs in approximately 5% of patients at 12 to 24 months and is typically secondary to intimal hyperplasia. These lesions are often amenable to repeat endovascular intervention.

The ROADSTER trial is a multicenter clinical study intended to support premarket approval of the Silk Road Medical Carotid Stent System and Neuroprotection System. The Silk Road procedure is a hybrid approach to carotid intervention, pairing the advantages of a transcervical approach with the benefits of reverse flow embolic protection and stenting technology. Direct carotid access is accomplished via a small 2- to 3-cm incision above the clavicle, and an arterial access sheath is placed. A second access sheath is placed in the femoral vein, and the two sheaths are connected by a flow controller to divert embolic debris from the ICA after clamping of the proximal common carotid artery. The debris is trapped in an inline filter and never enters the venous circulation. The results of the trial showed a 30-day composite stroke, death, and MI rate of 3.5%. The stroke rate of 1.4% to date represents the lowest rate for stenting in Òhigh-riskÓ patients, and is comparable to results of Òstandard-riskÓ patients in the CEA arm of CREST.

CHAPTER 26: CEREBROVASCULAR DISEASE

Multiple Choice Questions

1. Which one of the following is not considered a risk factor for stroke and TIA?

a. Age greater than 55 years

b. African-American or Hispanic race

c. Female gender

d. Hypertension

e. Smoking

View Answer

2. A 67-year-old male is referred to your clinic by his primary care physician after a routine check-up revealed a right carotid bruit. A subsequent duplex ultrasound revealed 50% stenosis of his right carotid artery. According to the ACAS trial, carotid endarterectomy for asymptomatic patients is indicated for stenoses defined by which of the following?

a. ≥ 90%

b. ≥ 80%

c. ≥ 70%

d. ≥ 60%

e. ≥50%

View Answer

3. According to the NASCET trial, what is the approximate reduction in stroke rate over 2 years if CEA is completed for a symptomatic patient with 70% stenosis of her left carotid artery?

a. 6% absolute risk reduction (i.e., 11% control vs. 5% CEA)

b. 7% absolute risk reduction (i.e., 22% control vs. 15% CEA)

c. 13% absolute risk reduction (i.e., 20% control vs. 7% CEA)

d. 17% absolute risk reduction (i.e., 26% control vs. 9% CEA)

e. 20% absolute risk reduction (i.e., 25% control vs. 5% CEA)

View Answer

4. Which of the following represents the major cranial nerve most commonly injured during carotid endarterectomy?

a. The vagus nerve

b. The hypoglossal nerve

c. The glossopharyngeal nerve

d. The facial nerve

e. The recurrent laryngeal nerve

View Answer

5. Which of the following veins is commonly ligated during carotid endarterectomy?

a. The facial vein

b. The internal jugular vein

c. The superior thyroid vein

d. The anterior jugular vein

e. The subclavian vein

View Answer

6. The first intracranial branch of the internal carotid artery is:

a. The superior thyroid artery

b. The inferior thyroid artery

c. The lingual artery

d. The ophthalmic artery

e. None of the above (there are no intracranial branches of the internal carotid artery)

View Answer

7. The first branch of the external carotid artery is:

a. The superior thyroid artery

b. The inferior thyroid artery

c. The lingual artery

d. The ophthalmic artery

e. None of the above

View Answer

8. A 72-year-old female with a history of TIAs is found to have 80% stenosis of the left carotid artery on duplex ultrasound. The most appropriate next step in management is:

a. Repeat ultrasound in 6 months

b. Start Aspirin and Clopidogrel

c. Start Aspirin only

d. Recommend carotid stenting

e. Recommend carotid endarterectomy

View Answer

9. The following, by themselves, are all acceptable indications for carotid artery stenting, except:

a. Surgically inaccessible lesion

b. Contralateral internal carotid artery occlusion

c. Contralateral vocal cord paralysis

d. Prior ipsilateral neck surgery

e. Prior neck radiation

View Answer

10. While undergoing carotid stenting, a patient develops bradycardia and hypotension, during predilatation of a stenotic region. This is most likely due to which process?

a. Global cerebral hypoperfusion

b. Myocardial infarction

c. Stretching of the carotid body

d. Stimulation of the hypoglossal nerve

e. Formation of atheroembolism

View Answer

27

Thoracoabdominal Vascular Disease

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The majority of vascular diseases are secondary to atherosclerotic changes of the arterial wall which are multifactorial and are influenced by genetic predisposition, age, and environmental factorsÑparticularly diet, smoking, and exercise. The arterial wall is comprised of the intimal, medial, and adventitial layers. Endothelium lines the intima. The media contains layers of smooth muscle cells and extracellular matrix (ECM) proteins. The adventitia is composed of loose connective tissue and fibroblasts. A true aneurysm is a permanent localized dilation of all three layers (intima, media, adventitia) greater than 50% of the normal or adjacent vessel diameter. Degenerative disorders of the thoracoabdominal aorta affect the media most, whereas occlusive disorders result in luminal narrowing from atherosclerosis.

I. ABDOMINAL AORTIC ANEURYSMS.

This is the most common type of aortic aneurysm, occurring in 3% to 9% of people above 50 years of age in the Western world *(Br J Surg.* 1998;85:155. PMID: 9501808). In the United States, ruptured abdominal aortic aneurysms (AAAs) are the 13th leading cause of death overall and the 10th leading cause of death in men above 55 years, a rate that has held steady for the past two decades despite improvements in operative technique and perioperative management.

A. Pathophysiology. Ninety percent of AAAs are degenerative in origin, 5% are inflammatory, and the remainder are idiopathic *(J Vasc Surg.* 2003;38:584). The most accepted predictor of rupture is maximal diameter, and the major risk factors for aortic dilation. Several trials (UKSAT; *Lancet.* 1998;352(9141):1649-1655; and ADAM; *Ann Intern Med.* 1997;126(6):441-449) have established a risk of rupture of 0.6% to 1%/year, with the risk rising to 10% at 6 cm, 20% at 6.5 cm, and 30% at 7.5 cm. Eighty-five percent of AAAs are infrarenal; 25% involve the iliac arteries, and 2% involve the renal or other visceral arteries *(J Cardiovasc Surg.* 1991;32:636). Fourteen percent are associated with peripheral (e.g., femoral or popliteal) aneurysms *(J Vasc Surg.* 2000;31:863). However, 62% of patients with popliteal aneurysms and 82% with femoral aneurysms have an associated AAA, mandating screening in these patients.

B. 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. Fifty percent of AAAs are identifiable

on physical examination as a pulsatile mass at or above the umbilicus. AAA rupture may mimic renal colic, peritonitis, duodenal perforation, pancreatitis, degenerative spine disease, acute disk herniation, or myocardial infarction.

2. Radiologic evaluation

a. Ultrasonography and computed tomography (CT) scanning. Ultrasound is an accepted screening study for the presence of AAAs with sensitivity of 87% and specificity nearing 100%; however for patients nearing size criteria for repair the study of choice is CT angiography *(J Vasc Surg.* 2009;50(4 Suppl):S2-S49).

b. Magnetic resonance (MR) scan is comparable to CT but avoids radiation exposure and is useful in patients with intravenous contrast contraindications.

c. Aortography is not sensitive for the diagnosis of AAA because the study by definition is a luminogram and as such will underestimate the total aortic size due to the presence of mural thrombus.

C. Elective Management of AAA. The risk of aneurysm rupture correlates best with aneurysm size *(Ann Surg.* 1966;164:678). However, even small aneurysms can rupture.

1. Medical management. Patients with small aneurysms (<4.5 cm in diameter) without risk factors for rupture can be followed using ultrasound or CT scan yearly, with larger ones being followed more frequently. Smoking cessation, exercise, control of hypertension, and treatment of chronic pulmonary obstructive disease are critical *(J Vasc Surg.* 2002;35:72).

2. Elective surgical treatment. Operative mortality ranges from less than 5% for uncomplicated AAA to greater than 50% for ruptured AAA *(Br J Surg.* 1998;85:1624). Five-year survival after elective repair 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 *(J Vasc Surg* 2009;50(4 Suppl):S2-S49. PMID: 19786250). Indications for **surgical management** include *(J Vasc Surg.* 2003;37:1106):

a. Symptomatic aneurysms of any size.

b. Aneurysms exceeding 5.5 cm diameter (5 cm for females).

c. Increase in diameter by more than 0.5 cm/6 months or 1 cm/year.

d. Saccular aneurysms.

e. Relative contraindications to elective repair include recent myocardial infarction, intractable congestive heart failure, unreconstructable coronary artery disease, life expectancy of less than 2

years, and incapacitating neurologic deficits after a stroke.

3. Open operative technique. The two open surgical approaches are the transabdominal and retroperitoneal approaches. In the transabdominal repair, the aneurysm is approached through a midline abdominal incision and exposed by eviscerating the small bowel, and mobilizing the left colon before incising the retroperitoneum. Next, the duodenum and left renal vein are dissected off the aorta. After systemic heparinization,

the aorta and iliac arteries are cross-clamped. An aortotomy is made and extended longitudinally to the aneurysm Òneck,Ó where the aorta is either transected or cut in a T fashion. The thrombus is removed, and any bleeding lumbar arteries are oversewn. The proximal anastomosis is performed to nonaneurysmal aorta using a graft. The distal anastomosis is completed at the aortic bifurcation (tube graft) or at the iliac or femoral arteries (bifurcated graft), as the extent of the aneurysmal disease dictates. The aneurysm wall is then closed over the graft. An alternative approach is through a left retroperitoneal incision. This approach is advantageous in obese patients, those with chronic pulmonary obstructive disease, patients with previous intraabdominal surgery, and is the preferred approach at Washington University in St. Louis. In addition, proximal suprarenal or supraceliac control of the aorta is more easily achieved via this latter approach.

D. Management of Ruptured AAA

1. Preoperative management. Unstable patients with a presumed diagnosis of a ruptured aneurysm (hypotension, abdominal or back pain, and a pulsatile abdominal mass or history of aneurysmal disease) are gently resuscitated with fluids (crystalloid, colloid, or blood) to maintain organ perfusion while avoiding over-resuscitation with the associated diluting of clotting factors, destabilization of thrombus, and hemorrhage (so called Òpermissive hypotensionÓ). Unstable patients are transferred immediately to the operating room for exploration, whereas those who are stable should undergo emergent CT scanning to confirm the diagnosis.

2. Operative management is aimed at rapidly controlling the aorta. Anesthetic induction is delayed until incision. Through a midline incision, the supraceliac aorta is controlled at the diaphragmatic hiatus by dividing the pars flaccida, mobilizing the left lateral segment of the liver toward the patient's right, and isolating the aorta from the adjacent esophagus and attaining vascular control with an aortic clamp, or direct pressure against the spine. The retroperitoneal hematoma is opened, and the proximal neck of the aneurysm is identified and cross-clamped. Distal vessel dissection continues, and management is similar to repair of an elective AAA. The use of bifurcated grafts should be avoided in favor of the more expeditious tube graft reconstruction. Heparin should also be avoided in these patients who are likely to already be coagulopathic due to the high risk of intraoperative and postoperative bleeding. However, in centers with EVAR capabilities, the endovascular approach has become the preference for initial approach to a ruptured AAA.

3. Complications from open AAA repair

a. Arrhythmia, myocardial ischemia, or infarction.

b. 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.

c. Management of resuscitation, electrolytes, and pressors is critically important during **aortic unclamping** due to sudden hypotension from a decrease in systemic vascular resistance and introduction of previously sequestered metabolites.

d. Renal insufficiency may be related to the use of intravenous contrast, inadequate hydration, hypotension, renal ischemia from a period of aortic clamping above the renal arteries, or embolization to the renal arteries.

e. Lower-extremity ischemia may result from embolism or thrombosis, especially in emergency operations for which heparin might not be used. This can be prevented by minimizing manipulation of the aneurysm prior to clamping. Use of Fogarty balloon catheters to remove distal emboli from lower-extremity vessels are indicated when leg ischemia is identified in the operating room.

f. Microemboli arising from atherosclerotic debris can cause cutaneous ischemia (trash foot), which is usually treated expectantly as long as the major vessels are patent. Amputation may be required if significant necrosis results.

g. Gastrointestinal complications consist of prolonged paralytic ileus, anorexia, periodic constipation, or diarrhea, which is diminished by using the left retroperitoneal approach. **Ischemic colitis of the sigmoid colon** is related to ligation of the inferior mesenteric artery (IMA) in the absence of adequate collateral circulation. Symptoms include leukocytosis, significant fluid requirement in the first 8 to 12 hours postoperatively, fever, and peritonitis. Diagnosis is confirmed by flexible sigmoidoscopy to 20 cm above the anal verge. Necrosis that is limited to the mucosa may be treated expectantly with intravenous antibiotics and bowel rest. 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.

h. Paraplegia, a rare complication of infrarenal aneurysm surgery, may occur after repair of a ruptured AAA due to spinal cord ischemia. Supraceliac cross-clamping, prolonged hypotension, and obliteration or embolization of important collateral flow to the spinal artery (internal iliac arteries or an abnormally low origin of the accessory spinal artery [artery of Adamkiewicz]) increases the risk.

i. Sexual dysfunction and retrograde ejaculation result from damage to the sympathetic plexus (ÒNervi erigentesÓ) during dissection near the proximal left common iliac artery.

E. Endovascular management of AAAs has dramatically decreased the acute morbidity of aneurysm surgery. **Indications** are no different than those for traditional open repair.

1. The most important **selection criterion** for endovascular treatment of an AAA is appropriate aortoiliac anatomy. Further advances in fenestrated and branched devices are allowing treatment of pararenal and

other more complex AAAs. At this time, preoperative CT assessment includes the following factors:

a. Current devices require at least 15 mm of healthy infrarenal aorta.

b. Angle between the neck and aneurysm.

c. Intraluminal thrombus in the proximal neck represents a relative contraindication for endovascular treatment.

d. A cone-shaped neck or reverse taper (i.e., widens more distally) may preclude adequate apposition of the endograft to the aortic wall.

e. Iliac artery tortuosity, calcification, and luminal narrowing are critical factors for endograft delivery and deployment.

f. Patent aortic branches may influence the decision as to whether to proceed with endovascular repair. Large accessory renal artery 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. A large patent IMA suggests abnormal mesenteric blood supply and risk of large-bowel ischemia with endograft coverage of the IMA orifice.

2. Technique. Endovascular devices are introduced retrograde through open or percutaneous femoral arterial access. Most commonly utilized devices are bifurcated and modular in design. Appropriate oversizing of stent grafts (by 20%) is based on preoperative assessment of arterial diameter to ensure adequate graft apposition to the aortic wall. Length can be tailored by overlapping extension segments. The distal end of the iliac limbs is typically positioned proximal to the hypogastric orifices to maintain pelvic perfusion.

3. Complications of endograft repairs of AAAs

a. Branch occlusion, distal embolization, graft thrombosis, and arterial injury (especially iliac artery avulsion at the iliac bifurcation).

b. Arterial dissection may occur. Additional stents can be used to treat the dissected vessel if necessary.

c. Bowel ischemia may occur postoperatively secondary to embolization or hypoperfusion, but this is rare compared to open surgical repair. **Renal dysfunction** may occur because of the

nephrotoxicity of the contrast agent used for intraoperative angiography or because of direct injury due to embolization or renal artery occlusion.

d. Graft migration occurs in 1% to 6% of patients, and is associated with challenging arterial anatomy and poor graft placement. This complication can typically be treated with secondary endovascular procedures.

e. Endoleak is defined as failure to fully exclude the aneurysmal sac from arterial blood flow, potentially predisposing to rupture *(J Endovasc Surg.* 1997;4:152. PMID: 9185003). Management strategies for endoleaks discovered on followup imaging studies are evolving. For any endoleak that is associated with aneurysm sac enlargement, intervention is required. Endoleaks are usually corrected by endovascular means but may require conversion to open surgical repair.

1. In general, endoleaks from the proximal or distal attachment sites **(type I)** warrant intervention because of continued direct flow into the AAA. Options include: conversion to open, or angioplasty with proximal stent placement.

2. Type II endoleaks are due to collateral flow (IMA, lumbar arteries) and may be closely observed in the absence of aneurysm expansion. Type II endoleaks may be treated with embolization through collateral vessels or directly through injection of the aneurysm sac via a translumbar approach, although the longterm outcomes are equivocal *(J Vasc Surg.* 2012;55(5):1263Ñ1267. PMID: 22322122).

3. Type III endoleaks are caused by inadequate seal between graft components. They should be corrected as soon as they are diagnosed as they too result in direct flow in the AAA.

4. Type IV endoleaks are due to porosity of the graft material. This is rare with newer generations of endografts but these must be treated if found.

4. Results. Relative to open surgical repair, endovascular treatment of AAA is associated with a reduction in perioperative morbidity, shorter duration of hospitalization *(J Vasc Surg.* 2003;37:262), and reduction in perioperative mortality (EVAR 1; *Lancet.* 2004;364:843-848; DREAM; *NEJM.* 2005;352:2398-2405). However, studies of long-term outcome comparing open versus endovascular repair have demonstrated similar rates of survival after 4 years (DREAM; *NEJM.* 2010;362:1881-1889; UK EVAR; *NEJM.* 2010;362:1863-1871). Close followup with CT scanning every 6 months for 1 year, and then yearly, is essential to maintaining long-term clinical success using this technique.

II. THORACIC AORTIC ANEURYSMS.

Thoracic aortic aneurysms (TAAs) are primarily a disease of the elderly, with an estimated incidence of 10.4 per 100,000 person-years *(JAMA.* 1998;280(22):1926). Ascending aortic aneurysms are most common (÷60%) followed by aneurysms of the descending aorta (÷35%) and of the transverse aortic arch (<10%). Most descending TAAs begin just distal to the orifice of

left subclavian artery.

A. Pathophysiology. TAAs are divided into five main types: Ascending, transverse, descending, thoracoabdominal, and traumatic. Ascending aortic aneurysms are usually caused by medial degeneration. Transverse, descending, and thoracoabdominal aortic aneurysms are related to atherosclerosis with hypertension contributing to their expansion. Traumatic aneurysms are usually due to blunt injury to the chest.

B. Diagnosis

1. Clinical manifestations are usually absent; most are detected as incidental findings on chest imaging obtained for other purposes. A minority of 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), erosion into adjacent structures (esophagus, lung, airway), or distal embolization.

2. Radiologic evaluation.

a. Chest x-ray may reveal a widened mediastinum or an enlarged calcific aortic shadow. Traumatic aneurysms may be associated with skeletal fractures.

b. MR or CT imaging with intravenous contrast provides precise estimation of the size and extent of aneurysms and facilitates surgical planning.

c. Echocardiography may be useful in evaluating aneurysms involving the aortic arch.

d. Aortography demonstrates the proximal and distal extent of the aneurysm and its relationship with aortic branch vessels arising from it.

C. Surgical management varies by 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. Ascending and transverse arches are repaired through a median sternotomy incision. Descending and thoracoabdominal aneurysms are approached through a left posterolateral thoracotomy or thoracoabdominal incision. Intraoperative management of patients undergoing thoracotomy is facilitated by selective ventilation of the right lung using a double-lumen endobronchial tube. Several adjuncts for limiting postoperative paraplegia following surgery for descending and thoracoabdominal aneurysm are employed, including cerebrospinal fluid drainage and retrograde perfusion.

1. Ascending aortic arch aneurysms

a. Size criteria for TAA repair are not as clearly defined as for infrarenal AAAs. **Indications** for surgical repair include symptomatic or rapidly expanding aneurysms, aneurysms greater than or equal to 6 cm in diameter, ascending (type A) aortic dissections, mycotic aneurysms, and

asymptomatic aneurysms greater than or equal to 5.5 cm in diameter in patients with Marfan syndrome *(Coron Artery Dis.* 2002;13:85).

b. Operative management. An aneurysm arising distal to the coronary ostia is replaced with an interposition graft. An aneurysm resulting in aortic valve incompetence is replaced with a composite valved conduit **(Bentall procedure)** or a supracoronary graft with separate aortic valve replacement. All ascending arch aneurysms due to connective tissue disorders 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 or equal to 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 a patch that incorporates the orifices 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 the 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 asymptomatic aneurysms greater than or equal to 6 cm in diameter and any symptomatic aneurysms.

b. Operative management. 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. Left heart (atriofemoral) bypass is often used, both to protect the heart from overdistention and to provide distal blood flow while the aorta is clamped. Cerebrospinal fluid drainage is used as an adjunct to decrease the incidence of postoperative paraplegia.

4. Thoracoabdominal aneurysms

a. Indications for repair include aneurysms greater than or equal to 6 cm in diameter and any symptomatic aneurysms.

b. Operative management consists of tube graft replacement along with anastomosis of the major visceral 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. Use of left heart (atriofemoral) bypass is a valuable adjunct to allow for both cerebral

perfusion and visceral perfusion off the bypass circuit via a femoral cannula. The thoracic aorta is clamped and opened to perform the proximal anastomosis. The aorta is clamped distally opening the remaining aneurysm. The orifices of all major aortic branches are occluded with balloon catheters or vascular clamps. Temporary perfusion can be maintained to those branches during aneurysm repair by using balloon catheters connected to the atriofemoral bypass. The anastomoses of significant aortic branches to the graft are performed as a patch or with separate bypasses. The clamp is moved to the graft below the renal arteries to reperfuse all visceral vessels in an antegrade fashion. The distal anastomosis is made either to the uninvolved aorta or to the iliac 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. Classification. Traumatic aortic injuries have the following categories:

(1) Grade I: Intimal tear

(2) Grade II: Intramural hematoma

(3) Grade III: Pseudoaneurysm

(4) Grade IV: Rupture

c. Management. Historically, these aneurysms were repaired by primary aortorrhaphy, aneurysmectomy, and end-to-end reanastomosis or by interposition grafting. In current practice, Grade I injuries may be observed on serial imaging and Grade IIÑIV injuries should undergo prompt endovascular repair, if amenable. Age should not disqualify from endovascular repair *(J Vasc Surg.* 2011;53(1):187Ñ192).

D. Possible **complications** of thoracic aortic surgery are similar to those for abdominal aortic surgery. The incidence of paraplegia may be as high as 30% with some types of TAAs *(Ann Thorac Surg.* 2007;83:S856). This risk can has been reduced to less than 2% by multimodal therapies used to minimize spinal cord ischemia: Distal aortic perfusion, intercostal and lumbar artery reimplantation, pre- or intraoperative localization of spinal cord blood supply, hypothermia, cerebrospinal fluid drainage, and pharmacotherapy *(World J Surg.* 2008;32(3):355-360).

E. Endovascular Management of TAA

1. Indications and technique. Because of the considerable morbidity and mortality associated with surgical repair of descending thoracic aneurysms, the endovascular approach to aneurysm exclusion is particularly attractive. Treatment with endovascular stent graft placement requires specific anatomic criteria: Adequate length (2 cm) and diameter (20 to 45 mm) of the proximal and distal aneurysm necks, absence of significant mural thrombus within the sealing zones, and aortic and iliofemoral anatomy amenable to device introduction. In situations in which the

proximal neck length is too short, coverage of the left subclavian artery can be performed with or without an adjunctive left carotidÑleft subclavian transposition or bypass.

2. Results and complications. Reported results for the use of endovascular devices are encouraging, with low morbidity and mortality with high rates of aneurysm exclusion. Fenestrated and branched grafts along with hybrid techniques using extra-anatomic bypass procedures are being used to approach more complex aneurysm anatomy.

III. OTHER ARTERIAL ANEURYSMS

A. Infected aneurysms have risen in incidence with the increased prevalence of immunocompromised patients and invasive transarterial procedures.

1. Pathophysiology. Infected aneurysms can be divided into four types: Mycotic aneurysm, microbial arteritis with aneurysm, infection of preexisting aneurysm, and posttraumatic infected false aneurysm. *Staphylococcus aureus* is the most common pathogen, although *Salmonella* species (arteritis), *Streptococcus* species, and *Staphylococcus epidermidis* (preexisting aneurysms) also may occur. Gram-negative infections have the highest risk of rupture.

2. Diagnosis

a. Clinical manifestations may be absent or include fever, tenderness, or sepsis. Physical examination may demonstrate a **tender, warm, palpable mass** in an infected peripheral aneurysm. Laboratory tests may

reveal leukocytosis. Aerobic and anaerobic blood cultures should be obtained, but are positive in only 50% of patients.

b. MRI or CTA can demonstrate an aneurysm and verify its rupture. **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.

B. Management

1. Preoperative. Broad-spectrum antibiotics should be administered intravenously after aerobic and anaerobic blood cultures have been obtained.

2. Intraoperative. Goals of surgery include (1) controlling hemorrhage; (2) obtaining arterial specimens for Gram stain, aerobic and anaerobic cultures, and drug sensitivities; (3) resecting the aneurysm with wide debridement and drainage; and (4) reconstructing major arteries through uninfected tissue planes. Extra-anatomic bypass may be necessary to avoid contamination of the graft. Inline reconstructions with antibiotic-impregnated grafts, cryopreserved homografts, or native veins are alternatives that can be used for arterial reconstructions depending on the location of the aneurysm and the extent of the infection.

3. Postoperative. Adequate drainage of the aneurysm cavity and longterm antibiotic therapy for

at least 6 weeks typically are required.

IV. ACUTE AORTIC SYNDROMES

A. Aortic dissection is a tear in the intima allowing blood to travel between the intima and the media resulting in the creation of two flow channels: The true lumen and the Òfalse lumen.Ó The incidence is ÷30 cases per million individuals per year *(J Cardiovasc Surg (Torino).* 2010;51:601-608), and the natural history is marked by a high mortality rate, as high as 1% per hour over the first 24 hours *(Circulation.* 1950;1:360-387). Dissections are classified according to the Stanford Classification system based on involvement of the ascending arch, which simplified the previously used DeBakey classification.

1. Diagnosis

a. Clinical manifestations include the hallmark sudden onset, ripping chest/abdominal pain, radiating to the back. There may be an associated blood pressure discrepancy between the upper extremities, new onset heart murmur, and, less commonly, paraplegia or paresthesias.

b. Risk factors include long-standing uncontrolled hypertension, family history, and collagen vascular diseases such as Marfan syndrome.

c. Radiographic tests

(1) CT Angiography remains the gold standard for diagnosis of dissections in the acute setting with modern techniques having a sensitivity and specificity of 100% *(Radiology.* 1996;199:347-352), and gives accurate information regarding the location of the intimal injury, and any associated involvement of branch vessels.

(2) MR Angiography has been supplanted by CT Angiography in the acute setting, but is used in long-term followup.

2. Type A dissections are those involving the proximal ascending aorta, and are treated as surgical emergencies. These are the most common type of dissection, accounting for approximately 60% of all dissections. The high mortality rate is due to the feared progression of retrograde dissection, hemopericardium, and cardiac tamponade.

a. Management of Type A dissection nearly universally involve emergent surgery with replacement of the proximal ascending arch with or without valve replacement.

3. Type B dissections are those isolated to the descending thoracic aorta distal to the left subclavian. The mortality rates approach 10% at 30 days, 25% at 3 years, and 50% at 5 years. These are subdivided in ÒacuteÓ (<14 days) and ÒchronicÓ (>14 days) and are further subdivided into ÒuncomplicatedÓ or ÒcomplicatedÓ depending on evidence of malperfusion of end organs.

a. Management of complicated Type B dissections is aimed at revascularization of the affected

segment through whatever means is most expedient, utilizing both endovascular and open surgical options.

b. Management of uncomplicated Type B dissections has recently been treated with medical management including β-blockade and long-term antihypertensive regimens. Despite this, uncomplicated Type B dissections may progress to become complicated, and up to 25% require intervention within 4 years (ADSORB trial. *Eur J Vasc Endovasc Surg.* 2014;48:285-291).

c. Surgical management either via direct graft replacement of the affected segment, removal of the flap via aortotomy, or endovascular fenestration and stenting have all been used as techniques to restore perfusion to malperfused end organs.

d. Endovascular coverage of the intimal tear in uncomplicated Type B dissections has become the standard of treatment. In the chronic setting, this has been shown to promote favorable aortic remodeling with improved mortality at 5 years (INSTEAD-XL, *Circ Cardiovasc Interv.* 2013;6:407-416). Trials are ongoing in the acute setting, and also demonstrate favorable aortic remodeling (ADSORB trial. *Eur J Vasc Endovasc Surg.* 2014;48:285-291).

B. Intramural hematomas are a focal hemorrhage in the wall of the aorta between the intima and media layer due to the rupture of the vasa vasorum without evidence of an intimal tear or dissection flap due to iatrogenic injuries, trauma, or hypertension. These are classified similar to dissections, with those involving the ascending aorta being corrected surgically, and the remainder being medically managed unless the hematoma is >1 cm think (ninefold higher progression), aortic diameter greater than 40 mm (30-fold higher risk of progression), or progression on serial imaging *(J Vasc Surg.* 2002;35:1179-1183).

C. Penetrating aortic ulcer is a projection into an intramural hematoma that is associated with higher rates of disease progression. These are associated with smoking and atherosclerotic disease. These are classified and treated similar to intramural hematomas.

V. RENOVASCULAR DISEASE.

Stenosis or occlusion of the renal arteries may result in hypertension, ischemic nephropathy, or both. **Renovascular hypertension is the most common form of surgically correctable secondary hypertension.**

A. There are several clinical features that may be used to identify patients with potential renovascular hypertension:

1. The onset of **hypertension** before the age of 18 or after the age of 55.

2. Accelerated, resistant, or malignant hypertension.

3. Unexplained impairment of renal function.

4. Refractory to appropriate multidrug therapy.

5. Hypertension in a patient with extensive coronary disease, cerebral vascular disease, or peripheral vascular disease.

B. The majority have a normal **physical examination;** however, an epigastric, subcostal, or flank bruit or findings of a unilateral small kidney on any imaging study are possible indicators.

C. Pathophysiology

1. Renal arterial stenosis (RAS) leads to activation of the **reninÑangiotensin-aldosterone system** by the ipsilateral kidney and results in volume expansion and peripheral vasoconstriction. Even in the **absence of hypertension,** RAS may lead to renal failure.

a. In **acute renal failure,** RAS should be considered in the differential diagnosis if the workup is unrevealing or the patient was recently started on an angiotensin-converting enzyme (ACE) inhibitor or another antihypertensive or diuretic.

b. RAS may account for up to 20% of **unexplained chronic renal failure** in patients older than 50 years of age.

c. Isolated unilateral RAS generally **do not** cause rise in serum creatinine.

2. Atherosclerosis accounts for nearly 90% of cases of renovascular HTN and usually affects the ostia and proximal renal artery.

3. The second-most common renovascular lesion is **fibromuscular dysplasia,** most commonly medial fibroplasia. These lesions are multifocal, with a **string-of-beads** appearance on angiography, and typically occur in young women.

D. Diagnosis. Testing for clinically significant renal artery disease must evaluate the **anatomic and physiologic changes.**

1. Arteriography remains the Ògold standardÓ for the diagnosis of anatomic RAS. However, the usual risks of arteriography, especially the nephrotoxic effects of the contrast agent, are important caveats to consider.

2. Duplex scanning is the preferred method for screening in patients with indicators of renovascular hypertension.

3. MR angiography is useful for evaluating kidney and main renal artery morphology without the use of nephrotoxic agents.

4. Two rarely used tests to determine the functional significance of a renal artery lesion are **captopril renal scintigraphy** and **selective renal vein renin measurement** when other workup is unrevealing.

E. Management of Fibromuscular Disease

1. Rarely causes renal failure.

2. Endovascular techniques (balloon angioplasty) have a high success rate for the treatment of this arterial pathology with 75% free from hypertension and 95% free from reduction in GFR at 5 years *(J Vasc Surg.* 2012;55(2):421Ñ427). For failure of endovascular treatment (see Section F.3), surgical therapy may be employed.

F. Management of atherosclerotic disease aims to control target organ damage from hypertension and avoid progressive ischemic renal failure. Response to therapy is difficult to predict because a patient's hypertension may be primarily essential and the renal failure due to hypertensive glomerulosclerosis.

1. Medical therapy is often successful in the management of patients with renovascular hypertension and remains the cornerstone of treatment. A combination of β-blockers and a calcium channel blocker, an ACE inhibitor, or an angiotensin II-receptor inhibitor is commonly used as first-line therapy.

2. Surgical therapy

a. Surgical revascularization is a durable option for patients with long segment disease and declining renal function *(Ann Surg.* 1999;230(4):524). In patients undergoing aortic surgery for aneurysmal or occlusive disease with concomitant renal stenoses, consideration should be given to renal revascularization.

b. Procedures

(1) Aortorenal bypass is the classic treatment of renal revascularization using saphenous vein, autologous hypogastric artery (in children), or prosthetic graft.

(2) Renal endarterectomy is another option and is often used for bilateral orificial lesions. A transverse arteriotomy is made over the both orifices.

(3) Alternative bypass procedures are used for unfavorable anatomy. Grafts can be taken from the supraceliac aorta or the superior mesenteric, common hepatic, gastroduodenal, splenic, or iliac arteries. Results are comparable to direct aortic reconstruction with less morbidity and mortality.

(4) Nephrectomy may be required in patients who have unreconstructable disease or a normal contralateral kidney and who are high-risk surgical candidates.

c. Postoperative care

(1) Immediately after operation, patients should be hydrated to maintain adequate urine output. Concern about the patency of the reconstruction may be addressed by a renal or duplex scan.

(2) Patient followup should consist of blood pressure 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 diagnostic imaging.

d. Complications of surgery include persistent hypertension, acute renal failure, renal artery restenosis, thrombosis, aneurysm formation, and distal embolization.

3. Endovascular management of renal artery stenosis

a. Indications for angioplasty of RAS include failure of medical management of renovascular hypertension. Renal artery stents are used for restenosis after previous angioplasty, procedural complications (e.g., dissection), and atherosclerotic ostial lesions.

b. Results. Technical success for renal artery angioplasty is defined as a less than 30% residual stenosis and a pressure gradient less than 10 mm Hg. For patients with atherosclerotic renal artery stenosis large studies (ASTRAL; *NEJM.* 2009;361:1953-1962 and CORAL; *NEJM.* 2014;370:13-22) have shown no impact on renal function or major adverse renal and cardiovascular events compared to medical management.

VI. MESENTERIC ISCHEMIA.

Mesenteric ischemia can be a difficult diagnosis to make because most patients are asymptomatic until late in the disease process. Although considerable advances have been made in the perioperative care as well as the diagnosis and treatment of intestinal ischemia, mortality remains 60% to 80% *(Langenbecks Arch Surg.* 2008;393:163).

A. Acute Mesenteric Ischemia

1. Pathophysiology. The most common cause of acute mesenteric ischemia (AMI) is embolization to the SMA; other causes include thrombosis of the SMA or portomesenteric venous thrombosis. Patients with AMI often have multiple **risk factors,** including significant cardiac disease (frequently atrial fibrillation) and severe atherosclerotic disease of nonmesenteric vessels, and may have a history consistent with chronic intestinal ischemia.

a. Abdominal pain is sudden in onset and intermittent at first, progressing to continuous severe pain. It is often described as **pain out-of-proportion to examination.** These patients may also have bloody diarrhea.

b. 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.

2. Diagnosis

a. Angiography of the mesenteric circulation, including lateral views of the celiac axis and SMA, remains the gold standard. However, most centers use **CT angiography** especially for the diagnosis of AMI. Abdominal plain radiographs are of limited utility.

b. Other laboratory findings can include elevated white blood cell count with a left shift, persistent metabolic acidosis, and lactic acidosis in more advanced cases, but are insensitive and nonspecific

for the diagnosis of mesenteric ischemia *(Langenbeck's Arch of Surg.* 2011;396:3-11).

3. Surgical therapy

a. Patients with AMI frequently require intestinal resection; therefore, laparotomy with open revascularization is the preferred method of treatment.

(1) 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 arterial pulsations are present in the adjacent vascular arcades. Other techniques have been described, including the use of fluorescein dye, Doppler studies, and tissue oximetry, but these are not substitutes for experienced clinical judgment.

(2) Second-look procedures are prudent when bowel viability is questionable. This is especially important in patients who have extensive bowel involvement and in whom resection of all questionable areas could result in short-bowel syndrome.

b. For **venous occlusion,** surgical intervention or lytic therapy rarely is helpful. Systemic anticoagulation should begin as soon as the diagnosis is made to limit progression of 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.

c. Nonocclusive mesenteric ischemia (NOMI) is intestinal ischemia in the absence of thromboembolic occlusion. It occurs in patients with a low-cardiac-output state and chronic intestinal angina. Mortality associated with NOMI is high and treatment is directed toward improving circulatory support and increasing cardiac output. In cases in which cardiac recovery is expected, intraarterial infusions of vasodilators (e.g., Papaverine, Prostaglandin E_1, nitroglycerin) have been attempted, without clear benefit.

4. Perioperative care usually requires maximal medical support; are frequently hemodynamically unstable and develop multiple organ system failure. Admission to the intensive care unit, prolonged endotracheal intubation, parenteral nutrition, and broad-spectrum antibiotic therapy are typically required.

B. Chronic Mesenteric Ischemia (CMI)

1. Patients with CMI present with **intestinal angina,** which is pain usually beginning within an hour after eating and abating within 4 hours (postprandial pain). Such patients experience significant weight loss related to the decreased intake secondary to recurrent pain (food fear). The diagnosis usually is made from obtaining a thorough history alone because physical findings

are usually lacking.

2. Surgical therapy should be reserved for symptomatic patients. Surgical revascularization via bypass or endarterectomy remains the treatment of choice. Studies comparing surgery and endovascular approaches show higher patency in the surgical group, with no significant difference in

2-year mortality, recurrence of symptoms or imaging findings, or reintervention *(J Vasc Surg.* 2007;45(6):1162-1171).

3. Perioperative care. These patients often are malnourished. Some advocate parenteral nutrition for 1 to 2 weeks before surgery, and 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 diagnostic imaging.

4. Complications include intestinal infarction, perforation, prolonged multisystem organ failure, and need for dialysis.

CHAPTER 27: THORACOABDOMINAL VASCULAR DISEASE

Multiple Choice Questions

1. Which of the following is not associated with development of an abdominal aortic aneurysm (AAA)?

a. DM

b. HTN

c. HLD

d. Smoking

e. Positive family history

View Answer

2. Which of the following is not a useful diagnostic modality for AAA?

a. CT

b. Ultrasound

c. MRI

d. Aortography

View Answer

3. Which of the following patients with AAA can be medically managed at this point in time?

a. A 75-year-old male with a 6.3-cm AAA

b. A 68-year-old female with an AAA that has grown from 4.9 to 5.3 cm in the last year

c. A 72-year-old male with a known 4.8-cm AAA and intractable pain

d. A 70-year-old male with an AAA that has grown from 4.8 to 5.1 cm in the last year

View Answer

4. 24 hours after undergoing elective AAA repair, the ICU nurse notices purple-blackish discoloration to the toes on both feet of the patient. He has palpable pedal pulses, what is the next step in management?

a. CT angiogram

b. Operative reexploration

c. Angioplasty

d. Expectant management

e. Guillotine amputation

View Answer

5. 10 hours after undergoing elective AAA, the patient becomes tachycardic, febrile to 39°C, and has required 5 L of fluid to maintain his blood pressure goals, and develops diarrhea. His abdomen is diffusely tender and sigmoidoscopy reveals transmural necrosis of the mucosa. What is the next step?

a. IV hydration, antibiotics, and bowel rest

b. Delayed segmental resection

c. Emergent resection of the involved segment

d. Endoscopic placement of a rectal tube

e. Repeat sigmoidoscopy in 24 hours

View Answer

6. During elective EVAR, both hypogastric arteries are inadvertently covered by the graft. All of the following are potential consequences of acute hypogastric artery occlusion EXCEPT:

a. Buttock claudication

b. Paraplegia

c. Small bowel ischemia

d. Colonic ischemia

e. Perineal skin necrosis

View Answer

7. Following endovascular repair of a ruptured AAA in which the patient received 12 units of blood, 2 units of platelets, and 10 units of plasma, he remains anuric, with peak airway pressures of 50 mm Hg leading to difficulty with adequate oxygenation, and his abdomen is distended. What is the next step?

a. Emergent laparotomy and decompression

b. Observation in ICU

c. Place the patient in prone positioning for oxygenation

d. Flush, and if necessary replace, Foley catheter

View Answer

8. Which of the following is true regarding the structure of the aorta?

a. The intima is composed of smooth muscle and extracellular matrix proteins.

b. The media is the layer most involved with atherosclerotic changes.

c. The adventia is composed of loose connective tissue and fibroblasts.

d. An aneurysm is dilation of the intima and the media, but not the adventia.

e. The majority of degeneration in an aneurysm develops in the adventia.

View Answer

9. All of the following are contraindications to endovascular repair of AAA (EVAR) EXCEPT:

a. A patient with 4 mm of healthy tissue between the renal arteries and the aneurysm

b. A patient with significant thrombus at the proximal landing zone

c. A patient with an occluded left hypogastric artery

d. A patient with a large IMA with a meandering mesenteric artery

e. A patient with a horseshoe kidney

View Answer

10. Which of the following is true with regard to endoleaks?

a. Type I endoleaks are via collateral circulation, and must be treated.

b. Type II endoleaks are due to leaks at proximal or distal components,

and may be observed.

c. Type III endoleaks are due to inadequate seal of the graft components.

d. Type IV endoleaks are due to neovascularization of the AAA sac.

e. Type V endoleaks are due to porosity of the graft material.

View Answer

11. Large-scale studies comparing EVAR to open AAA repair have shown reduction in all of the following with EVAR EXCEPT:

a. Perioperative morbidity

b. Duration of hospitalization

c. Perioperative mortality

d. 4-year survival rates

View Answer

12. A 78-year-old male is found to have a widened mediastinum on routine CXR. A CT scan shows a 5 cm aortic aneurysm beginning distal to the left subclavian artery. Which of the following is true?

a. This aneurysm should be repaired immediately

b. Open repair is accomplished via a median sternotomy

c. Repair requires hypothermic circulatory arrest

d. In the absence of symptoms, this can safely be watched until 6 cm

e. There is no role for endovascular treatment of this aneurysm

View Answer

13. A 68-year-old male presents to the ED with sudden onset ripping chest pain radiating to his back with a systolic blood pressure of 210. He has no EKG changes, but CT angiogram shows an intimal flap beginning in the ascending aortic segment. Which is the next step in management?

a. Transesophageal echocardiography to confirm location of flap

b. Emergent open repair with conduit replacement

c. Admission to ICU with anti-impulse control

d. Endovascular coverage of intimal flap

e. Observation, as this is a uniformly fatal diagnosis

View Answer

14. A 56-year-old male presents to the ED with a worsening of ripping chest pain radiating to his back. He reports the pain began 3 weeks ago, but has progressed this morning. His systolic blood pressure is 205. A CT angiogram shows an intimal flap beginning distal to the left subclavian artery. He is admitted to the ICU and his pain improves with blood pressure control. Which of the following is true?

a. This patient should be evaluated for endovascular coverage of the intimal tear

b. This patient has an acute Type B dissection

c. No further intervention is warranted, as this has a low long-term mortality rate

d. This is a complicated Type B dissection

e. β-blockers should never be used in the care of this disease

View Answer

15. All of the following are clinical features of patients that may be used to identify potential renovascular hypertension EXCEPT:

a. Onset in a young adult

b. Refractory to multidrug therapy

c. Accelerated onset

d. Unexplained renal impairment

e. Onset at the age of 45

View Answer

16. You are called to see a patient in the MICU with “abdominal pain out of proportion to examination.” A 72-year-old female was with marked heart failure due to viral myocarditis, requiring significant inotropic and vasopressor agents. She has begun passing bloody stools while in the MICU. Due to acute kidney injury, a noncontrast CT is obtained, that shows minimal calcification of the aorta or any of the visceral branches. Which of the following is true?

a. This condition has a low mortality rate

b. These patients are best managed with laparotomy and resection of viable bowel

c. Treatment is directed at increasing cardiac output and circulatory support

d. Open revascularization is the preferred therapy

e. Enteral nutrition is indicated

View Answer

28
Peripheral Arterial Disease

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Patrick J. Geraghty

The predominant etiology of occlusive disease of the lower extremities is atherosclerotic change of the arterial intima and media. Major risk factors for developing atherosclerosis include cigarette smoking, diabetes, dyslipidemia, hypertension, and hyperhomocysteinemia. 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 arteritis and Buerger disease).

ACUTE ARTERIAL OCCLUSION OF THE EXTREMITY

Symptoms of acute arterial insufficiency occur abruptly. The presentation generally includes the **six Ps of acute ischemia: Pain, pallor, pulselessness, paresthesias, poikilothermy (cold extremity), and paralysis.** The level of occlusion may be localized by the absence of pulses and the proximal extent of coolness and sensorimotor changes. If adequate collateral circulation is not present, irreversible changes may appear as early as 4 to 6 hours after onset. Therefore, priority must be given to prompt restoration of blood flow.

I. ETIOLOGY

A. The Most Common Cause of Acute Arterial Insufficiency is Embolization.

1. Cardiac sources account for more than 70% of emboli and are usually the result of mural thrombi that develop due to cardiac aneurysms following myocardial infarction or arrhythmias such as atrial fibrillation. Other cardiac sources of emboli include valvular heart disease, prosthetic heart valves, bacterial endocarditis, and atrial myxoma.

2. ArterialÑarterial 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 intact pulses and painful ischemic lesions in the distal extremity. Atheroemboli in the lower extremity can also occur secondary to plaque disruption by catheters. A severely diseased distal aorta in some of these patients is evident on computed tomography (CT) scan and arteriography and has been termed *shaggy aorta.* Upper-extremity ischemia/gangrene can occur due to emboli arising from

thoracic outlet syndrome. In these patients, first rib/anomalous cervical rib or band causes compression of subclavian artery with subsequent poststenotic dilation and mural thrombus formation.

3. VenousÑarterial emboli (paradoxical emboli) can result from an intracardiac shunt (e.g., patent foramen ovale) or intrapulmonary arteriovenous malformations (e.g., OslerÑWeberÑRendu 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. In this clinical scenario, the presence of contralateral pulses and the absence of a history of claudication direct suspicion toward an embolic source.

B. Direct arterial trauma is frequently obvious but may initially be occult. Arterial stenosis or occlusion may occur in a delayed fashion, after progression of an intimal flap or arterial wall hematoma. 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. See Table 28-1 for Rutherford classification of clinical categories of acute limb ischemia. If history and physical examination demonstrate clear evidence and location 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. **Computed tomography angiography (CTA)** provides a wealth of anatomic detail, and delayed sequences are needed to visualize arteries beyond the level of occlusion. Radiographically, embolic occlusions can be distinguished from thrombotic occlusions by their occurrence at vascular bifurcations and by the concave shadow formed at the interface with the contrast. 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. In patients who present with embolism, systemic anticoagulation and postoperative hypercoagulable workup are recommended.

B. Patients who present with penetrating **trauma,** long-bone fractures, or joint dislocations may have vascular injuries. Duplex scan of the injured area can be useful in the diagnosis of intimal flap, pseudoaneurysm, or arterial or venous thrombi. Patients with penetrating injuries who display ÒhardÓ signs of arterial injury need urgent surgical intervention without preoperative

angiography. **Hard signs** of arterial injury include the following:

1. Diminished or absent pulses distal to an injury

2. Ischemia distal to an injury

TABLE 28-1 Clinical Categories of Acute Limb Ischemia

		Clinical Findings		Doppler Signals	
Category	**Prognosis**	**Sensory Loss**	**Muscle Weakness**	**Arterial**	**Venous**
I. Viable	Not immediately threatened	None	None	Audible	Audible
II. Threatened					
a. Marginally	Salvageable if promptly treated	Minimal (toes) or none	None	Inaudible	Audible
b. Immediately	Salvageable with immediate revascularization	More than toes, associated with rest pain	Mild, moderate	Inaudible	Audible
III. Irreversible	Major tissue loss or permanent nerve damage inevitables	Profound, anesthetic	Profound, paralysis (rigor)	Inaudible	Inaudible

Adapted from Rutherford RB et al., *J Vasc Surg.* 1997.

3. Visible arterial bleeding from a wound

4. A bruit at or distal to the site of injury

5. Large, expanding, or pulsatile hematomas

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 pressure distal to the injury should be undertaken, along with comparison with the contralateral (uninjured) limb. A **difference of greater than 10% to 20% in the ankle-brachial indices (ABIs) suggests the need for arteriography or exploration.**

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/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 urgently in patients with an obvious embolus and acute ischemia. 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 documented with an intraoperative arteriogram, depending on the status of distal vessels and pulses after embolectomy. If adequate distal perfusion is not established and an angiogram demonstrates distal thrombus, the distal popliteal artery and tibial arteries may be explored via angiographic or surgical approach. In addition, it is not uncommon for vasospasm to occur following embolectomy and direct intra-arterial infusion of 50 to 100 µg of nitroglycerin may resolve it. In conjunction with steerable guidewires, Fogarty catheters can be used to select the tibial arteries to retrieve distal thrombus. When angiographic approaches fail, popliteal artery cutdown can allow direct access to these vessels. Patch angioplasty or bypass grafting may be required if significant preexisting arterial disease in the affected segment is discovered.

2. Thrombolytic therapy may be useful in patients with clearly viable extremities in whom thrombosis is the likely underlying cause of their acute ischemia. Thrombolytic agents work best on fresh clot. Thrombolysis and followup angiography frequently identify an underlying stenosis that may be treated by balloon angioplasty/stent or by surgical intervention.

Lytic agents, such as alteplase or reteplase, are instilled through an intra-arterial catheter positioned within the thrombosed vessel. These agents are also commonly used in conjunction with percutaneous mechanical thrombectomy (PMT) for large clot burdens.

3. There are several PMT devices available that work based on different principles. Rotational devices use a high-velocity rotating helix to break

the thrombus and include the Trerotola device (Arrow International, PA). The Trellis system (Medtronic, MN) uses an oscillating nitinol wire to agitate a lytic solution that is isolated between proximal and distal occlusion balloons to reduce systemic spread of the lytic agent. The AngioJet thrombectomy system (Boston Scientific, MA) uses a high pressure saline jet flow with aspiration of softened thrombus into the catheter. The EkoSonic catheter (EKOS Corporation, WA) delivers high frequency, low-energy ultrasound in a radial fashion to enhance the penetration of lytic agents by exposing plasminogen receptor sites. This mechanism probably has less hemolytic effect than with saline pressure thrombectomy and less endothelial damage than with rotational thrombectomy; however it requires multiple sessions.

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 prudent to reestablish arterial flow first, whether by direct repair, bypass grafting, or temporary shunting. If the decision is made to temporarily shunt, shunt patency should be assessed by handheld Doppler examination throughout the case. At the conclusion of the orthopedic repair, the arterial repair should be reexamined to ensure that it has not been disrupted and 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. It is essential to **obtain proximal and distal control** of the injured artery before exploring the hematoma or wound. When feasible, an end-to-end anastomosis is preferable. A few centimeters of the artery can usually be mobilized proximally and distally to accomplish 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. A completion angiogram can help to document distal flow. This is especially important if significant spasm is present and distal pulses are not readily palpable, which can be treated with an intra-arterial infusion of 50 to 100 µg of nitroglycerin.

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 the hand is well perfused. Similarly, isolated injuries to a solitary tibial artery may be ligated if one or more of the tibial arteries remain intact and the foot is well perfused.

IV. COMPLICATIONS

A. Reperfusion injury results from the formation of oxygen-free radicals that directly damage the tissue and cause white blood cell accumulation and sequestration in the microcirculation. This process prolongs the ischemic interval because it impairs adequate nutrient flow to the tissue, despite the restoration of

axial blood flow.

B. Rhabdomyolysis following reperfusion releases the by-products of ischemic muscle, including potassium, lactic acid, myoglobin, and creatine phosphokinase. The electrolyte and pH changes that occur can trigger dangerous arrhythmias, and precipitation of myoglobin in the renal tubules can cause pigment nephropathy and acute renal failure. The likelihood that a patient will develop these complications relates to the duration of ischemia and the muscle mass at risk. **Aggressive hydration, diuresis with mannitol, and intravenous infusion of bicarbonate to alkalinize the urine** are accepted methods of mitigating renal impairment secondary to rhabdomyolysis.

C. Compartment syndrome results when prolonged ischemia and delayed reperfusion cause cell membrane damage and leakage of fluid into the interstitium. Additional muscle and nerve necrosis occurs when the intracompartmental pressures exceed capillary perfusion pressure (generally >30 mm Hg). A four-compartment fasciotomy should be performed when there is concern about the possible development of lower leg compartment syndrome; less commonly, thigh compartment fasciotomies may be indicated. In the upper extremity, fasciotomies of the forearm and hand may be needed to prevent development of compartment syndrome after emergent revascularization.

Fasciotomy should be routinely considered in any patient with more than 6 hours of acute extremity ischemia, or in the presence of combined arterial and venous injuries.

D. Followup care is usually directed at treating the underlying cause of the obstruction. Patients with mural thrombi or arrhythmias require long-term anticoagulation. The in-hospital mortality rate associated with embolectomy is as high as 30%.

CHRONIC ARTERIAL OCCLUSIVE DISEASE

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,** 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 may be subdivided into three anatomic sections on the basis of symptoms and treatment options. Aortoiliac occlusive disease, or Òinflow disease,Ó affects the infrarenal aorta and the common and external iliac arteries. FemoralÑpopliteal occlusive disease, or Òoutflow disease,Ó affects the common femoral, superficial femoral, and popliteal arteries. Finally, tibialÑperoneal disease, or Òrunoff disease,Ó affects the vessels distal to the popliteal artery. An algorithm for the clinical approach to claudication is shown in Figure 28-1.

I. CLINICAL PRESENTATION

A. Aortoiliac disease presents with **symptoms of lower-extremity claudication,** usually of the **hip, thigh, or buttock.** It may coexist with

femoralÑpopliteal disease, contributing to more distal symptoms as well. The symptoms usually develop gradually, although sudden worsening suggests acute thrombosis of a diseased vessel. Patients ultimately develop incapacitating claudication but not rest pain unless distal disease is also present. *Leriche syndrome* (sexual impotence, buttock and leg claudication, leg musculature atrophy, trophic changes of the feet, and leg pallor) is a constellation of symptoms that results from the gradual occlusion of the terminal aorta.

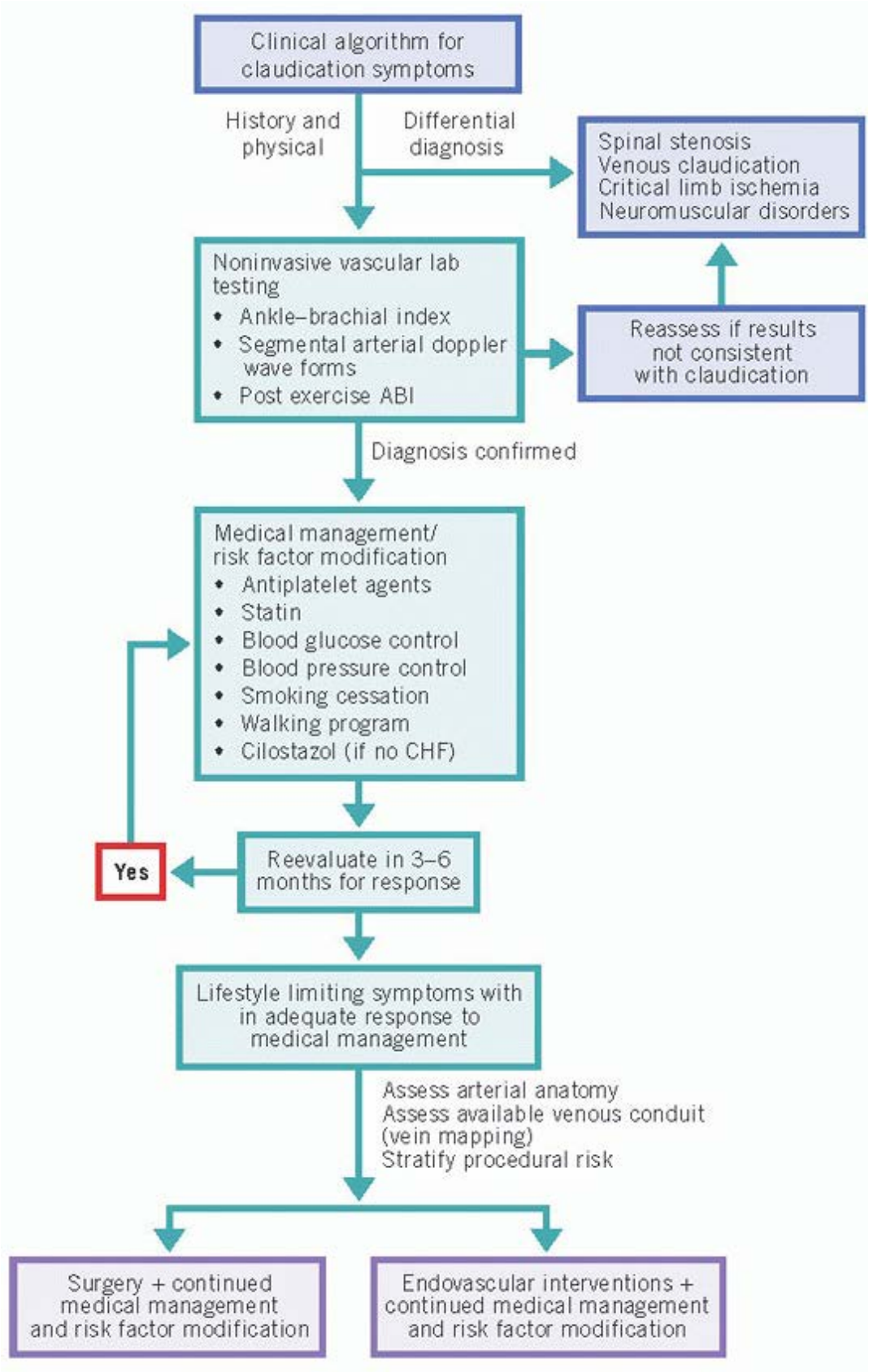

Figure 28-1 Clinical approach to claudication.

B. Patients with **femoralÑpopliteal** and **tibialÑperoneal 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, exacerbated by limb elevation, and often relieved by placing the leg in a dependent position. Examination findings of the chronically ischemic extremity include the following:

1. Decreased or absent distal pulses

2. Dependent rubor

3. Trophic changes that include thickening of the nails, loss of leg hair, shiny skin, and ulceration 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 fingerÑhand ischemia or necrosis. Occasionally, ulcerated plaques of the innominate or subclavian arteries can be a source of embolization to the hand.

2. Although many patients with proximal subclavian lesions are asymptomatic, **subclavian steal** can result when an occlusive subclavian artery lesion is located proximal to the origin of the vertebral artery. With exercise of the affected limb, the arm's demand for blood is supplied by retrograde flow in the ipsilateral vertebral artery, shunting blood from the posterior cerebral circulation and resulting in drop attacks, ataxia, sensory loss, or diplopia.

II. DIAGNOSIS.

Diagnosis of chronic arterial occlusive disease is concerned with determining the presence of **significant flow-limiting lesions** and distinguishing the disease from other conditions that may mimic it.

A. 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 stenoses have palpable pulses at rest that are lost after exercise. Bruits may also be appreciated over the lower abdomen or femoral vessels. It is also important to differentiate ulcers that arise from arterial insufficiency versus those generated by venous insufficiency and neuropathy.

1. Arterial insufficiency ulcers are usually painful and have an irregular appearance.

2. Neuropathic ulcers are painless and usually occur over bony prominences, particularly the plantar aspect of the metatarsophalangeal joints.

3. Venous stasis ulcers are located on the malleolar surface (ÒgaiterÓ distribution) and are dark and irregular in shape.

B. Segmental arterial Doppler readings with waveforms should be performed in all patients with suspected symptomatic arterial disease. The **ankle-brachial index (ABI),** the ratio of the systolic blood pressure in the leg to that in the arm, allows one to quantify the degree of ischemia. In general, patients without vascular disease have an ABI of 1.0, patients with claudication have an ABI of less than 0.8, and patients with rest pain and severe ischemia have an ABI of less than 0.4. Waveform changes help to localize the level of disease, and the severity of obstruction. Patients with history of claudication and normal resting waveforms require postexercise ABI measurements. In patients with diabetes and renal insufficiency, calcified vessels can result in a falsely elevated ABI measurement. Digit pressures are less affected by calcification and often provide a more accurate representation of arterial perfusion.

C. Digital subtraction arteriography is the gold standard for evaluating the arterial tree before planned revascularization. Typical digital subtraction arteriography of the lower extremities extends from the proximal abdomen to the toes. Noninvasive angiography using imaging modalities such as CTA and magnetic resonance angiography (MRA) has also gained widespread use. **CTA** produces high-resolution images of the vascular tree. However, diffuse calcifications may make interpretation of CT angiography images difficult. In addition, CT angiography does require iodinated contrast, which may adversely affect patients with renal insufficiency. **MRA** is an excellent imaging modality for assessing PAD and is useful for selecting patients who are endoluminal candidates. However, MRA may overestimate the degree of stenosis and may be inaccurate in stented arteries. Following the linkage of nephrogenic systemic fibrosis (NSF) to gadolinium administration in renal failure patients, alternative noncontrast MRA techniques have emerged (e.g., time-of-flight).

III. MANAGEMENT

A. The recently issued Society for Vascular Surgery Practice Guidelines for Atherosclerotic Occlusive Disease of the Lower Extremities (Conte et al., *J Vasc Surg.* 2015) offer detailed recommendations for management of patients with peripheral arterial disease. With adequate control of risk factors, intermittent claudication follows a benign course in most patients. In patients presenting with claudication alone, 70% to 80% remain stable or improve and 10% to 20% worsen over the ensuing 5-year period. Only 5% to 10% of patients develop gangrene and are at risk for limb loss. Therefore, **first-line treatment for patients with claudication should emphasize risk factor modification and structured exercise therapy.**

Despite this appropriate focus on medical optimization of the claudicant, **incapacitating claudication** that jeopardizes a patient's livelihood or

severely influences his or her quality of life may be considered for revascularization after failure of risk factor modification and exercise therapy.

Critical limb ischemia (CLI) is characterized by severely diminished arterial flow (ABI <0.4, and toe pressures <50 mm Hg), ischemic rest pain, and the development of ischemic ulceration or

pedal gangrene. The Society for Vascular Surgery Lower Extremity Threatened Limb Classification System (Mills et al., *J Vasc Surg.* 2013) uses the severity of limb ischemia, wound complexity, and foot infection to estimate the likelihood of limb salvage in CLI patients. In addition to maximal medical therapy, revascularization is indicated for symptom relief and limb preservation.

B. Medical Therapy. Society for Vascular Surgery Guidelines for Atherosclerotic Occlusive disease of the Lower extremities (Conte et al., *J Vasc Surg.* 2015) describe medical management and exercise therapy in detail.

1. Risk factor modification is the most important intervention for reducing 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. Lipid reduction is imperative in patients with PAD because the majority of the morbidity associated with PAD is related to cardiac events. On the basis of the Heart Protection Study involving statins, it is recommended to keep the low-density-lipoprotein level of patients with PAD less than 100 mg/dL, and <70 mg/dL for patients with disease in two vascular beds, to reduce the likelihood of morbidity associated with cardiac events *(J Vasc Surg.* 2007;45:645).

3. Antihypertensives should be administered to normalize blood pressure. The Intersociety consensus for the management of peripheral arterial disease (TASC II, *Eur J Vasc Endovasc Surg.* 2007) recommends blood pressure control to <140/90 mm Hg in all patients or <130/80 mm Hg if they also have diabetes or renal insufficiency.

4. Because many of these patients have concomitant coronary artery or cerebrovascular disease, daily **aspirin** therapy (81 or 325 mg) is indicated to reduce the risk of myocardial infarction or stroke.

5. Clopidogrel is an **antiplatelet agent** that has been shown to reduce cardiac and cerebral events in patients with systemic atherosclerosis. Although rigorous proof of its utility following peripheral arterial interventions is lacking, clopidogrel is frequently prescribed following these procedures.

6. Cilostazol is a type III **phosphodiesterase inhibitor** and the newest agent available for treatment of claudication. Cilostazol inhibits platelet aggregation and causes vasodilation. Given at 50 mg or 100 mg twice daily, it increases walking distances when compared with placebo and pentoxifylline. Early studies suggest that the drug is safe in most patients, although **its use is contraindicated in those with class III or IV heart failure** due to the toxicity of phosphodiesterase inhibitors in these patients.

C. Preoperative care of patients with PAD includes complete arterial imaging and procedural risk stratification. Myocardial complications account for the majority of early and late deaths. The American College of Cardiology/American Heart Association Guidelines on Perioperative

Cardiovascular Evaluation and Management (Fleisher et al., *Circulation.* 2014) offer a structured approach to the assessment and subsequent treatment of concomitant coronary artery disease.

D. Open Surgical Therapy

1. Aortoiliac occlusive disease

a. Aortobifemoral grafting is the treatment of choice in low-risk patients with diffuse aortoiliac stenoses and occlusions. Aortobifemoral bypass may be performed via transperitoneal or retroperitoneal approach. Distal endarterectomy and/or profundaplasty may be performed in conjunction with a bypass to improve outflow. Results are excellent, with reported patency rates of up to 95% at 5 years.

b. Femorofemoral bypass is an alternative in high-risk patients with unilateral iliac disease. The patency rates are lower than those achieved with aortobifemoral grafts.

c. Axillobifemoral bypass provides a less-invasive option for high-risk patients who need revascularization. This bypass avoids an intraabdominal procedure and the need for aortic cross-clamping. Patency rates are poorer than those achieved with aortobifemoral bypass.

d. Aortoiliac endarterectomy may be considered for patients who have disease localized to the distal aorta and common iliac vessels, although its use is now uncommon. Advantages include the avoidance of prosthetic material and preservation of antegrade flow into the hypogastric arteries.

2. Femoral, popliteal, and tibial occlusive disease

a. In patients with SFA occlusion, a femoralÑabove-knee popliteal bypass may be constructed. In patients who have disease below the knee, a distal bypass may be performed to the below-knee popliteal, posterior tibial, anterior tibial, or peroneal arteries. 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 unobstructed.

b. The best results are obtained with the use of autologous vein. Single-segment greater saphenous vein is the conduit of choice, but the lesser saphenous vein or the arm veins provide suitable alternatives. These autologous grafts can be used either in situ or reversed orientation. The advantages of the in situ bypass are that (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 advantage of the reversed-vein bypass is that endothelial trauma is minimized because valve lysis is not necessary.

c. When autologous vein is not available, polytetrafluoroethylene (PTFE) grafts and cryopreserved vein grafts can be used. Patency rates for PTFE above-knee grafts approach those achieved with venous conduit, but use of PTFE for more distal bypass procedures is associated with substantially lower patency and is reserved for patients with CLI who lack venous conduit. An alternative

technique when performing PTFE bypass is the use of a small cuff of vein (Miller cuff) or patch angioplasty (Taylor patch) at the distal anastomosis. These modifications appear to improve prosthetic graft patency by improving compliance match at the distal anastomosis. Cryopreserved vein graft patency also fares poorly in comparison to autologous conduit, but may prove useful when bypass is required in an infected field.

d. Endarterectomy is most commonly used to address severe stenosis or occlusion of the common femoral and profunda femoris arteries.

e. Amputation is reserved for patients with gangrene or persistent painful ischemia not amenable to vascular reconstruction. These patients often have severe coexistent vascular and cardiovascular disease, and the survival rate for patients undergoing major amputations is approximately 50% at 3 years and 30% at 5 years.

(1) The level of amputation is determined clinically. Important factors include the necessity of removing all the infected tissue and the adequacy of the blood supply to heal the amputation. A general principle is to preserve as much length of the extremity as safely possible, as this improves the patient's opportunity for rehabilitation.

(2) Digital amputations are performed for isolated gangrene and/or recalcitrant osteomyelitis.

(3) Transmetatarsal amputations are usually performed when several toes are involved in the ischemic process or after previous single-digit amputations.

(4) Below-knee amputation (BKA) is the most common type of amputation performed for patients with severe occlusive disease.

(5) Above-knee amputation (AKA) heals more easily than BKA and is useful in older patients who do not ambulate.

(6) Hip disarticulation is rarely 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, carotidÑsubclavian bypass is performed through a supraclavicular approach using a prosthetic graft (vein grafts are to be avoided). Subclavian artery transposition to ipsilateral carotid artery is an excellent alternative if anatomically feasible.

c. If the ipsilateral carotid artery is occluded, subclavianÑsubclavian bypass may be performed. This extraanatomic approach uses a longer segment prosthetic graft, with reduced patency.

4. Intraoperative anticoagulation is employed during most vascular reconstructions. Generally, unfractionated heparin (100 units/kg) is administered intravenously shortly before cross-clamping

and supplemented as necessary until the cross-clamps are removed. Anticoagulation can be monitored intraoperatively by following activated clotting time (ACT) levels, with a goal of greater than 250 seconds. The anticoagulant effect of heparin can be reversed with protamine administration. For patients with heparin-induced thrombocytopenia, direct thrombin inhibitors such as bivalirudin are preferred.

E. Postoperative Care

1. Open aortic procedures are initially managed in the intensive care unit, due to the need for continuous monitoring and rapid intervention. Assessment of distal pulses should be done regularly. Early ambulation is encouraged.

2. For distal bypass grafts, **pulses should be assessed frequently.** Antibiotics are continued for 24 hours, or longer if infected ulcers warrant additional treatment. Early ambulation is encouraged in patients without tissue necrosis. In patients who are unable to ambulate immediately, physical therapy can help to 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 instructed to elevate their legs while resting because this will mitigate the edema that develops in the revascularized extremity.

3. Perioperative antithrombotic therapy should include aspirin (81 to 325 mg/day) for all infrainguinal reconstructions. In patients sensitive to aspirin, clopidogrel (75 mg/day) may be substituted.

4. Postoperative oral anticoagulation has a more limited role. Owing in part to the increased risk of hemorrhage, anticoagulation with warfarin (INR 2 to 3) is generally limited to grafts considered to be at a high risk for thrombosis.

5. Following major amputations, weightbearing is delayed for 4 to 6 weeks. Some advocate the use of compressive wraps to aid in the maturation of the stump. In all cases, early consultation with a physical therapist and prosthetist is recommended.

6. Surveillance of distal bypass grafts consists of serial evaluations of vein graft patency by clinical examination and duplex ultrasound. Less frequent followup is necessary for aortoiliac bypasses. Detection of severe stenosis predicts impending graft failure, and such grafts should undergo arteriography and correction. Repair or revision of stenosed grafts results in higher long-term patency than repairing or replacing occluded grafts.

F. Complications

1. Early complications occur in approximately 5% to 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 dilation, 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** include graft kinks, retained valve leaflets, valvulotome trauma, intimal flaps, significant residual arteriovenous fistulas, and the use of a poor quality conduit.

G. Endovascular Options

1. Aortoiliac occlusive disease

a. Indications. Balloon angioplasty and intravascular stent placement for aortoiliac occlusive lesions produce excellent results. These procedures are indicated for symptomatic stenotic or occlusive lesions. Short-segment stenoses (less than 3 cm in length) of the common and external iliac arteries display excellent long-term patency rates when treated with angioplasty alone, or with stent placement. Angioplasty failure (defined as residual stenosis of ³30%, residual mean translesional pressure gradient of ³10 mm Hg, or flow-limiting dissection) is an indication for stent deployment.

b. Technique. Access for iliac artery angioplasty and stenting is generally via a femoral arterial approach. When the occlusive lesion is in the distal aorta or ostial common iliac artery, angioplasty should be performed using two balloons, one in each iliac artery and both partially projecting into the distal aorta (Òkissing balloonsÓ). The rationale for this technique is that lesions in proximity to the aortic bifurcation typically involve the distal aorta and both common iliac arteries. Unilateral balloon dilation may cause plaque shifting with compromise of the contralateral iliac artery lumen. Stenting may produce a more favorable result if postangioplasty dissection or lesion recoil is noted. Balloon-expandable and self-expanding stents are generally oversized 10% to 15% relative to the adjacent normal artery to ensure satisfactory stent apposition to the vessel wall. If stent deployment is required in proximity to the aortic bifurcation, Òkissing stentsÓ are utilized in a fashion similar to that described above.

c. Complications. Procedural complications of iliac angioplasty and stenting include arterial dissection, vessel occlusion, arterial rupture, and distal embolization, which may result in the need for surgical intervention or amputation.

d. Results. Immediate balloon angioplasty failure can result from elastic recoil of atherosclerotic plaque or arterial wall dissection. These complications are potentially amenable to stent placement. Early failure is usually due to intimal hyperplasia, whereas late failure may

also be caused by progressive atherosclerosis. Iliac artery balloon angioplasty 2-year patency rates between 60% and 70% have been reported. Reports of iliac artery stenting demonstrate 4-year patency rates as high as 85%. In general, the results of angioplasty and stenting are better for

common iliac artery lesions than for external iliac artery lesions, and are better for short-segment disease than for long-segment disease.

2. Infrainguinal occlusive disease

a. Indications. Balloon angioplasty and stenting of infrainguinal occlusive lesions has been widely applied for the treatment of claudication and CLI. See Figures 28-2, 28-3 and 28-4 for an example of angiographic intervention for superficial femoral artery occlusion. Aggressive modification of risk factors, institution of antiplatelet and statin medications, and a trial of exercise therapy are recommended prior to intervention, particularly in the setting of claudication. The Trans-Atlantic Inter-Society Consensus (TASC II) group has provided recommendations regarding the characteristics of femoropopliteal and infrapopliteal lesions that are best addressed by either endovascular

or surgical therapy (Table 28-2). Short, focal stenoses (TASC A) are felt to be amenable to endovascular therapy, whereas long-segment occlusions (TASC D) are best addressed by surgical bypass.

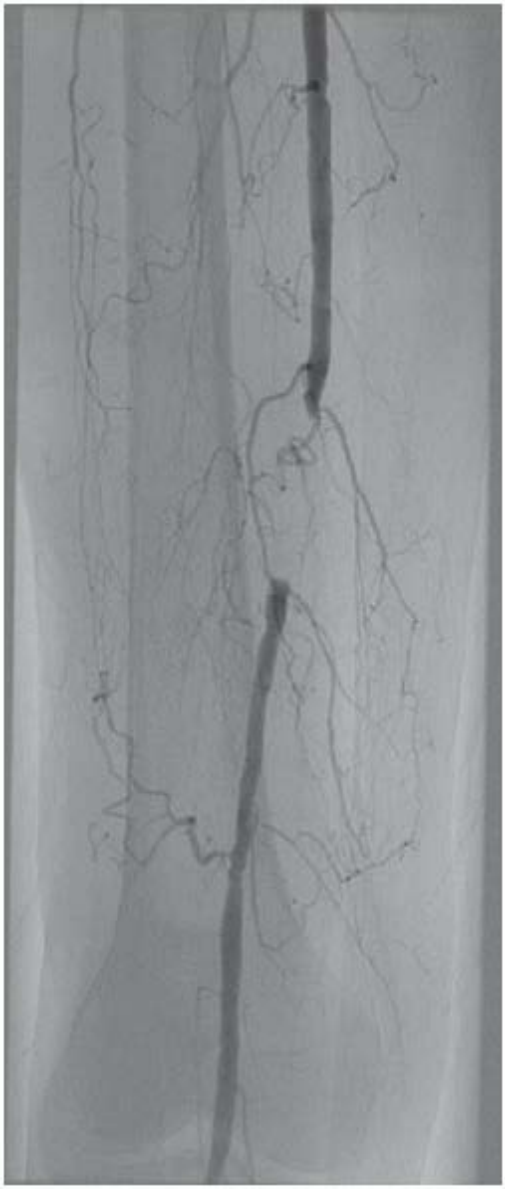

Figure 28-2 Superficial femoral artery occlusion prior to treatment.

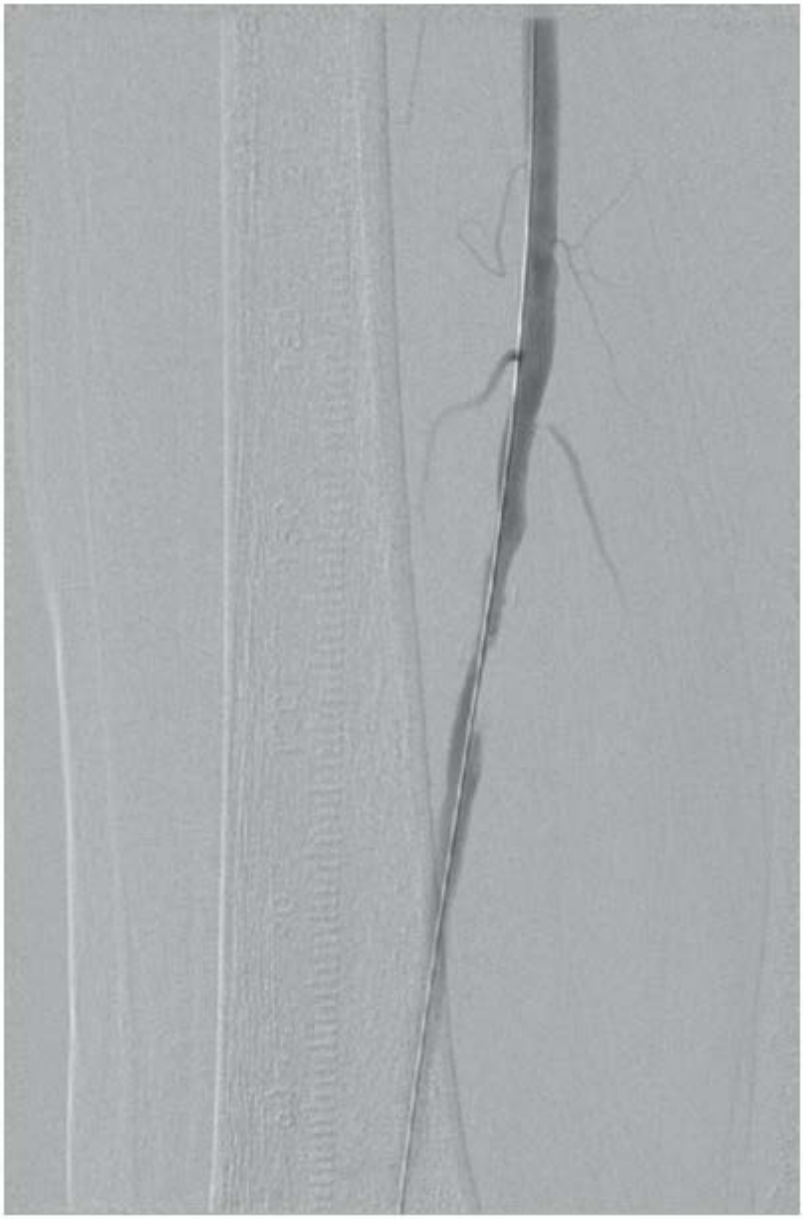

Figure 28-3 Recoil following balloon angioplasty.

b. Technique. Arterial access for infrainguinal intervention is usually accomplished via retrograde contralateral femoral artery approach or ipsilateral antegrade femoral artery approach. Retrograde tibial/pedal access is also being studied as an alternate access option. The most frequent cause of treatment failure is the inability to negotiate across the stenosis or occlusion and into the distal outflow target vessel. In general, once guidewire access to the distal target vessel has been established, technical success rates are excellent. Hydrophilic guidewires and catheters, occlusion crossing devices, lumen reentry devices, and specialized sheaths have been developed to facilitate this process.

c. Complications. Procedural complications of infrainguinal endovascular intervention include

perforation, flow-limiting dissection, arteriovenous fistula formation, and distal embolization. Severe complications may require surgical intervention or, rarely, amputation.

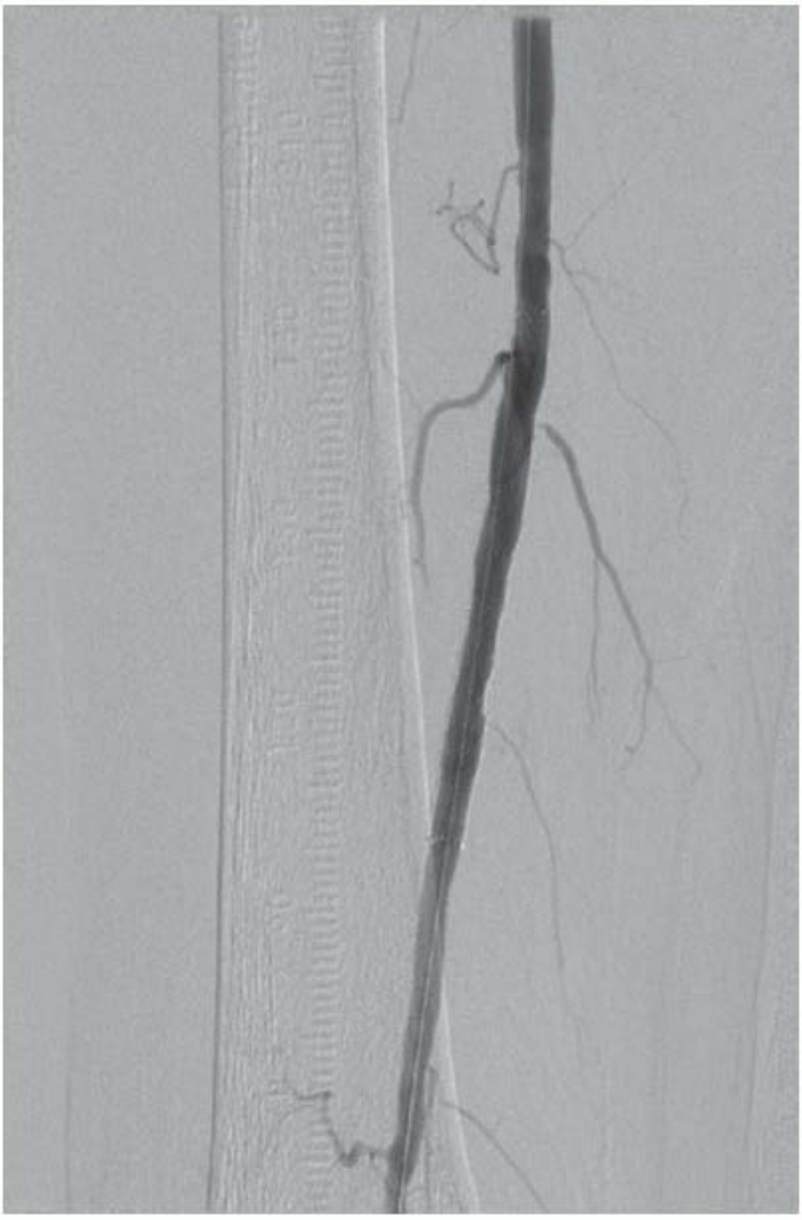

Figure 28-4 Completion angiogram following balloon angioplasty and stenting.

d. Results. Unfortunately, mid- and long-term outcomes data for endovascular intervention in the treatment of claudication and CLI remain relatively scarce. For moderate severity lesions of the femoropopliteal distribution, the ABSOLUTE trial demonstrated that primary nitinol stenting may provide a patency advantage over plain balloon angioplasty, and that this may be sustained

through 2 years of followup *(NEJM.* 2006;354:1879). Biologic modification of the intimal hyperplastic response to endovascular intervention appears to also hold promise. The ZILVER PTX randomized controlled trial of paclitaxel-eluting nitinol stents for femoropopliteal disease has shown superior 4-year patency rates compared to angioplasty alone or bare metal stent deployment. More recently, paclitaxel-eluting balloon angioplasty has been shown to increase 1-year patency rates

for SFA intervention. Perhaps the most compelling data regarding endovascular versus surgical intervention have been derived from the Bypass versus Angioplasty in Severe Ischemia of the Leg (BASIL) trial from the United Kingdom *(J Vasc Surg.* 2010;51:52S). Although the initial results reported from the trial were widely interpreted as demonstrating equivalency between angioplasty and bypass surgery for this patient cohort, longer-term followup has shown an advantage in both overall survival and amputation-free survival in those patients who underwent bypass surgery and survived beyond 2 years. Interestingly, the BASIL investigators noted that outcomes were worse for patients who underwent angioplasty followed by salvage bypass surgery, rather than a bypass-first approach. Good surgical candidates with complex anatomic lesionsÑin particular, those possessing good venous conduitÑshould be considered for surgical reconstruction. Endovascular intervention is the preferred approach for the medically compromised patient, particularly those lacking autologous venous conduit. Finally, hybrid open surgical/endovascular procedures are frequently utilized in the treatment of CLI. Vascular surgeons who are skilled in both open surgical reconstruction and endovascular interventions will therefore tailor their therapeutic approach based on each patient's unique risk factors and arterial anatomy.

TABLE 28-2 TASC II Classification

TASC Classification	Lesion Characteristics
A	Single stenosis <10 cm Single occlusion <5 cm
B	Multiple lesions <5 cm Single or multiple lesions in the absence of continuous tibial vessels Single stenosis/occlusion <15 cm Heavily calcified occlusion <5 cm Single popliteal stenosis

C	Multiple stenoses/occlusions totaling >15 cm Recurrent stenoses/occlusions needing intervention after two prior interventions
D	Chronic total occlusions of CFA or SFA Chronic total occlusion of popliteal and proximal trifurcation vessels

CFA, common femoral artery; SFA, superficial femoral artery; TASC, The Trans-Atlantic Inter-Society Consensus.

CHAPTER 28: PERIPHERAL ARTERIAL DISEASE

Multiple Choice Questions

1. A 55-year-old female patient with previous history of hypertension and smoking presents to the emergency room with severe pain in her left foot. Which of the following examination findings would make you suspicious of embolic phenomenon rather than progressive atherosclerotic disease?

a. Shiny skin with absence of hair and loss of subcutaneous fat in both feet

b. Palpable pedal pulses in right foot and no history of claudication

c. Palpable pedal pulses in left foot

d. Pale and cold left foot

e. Necrotic ulcer on left foot

View Answer

2. A 47-year-old male with medical history of smoking, diabetes, hypertension, and hyperlipidemia presents to your office with right calf claudication at 6 blocks of ambulation. He works as a lawyer and states that the symptoms do not hinder his desired activities. ABIs are R = 0.6, L = 1.1. What is the appropriate initial management of this patient?

a. Begin anticoagulation with Lovenox

b. Risk factor modification and structured exercise

c. Schedule angiography and stent placement in the interventional suite

d. Schedule CT angiography, and book OR for right femoral-popliteal bypass grafting

e. Prescribe 20 to 30 mm Hg graded compression stockings

View Answer

3. You have completed a femoral embolectomy on a patient in atrial fibrillation who presented with an 18-hour history of acute onset left leg pain accompanied by calf muscle weakness and loss of sensation. The pedal pulses are now palpable, but the calf muscle is tense. You should:

a. Apply nitropaste to the foot

b. Administer systemic thrombolytics

c. Perform four-compartment calf fasciotomies

d. Recommend early ambulation

e. Begin cilostazol

View Answer

4. A 51-year-old male smoker with past medical history of type 1 diabetes mellitus, hypertension, congestive heart failure, COPD presents to you with intermittent bilateral calf claudication for 6 months and upon further evaluation his ABIs are consistent with his symptoms (0.7 bilaterally). You counsel him regarding smoking cessation, blood pressure control, and glucose control. Which of the following medications should be added to his management first?

a. Statin

b. Cilostazol

c. Pentoxifylline

d. Coumadin

e. Clopidogrel

View Answer

5. A 19-year-old male restrained front seat passenger is brought by EMS following a head on motor vehicle collision. During an initial primary and secondary survey, no pulse can be felt in his left foot and there appears to be a deformity of his left knee. He is complaining of severe pain in his left knee and down to his foot. There is no obvious hematoma in the vicinity of left popliteal fossa. X-rays of the left knee are performed and reveal a posterior dislocation. What is the next step in management?

a. Perform a CT angiogram of left lower extremity

b. Perform an angiogram of left lower extremity in the operating room

c. Perform a reduction of left knee dislocation with sedation in the ER

d. Exploration of left popliteal fossa in the operating room

e. Perform formal ABIs in the vascular laboratory

View Answer

29

Venous and Lymphatic Disease

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VENOUS ANATOMY

Venous anatomy is divided into three compartments: Superficial, perforating veins, deep. In general, blood flows from the superficial to the deep veins through the perforating system. In the lower extremity, the major superficial veins are the *greater saphenous vein,* formed from the union of the dorsal vein of the great toe and the dorsal venous arch; the *small saphenous vein,* formed from the joining of the dorsal vein of the fifth toe and the dorsal venous arch; and the *posterior arch vein,* also called *Leonardo 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 deep veins of the calf typically are duplicated as venae comitantes with numerous communicating branches. The posterior tibial and peroneal veins also communicate with the soleal sinusoids. In the thigh, the deep venous system includes the femoral and deep femoral veins that join approximately 4 cm below the inguinal ligament. Perforating veins connect the superficial and deep systems through both direct and indirect mechanisms. Venous return from the lower extremities depends largely on compression of the deep veins by the muscles of the calf (gastrocnemius, soleus) during walking. Flow is unidirectional due to a series of one-way valves, which prevent reflux during this cycle of compression. Failure of these valves to close leads to pooling, stasis, and congestion of veins in the lower extremities, and subsequent dilation of the superficial veins.

CHRONIC VENOUS INSUFFICIENCY

Chronic venous disease includes telangiectasias, varicose veins, venous ulceration, and claudication.

I. PATHOPHYSIOLOGY

A. Etiology

1. Congenital

2. Primary (cause undetermined)

3. Secondary (postthrombotic, posttraumatic, or other)

B. Risk Factors

1. Obesity

2. Tobacco use

3. Multiparity

4. Hormone therapy

5. Obstruction (e.g., from adenopathy, arterial compression, or pregnancy)

6. History of deep venous thrombosis (DVT). DVT accounts for most secondary cases and may be responsible for a significant number of other cases because many deep vein thrombi are asymptomatic.

C. Reflux disease from venous valvular incompetence accounts for most (>80%) chronic venous disease.

1. Valve malfunction can be inherited or acquired through sclerosis or elongation of valve cusps or dilation of the valve annulus despite normal valve cusps.

2. 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 venous disease.

3. Valvular disease below the knee appears to be more critical in the pathophysiology of severe venous disease than disease above the knee.

4. 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.

5. All of the above components need evaluation in the workup of chronic venous insufficiency (CVI) *(Am Surg.* 2010;76:125).

D. Obstructive physiology is a less common cause of venous pathology, with reflux often being present simultaneously.

II. DIFFERENTIAL DIAGNOSIS

A. Arterial Disease

1. Ulcers with discrete edges and pale bases; more painful than venous ulcers, generally at the tips of the toes

2. Poor or absent pulses on examination

3. Dependent rubor

4. Pallor with elevation

5. Claudication

B. Lymphedema

1. Pitting edema without pigmentation and ulceration

2. Less responsive to elevation, usually requires several days to improve

C. Squamous Cell Carcinoma—can occur in patients with chronic wounds or de novo. A biopsy is required for diagnosis.

D. Trauma

E. Arteriovenous Malformation

F. Orthostatic Edema

III. NOMENCLATURE

A. CEAP Classification

1. Standardized nomenclature of chronic venous disease *(J Vasc Surg.* 2004;40:1248; *Eur J Vasc Endovasc Surg.* 1996;12:487).

2. CEAP: Clinical signs, Etiology, Anatomic distribution, and Pathophysiology (Table 29-1).

TABLE 29-1 Classification of Chronic Lower-extremity Venous Disease

Classification	Definition
C	Clinical classification
	C_0: No visible or palpable signs of venous disease
	C_1: Telangiectasias or reticular veins
	C_2: Varicose veins; distinguished from reticular veins by a diameter of 3 mm or more
	C_3: Edema
	C_4: Changes in skin and subcutaneous tissue secondary to CVD
	C : Pigmentation or eczema

	4a
	C_{4b}: Lipodermatosclerosis or atrophie blanche
	C_5: Healed venous ulcer
	C_6: Active venous ulcer
	S: Symptomatic (includes aching, pain, tightness, skin irritation, heaviness, muscle cramps, and other complaints attributable to venous dysfunction)
	A: Asymptomatic
E	Etiologic classification
	E_c: Congenital
	E_p: Primary
	E_s: Secondary (i.e., postthrombotic)
	E_n: No venous cause identified
A	Anatomic distribution
	A_s: Superficial veins involved
	A_p: Perforator veins involved
	A_d: Deep veins involved
	A_n: No venous location identified

P	Pathophysiologic dysfunction
	P_r: Reflux
	P_o: Obstruction
	$P_{r,o}$: Reflux and obstruction
	P_n: No venous pathophysiology identified

CVD, chronic venous disease.

B. Venous Clinical Severity Score (VCSS)

1. Developed by the American Venous Forum in 2000, and revised in 2010; expands the existing system.

2. Ten clinical descriptors: Pain, varicose veins, venous edema, skin pigmentation, inflammation, induration, number of active ulcers, duration of active ulceration, size of ulcer, and compressive therapy use.

3. Better assesses ongoing response to therapy.

IV. DIAGNOSIS

A. History

1. A history of any DVT or trauma.

2. Family history of varicose veins or CVI.

3. Complaint 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.

4. In rare instances, 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

1. Ankle edema

2. Subcutaneous fibrosis

3. Hyperpigmentation (brownish discoloration secondary to hemosiderin deposition)

4. Lipodermatosclerosis

5. Venous eczema

6. Subcutaneous vein dilatation, including telangiectasias (0.1 to 1 mm), reticular veins (1 to 4 mm), and varicose veins (>4 mm)

7. Ulcers, typically proximal to the medial malleolus

8. Any signs of infection should be noted

9. Pulse examination

C. Noninvasive Studies

1. Duplex scanning

a. B-mode ultrasound imaging combined with Doppler frequency shift display.

b. Used in assessing venous valvular competence and obstruction, presence of acute or chronic DVT.

c. With the leg in a dependant position, 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.

d. Competent valves generally take no more than 0.5 to 1 second to close.

e. Detailed mapping of valve competence of each segment of the venous system is possible, including the common femoral, greater saphenous, lesser saphenous, popliteal, posterior tibial, and perforator veins.

f. Has a PPV of 77% for diagnosing reflux leading to severe symptoms.

2. Continuous wave Doppler

a. Easily performed in the office using a handheld probe.

b. Helpful for screening reflux at the saphenofemoral and saphenopopliteal junctions.

c. Limited clinical use due to inability to quantitate reflux and to provide precise anatomic information.

3. Trendelenburg test

a. Largely replaced by the much more accurate duplex imaging studies.

b. Patient's leg is elevated to drain venous blood. An elastic tourniquet is applied at the saphenofemoral junction, and the patient then stands.

c. Rapid filling (<30 seconds) of the saphenous system from the deep system indicates perforator valve incompetence.

d. When tourniquet is released, additional filling of the saphenous system occurs if the saphenofemoral valve is also incompetent.

4. Descending phlebography has a positive predictive value of 44%, and is limited by its inability to study valves distal to a competent proximal valve.

V. NONSURGICAL TREATMENT

A. Infected Ulcers

1. Necessitates treatment of the infection first.

2. *Staphylococcus aureus, Streptococcus pyogenes,* and *Pseudomonas* species are responsible for most infections.

3. Usually treated with local wound care, wet-to-dry dressings, and oral antibiotics.

4. Topical antiseptics should be avoided.

5. 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 an adjunctive treatment for CVI.

1. Elastic compression stockings

a. Fitted to provide a compression gradient from 30 to 40 mm Hg, with the greatest compression at the ankle.

b. Should be worn from awakening and removed at bedtime.

c. Effective in healing ulcers, *but can take months* to obtain good results.

d. Study of 113 patients treated with initial bed rest, local wound care, and elastic compression stockings demonstrated a 93% ulcer healing rate in a mean of 5.3 months *(Surgery.* 1991;109:575).

e. Stockings do not correct the abnormal venous hemodynamics and must be worn after the ulcer has healed to prevent recurrence.

f. Patient compliance is principal limiting factor.

g. Recurrence for compliant patients in the same study was 16% at a mean followup of 30 months.

2. Unna boots

a. Employed when there is actual skin ulceration, they combine compression therapy with a zinc oxide paste that assists in wound healing. Used to help prevent further skin breakdown.

b. Provide nonelastic compression therapy.

c. Changed once or twice a week.

3. Pneumatic compression devices

a. Provide dynamic sequential compression.

b. Used primarily in the prevention of deep vein thrombi in hospitalized patients.

c. Also used successfully to treat venous insufficiency.

D. Topical Medications

1. Largely ineffective as a stand-alone therapy for venous stasis ulcers.

2. Topical therapy is directed at absorbing wound drainage and avoiding desiccation of the wound.

3. 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.

VI. SURGICAL THERAPY.

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 sclerotherapy, saphenous vein stripping, endovenous ablation of the saphenous vein, Subfascial Endoscopic Perforating Vein Surgery (SEPS), and varicose vein stab avulsion.

A. Sclerotherapy

1. Effective in treating telangiectasias, reticular varicosities, and small varicose veins.

2. If saphenous reflux is present, it should be corrected first.

3. Contraindications include arterial occlusive disease, immobility, acute thrombophlebitis, and hypersensitivity to sclerosing agent.

4. Sclerosing agents

a. Sodium tetradecyl sulfate

b. Sodium morrhuate

c. Hypertonic saline

d. Polidocanol

5. Varices are marked while the patient is standing. A 25-gauge needle is used to inject 0.25 to 0.50 mL of sclerosant slowly into the lumen of larger veins. A 30-gauge needle is used for sclerosing reticular veins and telangiectasias in supine patients.

6. 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.

7. Complications include cutaneous necrosis, hyperpigmentation, telangiectatic matting (new, fine, red telangiectasias), thrombophlebitis, anaphylaxis, allergic reaction, visual disturbances, venous thromboembolism

(VTE), and even death *(J Vasc Surg.* 2010;52:939; *Dermatol Surg.* 1995;21:19).

B. Saphenous vein stripping, once considered the gold standard for superficial venous surgery, has since been replaced by the use of minimally invasive techniques.

C. Endovenous Ablation of the Saphenous Vein

1. This was shown to effectively treat saphenous reflux and associated varicose veins with less morbidity than saphenectomy *(J Vasc Surg.* 2003;38:207).

2. A probe is inserted into the greater saphenous vein under ultrasound guidance. The probe emits either laser or radiofrequency energy, which coagulates and coapts the vein walls, causing complete obliteration of the lumen.

3. Potential complications

a. Skin burns

b. DVT

c. Pulmonary thromboembolism

d. Vein perforation and hematoma

e. Paresthesias

f. Phlebitis

4. Reported outcomes achieved with endovenous radiofrequency and laser obliteration are comparable to those resulting from saphenectomy *(Ann Vasc Surg.* 2010;24:360; *J Vasc Interv Radiol.* 2009;20:752; *J Vasc Surg.* 2008;47:151). Incomplete obliteration and recanalization occur in a small percentage of patients.

5. A contraindication to endovenous obliteration is saphenous vein thrombosis.

D. Subfascial Endoscopic Perforating Vein Surgery (SEPS)

1. This is associated with decreased morbidity as compared to vein stripping and has gained recognition as an alternative treatment option.

2. 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) can 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, 3 to 14 perforators are identified and ligated.

3. Most patients are discharged within 24 hours of surgery.

E. Varicose Vein Stab Avulsion

1. Preoperatively, the patient's varicose veins are carefully marked with indelible ink while the patient is standing.

2. Small incisions (2 to 3 mm) are made next to the markings. The vein is elevated from the incision with a small vein hook, divided, and avulsed from the subcutaneous tissue. This process 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 wrapped with a compression stocking for several days to weeks. This technique is often used in conjunction with other modalities to provide optimal results.

VENOUS THROMBOEMBOLISM

I. EPIDEMIOLOGY.

Venous thromboembolic disease represents a significant problem, with 250,000 hospitalizations for deep vein thrombosis/pulmonary embolus (DVT/PE) annually. Approximately 50% to 60% of DVT episodes are asymptomatic. Of those patients with DVTs, 30% will have a symptomatic PE with a mortality of 17.5% if untreated. DVT and PE can occur in approximately 10% to 40% of general surgical patients without perioperative prophylaxis, and 40% to 60% following major orthopedic surgery *(Chest.* 2008;133:381S-453S).

II. PATHOPHYSIOLOGY.

DVT starts as a platelet nidus, usually on the venous valves of the calf. The thrombogenic nature of the nidus activates the clotting cascade, leading to platelet and fibrin accumulation. The fibrinolytic system is subsequently activated, with thrombus propagation if thrombogenesis predominates over thrombolysis. A thrombus can detach from the endothelium and migrate into the pulmonary system, becoming a PE; alternatively, it can also organize and grow into the endothelium, resulting in venous incompetency and phlebitis. Thrombi localized to the calf have less tendency to embolize than thrombi that extend to the thigh veins *(Am Rev Respir Dis.* 1990;141:1). Approximately 20% of cases of calf DVT propagate to the thigh, and 50% of cases of thigh or proximal DVT embolize.

III. RISK FACTORS FOR VENOUS THROMBOEMBOLISM

A. Malignancy

B. Endothelial Injury

C. Venous Stasis

D. Oral Contraceptives (OCPs)

1. These have been linked to increased risk of venous thrombus formation. Many studies have found an odds ratio of 3 to 5 for risk of DVT in patients taking OCPs compared to nonÑOCP-using patients. Smoking and increased age increase the risk of DVT formation for patient taking OCPs.

E. Hypercoagulable States

1. Primary hypercoagulable states are inherited conditions that can lead to abnormal endothelial cell thromboregulation.

2. Secondary hypercoagulable states are states in which endothelial activation by cytokines leads to an inflammatory, thrombogenic vessel wall.

IV. DIAGNOSIS

A. Initial Evaluation

1. Approximately 75% of patients with suspected DVT or PE do not have a hypercoagulable condition.

2. Assessment of risk factors (see Section III).

3. Clinical presentation

a. Extremity pain

b. Increased calf circumference with respect to contralateral extremity

c. Dilation of superficial veins of the suspected extremity only

d. Calf pain on dorsiflexion of the ankle

e. Phlegmasia cerulea dolens represents a more severe manifestation of DVT in which the deep venous channels of the extremity are affected *while sparing collateral veins* and therefore maintaining some degree of venous return. Patients present with blanching of the extremity, edema, and discomfort.

f. Phlegmasia alba dolens occurs with extension of thrombus into the collateral venous system, resulting in limb pain and swelling, accompanied by cyanosis, a sign of arterial ischemia.

B. Suspected DVT

1. Duplex 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.

2. It represents the preferred diagnostic modality because it is less invasive than the reference standard of venography and is more sensitive than

impedance plethysmography.

3. Approximately 2% of patients with initial normal ultrasound results have positive results on repeat tests performed 7 days later. Delayed detection rate is attributed to extension of calf vein thrombi or small, nonocclusive proximal vein thrombi.

C. Assessment of PE

1. Contrast-enhanced spiral chest computed tomography (CT) has sensitivity (70% to 90%) comparable to that of pulmonary angiography. Chest CT is preferable to pulmonary angiography (less invasive and less expensive).

2. Chest CT can be combined with CT angiography of pelvic and deep thigh veins to detect DVT as well as PE.

3. Radionucleotide ventilation and perfusion lung imaging (V/Q scan) has been replaced by chest CT as the initial imaging test for suspected PE. V/Q scanning is used in situations in which CT is deemed not feasible. A V/Q scan result of "high probability" 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.

4. Pulmonary angiography, the reference test, is reserved for patients in whom diagnosis is still uncertain.

V. PREVENTION AND TREATMENT OF VENOUS THROMBOEMBOLISM.

For anticoagulation treatments following specific procedures, please see the recent guidelines published by the American College of Chest Physicians *(Chest.* 2012;141(2 Suppl):7S-47S).

A. Low-Dose Unfractionated Heparin (LDUH)

1. Administered subcutaneously at 5,000 units 1 to 2 hours preoperatively, and every 8 or 12 hours postoperatively *(N Engl J Med.* 1988;318:18).

2. LDUH reduces the risk of VTE by 50% to 70% *(N Engl J Med.* 1988;318:18) 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.

B. Graduated Compression Stockings

1. Effective in preventing DVT formation by reducing venous stasis.

2. In surgery patients, the use of graduated compression stockings appears to augment the protective benefit of low-dose heparin by nearly 75%. The combination of graduated compression

stockings and LDUH is significantly more effective than LDUH alone, with DVT rates of 4% and 15%, respectively *(Cochrane Database Syst Rev.* 2000;1:CD001484; *Br J Surg.* 1985;72:7).

3. Graduated compression stockings are relatively inexpensive and should be considered for all high-risk patients, even when other forms of prophylaxis are used. Furthermore, the early use of either over-thecounter or custom-fit stockings following diagnosis of DVT results in a reduction in the incidence of postthrombotic syndromes *(Lancet.* 1997;349:759; *Ann Intern Med.* 2004;141:249).

C. Intermittent Pneumatic Compression of the Extremities

1. Enhances blood flow in the deep veins, and increases blood fibrinolytic activity through upregulation of thrombomodulin, fibrinolysin, t-PA, and endothelial nitric oxide synthase expression *(Acta Anaesthesiologica Scandinavica.* 2005;49:660).

2. For patients with significant bleeding risk with anticoagulation, pneumatic compression is an effective alternative.

3. Compression devices should not be placed on an extremity with known DVT.

4. In the case of known bilateral lower-extremity DVT, compression devices can be placed on the upper extremity, as the upregulated agents have systemic effects.

D. Low-Molecular-Weight Heparins (LMWHs)

1. Several advantages over unfractionated heparin, including longer half-lives, a more predictable doseÑresponse curve, a lower risk of heparin-induced thrombocytopenia (HIT), and it allow for ambulatory therapy.

2. In large randomized trials of patients with DVT, outpatient treatment with an LMWH was as safe and effective as inpatient treatment with intravenous unfractionated heparin *(J Thromb Thrombolysis.* 2005;19(3):173-181). Per CHEST Guidelines published in 2012, enoxaparin is recommended over unfractionated heparin.

E. Other medications such as the direct thrombin inhibitors (DTIs) and fondaparinux represent a possible alternative to the unfractionated and LMWHs in the prevention of thromboembolic disease.

F. Caval Interruption with Intracaval Filters. The American College of Chest Physicians recommends inferior vena cava (IVC) filter placement only in those patients with proven VTE with a contraindication for anticoagulation, a complication of anticoagulation, or recurrent VTE despite adequate anticoagulation. No randomized trials have examined the prophylactic use of IVC filters in any patient population. In fact, several meta-analyses found no difference in the rates of PE among patients with and without prophylactic IVC filters *(J Trauma.* 2000;49:140; *J Am Coll Surg.* 1999;189:314). Absolute and relative indications for caval interruption are listed in Table 29-2 *(Chest.* 2012;141(2 Suppl):7S-47S; *Am J Med.* 2007;120:S13; *Prog Cardiovasc Dis.* 2006;49:98; *J*

Am Coll Surg. 2005;201:957; *Chest.* 2004;126:401S; *J Vasc Interv Radiol.* 2003;14:425; *Blood.* 2000;95:3669). Complications related to filter insertion occur in 4% to 11% of patients. The most common complications are thrombotic in nature: Insertion site thrombosis (2% to 28%); IVC thrombosis (3% to 11%); and recurrent DVT (6% to 35%). Other complications include filter migration, penetration of the IVC, filter fracture, vena caval obstruction, and guidewire entrapment. The specific types of retrievable and permanent filters are beyond the scope of this chapter, but the use of retrievable filters can reduce

the incidence of thrombotic complications *(Am J Med.* 2007;120:S13). A simplified algorithm for IVC filter placement is found in Figure 29-1.

TABLE 29-2 Use of Inferior Vena Cava Filters

Absolute indications *(strongly* **recommended according to evidence-based guidelines)**

Proven VTE with contraindication for anticoagulation.

Proven VTE with complication of anticoagulation treatment.

Recurrent VTE despite anticoagulation treatment (Òfailure of anticoagulationÓ).

Relative indications *(expanded* **use; not guideline recommended)**

Recurrent PE complicated by pulmonary hypertension.

Patients with DVT and limited cardiopulmonary reserve or chronic obstructive pulmonary disease.

Patients with large, free-floating ileofemoral thrombus.

Following thrombectomy, embolectomy, or thrombolysis of DVT.

High-risk trauma patients (head and spinal cord injury, pelvic or lower-extremity fractures) with a contraindication for anticoagulation.

Patients with DVT who have cancer or burns, or are pregnant.

Contraindications for filter placement

Chronically thrombosed IVC.

Anatomical abnormalities preventing access to the IVC for filter placement.

VTE, venous thromboembolism; PE, pulmonary embolism; DVT, deep venous thrombosis; IVC, inferior vena cava.

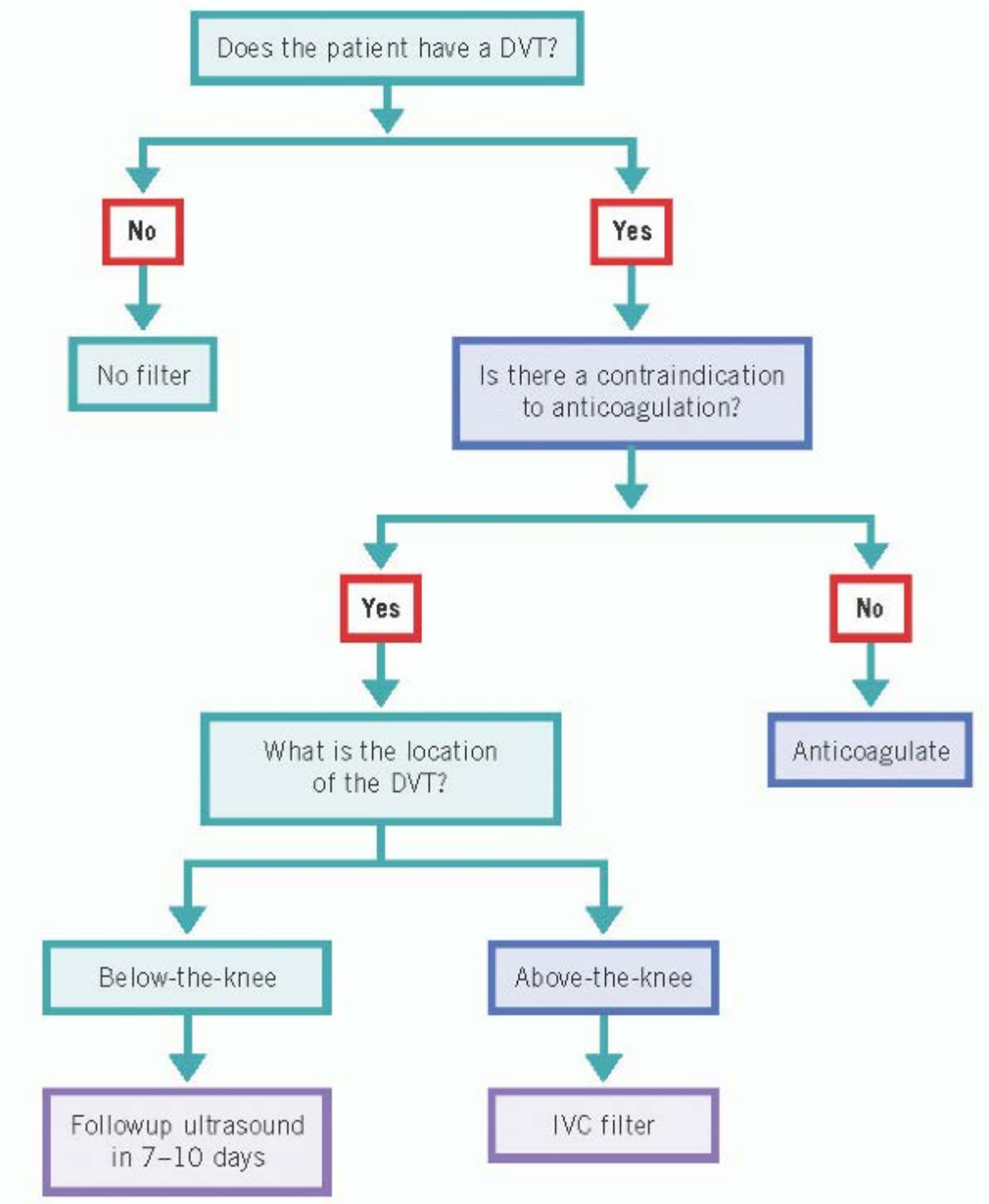

Figure 29-1 IVC Filter Placement Algorithm.

G. Catheter-directed thrombolysis of acute DVT with or without mechanical thrombectomy devices has been advocated to avoid adverse sequelae of DVT. The goals are to restore venous flow, preserve venous valve function, and eliminate the possibility of thromboembolism. Technical success and early clinical benefit have been reported, but long-term data are unavailable. In patients with migration of DVT resulting in severe PE and hemodynamic instability, potentially life-

saving thrombolytics should be considered *(Curr Opin Anaes.* 2006;19:52; CHEST guidelines. 2012).

VI. UPPER-EXTREMITY DVT

A. In patients with upper-extremity DVT that involves axillary or more proximal veins, the Chest Guidelines suggest anticoagulation *(CHEST.* 2012;141(2)).

B. If the upper-extremity DVT is associated with a catheter, it does not need to be removed if the catheter is functional and necessary. Rather, anticoagulation should be started and continued for 3 months after.

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.

1. Primary lymphedema is classified according to age at presentation.

a. Congenital primary lymphedema (present at birth) represents 10% to 15% of all cases, which can be hereditary (Milroy disease) or nonhereditary.

b. Praecox (early in life) or Meige disease represents 70% to 80% of cases.

(1) 80% to 90% of patients are female.

(2) Presents during the second and third decades of life, typically with localized swelling of the foot and ankle. Such swelling is worsened by prolonged standing.

(3) A single lower extremity is affected in 70% of patients.

c. Tarda (late in life) primary lymphedema, representing 10% to 15% of cases, is seen equally in men and women and presents after the third or fourth decade of life.

B. Secondary lymphedema results from impaired lymphatic drainage secondary to a known cause and is the most common cause of lymphedema in the United States. 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. Secondary lymphedema in the context of filariasis, caused by the parasite *Wuchereria bancrofti,* represents the most common worldwide etiology of the disease.

II. DIAGNOSIS

A. Clinical Presentation

1. Symptoms

a. 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 with elevation. Significant pain is unusual.

b. Patients with secondary lymphedema commonly present with repeated episodes of cellulitis secondary to high interstitial protein content.

2. Physical examination

a. When a lower extremity is involved, the toes are often spared.

b. With advanced disease, the extremity becomes tense, with nonpitting edema.

c. 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 lymphatic collaterals. The study has a sensitivity and specificity of 92% and 100%, respectively in the diagnosis of lymphedema *(J Vasc Surg.* 1989;9:683).

2. CT and magnetic resonance (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.

III. DIFFERENTIAL DIAGNOSIS.

Differential diagnosis includes all other causes of a swollen extremity.

A. Trauma

B. Infection

C. Arterial Disease

D. CVI

E. Lipedema

F. Neoplasm

G. Radiation Effects

H. 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. The 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.* 2009;42:51). CPT consists of a two-stage treatment program, beginning with skin care, followed by the application of compression bandages.

2. Sequential pneumatic compression has been shown to improve lymphedema.

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 derive from enhanced macrophage activity and extralymphatic absorption of interstitial proteins.

5. Cellulitis and lymphangitis should be suspected when sudden onset of pain, swelling, or erythema of the leg occurs. Intravenous antibiotics should be initiated to cover staphylococci and β-hemolytic streptococci. 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. Surgical intervention is an alternative approach for patients whose lymphedema has been refractory to nonoperative therapies. 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.

2. Closure of disrupted lymphatic channels.

3. Omental transposition.

4. Lymphatic transposition includes direct (lymphovenous bypass, lymphatic grafting) and indirect (mesenteric bridge, omental flap) procedures. Lymphatic grafting is performed for upper-extremity or unilateral lower-extremity lymphedema. Good results have been reported in 80% of patients *(Plast Reconstr Surg.* 1990;85:64).

5. Microsurgical lymphovenous anastomoses bypass the obstructed lymphatic system in patients with chronic lymphedema. With improved microvascular techniques, patency rates of 50% to 70% can be expected many months after surgery *(J Vasc Surg.* 1986;4:148).

CHAPTER 29: VENOUS AND LYMPHATIC DISEASE

Multiple Choice Questions

1. A 72-year-old man presents to your clinic with 3 days of right leg swelling. After completing a thorough history and physical examination you determine that a venous duplex is indicated. The results confirm your suspicion of proximal DVT. Which of the following statements is true regarding DVT?

a. The diagnosis of DVT is easily made by clinical examination.

b. Only half of patients with DVT have even one identifiable risk factor.

c. IVC filters are indicated in all patients with iliofemoral DVT.

d. Male gender is an independent risk factor for DVT.

e. DVT is a rare complication of orthopedic surgery.

View Answer

2. A 65-year-old female returns to your wound clinic for routine followup of her venous ulcer. She had a venous duplex showing reflux at her saphenofemoral junction and you performed a radiofrequency ablation of her left GSV 1 year ago. Today you observe that her ulcer is well healed. What is the CEAP classification of her venous disease ?

a. C_5 E_s A_s P_r

b. C_5 E_s A_d P_r

c. $C4_5$ E_s Ad P_r

d. C_5 E_p A_s P_r

e. C_5 E_p A_s P_p

View Answer

3. You are part of a hospital task-force dedicated to reducing the incidence of DVT. As part of your presentation you recommend giving all patients low-dose subcutaneous heparin TID. A hospital administrator asks you how effective this treatment is for preventing DVT as this proposal is projected to cost the hospital a million dollars/year. Low-dose unfractionated heparin reduces the risk of DVT by how much?

a. 10% to 30%

b. 30% to 50%

c. 50% to 70%

d. 70% to 90%

e. 90% to 100%

View Answer

4. You perform radiofrequency ablation of the right greater saphenous vein for symptomatic varicose veins. A venous duplex obtained 7 days after the procedure reveals a thrombosed GSV but there is propagation of clot into the common femoral vein. What is the appropriate treatment?

a. Prescribe Warfarin and refer to anticoagulation clinic for further management

b. No medications, this clot is a known risk of RFA and will resolve on its own

c. No medications, repeat duplex in 1 week to monitor clot

d. Enoxaparin with bridge to Coumadin therapy for 3 months for provoked DVT

e. Repeat radiofrequency ablation, this time on the femoral vein

View Answer

5. You are serving as the vascular surgery consult resident. You are called by the neurosurgery service to place an IVC filter on a patient prior to spine surgery, there is no evidence of DVT on the duplex but the patient will be nonambulatory for 7 days postop. Which of the following are complications of IVC filter placement?

a. Filter fracture

b. Filter embolus

c. DVT

d. Hematoma

e. All of the above

View Answer

6. A 43-year-old female presents to your Vascular Surgery clinic for a newly diagnosed peroneal vein DVT. Patient has no symptoms associated with this DVT. What is the correct recommendation for treatment?

a. IVC filter placement

b. Lovenox for 2 weeks with a repeat ultrasound

c. Repeat ultrasound in 1 to 2 weeks

d. Lovenox with bridge to Coumadin for 3 months

e. Lovenox with bridge to Coumadin for 6 months

View Answer

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Hemodialysis Access

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Surendra Shenoy

Hemodialysis (HD) is the most common renal replacement therapy used to prolong survival in patients with end-stage renal disease (ESRD). In December 2012, there were 636,905 ESRD patients in the United States. Approximately 408,711 (91% of all dialysis dependent patients) were on HD, 40,631 on peritoneal dialysis (PD), and 186,303 had a working renal transplant (USRDS. 2014). HD, based on body mass and the type of dialysis prescription (intermittent, daily, nocturnal etc.), requires 200 to 500 mL/minute of patient's blood circulation through the dialyzer. ÒVascular AccessÓ (VA) is a port or site in the body capable of supporting the blood flow required for dialysis. Adequacy of HD, responsible for the longevity of the ESRD patient, is directly dependent on the function of the dialysis access.

I. INDICATIONS FOR DIALYSIS

A. Short-term HD (temporary) is often urgently indicated in patients with acute renal failure in clinical situations such as (i) severe fluid overload, (ii) refractory hypertension, (iii) symptomatic hyperkalemia, (iv) uremiainduced GI symptoms or bleeding, encephalopathy, seizures, or pericarditis, (v) intractable metabolic acidosis, and (vi) occasionally with medication overdose or intoxication.

B. Long-term HD (permanent) is initiated in patients with stage 5 chronic kidney disease (CKD) presenting with intractable (i) fluid overload, (ii) electrolyte imbalance, (iii) hypertension, and/or (iv) uremia.

II. TYPES OF ACCESS

A. VA. Short-term or urgent HD needs are met by placing HD catheters temporarily in the central veins. Long-term dialysis requires surgical creation of subcutaneous conduits with high volume blood flow that can be accessed with needles to circulate blood through the dialyzer and back to the body.

B. PD Access. PD uses a silicone (Tenckhoff) catheter or polyurethane catheter, inserted with its tip placed in the dependent part of the peritoneal cavity (pelvis), that allows free in- and outflow of dialysate. The peritoneal membrane acts as a blood filter to exchange electrolytes and uremic toxins retained from renal failure.

III. DIALYSIS ACCESS CATHETERS

A. HD Catheters. HD catheters are large (approximately 10F to 15F) double lumen tubes (polyurethane, silicone, or copolymers) available in varying lengths (11 to 55 cm) intended to provide immediate VA for HD. The

arterial lumen (marked with a red Luer lock) draws blood to the dialyzer, and the venous lumen (marked with a blue Luer lock) returns blood to the body circulation. HD catheter placement should be performed under sterile conditions, using ultrasound guidance. The preferred site is the right internal jugular vein because it enters the superior vena cava in a straight path (*Tech Vasc Interv Radiol.* 2008;11:181-185).

1. HD catheter indications and types. Multiple HD catheters (Tesio, Duramax, Nextstep, Ultrastream, HemoSplit, Palindrome, Mahurkar, Proguide, Vaxcel, etc.), differing in their luminal and tip design (for providing uninterrupted blood flow and proper mixing of dialyzed blood with circulating blood to minimize recirculation during HD), are currently available (*Tech Vasc Interven Radio.* 2008;11:186-191). They are classified as nontunneled and tunneled dialysis catheters (TDCs). Table 30-1 compares the two types.

2. HD catheter complications

a. Insertion complications result from inadvertent injury to adjacent structures (vessels, nerves, and muscles) during needle puncture, guidewire/catheter introduction, or their misplacement. Complications are less common with the use of ultrasound guidance (*Semin Interv Radiol.* 2008;25:432-446).

TABLE 30-1 Differences between Tunneled and Nontunneled Dialysis Catheters

Dialysis Catheter	Nontunneled	Tunneled
Location need for placement	Bed side, dialysis unit	Setup with surgical sterility
Site of skin and vein puncture	Close to each other	Separated by a subcutaneous tunnel
Cuff as infection barrier and catheter	No cuff on the catheter	Often present

securing point		
Recommended duration of use	Short term (7-21 days)	Intermediate term (months)
Management of exit site infections	Often needs catheter removal and antibiotics	Often treated with antibiotics and no catheter removal
Blood flow rates	Stiff material, smaller internal diameter, and support lower flows	Soft material, larger internal diameter, and may support higher flows

b. Catheter dysfunction. *Early dysfunction* of HD catheters is usually due to a luminal thrombus, subtle kinks in the catheter, or malpositioning of the tip. *Late dysfunctions* are usually secondary to fibrin sheath formation around the catheter, tip migration, or luminal thrombosis.

c. Catheter infection is a major cause of morbidity and mortality. HD using a TDC has a 10 time higher relative risk of infection when compared to an AVF (*Am J Infect Control.* 2004;32:155). Catheters may present with exit site or systemic infection. While most exit site infections in TDC may respond to antibiotics, cuff, tunnel, and systemic infections usually need catheter exchange or removal based on severity of symptoms during hospitalization, response to IV antibiotic, and the etiology of infection. Catheter-related bacteremia has a high rate (20% to 35%) of systemic infection that can lead to death.

d. Central vein stenosis, thrombosis, or stricture. Catheter-related acute venous thrombosis often requires systemic anticoagulation therapy. The incidence of catheter-induced central vein stenosis varies (5% to 50%) based on site, type, and duration of access, but may be precipitated by a single catheter insertion. Despite treatment they frequently result in loss of VA in the ipsilateral extremity.

Catheter-based HD has double the relative risk of mortality compared with AVF. Hence, attempts should be made to either avoid catheter usage or remove them as early as possible. Catheters should be limited to ESRD patients either (a) awaiting permanent VA (AVF/AVG), (b) with living donors awaiting transplant, or (c) suffering acute failure of AVF, AVG, or PD catheter. They may be indicated for long-term access in patients with limited life expectancy (e.g., metastatic tumors) or when all other permanent access modalities have failed.

B. PD Catheters

1. PD catheter indications and types. PD catheters are used in acute (short-term) or chronic

renal failure (long-term). PD catheter designs vary in cuff, body, and tip configuration (straight with disc, weight balloon and fluting, or coiled with similar variations, Swan-neck presternal catheters etc.). None have shown convincing superiority in function or complication rates (*Perit Dial Int.* 2007;27:S119-S125). Surgeons, nephrologists, or interventional radiologists (based on expertise available) can place single cuff Tenckhoff catheters for short-term immediate use at the bed side. Long-term PD catheters (usually two cuffs) are placed surgically with or without the use of radiologic imaging, peritoneoscopy, or laparoscopy. PD is the preferred technique for children. Some surgical contraindications for PD catheter placement include recent abdominal surgery, peritoneal infections, adhesions, large abdominal wall hernia, significant gastroesophageal reflux, and diaphragmatic hernia.

2. PD catheter complications

a. Early complications

(1) Injury to intraperitoneal[u3] structures resulting in intestinal perforation, peritonitis, and bleeding.

(2) Dialysate infusion resulting in hydrothorax, exacerbation of hernia (scrotal swelling), and abdominal wall edema.

(3) Catheter dysfunction due to malposition or kink, fibrin sheath or coagulum, or constipation.

b. Late complications

(1) Catheter related: Leakage[u4] from fractures, intra-abdominal bleeding from tugging, intestinal obstruction, and intestinal perforations from erosions.

(2) Dialysate-related problems (listed above) can occasionally manifest late.

c. Infections

(1) Exit site infection[u5] or cuff extrusion can be treated with antibiotics and cuff removal in double cuff catheters.

(2) Peritonitis is a major cause of hospitalization, PD failure, and mortality (35 deaths per 1,000 years at risk). Infections can include Gram-positive bacteria, Gram-negative bacteria including Pseudomonas, fungal infections, or tuberculous infections. First cultures should be obtained, then empiric intraperitoneal therapy should be initiated with vancomycin or a cephalosporin to cover Gram-positive bacteria and third-generation cephalosporin or an aminoglycoside to cover Gram-negative bacteria. Once cultures and sensitivities are reported, the regimen should be targeted to the specific microbe (*Perit Dial Int.* 2010;30:393). Severe, recurrent, or resistant infections require catheter removal.

IV. ARTERIOVENOUS ACCESS.

An arteriovenous access (AVA) is a subcutaneous conduit that can deliver blood flow required to provide adequate dialysis. The increased conduit flow is a physiologic response to the *permanent* arteriovenous communication created between a peripheral artery and a vein. Functionally, it is a complete circuit starting at the left ventricle (pump) and ending on the right atrium. The important functional components include the inflow, needle access segment (NAS or conduit), and outflow. Based on the conduit used, AVA is classified as AVF or AVG.

A. Inflow. A normal (without atherosclerosis or calcification) radial artery with a diameter over 2 mm is the most common vessel used to obtain inflow to a VA. Due to the narrow caliber, VA with distal (at the wrist) or proximal (at the elbow) radial inflow have low flows and low risk of flow-induced distal ischemic complications (steal). Larger arteries such as brachial or axillary artery are also used as inflow for AVA. Any stenosis in the inflow beyond the heart (pump) can lead to low VA flow. The most common site to encounter flow limiting stenosis is the juxta-anastomotic area.

B. Conduit. For easy placement of two needles during dialysis, the NAS (conduit) should be 6 mm in diameter, less than 5 mm deep from the skin surface, and have a 10 cm long straight segment. This allows two 2.6-cm needles (one to draw blood to the dialyzer and one to return blood to body circulation) to be placed 4 to 5 cm apart for proper mixing and to prevent recirculation of dialyzed blood inside the conduit. Inadequate conduits often cause needle access problems during dialysis.

With the increased flow, superficial veins (usually cephalic) in the forearm or upper arm dilate in 4 to 6 weeks and serve as a good NAS. An AVA constructed by anastomosing a native vein (to develop as conduit and outflow) to a native artery (inflow) is termed an **arteriovenous fistula (AVF)** or **autogenous fistula.** When veins are not available, or fail to develop (mature) as good conduits, tubes made of biocompatible materials are used as NAS. Such AVA are termed **arteriovenous graft (AVG)** or **nonautogenous access.** Expanded polytetrafluoroethylene (ePTFE) is the most common material used for AVG. Composite grafts coated with ePTFE, biologic tissue such as formaldehyde fixed bovine arteries and veins, and cryopreserved human veins are also available.

C. Outflow. Veins beyond the NAS carrying blood flow back to the heart are termed the outflow for an AVA. Flow restriction caused by stenosis developing in the peripheral aspect of the outflow results in increased pressure (pulsatile access) within the NAS and causes prolonged bleeding from needle holes following dialysis or aneurysmal dilation. When the stenosis develops more centrally (within the chest or abdominal cavity), due to paucity of collaterals, the increased pressure is reflected in the entire venous system of the ipsilateral limb with AVA resulting in symptoms of venous hypertension.

D. Timing. All CKD patients likely to need dialysis should have a conscious attempt at vein preservation or Òsave the veinÓ (identify and preserve veins by minimizing venipuncture and central vein instrumentation) for VA. VA evaluation and planning requires approximately 6

months. Planning renal replacement when the patient reaches CKD stage 4 (GFR < 30 mL/minute) should provide ample opportunity for VA referral and placement.

V. PREOPERATIVE EVALUATION

A. History. Obtain demographics, previous VA attempts, and details related to arterial disease (claudication, rest pain, limb/digit loss, neuropathy etc.) and venous problems (venipunctures, catheter placements, percutaneously introduced central catheters [PICC], cardiac implantable electronic device [CIED], etc.). Evaluate comorbidities such as smoking, diabetes, cardiac problems, peripheral neuropathy, and/or thrombophilia that may contribute to VA failure.

B. Physical Examination

1. Arterial (inflow) evaluation. Palpate and compare strength of pulse and quality of arterial wall (radial, brachial, and axillary) bilaterally.

When indicated, the femoral, popliteal, and pedal arteries should be evaluated. Bilateral blood pressures should be measured (difference >20 mm Hg is significant for proximal stenosis). The role of the Allen test, intended to assess the integrity of palmar circulation, is not certain for end to side VA placement. Scars from previous surgery and catheters should be noted.

2. Venous (outflow) evaluation. Using vein dilation techniques (tourniquets, warmth, tapping, use of gravity etc.) is critical for peripheral vein evaluation. Presence of edema, unequal extremities, and/or chest and shoulder collaterals may suggest central venous stenosis. Patency and continuity of the vein can be assessed by eliciting fluid thrill.

C. Doppler Duplex ultrasound scanning (DDUS) should always complement clinical examination. Best results from ultrasound vessel mapping are obtained when the operating surgeon evaluates the scan in real time. DDUS provides both functional (distensibility, flow pattern, and volumes) and structural evaluation of the peripheral arteries and veins (superficial and deep). It is not very reliable in evaluation of central veins.

D. Diagnostic Imaging

1. Contrast venography is the gold standard for determining the patency (luminal dimension) and adequacy of central venous anatomy. Peripheral vein visualization is limited to downstream tributaries receiving contrast from the distal peripheral vein injection site.

2. Arteriography remains the gold standard for the evaluation of a suspected arterial inflow stenosis or occlusion. **MRA** (noncontrast) or **CTA** may be used to evaluate larger veins and arterial run off.

E. Laboratory Studies. Hyperkalemia and acidosis are the most common electrolyte abnormalities seen in ESRD patients. Testing (day of surgery) should include hemoglobin, serum potassium, and glucose levels to avoid possible procedural or anesthesia-related complications.

VI. ARTERIOVENOUS FISTULA.

AVF is the preferred access, as once established, it has superior longevity, patency, and resistance to infections requiring minimum interventions. Disadvantages of AVF include wide variation in maturation failure (13% to 58%), duration for maturation (4 weeks to 4 months), and need for additional procedures to help maturation (~40%). The National Kidney Foundation, Kidney Disease Outcomes Quality Initiative (NFKDOQI) suggests a goal of 65% fistula prevalence. In the United States, following the initiation of Òfistula firstÓ initiative, the prevalence of AVF has steadily increased to 62%.

A. AVF Locations and Planning. There are multiple established locations (Fig. 30-1) in the upper limb where the anatomy may lend itself to AV anastomosis. When suitable, the nondominant limb is used first. Using distal sites has less risk of ischemic complication, and allows proximal veins to dilate for future use as secondary options. Using isolated segments of superficial veins preserves proximal veins for future use.

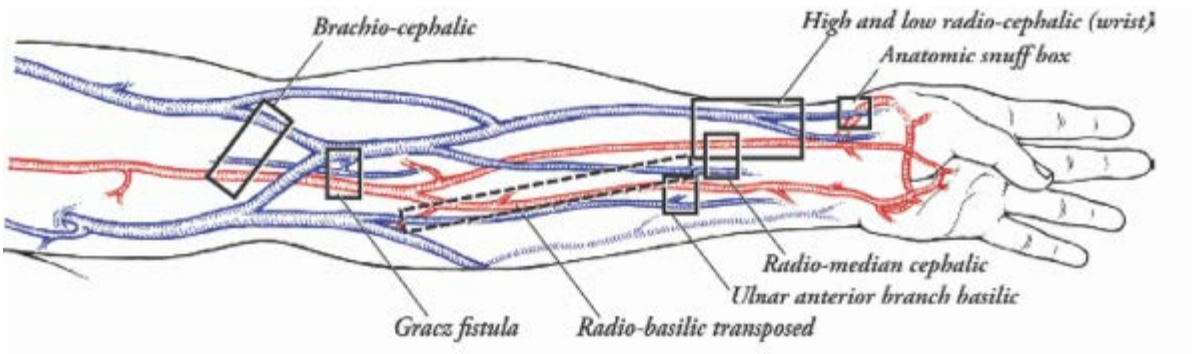

Figure 30-1 Possible sites of arteriovenous anastomosis in the upper extremity. (Reprinted from *J Vasc Access.* 2009;10(4):223-232, with permission of the publisher.)

Surgical procedure. All AVF procedures are generally performed on an outpatient basis, using intravenous conscious sedation and local anesthetics or regional blocks. The end of a superficial vein is anastomosed to the side of the artery using conventional or piggy back straight line onlay technique (*J Vasc Surg.* 2012;55:274-280) that provides an opportunity to tailor anastomotic length. Anastomotic length in conventional techniques is often dictated by the size of the vessels used and need for spatulation. Computational modeling suggests that a diameter of 3 mm is sufficient to provide flows necessary for AVF. Larger anastomotic diameters in larger inflow arteries (e.g., brachial) have a higher propensity for high flow related distal ischemia (steal).

B. Postoperative Evaluation. Early assessment (7 to 10 days) is necessary to evaluate the surgical wound, fistula patency, and detect any distal vascular or neurologic problems. It also provides an opportunity to further evaluate early postoperative problems that may interfere with maturation. A **maturation evaluation** (assess inflow, conduit, and outflow) should be performed between 4 and 6 weeks postoperatively using clinical evaluation and DDUS. Any problems detected should be further evaluated. AVF meeting maturation criteria can then be accessed. Fistulae that have good inflow but are deep and not accessible need superficialization (VA: Principle and Practice. Williams and Wilkins. 2010:1996-205). Occasionally, fistulae may need a few additional weeks to mature.

VII. AV GRAFT.

AV grafts are conduits (anastomosed to an artery to obtain inflow and a vein for outflow) placed in a subdermal location to provide NAS for dialysis. Based on the configuration of the conduit they are termed loop or straight grafts. The graft limb receiving arterial inflow, termed arterial limb, provides blood to the dialysis circuit. The graft limb delivering dialyzed blood into the outflow vein is termed the venous limb.

A. AVG Locations and Planning. The proximal radial, brachial, and axillary arteries usually provide graft inflow. Since patients requiring AVG often lack sufficient superficial venous anatomy for AVF, the venae comitantes or the deep veins of the upper arm (basilic, brachial, or axillary) provide the outflow for AVG. They can be placed in the thigh using femoral artery

and saphenous or femoral veins. Distal ischemia becomes more of an issue when larger vessels are used for inflow in AVG. Lower-extremity grafts are at a higher risk for infection.

B. Surgical Procedure. AV grafts are placed under local anesthesia with conscious sedation. Prophylactic antibiotics (e.g., second-generation cephalosporins) are commonly administered immediately prior to the surgery.

C. Postoperative Evaluation. While conventional grafts need good tissue incorporation prior to cannulation (3 to 6 weeks), newer composite graft materials permit early cannulation when indicated. A standard 6 mm caliber graft, placed in a subdermal location, and with rigid walls make graft cannulation easier than many AVF. Once placed, grafts are more predictably available for dialysis; however, they are prone to repeated thrombosis requiring a significantly higher number of interventions and have poor longevity compared to a well-matured AVF.

VIII. AVA PLANNING.

The goal of clinical evaluation is to plan a sequence of options to provide VA throughout the life span of the ESRD patient. Access planning should take into consideration the life expectancy of the ESRD patient, renal function at the time of referral (CKD 4 or 5 or already on dialysis), and the available access options based on clinical and ultrasound evaluation. Based on these factors, the surgeon should plan to create a VA that has the longest durability, needing the least

interventions, in a timely fashion, without jeopardizing future access options (*J Vasc Access.* 14;15:S1-S5). When the anatomy is suitable, AVF is the preferred access. AVG should be considered when AVF is not an option or is not able to provide a timely access.

IX. COMPLICATIONS OF AVA.

VA dysfunction is a major cause of morbidity and mortality in ESRD. Stenosis (responsible for over 90% of VA dysfunctions) can present as thrombosis, infection, and aneurysm. NKF-KDOQI guidelines recommend prospective monitoring and surveillance using physical examination, ultrasound evaluation of flow, and assessment of dialysis adequacy to help detect access dysfunction. Interventions guided by DDUS/angiography imaging or surgical techniques are used to prolong access patency.

A. Stenosis. The functional impact of stenoses depend on their location, diameter relative to the inflow volume, and pressure (*J Vasc Acc.* 2014;15:409-414). Development of venous neointimal hyperplasia (VNH), the cause for luminal narrowing, is common in areas of outflow (e.g., graft vein anastomosis of AVG and JAS of AVF) experiencing hemodynamic stress resulting from blood flow increase. Balloon angioplasty, using DDUS or contrast angiography, prior to development of thrombosis helps improve access patency and delay failure. Surgical venoplasty and vascular stents are also used based on indications or when angioplasty fails.

B. Thrombosis. Thrombosis seen early after AVA placement is attributed to flow interruption due to technical factors, inflammatory narrowing of outflow veins, preexisting vein occlusions, or kinked grafts. Occasionally

patient-related factors such as thrombophilia and hypotension may be responsible. Early thrombosis of AVF often results in loss of the access site. AVG with early thrombosis may be salvaged by surgical or radiologic image-guided thrombectomy. Late thrombosis both in AVG and AVF are mostly due to development of stenosis in the circuit. They are salvaged by thrombectomy and dilation of stenotic lesions using surgical or interventional means.

C. Infection. Infections in AVA are caused by cutaneous flora (commonly *Staphylococcus* species) contamination, mostly through the needle access sites, presenting as skin ulceration, infections in chronic thrombi within aneurysms, or seeding of graft material. Management of infection depends on severity and presentation. Most graft infections result in removal of the graft and loss of the access site. Focal infections may be salvaged with resection of the infected portion and reconstruction with a bypass graft. Most AVF infections are treated with antibiotics, with or without surgical intervention.

D. Aneurysms and Pseudoaneurysms. These develop secondary to loss of AVG integrity, repeated needle puncture, and extravasation of blood which is walled off by surrounding tissue (pseudoaneurysms). AVF aneurysms are often true dilations of the vein developing over time due to stretch on the healing puncture site from the pressure within. Aneurysms can develop chronic

thrombi resulting in access difficulty. They may also be at risk of rupture and bleeding due to thinning of the overlying skin. In such situations both AVG and AVF aneurysms can be electively repaired.

E. Arterial ÒStealÓ Syndrome. All AV accesses divert or ÒstealÓ a fraction of blood from the distal circulation which is usually well tolerated by the distal extremity. In approximately 1% to 4% of patients (diabetics, underlying neuropathy, and vascular disease) this may precipitate ischemic pain, worsening neuropathy, ulceration, or gangrene (*Ann Vasc Surg.* 2000;14(2):138-144). Mild symptoms such as subjective coolness and paresthesias without sensory or motor loss may be managed expectantly with increasing exercise tolerance. Failure to improve or increasingly severe symptoms require further evaluation. Reduction of AVA flow may improve symptoms due to high VA flows. Symptomatic patients with low AVA flows may benefit from distal flow enhancement techniques. Severe ischemia requires immediate evaluation and management including access ligation to avoid irreversible nerve injury.

F. Venous Hypertension. Venous hypertension caused by central vein stenosis manifests as edema, skin discoloration, and/or hyperpigmentation in the affected limb. Management options include venoplasty of the stenosis, stent graft repair, or surgical provision of improved outflow drainage.

G. Congestive Heart Failure (CHF). High volume flow, often resulting from large artery based access AVA can occasionally precipitate CHF. This is more common in patients with underlying heart problems or preexisting fluid overload. Based on the situation, this may necessitate flow reduction or access ligation.

CHAPTER 30: HEMODIALYSIS ACCESS

Multiple Choice Questions

1. What is the appropriate flow rate for adequate hemodialysis?

a. 100 to 200 mL/minute

b. 500 to 700 mL/minute

c. 800 to 1,000 mL/minute

d. 200 to 500 mL/minute

View Answer

2. What is the most common cause of AV graft dysfunction?

a. Infection

b. Thrombosis

c. Stenosis

d. Maturation failure

View Answer

3. What is the adequate time frame for dialysis access planning?

a. 6 months

b. 3 months

c. 2 months

d. 1 month

View Answer

4. What is the most common reason for abandoning peritoneal dialysis for hemodialysis?

a. Inconvenience

b. Hydrothorax

c. Bleeding

d. Peritonitis

View Answer

5. What is an acceptable initial empiric antibiotic regimen for catheterassociated peritonitis?

a. Oral vancomycin and ceftriaxone

b. Intraperitoneal cefazolin and gentamicin

c. Intraperitoneal vancomycin and metronidazole

d. Intraperitoneal cefazolin and vancomycin

View Answer

6. What is the most common site of flow-limiting stenosis in AV fistulae?

a. Central vein

b. Proximal artery

c. Juxta-anastomotic area

d. Previous central line site

View Answer

7. What is the maximum depth beneath the skin surface recommended for easy AV fistula access?

a. 2 mm

b. 10 mm

c. 7 mm

d. 5 mm

View Answer

8. What is the correct terminology for the implanted synthetic tube placed during creation of an AV graft?

a. Autogenous fistula

b. Nonautogenous conduit

c. Autogenous outflow

d. Nonautogenous inflow

View Answer

9. Salvage procedures used for vascular steal in a high-flow AVF access include:

a. Distal revascularization with interval ligation (DRIL)

b. Banding the access

c. Proximalization of arterial inflow (PAI)

d. Fistula ligation

View Answer

10. Which vascular access poses the highest risk of infection?

a. AV fistula

b. Nontunneled dialysis catheter

c. Tunneled dialysis catheter

d. AV graft

View Answer

31
Transplantation

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PHYSIOLOGIC IMMUNITY

Our immune system did not evolve to stymie the efforts of transplant surgeons, but rather to evade microbes that aim to overrun us. To understand why tissues from one individual are rejected by another, one has to appreciate the components of our immune system in its physiologic state.

I. THE IMMUNE SYSTEM ENCOMPASSES 2 COMPLEMENTARY ARMS

A. Innate immune system recognizes general distress as well as conserved moieties from ubiquitous pathogens, such as lipopolysaccharides of gramnegative bacteria. The response is **direct, nonspecific, and lacks memory.**

1. Mediators

a. The **complement cascade** is a soluble group of proteins whose activation promotes formation of the membrane attack complex (MAC). This embeds itself within cell membranes of pathogens causing disruption and cell lysis. In addition, byproducts of the complement cascade opsonize pathogens, which promotes phagocytosis by antigen presenting cells (APCs).

b. Natural Killer cells recognize cells that lack self-Major Histocompatibility Complex (MHC) and are part of the body's immunosurveillance for cancer.

B. The **adaptive immune system** recognizes **specific,** pathogenic antigens in the context of MHC, which help the immune system distinguish self from non-self. Foreign antigens presented in the context of the MHC are targets of the adaptive immune system. In humans, these complexes are referred to as **Human Leukocyte Antigens (HLA**) and are located on chromosome 6.

1. Classes of HLA

a. Class I (A, B, C) are present on all nucleated cells and are targets for cytotoxic (CD8) T cells

b. Class II (DR, DP, and DQ) are present on APCs and are targets for helper (CD4) T cells. They trigger an antibody (humoral) mediated immune response.

The most important HLA in solid organ transplantation are A, B, and DR. Since each

person has 2 MHC complexes, one on each copy of chromosome 6, everyone has a total of 6 HLA antigens that are relevant to organ transplantation.

2. Adaptive immune responses

a. Cell-mediated. Antigens in the peripheral tissues are presented to T-cells located in lymph nodes and the spleen. The T-cell receptor (TCR)

recognizes a specific antigen in the context of MHC. Formation of the TCR is the result of DNA rearrangement that occurs within the thymus during fetal development. Following rearrangement, T-cells are selected based on their ability to bind self MHC without activating a response. MHC encountered in the tissues not involved in thymic education activates an immune response. *This is the basis of alloreactivity.*

(1) Helper T cells (CD4) recognize exogenous antigens presented in the context of MHC class II on the surface of APCs (B cells, dendrites, and macrophages). Activation releases IL-2, which causes B cell maturation into Plasma cells and IL-4, which causes maturation of cytotoxic T cells.

(2) Cytotoxic T cells (CD8) recognize endogenous (i.e., TB, viruses) pathogens presented in the context of MHC class I.

b. Antibody-mediated (humoral). B cells (Bone) activate antibody-mediated (humoral) immunity. IL-4 from helper T cells transforms B cells into plasma cells, which secrete antibodies specific to the offending pathogen.

TRANSPLANT IMMUNOLOGY

A. Isografts. Tissue Transfer from Genetically Identical Individuals (i.e., Twins)

B. Xenografts. Tissue Transfer between Species

C. Allografts. Tissue Transfer among Members of the same Species

1. Alloreactivity/Histocompatibility

a. ABO blood compatibility is necessary for all transplants, except liver.

b. HLA Ð A, -B, -DR are the most important for compatibility; -DR is most important overall.

(1) Cross-matching detects preformed antibodies against donor HLA. It involves mixing recipient serum with donor lymphocytes.

(2) Panel reactive antibodies (PRA) help to predict the likelihood of 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 with which the patient's sera react is the PRA. Patients who have been exposed to other HLAs via blood transfusion, pregnancy, or prior transplantation will have higher PRAs.

TRANSPLANT REJECTION

I. TYPES OF REJECTION

A. Hyperacute rejection is the result of preformed anti-HLA antibodies that bind the allograft endothelium to initiate a cascade of events culminating in vascular thrombosis and ischemic necrosis. The only therapeutic option is to remove the allograft immediately. This is extraordinarily uncommon in the modern era of cross-matching.

B. Accelerated rejection is caused by sensitized T cells that produce a secondary immune response. This generally occurs within 1 week of transplantation. Treatment includes pulse steroids and OKT3.

C. Acute rejection is cell-mediated and involves T lymphocytes (cytotoxic and helper). This typically occurs 1 week to 1 month after transplantation. There are two basic treatment modalities: High-dose methylprednisolone and an antilymphocyte preparation.

D. Chronic rejection is a poorly understood phenomenon that can occur weeks to years after transplantation. Emerging evidence suggests that the humoral immune response is an important contributor. Plasmapheresis, intravenous immunoglobulin, and rituximab have been used to treat antibody-mediated rejection.

IMMUNOSUPPRESSION

I. CLASSES OF IMMUNOSUPPRESSIVE DRUGS

A. Corticosteroids (Prednisone or methylprednisolone/Solu-Medrol) play the broadest role in immunomodulation and are used for induction, maintenance, and treatment of rejection. Steroids inhibit transcription of cytokines that promote lymphocyte proliferation, which dampens the inflammatory response and migration of neutrophils. Toxicities include poor wound healing, hyperglycemia, infections, cataracts, hypertension, weight gain, and bone disease.

B. Antiproliferative Agents/Antimetabolites

1. Azathioprine (Imuran) is an antimetabolite that is a thioguanine derivative of mercaptopurine. This purine analog alters the function or synthesis of DNA and RNA, inhibiting T- and B-lymphocyte proliferation. Myelosuppression manifested as leukopenia and thrombocytopenia are its main toxicities.

2. Mycophenolic acid (CellCept, Myfortic) inhibits inosine monophosphate dehydrogenase (rate-limiting enzyme in guanine monophosphate synthesis) inhibiting RNA synthesis. In doing so, this drug selectively inhibits T- and B-cell proliferation, cytotoxic T-cell generation, and antibody formation. Toxicities include GI disturbance and leukopenia.

C. Calcineurin Inhibitors

1. Cyclosporine (Sandimmune, Neoral, Gengraf) binds cyclophilin protein and inhibits IL-2 production. This blocks T cell activation and proliferation. Nephrotoxicity, hypertension, tremors, seizures, hyperkalemia, hyperuricemia, hypercholesterolemia, gingival hyperplasia, and hirsutism are the main toxicities.

2. Tacrolimus (FK506, Prograf) binds FK binding protein; action similar to CSA but is 10 to 100 times more potent. Toxicities are similar to CSA, but include more GI and neurologic changes.

3. Sirolimus (Rapamune) is an anti-T-cell agent that inhibits the mTOR molecule, blocking T-cell signal transduction. Toxicity includes

thrombocytopenia, hyperlipidemia, oral ulcers, anemia, proteinuria, and impairment of wound healing.

D. Co-Stimulation Blockade. Belatacept (Nulojix) is a fusion protein that is homologous to CD27 and binds to the B7 complex to irreversibly block the Signal 2 pathway associated the MHC/APC binding process.

E. 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 class I and II MHC molecules. These agents are used as induction therapy in the perioperative period or as rescue therapy following acute rejection. The most commonly used antithymocyte immunoglobulin in the United States is **thymoglobulin,** which is derived from rabbit serum.

II. COMPLICATIONS OF IMMUNOSUPPRESSION

A. Bacterial Infections. Pneumonia and urinary tract infections (UTIs) occur fairly commonly after transplantation. Infectious complications from opportunistic organisms are now uncommon because of appropriate prophylactic strategies.

B. Viral Infections

1. CMV infection can occur at any time but is most common 1 to 4 months posttransplant, in the absence of prophylaxis. CMV may infect the recipient's liver, lungs, or GI tract. Signs and symptoms include fever, chills, malaise, anorexia, nausea, vomiting, cough, abdominal pain, hypoxia, leukopenia, and elevation in liver transaminases. CMV peripheral blood PCR or serologic assays are the most common tools for diagnosis. Prophylaxis 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, which inhibits DNA synthesis.

2. EBV can infect B cells at any time after transplantation and may be associated with the development of a type of lymphoma, termed posttransplant lymphoproliferative disorder (PTLD), usually of monoclonal B-cell origin. Infiltration of the hematopoietic system, central nervous

system (CNS), lungs, or other solid organs may occur. The patient usually presents with fever, chills, sweats, enlarged lymph nodes, and elevated uric acid. Diagnosis is made by physical examination; EBV serology; computed tomography (CT) scan of the head, chest, and abdomen (to evaluate lymph nodes or masses); and biopsy of potential sites or lesions. Treatment consists of reducing or withdrawing immunosuppression.

3. HSV causes characteristic ulcers on the oral mucosa, in the genital region, and in the esophagus. Renal transplant patients, if not on ganciclovir, are given prophylactic acyclovir. Active HSV infections are treated by decreasing the patient's immunosuppression and instituting acyclovir therapy.

4. BK virus is a member of the polyoma virus family. Approximately 90% of individuals are seropositive. BK viruria develops in 30% of kidney transplant recipients and progresses to viremia in 15% of recipients within the first year. Persistent viremia leads to BK nephropathy, which occurs in up to 10% of kidney transplant recipients during the first year. There is no known effective treatment.

C. Fungal infections can range from asymptomatic colonization to lethal invasive infections. Oral **candidiasis** can be prevented and treated with oral nystatin or fluconazole. Esophageal candidiasis can be treated with a short course of intravenous amphotericin B or fluconazole. Serious fungal infections are treated with intravenous amphotericin B, although use of less nephrotoxic agents such as caspofungin and anidulafungin is increasing.

D. Malignancies. Cancers that occur at a higher frequency in transplant recipients include squamous cell carcinoma, basal cell carcinoma, Kaposi sarcoma, lymphomas, hepatobiliary carcinoma, and cervical carcinoma.

ORGAN ALLOCATION

Over 120,000 individuals in the United States are currently waiting for the life-saving gift from an organ donor. The gap between supply and demand grows daily, so a system exists that allocates solid organs to individuals based on two overarching themes (*unos.org*):

A. Justice. Each candidate is given fair consideration based on individual circumstances and medical need.

B. Utility. The system tries to maximize the number of transplants performed and the survival of both patients and allografts.

The United Network of Organ Sharing is the organization contracted by the federal government to oversee organ allocation in the United States. Fifty-eight local Organ Procurement Organizations (OPOs) serve 11 UNOS regions.

Livers are allocated based on the **MELD** scoring system. The MELD score is derived from a logarithmic formula that incorporates the values for bilirubin, serum creatinine, and the

international normalized ratio (INR) and ranges from 6 to 40 (*unos.org*). Livers are allocated to appropriate patients with the highest MELD scores. Special exception points may be granted, such as in cases of hepatocellular carcinoma (HCC), hilar cholangiocarcinoma, hepatopulmonary syndrome, or hepatorenal syndrome. Children receive a Pediatric End-Stage Liver Disease (PELD) score.

Given the effectiveness of renal replacement therapy in the form of hemodialysis, kidney transplantation is never a medical emergency and **kidney** allocation is largely based on waiting times, the degree of sensitization (PRA score), and histocompatibility.

ORGAN DONATION

I. TYPES OF ORGAN DONATION

A. Living Donation. Given the stability in the number of deceased donors, living donation is an important means for increasing the donor pool and

has become an integral part of renal transplant practice. Advantages of living-donor transplantation include improved short- and long-term graft survival (1-year survival >95%), improved immediate allograft function, planned operative timing to allow for medical optimization (and, often, avoidance of dialysis). The evaluation of potential living donors includes assessment of their overall health, co-morbid conditions and psychosocial influences. Compatibility with their intended recipient is determined though ABO blood typing and HLA histocompatibility. Donors who are not compatible with their intended recipient may still donate through paired exchange and ABO-incompatible protocols.

B. Deceased Donation

1. Heart beating (brain dead). Strict criteria for establishing brain death include irreversible coma and the absence of brain stem reflexes (i.e., pupillary, corneal, vestibulo-ocular, and gag reflexes). Other useful diagnostic tests include blood flow scan, arteriography, and an apnea test.

2. Nonheart beating (Donation after cardiac death DCD) refers to those potential organ donors who do not meet strict brain death criteria but who are considered to have nonrecoverable devastating neurologic insults. Life support is discontinued in the operating room, and organ procurement is initiated after a specified interval following cardiac asystole. While effectively increasing the donor pool, 16% to 28% of DCD livers have biliary complications, including ischemic cholangiopathy (*World of Gastroenterol.* 2014;20(20):6159-6169).

*II. SUITABILITY FOR TRANSPLANTATION***

A. Contraindications

1. Active infection. While evidence of HIV or TB are absolute contraindications to organ donation, localized infections such as UTIs and pneumonia, in absence of dissemination, are

routinely given consideration. Even in the presence of bacteremia, appropriate initiation of antibiotic therapy ensures a small risk of transmission.

2. Cancer. With the exception of primary CNS tumors, active cancer, whether treated or not, is an absolute contraindication to organ donation. While the blood-brain barrier protects CNS tumor cells from widespread dissemination in the heavily immunosuppressed patient, this is not the case for other malignancies. Depending on the type of cancer, patients may be listed after a cancer free wait time ranging from 2 to 5 years.

B. General Considerations

1. Age. As experience with less than ideal donors has grown, it has become apparent that arbitrary limits on donor age are unnecessary. Good allograft function has been achieved with kidney and liver donors with advanced age.

2. Overall health. As the donor pool ages, systemic diseases that can have an effect on specific organ function must be taken into consideration. Hypertension and atherosclerosis can hinder the suitability of kidney

allografts, while obesity with hepatic steatosis limits the suitability of liver allografts.

3. Social behaviors. While all donors are tested for HIV, hepatitis, and other viral infections, donors who engage in high-risk behaviors may still transmit an infection if donation were to occur within the window period prior to seroconversion. Potential recipients are counseled regarding these socially high-risk donors and given the option whether to consider organs allocated from this group.

ORGAN PROCUREMENT AND PRESERVATION

Initial dissection aims to control the abdominal aorta and IMV for the placement of cannulae. Identification of hepatic hilar structures aids later dissection in the cold. After cross-clamping the supraceliac aorta, the abdominal viscera are then flushed and cooled with University of Wisconsin (UW) preservation solution or HTK (histidine-tryptophan-ketoglutarate). The organs are packed with ice, while the solution infuses. Ventilation of blood is into the chest via the IVC. 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 preservation solution and surrounded by iced saline during transportation.

The donor kidneys are removed separately or en bloc. The ureters are dissected widely to minimize devascularization and are divided near the bladder.

The pancreas may also be removed for transplantation, with the pancreas, duodenum, and spleen removed en bloc. The blood supply for the pancreas allograft comes from the donor splenic and superior mesenteric arteries, and outflow is via the portal vein (Fig. 31-1).

With the advent of modern preservation solutions, donor livers can be preserved for up to 12 hours before reperfusion (kidneys up to 40 hours), with a low incidence

of allograft dysfunction. Ideally, cold ischemia time is minimized to less than 6 hours for livers and 24 hours for kidneys.

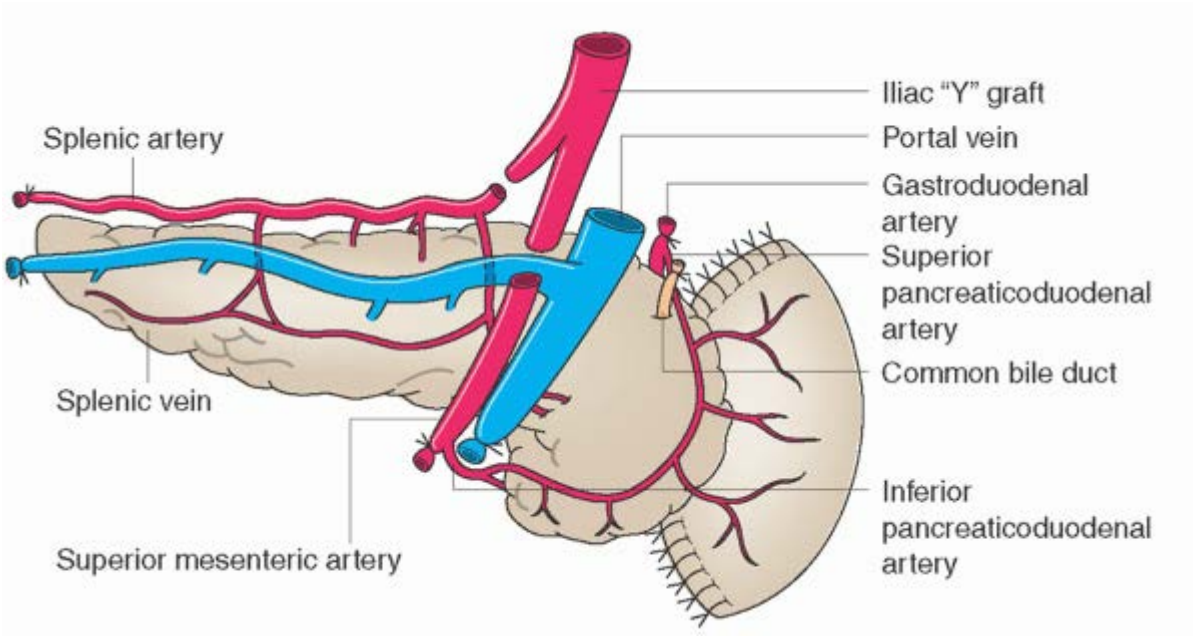

Figure 31-1 Anatomy of a Pancreatic Allograft. (From: Sung R. Pancreas and Islet Transplantation. *Greenfield's Surgery: Scientific Principles and Practice.* Philadelphia, PA: Lippincott Williams and Wilkins, 2011.)

ORGAN TRANSPLANTATION

I. KIDNEY

A. End-Stage Renal Disease (ESRD) is the consequence of multiple disease processes; however, diabetes, hypertension, polycystic kidney disease account for majority of cases (Table 31-1).

1. Transplantation

a. Recipient selection. 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.

b. Recipient operation. 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. The transplant renal vein and artery typically are anastomosed to the external iliac vein and artery, respectively. A heparin bolus of 3,000 units may be administered before clamping the iliac vessels. Before reperfusion of the kidney, mannitol (25 g) and furosemide (100 mg) are administered intravenously, and the patient's systolic blood pressure (BP) is maintained above 120 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 preferred. Establishing

an antireflux mechanism is essential for preventing posttransplantation reflux pyelonephritis. This is accomplished by performing an extravesical ureteroneocystostomy (Litch). A double-J ureteral stent is commonly used.

TABLE 31-1 Causes of Renal Failure Requiring Transplantation

Type	Characteristics
Congenital	Aplasia, obstructive uropathy
Hereditary	Alport syndrome (hereditary nephritis), polycystic kidney disease, tuberous sclerosis
Neoplastic	Renal cell carcinoma, Wilms tumor
Progressive	Diabetic neuropathy, chronic pyelonephritis, Goodpasture syndrome (antiglomerular basement membrane disease), hypertension, chronic glomerulonephritis, lupus nephritis, nephrotic syndrome, obstructive uropathy, scleroderma, amyloidosis
Traumatic	Vascular occlusion, parenchymal destruction

c. Postoperative considerations

(1) Intravenous fluid replacement. In general, the patient should be kept euvolemic or mildly hypervolemic in the early posttransplantation period with a goal of 130 mm Hg systolic BP to

ensure adequate perfusion to the new allograft. 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 to 80 mEq/L (60 to 80 mmol/L).

(2) Renal allograft function or nonfunction. If the patient's urine output is low in the early postoperative period (<50 mL/hour), perfusion to the new allograft must be assessed. After adequate volume resuscitation, low dose (²5 mg/hour) dopamine infusion may be added to augment vasomotor tone and perfusion pressure. Early poor function of a transplanted kidney is most commonly due to reversible ATN secondary to reperfusion injury. 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 (renal ultrasonography) or urinary leak must be obtained. If flow is confirmed, dialysis can be continued until allograft function recovers.

d. Complications

(1) Lymphoceles are lymph collections that occur because of lymphatic leaks in the retroperitoneum. They present 1 week to several weeks after transplantation and are best diagnosed by ultrasonography. Most are asymptomatic and are found incidentally. Treatment of symptomatic lymphoceles consists of drainage into the peritoneum, laparoscopic or open.

(2) Renal artery and vein thrombosis. Arterial and venous thromboses most often occur in the first 1 to 3 days after transplantation. If the kidney had been functioning but a sudden cessation of urine output occurs, graft thrombosis should be suspected. A rapid rise in serum creatinine, graft swelling, and local pain ensues. The diagnosis is made by technetium-99m renal scan or Doppler ultrasonography. Unless the problem is diagnosed and repaired immediately, the graft will be lost and transplantation nephrectomy will be required.

(3) 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. A renal scan demonstrates radioisotope outside the urinary tract. Urine leaks are treated by placing a bladder catheter to reduce intravesical pressure and subsequent surgical exploration.

(4) Rejection is infrequent and inversely correlated with degree of HLA matching. Treatment of ACR is pulse steroids, while antithymocyte preparations can be used in situations involving steroid resistant rejection (Table 31-2).

TABLE 31-2 Treatment of Rejection

Corticosteroids

Intravenous pulse, methylprednisolone

7 mg/kg QD for 3 days

Consider if rejection is early (<3 mo) or mild

Oral pulse, prednisone

3 mg/kg QD in 2-4 divided doses for 3-5 days

After pulse, restart steroids at previous dose

Use if patient is reliable and rejection is early or mild

Tacrolimus

Target 12-hr trough level 5-15 ng/mL

Antilymphocyte preparations

Thymoglobulin

2-3 mg/kg IV for 3-4 days

Myfortic

720 mg PO BID

Rapamycin

4 mg PO QD, target level 8-20 ng/mL

Plasmapheresis

Consider for antibody-mediated rejection

BID, twice a day; IV, intravenously; PO, orally; QD, daily.

TABLE 31-3 Long-term Maintenance Immunosuppression for Renal Transplantation

Myfortic 720 mg PO BID

Reduce to 360 mg PO BID when used with tacrolimus and for WBC <5,000/mm^3, diarrhea, first week posttransplant

Prednisone

1 mg/kg QD for days 1-3

20 mg QD for days 4-14

15 mg QD for week 3

10 mg QD for week 4

5 mg QD for week 5 and onward

Tacrolimus

Dose titrated to achieve trough level 5-7 ng/mL (FPIA)[a]

Levels >15 ng/mL are considered toxic

[a] [a]FPIA, fluorescence polarization immunoassay.

e. Graft surveillance. After the initial 3-month period, when acute rejection becomes less of a risk, tacrolimus and steroid doses are tapered. Chronic long-term immunosuppression can be maintained at lower levels than those required for induction (Table 31-3). Rarely immunosuppression can be discontinued completely.

II. LIVER

A. Indications include complications attributable to ESLD (Table 31-4). While Hepatitis C has been the leading contributor to ESLD, Nonalcoholic Steatohepatitis (NASH) has become increasingly prevalent. At the largest single institution experience, OLT for NASH increased five-fold from 2002 to 2011 (*Ann Surg.* 2012;256(4):624-633). Although outcomes were comparable with other indications for OLT, there are significantly more healthcare resources consumed for this group of recipients.

1. Transplantation for hepatic malignancy. Cirrhosis is a risk factor for HCC. Given that most patients who develop HCC die from their underlying cirrhosis rather than from metastatic disease, it was

reasoned that transplantation may be a potentially curative approach to the primary tumor as well as the underlying pathology. The **Milan Criteria** are outcome driven and establish guidelines for considering OLT in patients who present with **early stage I or II HCC** and underlying cirrhosis (Table 31-4). Given the concern for HCC progression while awaiting transplantation, candidates receive MELD exception points beyond what is calculated from their cirrhosis.

TABLE 31-4 Most Common Indications for Orthotopic Liver Transplantation

Adults	
Chronic hepatitis C	Primary biliary cirrhosis
Alcoholic liver disease	Primary sclerosing cholangitis
Chronic hepatitis B	Autoimmune hepatitis
Children	

Extrahepatic biliary atresia	Primary hepatic tumors
α_1-Antitrypsin deficiency	Metabolic liver disease
Cystic fibrosis	

B. Recipient Operation. OLT comprises three distinct sequential phases. The *first phase* involves the dissection and removal 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 (Fig. 31-2). VVB shunts blood from the portal vein and infrahepatic inferior vena cava (IVC) to the axillary, subclavian, or jugular veins. Alternatively, many transplant surgeons will create a temporary portocaval shunt, which has the advantages of VVB with much less risk and cost. Maintenance of venous return from the kidneys and lower extremities 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. Finally, 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 or a choledochojejunostomy. A duct-to-duct anastomosis is preferable, but 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, or secondary biliary cirrhosis).

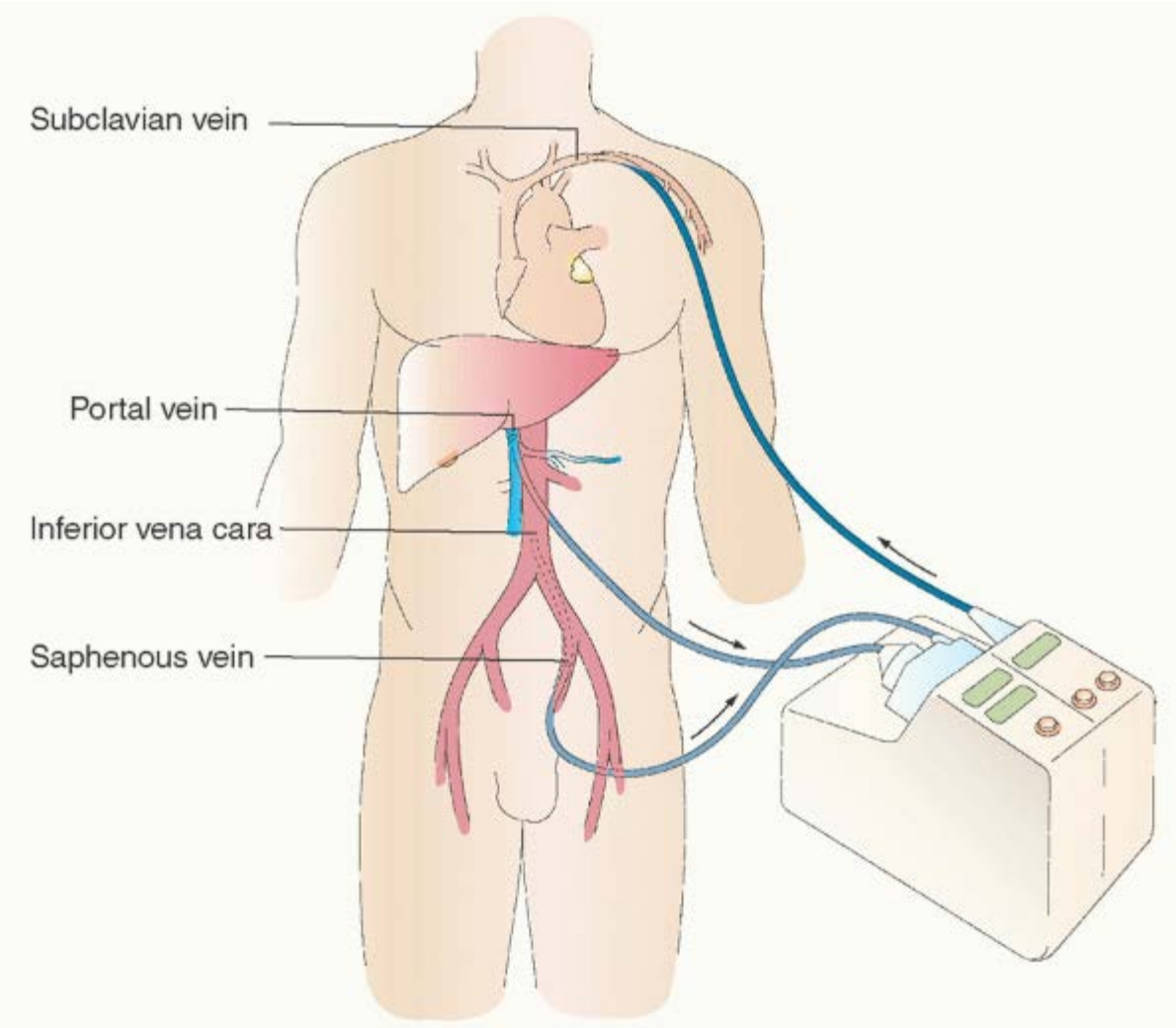

Figure 31-2 Hepatic Transplantation with Veno-Venous Bypass. (From: Welling T, Pelletier S. Hepatic Transplantation. *Greenfield's Surgery: Scientific Principles and Practice.* Philadelphia, PA: Lippincott Williams and Wilkins, 2011.)

C. Postoperative Considerations

1. 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 allograft function continues postoperatively, initially occurring every 12 hours. Satisfactory 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 to

48 hours postoperatively. Persistent transaminitis should prompt a liver ultrasound to assess vessel patency and flow.

D. Complications

1. 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). The incidence is approximately 1% and without retransplantation, death ensues.

2. Hepatic artery thrombosis in the early posttransplantation period may lead to fever, hemodynamic instability, and rapid deterioration, 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 thrombosis may be treated by attempted thrombectomy; however, this is usually unsuccessful and retransplantation is needed.

3. Portal vein stenosis or thrombosis is rare. When it occurs, the patient's condition may deteriorate rapidly, with profound hepatic dysfunction, massive ascites, renal failure, and hemodynamic instability. Although surgical thrombectomy may be successful, urgent retransplantation is often necessary.

4. Biliary stricture 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 anastomosis.

III. PANCREAS

A. Indications. Most patients are type I diabetics with concomitant nephropathy who are evaluated for a pancreas transplant in conjunction with kidney transplantation. Ninety-five percent of all pancreas transplants are performed in conjunction with a kidney transplant. Whole organ pancreas transplantation represents the only therapeutic option for long-term insulin independence.

B. Graft Selection

1. Simultaneous kidney-pancreas transplantation (SPK) may be considered in insulin-dependent diabetic patients who are dialysis dependent (or imminent) and have a creatinine clearance of less than 30 mL/minute. Since alloreactivity among donor organs is concordant, an advantage of combined transplantation includes the ability to monitor pancreas rejection by monitoring renal rejection.

2. Pancreas after kidney transplantation (PAK). Patients with living donors can be listed separately for deceased pancreas transplantation; however, since the organs are immunologically distinct, pancreas allograft monitoring is more difficult and outcomes are worse.

3. Pancreas alone transplantation (PAT). Few patients with complications from type I diabetes who are not uremic are considered for pancreas only transplantation. Patients must be brittle diabetics who have experienced life-threatening hypoglycemic episodes.

4. Pancreatic islet cell transplantation is still investigational and has not received widespread acceptance. Pancreatic islet cells are isolated and injected into the portal vein for engraftment in the liver. The major problems have been in obtaining enough islet cells to attain glucose homeostasis and failing to achieve long-term insulin independence.

C. Recipient Operation. The most widely accepted technique of pancreatic transplantation in the United States uses whole-organ pancreas with venous drainage into the systemic circulation and enteric exocrine drainage. Some centers advocate portal venous drainage. 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 (Fig. 31-1). 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 graft is anastomosed to the recipient's external iliac artery. The duodenal segment of the transplant is then opened, and a duodenojejunostomy is created. Alternatively, the duodenal segment can be anastomosed to the bladder. The pancreas transplant is placed in the right paracolic gutter, and if kidney transplantation is to be performed, it is done on the left side.

D. Postoperative Considerations

1. Serum glucose is followed during and after the transplantation. Intravenous insulin infusions should not be needed from the time in the operating room or are stopped within the first few hours after pancreas transplantation.

2. 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 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. Rejection is treated with corticosteroids or antilymphocyte preparations.

3. 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. Other common complications include pancreatitis, UTIs, urethritis, and anastomotic leak from the duodenocystostomy. Infections with CMV also may occur.

IV. INTESTINE.

Intestinal failure refers to the loss of nutritional autonomy due to gut dysfunction (*N Engl J Med.*

2009;361:10). Both anatomic and functional disease states can result in intestinal failure; however, loss of intestinal length resulting in malabsorption is the leading cause.

A. Total parenteral nutrition (TPN) is the mainstay for intestinal replacement, although long-term therapy can be associated with significant morbidity and mortality. While the 1-year survival rate of patients on TPN is

approximately 90%, the 3- and 5-year rates drop to 70% and 63%, respectively (*N Engl J Med.* 2009;361:10).

1. Complications of long-term TPN

a. Cholestatic liver disease develops in approximately one-half of adults and children who remain on continuous therapy. Recent lipid-sparing PN strategies have shown promise in improving their hepatic, biochemical profile associated with cholestasis (*J Ped Surg.* 2013;48:228-232). If left untreated, PN-associated liver disease can result in liver failure.

b. Line infections are costly complications that are the result of chronic indwelling catheters. This can lead to multiple hospital readmissions for IV antibiotics and significant morbidity if sepsis ensues.

c. Central vein stenosis/thrombosis can occur and result in the loss of vascular access for PN administration.

B. Intestinal Transplantation. The Centers for Medicare and Medicaid Services (CMS) recognize intestinal transplantation as the standard of care for patients who have failed TPN. Approved indications for intestinal transplant include the life-threatening complications associated with TPN therapy (Table 31-5). Timing of referral to an intestinal center can be difficult and is aided by a patient-centered, clinical algorithm (*Nat Rev Gastroenterol Hepatol.* doi: 10.1038/nrgastro.2014.216; Fig. 31-3)

1. Types of grafts

a. Isolated intestinal allograft. Transplantation of the jejunoileum

b. Composite liver and intestinal graft. In cases of severe hepatic dysfunction, composite grafts are necessary. Inclusion of the pancreas and duodenum facilitates en bloc procurement and engraftment (*N Engl J Med.* 2009;361:10).

TABLE 31-5 Failure of Parenteral Nutrition, as Defined by the Centers for Medicare and Medicaid Services[a]

Impending or overt liver failure due to TPN-induced liver injury

Thrombosis of two or more central veins	
Two or more episodes per year of catheter-related systemic sepsis that requires hospitalization	
A single episode of line-related fungemia, septic shock, or acute respiratory distress syndrome	
Frequent episodes of severe dehydration despite intravenous fluid supplementation in addition to TPN	

[a][a]TPN denotes total parenteral nutrition.
From: Fishbein TM. Intestinal transplantation. *N Engl J Med.* 2009;361(10):998-1008, with permission.

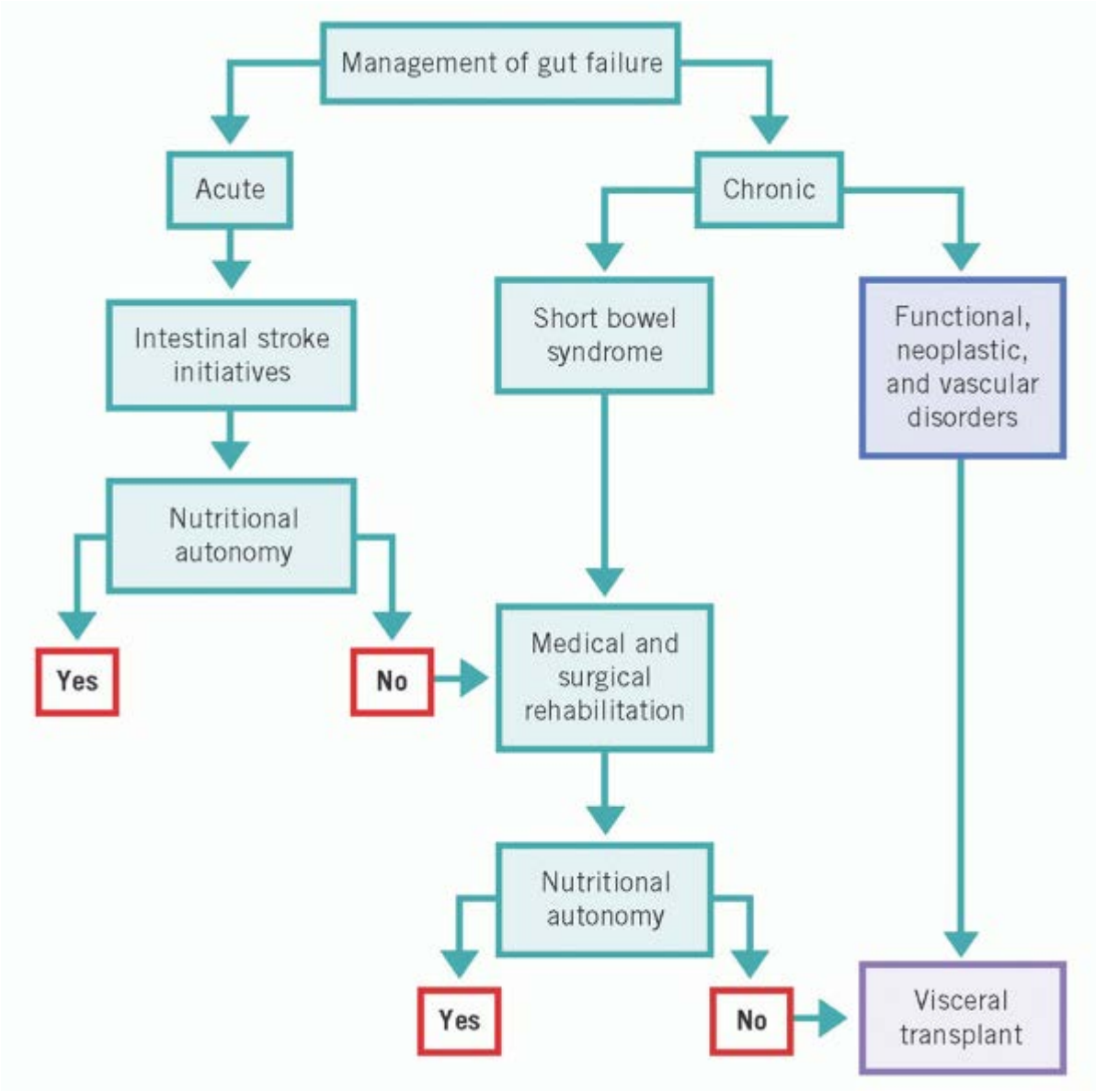

Figure 31-3 Decision making algorithm in the management of intestinal failure. (Abu-Elmagd K. The concept of gut rehabilitation and the future of visceral transplantation. *Nat Rev Gastroenterol Hepatol* 2015;12:108(2)-120, with permission.)

c. Multivisceral graft. Exenteration of the native foregut allows en bloc engraftment of donor stomach, duodenum, pancreas, small intestine and liver. Inclusion of the colon is sometimes performed and is currently under investigation.

(1) Recipient operation. Patients who receive isolated intestinal allografts have vascular anastomoses created between the donor superior mesenteric vein and the recipient portal vein, and between the donor SMA and the recipient aorta. Vascular reconstruction for patients who receive combined liver-intestinal grafts parallels that for patients undergoing a standard 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.

(2) Complications

d. Graft rejection. The introduction of antithymocyte antibodies into induction protocols has correlated with reduction in early acute cellular rejection (ACR). Previously reported in 70% to 90% of intestinal transplant recipients, now only one-third to one-half of patients experience ACR (*N Engl J Med.* 2009;361:10). Given the lack of a reliable noninvasive marker of rejection, intestinal stomas created during transplantation give an access to serial endoscopic biopsies for graft surveillance.

e. Infection. The foremost clinical conundrum that faces intestinal transplant surgeons is distinguishing infectious enteritis from allograft rejection. Both present similarly and have masquerading features on biopsy specimens. Adenovirus, calicivirus, *Clostridium difficile,* and CMV must all be distinguished from rejection.

f. Renal dysfunction. Intestinal transplant recipient are at a higher risk of nephrotoxicity from calcineurin inhibitors because of the higher dose used for immunosuppression. Ojo et al. reported 21.3% of intestinal transplant recipients at 5 years experienced renal dysfunction (*N Engl J Med.* 2003;349:931-940). Recent use of antithymocyte antibody induction therapy associated with decreased target levels of tacrolimus is anticipated to preserve renal function of future recipients (*AJT.* 2014;14:1976-1984).

2. Outcomes

a. Survival. Experienced centers report 1-year patient survival between 86% to 93%, which is on par with other solid organs (*AJT.* 2014;14:1976-1984). Unfortunately, these results do not persist as 3- and 5-year survival rates are a modest 61% and 47%, respectively (*N Engl J Med.* 2009;361:10).

b. Nutritional autonomy. Most recipients wean from TPN and achieve nutritional autonomy after transplantation (*OPTN/SRTR 2011 Annual Data Report*). Oral aversion can be an issue in children who have never developed feeding skills.

c. Cost. Similar to renal transplantation, intestinal transplantation becomes cost effective over continuous PN after 2 years (*Gastroenterology.* 2006;130:s158-s162).

d. Quality of Life (QOL). Patients who receive an intestinal transplant seem to enjoy a better QOL than those who remain on continuous PN (*AJT.* 2014;14:1976-1984). Factors that diminish QOL after transplantation include persistent g-tube or ostomies and repeat hospitalizations.

CHAPTER 31: TRANSPLANTATION

Multiple Choice Questions

1. During preoperative evaluation of a 33-year-old male with ESRD from polycystic kidney disease, his mother comes forward and wishes to donate one of her kidneys to her son. What factors are taken into consideration when evaluating potential living donors?

a. Blood type

b. HLA type

c. Co-morbid conditions

d. Psychosocial influences

e. All of the above

View Answer

2. A 65-year-old male is 3 days status postorthotopic liver transplantation. On rounds you note new bilious output from his surgical drain. This finding along with a persistent transaminitis should prompt an evaluation for:

a. Portal vein thrombosis

b. Enterocutaneous fistula

c. Hepatic arterial thrombosis

d. Rejection

e. All of the above

View Answer

3. A 55-year-old male with cirrhosis from HCV visits your office to discuss his recent surveillance imaging. “They told me I have a few spots on my liver.” What findings would preclude this patient from being considered for transplantation for HCC?

a. Single 4.5 cm lesion

b. 3 lesions: 2.5 cm, 2 cm, 3.5 cm

c. 2 lesions: 2.5 cm, 2 cm

d. Single 3 cm lesion

e. All of the above are contraindications for transplantation

View Answer

4. A 76-year-old female s/p left-sided brachiobasilic AVF placement over 1 year ago presents to the emergency department complaining of left upper arm swelling and pain. On examination, she has good distal

perfusion, but you are not able to palpate a thrill, only a dopplerable signal. What is the next appropriate step in management of venous hypertension with outflow stenosis?

a. Percutaneous fistulogram with therapeutic dilation of stenotic outflow segment

b. Open thromboembolectomy

c. Anticoagulation

d. Arm elevation and physical therapy

e. Watchful waiting

View Answer

5. A 45-year-old female with type I diabetes complicated by nephropathy and ESRD is referred to your office for kidney transplant evaluation. She asks you whether she would benefit from a combined kidney-pancreas transplant. Which of the following statements regarding whole-organ pancreas transplantation are true?

a. It is usually performed alone without concomitant kidney transplantation

b. It is the only therapeutic modality that can achieve long term insulin independence

c. Compared to islet cell transplantation, there is a greater chance of requiring insulin after whole-organ pancreas transplantation

d. Sequela of diabetes, such as neuropathy and retinopathy are unaffected after pancreas transplantation

e. All of the above

View Answer

6. A 55-year-old male is 2 days status postliver transplantation. Despite normalization of his coagulation profile and bilirubin level, his transaminases are persistently elevated. Which noninvasive test would be most appropriate in the workup of this patient?

a. MRCP

b. CT angiography

c. KUB

d. Liver duplex ultrasound

e. No further testing is indicated at this time

View Answer

7. Three days following deceased-donor renal transplantation, your patient begins to complain of swelling and tenderness over her graft. Despite having adequate urine output over the last 12 hours, you notice a moderate rise in her serum creatinine. Which study would be most helpful in making the diagnosis?

a. Ultrasound of the graft

b. Nuclear medicine scan

c. Retrograde urethrogram

d. CT scan

e. MRI

View Answer

8. A 45-year-old female with intestinal failure secondary to short gut syndrome is hospitalized with her third serious line infection in the last 12 months. Which of the following are indications for intestinal transplantation?

a. Frequent episodes of dehydration despite IV fluid supplementation with TPN

b. Two or more serious line-related infection per year, 1 line-related fungal infection or episode of shock due to line sepsis

c. Loss of two or more central venous access sites

d. Peripheral nutrition associated liver disease (PNALD)

e. All of the above

View Answer

9. A 55-year-old female with ESRD secondary to Alport syndrome is undergoing evaluation for kidney transplantation. Which of these factors should not raise her PRA?

a. Previous pregnancy

b. History of blood transfusion

c. Previous transplant

d. Prior blood donation

e. All of the above will raise one's PRA

View Answer

32
Pediatric Surgery

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Care of pediatric surgical patients presents unique challenges for the general surgeon. A thorough understanding of pre- and postnatal development, nutritional needs, and pathophysiology that is quite distinct from adult conditions is essential to successfully managing such patients.

I. FLUIDS AND NUTRITION

A. Fluid Requirements. Normal daily fluid requirements for children are higher than those of adults per unit of body weight due to greater insensible and urinary losses. Infants have a particularly high ratio of body surface area to volume and a limited ability to concentrate urine due to immature renal function. In addition, total body water is a higher percentage of body weight (75% in children vs. 60% in adults). Postoperative fluid replacement can be calculated using the Ò4-2-1Ó rule (4 mL for the first 10 kg of body weight, 2 mL for the next 10 kg, and 1 mL for each additional kg after the first 20 kg body weight) and should be adjusted to support hemodynamic stability and urine output between 1 and 2 mL/kg/hour.

1. Fluid boluses are carefully calculated using total weight as a surrogate for total blood volume, which is approximately 80 mL/kg. Isotonic fluid boluses should be in a volume of 10 to 20 mL/kg. Initial transfusion of packed red blood cells is typically 10 mL/kg.

B. Nutrition. The unique caloric requirements for the pediatric surgical patient are a critical component of perioperative care and one of the areas in which perioperative care varies most from that of adult patients. Nutritional intake for infants and children must carefully account for the needs for both growth and maintenance. Calculations of daily nutritional needs are usually based on calories per kilogram body weight.

1. Parenteral nutrition is a critical component in the management of pediatric surgery patients. Several surgical conditions, particularly neonatal gastrointestinal anomalies, require the surgeon to possess a basic understanding of nutritional values and titration of TPN (Table 32-1).

a. Common nutritional values in pediatric surgery:

(1) Newborn enteral caloric requirements are 100 to 120 kcal/kg/day, while parenteral are 90 to 100 kcal/kg/day.

(2) Daily caloric needs decrease throughout childhood to adult values of roughly 25 to 30

kcal/kg/day (Table 32-2).

(3) A newborn is expected to gain weight at about 15 to 30 g/day.

(4) Most enteral infant formulas and breast milk contain 20 kcal/oz.

TABLE 32-1 Components of Parenteral Nutrition and Titration in Neonates[a],[b]

	Caloric Density	% of Daily Calories	Starting Value	Daily Titration	Goal
Carbohydrate	3.4 kcal/g	60%	5.5 mg/kg/min	2-3 mg/kg/min	12-14 mg/kg/min
Fat	4 kcal/g	30%	1 g/kg/day	1 g/kg/day	3 g/kg/day
Protein	9 kcal/g	10%	2.5 g/kg/day	0.5-1 g/kg/day	Term infant: 3 g/kg/day Preterm infant: 3.4-4 g/kg/day

[a]Please note that starter TPN is indicated when neonates are <1,500 g or <32 weeks. This helps promote an anabolic state after withdrawal of placental nutrients and protein and aids in glucose utilization, overall improving neurodevelopment.

[b]Addition of insulin is not routine in pediatric TPN infusions, as exogenous insulin-naive cells are particularly sensitive to its effects and risks hypoglycemia. However, blood glucose should be monitored to prevent seizures and impaired neurodevelopment from hypoglycemia and osmotic diuresis with possible resultant intraventricular hemorrhage in the setting from hyperglycemia.

TABLE 32-2 Daily Caloric Needs in Children

Age (yrs)	REE (kcal/kg/day)	Average (kcal/kg/day)
<36 wks	63	120
0-0.5	53	108
0.5-1	56	98
1-3	57	102
4-6	48	90
7-10	40	70
11-14	32	55
15-18	27	45

REE, resting energy expenditure.

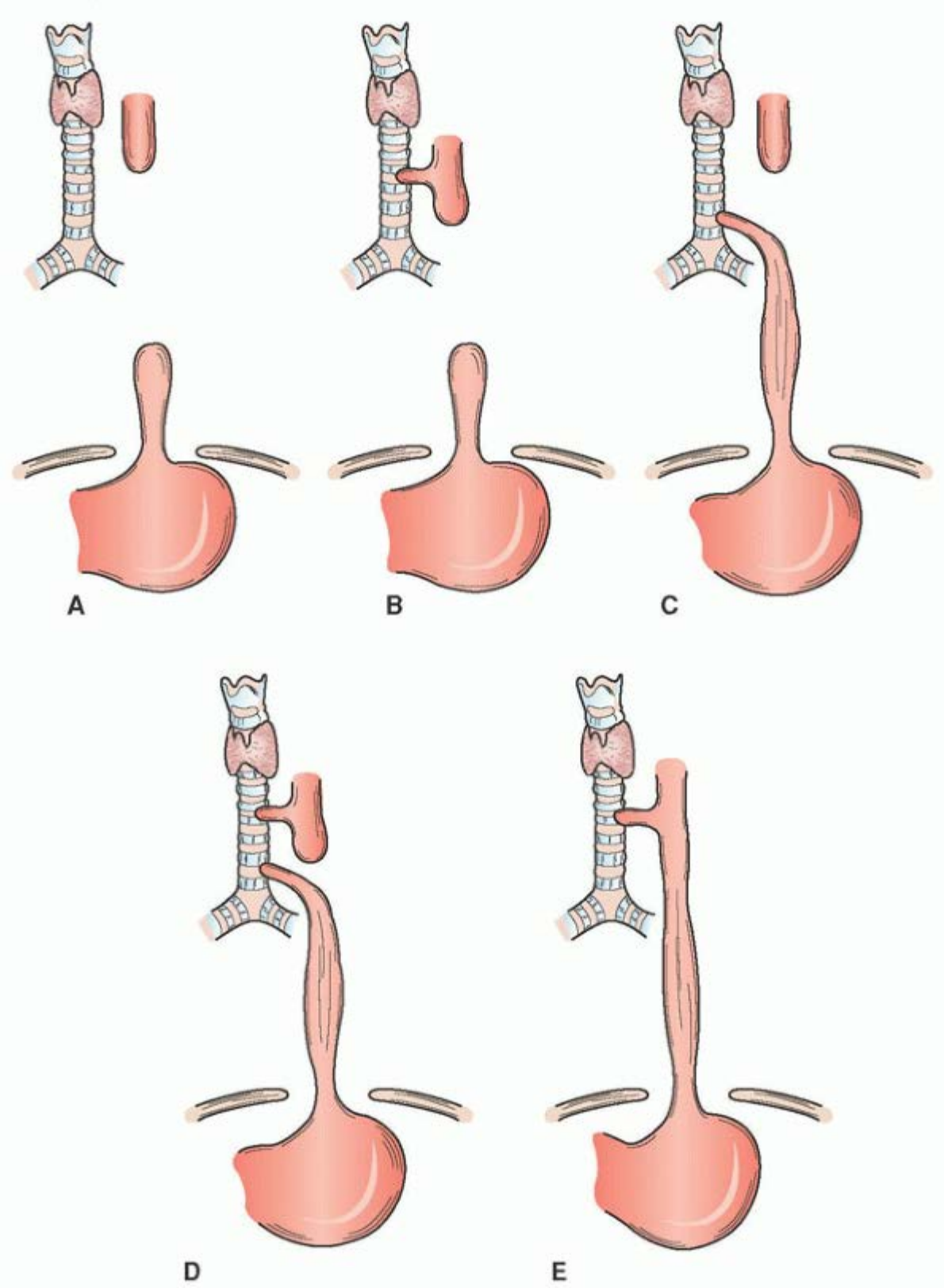

Figure 32-1 Variants of TEF. **A:** Pure esophageal atresia without fistula (5% to 7% of cases). **B:** Proximal fistula and distal pouch (<1% of cases). **C:** Proximal pouch with distal fistula (85% to 90% of cases). **D:** Atresia with proximal and distal fistulas (<1% of cases). **E:** Fistula without atresia (ÒH typeÓ) (2% to 6% occurrence).

II. NEONATAL SURGICAL CONDITIONS

A. Tracheoesophageal Malformations. Tracheoesophageal malformations describe a spectrum of anomalies including esophageal atresia (EA) alone or as a component of a tracheoesophageal fistula (TEF) (Fig. 32-1). The incidence of these disorders is 3 in 10,000 births, with a slight male predominance.

1. Diagnosis

a. Clinical examination. Suspicion should be raised for EA/TEF in the neonatal patient with difficulty clearing secretions, regurgitation of saliva, or respiratory difficulty during feeds. The classic clinical finding in EA/TEF is inability to pass an oro- or nasogastric tube.

b. Imaging. Visualization of a coiled orogastric tube in the upper chest on plain radiograph implies EA (Fig. 32-2), and associated presence

of gas in the GI tract confirms a distal communication between the respiratory and GI systems (TEF). Imaging should also be reviewed with concern for aspiration of feeds and secretions. These imaging modalities are often sufficient. Contrast studies are rarely necessary to visualize the level of EA and/or TEF, and are associated with risk for aspiration.

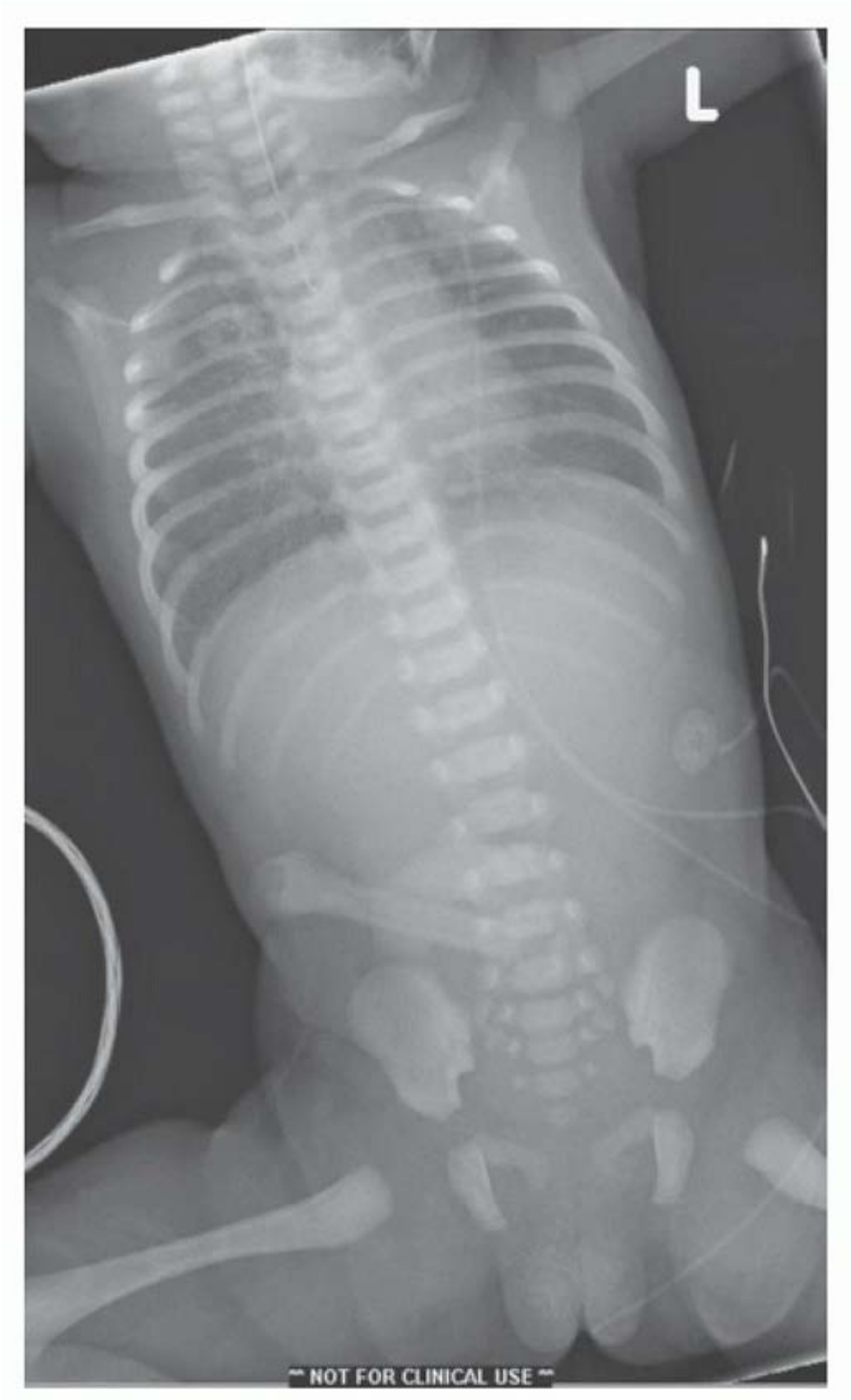

Figure 32-2 Radiographic findings of esophageal atresia. Note the orogastric tube ending in upper chest and the absence of bowel gas, suggesting pure esophageal atresia without any communication between the airway and gastrointestinal tract.

2. Workup. Up to two-thirds of EA/TEF patients have associated anomalies. Physical examination and imaging should be used to search for VACTERL anomalies (*V*ertebral, *A*norectal, *C*ardiac, *T*racheal, *E*sophageal, *R*enal, and *L*imb).

3. Management

a. Preoperative management should include decompression of the proximal esophageal pouch along with elevation of the head of the bed to 30 degrees to reduce the risk of aspiration from reflux of gastric contents through the TEF. Ventilation may prove challenging in the presence of a TEF, as positive-pressure can be directed through the TEF into the GI system, resulting in increased abdominal distention and pressure. Passage of an endotracheal tube beyond the fistula, right mainstem intubation, or high frequency oscillatory ventilation may be necessary to appropriately ventilate the patient.

b. Operative approach is typically determined by the side of the aortic arch. A TEF with a normal left-sided aortic arch is approached through a right fourth intercostal space, retropleural thoracotomy. The fistula is ligated, and the proximal esophageal stump is mobilized in an attempt to create a tension-free primary anastomosis. Pure EA without TEF is typically associated with a long-gap, and a gastrostomy is the usual first procedure. Later, long-gap EA can be reconstructed with a variety of techniques including lengthening myotomy, colonic interposition, gastric pull-up or delayed repair after a period of growth or traction (the Foker process). An isolated TEF without EA is often more proximal and can be approached via a cervical incision.

B. Congenital Diaphragmatic Hernia (CDH). CDH has an incidence of 1 in every 2,000 to 5,000 births and an equal male to female distribution. CDH develops as a result of incomplete diaphragm development at 8 weeks of gestation. Abdominal contents herniate into the chest, resulting in lung compression and subsequent hypoplasia that is worse on the affected side. Moreover, the pulmonary vasculature develops increased tone in the muscular arterioles, which predisposes to pulmonary hypertension and vasospasm. The majority (80% to 90%) of hernias are posterolateral (Bochdalek), with the remainder presenting anteriorly (Morgagni). Approximately 90% of hernias occur on the left.

1. Diagnosis of CDH is frequently made during routine prenatal screening ultrasounds. Prenatal diagnosis should set in motion a team of critical care neonatologists and surgeons available for respiratory management at the time of birth. For patients not diagnosed prenatally, significant respiratory distress including tachypnea, cyanosis, and retractions should

raise immediate concern. Asymmetric chest wall diameter and a scaphoid abdomen as a result of abdominal contents herniating into the chest are additional clinical clues. A second set of patients will present after an initial ÒhoneymoonÓ phase of several hours that can occur before signs of pulmonary hypertension and hypoxemia present. Finally, less than 20% of cases present after the first 24 hours of life with respiratory distress, pneumonia, and feeding intolerance or intestinal

obstructions. In all patients, a plain chest radiograph demonstrating bowel gas patterns in the chest is sufficient for diagnosis (Fig. 32-3). Major negative predictors of outcome for prenatally diagnosed CDH include an intrathoracic liver, presence of major congenital heart disease, and other associated anomalies.

2. Management

a. Immediate postnatal care must focus on cardiopulmonary stabilization. Endotracheal intubation is often required, as is orogastric or nasogastric tube decompression to reduce gastric distension. Conventional ventilation with low positive-pressure, permissive hypercapnea, and stable hypoxemia (tolerance of pre-ductal oxygen saturations above 80%) has been shown to improve survival (*J Pediatr Surg.* 2002;37:357-366). Complex cases may require further cardiopulmonary stabilizing measures such as high frequency oscillator ventilation, inhaled pulmonary vasodilators such as nitric oxide, epoprostenol, or sildenafil, and extracorporeal membrane oxygenation.

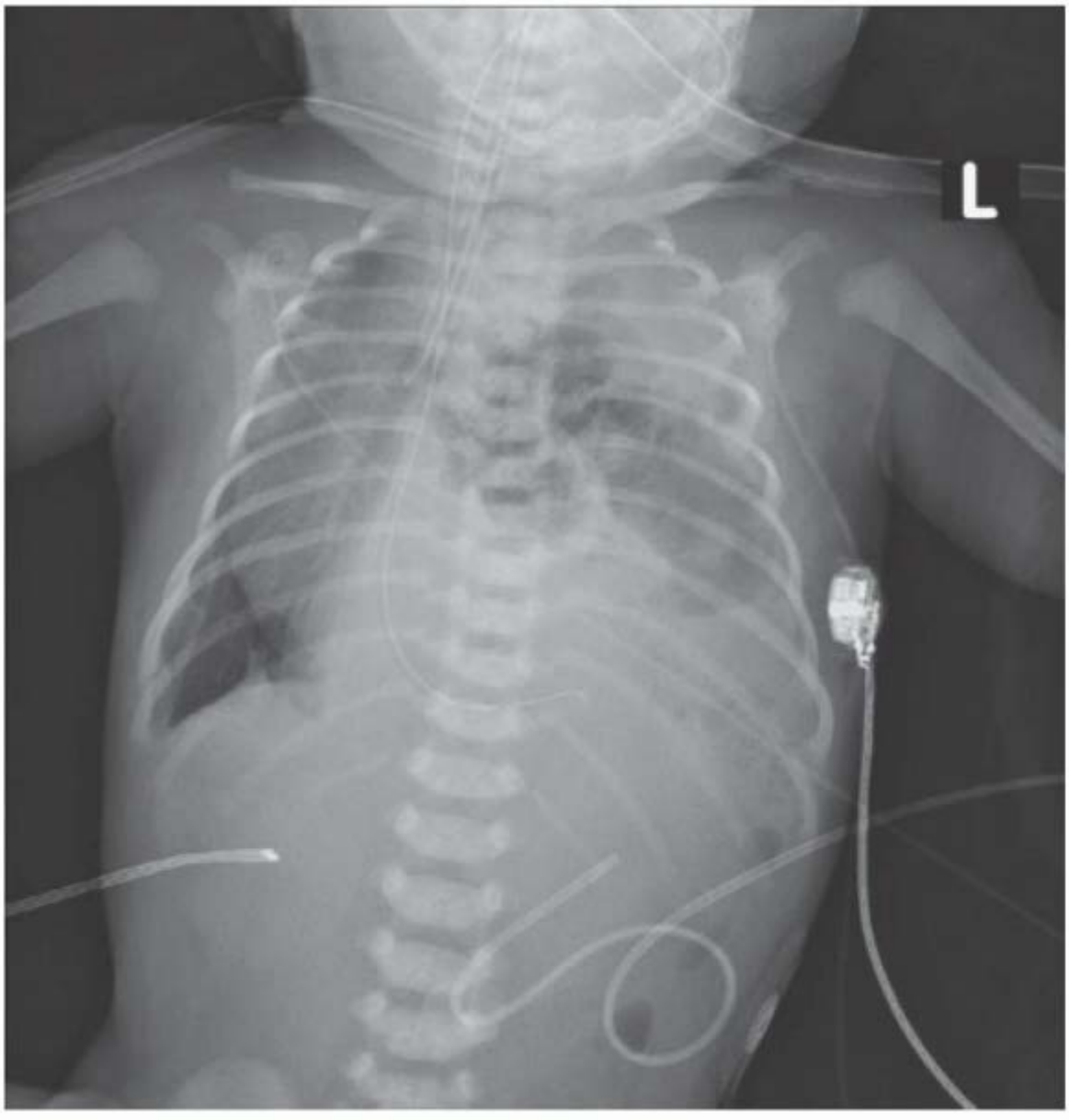

Figure 32-3 A case of congenital diaphragmatic hernia showing bowel filling the left chest.

(1) Extracorporeal membrane oxygenation (ECMO), also known as Extracorporeal Life Support (ECLS), is frequently employed in cases of respiratory failure, with the greatest experience reported being in neonatal patients (*Semin Perinatol.* 2005;29:24-33). Frequent indications for ECMO in addition to CDH include meconium aspiration, respiratory distress syndrome, pulmonary hypertension, and sepsis. Selection criteria commonly include patients with a reversible cardiopulmonary process, greater than 2 kg body weight, and greater than 34 weeks of gestation. Exclusion criteria include irreversible cardiopulmonary congenital anomalies and contraindications to anticoagulation, such as the presence of greater than Grade 1 intracranial hemorrhage.

The ECMO circuit is intended to remove carbon dioxide and provide oxygenated blood while supporting or temporarily replacing the normal function the lungs (veno-veno ÒV-VÓ ECMO) or both the lungs and heart (veno-arterial ÒV-AÓ ECMO). Veno-arterial bypass is used most commonly, and the right internal jugular vein and common carotid artery are typically chosen for cannulation because of their large size, accessibility, and adequate collateral circulation. Systemic anticoagulation is required, necessitating serial coagulation monitoring, platelet and hematocrit trends, and daily head ultrasounds to screen for intracranial hemorrhage.

Once the patient has achieved acceptable arterial blood gas measurements on circuit, ECMO circuit flow rates are maintained until there are signs of pulmonary improvement (increased pO_2 levels, clearing chest radiographs, and improved lung compliance). At this point, ECMO flow rates can be weaned slowly. Ultimately, the ECMO circuit can be clamped under close observation to ensure clinical stability and adequate postclamp blood-gas values. Once the patient has tolerated clamping, the cannulas are surgically removed with subsequent ligation of the cannulated vein and/or artery. Survival rates on ECMO with appropriate patient selection are greater than 80%. Morbidity is highest in patients of low birth weight and gestational age less than 35 weeks and includes sequelae of bleeding, exposure to blood products, neurologic deficits or seizures, renal failure, infection, and risk of mechanical failure of the circuit.

b. Operative intervention. Once the patient has been shown stability on lower ventilator settings, operative fixation of the hernia should be undertaken. A subcostal incision or thoracotomy is made on the affected side. Herniated abdominal contents are dissected free and reduced into the abdomen. The defect is repaired primarily or with a synthetic patch if needed.

3. Outcomes. Mortality of CDH is difficult to assess, as the outcome of cases presenting with profound respiratory distress at birth contrast greatly with patients presenting greater than 24 hours after birth.

Overall survival rates greater than 70% are frequently reported (*J Pediatr Surg.* 2004;39:657-660). Significant respiratory disease at birth can result in neurologic deficits, including developmental delay and seizures. In addition, gastroesophageal reflux is common. Patients must be followed to observe for respiratory symptoms, management of chronic lung disease, surveillance for hernia recurrence, and the possibility of reoperation if a patch was used in the initial operation.

C. Abdominal Wall Defects. During normal development of the human embryo, the midgut herniates outward through the umbilical ring and continues to grow. By the 11th week of gestation, the midgut returns back into the abdominal cavity and undergoes counterclockwise rotation and fixation, along with closure of the umbilical ring. **Omphalocele** is the failure of the abdominal contents to reduce back into the abdomen, resulting in a large hernia covered by a peritoneal sac. In contrast, **gastroschisis** is believed to be the result of an isolated intrauterine vascular insult resulting in an abdominal wall defect to the right of the umbilical cord. The bowel herniates through the defect but is not covered by a sac. Gastroschisis defects tend to be smaller than those of an omphalocele. Although there is a small incidence of associated anomalies with gastroschisis (10% rate of associated intestinal atresias), omphalocele is more typically associated with congenital anomalies, with 50% of cases having an associated genetic and/or cardiac anomaly that often has a major impact on the prognosis of the infant.

1. Diagnosis is often made at the time of prenatal ultrasound after 13 weeks of gestation or simply by clinical examination at the time of birth.

2. Management. Naso- or orogastric tube decompression and broad-spectrum prophylactic antibiotics should be initiated. Heat and fluid losses from exposed viscera should be corrected with intravenous fluids and warming while covering the exposed organs and lower body in a clear plastic bag. Covering the bowel with gauze and pouring warm saline should be avoided as the gauze prevents visualization of the bowel prior to surgery, and the warm saline ultimately drops to room temperature, thereby cooling the infant. In cases of gastroschisis, the bowel may be placed within a silastic silo at the bedside, with gradual reduction into the abdomen while carefully monitoring for abdominal compartment syndrome (*J Pediatr Surg.* 2009;44(11):2126-2129). In cases of omphalocele, the sac serves as sufficient coverage until operative closure is undertaken.

a. Operative repair. Once the herniated abdominal contents can be reduced into the abdomen, the abdominal wall defect may be closed primarily. In omphalocele, the sac should be excised with care to dissect out and ligate the umbilical vessels. Repair of gastroschisis should involve careful examination of the length of the bowel for atresias, which may be managed primarily or with diversion and subsequent repair. In the case of large abdominal wall defects that cannot be closed primarily, the skin can be closed over the bowel or, if this is not possible, the exposed bowel can be allowed to granulate. Either management method requires operative repair of the fascial defect at a later date.

D. Necrotizing Enterocolitis (NEC). NEC is the most common neonatal gastrointestinal emergency. It is characterized by an acute inflammatory disease of the intestine associated with ulceration and necrosis of the gastrointestinal tract most frequently affecting the small bowel. The pathogenesis is believed to be multifactorial involving prematurity, an immature gut barrier defense, bacteria, enteral feeding, and hypoxia/ischemia or low-flow states. The incidence of NEC is 1 to 3 per 1,000 live births, with prematurity being the single most prominent risk factor.

1. Diagnosis requires a high clinical suspicion. NEC is unusual within the first few days of life but approximately 80% of cases occur within the first month of life. Clinical examination may demonstrate a lethargic or irritable patient with abdominal distention, feeding intolerance, or passage of bloody stools. Advanced cases may show signs of peritonitis including erythema of the abdominal wall. Temperature instability is common, as well as increasing incidence of apneic or bradycardic episodes. Laboratory studies may reveal increasing leukocytosis or leukopenia as well as metabolic acidosis and thrombocytopenia. Plain radiographs often reveal dilated loops of bowel with evidence of ischemia including pneumatosis intestinalis (Fig. 32-4), portal venous gas, or pneumoperitoneum. Based on clinical examination and laboratory and imaging data, patients and their expectant management can be guided by Bell staging (*Ann Surg.* 1978;187(1):1-7).

2. Initial management of the hemodynamically stable patient should focus on bowel rest, nasogastric decompression, parenteral nutrition, and broad-spectrum antibiotics. Antibiotic regimen historically was a

combination of ampicillin, gentamicin, and clindamycin (*J Pediatr Surg.* 1980;15(4):569-573) but studies have failed to show any single superior regimen (*Cochrane Database Syst Rev.* 2012;8:CD007448). Serial abdominal examinations, laboratory studies including CBC and blood gas, and plain radiographs are valuable studies to determine the success of nonoperative management (Fig. 32-5). Fifty percent of patients will experience improvement and resolution of signs of NEC with nonoperative management.

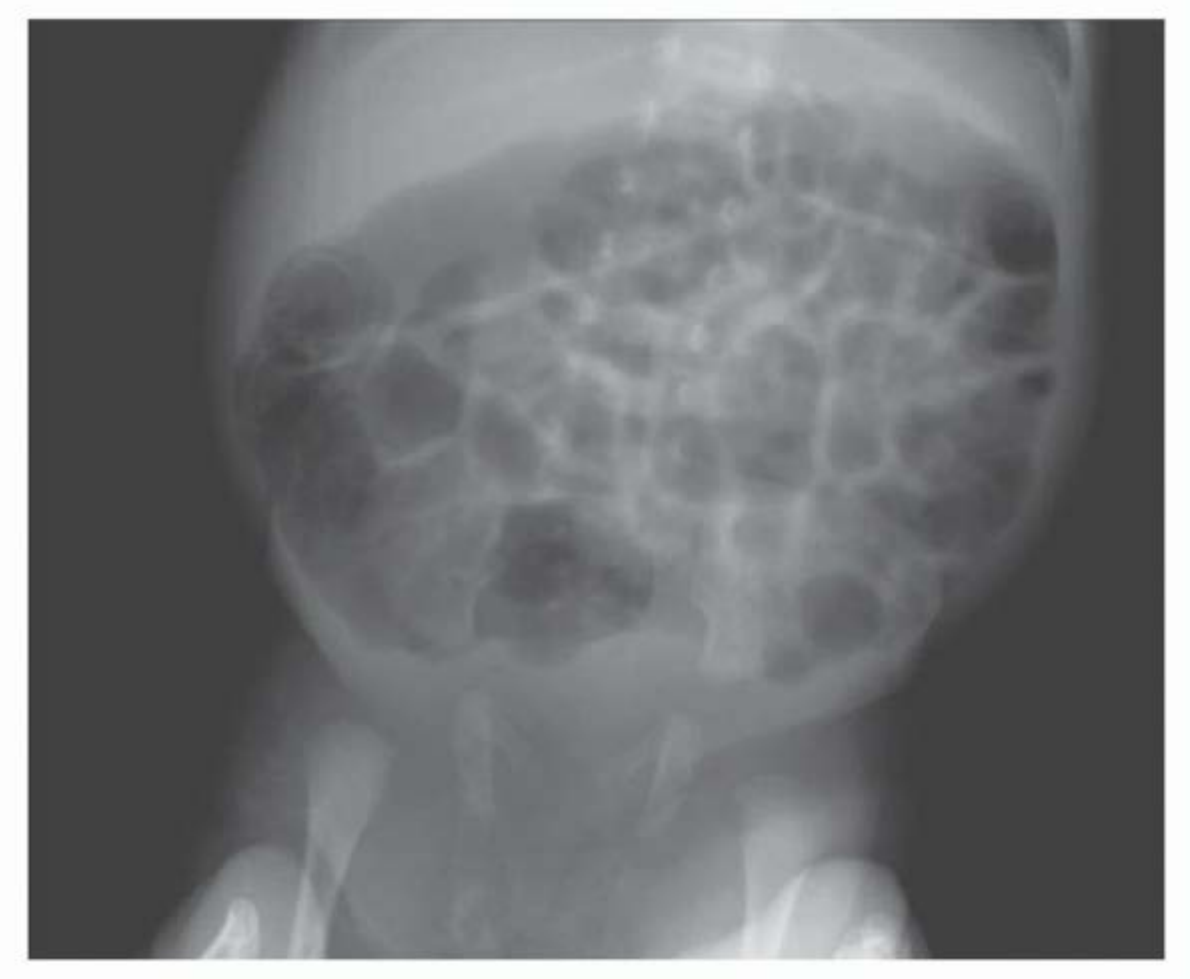

Figure 32-4 Radiographic findings of necrotizing enterocolitis. Pneumatosis intestinalis is visualized in the right upper quadrant.

3. Operative management is indicated for intestinal perforation, as indicated by free air on abdominal radiograph. Relative indications for operative management include overall clinical deterioration, abdominal wall cellulitis, worsening acidosis, falling white blood cell or platelet count, a palpable abdominal mass, or a persistent fixed loop on repeated abdominal radiographs. Improvement of clinical status can be achieved with laparotomy, including resection of nonviable bowel and anastomoses or stomas as necessary, or with peritoneal drainage, a bedside procedure performed under local anesthesia that has shown comparable outcomes to laparotomy in select patients (*N Engl J Med.* 2006;354(21):2225-2234).

E. Meconium Syndromes, Malrotation, and Intestinal Atresias. Meconium plug or failure to pass meconium within the first 24 hours of life can be associated with a range of diagnoses including cystic fibrosis (CF) and Hirschsprung disease. Unrecognized surgical causes of obstruction can quickly progress to enterocolitis, sepsis, and perforation if unrecognized. Water-soluble contrast enemas can be both therapeutic and diagnostic. Testing for CF and Hirschsprung's is often recommended even in the setting of simple and resolved meconium plug.

1. Meconium ileus represents the earliest manifestation of CF. Plain radiographs will demonstrate dilated loops of small bowel filled with meconium, resulting in an absence of air-fluid levels. Relief of the obstructed bowel, frequently located in the distal ileum, may require laparotomy, in which case an enterotomy is made to remove and irrigate inspissated meconium using saline or *N*-acetylcysteine. Postoperative confirmation of CF diagnosis by sweat testing or genetic analysis is then performed.

2. Malrotation results when the intestine fails to complete its normal 270 degrees counterclockwise rotation about the superior mesenteric artery from the fourth to tenth weeks of gestation. Patients may present later in life but often exhibit bilious emesis and abdominal distention in the neonatal period. Plain abdominal radiographs can shows dilated or normal bowel patterns, but an upper GI contrast study demonstrating failure of the duodenojejunal junction to cross midline with jejunum in the right side of the abdomen is consistent with malrotation. Volvulus may appear as a classic Òbird's beakÓ or corkscrew appearance of the intestine. Mesenteric attachments (Ladd bands) can result in mechanical obstruction.

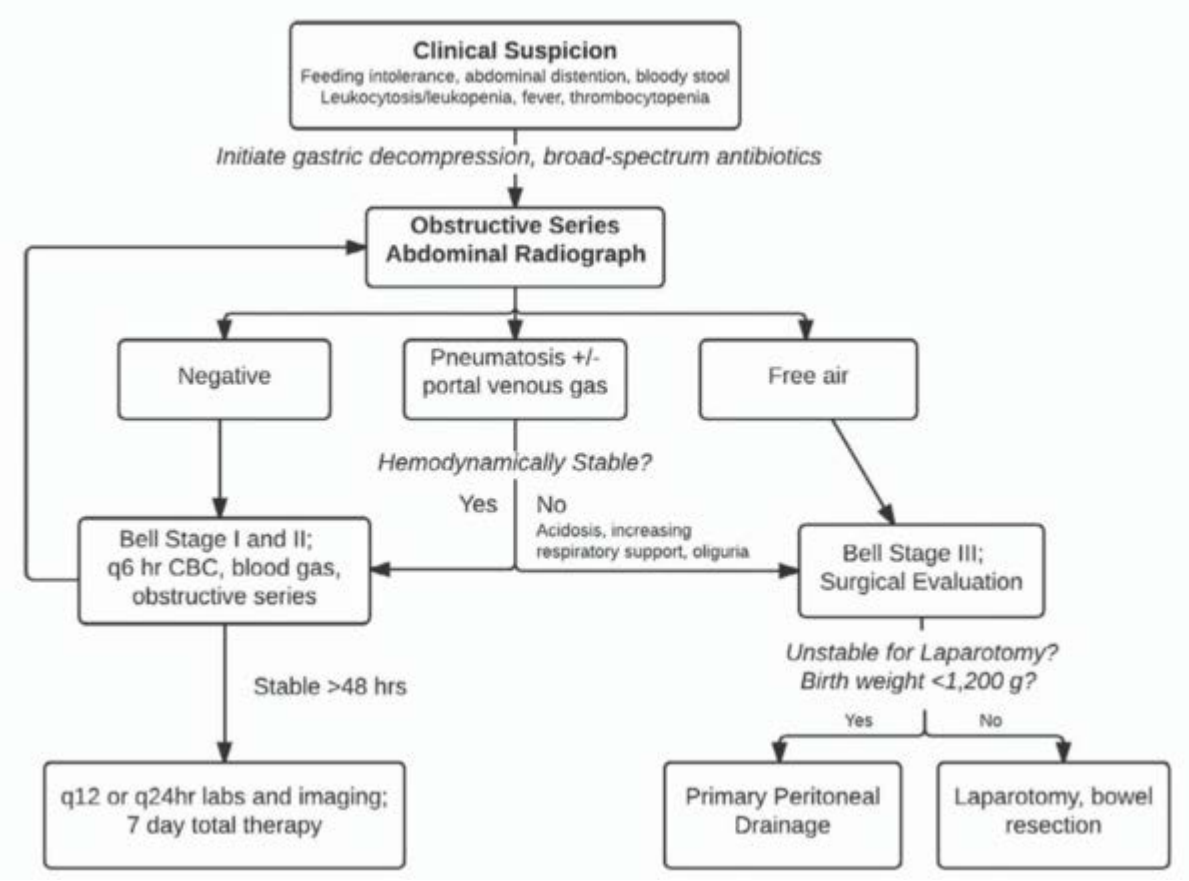

Figure 32-5 Algorithm for management of patients with suspected necrotizing enterocolitis.

a. Surgical treatment for malrotation is the Ladd procedure, during which the bowel is untwisted in a counterclockwise manner (Òturning back the hands of timeÓ). Ladd bands are then divided between the duodenum and colon, which allows for broadening of the mesenteric base and reducing the risk for volvulus. There is no benefit to pexying the duodenum or cecum. An appendectomy is performed to prevent future diagnostic uncertainty since the cecum is ultimately located in an aberrant location.

3. Intestinal atresias, believed to be the result of an intrauterine vascular insult, present as either distal obstructive symptoms such as failure to pass meconium or proximal symptoms including feeding intolerance and bilious emesis. The most common location is the distal small bowel. Prenatal ultrasound may demonstrate polyhydramnios and postnatal imaging with plain abdominal radiograph will show dilated proximal loops with absence of air distally. Upper gastrointestinal contrast studies are helpful in identifying the suspected level of the atresia and ruling out volvulus, which is a surgical emergency.

a. Duodenal atresia results after failure of recanalization of the duodenal lumen. Plain radiographs demonstrate the Òdouble bubbleÓ sign caused by air within both the stomach and duodenum. Duodenal atresia has a much higher association with other conditions than jejunoileal atresias, including prematurity, Down syndrome, maternal polyhydramnios, malrotation, annular pancreas, and biliary atresia. Operative repair may be through duodenoduodenostomy with or without duodenoplasty to account for proximal dilatation of the bowel.

(1) Duodenal web represents a subcategory of duodenal atresia that can present later in life and can be repaired with simple transduodenal excision.

b. Anorectal anomalies include a variety of congenital defects of development that can result in intestinal obstruction. These anomalies are often associated with other congenital defects as part of the VACTERL syndromes. Lesions are characterized by their levelÑlow or highÑand that distinction determines whether or not repair can be done in the newborn period without the need for a colostomy. Low lesions are typically indicated by an anocutaneous fistula (meconium noted at the perineum) and are often amenable to perineal anoplasty in the newborn period. Management follows an algorithmic approach in the majority of cases (Fig. 32-6).

4. Hirschsprung disease occurs in 1 out of every 5,000 live births and is characterized by absent ganglion cells in the myenteric (Auerbach) and submucosal (Meissner) plexuses starting in the rectum and extending proximally. This neurogenic abnormality is associated with nerve hypertrophy and muscular spasm of the distal colon and internal anal sphincter resulting in a functional obstruction. There is a predisposition for patients with affected family members as well as those with trisomy 21.

a. Diagnosis. The most common presentation is abdominal distention and failure to pass meconium within 24 hours. Older patients may

present with chronic constipation. Plain radiographs demonstrate absence or paucity of air distal to the obstruction and contrast enema reveals a transition zone between proximally dilated and distally decompressed pathologically abnormal bowel. Rectal biopsy, which can be performed transanally with a suction device or on the seromuscular surface of the bowel via an abdominal approach is required for pathologic confirmation of the diagnosis.

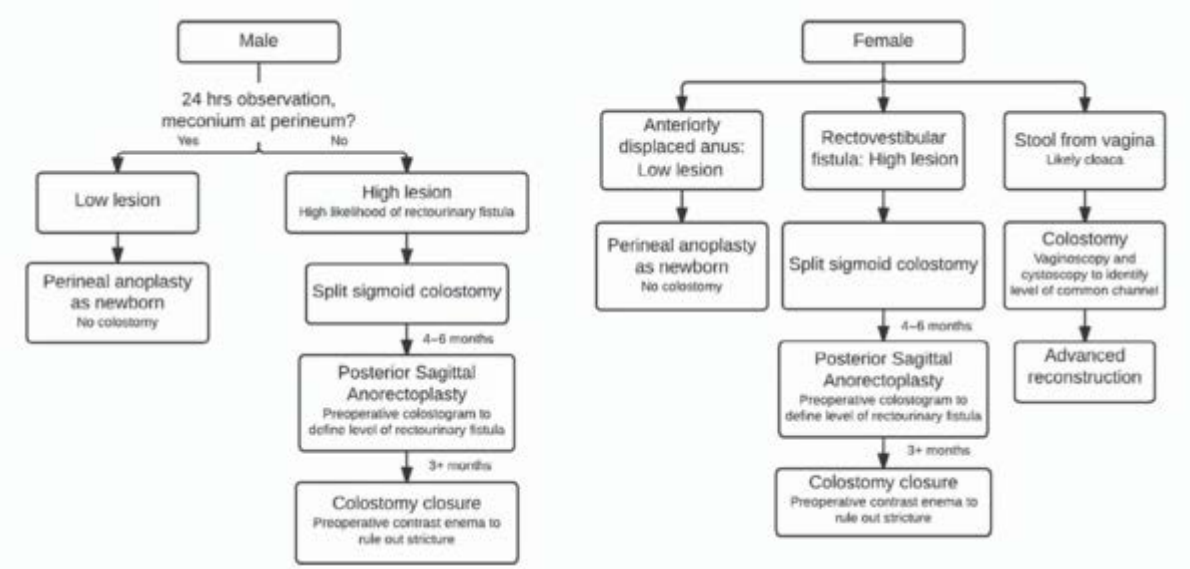

Figure 32-6 Algorithm for management of neonatal anorectal anomalies.

b. Management. Preoperative management includes decompression and saline colonic irrigations to evacuate impacted stool. Operative technique is aimed at identifying the transition zone (ÒlevelingÓ) with serial biopsies, then bringing ganglionated bowel down to the anus while preserving sphincter function. The definitive management of Hirschsprung disease involves variations among three main procedures. In the **Swenson procedure,** the aganglionic bowel is removed down to the level of the internal sphincters and a coloanal anastomosis is performed on the perineum. In the **Duhamel procedure,** the aganglionic rectal stump is left in place and the ganglionated, normal colon is pulled behind and anastomosed to this stump. Finally, the **Soave procedure** involves an endorectal mucosal dissection within the aganglionic distal rectum. The normally ganglionated colon is then pulled through the remnant muscular cuff and a coloanal anastomosis performed. Transanal procedures involving an endorectal pull-through are associated with fewer complications and fewer episodes of enterocolitis without higher rates of incontinence when compared to transabdominal approaches (*J Pediatr Surg.* 2010;45(6):1213-1220).

c. Surgical results are quite favorable with minimal risk of mortality, but morbidity involving constipation and/or incontinence as well as enterocolitis remains.

III. THORACIC PATHOLOGY

A. Congenital Airway Malformations

1. Pulmonary sequestrations are lung malformations with an aberrant blood supply and no bronchial communication.

a. Intralobar sequestrations are contained within lung parenchyma, commonly in the medial and posterior segments of the lower lobe. Two-thirds of these sequestrations are found on the left side, and 85% receive anomalous arterial supply from the infradiaphragmatic aorta via the inferior pulmonary ligament. Indications for surgery include risk of hemorrhage or infection. CT and MRI are useful preoperative imaging studies to elucidate the vascular supply.

b. Extralobar sequestrations are surrounded by a separate pleural layer and are a predominantly male disease (3:1). In contrast to intralobar, extralobar sequestrations are associated with other anomalies in 40% of cases, including diaphragmatic hernia, chest wall deformities, and congenital heart disease. Lesions present less of an infection risk, allowing for a period of observation if the patient is asymptomatic.

2. Congenital pulmonary airway malformations (CPAM), formerly referred to as congenital cystic adenomatoid malformations (CCAM), describe multicystic lesions of bronchial tissue with relatively little alveoli. These typically do not have normal bronchial communication. CPAMs are classified by size, with Type I considered macrocystic (>2 cm), Type II describing epithelial-lined cysts <1 cm (frequently associated with other anomalies including CDH or cardiac malformations), and Type III being microcystic and having a poor prognosis. Pulmonary resection in the newborn period should be performed due the potential for increase in size, infection, or malignancy.

3. Bronchogenic cysts are lined by ciliated cuboidal or columnar epithelium and mucus glands. Two-thirds of cysts are within lung parenchyma and the remainder are within the mediastinum. These should be resected due to symptoms or concern for malignant potential.

4. Congenital lobar emphysema (CLE), which occurs most commonly in the left upper lobe, is the result of overdistention of one or more lobes within a histologically normal lung due to abnormal cartilaginous support of the feeding bronchus. Bronchial collapse creates a one-way valve promoting air trapping. Although many patients are asymptomatic, acute cases of respiratory distress with radiographic imaging mimicking tension pneumothorax may actually be the result of CLE. Placement of a chest tube in these instances would be detrimental and, instead, emergent lobar resection may be needed.

B. Chest Wall Deformities. The two major types of chest wall deformities are **pectus excavatum** and **pectus carinatum.** Pectus excavatum, also referred to as sunken chest, has an incidence roughly five times higher than carinatum and is found in a 3:1 male:female ratio. The etiology of these conditions is believed to be related to abnormal and asymmetric costal

cartilage development.

1. Preoperative evaluation must include examination for scoliosis, which is present in 15% of cases, as well as any cardiorespiratory abnormalities. Chest radiograph is considered a standard component of the preoperative assessment. Additional workup may require an echocardiogram and an ophthalmologic examination if concern for cardiac abnormalities or Marfan syndrome exists. Pulmonary function tests should be obtained if there is concern for the condition causing pulmonary impairment. CT scan allows evaluation of the Haller Index, the transverse chest diameter divided by the anterior-posterior diameter, to document the severity of the defect, but it has little bearing on operative consideration.

2. Indications for surgery are largely cosmetic, as the condition becomes exaggerated both physically and psychosocially in the pubescent years. Correction is generally performed after 8 years of age in order to prevent restrictive chest wall deformities. Two standard techniques, the **Ravitch and Nuss procedures,** offer effective approaches, with both requiring subsequent surgery to remove hardware. Carinatum defects can be similarly repaired with cartilage resection and fixation or more frequently with external chest compression (*J Pediatr Surg.* 2006;41:40-45).

IV. ABDOMINAL PATHOLOGY

A. Acquired Alimentary Obstruction

1. Hypertrophic pyloric stenosis (HPS) is the most common surgical cause of nonbilious vomiting, occurring in 1 of every 400 births, commonly between 2 and 8 weeks of age and with a male to female ratio of 4:1. Infants of Northern European descent are most commonly affected and there appears to be an increased incidence for children of parents with HPS. Pyloric stenosis has no known causative factor and is characterized by hypertrophy of the circular muscle of the pylorus resulting in obstruction of the gastric outlet leading to nonbilious projectile emesis.

Diagnosis is the result of suspicion based on a history of an appropriately aged infant presenting with projectile nonbilious emesis associated with feeding. Persistent fussiness or presumed hunger are all characteristic. Occasionally, the emesis may be coffee-ground. Parents may initially present to a pediatrician for evaluation of their child and can be given more common diagnoses such as reflux or intolerance of feeds requiring a change in formula. Patients must be evaluated for dehydration by sufficient urine production and electrolyte abnormalities. Hypokalemic, hypochloremic metabolic alkalosis is the most common electrolyte abnormality due to a loss of hydrochloric acid from gastric secretions and potassium from the kidney in attempt to compensate for hypovolemia. Physical examination may reveal the Òolive sign,Ó the palpable pylorus to the right of and superior to the umbilicus.

a. Abdominal ultrasound is used for **diagnosis,** with criteria including a pyloric channel length greater than 14 mm and single-wall muscular thickness of 4 mm or greater. These criteria have 99.5% sensitivity and 100% specificity for identifying HPS (*J Pediatr Surg.* 2007;16(1):27-33).

b. Management should initially include evaluation and treatment of dehydration and electrolyte abnormalities. Patients are made NPO to prevent further episodes of emesis and are administered intravenous fluids titrated to urine output greater than 1 mL/kg/hour. Addition of potassium into fluids should be withheld until urine output has been restored. Surgical timing is largely decided by the severity of electrolyte abnormalities, particularly the HCO_3, which, if significantly elevated, results in a compensatory respiratory acidosis that may result in apnea, a devastating consequence particularly exaggerated by postanesthetic respiratory depression.

c. Pyloromyotomy, separation of the external muscular layers down to the level of mucosa, is performed either through right upper quadrant or periumbilical incision, or with laparoscopy.

d. Postoperative feeding protocols typically start with an electrolyte solution with gradual advancement over the first 24 hours postoperatively to formula or breast milk volumes that are appropriate for age. Vomiting is a common postoperative issue, but, rarely, may be an indicator of incomplete myotomy. Additional complications include

perforation of the mucosa, which is treated by mucosal repair and nasogastric drainage.

2. Intussusception is another common acquired cause of intestinal obstruction, resulting from invagination or telescoping of the proximal intestine into the distal bowel. This is most common in infants 3 months to 3 years of age and most frequently originates at the ileocecal junction. Although a lead-point (pathologic or anatomic source) is most commonly not present, definitive identification of a contributing structure may occur in up to 12% of patients, most frequently either a Meckel diverticulum (remnant of the omphalomesenteric duct), intestinal polyp, or tumor. Affected infants present with periods of intense crying during which they retract their legs up in pain; these ÒattacksÓ subside within a few minutes. Recent gastrointestinal or upper respiratory illness may also be part of the patient's history. Obstruction may result in ischemia, which produces stool with a mix of blood and mucous (i.e., the pathognomonic Òcurrant jellyÓ stools).

a. Diagnosis. Plain abdominal radiographs may be sufficient for diagnosis in as many as 50% of cases, revealing abnormal gas patterns concerning for a mass with subsequent paucity of gas in the right lower abdomen. Ultrasound may identify a ÒtargetÓ lesion representing a transverse view of the intussuscepted layers of bowel.

b. Nonoperative management of active intussusception is performed by contrast or air enema. This is successful in 80% of cases, with recurrence rates of approximately 10% within the first 24 hours. Management of recurrence is usually again attempted nonoperatively, but failure or a second recurrence are indications for surgery, as are peritonitis or concern for bowel ischemia at any stage of presentation.

c. Operative management can be approached via either open or laparoscopic techniques. With open surgery, reduction is achieved with retrograde (distal to proximal) squeezing or milking of the intussusceptum until reduced. During laparoscopy, the bowel is pulled apart. The affected

bowel should be examined closely for signs of ischemia. Lymphoid tissue is often noted to be hypertrophic, but it is unclear if this is causative or reactive. If manual reduction is unsuccessful, the involved bowel is resected. Incidental appendectomy may also be performed.

B. Hepatopancreatobiliary. Although jaundice may be a normal physiologic condition of the newborn, it should only be transient. Any infant with direct, conjugated hyperbilirubinemia (greater than 2 mg/dL) beyond 2 weeks of age should undergo further workup. Unconjugated hyperbilirubinemia is often caused by nonsurgical conditions, including physiologic jaundice of the newborn, hemolytic conditions, and breast-milk jaundice. Conjugated hyperbilirubinemia may be caused by hepatitis or biliary obstruction. There are two common obstructive surgical conditions.

1. Choledochal cysts are a spectrum of abnormalities characterized by cystic dilatation of the biliary system of uncertain etiology. Fifty percent

of affected children present within the first 10 years of life. Jaundice in the setting of abdominal pain and a right upper quadrant mass are highly suspicious. Pancreatitis or cholangitis may also occur. Diagnosis is frequently made using ultrasound, which can demonstrate biliary dilatation in one of five types. Surgical repair is undertaken for either resolution of symptoms or due to the potential for malignant degeneration, and the procedure is specific to the type of cyst (Table 32-3).

2. Biliary atresia is the result of progressive obliteration of the extrahepatic bile ducts. There is no causative factor. The incidence is estimated to be 1 in 15,000 births. Patients typically present with jaundice, acholic stools, dark urine, and/or hepatomegaly.

a. Diagnosis is frequently made with ultrasound, which demonstrates a shrunken or absent gallbladder and incomplete or nonvisualized extrahepatic bile ducts. Percutaneous liver biopsy is then performed to confirm the diagnosis (bile duct plugs, biliary epithelial proliferation). Technetium-99m hepatobiliary iminodiacetic acid (HIDA) scan aids in differentiating liver parenchymal disease and biliary obstructive disease. In biliary atresia, the liver readily takes up the tracer molecule, but no excretion into the extrahepatic biliary system or duodenum is seen. Cholangiography may also be performed.

b. Kasai hepatoportoenterostomy is the only surgical management for biliary atresia. The distal bile duct and gallbladder remnant are excised and a Roux-en-Y limb of jejunum is anastomosed to the divided portal plate (the scarred remnant of the biliary tract).

c. Outcomes are best for patients if surgical correction takes place within the first 60 days of life with 30% of these patients

requiring no further surgical intervention (*J Pediatr Gastroenterol Nutr.* 2006;42:93-99). The remainder of patients inevitably progress to fibrosis, portal hypertension, and cholestasis and will require liver transplantation. Older patients or those with significant fibrosis at initial presentation may require immediate transplantation.

TABLE 32-3 Choledochal Cyst Classification

Type	Description	Surgical Management
I	Fusiform dilation of the common bile duct	Resection; hepaticojejunostomy if unable to perform primary bile duct anastomosis
II	Isolated cystic diverticulum of the common bile duct	
III	Intraduodenal or intrapancreatic dilatation of common bile duct (ÒcholedochoceleÓ)	Resection often requiring duodenotomy
IV	Intra- and extrahepatic cystic disease	Resection may involve partial hepatectomy, hepaticojejunostomy
V	Intrahepatic cystic disease	Often requires liver transplantation

V. HEAD AND NECK MASSES

A. Branchial Cleft Cyst. Head and neck structures are embryologically derived from six pairs of branchial arches and the corresponding external clefts and internal pouches. Congenital cysts, sinuses, or fistulae result from failure of appropriate migration or regression of these structures. Branchial remnants are present at the time of birth but may not become clinically evident until later in life. In children, fistulas are more common than external sinuses, which are more frequent than cysts. Patients present with a spectrum of symptoms including visible or palpable lesions, mucoid drainage, or development of cystic masses that may become infected.

1. First branchial remnants are typically located in the front or back of the ear, or in the upper neck in the region of the mandible and may involve the parotid gland, facial nerve, or external auditory canal.

2. Second branchial clefts remnants are the most common. These are usually located along the anterior border of the sternocleidomastoid muscle, invade the platysma, ascend along the

carotid sheath to the level of the hyoid bone, and extend medially between the carotid artery bifurcation. The fistula then courses behind the posterior belly of the digastric and stylohyoid muscles to end in the tonsillar fossa.

3. Third branchial cleft remnants usually do not have associated sinuses or fistulae. These most often contain cartilage, are located in the suprasternal notch or clavicular region, and present as a firm mass or as a subcutaneous abscess.

B. Thyroglossal duct cysts are common lesions in the midline of the neck and frequently present in preschool-age children. These cysts represent incomplete thyroid gland formation and, as such, are located along the normal course of thyroid migration: From the foramen cecum to the anatomically normal site of the pyramidal lobe of the thyroid gland. Frequently the tongue or hyoid bone are involved.

1. Indications for surgery include increasing size, infection or risk for infection, and risk for carcinoma (1% to 2%). Preoperative ultrasound can aid significantly in identifying involved structures including the site of normal thyroid tissue, which should be preserved if possible. Standard surgery, the **Sistrunk procedure,** involves excision of the central portion of the hyoid bone in addition to the cyst in continuity with its tract.

C. Cystic hygroma is a lymphatic malformation resulting from abnormal development of a lymphatic network that fails to drain into the venous system. Seventy-five percent of these lesions involve the lymphatic jugular

sacks, presenting as a posterior neck mass. The majority of cystic hygromas present at birth (50% to 65%), with most becoming apparent by the second year of life.

1. Indications for resection are largely cosmetic, although expansion that threatens compression of the airway; infection; or pain, possibly due to hemorrhage, are also acceptable indications for treatment. Complete surgical excision is preferred. Preoperative magnetic resonance imaging can assist in determining the extent of the lesion and any neurovascular involvement. Postoperative morbidity includes recurrence, lymphatic leak, infection, and neurovascular injury. Additional therapeutic modalities include injection of sclerosing agents such as bleomycin.

D. Cervical lymphadenopathy is a frequent diagnosis for referral to a pediatric surgeon. Infectious etiologies are the overwhelming majority of diagnoses, including cat-scratch fever as a result of Bartonella, which contributes to approximately 3% of lymphadenopathy cases. Lymph nodes that are unilateral, firm, fixed, or greater than 2 cm should prompt additional workup, including a chest radiograph to evaluate for mediastinal lymphadenopathy or masses.

VI. TUMORS AND NEOPLASMS

A. Neuroblastoma is the most common neoplasm of childhood, accounting for 6% to 10% of all childhood cancers with an incidence of 1 in 10,000 cases annually and a median age of diagnosis

of 2 years old. Only 25% of patients present with isolated disease, often found incidentally on examination or imaging, for which surgical therapy is standard of care. The remainder present with metastatic disease and have a poor prognosis. These neoplasms are of neural crest origin and are thus found along the sympathetic nervous system, with 75% in the abdomen or pelvis and half of these within the adrenal medulla.

1. Preoperative evaluation should include routine laboratory work such as CBC and CMP. Blood pressure should be checked to investigate the potential for hypertension, and urine should be tested for catecholamine metabolites. CT and MRI assist in preoperative planning, particularly as neuroblastoma is capable of invading into adjacent vascular structures. They are also useful for metastatic workup, with frequent sites of metastases being bone and lung. Radiolabeled metaiodobenzyl guanidine (MIBG) is a high-yield study to document the presence of metastatic disease. Bone marrow aspirate and biopsy complete the staging evaluation. Staging and survival are determined by burden of disease as well as genetic markers including N-*myc* overexpression and chromosomal deletions, which portend a worse prognosis. Young patients with early disease and favorable pathology have >90% survival, while older patients with unfavorable or metastatic disease can have survival as low as 10%.

2. Surgical management is reserved for anatomically resectable cases of early nonmetastatic disease or can be undertaken after neoadjuvant therapies.

B. Wilms tumor accounts for 6% of all malignancies in children and is the most common renal malignancy in children. Approximately 500 cases are diagnosed annually in the United States, with an average age of diagnosis of 3 or 4 years old. There is no gender predominance, and 5% of cases are bilateral. Although there are heritable forms of Wilms tumor including an association with Beckwith-Wiedemann syndrome, most cases are sporadic and present as incidental abdominal masses. Patients may have hypertension or hematuria.

1. Preoperative evaluation includes ultrasound to confirm renal origin of the mass and to evaluate for any potential intravascular extension. CT or MRI may further assist in differentiating Wilms tumor from neuroblastoma, as will urine catecholamines. Chest imaging via CT scan is needed for complete staging.

2. Surgical intervention requires radial nephroureterectomy and lymph node sampling. Intraoperative spillage of tumor contents must be carefully avoided, as it upstages the patient's disease (Table 32-4). For resectable disease, surgery and chemotherapy together result in a greater than 90% cure rate.

C. Hepatic tumors make up fewer than 5% of all intraabdominal pediatric malignancies but are malignant in up to 70% of cases.

1. Hepatoblastoma typically presents before 3 years of age and is often unifocal. Chemotherapy is somewhat effective and overall survival is dictated primarily as to whether the

tumor is resectable or not.

2. Hepatocellular carcinoma is often multifocal, with an overall survival of 25%. Surgical resection or liver transplantation can be curative.

D. Teratomas are tumors containing tissue from more than one of the three embryonic germ cell layers. A frequent presentation is as a **sacrococcygeal teratoma** in the neonatal period, which has a 4:1 female:male ratio.

Ultrasound and rectal examination should be performed preoperatively to rule out pelvic or pre-sacral extension. The majority of these are benign and are removed along with the sacrum to prevent recurrence.

TABLE 32-4 Wilms Tumor Staging System

Stage	Characteristics
I	Tumor confined to the kidney and completely excised intact
II	Tumor extends through the renal capsule into adjacent tissues (fat, vessels etc.), but all affected tissue is completely excised intact. Stage II also includes cases in which the kidney is biopsied preoperatively or localized spillage occurs during resection
III	Tumor without hematogenous or extraabdominal spread. Includes cases of positive lymph nodes, positive resection margins, or peritoneal implants
IV	Hematogenous metastases
V	Bilateral renal involvement

E. Soft-tissue sarcomas account for 6% of childhood malignancies, with half of these being **rhabdomyosarcomas.** Wide local excision depending on the anatomic location with or without chemotherapy and lymph node sampling should be performed.

VI. PEDIATRIC GENERAL SURGICAL CONDITIONS

A. Appendicitis is one of the most common pediatric surgical conditions. History of abdominal

pain followed by nausea along with physical examination findings of tenderness at McBurney point and obturator, iliopsoas, and Rovsing signs can be helpful clinical cues. Scoring systems incorporating history, physical, and laboratory values such as the Alvarado score (*Ann Emerg Med.* 1986;15(5):557-564) can aid in objectively risk-stratifying patients.

Ultrasound is routinely the first imaging modality of choice in the pediatric population. A positive study should result in surgical management, whereas a negative study or one in which the appendix was nonvisualized should prompt either observation, evaluation of other diagnoses, or consideration for obtaining a CT scan, particularly in patients showing signs of advanced illness.

B. Indirect inguinal hernias affect approximately 1% to 5% of children, with a predominance in males (8:1) and an increased rate in premature infants (7% to 30%). Bilateral hernias are present in 10% to 40% and occur more frequently in premature infants and girls. Due to an increased risk of recurrence and post-anesthesia apnea in premature patients, neonatal inguinal hernias are frequently repaired prior to the patient leaving the hospital or at 50 weeks postconception (*J Pediatr Surg.* 1996;31:1166-1169).

C. Hydroceles are fluid collections within the processus vaginalis that envelop the testicles. They occur in approximately 6% of full-term male newborns.

1. Communicating hydroceles allow the free flow of peritoneal fluid down to the scrotum through a patent processus vaginalis. This must be regarded as a hernia and repaired as such.

2. Noncommunicating hydroceles contain fluid confined to the scrotum due to an obliterated processus vaginalis. This is usually a self-limiting process that resolves in 6 to 12 months.

D. Umbilical hernia is common in children due to a persistence of the umbilical ring. In the vast majority of cases, these close spontaneously by age 4 to 5 but can be repaired if they are exceptionally large (>2 cm, due to low likelihood of spontaneous closure) or if the patient has had incarceration of bowel within the hernia.

E. Trauma

1. Imaging. Limiting radiation exposure and unnecessary testing is important in children. However, the inability to obtain reliable examinations

in this population, particularly in trauma settings, complicates the decision to obtain imaging. Diffuse abdominal pain, a seatbelt sign, or distracting injuries can be indications for computed tomography scan. In addition, elevated serum glutamic-oxaloacetic transaminase (SGOT) or serum glutamic-pyruvic transaminase (SGPT) levels higher than 200 or 100 IU/L, respectively, are accepted thresholds for obtaining a CT scan in the setting of blunt trauma.

TABLE 32-5 Nonoperative Management of Pediatric

Blunt Traumatic Isolated Spleen or Liver Injury

	CT Grade			
	I	II	III	IV
ICU stay (days)	-	-	-	1
Hospital stay (days)	2	3	4	5
Return to age-appropriate activity (wks)	3	4	5	6

From Stylianos et al., 2000, Table 3.

2. Treatment of injury. Indications for exploratory laparoscopy or laparotomy are similar to those utilized in adults and include hemodynamic instability with either visible or suspected organ or vascular injury, penetrating abdominal injury, imaging findings consistent with bowel injury, or the presence of pelvic free fluid after blunt trauma without solid organ injury, suggestive of small bowel injury.

a. Conservative or nonoperative management of isolated solid organ injury is covered by the guidelines of the Liver/Spleen Trauma Study Group of the American Pediatric Surgical Association (Table 32-5). Regardless of the injury grade and in the absence of specific indications, follow-up imaging either at the time of discharge or prior to resumption of normal activities is not indicated.

CHAPTER 32: PEDIATRIC SURGERY

Multiple Choice Questions

1. A 10-year-old 35-kg girl presents to your Emergency Room after being in a highway-speed motor vehicle crash. She was a restrained back seat passenger and airbags did deploy. She is tachycardic, hypotensive, and has diffuse abdominal pain with a positive seatbelt sign. Focused Assessment with Sonography in Trauma (FAST) is positive for intraabdominal fluid. Her hypotension has not improved after 2 boluses of isotonic fluid. You next ask for rapid transfusion of blood as you prepare to take her to the operating room. What blood

volume should you initially transfuse?

a. 100 mL

b. 175 mL

c. 250 mL

d. 350 mL

e. 475 mL

View Answer

2. You receive a call from a primary care physician who is working up a 4-year old female for chronic abdominal pain, distention, and jaundice. The physician describes a CT scan finding of a cystic structure arising from the common bile duct that is separate from the gallbladder. You suspect choledochal cyst as a possible diagnosis. This imaging is consistent with what type of choledochal cyst?

a. Type I

b. Type II

c. Type III

d. Type IV

e. Type V

View Answer

3. You are caring for a 14-year-old boy who was presented to the Emergency Room after injuring himself in an ATV accident. He had been complaining of abdominal pain and eventually received a CT scan showing a blunt splenic injury with a subcapsular hematoma occupying <50% of the surface and associated with a 3-cm splenic laceration. He has been hemodynamically stable. You inform the family that the boy should be admitted. His family asks how long he will need to be admitted and how long he will have to engage in only light activity after discharge. You tell them:

a. 24 hour overnight hospitalization, return to activity as tolerated

b. 2 day hospitalization, 3 weeks light activity

c. 3 day hospitalization, 4 weeks light activity

d. 4 day hospitalization, 5 weeks light activity

e. 5 day hospitalization including 24 hours in the ICU, 6 weeks light activity

View Answer

4. You are asked to see a patient in the Neonatal Intensive Care Unit. The infant was born just hours ago and has progressed into significant respiratory distress. The intensivists have intubated the patient but are unsuccessful in passing an orogastric tube due to resistance. Your attempts also fail. Plain chest x-ray shows coiling of the gastric tube in the upper chest. What additional workup would you request to make a diagnosis?

- **a.** CT chest
- **b.** Spinal ultrasound
- **c.** Abdominal x-ray
- **d.** CT abdomen and pelvis
- **e.** Upper GI study with contrast

View Answer

5. The plain abdominal x-ray shows air in the bowel. You determine that there is likely both an esophageal atresia and a distal tracheoesophageal fistula. Bronchoscopy and endoscopy are performed and fail to reveal a fistula proximally. This is classified as what type of tracheoesophageal fistula?

- **a.** A
- **b.** B
- **c.** C
- **d.** D
- **e.** E

View Answer

6. Which one of these descriptors is associated with gastroschisis versus other abdominal wall defects?

- **a.** Covered by peritoneal sac
- **b.** Result of intrauterine vascular insult
- **c.** High (~50%) incidence of associated anomalies
- **d.** Typically large defects

View Answer

7. You are being asked to see a newborn with respiratory distress after 8 hours of life. The infant is tachypneic with significant degree of

retractions. Oxygen saturations are below 80%. Chest x-ray shows herniated abdominal contents in the left chest. What treatment option is the next best choice?

a. Emergent thoracotomy

b. ECMO cannulation

c. Intubation

d. Bedside echocardiogram

View Answer

8. Hirschsprung disease results from a failure of neural crest cell development in the Meissner and Auerbach plexi. In what layers of the bowel wall are these plexi contained?

Choice	Meissner	Auerbach
A	Mucosal	Mucosal
B	Submucosal	Mucosal
C	Muscular	Submucosal
D	Submucosal	Muscular
E	Serosal	Muscular

View Answer

9. Twenty-four hours after a pyloromyotomy and tolerating a normal volume of formula every 3 to 4 hrs, your patient develops feeding intolerance, mild tachypnea, and abdominal distention. The infant is otherwise stable. What is your next step?

a. Continue feeding

b. Change formula

c. Make NPO, start nasogastric drainage

d. Laparotomy

View Answer

10. A young patient presents to you with <12 hours of severe abdominal pain, diffuse tenderness on examination, and bilious emesis. You are concerned about malrotation and, while awaiting imaging, plan

your operative approach. Which step is not a common component of Ladd procedure?

a. Reduction of volvulus by counterclockwise rotation

b. Division of Ladd bands

c. Appendectomy

d. Pexying the cecum to the peritoneal sidewall

View Answer

33
Cardiac Surgery

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This chapter focuses on the preoperative evaluation, surgical indications, procedures, and postoperative management of adult cardiac surgery patients.

I. ANATOMY

A. Coronary Arteries. The coronary arteries are visible and accessible on the epicardial surface of the heart or may be found intramyocardially. The left and right coronary arteries arise from within the sinuses of Valsalva just above the right and left coronary cusps of the aortic valve. The **left main coronary artery** travels posterior toward the pulmonary artery, then divides into its main branches, the **left anterior descending artery (LAD)** and the **left circumflex artery (LCx).** In 10% to 15% of patients, the LCx gives off the **posterior descending artery (PDA),** termed a **left dominant coronary circulation.** The **right coronary artery (RCA)** begins on the anterior surface of the aorta and descends in the anterior AV groove, where, in **right dominant coronary circulation** (80% to 85% of cases), it gives off the PDA (Fig. 33-1).

Clinical correlation: Dominance is important in selecting targets to graft during coronary artery bypass grafting (CABG) surgery and in predicting location (anterior or posterior) of culprit vessel in the diagnosis of post myocardial infarction (MI) ventricular septal defect (VSD).

Clinical correlation: Because of its anterior location, intracardiac air preferentially enters the orifice of the RCA when weaning from cardiopulmonary bypass (CPB), resulting in right ventricular dysfunction.

B. Coronary Veins. The **coronary sinus** is located in the posterior AV groove and receives venous drainage mainly from the left ventricular system. Its main tributaries are the great, middle, and small cardiac veins. The **anterior cardiac veins** drain the right coronary system, and ultimately into the right atrium.

Clinical correlation: The coronary sinus is an important structure in the myocardial protection strategy for most cardiac surgery. Infusion of hyperkalemic solution into this venous structure (retrograde cardioplegia) provides protection to all territories (except the right heart as the venous drainage is into the right atrium) in a more uniform fashion versus the antegrade direction in patients with diffuse coronary

disease.

C. Valves

1. AV valves. These valves are continuous with the **annuli fibrosi** at the base of the heart and secured by **chordae tendineae,** which attach the free leaflets to the intraventricular papillary muscles. The **tricuspid valve** separates the right chambers and consists of a large

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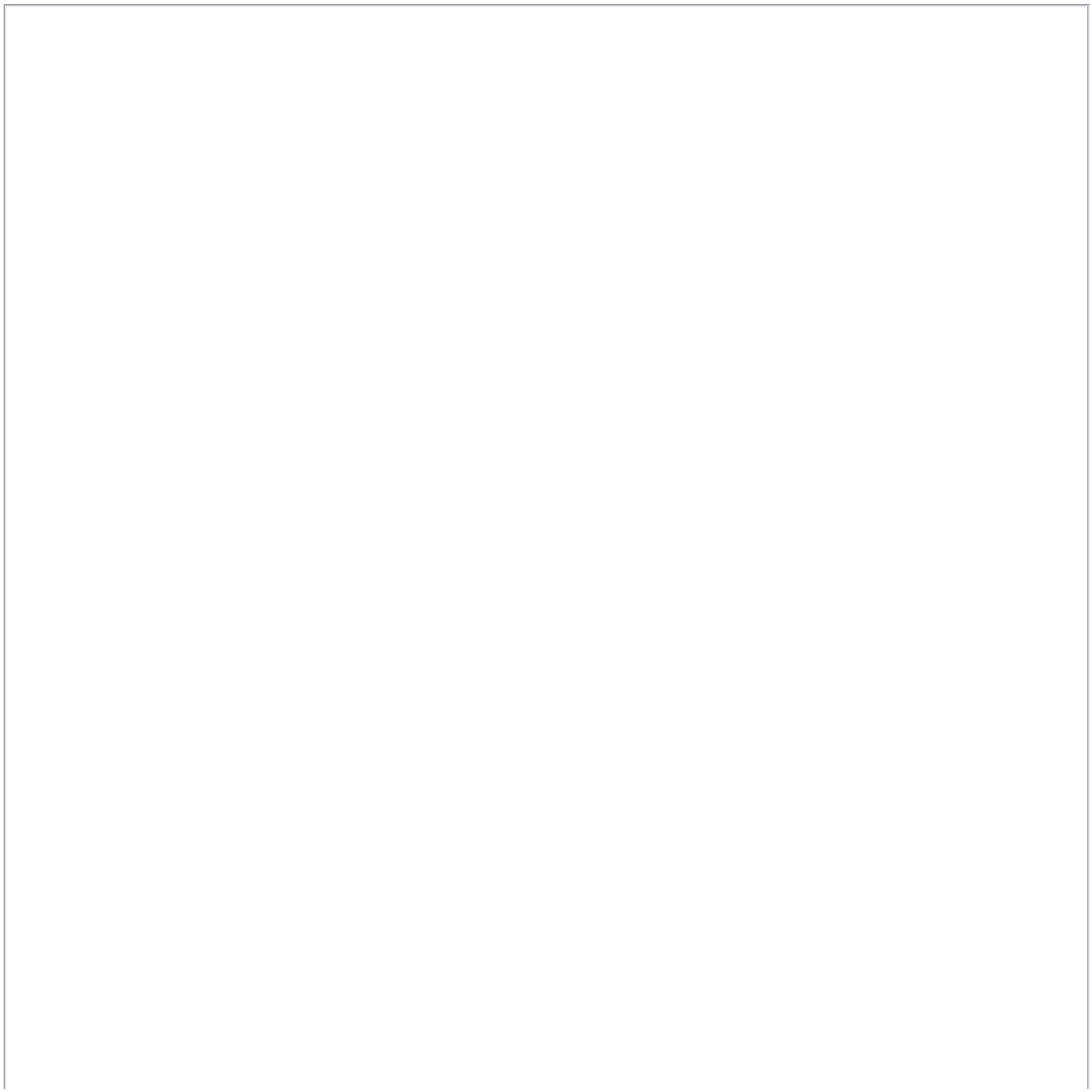
anterior leaflet, a posterior leaflet, and a septal leaflet attached to the interventricular septum. The **mitral (bicuspid) valve** separates the left chambers and consists of a large anterior (aortic) leaflet and a posterior (mural) leaflet.



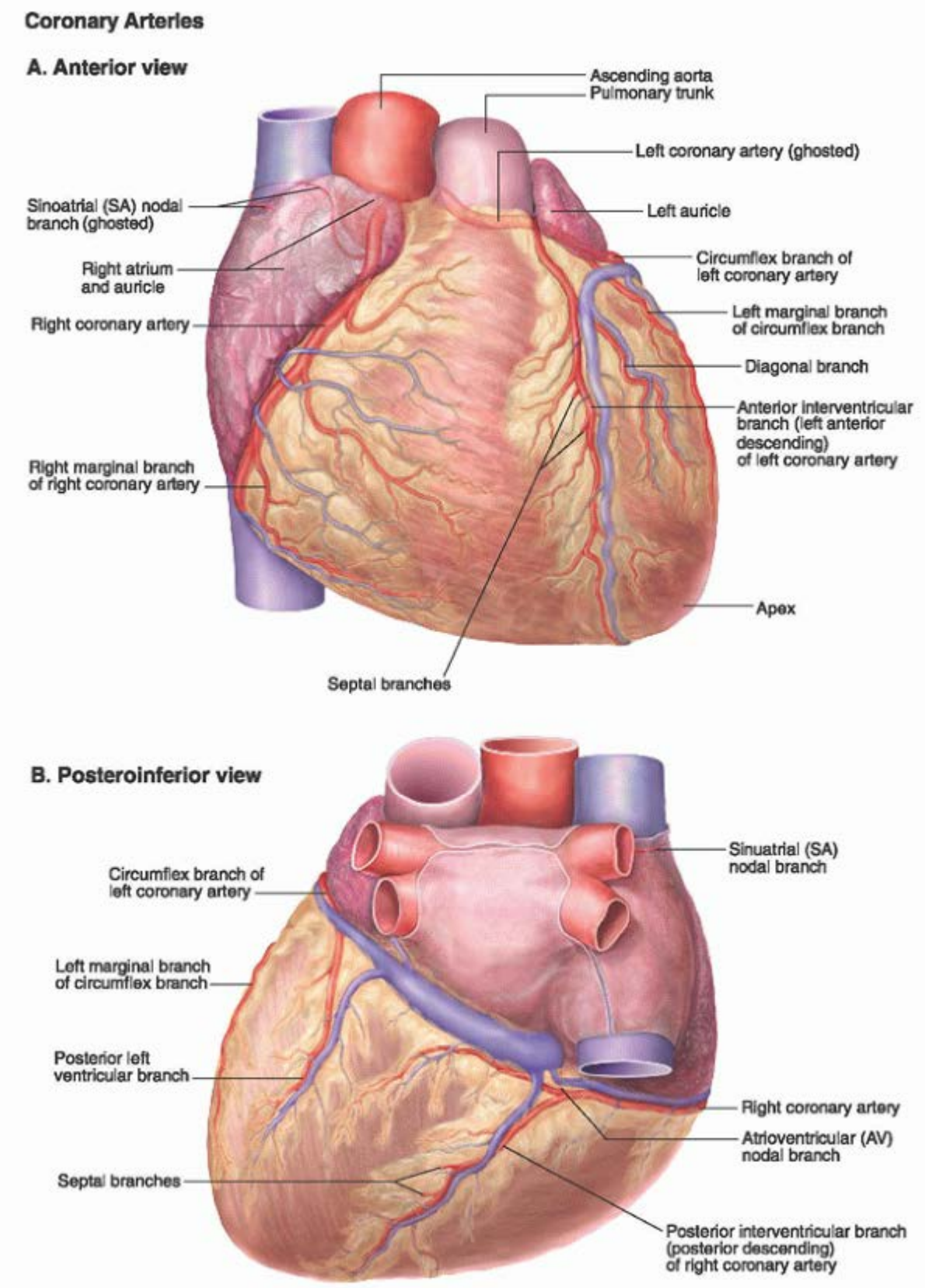


Figure 33-1 Coronary artery anatomy. (Reprinted with permission from Tank PW, Gest TR. Lippincott Williams & Wilkins Atlas of Anatomy, 1st ed. 2008.)

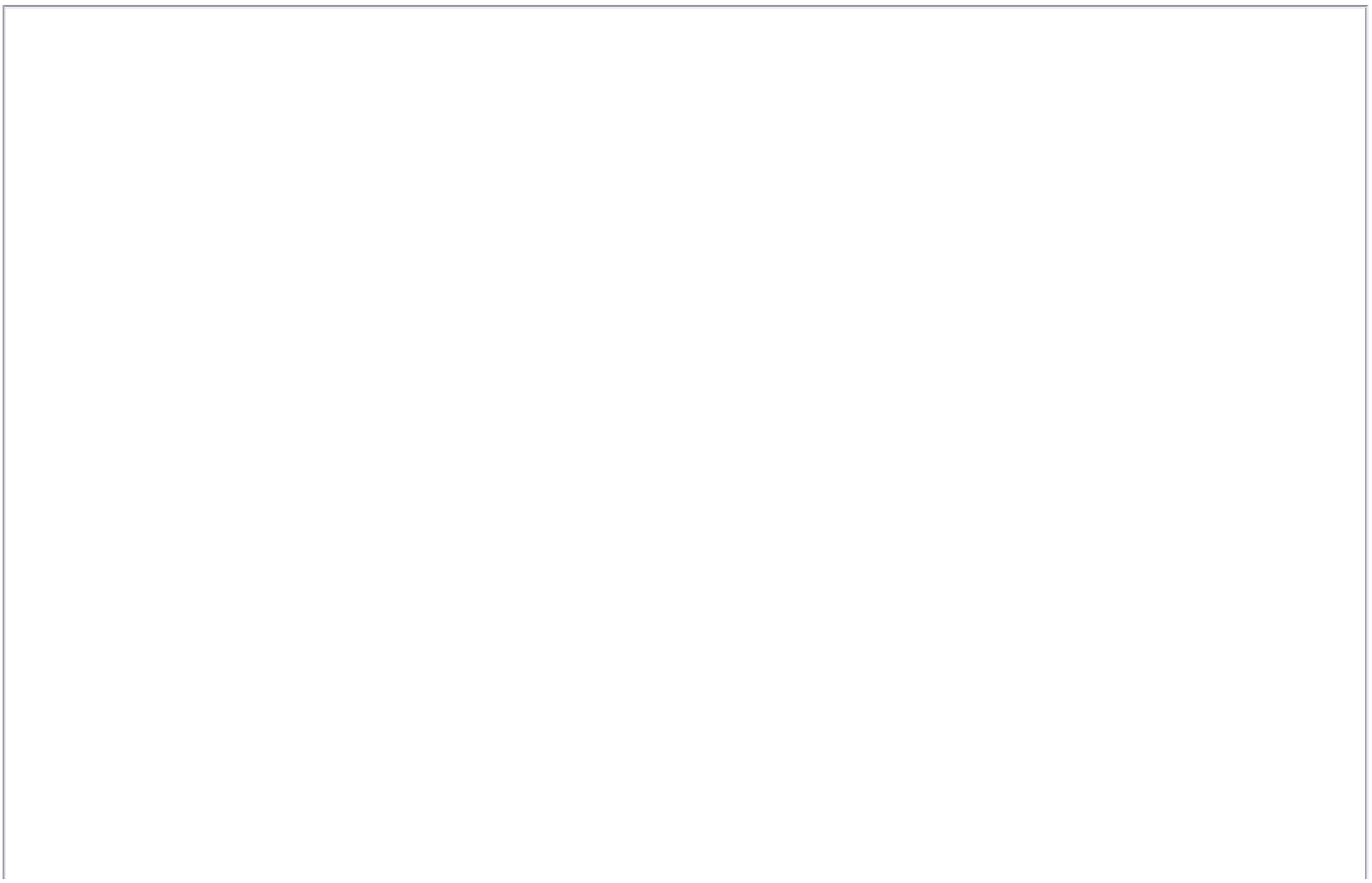
2. Semilunar valves. The **pulmonary** and **aortic** valves are essentially trileaflet and the coronary arteries arise just distal to the aortic valve. Just distal to the valves are gentle dilations of the ascending aorta, known as **sinuses of Valsalva.**

D. Electrophysiology. Electrical activation of the heart begins in the **sinoatrial (SA) node** (located at the junction of the anteromedial aspect of the superior vena cava and the right atrium), travels to the **AV node** (located in the triangle of Koch; Fig. 33-2) defined by the coronary sinus, tendon of Todaro, and the septal leaflet of the tricuspid valve. The AV node protects the ventricle from atrial tachyarrhythmias. From the AV node, conduction travels to the right and left **bundles of His.**

Clinical correlation: The AV node is of particular importance as it may be injured during cardiac surgery. Specifically, during tricuspid valve repair or replacement, during aortic valve replacementÑin the commissure between the noncoronary and right coronary cusps, and in mitral valve repair or replacementÑin the annulus near the A1 portion of the anterior leaflet just under the aortic valve.

E. Myocardium. The working chambers of the heart (ventricles) provide cardiac output to the lungs and body. Cardiac output is the volume of blood

ejected by the heart per minute and is mainly influenced by heart rate and stroke volume (volume of blood ejected from one ventricle per heart beat). Cardiac output may be manipulated by altering heart rate, preload, afterload, and contractility (Fig. 33-3).



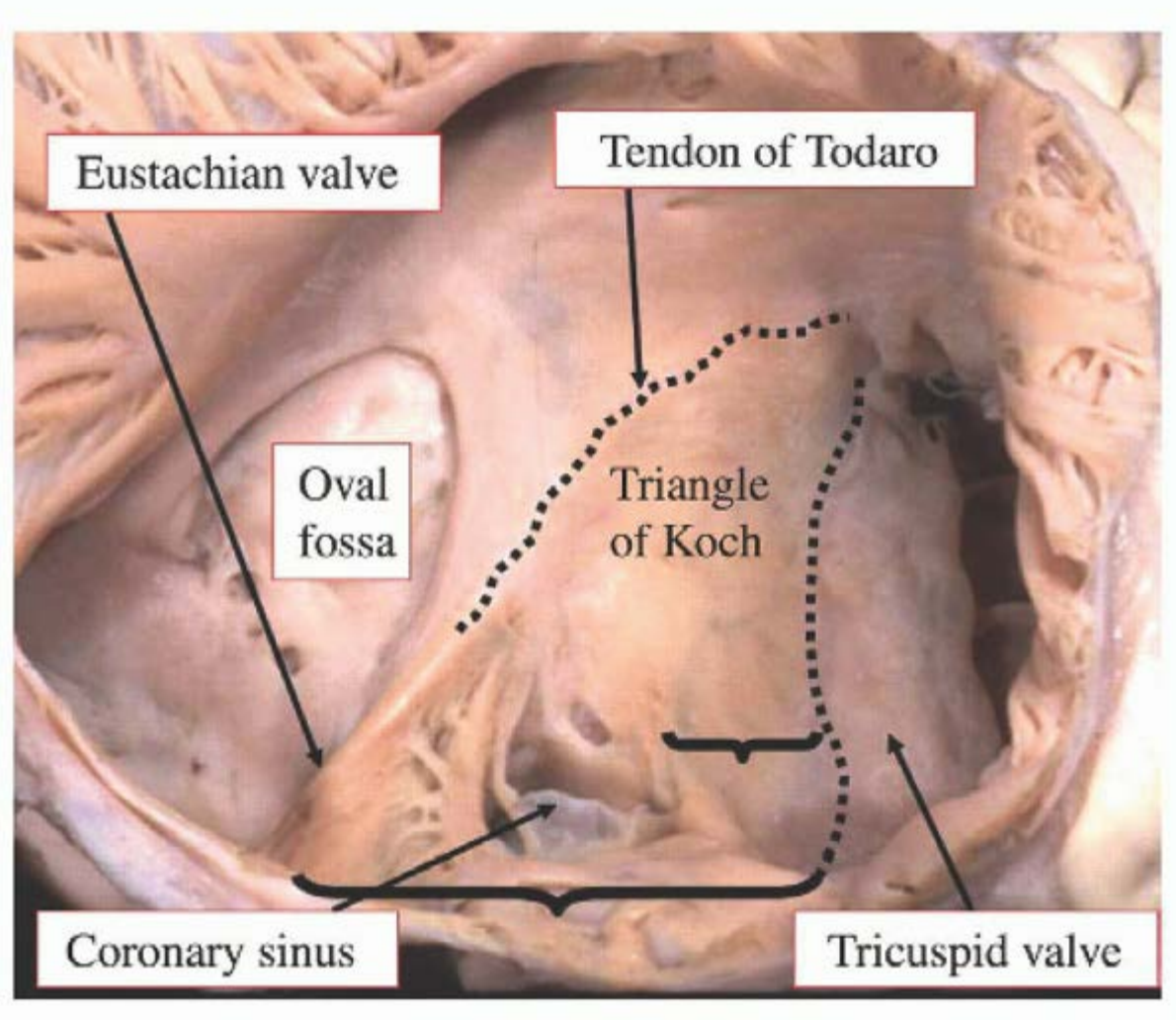


Figure 33-2 Triangle of Koch viewed from the inside the right atrium. Patient head at top and feet at bottom of page. (Reprinted with permission from Anderson RH, Cook AC. The structure and components of the atrial chambers. *Europace.* 2007;9 Suppl 6:vi3-vi9.)

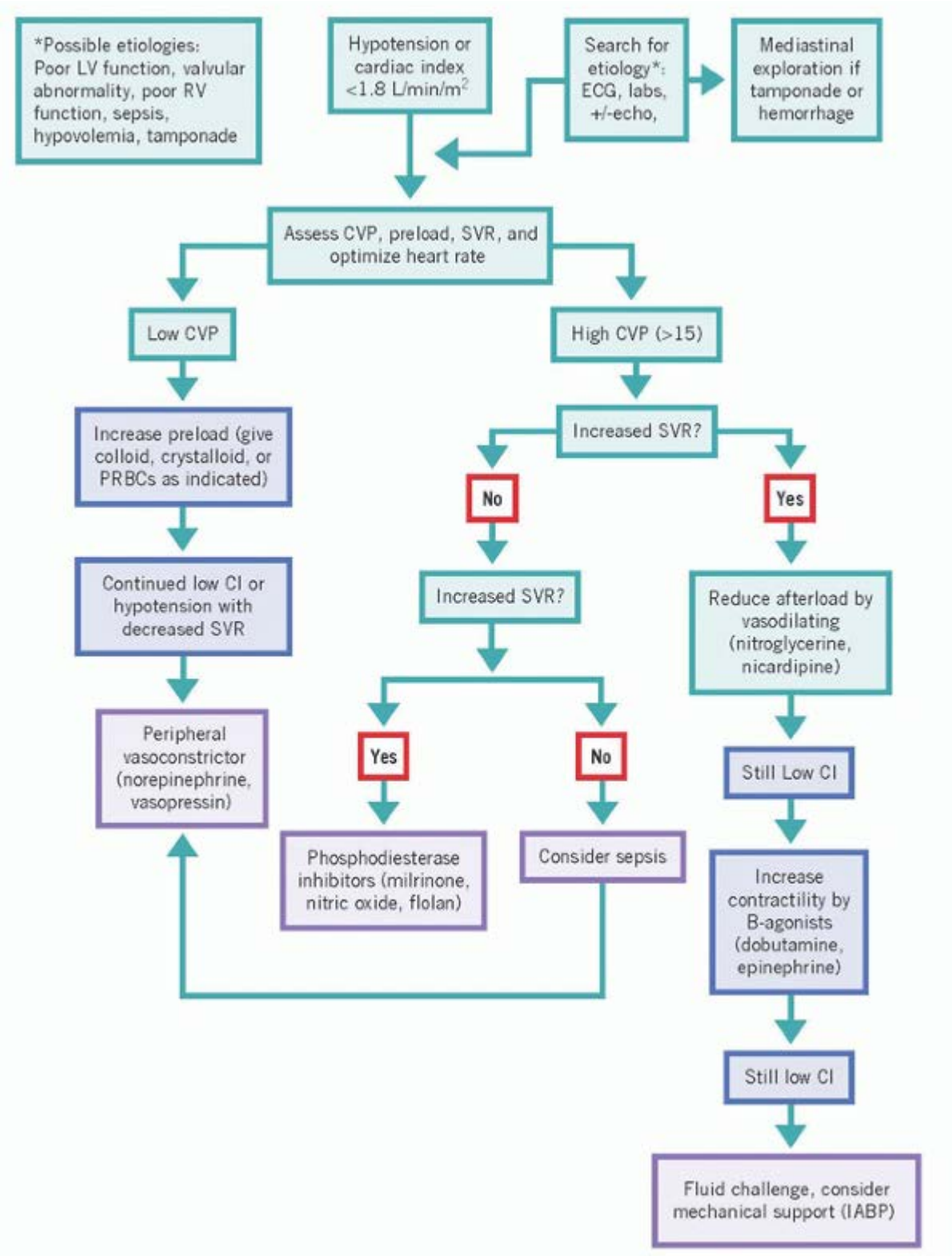


Figure 33-3 Postoperative evaluation and treatment of low cardiac output state. (Adapted from Cohn LH, Dody DB, McElvein RB. Decision making in Cardiothoracic. *Surgery.* 1993;2:83.)

II. PREOPERATIVE RISK ASSESSMENT.

Many complications encountered during cardiac surgery may be avoided by careful preoperative assessment and planning. This assessment is also extremely important in communicating operative risk and possible alternatives to the patient.

History and Physical Examination

The best history and physical examination is usually obtained directly from the patient and Table 33-1 highlights key historical and physical findings that may prompt additional testing or evaluation and alert one to the possibility of future complications.

Several preoperative assessment tools are available to determine a numerical and objective evaluation of operative risk. These tools include the *Euro*SCORE and the Society of Thoracic Surgeons Risk Model Score (STS score at: http://riskcalc. sts.org/stswebriskcalc/#/; *Euro*SCORE at: http://www.euroscore.org/calculators. htm).

A careful, *personal* review of available data is imperative prior to planning any operative procedure. Anticipation of potential complications during this review may reduce their magnitude and severity.

PA and Lateral CXR

A careful review of the CXR will alert the surgeon to aortic calcification, significant lung disease, and mediastinal pathology. Significant aortic calcification (Fig. 33-4) increases the risk of embolization and indicates a potential need for alternate cannulation sites or even inoperability. Aortic calcification can be further evaluated utilizing CT imaging (Fig. 33-5) and by careful review of the left ventriculogram during left heart catheterization (Fig. 33-6).

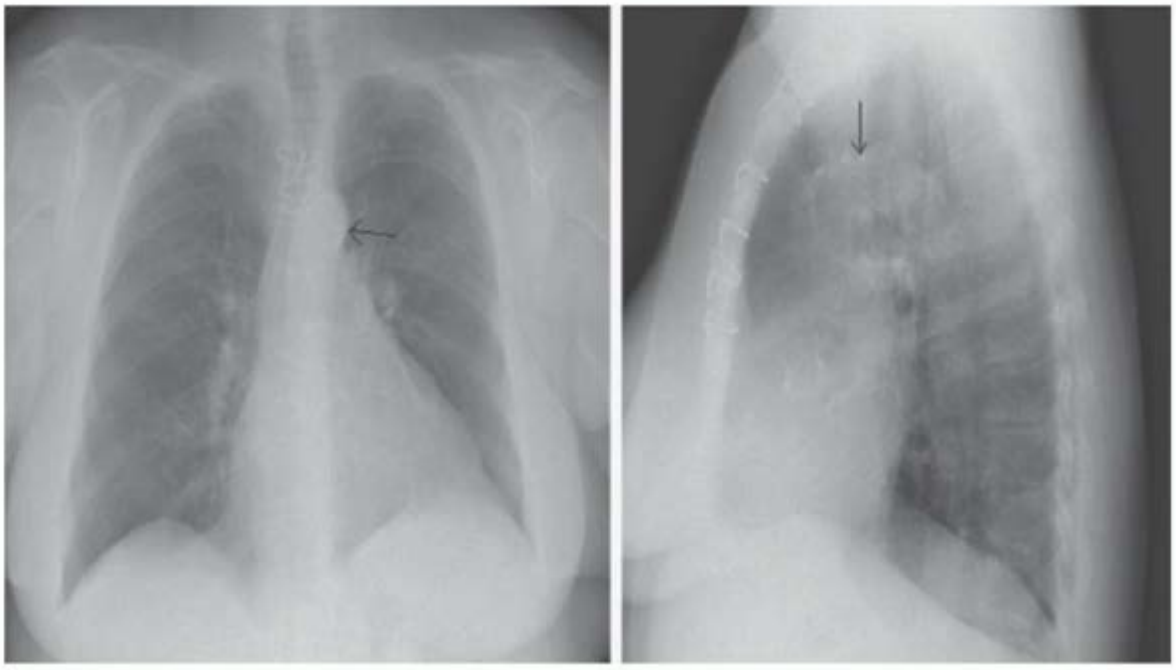


Figure 33-4 AP (**left**) and lateral (**right**) chest x-ray demonstrating a calcified aorta (*black arrows*).

TABLE 33-1 Preoperative Risk Assessment

History	Potential Complication and Evaluation
Diabetes Mellitus Immunosuppression Abnormal BMI	Increased risk of infection and poor wound healing
Poorly controlled DM Increased age Gender (female)	Increased mortality
Hypertension	Increased risk of CVA

Recent cath	Increased risk of renal failure
Home oxygen use Lung disease Tobacco use Previous tracheostomy	Increased risk of prolonged ventilation; consider PFTs
Liver disease Recent antiplatelet agent use (Plavix, IIb-IIIa inhibitors, thrombin inhibitors) Thrombocytopenia	Increased risk of bleeding, consider hematology or hepatology consults
Previous sternotomy Chest wall XRT Pericarditis	Anticipate difficult dissection
Poor social support Neurologic dysfunction	Difficult rehabilitation
Exercise tolerance	Good indicator of outcome
Pulsatile abdominal mass	Abdominal ultrasound
Carotid bruit History of carotid endarterectomy TIA symptoms History of stroke	Carotid Doppler
Poor dental hygiene	Panorex prior to valve procedure

BMI, body mass index; DM, diabetes mellitus; XRT, radiation therapy; CVA, cerebrovascular accident; PFT, pulmonary function test; TIA, transient ischemic attack. Adapted from Lawton JS and Gay WA. On Pump Coronary Artery Bypass Grafting in Complications in Cardiothoracic Surgery Avoidance and Treatment, 2010 Blackwell Publishing, 334-335.

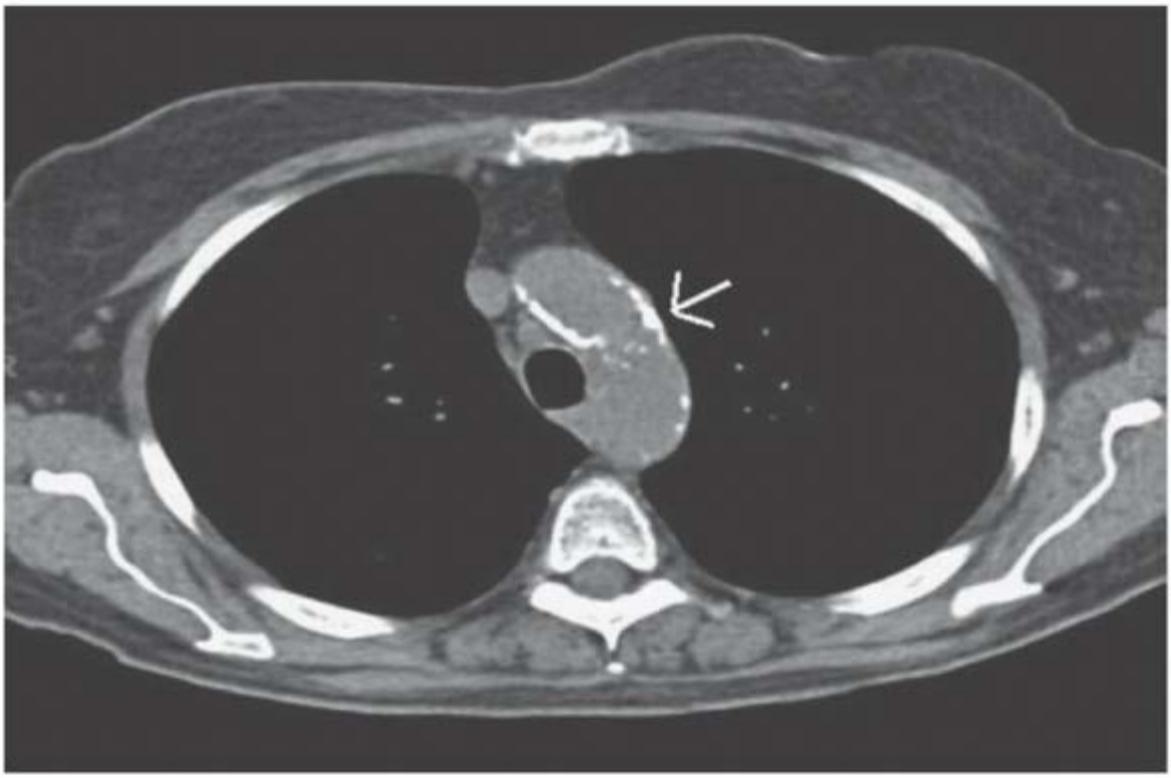


Figure 33-5 CT scan demonstrating a calcified aorta (*white arrow*).

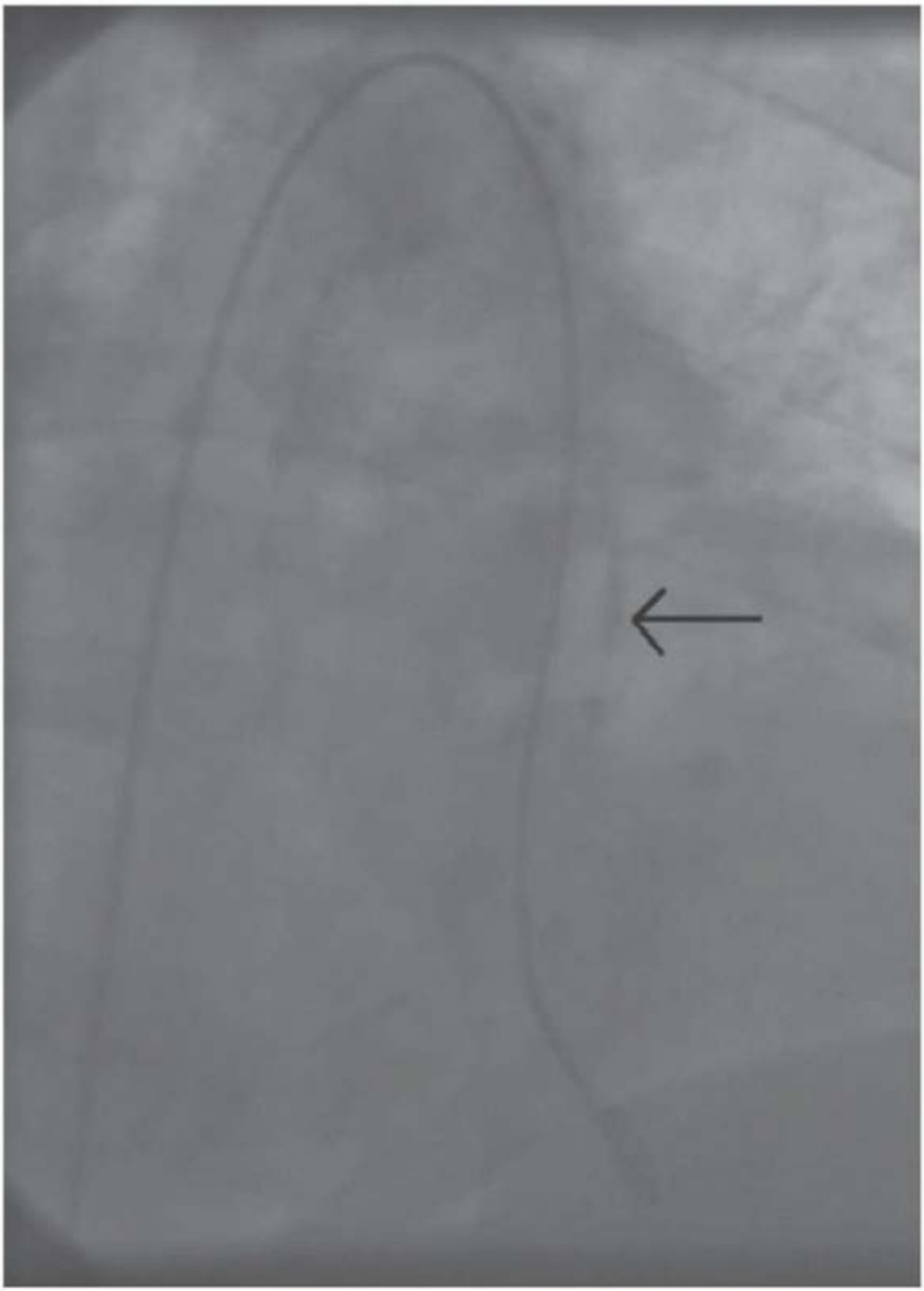


Figure 33-6 Calcified aorta demonstrated on fluoroscopy after catheter placed in the left ventricle (*black arrow*).

Cardiac Catheterization

Nearly all patients undergoing cardiac surgery undergo cardiac catheterization to determine the presence of coronary disease and to delineate coronary anatomy for bypass planning. Prior to the injection of dye, calcified valve annuli and coronary arteries may be visualized. Significantly stenotic coronary arteries must be of sufficient size and have a patent, anatomically accessible location in order to accept a bypass graft. Knowledge of the size of the injection catheter utilized can indicate the size of the vessel opacified, particularly if magnification has been utilized to produce the images. Proposed target sites for bypass grafts must be free of significant calcium and stent material. Intramyocardial vessels may also be anticipated by viewing the injection through the cine cycle. The left ventricular injection is particularly valuable as it provides an estimation of wall motion and ejection fraction (EF) as well as the size of the ascending aorta and a visual estimation of mitral valve competence. In addition, injections of the descending aorta, renal arteries, or iliac vessels provide information regarding significant peripheral vascular or renal artery occlusive disease.

Transthoracic or Transesophageal Echocardiogram

Transthoracic or transesophageal echocardiogram (TEE) will provide information in the assessment of EF and valvular pathology. TEE is vital to the planning of mitral valve repair versus replacement.

Clinical correlation: Preop TEE is considered mandatory by many surgeons prior to mitral valve surgery to determine the extent of regurgitation. The extent of regurgitation may be underestimated intraoperatively during assessment under general anesthesia.

Viability Study

If a significantly reduced EF is noted, then an assessment of viability should be considered. Pharmacologic as well as radiologic examinations may be obtained to determine *myocardial viability.* Large areas of nonviable or infarcted myocardium should alert the surgeon to heavily weigh the risk of surgery.

A *computerized tomography scan* of the chest or abdomen may provide valuable information regarding calcification of the aorta, the presence or lack of a tissue plane between the heart and the sternum in the case of redo surgery, significant lung pathology, and unexpected findings in other organs.

Conduit Selection for CABG or Concomitant Bypass during Other Cardiac Surgery

Plans for conduit use for coronary artery bypass are typically determined during the initial patient

evaluation. Considerations include the patient's age (with consideration of graft patency rates), comorbidities, and quantity and quality of vein available. Obesity, poorly controlled diabetes mellitus, emergency surgery, significant subclavian artery stenosis, and history of radiation to the chest wall may be factors indicating caution when planning the use of one or two internal mammary arteries. Factors involved in the decision to utilize the radial artery include degree of coronary artery stenosis (patency is reduced in noncritical lesions), the result of an Allen test (abnormal result indicates poor flow to the hand and the radial should not be harvested),

perceived patient life span, emergent nature of surgery, patient's occupation (upper extremity fine motor skills and manual dexterity may be vital and may be reduced in rare patients following radial harvest), presence of severe diabetes or peripheral vascular disease, patient preference, and the patient's desire to avoid a visible arm scar. *Preoperative venous mapping* may be obtained to locate and determine the presence and size of bilateral greater and lesser saphenous veins (particularly in redo CABG cases). Preoperative *lower-extremity arterial Doppler* examination may guide vein harvest, as the risk of wound infection and poor wound healing is increased in a lower extremity with peripheral vascular disease.

Left internal mammary artery patency is 94% at 10 years and its use has been shown to prolong patient survival (*J Thorac Cardiovasc Surg.* 1999;117:855). **Use of the left internal mammary artery is now a Medicare quality measure. The use of two internal mammary arteries has been documented to prolong survival compared to that of one internal mammary artery** (*Ann Thorac Surg.* **2004;78:2005). Patency of a free (attached to the aorta) left internal mammary artery graft offers the same patency of an in situ graft (still attached at the subclavian artery).** Patency of radial artery grafts is approximately 80% at 10 years and that of saphenous vein grafts is approximately 50% to 60% at 10 years (*Ann Thor Surg.* 2004;77:93; *J Thorac Cardiovasc Surg.* 2010;140:73). **The use of a radial artery has also been documented to prolong survival in propensity matched studies** (*Circulation.* **2003;108:1350-1354).**

Timing of Surgery

Preoperative hemodynamic instability and/or unrelenting unstable angina often prompt urgent or emergent surgery. In stable patients, factors for consideration include the time since cardiac catheterization, the administration of nephrotoxic medications or agents, and the baseline creatinine due to the risk of postoperative renal dysfunction. Caution should be taken in the stable patient with an acute MI in the setting of poor left ventricular function.

Systemic illnesses and preoperative antithrombotic therapy may also indicate a need for further workup or delay in elective surgery. The Report from the STS Workforce on Evidence Based Surgery states that it is reasonable to discontinue clopidogrel for 5 to 7 days before CABG due to the increased risk of bleeding and that patients on glycoprotein IIb/IIIa inhibitors should be considered high risk for bleeding depending on the half-life of the agent administered (*Ann*

Thorac Surg. 2012;94:1761-1781).

Operative Plan

Careful planning of the procedure in advance will equip the surgeon with the opportunity to provide a calm and systematic resolution to unexpected operative findings. Prior to surgery, a Òback up planÓ should be considered in the event that the procedure does not proceed as planned. For example, the discovery of a porcelain aorta or a significantly dilated ascending aorta would quickly warrant a change in the operative plan.

III. CARDIOPULMONARY BYPASS.

Extracorporeal circulation, in the form of CPB, allows for the performance of cardiothoracic surgery in a quiet, bloodless field. Technologic advances have made the use of CPB readily available and

technically feasible, thereby reducing the morbidity and mortality of cardiac surgery in the current era.

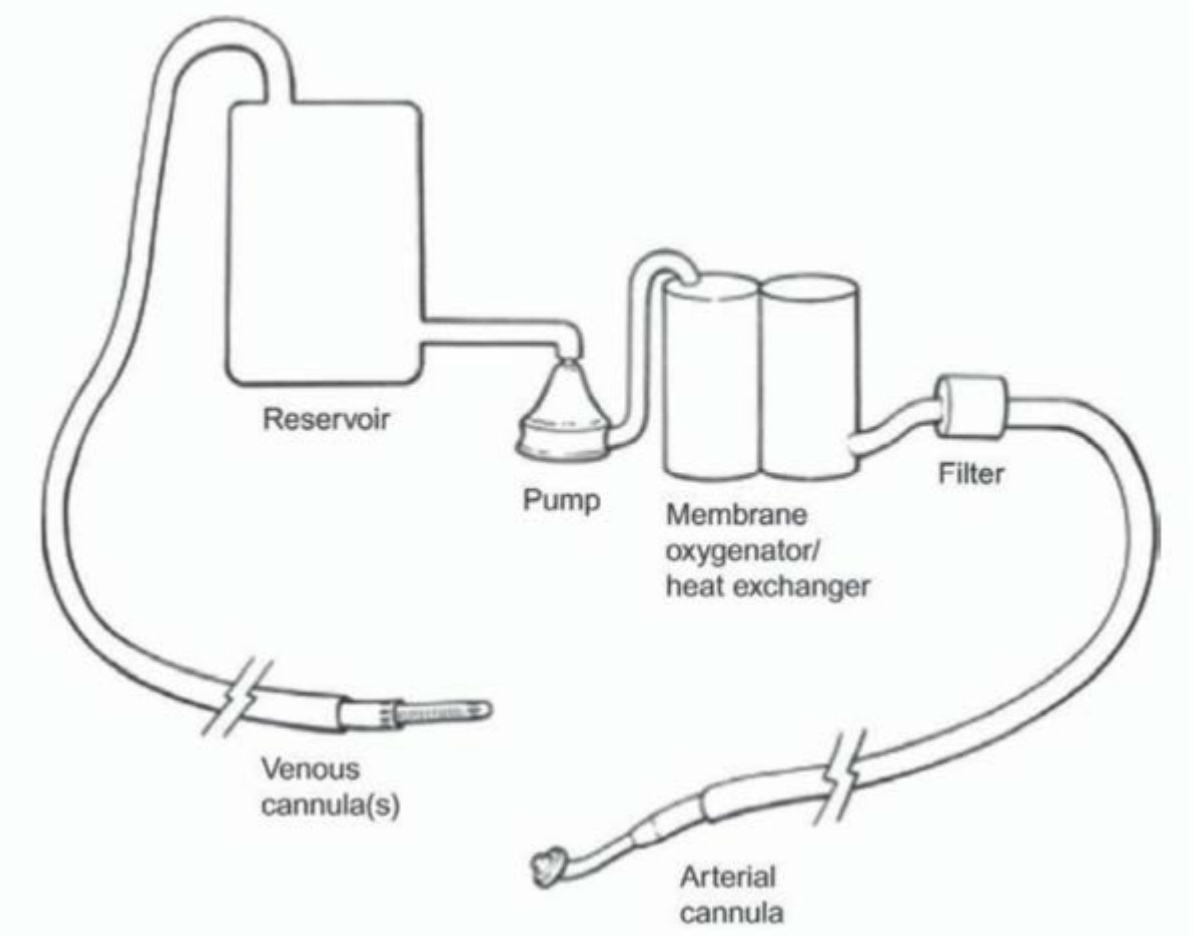


Figure 33-7 Cardiopulmonary bypass circuit. (Reprinted with permission from Punjabi PP

and Taylor KM. The science and practice of cardiopulmonary bypass: From cross circulation to ECMO and SIRS. *Glob Cardiol Sci Pract.* 2013;2013(3):249-260.)

The CPB circuit consists of a venous cannula, a venous line (tubing), a reservoir, a heat exchanger, a membrane oxygenator, an air vent, an arterial line (tubing), and the arterial cannula. The **venous reservoir** stores the blood volume that drains by gravity and allows for the escape of air via the reservoir. All pump suction devices also drain to this reservoir. A **membrane oxygenator** performs gas exchange. A **heat exchanger** regulates patient temperature. The **arterial pump** is usually a roller pump and requires calibration to ensure accurate flow (Fig. 33-7). This serves to pump the blood volume back to the patient via the arterial cannula.

The use of CPB introduces the blood to nonendothelial surfaces that result in detrimental changes manifested throughout the body. The mechanisms responsible include a generalized low flow state, relative ischemia and reperfusion injury, exposure to anticoagulation and its reversal, hemodilution and the destruction of the blood constituents, the embolization of gas or particulate matter, the placement of intravascular cannulae, and the activation of a systemic inflammatory response. Complications directly related to the CPB machine include power interruption, air introduction via physical jarring of the machine or circuit manipulation, oxygenator failure, dislodgement of a connection, pump head reversal, tubing rupture, improper occlusion, and stopcock dislodgement

or turning. It may be difficult to ascertain what portion of observed complications are attributed to CPB alone; however, it is clear that prolonged CPB time is a significant risk factor for all postoperative complications.

IV. SURGICAL PROCEDURES AND INDICATIONS FOR SURGERY

A. Coronary Artery Bypass Grafting (CABG). Coronary artery disease (CAD) is the No. 1 cause of death in men and women in the United States and in most developed countries. Risk factors for CAD include tobacco abuse (including passive exposure to smoke), hypertension, diabetes mellitus, hyperlipidemia, obesity, sedentary lifestyle, advanced age, rheumatoid arthritis or lupus, and a family history of premature CAD in a first-degree relative. Risk factors unique to women in include preeclampsia, gestational diabetes, and pregnancy-induced hypertension (*J Am Coll Cardiol.* 2012;59:1663). Patients may present with angina pectoris, MI, or chronic ischemic cardiomyopathy.

Angina pectoris, or chest pain, occurs when reversible myocardial ischemia is present without cellular necrosis. Angina may manifest as a pain or pressure that often radiates to the left shoulder and down the left arm or into the neck. An anginal equivalent may manifest as epigastric discomfort or shortness of breath. Angina occurs during times of increased myocardial

oxygen demand (exercise) and typically resolves with rest or the administration of nitrates. Unstable angina refers to chest pain that occurs at rest or with increasing frequency, duration, or severity. *MI* results when there is a lack of myocardial oxygen supply with irreversible muscle injury and cell death. Increases in cardiac specific enzymes and ECG changes (ST-segment elevation, T-wave inversions, and new Q waves) are observed. Early and late sequelae of MI can include arrhythmias, congestive heart failure (CHF), VSD, papillary muscle rupture, left ventricular aneurysm, and ischemic cardiomyopathy.

Clinical correlation: Continued angina following MI warrants consideration for urgent or emergent CABG. Medical optimization includes nitrates, intravenous heparin, and placement of an IABP prior to revascularization.

Arrhythmias (potentially fatal ventricular arrhythmias, atrial fibrillation [AF], atrial flutter, heart block, or junctional rhythm) are common during the first 24 hours after acute MI.

CHF may result when a large portion of the left ventricle is infarcted. The extent to which the patient's activity is limited can be graded according to the New York Heart Association (NYHA) classification: Class I, no symptoms; class II, symptoms with heavy exertion; class III, symptoms with mild exertion; class IV, symptoms at rest.

VSD occurs in approximately 2% of patients after MI (anterior wall in 60%, inferior wall in 40%). An acute VSD occurs in the anterior or posterior septum depending upon the location of the coronary artery occlusion and coronary artery dominance (right or left) between 5 days and 2 weeks or more after an acute MI.

Clinical correlation: The onset of a new postinfarction VSD is noted when a new holosystolic murmur is found on physical examination with hemodynamic deterioration (differential diagnosis also includes

papillary muscle rupture as noted below). Diagnosis is made by cardiac cath (oxygen step up in the right heart and left to right shunt noted on left ventriculogram) and by TEE. Treatment is intra-aortic balloon pump (IABP) placement and emergent surgery.

Papillary muscle rupture with severe mitral regurgitation (MR) results when MI involves a papillary muscle and often the adjacent myocardial wall.

Clinical correlation: Acute papillary muscle rupture is noted when a new holosystolic murmur is found on physical examination with hemodynamic deterioration (differential diagnosis also includes VSD as noted above). Diagnosis is made by echocardiography and treatment includes IABP and emergent surgery.

Left ventricular aneurysm occurs late following MI. A well-defined fibrous scar that is dyskinetic develops in 5% to 10% of patients in the infarcted territory. Large dyskinetic left ventricular aneurysms reduce left ventricular EF, result in symptoms of CHF, serve as the substrate for ischemic reentrant ventricular arrhythmias, and create an area of stagnant blood that may lead to

thrombus and peripheral emboli.

Clinical correlation: Akinetic left ventricular walls (which are related to ischemic nontransmural infarcted or hibernating myocardium) must be differentiated from dyskinetic (where blood moves in the opposite direction of normal blood flow) in true left ventricular aneurysms. Left ventricular aneurysmectomy (DOR procedure) may be indicated in appropriate patients who may benefit from the removal of the dyskinetic walls.

Ischemic cardiomyopathy may develop after multiple MIs and is associated with signs and symptoms of heart failure. Treatment is optimal medical management, placement of an automatic implantable cardioverter defibrillator for prevention of sudden cardiac death in patients with significantly reduced EF, and consideration of other support (see section on heart failure).

Indications for CABG

Myocardial revascularization may be accomplished via percutaneous coronary intervention (PCI) (percutaneous transluminal coronary angioplasty, stent, atherectomy) or CABG. Goals are to relieve angina, prevent repeat revascularization and MI, and to prolong survival. The American Heart Association (AHA) and American College of Cardiology (ACC) have established *guidelines for CABG and PCI* (*Circulation.* 2011;124:e652). Current guidelines recommend that CABG be performed to improve survival in patients with significant (³50% stenosis) left main coronary artery stenosis, in patients with significant (³70% stenosis) in three major coronary arteries or in the proximal LAD plus one other major artery, in patients with significant stenosis in two major coronary arteries with severe or extensive myocardial ischemia or target vessels supplying a large area of viable myocardium, in patients with LV dysfunction (EF, 35% to 50%) and significant stenosis when viable myocardium in the area of intended revascularization, in patients with significant stenosis in the proximal LAD and evidence of extensive ischemia, in patients with complex three-vessel CAD with or without involvement of the LAD who are good candidates for surgery, and in patients with multivessel CAD with diabetes mellitus.

CABG versus Medical Therapy

CABG offers symptom relief, reduced risk of MI, less repeat revascularization, survival advantage and improvement in functional status over medical therapy in patients with LMCA disease, multi-vessel disease, and those with left ventricular dysfunction (*Circulation.* 1979;60:888; *Lancet.* 1982;2:1173; *Circulation.* 1983;68:939; *N Engl J Med.* 1984;311:1333; *N Engl J Med.* 1988;319:332; *Circulation.* 1990;82:1629; *J Am Coll Cardiol.* 2004;43:1743).

CABG versus PCI

The SYNTAX trial, the largest, prospective randomized controlled trial, compared CABG to PCI (with drug-eluting stents) in patients with untreated left main or three-vessel CAD. At 1 year, PCI

was associated with higher rates of major adverse cardiac or cerebrovascular events (MACCE) (17.8% PCI vs. 12.4% CABG, p = 0.002) and higher rates of repeat revascularization (13.5% PCI vs. 5.9% CABG, p<0.001) but CABG was associated with higher rates of stroke. By 3 years, there were no statistically significant differences in stroke rates between groups (2.9% CABG vs. 2.6% PCI, p = 0.64). The 5-year results demonstrated persistent divergence in all event rates except stroke in patients with three-vessel CAD (MACCE 37.5% PCI vs. 24.2% CABG, p<001; all cause death 14.6% PCI vs. 9.2% CABG, p = 0.006; MI 10.6% PCI vs. 3.3% CABG, p<0.001; repeat revascularization 25.4% PCI vs. 12.6% CABG, p<0.001; and stroke 3.0% PCI vs. 3.4% CABG, p = 0.66) (*Eur Heart J.* 2014;35:2821).

Contraindications for CABG

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. The risks and benefits of the procedure must be individualized for each patient before proceeding with surgery.

Clinical correlation: Questions to consider when evaluating a patient for CABG include:

1. Does the patient have suitable conduit and targets for the procedure?

2. Are there any particular anatomic issues that that may either prevent the procedure or increase the complexity to an unacceptably high level (i.e., porcelain aorta, mediastinal radiation)?

3. Do the expected perioperative risks outweigh the potential benefit of the procedure? Consider factors that may be prohibitive (preexisting stroke, frailty, dementia, severe COPD, bleeding dyscrasias, malnutrition, or liver insufficiency).

Minimally invasive CABG

Minimally invasive approaches for CABG surgery include off pump (OPCAB), robotic or endoscopic CABG, hybrid CABG, (includes CABG to LAD and DES placed to other vessels), and CABG with only a left anterior thoracotomy for bypass of the LAD.

OPCABG is technically more challenging and is performed on the beating heart with the use of suction stabilizers or other techniques. OPCAB has been shown to decrease morbidity (*J Thorac Cardiovasc Surg.* 2003;125:797; *BMJ.* 2006;332:1365), while others have reported reduced long-term survival (*J Thorac*

Cardiovasc Surg. 2014;148:5). Graft patency rates following OPCAB vary between studies (*JAMA.* 2004;291:1841-1849; *Br Med J.* 2006;332:1365; *J Thorac Cardiovasc Surg.* 2009;137:295; *N Engl J Med.* 2009;361:1827-1837). Fewer blood transfusions, shorter hospital length of stay, shorter duration of intubation, reduced pulmonary complications, and reduced cost have been consistently associated with off pump CABG in randomized trials (*Chest.* 2005;127:892; *B Med J.*

2006;332:1365; *J Card Surg.* 2006;21:35). Guidelines from the International Society of Minimally Invasive Cardiac Surgery (ISMICS) have been published and will be updated in 2015 (*Innovations.* 2012;7:229). Patients at high risk (severe atheromatous aortic plaque, renal failure, or the elderly) may derive the greatest benefit from off pump CABG.

Clinical correlation: Appropriate patient selection is important. Considerations include surgeon experience and learning curve, size, and disease of target vessels, location of coronary stenoses, and need for Òno-touchÓ aortic technique in the case of a porcelain aorta (Fig. 33-5).

B. Valvular Heart Disease

1. Aortic valve disease

a. Aortic stenosis (AS) may result from senile degeneration and calcification of a normal valve, a congenitally bicuspid aortic valve, or an abnormal valve (rheumatic disease). The degree of valvular stenosis is graded using echocardiography from mild to severe. AS places a pressure overload on the left ventricle, resulting in left ventricular hypertrophy. Symptoms include chest pain or angina, shortness of breath or CHF, and syncope.

Clinical correlation: Physical examination findings include a loud systolic murmur across the precordium and a murmur audible in the carotid arteries.

Aortic valve replacement provides relief of symptoms as well as a survival benefit in patients with symptomatic severe (AVA <1 cm^2) AS compared to medical management alone (*Circulation.* 1982;66:1105; *J Thorac Cardiovasc Surg.* 2014;64:1763). Aortic valve replacement is also indicated in asymptomatic patients with severe AS undergoing CABG or other cardiac surgery and in patients with severe AS and left ventricular systolic dysfunction (EF < 0.50) (*J Thorac Cardiovasc Surg.* 2014;64:1763).

Clinical correlation: Patients who undergo surgical aortic valve replacement often have significant left ventricular hypertrophy. Because of this hypertrophy, initial consideration of treatment of hypotension should include volume resuscitation rather than vasoconstrictor agents.

b. *Aortic insufficiency* (AI) is often the result of valve leaflet pathology resulting in thickening, calcification and fixation (rheumatic heart disease or combined with AS), leaflet redundancy or destruction (myxomatous degeneration), maladaptation (aortic root dilatation, aortic dissection), inflammatory disease (ankylosing spondylitis), trauma (blunt chest injury, balloon dilatation, or transaortic valve implantation), or destruction (endocarditis). Chronic AI

results in volume overload of the left ventricle, causing chamber enlargement and wall thickening and pulmonary congestion. Severity of insufficiency is graded from mild to severe by echocardiography. Acute AI is not well tolerated and often results in fulminant pulmonary edema, myocardial ischemia, and cardiovascular collapse.

Clinical correlation: IABP is contraindicated with severe AI as the balloon inflates during diastole, resulting in increased AI.

Indications for surgery in symptomatic patients include severe AI, chronic moderate to severe AI and left ventricular dysfunction (EF < 50%), and patients with chronic severe AI who are undergoing other cardiac surgery. Surgery is reasonable in patients without symptoms and normal left ventricular function but who have severe left ventricular dilatation (left ventricular end systolic dimension >50 mm or indexed left ventricular end systolic dimension >25 mm/m^2 (*J Thorac Cardiovasc Surg.* 2014;64:1763).

Surgical treatment may include open valve repair or replacement or alternative access approaches depending upon the pathology, leaflet quality, and patient comorbidities. One alternative access approach is transcatheter aortic valve implantation (TAVI). TAVI is the replacement of the aortic valve through a transfemoral, transaortic, or transapical approach. The first-generation devicesÑ the balloon-expandable **SAPIEN valve** (Edwards Lifesciences, Irvine, CA) and the self-expandable **CoreValve** (Medtronic, Minneapolis, MN)Ñhave been thoroughly tested in the United States by large, multicenter trials demonstrating their safety and efficacy in high-risk patients. Second-generation devices have been developed to reduce the shortcomings of the first-generation devices and are currently being tested in large, multicenter trials in the United States.

2. Mitral valve disease

a. Mitral stenosis (MS) is caused by valve leaflet thickening and calcification due to rheumatic fever or senile calcification, collagen vascular diseases, amyloidosis, congenital stenosis, or mitral inflow obstruction (tumors or masses). MS places a pressure overload on the left atrium, with relative sparing of ventricular function. Left atrial dilation to more than 45 mm is associated with a high incidence of AF and subsequent thromboembolism. Critical MS occurs when the valve area is 1.5 cm^2 or less.

Clinical correlation: Physical examination findings include an apical diastolic murmur and a loud S1.

Symptoms usually develop late and reflect pulmonary congestion (dyspnea), reduced left ventricular preload (low-cardiac-output syndrome), or AF (thromboembolism). Surgery is indicated in patients with severe MS and severe symptoms, patients with moderate MS undergoing cardiac surgery for other indications, or in patients who have had recurrent embolic events while receiving adequate anticoagulation and excision of the left atrial appendage may be

considered. Percutaneous mitral balloon valvuloplasty is indicated in symptomatic patients with severe mitral rheumatic stenosis who have favorable valve morphology in the absence of contraindications (*J Thorac Cardiovasc Surg.* 2014;64:1763). **b.** MR may be categorized as primary (degenerative) or chronic secondary (functional). Primary causes include those that involve the leaflets (mitral valve prolapse, fibroelastic deficiency, connective tissue disorders,

rheumatic heart disease, cleft mitral valve, radiation injury, trauma following mitral valvuloplasty, or endocarditis), the chordae tendineae (rupture [endocarditis or MI], fusion, or elongation), or the papillary muscles (ischemic papillary muscle dysfunction or rupture secondary to MI [noted under MI]) (*J Thorac Cardiovasc Surg.* 2014;64:1763). Chronic secondary MR is due to either chronic ischemic cardiomyopathy or idiopathic myocardial disease causing secondary dilation of the annulus itself.

Clinical correlation: Physical examination findings include loud systolic murmur loudest at the apex.

MR places a volume overload on the left ventricle and atrium, causing chamber enlargement and wall thickening and pulmonary congestion. AF often develops due to left atrial dilation. Acute severe MR results in pulmonary congestion and low cardiac output.

Clinical correlation: In acute, severe MR, IABP is beneficial as it decreases afterload, thereby increasing forward flow and decreasing regurgitant volume.

Mitral valve repair or replacement is recommended for symptomatic patients with chronic severe MR and left ventricular EF greater than 30%, and is recommended in asymptomatic patients with chronic severe primary MR and left ventricular dysfunction (EF 30% to 60%) (*J Thorac Cardiovasc Surg.* 2014;64:1763). Specific indications and recommendations for repair of the mitral valve continue to evolve (*J Thorac Cardiovasc Surg.* 2014;64:1763). Mitral surgery in patients with chronic ischemic MR is challenging and is associated with increased operative risk. In a recent randomized trial, mitral valve replacement provided a more durable freedom from MR compared to mitral repair in patients with severe ischemic MR (*N Eng J Med.* 2014;370:1).

3. Tricuspid valve disease

a. Tricuspid stenosis (TS) is most commonly secondary to rheumatic disease. TS may be associated with regurgitation and may not be detectable on bedside examination (*J Thorac Cardiovasc Surg.* 2014;64:1763). Tricuspid valve surgery is recommended in patients with severe TS at the time of operation for left-sided valve disease and in isolated symptomatic severe TS (*J Thorac Cardiovasc Surg.* 2014;64:1763). Tricuspid balloon commissurotomy might be considered in patients with isolated severe TS without TR (*J Thorac Cardiovasc Surg.* 2014;64:1763).

b. Tricuspid insufficiency (TI) most often results from a secondary (functional) dilation of the valve annulus caused by pulmonary

hypertension, caused by intrinsic mitral or aortic valve disease. Causes of primary TI include rheumatic heart disease, bacterial endocarditis (often in IV drug users), carcinoid tumors, Ebstein anomaly, and blunt trauma. Mild-to-moderate TI usually is well tolerated. Tricuspid repair is indicated in the case of moderate to severe TI at the time of surgery for other cardiac anomalies. The majority of tricuspid valves can be repaired with annuloplasty techniques rather than replacement.

Clinical correlation: TI may be associated with a systolic murmur, a prominent jugular venous pulse, and a pulsatile liver.

Clinical correlation: The AV node is of particular importance during tricuspid valve surgery because it may be injured resulting in heart block. Tricuspid valve repair techniques involve incomplete rings to avoid injury to the conduction system and sutures in the annulus at the time of valve replacement are preferentially placed in the leaflet tissue only (Fig. 33-2).

4. Pulmonic valve disease

a. Pulmonic insufficiency (PI) that is mild to moderate does not require intervention if asymptomatic and if associated with normal right ventricular function and size (*J Thorac Cardiovasc Surg.* 2014;64:1763). PI in adults more commonly requires treatment in patients following childhood repair of Tetralogy of Fallot. The pulmonic valve may be affected by endocarditis, tumors (carcinoid, papillary fibroelastoma), or radiation injury.

b. Pulmonic stenosis (PS) is mostly seen as a congenital disorder and is rarely seen in adults.

5. Infective endocarditis. Endocarditis may involve infection of native valves (typically abnormal: Elderly sclerotic disease or rheumatic valvular disease) or prosthetic valves. Endocarditis may be related to bacteremia (poor dental hygiene, intravenous drug abuse [IVDA], nosocomial infection, indwelling intravenous catheters or dialysis). Surgical treatment is associated with increased survival compared to antibiotic therapy alone and should be aggressive in order to eradicate all infected tissues (*Heart.* 2001;89:269; *Heart.* 2001;88:61). The risk of embolization is reduced significantly following the initiation of appropriate antibiotics. Indications for surgery due to endocarditis may include hemodynamic instability, CHF, large foci of disease (>1 cm), aggressive bacterial organisms or fungal organisms, prosthetic valve endocarditis, aortic root abscess, and persistent evidence of infection despite appropriate antibiotic therapy (persistent fever, recurrent septic emboli, persistent positive blood cultures). The risk for reoperation for recurrent infective endocarditis is about 17% for IV drug users and 5% for non-IV drug users (*Ann Thorac Surg.* 2007;83:30).

Clinical correlation: Aortic root abscess is a surgical emergency. Daily ECGs should be monitored for signs of heart block that may occur with destruction of the AV node between the right and noncoronary cusps of the aortic annulus.

6. Prosthetic valve selection. *Tissue bioprostheses* include porcine aortic valves or valves constructed with bovine pericardium. These prostheses are associated with a low rate of thromboembolism, even without longterm anticoagulation (a daily aspirin is recommended). They are less durable than mechanical valves (mean time to failure is approximately 10 to 15 years) and this can potentially be prolonged in newer valves due to modern preservation methods. Bioprostheses are often the preferred valves for older patients (>65 years) or patients with a

contraindication to anticoagulation.

Mechanical valves have excellent long-term durability; however, the rate of thromboembolic complications (0.5% to 3% per year) is cumulative and these valves require lifelong anticoagulation. Newer valve options (On-X) with alternative construction allow for a lower target INR (*J Thorac Cardiovasc Surg.* 2014;147:1202).

Homograft/allograft and autografts are useful for replacement of the aortic valve, particularly in the setting of endocarditis or for the Ross procedure (*J Heart Valve Dis.* 1994;3:377). These tissues have reasonable durability and a low incidence of thromboembolism. Their use is limited due to supply and technical expertise needed for implantation and to the drawbacks of long-term function noted in autografts in the aortic position.

7. New technology. Following the introduction of TAVI, the treatment of valvular disease continues to rapidly evolve. New transcatheter therapies for the mitral valve have recently been introduced that allow for mitral valve repair via a transfemoral approach by clipping the two leaflets of the mitral valve (MitraClip, Abbott Laboratories, Abbott Park, Il). Transcatheter mitral valve replacement is also on the horizon and first-in-man trials are underway. Suture-less valves have been designed for rapid surgical deployment. The trend toward minimally invasive procedures and the combination of transcatheter and surgical techniques have mandated a multidisciplinary approach to the treatment of valvular disease.

C. Hypertrophic Cardiomyopathy (HCM). HCM characterized by asymmetric hypertrophy and fibrosis of the myocardium, causing obstruction of the left ventricular outflow tract. Medical therapy with β-blockade or calcium channel blockade is the preferred first-line treatment. Nifedipine, nitroglycerin, angiotensin-converting enzyme inhibitors, and angiotensin II blockers are all generally contraindicated due to their vasodilatory properties, which can exacerbate the outflow tract obstruction. Surgical treatment of HCM is myectomy, with a postoperative mortality of 1% or less (*Ann Thorac Surg.* 2000;69:1732). By convention, surgery is recommended for symptomatic patients who have failed medical therapy or septal ablation with a documented at-rest outflow tract gradient of at least 30 mm Hg.

D. Atrial Fibrillation. AF affects 1% to 2% of the general population and it affects nearly 10% of individuals >80 years of age. Morbidity includes patient discomfort, hemodynamic compromise, and thromboembolism.

Nonsurgical management of AF includes antiarrhythmic drugs, cardioversion, and catheter ablation. Indications for surgery include symptomatic AF in patients undergoing other cardiac procedures, selected asymptomatic AF patients undergoing cardiac surgery in whom ablation can be performed with minimal risk, and for symptomatic AF patients who prefer a surgical approach, have failed one or more attempts at catheter ablation, or are not candidates for catheter ablation (*Heart Rhythm.* 2007;4:816). The Coxmaze procedure is the gold standard for surgical AF ablation and its current iteration, the Cox-maze IV, utilizes a combination of cryoablation and

bipolar radiofrequency ablation. Postoperative freedom from AF at 93%, 90%, and 90% at 6, 12, and 24 months, respectively, has been demonstrated (*J Interv Card Electrophysiol.* 2011;31:47). Results have been variable when less complete surgical ablations are performed.

E. Surgical Treatment of Heart Failure. It is estimated that approximately 250,000 people suffer from advanced heart failure in the United States (*Curr Heart Fail Rep.* 2010;7:140). The management of heart failure involves aggressive medical and surgical care.

The *IABP* (see myocardial infarction) is used as the first-line device to provide circulatory support in *acute* heart failure (*Ann Thorac Surg.* 1992;54:11). Indications may include preoperative low-cardiac-output states, preoperative unstable angina refractory to medical therapy, intraoperative weaning from CPB after inotropic agents are maximized, and postoperative low cardiac output states. 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). With clinical improvement the IABP is gradually weaned by decreasing the augmentation frequency from 1:1 to 1:3 in steps. Placement of an IABP is not without risk and potential complications include incorrect placement resulting in abdominal organ malperfusion, perforation of the aorta, injury to the femoral artery, and ischemia of the lower extremity. Rupture of the balloon is an indication for immediate removal due to risk of thrombosis.

Clinical correlation: Contraindications for IABP include AI, aortic dissection, and severe peripheral vascular disease.

Percutaneous assist devices are devices that can be deployed via catheter-based approach and function as ventricular assist devices (VADs) without the need for open surgery. They are typically used only for short-term support. Examples of these include the TandemHeart, the Impella, and the CentriMag (*Expert Rev Cardiovasc Ther.* 2010;8:1247).

Cardiac resynchronization therapy (biventricular pacing) has emerged as a valid treatment modality for patients with heart failure and concomitant intraventricular conduction delay manifested by QRS complex greater than 120 milliseconds.

Extracorporeal membrane oxygenation (ECMO) is a temporary circulatory support that is nonphysiologic and usually nonpulsatile. It consists of a centrifugal pump and an oxygenator; thus, anticoagulation is necessary. Most commonly, it is used to allow patients to recover from reversible myocardial

dysfunction (stunning), adult respiratory distress syndrome, or pulmonary insufficiency of various etiologies.

VADs may be used to support the left (LVAD) or the right ventricle (RVAD). When both an LVAD and an RVAD are used, the combination is termed a biventricular assist device (BiVAD). They provide mechanical decompression of the ventricle and restoration of cardiac output, resulting in

decreased myocardial oxygen consumption. Indications include three separate broad categories: Inability to separate from CPB despite inotropic and IABP support (Òbridge to recoveryÓ), intermediate-term cardiac support (Òbridge to transplantÓ), and permanent replacement therapy in nontransplant candidates (Òdestination therapyÓ) (*J Heart Lung Transplant.* 2013;32:1147-1162).

VAD subtypes include *nonpulsatile devices* (centrifugal or axial flow pumps) and *pulsatile devices* (external and long-term implantable devices). Device selection depends on surgeon familiarity and preference, practical advantages and disadvantages of each device, and unique patient characteristics and body habitus. Devices currently in use include the HeartMate II (Thoratec Corp., Pleasanton, CA), which is a second-generation nonpulsatile axial flow continuous flow VAD, and the *HVAD* (Heartware, Inc., Framingham, MA), which is a third-generation nonpulsatile centrifugal continuous flow VAD.

Following VAD placement, the activated clotting time should be maintained at approximately 200 seconds once postoperative bleeding is acceptable. Factors that affect a low-flow-state status post LVAD include right ventricular dysfunction, pulmonary hypertension, hypovolemia, and tamponade. Pulmonary vasodilators (nitric oxide or inhaled prostacyclin) are frequently used to lower pulmonary vascular resistance and load on the right ventricle. Complications include bleeding, thrombus formation, embolization, hemolysis, infection at the drive line site, and device failure.

Cardiac transplantation can provide relief from symptoms in patients with end-stage cardiomyopathy who are functionally incapacitated despite optimal medical therapy and who are not candidates for other procedures. Heart transplantation is typically utilized in patients with ischemic, idiopathic, postpartum, or chemotherapy-induced cardiomyopathy and is associated with a 1-year survival of 84.5% and a 5-year survival of 72.5% (*J Heart Lung Transplant.* 2014;33:996-1008). *Relative indications* include refractory cardiogenic shock, instability in fluid balance or renal function despite optimal medical therapy, severe persistent angina not amenable to revascularization, markedly reduced exercise capacity (peak VO_2 <10 to 14 mL/kg/minute), and recurrent refractory ventricular arrhythmias (*Mayo Clin Proc.* 2014;89:662).

Relative contraindications to transplantation include age older than 65 years, irreversible pulmonary hypertension, active infection or malignancy, recent pulmonary embolus, and excessive comorbidity (renal dysfunction, hepatic dysfunction, systemic disease such as amyloidosis, significant peripheral vascular disease, active peptic ulcer disease, uncontrolled diabetes mellitus, morbid obesity, mental illness, active substance abuse,

inadequate social support, or psychosocial instability) (*Mayo Clin Proc.* 2014;89:662).

Complications of heart transplant include acute and chronic rejection that may be diagnosed by endomyocardial biopsy or echocardiography. Coronary artery vasculopathy (CAV), thought to represent chronic vascular rejection**,** occurs in a significant percentage of cardiac transplant

recipients and is a major limitation on the long-term success of cardiac transplantation, being responsible for 30% of deaths in transplanted patients after 5 years (*J Heart Lung Transplant.* 2007;26:769). CAV is usually not amenable to conventional revascularization owing to small-vessel, nonfocal disease and often requires retransplantation.

V. POSTOPERATIVE MANAGEMENT AND COMPLICATIONS

A. Monitoring and Management. Early extubation following cardiac surgery is preferred if the patient is hemodynamically stable and bleeding is minor. Neurologic assessment is obtained as soon as possible. Normal hemodynamic values obtained from a Swan-Ganz catheter are listed in Table 33-2. A *cardiac index* of 2 L/minute/m^2 is generally a minimum acceptable value. A *mixed-venous oxygen saturation* of less than 60% suggests inadequate peripheral tissue perfusion and increased peripheral oxygen extraction. Etiologies of low cardiac output must be aggressively corrected by manipulation of factors affecting cardiac output (heart rate, preload, afterload, and contractility). Myocardial stunning is common following heart surgery and requires additional support (pharmacologic and mechanical). Stunning is defined as a transient postischemic myocardial dysfunction that persists despite adequate reperfusion and in the absence of irreversible damage (*Circulation.* 1982;66:1146). One of the easiest methods to increase cardiac output is to increase the heart rate. This may be accomplished by using the temporary epicardial pacing electrodes (placed at the time of operation) at 80 to 100 beats/minute. **Optimal pacing always involves maintaining AV synchrony.**

B. Complications. Complications related to CPB were detailed in the cardiopulmonary bypass section.

Arrhythmias are common following cardiac surgery. *Supraventricular arrhythmias* (AF, atrial flutter, atrial tachycardia) are most common and are associated with an increased risk of transient or permanent neurologic deficits (*J Card Surg.* 2005;20:425). Postoperative AF occurs in approximately 30% of patients and has a peak incidence on postoperative day 2 (*J Thorac Cardiovasc Surg.* 2011;141:559). Supraventricular arrhythmias with hemodynamic compromise can rapidly be treated with electrical cardioversion (with 50 to 100 J). For patients with hemodynamic stability and *atrial flutter,* overdrive pacing may be used to terminate the arrhythmia. For AF and flutter, ventricular rate control may be facilitated with β-blockade and amiodarone. AF or flutter that persists beyond 12 hours or that is recurrent despite amiodarone, anticoagulation (IV heparin as a bridge to oral Coumadin with a goal INR of 2.0 to 3.0) should be considered to avoid the complication of stroke. Sustained *ventricular arrhythmias* other than

premature ventricular contractions suggest underlying ischemic pathology and etiology should be investigated.

TABLE 33-2 Normal Hemodynamic Parameters

Parameter	Normal Value	Unit
Central venous pressure	2-8	mm Hg
Right ventricular pressure (syst/diast)	15-30/2-8	mm Hg
Pulmonary artery pressure (syst/diast)	15-30/4-12	mm Hg
Pulmonary capillary wedge pressure	2-15	mm Hg
Left ventricular pressure (syst/diast)	100-140/3-12	mm Hg
Cardiac output	3.5-5.5	L/min
Cardiac index	2-4	L/min/m^2 BSA
Pulmonary vascular resistance	20-130	dynes sec/cm^5
Systemic vascular resistance	700-1,600	dynes sec/cm^5
Mixed-venous oxygen saturation	65-75	Percent

BSA, body-surface area; diast, diastolic; syst, systolic

Respiratory failure may occur following cardiac surgery. A prompt postoperative trial of extubation should be attempted along with the limitation of sedation, tracheostomy, and early nutritional support. Appropriate antibiotics are

administered for ventilator-associated pneumonia.

Postoperative bleeding is relatively common after cardiac surgery and necessitates reexploration in up to 5% of patients. A treatment algorithm for the management of postoperative bleeding is represented in Figure 33-8. *Cardiac tamponade* is a potentially lethal cause of low cardiac output with associated narrowed pulse pressure, increased jugular venous distention, rising CV, muffled heart sounds, pulsus paradoxus, widened mediastinal silhouette on chest radiograph, and decreased urine output. Diagnosis is clinical and may be confirmed by echocardiography. Treatment is emergent drainage in the operating room. *Delayed tamponade* occurs after the acute postoperative period and may be extremely difficult to diagnose. A high index of suspicion for this diagnosis must be maintained. A decline in renal or liver function, poor urine output, or an elevated coagulation profile may be the initial clue. An echocardiogram is helpful in making the diagnosis.

This complication must be rapidly treated with reexploration of the chest and evacuation of mediastinal fluid.

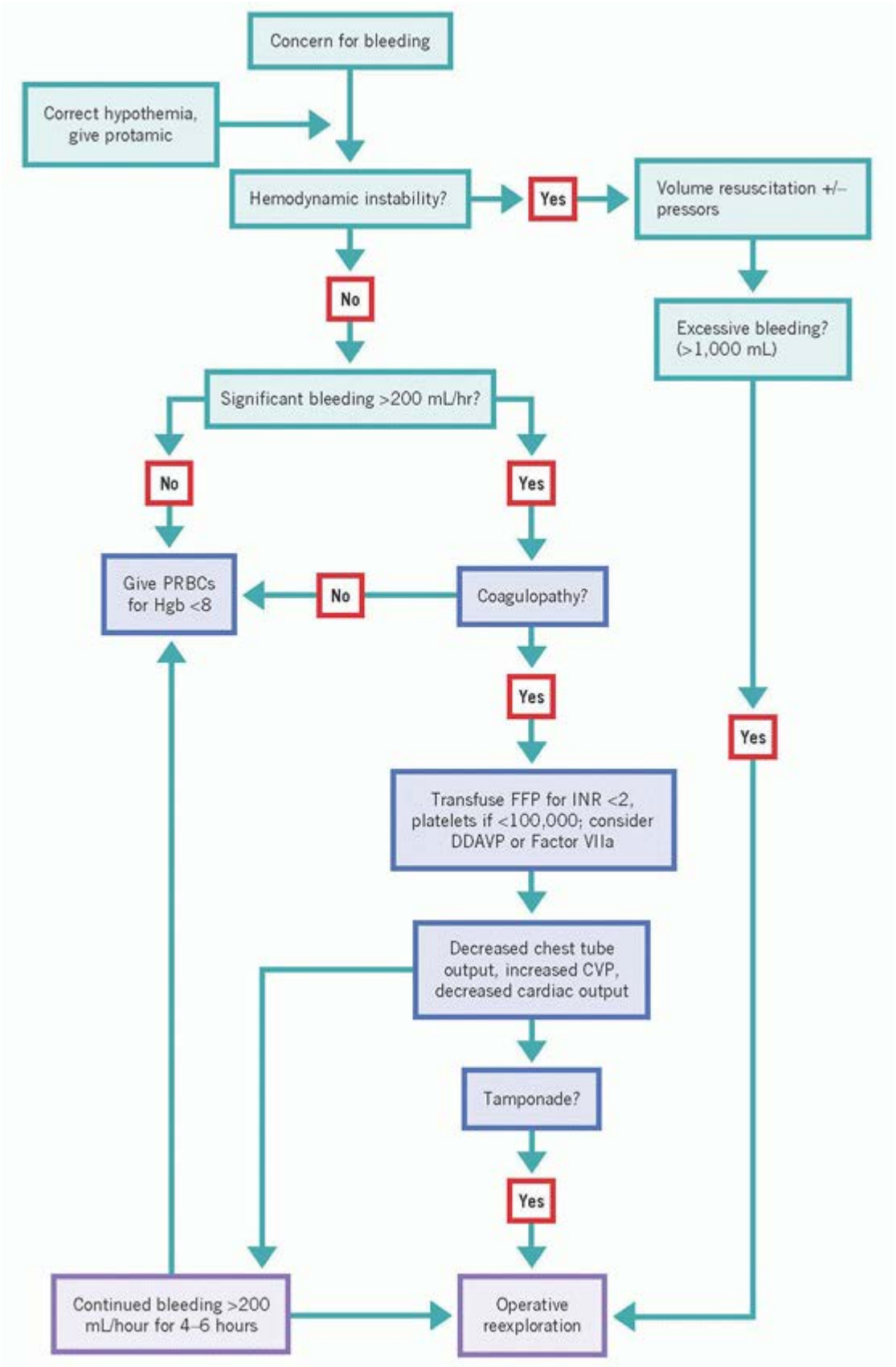


Figure 33-8 Postoperative bleeding algorithm.

Perioperative MI occurs in approximately 1% to 2% of patients and can be diagnosed by chest pain, ventricular arrhythmias, ECG changes, elevated cardiac specific enzymes, or new changes on echocardiography. Newly constructed bypass grafts can be occluded due to thrombosis or spasm. Cardiac catheterization can delineate appropriate treatment, which may include return to the operating room or stenting for myocardial revascularization.

Renal dysfunction in the postoperative period significantly increases mortality. To limit renal injury, an acceptable cardiac index and mean arterial pressure must be maintained. Nephrotoxic agents should be avoided or limited, and any renally excreted medication should have dose adjustment.

Cerebrovascular accident (CVA) is the most devastating of neurologic complications following cardiac surgery. Neurologic complications may be manifested by a wide range of signs and symptoms from delirium and confusion to permanent stroke and may be related to intraoperative (aortic atherosclerotic or air emboli) or perioperative events. An acceptable cardiac output and mean arterial pressure must be maintained. Patients with postoperative AF or documented poor EF with intracardiac clot should be anticoagulated to prevent cerebral embolization.

Postoperative infection may be clinically significant in the sternal wound, urinary tract, conduit harvest sites, or lungs. Perioperative intravenous antibiotics are administered prior to incision and continued for only 48 hours postoperatively. Prophylaxis with a second-generation cephalosporin has been associated with a fivefold decrease in wound infection rates compared to placebo in a meta-analysis (*J Thorac Cardiovasc Surg.* 1992;104:590). Additional antibiotic use should be guided by specific culture and sensitivity results. In addition, aggressive blood sugar management (often with insulin infusion) is utilized to limit the occurrence of sternal wound infection. Central lines and chest tubes are promptly discontinued when no longer necessary or useful. In addition, other sites (conduit harvest sites, urinary tract, and lungs) are closely monitored and promptly treated should an infection occur.

Serous drainage from the sternal incision is worrisome and should be treated by application of a sterile dressing twice daily and the administration of IV or oral antibiotics. Purulent wound drainage, a sternal click, gross movement of the sternal edges, or substernal air on the chest x-ray may indicate a deep sternal infection. A CT scan of the chest can confirm this diagnosis. Deep sternal infections require operative debridement of devitalized sternal and soft tissue, administration of broad-spectrum IV antibiotics, and muscle flap closure of the soft tissue defects.

Gastrointestinal (GI) complications are uncommon after cardiac surgical

procedures. Prompt diagnosis and treatment requires a high index of suspicion. GI ischemia may occur following periods of hypoperfusion due to low cardiac output, hypotension from blood loss, or as a result of vascular

emboli. GI bleeding may be exacerbated by the use of antiplatelet agents and anticoagulation. Adequate gastric mucosal protection should be provided with proton pump inhibitors to prevent stress ulceration. In addition, early enteral feeding should be initiated in the case of prolonged ventilation. Acute cholecystitis, usually acalculous, is associated with a high mortality. Antibiotics should be discontinued promptly when they are no longer clinically indicated to prevent *Clostridium difficile* colitis.

CHAPTER 33: CARDIAC SURGERY

Multiple Choice Questions

1. A 75-year-old female underwent coronary bypass grafting with four grafts and returns to the ICU in stable condition on no pressors. Upon arrival to the ICU, the patient is noted to have a cardiac index of 1.7 but is normotensive and there is no evidence of bleeding. The admission ECG is unchanged, an echo shows no effusion, the CVP is found to be 20 mm Hg, and the admission laboratory values are unremarkable. What is the next best step in management?

a. Transfuse 1 unit of blood

b. Start Dobutamine

c. Bolus 1 L of crystalloid

d. Place an intra-aortic balloon pump

e. Return to the operating room for exploration

View Answer

2. A 60-year-old male returns to the ICU after a redo aortic valve replacement. In the first 10 minutes after the arrival the ICU, the patient has 150 mL of sanguineous output from his mediastinal chest tubes, but is hemodynamically stable. What is the best next step in management?

a. Return to the operating room for exploration

b. Re-open the chest in the ICU

c. Transfuse platelets

d. Transfuse FFP

e. Place a warmer on the patient and give protamine

View Answer

3. A 55-year-old male comes to the emergency room with severe chest pain and is diagnosed with an ST-elevation myocardial infarction. On the way to the cath laboratory, he becomes hypoxic and has to be intubated. He undergoes left heart cath and he is found to have 95% left main and 99% RCA lesions, as well as an EF of 10% on ventriculogram. The patient is then referred to a cardiac surgeon for a CABG, and the surgeon asks the interventionalist to place an IABP. Which of the following findings on echo would be a contraindication to placement of IABP?

a. Aortic stenosis

b. Aortic insufficiency

c. Mitral stenosis

d. Mitral insufficiency

e. Tricuspid insufficiency

View Answer

4. Which of the following patients should be referred for CABG?

a. A 48-year-old male with an NSTEMI and 70% mid left anterior descending artery lesion and otherwise clean coronaries.

b. A 60-year-old female with unstable angina and a failed dobutamine stress test.

c. A 55-year-old male with chronic CAD s/p multiple PCI who presents with angina and on a cath has a chronically occluded right coronary, 40% in-stent stenosis of the LAD.

d. A 52-year-old male with a STEMI and 90% proximal LAD and 90% proximal RCA lesions.

e. A 78-year-old s/p cardiac arrest and 30 minutes of CPR in the field who had a cath on arrival that showed 90% LAD, 95% left main coronary, and 95% right main coronary lesions, and a porcelain aorta.

View Answer

5. A 59-year-old male with a history of CAD s/p CABGx4, 10 years ago comes to the hospital complaining of shortness of breath. He is admitted to the ICU where an echo shows an EF of 10% and a left heart

cath shows patent grafts to RCA and LAD, but otherwise diffuse disease throughout the left and right coronary systems. A balloon pump is placed, he is placed on a lasix drip, and high doses of inotropes, but he does not improve. Regarding cardiac transplantation, which of the following is true?

a. His age of 59 years precludes him from being listed for transplant.

b. Acute renal failure would be a relative contraindication for transplant.

c. If he were to get an LVAD, he no longer would be a transplant candidate.

d. If he were to get pneumonia, he would no longer be a transplant candidate until the infection is treated.

e. Ventricular tachycardia would preclude him from transplant.

View Answer

34
Lung and Mediastinal Diseases

Stephanie H. Chang

Varun Puri

I. PNEUMOTHORAX

A. Pneumothorax is the presence of air in the pleural cavity, leading to separation of the visceral and parietal pleura. This results in abnormal pulmonary mechanics that can progress to tension pneumothorax, which causes cardiac compromise and is a true emergency. Pneumothoraces may be spontaneous, iatrogenic, or due to trauma.

1. Spontaneous pneumothoraces are usually caused by the rupture of an apical bleb. The typical patients are tall young males who present with acute shortness of breath and chest pain. Older patients usually have significant parenchymal disease, such as emphysema. These patients present with a ruptured bulla and can have a more dramatic presentation, including tachypnea, cyanosis, and hypoxia. Other etiologies of spontaneous pneumothorax include cystic fibrosis (CF) and, rarely, lung cancer. The risk of ipsilateral recurrence of a spontaneous pneumothorax is 50%, 62%, and 80% after the first, second, and third episodes, respectively, if managed conservatively without surgery.

2. Iatrogenic pneumothoraces are usually the result of pleural injury during central venous access attempts, pacemaker placement, or transthoracic or transbronchial lung biopsy.

3. Traumatic pneumothoraces will be discussed in Chapter 11 (Chest Trauma).

B. Physical examination demonstrates decreased breath sounds on the involved side. Examination for signs of tension pneumothorax, including deviation of the trachea to the opposite side, respiratory distress, and hypotension, must be performed. An upright chest x-ray (CXR) can establish the diagnosis of pneumothorax (Fig. 34-1); however, confirming the diagnosis of tension pneumothorax is unnecessary and leads to dangerous delays. Smaller pneumothoraces may only be evident on expiratory CXRs or CT scan.

C. Management options include observation, aspiration, needle thoracostomy, chest tube placement with or without pleurodesis, and surgery.

1. Observation is an option in a healthy, asymptomatic patient with a small pneumothorax. Supplemental oxygen may help to reabsorb the pneumothorax by affecting the gradient of nitrogen in the pneumothorax.

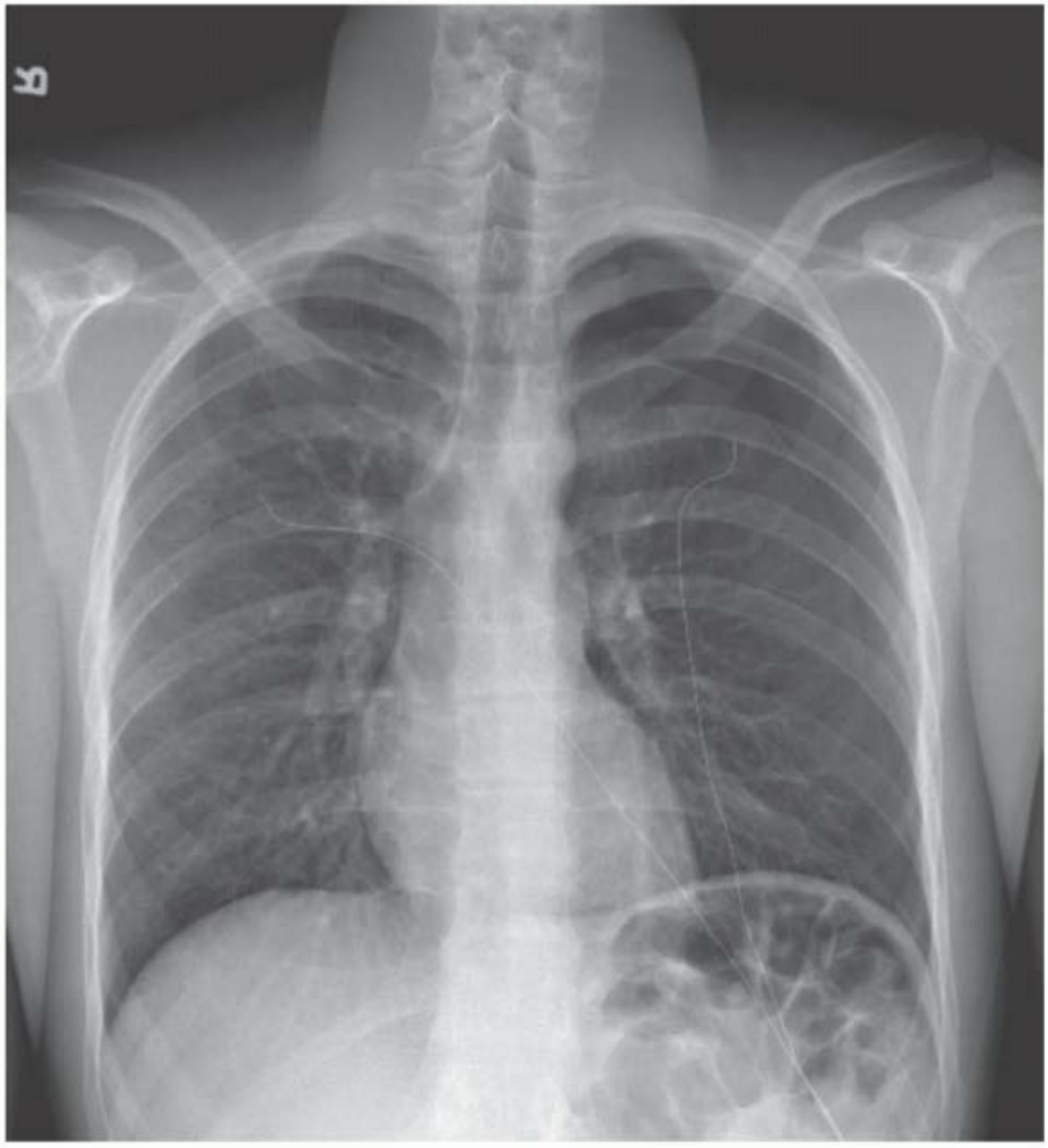


Figure 34-1 Spontaneous left-sided pneumothorax.

2. Aspiration of the pneumothorax may be performed using a small catheter attached to a three-way stopcock. This should be reserved for small to moderate pneumothoraces with low suspicion of an ongoing air leak.

3. Needle thoracostomy should be performed for suspected tension pneumothorax in the setting of hemodynamic compromise. A large-bore IV catheter (14 to 16 gauge) is placed in the second intercostal space in the mid-clavicular line, with the needle advanced until air is freely aspirated into a syringe. The needle is then removed and the catheter is kept open and in place

until a formal chest tube is expeditiously placed.

4. Percutaneous catheters are essentially mini chest tubes that may be placed using Seldinger technique. Multiple commercial kits exist and allow for the catheter to be placed to a water seal or suction. The catheters are of small caliber, so use is limited to situations of simple pneumothorax. If there is concern for lung adhesions, bedside percutaneous catheters should be avoided.

5. Tube thoracostomy remains the gold standard for large pneumothoraces, associated effusion, or when there is an expected need for pleurodesis. Chest tubes may be connected to a Heimlich one-way valve, a

simple underwater-seal system, or to vacuum suction (typically -20 cm). If the water-seal chamber bubbles with expiration or with coughing, it is evident that an air leak persists.

6. Bedside pleurodesis. Sclerosing agents may be administered through the chest tube to induce fusion of the parietal and visceral pleural surfaces. Doxycycline, bleomycin, and talc have all been described. Bedside pleurodesis can be associated with an inflammatory pneumonitis in the lung on the treated side. In patients with limited pulmonary reserve, this may present as clinically significant hypoxia.

7. Surgery is performed using a video-assisted thoracoscopic surgery (VATS) or rarely via thoracotomy.

a. Indications for operation for pneumothorax include

(1) Recurrent spontaneous ipsilateral pneumothoraces

(2) Bilateral pneumothoraces

(3) Persistent air leaks on chest tube suction (usually >3 to 5 days)

(4) First spontaneous pneumothorax in patients with high-risk occupations (e.g., pilots and divers) or live at a great distance from medical facilities

b. 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 performed to promote formation of adhesions between visceral and parietal pleurae. In older patients, intraoperative talc insufflation in the pleural space provides reliable pleurodesis.

II. PLEURAL EFFUSION

A. Pleural effusions are buildup of fluid in the pleural space and may result from a wide spectrum of benign, malignant, and inflammatory conditions. They are broadly categorized as either **transudative** (protein-poor fluid from increased intravascular pressure) or **exudative** (protein or cell rich fluid resulting from increased vascular permeability).

B. Presenting symptoms of pleural effusions can include dyspnea, cough, or pleuritic chest pain, as well as a variety of symptoms specific to the underlying etiology. Small pleural effusions are often asymptomatic.

C. Diagnosis

1. Most pleural effusions are first diagnosed on **CXRs** (Fig. 34-2). **CT scan** and **ultrasound** can be helpful if history suggests a more chronic organizing process such as empyema.

2. Thoracentesis is frequently used to evaluate large, recurrent, or symptomatic pleural effusions. The fluid should be sent for pH, glucose, amylase, lactate dehydrogenase (LDH), protein levels, culture and Gram stain, a differential cell count and cytology, to aid in diagnosis (Fig. 34-3). Appearance of the fluid often indicates etiology: Thin yellow or clear fluid is common with transudative effusions; cloudy, foul-smelling fluid usually signals infection; milky white fluid suggests chylothorax.

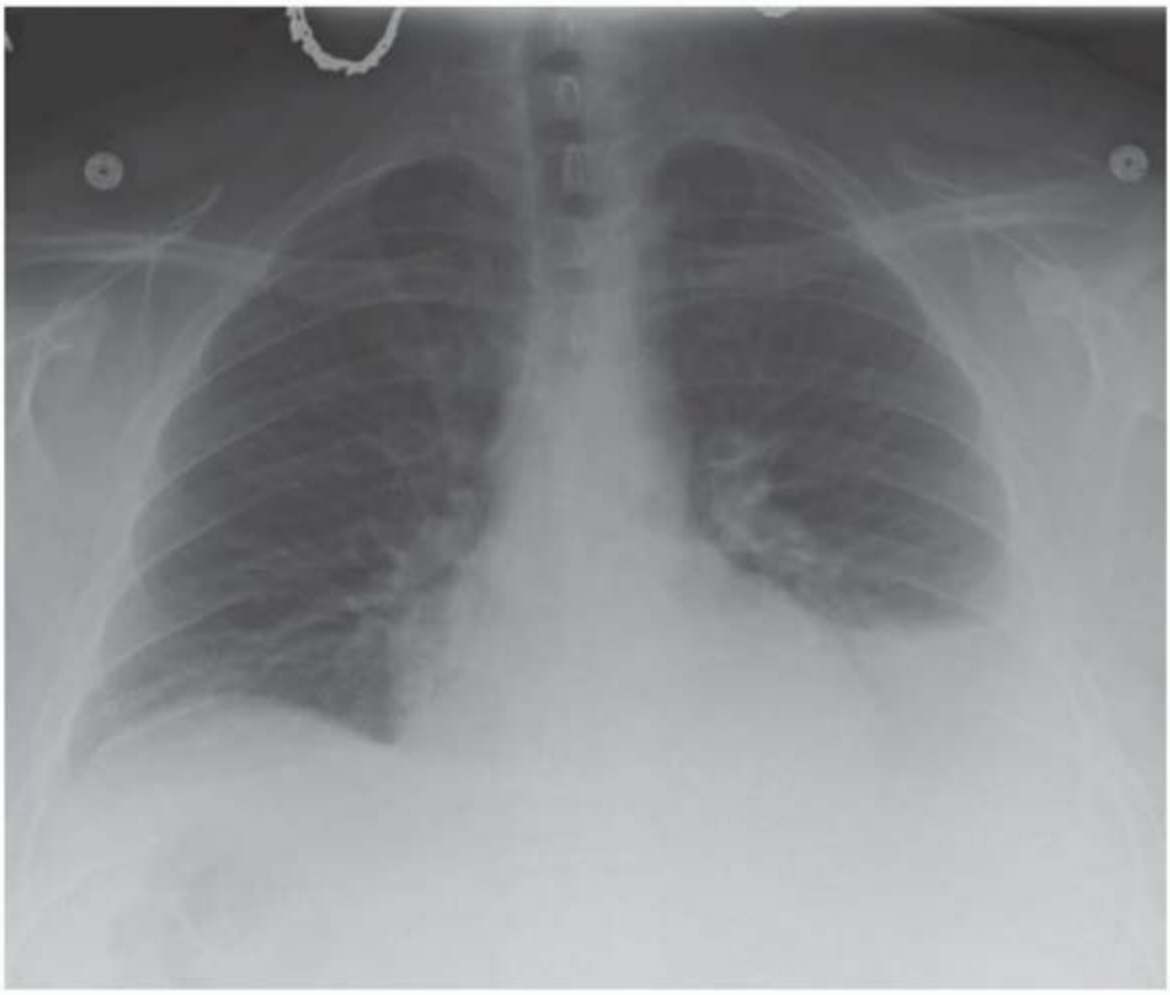


Figure 34-2 Left-sided moderate pleural effusion.

D. Management

1. Transudative pleural effusions are considered a secondary diagnosis; therefore, therapy should be directed at the underlying problem (e.g., congestive heart failure, cirrhosis, or nephrotic syndrome). PleurX indwelling catheters can be used for drainage of recurrent symptomatic effusions (Fig. 34-4).

2. Exudative pleural effusions may be broadly classified based on whether the cause is benign or malignant.

a. Benign exudative effusions are often a result of pneumonia (parapneumonic). They can also be a true empyema (see next section), tuberculous, chylous, or pancreatic reactive effusions. Treatment of parapneumonic and tuberculous effusions is adequate pleural drainage and appropriate antibiotics. Chylothorax can be controlled with diet changes and drainage, pleurodesis, or thoracic duct ligation. A pancreatic reactive effusion usually disappears with resolution of the pancreatitis.

b. Malignant effusions are most often associated with cancers of the breast, lung, and ovary and with lymphoma. Diagnosis is often made by cytology. In the event that cytology is not diagnostic, pleural biopsy may be indicated. Given the overall poor prognosis in these patients, **therapy offered by the thoracic surgeon is generally palliative.**

(1) Drainage of effusion to alleviate dyspnea and improve pulmonary mechanics by reexpanding the lung is performed with a PleurX catheter.

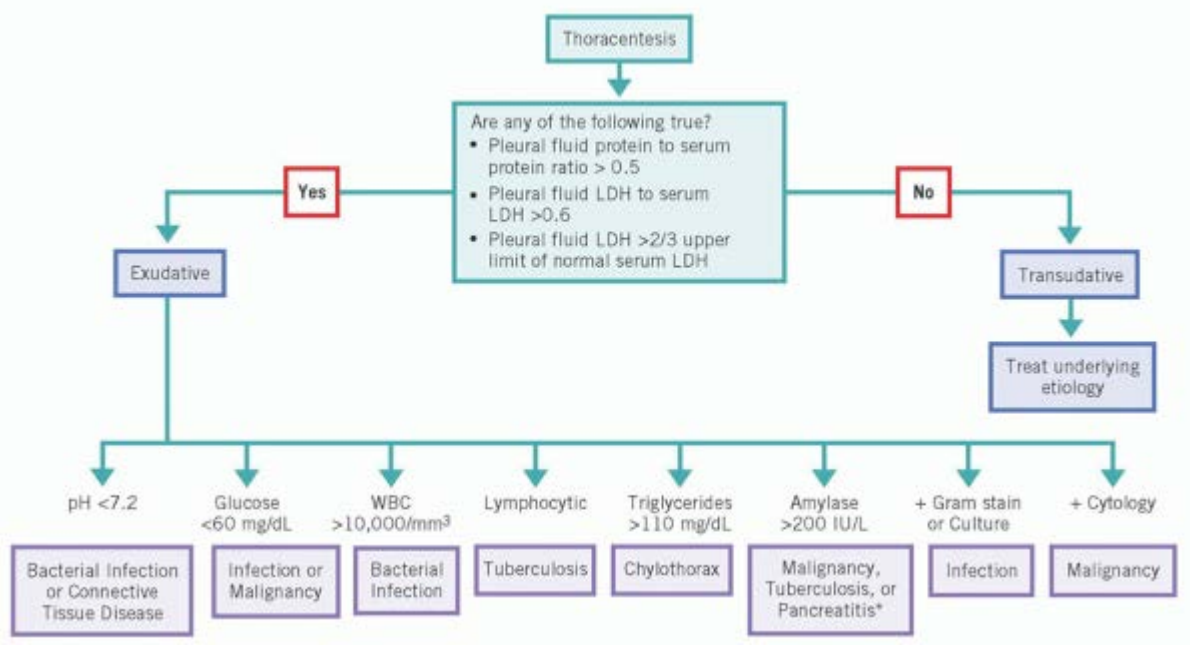


Figure 34-3 Interpreting laboratory results from a thoracentesis. *(*Chest.* 2002:121:470-474).

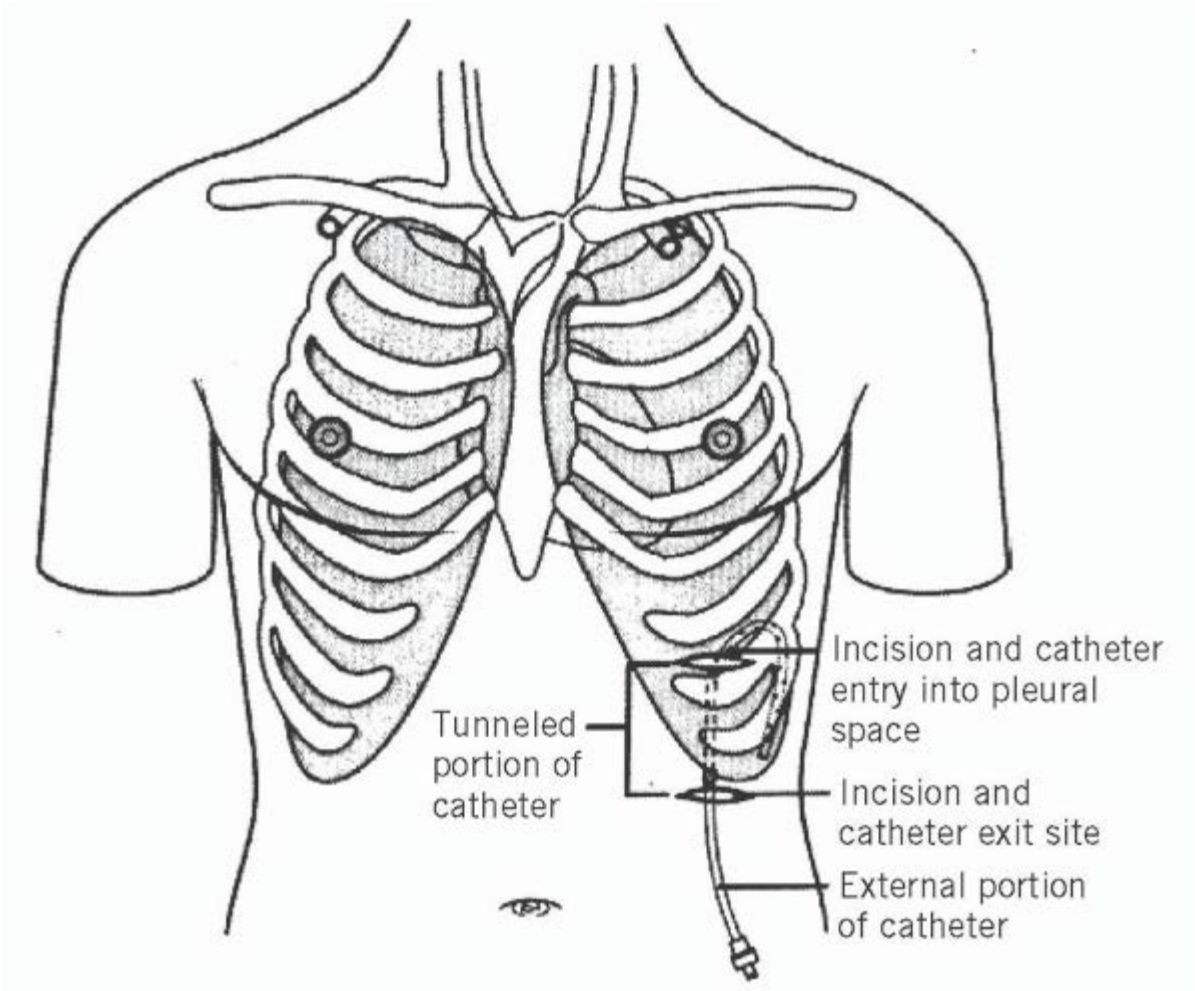


Figure 34-4 PleurX catheter.

(2) Pleurodesis, usually with talc, can prevent reaccumulation of the effusion.

III. EMPYEMA

A. Empyema is a purulent collection in 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. Common Gram-positive bacteria are *Staphylococcus aureus* and *Streptococci,* Gram-negative bacteria are *Escherichia coli, Pseudomonas,* and *Klebsiella,* and anaerobic bacteria are *Bacteroides* species.

B. Presentation of empyema ranges from chronic loculated effusion in a patient with fatigue to systemic sepsis requiring emergent care. Other symptoms include pleuritic chest pain, fever, cough, and dyspnea. Empyemas are diagnosed via thoracentesis and evaluated by CT scan.

C. Management includes control of the infection by appropriate antibiotics, drainage of the pleural space, and obliteration of the empyema space.

1. 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 and may require thoracoscopic drainage. In advanced or **organizing empyema,** the fluid is thicker and a fibrous peel encases the lung. Thoracotomy may be

necessary to free the entrapped lung. If a patient has a persistent fluid collection with an adequately placed tube as evidenced by chest CT, intrapleural fibrinolytic therapy may be useful to break down thin adhesions. Failure of intrapleural fibrinolytics usually requires operative intervention.

2. A **postpneumonectomy empyema** can result from a bronchial stump dehiscence with contamination of the pneumonectomy space. Increasing air in the pneumonectomy space on CXR is often diagnostic. Bronchopleural fistula has a high mortality rate and is managed via drainage of the space, antibiotics, and surgical repair of the fistula. The residual pleural cavity can be obliterated by a muscle transposition, thoracoplasty, or delayed Clagett procedure. **Great caution should be taken in inserting chest tubes into postpneumonectomy empyemas.**

IV. HEMOPTYSIS

A. Hemoptysis is the expectoration of blood and can originate from a number of causes, including infectious, malignant, and cardiac disorders (e.g., bronchitis or tuberculosis, bronchogenic carcinoma, and mitral stenosis, respectively).

B. Workup of hemoptysis includes a focused history and physical examination to assess for symptoms that suggest infection or systemic disease, in conjunction with a CXR.

1. If the CXR is abnormal, appropriate workup and management is indicated.

2. In the setting of a negative CXR and hemoptysis that is likely due to acute bronchitis, observation can be appropriate.

3. For negative CXR and recurrent hemoptysis, a CT scan is warranted to further assess the underlying etiology, and can be used as an adjunct to bronchoscopy to localize and potentially treat the source of bleeding.

C. Massive hemoptysis is a surgical emergency. The first steps are to assess the patient and secure the airway.

1. Bronchoscopy can identify the bleeding side and allow for prompt protection of the

remaining lung parenchyma, either by controlling the area of bleeding or selective ventilation. Bleeding can be controlled by topical or injected vasoconstrictors or by placement of a balloon-tipped catheter in the lobar orifice. Selective ventilation can be achieved with a double-lumen tube or by direct intubation of the contralateral mainstem bronchus.

2. If unable to identify the site of bleeding or after temporizing the bleed, **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 or some lung cancers.

3. Definitive therapy may require thoracotomy with lobar resection or, rarely, pneumonectomy. Rarely, 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.

V. LUNG CANCER

A. Lung cancer is the second most common nonskin malignancy, and is the leading cause of cancer death. An estimated 222,520 cases will be diagnosed and 158,040 patients will die of the disease in 2015 (*www.cancer.org*). Cigarette smoking is the leading risk factor for the development of lung cancer.

B. Pathology. The two main classes of lung tumors are small-cell carcinoma and non-small-cell carcinoma (NSCLC).

1. Small-cell carcinoma accounts for approximately 20% of all lung cancers. It is an aggressive tumor, usually occurs near the hilum, is almost exclusive to 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 less than 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 main subtypes are **adenocarcinoma** (30% to 50% of cases), **squamous cell** (20% to 35%), and **large cell** (4% to 15%). **Bronchioloalveolar carcinoma** is a variant of adenocarcinoma, produces mucin, and can be multifocal. Over the last decade, it has been appreciated that typical carcinoid tumors (grade I neuroendocrine carcinoma), atypical carcinoid tumors (grade II neuroendocrine carcinoma), large-cell carcinoma, and small-cell tumors represent important subgroups of bronchogenic neuroendocrine carcinoma. This may explain the more aggressive behavior of large-cell carcinoma relative to other non-smallcell cancers.

C. Radiographic presentation may occur during a diagnostic evaluation for symptoms or as an incidental finding.

1. Solitary pulmonary nodules are circumscribed lung lesions in an asymptomatic individual,

with lesions greater than 3 cm labeled masses. An estimated 200,000 SPNs are diagnosed each year on CT scans.

2. Radiographic imaging by CT is used to follow lesions and predict outcome. Factors favoring a benign lesion include no growth over a 2-year period, lesion <2 cm, and certain patterns of calcification such as diffuse, centrally located, laminar, or popcornlike calcifications. Malignancy is favored with intravenous contrast enhancement, irregular borders, and eccentric or stippled calcifications (Fig. 34-5).

3. Positron emission tomography (PET) scanning is 95% sensitive and 80% specific in characterizing nodules. However, bronchoalveolar carcinoma and carcinoid tumor can be falsely negative, and inflammatory and infectious processes can be falsely positive. The patient's overall risk factor profile must also be considered. In the setting of low risk (e.g., young age, nonsmoker, favorable CT finding), a negative PET scan has

a high negative predictive value, but the same result in an elderly smoker is less reassuring, and further evaluation is warranted.

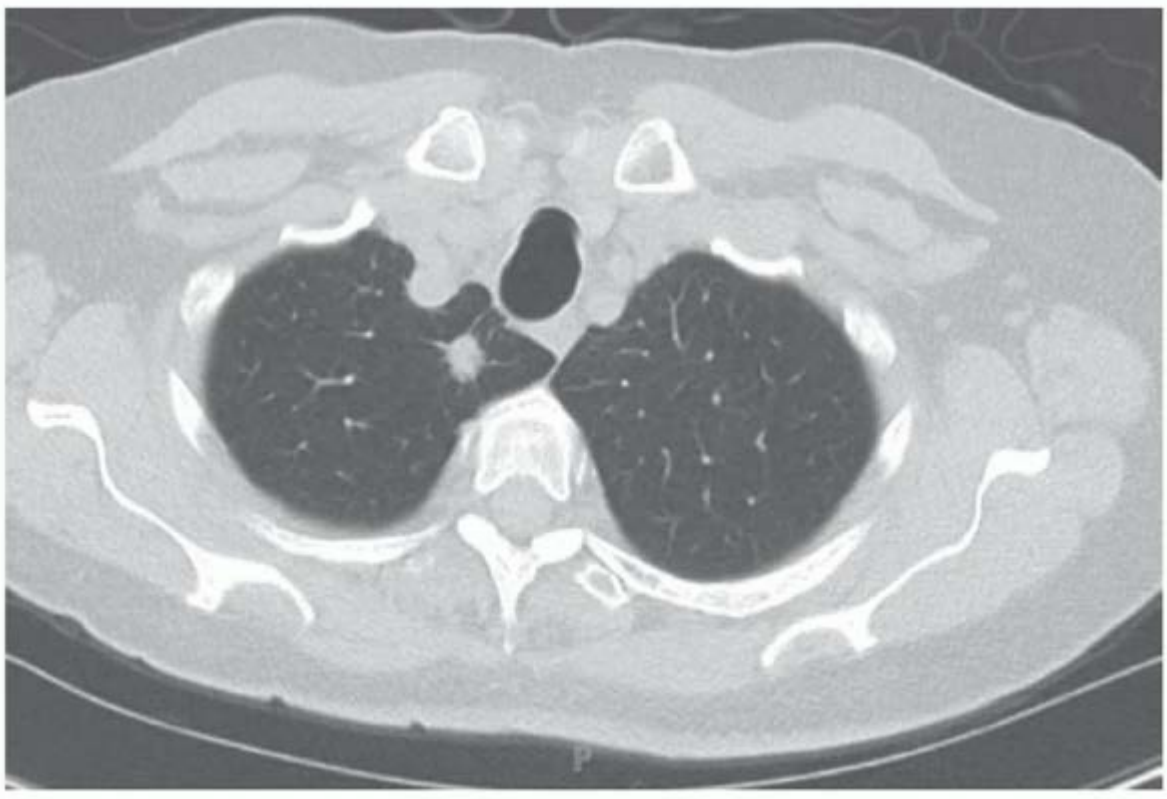


Figure 34-5 Solitary pulmonary nodule in right upper lobe.

4. Tissue biopsy remains the gold standard for diagnosis. Tissue may be obtained by

bronchoscopy in patients with central lung lesions or by CT-guided biopsy. Surgical biopsy by minimally invasive surgical techniques like video thoracoscopy and radial endobronchial ultrasound (EBUS) can provide a definitive diagnosis.

D. Symptomatic presentation of lung cancer implies a more advanced stage and is associated with an overall lower rate of survival.

1. Bronchopulmonary features include cough or a change in a previously stable smoker's cough, increased sputum production, dyspnea, and wheezing. Minor hemoptysis should be investigated with bronchoscopy 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 or phrenic nerve invasion, and superior vena cava syndrome causing facial, neck, and upper-extremity swelling. A Pancoast tumor (superior sulcus tumor) can lead to invasion of the brachial plexus or the cervical sympathetic ganglia, causing an ipsilateral Horner syndrome (ptosis, miosis, and anhidrosis). Rarely, lung cancer can present as dysphagia from compression or invasion of the esophagus by mediastinal nodes or the primary tumor.

3. The most frequent **sites of distant metastases** include the liver, bone, brain, adrenal glands, and the contralateral lung. Symptoms may

include pathologic fractures from bony involvement. Brain metastasis may cause headache, vision changes, or changes in mental status. Adrenal involvement infrequently presents with Addison disease. Lung cancer is the most common tumor causing adrenal dysfunction.

4. Paraneoplastic syndromes are frequent and occur due to release of endocrine substances by tumor cells. They include Cushing syndrome (ACTH secretion in small-cell carcinoma), SIADH, hypercalcemia (PTH-related protein secreted by squamous cell carcinomas), hypertrophic pulmonary osteoarthropathy (clubbed fingers, joint stiffness, and periosteal thickening on XR), and various myopathies.

E. Accurate clinical and pathologic staging is critical in the management of patients with non-small-cell carcinoma. Surgery is the primary therapy for many stage I and II patients and selected stage III patients. The essential elements of staging include evaluation for lymph node involvement and evaluation for distant metastasis. The staging system was most recently modified in 2009 (AJCC 7th edition; Table 34-1).

1. Chest CT to include the upper abdomen examines the size, location, and local involvement of tumor, identifies mediastinal lymphadenopathy, and evaluates for liver and adrenal metastasis. The sensitivity for identifying metastatic lymph nodes by CT is 65% to 80% and the specificity is only 65%.

2. PET imaging is often used to stage patients with NSCLC, but its accuracy for detecting primary tumors and metastatic disease may be limited by the presence of inflammation and ongoing infection. In regions endemic for inflammatory processes such as tuberculosis and histoplasmosis, the usefulness of PET imaging for investigating mediastinal lymph nodes is limited. However, it can be useful for identifying occult distant metastatic disease to the liver, adrenals, and bone.

3. Lymph node staging of the mediastinum is done using either **EBUS**guided fine needle aspiration or **mediastinoscopy.** The pretracheal, paratracheal, and subcarinal lymph nodes can be easily accessed by these techniques. Aortopulmonary nodes can be sampled via endoscopic (transesophageal) ultrasound (EUS), VATS, or, less commonly, anterior mediastinoscopy (Chamberlain procedure). Routine use of these techniques in the staging of patients with NSCLC should be favored, with the exception of select patients with clinical stage I lung cancer staged by CT and PET with no abnormal lymphadenopathy (*J Thorac Cardiovasc Surg.* 2006;131:822-829). The timing of mediastinoscopy, whether before or during a planned resection, depends on surgeon preference and the availability of accurate pathologic evaluation of mediastinal lymph node frozen sections.

4. CT or magnetic resonance (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 of asymptomatic patients.

5. Bone scan is sometimes obtained in patients with specific symptoms of skeletal pain and selectively as part of the general preoperative metastatic workup.

TABLE 34-1 American Joint Committee on Cancer Staging System of Lung Cancer

Tumor Status (T)

T1a	²2 cm
T1b	>2-3 cm
	No invasion of visceral pleura or more proximal than lobar bronchus
T2a	>3-5 cm
T2b	>5-7 cm
	Involvement of bronchus ³2 cm distal to the carina
	Invasion of visceral pleura

	Associated atelectasis or obstructive pneumonitis not involving entire lung
T3	>7 cm or tumor with any of the following characteristics: Invasion of chest wall, diaphragm, phrenic nerve Invasion of mediastinal pleura or parietal pericardium Associated atelectasis or obstructive pneumonitis of entire lung Tumor within main bronchus <2 cm from carina but does not involve carina Satellite nodules in the same lobe
T4	Tumor with any of the following characteristics: Mediastinal invasion Invasion of heart of great vessels Invasion of carina, trachea, esophagus, or recurrent laryngeal nerve Invasion of vertebral body Separate tumor nodules in a different but ipsilateral lobe

Nodal Involvement (N)

N0	None
N1	Hilar, interlobar, or peripheral lymph node zones
N2	Ipsilateral mediastinal lymph nodes, subcarinal, or aortopulmonary lymph nodes
N3	Contralateral mediastinal, hilar, or aortopulmonary lymph nodes; ipsilateral or contralateral scalene or supraclavicular lymph nodes

Distant Metastases (M)

M0	None
M1	Distant metastases present

Stage	**5-yr Survival**

Ia	T1a/T1b N0 M0	50-80%
Ib	T2a N0 M0	47%
IIa	T1a/T1b, N1 M0 T2a N1 M0 T2b N0 M0	36%
IIb	T2b N1 M0 T3 N0 M0	26%
IIIa	T1/T2 N2M0 T3 N1/N2 M0 T4 N0/N1 M0	19%
IIIb	T4 N2 M0 Any T with N3 M0	7%
IV	Any T, any N, M1	2%

6. 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 for excluding synchronous lung cancers (found in approximately 1% of patients) prior to resection.

F. Management of lung cancer includes a combination of surgery, chemotherapy, and radiation.

1. Stage I disease is generally treated with surgical resection alone. Stage II cancers are also treated with surgery, but chemotherapy increases 5-year survival and is recommended in patients with stage II and stage III disease. Adjuvant radiation therapy is considered in patients with close surgical margins. Certain patients with stage IIIA disease appear to benefit from surgical resection alone (T3N1M0). Selected patients with mediastinal lymph node metastasis (N2 disease) may be candidates for surgical resection after neoadjuvant chemoradiation therapy. Patients with

bulky, diffuse mediastinal lymphadenopathy are typically treated using definitive chemoradiation. Stage IV tumors have distant metastases and are considered unresectable, though some patients with node-negative lung cancer and a solitary brain metastasis have achieved long-term survival with combined resection.

2. Operative principles. In the patient able to tolerate any resection (see **Section IX.**A), the minimum extent of resection is usually an anatomic lobectomy. Even in stage I disease, a wedge resection results in a threefold higher local recurrence rate and a decreased overall and disease-free survival. Patients with limited pulmonary reserve may be treated by segmental or wedge resection. Most centers report operative mortality of less than 2% with lobectomy and 6% with

pneumonectomy. VATS has become a widespread technique for lung resections.

3. Stereotactic body radiation therapy (SBRT) is external beam radiation therapy that precisely delivers high-dose radiation to a target in the body over one or more treatments. SBRT has been shown to be effective for early stage lung cancer in patients that are poor candidates for surgical resection.

G. Five-year survival rates are summarized in Table 34-1.

VI. TUMORS OF THE PLEURA

A. The most common tumor of the pleura is **mesothelioma.** Malignant mesothelioma is a rare and aggressive cancer that has been linked to asbestos exposure with a latency period of decades.

1. Patient presentation varies, but can include chest pain, malaise, cough, weakness, weight loss, and shortness of breath with pleural effusions. One-third of patients report paraneoplastic symptoms of osteoarthropathy, hypoglycemia, and fever.

2. Tissue for diagnosis can be obtained via cytology of pleural fluid, needle biopsy of the pleura, or biopsy via thoracoscopy. The most favorable subtype is epithelioid (50%), followed by sarcomatoid (20%), mixed, and desmoplastic.

3. CT scans can differentiate pleural from parenchymal disease. Routine use of MRI is not recommended, while PET scan is used to identify distant metastatic disease (*J Thorac Cardiovasc Surg.* 2003;126: 11-15).

4. Treatment consists of a multimodal therapy comprising surgery, chemotherapy, and radiation. Surgical options include extrapleural pneumonectomy (EPP) or pleurectomy/decortication. For early stage cases, EPP may offer the best chance of cure. The best reported 5-year survival following completion of the multimodal therapy in patients without nodal metastasis is 53% but survival is typically lower than that (*J Clin Oncol.* 2009;27:1413-1418).

B. Less common tumors include lipomas, angiomas, soft-tissue sarcomas, and fibrous histiocytomas.

VII. TUMORS OF THE MEDIASTINUM.

The location of a mass in relation to the heart helps the surgeon to form a differential diagnosis (Table 34-2). On the lateral CXR, the mediastinum is divided into thirds, with the heart comprising the middle segment.

A. Presentation of mediastinal tumors vary, and symptoms are only present in one-third of patients. Symptoms are often nonspecific and include dyspnea, cough, hoarseness, vague chest pain, and fever.

B. Radiographic evaluation includes CXR, which should be followed by a CT scan to further delineate the anatomy. Malignant germ cell tumors are further evaluated with abdominal CT and scrotal ultrasound.

TABLE 34-2 Differential Diagnosis of Tumors Located in the Mediastinum

Anterior		Middle	Posterior
Thymoma	Lymphoma	Congenital cyst	Neurogenic
Germ cell	Parathyroid	Lymphoma	Lymphoma
Teratoma	Lipoma	Primary cardiac	Mesenchymal
Seminoma	Fibroma	Neural crest	
Nonseminoma	Lymphangioma		
Aberrant thyroid			

Modified from Young RM, Kernstine KH, Corson JD. Miscellaneous cardiopulmonary conditions. In: Corson JD, Williamson RCN, eds. *Surgery.* Philadelphia: Mosby, 2001.

C. Anterior mediastinal masses are represented by the ÒTerrible TsÓ: Thymoma,

teratomas/germ cell tumors, ÒterribleÓ lymphoma, or thyroid tumors.

1. Thymomas are malignant in 15% of cases, and are staged by the Masaoka system (Table 34-3). Approximately 50% of patients with a thymoma have paraneoplastic syndromes, including myasthenia gravis (MG), hypogammaglobulinemia, and red cell aplasia.

a. The thymus gland plays a role in **MG** by generating autoreactive antibodies against the acetylcholinesterase receptor. Roughly 15% of MG patients have a thymoma, though 80% of cases demonstrate complete or partial response to thymectomy.

TABLE 34-3 Masaoka Staging System of Thymomas and 5-year Survival

Stage		5-yr Survival
Stage I	Macroscopic complete encapsulation and no microscopic capsular invasion	100%
Stage II	II-A Microscopic invasion into surrounding fatty tissue or mediastinal pleura II-B Macroscopic invasion into the capsule	98.4%
Stage III	Macroscopic invasion into neighboring organs	88.7%
Stage IV	IV-A Pleural or pericardial implants IV-B Lymphogenous or hematogenous metastasis	70.6% 52.8%

From Kondo K, Monden Y. Therapy for thymic epithelial tumors: a clinical study of 1,320 patients from Japan. *Ann Thorac Surg.* 2003;76(3):878-884.

b. Preoperative preparation for patients with MG includes decreasing corticosteroids and weaning of anticholinesterases. Plasmapheresis can be performed preoperatively to aid in this. Muscle relaxants and atropine should be avoided during anesthesia.

c. Operative approach includes transcervical thymectomy, median sternotomy, VATS resection, and robotic resection. For more advanced stages of thymoma, neoadjuvant chemotherapy may be indicated.

2. Biochemical evaluation with β-human chorionic gonadotropin (β-HCG) and α-fetoprotein (AFP) if the lesion is suspected to be germ cell tumors.

a. Teratomas are usually benign and often contain ectodermal components such as hair, teeth, and bone. Elevation of both β-HCG and AFP is very rare, and suggests a malignant teratoma. Treatment is surgical resection.

b. Seminomas do not have elevated AFP, and fewer than 10% present have elevated β-HCG. Their treatment is primarily nonsurgical (radiation and chemotherapy), except in the case of localized disease.

c. Nonseminomatous germ cell tumors present with an elevation of both tumor markers. The treatment is with platinum-based chemotherapy, with resection of residual masses after definitive chemotherapy.

3. Tissue diagnosis is often crucial for the diagnosis and treatment of **lymphoma.** Treatment is primarily nonsurgical. Cervical lymph node biopsy, CT-guided biopsy, or mediastinoscopy with biopsy may be required. These lesions often present as irregular masses on CT scan.

D. Bronchogenic and enteric cysts present as **middle mediastinal masses,** and should be resected for tissue diagnosis and due to risk of future infection or malignant degeneration.

E. Patients with **posterior mediastinal masses** or paravertebral masses should have their catecholamine levels measured to rule out **pheochromocytomas.**

VIII. LUNG TRANSPLANTATION

A. Lung transplantation has been increasing steadily over the last decade, with the leading indications for lung transplant being chronic obstructive pulmonary disease (COPD), idiopathic pulmonary fibrosis (IPF), and CF. Other common indications include α_1-antitrypsin deficiency (alpha-1) and pulmonary arterial hypertension (PAH).

B. COPD is a long-term consequence of smoking and is the result of nonuniform destruction of lung parenchyma. As lung tissue loses its elastic recoil, the areas of destruction expand. This expansion of diseased areas, in combination with inflammation, leads to poor ventilation of relatively normal lung.

1. This leads to the typical findings of **hyperexpanded lungs** on CXR: Flattened diaphragms, widened intercostal spaces, and horizontal ribs. On pulmonary function testing, patients present with increased residual volumes and decreased FEV_1 (forced expiratory volume in 1 second). Surgical treatment is generally reserved for the symptomatic patient who has failed maximal

medical treatment.

2. The **goals of resectional surgery** for COPD are to remove diseased areas of lung and allow improved function of the remaining lung tissue. Previously, bullectomy or lung volume reduction surgery were more commonly offered; however, currently lung transplantation is the most common surgical approach.

C. Both **single lung** and **bilateral lung transplants** are options. Nationally, the number of single lung transplants is steady, while the number of double lung transplants is increasing annually. Single lung transplants offer greater use of donor organs, shorter ischemic time, and allow for the ability to replace the other native lung if rejection develops. Bilateral lung transplants allow for more pulmonary reserve and the ability to use more marginal donor lungs. Our institution favors bilateral lung transplant as patients have improved long-term survival (Fig. 34-6). The only absolute indication for bilateral lung transplantation is CF because single-lung transplantation would leave a chronically infected native lung in an immunocompromised patient.

D. Current problems with lung transplantation include donor shortage, primary graft dysfunction, and chronic rejection. Long-term, chronic allograft dysfunction in the form of bronchiolitis obliterans occurs in 50% of patients.

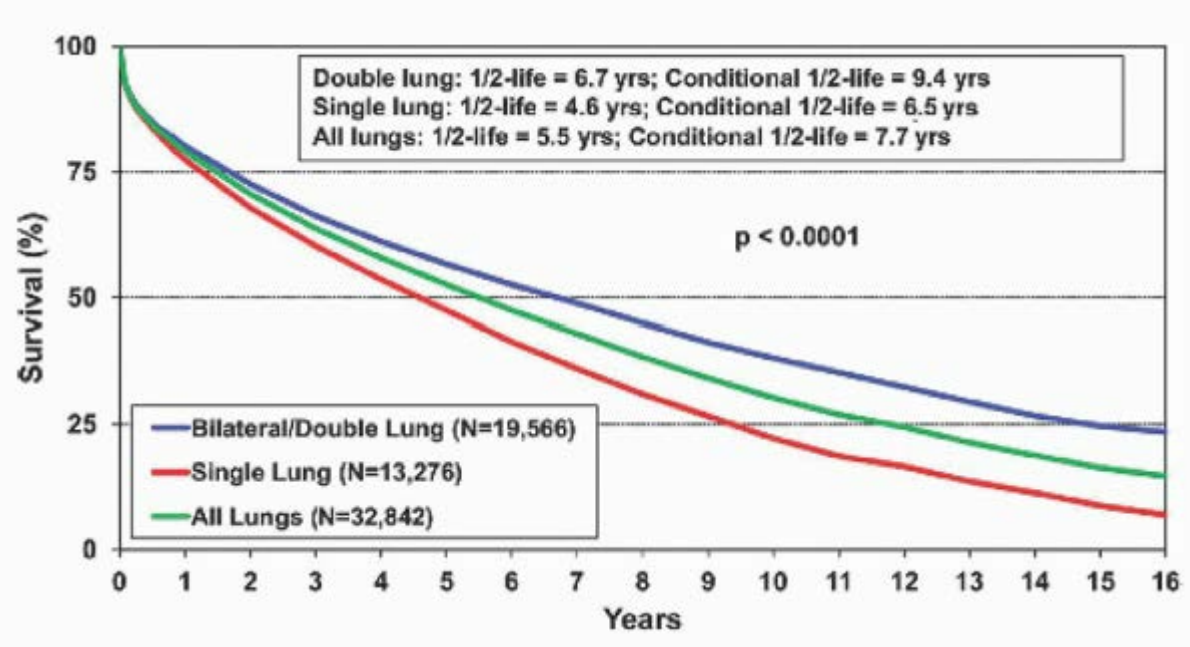


Figure 34-6 Chart showing lung transplant graft survival of bilateral, single, and all lung transplants from Jan 1994 to June 2010 and by diagnosis from Jan 1990 to June 2010. Conditional half-life is the 50% survival time for recipients who were alive 1 year after lung transplants. (From Christie JD, Edwards LB, Kucheryavaya AY, et al.; International Society of Heart and Lung Transplantation. The Registry of the International Society for Heart and Lung Transplantation: 29th adult lung and heart-lung transplant reportÑ2012. *J Heart Lung*

Transplant. 2012;31(10):1052-1064, with permission.)

IX. MANAGEMENT OF THORACIC SURGICAL PATIENTS

A. Preoperative assessment of pulmonary function and estimation of postoperative pulmonary assessment is the most critical factor in planning lung resection.

1. Pulmonary function tests (PFTs), which include FEV_1, and **arterial blood gas (ABG)** are used to assess risk of postoperative pulmonary failure (Fig. 34-7). **Diffusion capacity (DLCO)** and **cardiopulmonary exercise testing (CPET)** are indicated in patients with marginal function for accurate assessment of postoperative function. In general, average risk for pulmonary resection is associated with a 1% to 2% mortality.

Preoperative hypercapnia (arterial carbon dioxide tension >45 mm Hg) may preclude resection.

2. Evaluation of cardiac disease starts with a detailed history and physical examination to elicit symptoms of ischemia and a baseline EKG. Abnormal findings should be pursued with stress tests or coronary catheterization.

3. Smoking cessation preoperatively for as little as 2 weeks can aid in the regeneration of the mucociliary function and has been associated with fewer postoperative respiratory complications.

B. Postoperative care of the thoracic surgery patient focuses on pain control, fluid management, and maintenance of pulmonary function.

1. The **thoracotomy incision is one of the most painful and debilitating** in surgery. Inadequate pain control contributes heavily to 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 during the early recovery period significantly improves pain management. Other effective analgesic maneuvers include intercostal blocks with long-acting local anesthetic before closure of the chest and subpleural administration of local anesthetic via catheters placed adjacent to intercostal nerves at the time of thoracotomy or thoracoscopy.

2. Perioperative fluid management of thoracic surgery patients differs from that of patients after abdominal surgery. Pulmonary surgery does not induce large fluid shifts, while collapse and reexpansion of lungs during surgery can lead to pulmonary edema. Judicious fluid management to avoid fluid overload and pulmonary edema is critical in patients with limited pulmonary reserve. Discussions regarding 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 α-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.

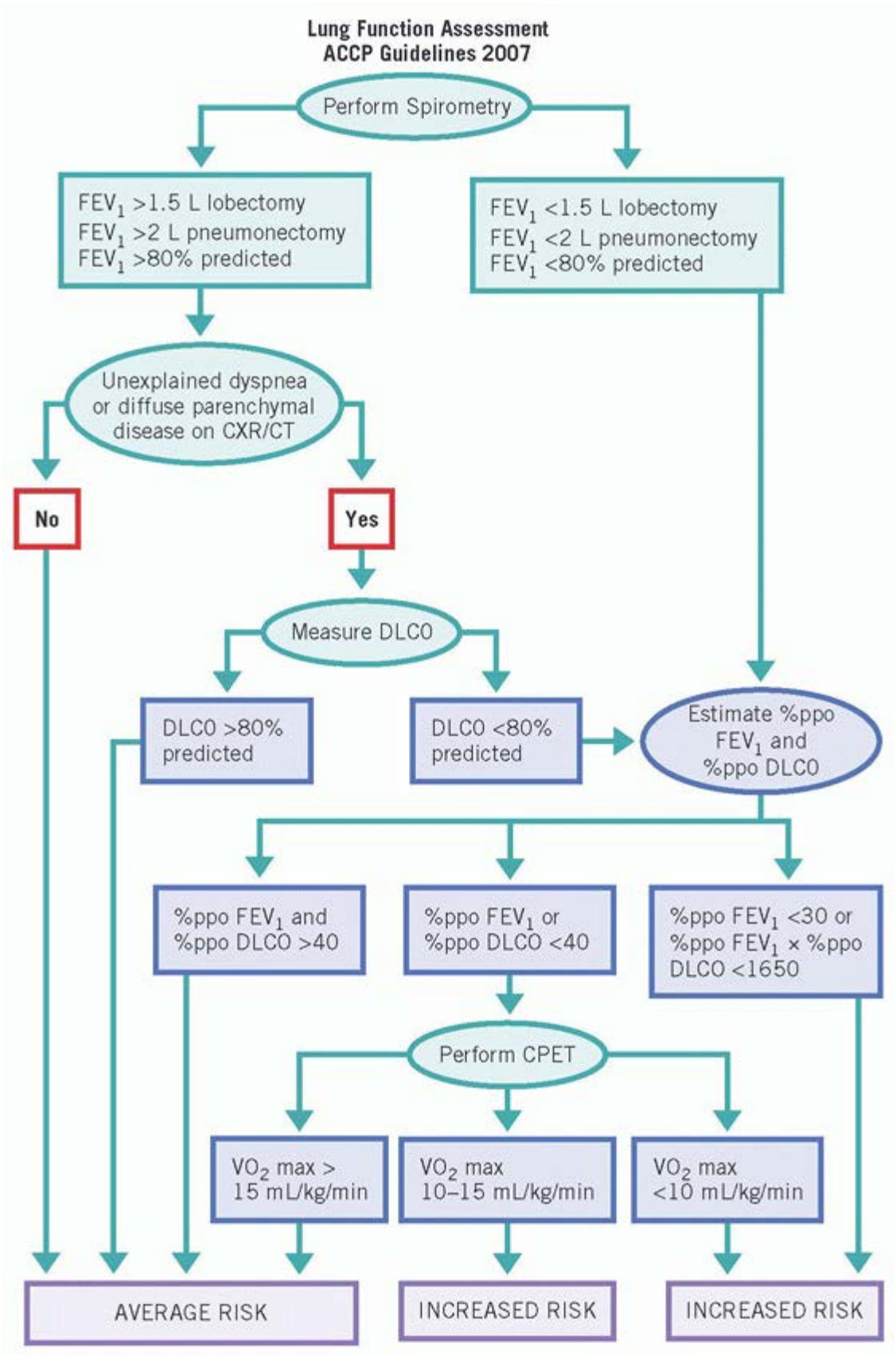


Figure 34-7 Preoperative lung function assessment algorithm. (From Colice GL et al. Physiologic evaluation of the patient with lung cancer being considered for resectional surgery: ACCP evidenced-based clinical practice guidelines (2nd edition). *Chest.* 2007;132(3

Suppl): 161S-177S, with permission.)

3. 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. Adequate analgesia along with aggressive pulmonary toilet, incentive spirometry, and chest physiotherapy delivered by the respiratory therapist are essential. Occasionally nasotracheal suctioning or bedside flexible bronchoscopy can be indicated for bronchial toilet.

4. Cardiovascular complications are common 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. Cardiac arrhythmias occur in up to 30% of patients undergoing pulmonary surgery. The most common arrhythmia is atrial fibrillation. The highest incidence occurs in elderly patients undergoing pneumonectomy or intrapericardial pulmonary artery ligation.

b. Treatment of any rhythm disturbance begins with an assessment of the patient's hemodynamic status. Manifestations of these arrhythmias vary in acuity from palpitations to hemodynamic collapse. If the patient is hemodynamically unstable, the advanced cardiac life support protocol should be followed. After the patient has been examined and hemodynamic stability confirmed, an ECG, ABG sample, and serum electrolyte panel should be obtained. Frequently, supplementary oxygen and aggressive potassium and magnesium replenishment are the only treatment necessary. They should be treated expediently with electrolyte correction, optimization of oxygenation, and evaluation for cardiac ischemia.

CHAPTER 34: LUNG AND MEDIASTINAL DISEASES

Multiple Choice Questions

1. Which of the following patients can be initially managed by observation?

a. A 45-year-old pilot with a small pneumothorax on room air.

b. A 62-year-old teacher with a small pneumothorax on room air.

c. An 18-year-old college student with a recurrent small pneumothorax on room air.

d. A 31-year-old police officer with a small pneumothorax and shortness of breath on room air.

View Answer

2. A patient with COPD has acute respiratory failure from pneumonia and is intubated in the ICU. The nurse calls you to inform you that the patient's oxygen requirement has increased, in addition to his blood pressure slowly trending down, with his MAP going from 70 to 60 over the last hour. On physical examination, you notice significantly decreased breath sounds on the right. The next step is:

a. Obtain a CXR

b. Needle decompression

c. Percutaneous catheter placement

d. Chest tube placement

View Answer

3. A 50-year-old female with hepatitis C cirrhosis has a recurrent rightsided pleural effusion that is being managed with drainage from a PleurX catheter. His serum laboratory results are: Protein 6.8, LDH 100, amylase 20, WBC 9,000. Which of the following laboratory results would be expected in this effusion?

a. Pleural fluid protein is 2.0

b. Pleural fluid LDH is 75

c. Pleural amylase is 250

d. Pleural WBC is 12,000

View Answer

4. A 35-year-old male presents with an incidentally found mediastinal mass on CXR. Further workup with a CT scan demonstrates an anterior mediastinal mass. AFP and β-HCG are both elevated. Treatment of this mass is:

a. Observation as he is asymptomatic

b. Radiation and chemotherapy without surgery

c. Surgical resection

d. Chemotherapy followed by surgery

View Answer

5. A 70-year-old patient presents to the ER after a motor vehicle collision. In the ER, he undergoes a chest CT that demonstrates an incidentally found 3 cm peripheral mass in the RUL. What is the next step in management?

a. Observation with repeat CT in 6 to 12 months

b. PET scan

c. Navigational bronchoscopy

d. Wedge resection

View Answer

6. A 65-year-old patient had a chronic cough with a 40 pack year smoking history. CXR demonstrated a RLL mass with staging by PET/CT concerning for a T1, N2, M0 lesion. A percutaneous biopsy indicates that the patient has lung adenocarcinoma. What is the appropriate management?

a. Mediastinoscopy or EBUS

b. Surgical resection alone

c. Surgical resection followed by chemotherapy

d. Neoadjuvant chemotherapy

View Answer

7. Which of the following lung function evaluations has the highest risk for undergoing a lobectomy?

a. FEV_1 80% predicted, DLCO 40% predicted

b. FEV_1 40% predicted, DLCO 70% predicted

c. FEV_1 20% predicted, VO_2 max 12 mL/kg/minute

d. FEV_1 30% predicted, VO_2 max 15 mL/kg/minute

View Answer

35
Breast

Oluwadamilola M. Fayanju

Julie A. Margenthaler

ANATOMY

I. THE BREAST.

Located between the subcutaneous fat and the fascia of the **pectoralis major** and **serratus anterior** muscles, it is bounded superiorly by the second/third rib, inferiorly by the inframammary fold, and medially by the lateral edge of the sternum, with its lateral border lying between the anterior and midaxillary lines. Suspensory ligaments **(Cooper ligaments)** running through the breasts from the deep fascia to the skin may cause skin dimpling when associated with a malignancy.

A. Vasculature. Arterial supply is predominantly **internal thoracic artery (or internal mammary artery)** via perforating branches **(perforators).** Venous drainage is mainly to the **axillary vein.**

B. Lymphatic Drainage. The superficial **Sappey plexus** converges with a deep lymphatic plexus, and they ultimately drain into the axillary (75%) and internal mammary lymph nodes.

C. Innervation. Lateral and anterior cutaneous branches of the second to sixth intercostal nerves provide sensory innervation.

II. THE AXILLA.

The borders of the axilla are defined as the **axillary vein** superiorly, **latissimus dorsi** laterally, and the **serratus anterior** muscle medially.

A. Axillary lymph nodes are classified according to their anatomic location relative to the **pectoralis minor** muscle.

1. Level I nodes: *Lateral* to the pectoralis minor muscle.

2. Level II nodes: *Posterior* to the pectoralis minor muscle.

3. Level III nodes: *Medial* to the pectoralis minor muscle and most accessible with division of the muscle.

4. Rotter nodes: *Between* the pectoralis major and the minor muscles.

B. Axillary Nerves. Three motor and several sensory nerves are located in the axilla. Preservation of all is preferred during an axillary lymph node dissection (ALND); however, direct tumor invasion may require resection along with the specimen.

1. Long thoracic nerve travels superior to inferior along chest wall and medially within the axilla, innervating the **serratus anterior muscle.** Injury to this nerve causes a ÒwingedÓ scapula.

2. Thoracodorsal nerve courses along the posterior border of the axilla superior to inferior on the subscapularis muscle and innervates the

latissimus dorsi. Injury to this nerve causes weakness in arm adduction and medial rotation.

3. Medial pectoral nerve travels from the posterior aspect of the pectoralis minor muscle around the lateral border of the pectoralis minor to the posterior aspect of the pectoralis major muscle. It innervates the lateral third of the pectoralis major; injury to this nerve results in atrophy of the lateral pectoralis major muscle.

4. Intercostal brachial sensory nerves travel laterally in the axilla from the second intercostal space to the medial upper arm. Transection causes numbness in the posterior and medial surfaces of the upper arm.

CLINICAL ASSESSMENT

I. HISTORY.

Patients seek medical attention most commonly for an abnormal mammogram, a breast mass, breast pain, nipple discharge, or skin changes. History should include:

- Description and duration of signs and symptoms and their temporal relationship to pregnancy, menstrual cycle, or previous trauma.
- Date of last menstrual period and regularity of the menstrual cycle.
- Age of menarche.
- Number of pregnancies and age at first full-term pregnancy.
- Lactational history.
- Age at natural or surgical menopause (i.e., oophorectomy).
- Previous history of breast biopsies and mammoplasties.
- Mammogram history.
- History of oral contraceptive and/or hormone replacement therapy (HRT).
- Personal and family history of breast and gynecologic cancer, including age at diagnosis. This

should include at least two generations as well as any associated cancers, such as ovary, colon, prostate, gastric, or pancreatic.

A. Assessment of Cancer Risks

1. Risk factors. Hormonal and environmental exposures, genetics, and certain types of breast tissue histology can all be associated with an increased risk for breast cancer (Table 35-1).

2. Hereditary breast cancer. Approximately 5% to 10% of breast cancers are hereditary, that is, can be attributed to a mutation in a single, highly penetrant gene, and approximately 80% of hereditary breast cancers are the result of mutations in **BRCA1** and **BRCA2.** Women with BRCA1 mutations have an estimated risk of 85% for breast cancer by the age of 70 years, a 50% chance of developing a second primary breast cancer, and a 20% to 40% chance of developing ovarian cancer. BRCA2 mutations carry a slightly lower risk for breast and ovarian cancer and account for 4% to 6% of all male breast cancers. Screening for BRCA gene mutations should be reserved for women who have a strong family history of breast or ovarian cancer, and referral for genetic counseling should be based on the National Comprehensive Cancer Network (NCCN) guidelines (*J Natl Compr Canc Netw.* 2014;12:1326-1338).

TABLE 35-1 Risk Factors for Breast Cancer and Approximate Strength of Association

Reproductive	Hormonal	Nutritional/Lifestyle/Body Habitus	Other
Early menarche [+]	OC use (current vs. none) [+]	Obesity (>30 BMI vs. <25) Premenopausal [-] Postmenopausal [+]	Family history (mother and sister)[a] [+++]
Age at first birth (>35 vs. <20) [++]	Estrogen replacement (10+ yr vs. none) [+]	Adult weight gain (postmenopausal) [++]	Family history (first-degree relative)[b] [++]
No. of births (0 vs. 1 child) [+]	Estrogen plus progesterone replacement (>5 yrs vs. none)	Alcohol (1 or more drink/day vs. none) [+]	Jewish heritage (yes vs. no) [+]

	[++]		
Age at menopause (5-yr increment) [+]	High blood estrogens or androgens (postmenopause) [+++]	Height (>5 ft 7 in) [+]	Ionizing radiation (yes vs. no) [+]
Breast-feeding (>1 yr vs. none) [-]	High blood prolactin [++]	Physical activity (>3 hrs/wk) [-] Monounsaturated fat[c] (vs. saturated fat) [-] Low intake of fruits and vegetables[c] (specifically for ER-breast cancer) [+]	Benign breast disease (MD diagnosed)[d] [++] Mammographic density (highest category vs. lowest) [+++]

[a] Two first-degree relatives who have a history of breast cancer before age 65 years versus no relative.

[b] First-degree relative who has a history of breast cancer before age 65 years versus no relative.

[c] Upper quartile (top 25%) versus lower quartile (lowest 25%).

[d] Clinically recognized chronic cystic, fibrocystic, or other benign breast disease versus none.

BMI, body mass index; OC, oral contraceptives;
[+] = relative risk (RR) 1.1-1.4; [++] = RR 1.5-2.9; [+++] = RR 3.0-6.9; [-] = RR 0.7-0.8.
Adapted with permission from Table 18.2 in Chapter 18 : Nongenetic Factors in the Causation of Breast Cancer, *Diseases of the Breast,* LWW 2014.

3. Modeling breast cancer risk. The **Gail model** is one of several prediction modelsÑincluding the BOADICEA, BRCAPRO, Claus, and Tyrer-Cuzick, modelsÑthat estimate the absolute risk (probability) that a woman in a program of annual screening will develop breast cancer over a defined age interval (*J Nat Cancer Inst.* 1989; 81:1879-1886). A modified Gail model focusing only on the risk of invasive cancer has been used to define eligibility criteria for entry into chemoprevention trials. The National Surgical Adjuvant Breast and Bowel Project (NSABP) and the

National Cancer Institute (NCI) offer an interactive online risk assessment tool (http://www.cancer.gov/bcrisktool). In addition, a number of genome-based prediction tools have also been developedÑthe most widely used being **Oncotype Dx**Ñin order to help predict who is at risk for malignancy and who would benefit from chemotherapy.

II. PHYSICAL EXAMINATION.

Inspect the patient's breasts in both the **upright** and **supine** positions. With the patient in the upright position, examine with the patient's arms relaxed and then raised, looking for shape asymmetry, deformity, skin changes (erythema, edema, dimpling), nipple changes or discharge, and lymphadenopathy (axillary, supraclavicular, and infraclavicular). With the patient in the supine position, examine the entire breast systematically with the patient's ipsilateral arm raised above and behind the head.

III. BREAST IMAGING

A. Screening mammograms are performed in the **asymptomatic** patient, and consist of two standard views, mediolateral oblique (MLO) and craniocaudal (CC). **Tomosynthesis** became available in the United States in 2011 and improves the sensitivity and specificity of mammography, particularly for women with nonfatty breasts and in the assessment of noncalcified lesions. Although the United States Preventative Task Force (USPTF) changed its screening recommendation in 2009 to annually for women beginning at age 50, the current recommendation from the NCI, the American Congress of Obstetricians and Gynecologists (ACOG), and the American Cancer Society (ACS) is **annual screening mammography for women aged 40 years and older,** and we agree with these societies' recommendation. Breast lesions on mammograms are classified according to the American College of Radiology by **BI-RADS (Breast Imaging Reporting and Database System)** scores:

- **0** = Needs further imaging; assessment incomplete.
- **1** = Normal; continue annual followup (risk of malignancy: 1/2,000).
- **2** = Benign lesion; no risk of malignancy; continue annual followup (risk of malignancy: 1/2,000).
- **3** = Probably benign lesion; needs 4 to 6 months followup (risk of malignancy: 1% to 2%).
- **4** = Suspicious for breast cancer; biopsy recommended. This category can be further subdivided into
  - 4A = Low suspicion for malignancy (>2% to ² 10%)

  - 4B = Moderate suspicion for malignancy (>10% to ² 50%)
  - 4C = Finding of moderate concern of being cancer, but not as high as Category 5 (>50% to

²95%)

- 5 = Highly suspicious for breast cancer; biopsy strongly recommended (³95% are malignant).
- 6 = Known biopsy-proven malignancy.

1. Malignant mammographic findings

a. New or spiculated masses.

b. Clustered microcalcifications in linear or branching array.

c. Architectural distortion.

2. Benign mammographic findings that might be mistaken for malignancy include **radial scar** (biopsy needed), **fat necrosis** (i.e., oil cysts; biopsy may be needed), and **milk of calcium** (no biopsy needed). **Cysts** cannot be distinguished from solid masses by mammography; **ultrasound** is needed to make this distinction.

3. Screening in high-risk patients. For patients with *known BRCA mutations,* annual mammograms and semiannual physical examinations should **begin at the age of 25 to 30 years.** In patients with a *strong family history of breast cancer but undocumented genetic mutation,* annual mammograms and semiannual physical examinations should begin **10 years earlier than the age at diagnosis of the youngest affected relative and no later than the age of 40 years.**

4. Magnetic resonance imaging (MRI) is recommended for screening in *select* **high-risk patients** including those with an elevated lifetime risk per a validated risk assessment model, a personal or family history of BRCA mutations or other predisposing genetic syndromes (Li-Fraumeni, Cowden, or BannayanÑRileyÑRuvalcaba), or a history of chest wall radiation between the ages of 10 and 30.

B. Diagnostic Imaging

1. Diagnostic mammograms are performed in the **symptomatic** patient or to **follow up an abnormality noted on a screening mammogram.** Additional views (spot-compression views or magnification views) may be used to further characterize any lesion. A normal mammogram in the presence of a palpable mass does *not* exclude malignancy and further workup should be performed with an ultrasound, MRI, and/or biopsy.

2. Ultrasonography can determine whether a lesion is **solid or cystic** and can define the size, contour, or internal texture of the lesion. Although not a useful screening modality by itself due to significant false-positive 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** due to denser breast tissue. In patients with known cancer, ultrasound is sometimes used to detect additional suspicious lesions and/or to map the extent of disease.

3. MRI is useful as an adjunct to mammography to determine extent of disease, to detect multicentric disease in the dense breast, to assess the contralateral breast, to evaluate patients with axillary metastases and an

unknown primary (i.e., occult primary breast cancer), and in patients in whom mammogram, ultrasound, and clinical findings are inconclusive. It is also useful for assessing chest wall involvement. Patients should be counseled about the relatively high false-positive rates associated with this modality (Fig. 35-1).

IV. BREAST BIOPSY

A. Palpable masses (Figs. 35-2 and 35-3)

1. Fine-needle aspiration biopsy (FNAB) is reliable and accurate, with sensitivity greater than 90%. FNAB can determine the presence of malignant cells and estrogen receptor (ER) and progesterone receptor (PR) status but does not give information on tumor grade or the presence of invasion. Nondiagnostic aspirates require an additional biopsy, either surgical, that is, excisional or core needle biopsy (*Am J Surg.* 1997;174:371-385).

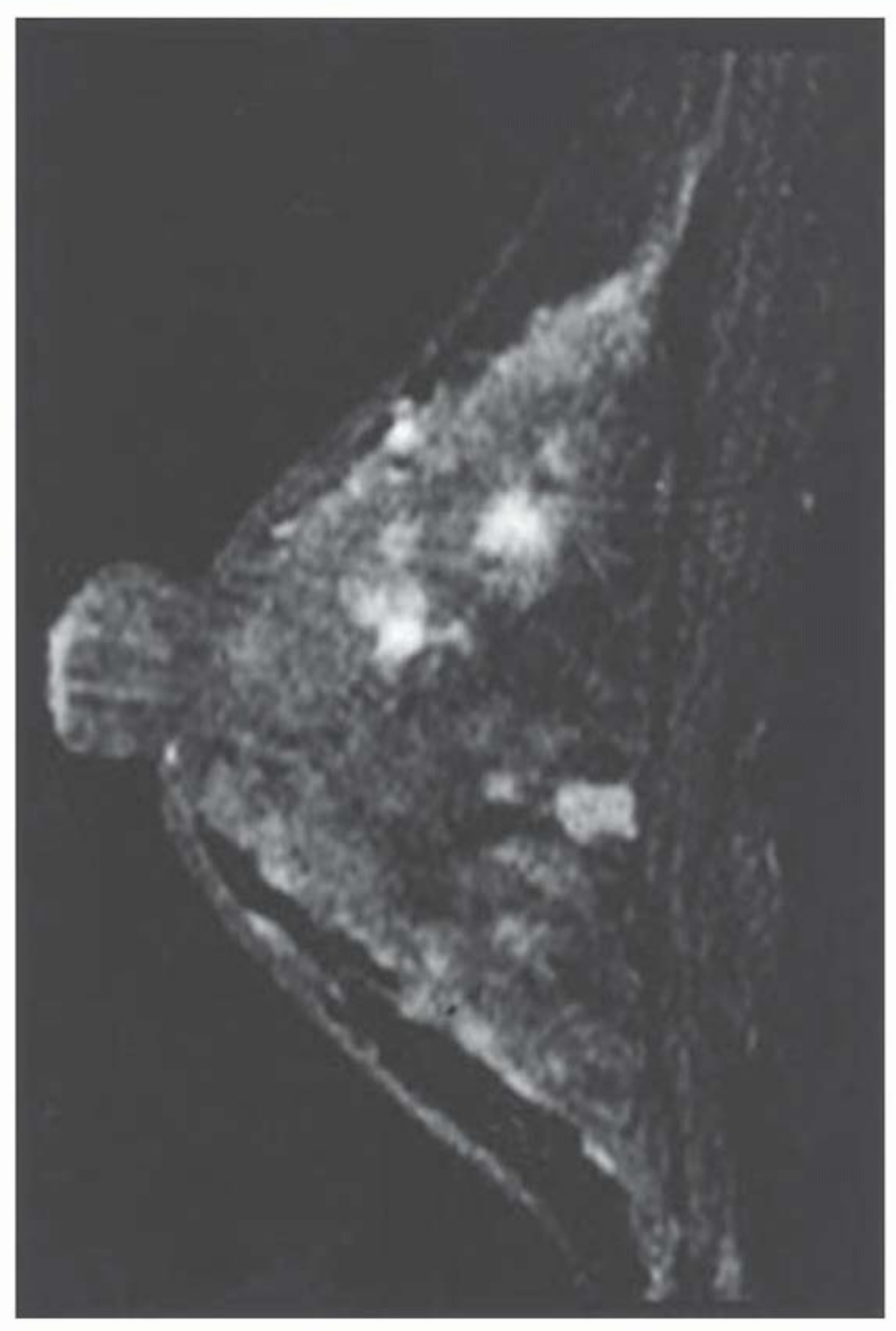


Figure 35-1 Magnetic resonance imaging (MRI)-detected multiple enhancing Òlesions.Ó (Reproduced with permission from Harris JR, Lippman ME, Osborne CK, et al., eds. *Diseases of the Breast.* Philadelphia, PA: Wolters Kluwer Health, 2014.)

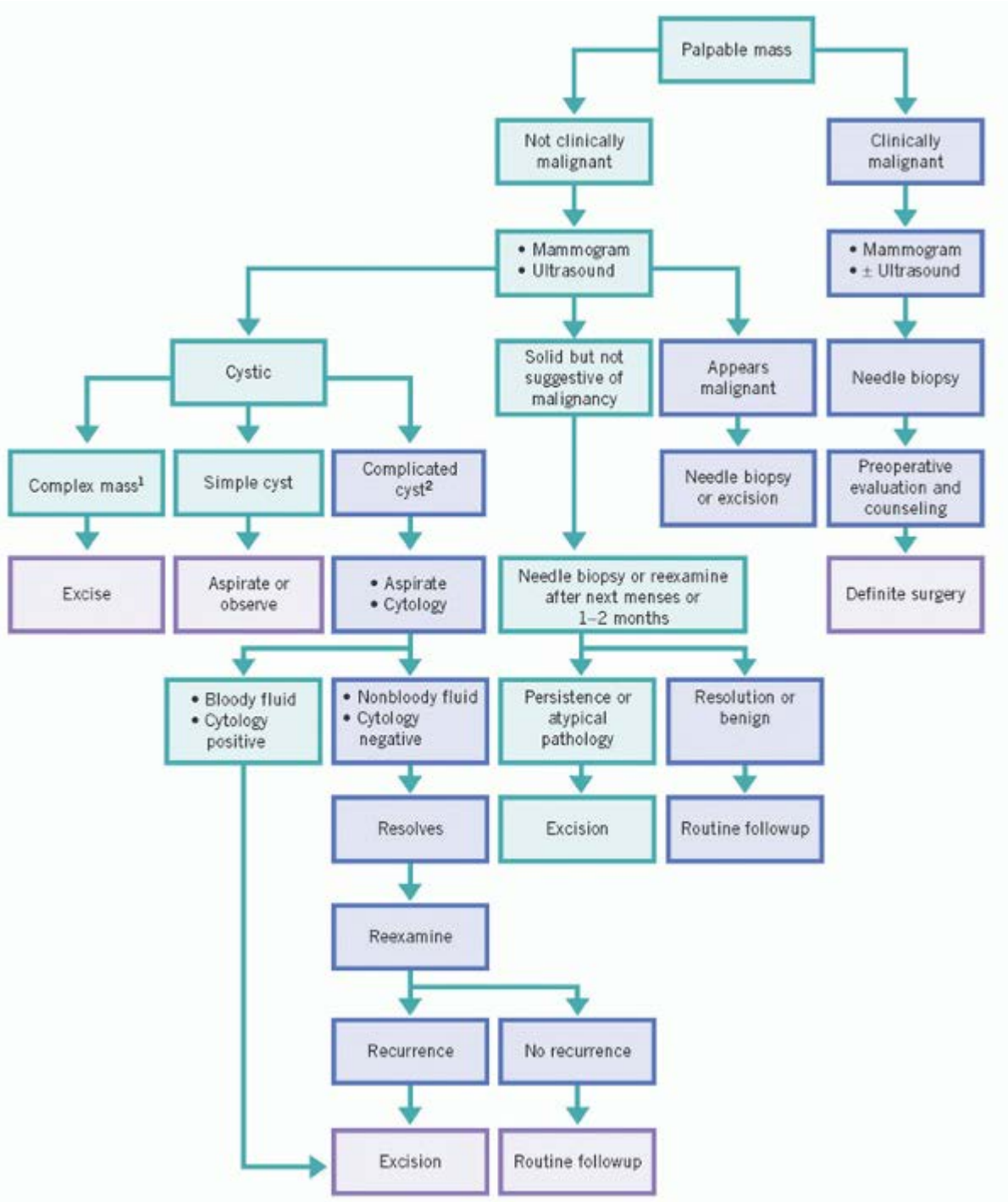


Figure 35-2 Algorithm for management of breast masses in premenopausal women. (Reproduced with permission from Berek JS. *Berek & Novak's Gynecology.* Philadelphia, PA: Wolters Kluwer Health, 2012.)

2. Core needle biopsy is preferred over FNAB. It can distinguish between invasive and noninvasive cancer and provides information on tumor grade as well as receptor status. For indeterminate specimens, a surgical biopsy is necessary.

3. Excisional biopsy should primarily be used when a core biopsy cannot be done. In general, this should be an infrequent diagnostic method. It is performed in the operating room; incisions should be planned so that they can be incorporated into a future mastectomy incision if necessary.

Masses should be excised as a single specimen and labeled to preserve three-dimensional orientations.

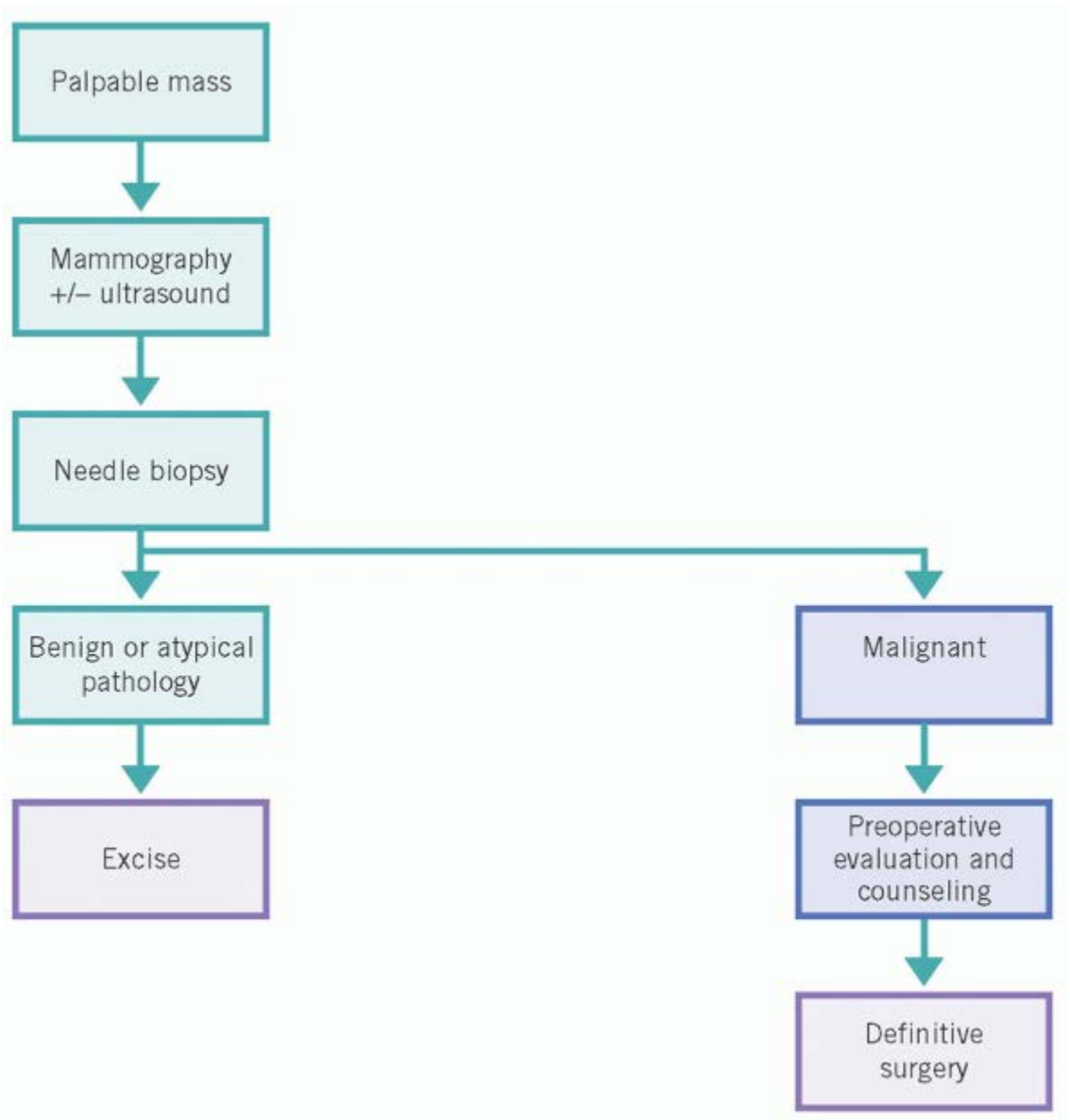


Figure 35-3 Algorithm for management of breast masses in postmenopausal women.

(Reproduced with permission from Berek JS. *Berek & Novak's Gyn*ecology. Philadelphia, PA: Wolters Kluwer Health, 2012.)

4. Incisional biopsy is indicated for the evaluation of a large breast mass suspicious for malignancy but for which a definitive diagnosis cannot be made by FNAB or core biopsy. For **inflammatory breast cancer** with skin involvement, an incisional biopsy can consist of a **skin punch** biopsy.

B. Nonpalpable Lesions. Minimally invasive breast biopsy is the optimal initial tissue acquisition method and procedure of choice for obtaining a pathologic diagnosis of image-detected abnormalities. Correlation between pathology results and imaging findings is mandatory. Patients with histologically benign findings on percutaneous biopsy do not require open biopsy if imaging and pathologic findings are concordant. Patients with

high-risk lesions on image-guided biopsy **(atypical ductal hyperplasia [ADH], atypical lobular hyperplasia [ALH], lobular carcinoma in situ [LCIS], radial scar)** may have malignancy at the same site and should undergo a **surgical biopsy.**

1. Stereotactic core biopsy is used for nonpalpable mammographically detected lesions, such as microcalcifications that cannot be seen with ultrasonography. In a vacuum-assisted approach, a **metallic marking clip** is usually placed through the probe after sampling is complete to allow for identification of the biopsy site if excisional biopsy or partial mastectomy becomes necessary; this is the preferred approach for lesions presenting with microcalcifications without a visible or palpable mass. Contraindications include lesions close to the chest wall or in the axillary tail and thin breasts that may allow needle strikethrough into the thorax. Superficial lesions and lesions directly beneath the nippleÑareolar complex are also often not approachable with stereotactic techniques. Nondiagnostic and insufficient specimens necessitate **needle-localized excisional biopsy** (**NLB,** see later discussion).

2. Ultrasound-guided biopsy is generally easier to perform than stereotactic core biopsy and is the preferred method for lesions with a cystic component, as it can be used to aspirate the cyst as well as to provide core biopsy specimens.

3. NLB. A needle and hookwire are placed into the breast adjacent to the concerning lesion under mammographic guidance. The patient is then brought to the operating room for an excisional biopsy. Using localization mammograms as a map, the whole hookwire, breast lesion, and a rim of normal breast tissue is removed en bloc. The specimen is oriented, and a radiograph is performed to confirm the presence of the lesion within the specimen.

BENIGN BREAST CONDITIONS

I. FIBROCYSTIC BREAST CHANGE.

Fibrocystic breast change refers to a variety of pathologic features including stromal fibrosis, macro- and microcysts, apocrine metaplasia, hyperplasia, and adenosis (which may be sclerosing, bluntduct, or florid).

A. FBC is common and may present as breast pain, a breast mass, nipple discharge, or abnormalities on mammography.

B. Patients presenting with suspected FBC should be reexamined in a short interval, preferably on day 10 of the menstrual cycle, when hormonal influence is lowest and the mass may have diminished in size.

C. A persistent dominant mass must undergo further radiographic evaluation to exclude cancer.

II. BREAST CYSTS.

Breast cysts frequently present as tender masses or as smooth, mobile, well-defined masses on palpation. **Aspiration can determine the nature of the mass (solid vs. cystic) but is not routinely necessary.**

Cyst fluid color varies and can be clear, straw-colored, or even dark green. If discovered by mammography and confirmed as simple cysts by ultrasound, **asymptomatic cysts can be observed. Symptomatic simple cysts should be aspirated.** If no palpable mass is present after drainage, the patient should be evaluated in 3 to 4 weeks. If the cyst recurs, does not resolve completely with aspiration, or yields bloody fluid with aspiration, then mammography or ultrasonography should be performed to exclude intracystic tumor. Nonbloody clear fluid does not need to be sent for cytology.

III. FIBROADENOMA.

Fibroadenoma is the most common discrete mass in women younger than 30 years of age. They typically present as **smooth, firm, mobile masses,** and can be multiple in 20% of cases.

A. They may enlarge during pregnancy and involute after menopause.

B. They have well-circumscribed borders on mammography and ultrasound.

C. They may be managed conservatively if clinical and radiographic appearance is consistent with a fibroadenoma and it is less than 2 cm. If the mass is symptomatic, greater than 2 cm, or enlarges, it should be excised.

IV. MASTALGIA.

Most women (70%) experience some form of breast pain or discomfort during their lifetime. The pain may be **cyclic** (worse before a menstrual cycle) or **noncyclical,** which is more suspicious for malignancy, especially if focal and in association with a mass or bloody discharge. Benign

disease is the etiology in the majority of cases. However, pain may be associated with cancer in up to 10% of patients. *Once cancer has been excluded,* most patients can be managed successfully with symptomatic therapy and reassurance, as it resolves in up to 30% of women, though it does recur in 60%. In 15% of patients, the pain may be so disabling that it interferes with activities of daily living. A **well-fitting supportive bra** is an important first step in pain relief. **Topical nonsteroidal anti-inflammatory drugs (NSAIDs, e.g., diclofenac gel)** is considered first-line therapy, having been proven effective with minimal side effects in a randomized trial (*J Am Coll Surg.* 2003;196:525-530). Lowdose **tamoxifen** (an estrogen antagonist) has been shown to provide good pain relief in placebo-controlled trials with tolerable side effects (*Br J Surg.* 1988; 75: 845Ñ846), although concerns over increased risks of endometrial cancer limit long-term use. **Danazol** (a derivative of testosterone), **bromocriptine, and gonadorelin analogs** have significant side effects, and their use should be limited to refractory cases. Many patients experience symptomatic relief by reducing caffeine intake or by **taking vitamin E or evening primrose oil,** although there is no scientific evidence supporting these lifestyle modifications.

A. Superficial thrombophlebitis of the veins overlying the breast **(Mondor disease)** may present as breast pain. Treatment is conservative with **NSAIDs** and **hot compresses.** Antibiotics are not generally indicated.

B. Breast pain in pregnancy and lactation can occur from engorgement, clogged ducts, trauma to the areola and nipple from pumping or nursing,

or any of the aforementioned sources. Clogged ducts are usually treated with **warm compresses, soaks, and massage.**

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 breastfeeding. In parous nonlactating women, a small amount of milk may be expressed from multiple ducts. This requires no treatment.

B. Galactorrhea is milky discharge unrelated to breastfeeding. Physiologic galactorrhea is the continued production of milk after lactation has ceased and menses has resumed and is often caused by continued mechanical stimulation of the nipples.

1. Drug-related galactorrhea is caused by medications that affect the hypothalamicÑpituitary axis by depleting dopamine (**tricyclic antidepressants, reserpine, methyldopa, cimetidine, and benzodiazepines**), blocking the dopamine receptor **(phenothiazine, metoclopramide, and 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 **prolactinoma,** and may be associated with amenorrhea. The diagnosis is established by

measuring the **serum prolactin level** and performing a **computed tomography (CT) or MRI scan of the pituitary gland.** Treatment is **bromocriptine** or **resection** of the prolactinoma.

C. Pathologic nipple discharge is usually **bloody** (can be confirmed with guaiac test), **spontaneous, unilateral,** and/or originates from a **single duct.** *Normal physiologic discharge* is usually nonbloody, is from multiple ducts, can be a variety of colors (clear to yellow to green), and requires breast manipulation to produce. Cytologic evaluation is generally not useful.

1. Malignancy is the underlying cause in 10% of patients.

2. If not associated with a mass, the most likely etiologies are **benign intraductal papilloma** (peripheral papillomas put patients at slightly higher risk of malignancy), **duct ectasia,** and **fibrocystic changes.** In lactating women, serosanguineous or bloody discharge can be associated with duct trauma, infection, or epithelial proliferation associated with breast enlargement.

3. Patients with persistent spontaneous discharge from a single duct require a surgical **microdochectomy** (excision of a single duct and its associated lobule) using a ductogram or ductoscopy or **major duct excision** (excision of all retroareolar ducts).

VI. BREAST INFECTIONS

A. Lactational Mastitis

1. The most common causative organism is *Staphylococcus aureus.*

2. It presents as a swollen, erythematous, and tender breast; purulent discharge from the nipple is *uncommon.*

3. In the early cellulitic phase, the treatment is **antibiotics,** and the frequency of **nursing or pumping should be** *increased.* Approximately 25% progress to abscess formation.

4. Breast abscesses occur in the later stages and are often *not* fluctuant. The diagnosis is made by failure to improve on antibiotics, abscess cavity seen on ultrasound, or aspiration of pus. Treatment is **cessation of nursing** and **surgical drainage.**

B. Nonpuerperal abscesses result from duct ectasia with periductal mastitis, infected cysts, infected hematoma, or hematogenous spread from another source.

1. They usually are located in the peri/retroareolar area.

2. Anaerobes are the most common causative agent, although antibiotics should cover both **anaerobic and aerobic** organisms.

3. Treatment is **surgical drainage.**

4. Unresolved or recurring infection requires biopsy to exclude cancer. These patients often have a chronic relapsing course with multiple infections requiring surgical drainage.

5. Repeated infections can result in a **chronically draining periareolar lesion or a mammary fistula** lined with squamous epithelium. Treatment is **excision of the central duct along with the fistula** once the acute infection resolves. The fistula can recur even after surgery.

VII. GYNECOMASTIA.

Gynecomastia is hypertrophy of breast tissue in men that is usually secondary to an imbalance between the breast stimulatory effects of estrogen and the inhibitory effects of androgens.

A. Pubertal hypertrophy occurs in adolescent boys, is usually bilateral, and resolves spontaneously in 6 to 12 months.

B. Senescent gynecomastia is commonly seen after the age of 70 years, as testosterone levels decrease.

C. Drugs associated with this are similar to those that cause galactorrhea in women: **Digoxin, spironolactone, methyldopa, cimetidine, tricyclic antidepressants, phenothiazine, reserpine, and marijuana.** Drugs used for androgen blockade, such as **luteinizing hormone releasing hormone analogues** for the treatment of prostate cancer and **5-alpha reductase inhibitors** (e.g., finasteride) for the management of benign prostatic hypertrophy, may also result in gynecomastia.

D. Tumors can cause gynecomastia secondary to excess secretion of estrogens. These include testicular teratomas and seminomas, bronchogenic carcinomas, adrenal tumors, and tumors of the pituitary and hypothalamus.

E. Gynecomastia may be a manifestation of **systemic diseases** such as hepatic cirrhosis, renal failure, hyperthyroidism, and malnutrition.

F. During the workup of gynecomastia, cancer should be excluded by mammography and subsequently by biopsy if a mass is found. If workup fails

to reveal a medically treatable cause or if the enlargement fails to regress, **excision of breast tissue via a periareolar incision** can be performed.

VIII. HIGH-RISK AND PREMALIGNANT CONDITIONS

A. ADH and **ALH** are proliferative lesions with cell atypia that arise within breast ducts and lobules, respectively. ADH confers a 4 to 5 times increased relative risk of developing an invasive breast malignancy. Historically, ALH was thought to have a weaker association with malignancy, but a recent study suggested that both lesions confer equal levels of risk (*Cancer Prev Res.* 2014;7:211-217). If atypical hyperplasia is found on needle biopsy, **excisional biopsy** is warranted to rule out associated malignancy. If no malignancy is found on postoperative pathology, patients with these conditions can simply undergo surveillance with imaging and

physician examination at increased intervals as compared to low-risk patients. **Chemoprevention with tamoxifen** is also an option.

B. LCIS is not considered a preinvasive lesion but rather an indicator for increased breast cancer risk of approximately 1% per year (÷20% to 30% at 15 years) (*J Natl Compr Canc Netw.* 2006;4:511-522).

1. It may be **multifocal** and/or **bilateral.**

2. The cancer that develops **may be invasive ductal or lobular** and may occur in either breast.

3. LCIS has loss of **E-cadherin** (involved in cellÑcell adhesion), which can be stained for on pathology slides to clarify cases that are borderline ductal carcinoma in situ (DCIS) from LCIS.

4. Pleomorphic LCIS is a particularly aggressive subtype of LCIS that is treated more like DCIS; it tends to have less favorable biologic markers.

5. Treatment options are (1) lifelong close **surveillance,** (2) **bilateral total mastectomies** with immediate reconstruction for selected women with a strong family history after appropriate counseling, or (3) **chemoprevention** with **tamoxifen, raloxifene** (which has been validated in the postmenopausal setting), or an **aromatase inhibitor.**

MALIGNANCY OF THE BREAST

I. EPIDEMIOLOGY.

Breast cancer is the **most common noncutaneous cancer in women,** with a lifetime risk of **one in eight women.** In the United States in 2014, an estimated 235,030 new cases of invasive breast cancer and 62,570 new cases of noninvasive in situ carcinoma of the breast were diagnosed (*CA Cancer J Clin.* 2014;64:9-29). In that same period, approximately 40,000 women died of breast cancer, making it the second-leading cause of cancer death in women, exceeded only by lung cancer.

II. STAGING.

Breast cancer is staged by using the **American Joint Committee on Cancer (AJCC)** system, which is both a **clinical and pathologic staging system** and is based on the TNM system (tumor, node, and metastasis) system (Tables 35-2, 35-3, 35-4, 35-5 and 35-6). The most recent seventh edition marks a significant departure from the previous version with its inclusion of pathologic staging, which can be performed in patients treated initially with definitive surgery or in patients treated with neoadjuvant systemic therapy followed by definitive surgery. Workup should include the following in addition to breast-specific imaging:

TABLE 35-2 AJCC StagingÑPrimary Tumor (T)[a]

TX		Primary tumor cannot be assessed
T0		No evidence of primary tumor
Tis		Carcinoma in situ
	Tis (DCIS)	DCIS
	Tis (LCIS)	LCIS
	Tis (Paget)	Paget disease of the nipple NOT associated with invasive carcinoma and/or carcinoma in situ (DCIS and/or LCIS) in the underlying breast parenchyma. Carcinomas in the breast parenchyma associated with Paget disease are categorized based on the size and characteristics of the parenchymal disease, although the presence of Paget disease should still be noted.
T1		Tumor ²20 mm in greatest dimension
	T1mi	Tumor ²1 mm in greatest dimension
	T1a	Tumor >1 mm but ²5 mm in greatest dimension
	T1b	Tumor >5 mm but ²10 mm in greatest dimension
	T1c	Tumor >10 mm but ²20 mm in greatest dimension
T2		Tumor >20 mm but ²50 mm in greatest dimension
T3		Tumor >50 mm in greatest dimension

T4	Tumor of any size with direct extension to the chest wall and/or to the skin (ulceration or skin nodules)[b]
T4a	Extension to the chest wall, not including only pectoralis muscle adherence/invasion
T4b	Ulceration and/or ipsilateral satellite nodules and/or edema (including peau d'orange) of the skin, which do not meet the criteria for inflammatory carcinoma
T4c	Both T4a and T4b
T4d	Inflammatory carcinoma

[a]The T classification of the primary tumor is the same regardless of whether it is based on clinical or pathologic criteria, or both. Size should be measured to the nearest millimeter. If the tumor size is slightly less than or greater than a cutoff for a given T classification, it is recommended that the size be rounded to the millimeter reading that is closest to the cutoff. For example, a reported size of 1.1 mm is reported as 1 mm, or a size of 2.01 cm is reported as 2 cm. Designation should be made with the subscript ÒcÓ or ÒpÓ modifier to indicate whether the T classification was determined by clinical (physical examination or radiologic) or pathologic measurements, respectively. In general, pathologic determination should take precedence over clinical determination of T size.

[b] Invasion of the dermis alone does not qualify as T4.

DCIS, ductal carcinoma in situ; LCIS, lobular carcinoma in situ.

TABLE 35-3 AJCC StagingÑRegional Lymph Nodes (N)

		Clinical
NX		Regional lymph nodes cannot be assessed (e.g., previously removed)
N0		No regional lymph node metastases
N1		Metastases to movable ipsilateral level I, II axillary lymph node(s)
N2		Metastases in ipsilateral level I, II axillary lymph nodes that are clinically fixed or matted OR Metastases in clinically detected[a] ipsilateral internal mammary nodes in the absence of clinically evident axillary lymph node metastases
	N2a	Metastases in ipsilateral level I, II axillary lymph nodes fixed to one another (matted) or to other structures
	N2b	Metastases only in clinically detected[a] ipsilateral internal mammary nodes and in the absence of clinically evident level I, II axillary lymph node metastases
N3		Metastases in ipsilateral infraclavicular (level III axillary) lymph node(s) with or without level I, II axillary lymph node involvement OR Metastases in clinically detected[a] ipsilateral internal mammary lymph node(s) with clinically evident level I, II axillary lymph node metastases OR Metastases in ipsilateral supraclavicular lymph node(s) with or without axillary or internal mammary lymph node involvement

N3a	Metastases in ipsilateral infraclavicular lymph node(s)
N3b	Metastases in ipsilateral internal mammary lymph node(s) and axillary lymph node(s)
N3c	Metastases in ipsilateral supraclavicular lymph node(s)

[a] Clinically detected is defined as detected by imaging studies (excluding lymphoscintigraphy) or by clinical examination and having characteristics highly suspicious for malignancy or a presumed pathologic macrometastasis based on fine-needle aspiration biopsy with cytologic examination. Confirmation of clinically detected metastatic disease by fine-needle aspiration without excision biopsy is designated with an (f) suffix, for example, cN3a(f). Excisional biopsy of a lymph node or biopsy of a sentinel node, in the absence of assignment of a pT, is classified as a clinical N, for example, cN1. Information regarding the confirmation of the nodal status will be designated in site-specific factors as clinical, fine-needle aspiration, core biopsy, or sentinel lymph node biopsy. Pathologic classification (pN) is used for excision or sentinel lymph node biopsy only in conjunction with a pathologic T assignment.

TABLE 35-4 AJCC StagingÑPathologic Lymph Node Status (pN)[a,b,c]

pNX	Regional lymph nodes cannot be assessed (e.g., previously removed or not removed for pathologic study)
pN0	No regional lymph node metastasis identified histologically

Note: ITCs are defined as small clusters of cells ²0.2 mm, or single tumor cells, or a

cluster of <200 cells in a single histologic cross-section. ITCs may be detected by routine histology or by IHC methods. Nodes containing only ITCs are excluded from the total positive node count for purposes of N classification but should be included in the total number of nodes evaluated.

pN0(i-)	No regional lymph node metastases histologically, negative IHC
pN0(i+)	Malignant cells in regional lymph node(s) ²0.2 mm (detected by H&E or IHC including ITC)
pN0(mol-)	No regional lymph node metastases histologically, negative molecular findings (RT-PCR)
pN0(mol+)	Positive molecular findings (RT-PCR), but no regional lymph node metastases detected by histology or IHC
pN1	Micrometastases
	OR
	Metastases in 1-3 axillary lymph nodes
	AND/OR
	Metastases in internal mammary nodes with metastases detected by sentinel lymph node biopsy but not clinically detected[b]
pN1mi	Micrometastases (>0.2 mm and/or >200 cells but none >2.0 mm)
pN1a	Metastases in 1-3 axillary lymph nodes, at least one metastasis >2.0 mm
pN1b	Metastases in internal mammary nodes with micrometastases or macrometastases detected by sentinel lymph node biopsy but not clinically detected[b]

pN1c	Metastases in 1-3 axillary lymph nodes and in internal mammary lymph nodes with micrometastases or macrometastases detected by sentinel lymph node biopsy but not clinically detected
pN2	Metastases in 4-9 axillary lymph nodes OR Metastases in clinically detected[b] internal mammary lymph nodes in the *absence* of axillary lymph node metastases
pN2a	Metastases in 4-9 axillary lymph nodes (at least 1 tumor deposit >2 mm)
pN2b	Metastases in clinically detected[c] internal mammary lymph nodes in the *absence* of axillary lymph node metastases
pN3	Metastases in ³10 axillary lymph nodes OR Metastases in infraclavicular (level III axillary) lymph nodes OR Metastases in clinically detected[c] ipsilateral internal mammary lymph nodes in the *presence* of one or more positive level I, II axillary lymph nodes OR Metastases in >3 axillary lymph nodes and in internal mammary lymph nodes with micrometastases or macrometastases detected by sentinel lymph node biopsy but not clinically detected[b]

	OR
	Metastases in ipsilateral supraclavicular lymph nodes
pN3a	Metastases in ³10 axillary lymph nodes (at least 1 tumor deposit >2.0 mm)
	OR
	Metastases to the infraclavicular (level III axillary lymph) nodes
pN3b	Metastases in clinically detected[c] ipsilateral internal mammary lymph nodes in the *presence* of one or more positive axillary lymph nodes
	OR
	Metastases in >3 axillary lymph nodes and in internal mammary lymph nodes with micrometastases or macrometastases detected by sentinel lymph node biopsy but not clinically detected[b]
pN3c	Metastases in ipsilateral supraclavicular lymph nodes

Posttreatment ypN

- Posttreatment yp ÒNÓ should be evaluated as for clinical (pretreatment) ÒNÓ methods above. The modifier ÒsnÓ is used only if a sentinel node evaluation was performed after treatment. If no subscript is attached, it is assumed that the axillary nodal evaluation was by AND.
- The X classification will be used (ypNX) if no yp posttreatment sn or AND was performed.
- N categories are the same as those used for pN.

[a]Classification is based on axillary lymph node dissection with or without sentinel lymph node biopsy. Classification based solely on sentinel lymph node biopsy without subsequent axillary lymph node dissection is designated (sn) for Òsentinel node,Ó for example, pN0(sn).

[b]Not clinically detectedÓ is defined as not detected by imaging studies (excluding lymphoscintigraphy) or not detected by clinical examination.

[c]ÒClinically detectedÓ is defined as detected by imaging studies (excluding lymphoscintigraphy) or by clinical examination and having characteristics highly suspicious for malignancy or a presumed pathologic macrometastasis based on fine-needle aspiration biopsy with cytologic examination.
AND, axillary node dissection; H&E, hematoxylin and eosin stain; IHC, immunohistochemical; ITC, isolated tumor cells; RT-PCR, reverse transcriptase/polymerase chain reaction.

TABLE 35-5 AJCC StagingÑDistant Metastases (M)

M0	No clinical or radiographic evidence of distant metastases
cM0(i+)	No clinical or radiographic evidence of distant metastases, but deposits of molecularly or microscopically detected tumor cells in circulating blood, bone marrow, or other nonregional nodal tissue that are ²0.2 mm in a patient without symptoms or signs of metastases
M1	Distant detectable metastases as determined by classic clinical and radiographic means and/or histologically proven >0.2 mm

Posttreatment yp M classification. The M category for patients treated with neoadjuvant therapy is the category assigned in the clinical stage, prior to initiation of neoadjuvant therapy. Identification of distant metastases after the start of therapy in cases where pretherapy evaluation showed no metastases is considered progression of disease. If a patient was designated to have detectable distant metastases (M1) before chemotherapy, the patient will be designated as M1 throughout.

A. Complete blood cell count (CBC), **complete metabolic panel** including **liver function tests (LFTs),** and **chest x-ray.**

B. A **bone scan,** if the alkaline phosphatase or calcium level is elevated.

C. CT scan of the abdomen if LFTS are abnormal.

D. Patients with clinical stage III or IV disease should undergo bone scan and CT scan of the chest/abdomen/pelvis due to a high probability of distant metastases.

III. TUMOR BIOMARKERS AND PROGNOSTIC FACTORS.

These should be evaluated on all tumor specimens. The **presence or absence of disease in axillary lymph nodes** is the single most important prognostic factor in breast cancer. **Tumor size** and **grade** (which is based on degrees of *glandular differentiation, mitotic count,* and *nuclear grade*) are the most reliable pathologic predictors of outcome for patients without axillary nodal involvement. **High grade** (**i.e., grade 3** on a scale of 1 to 3) is a *poor* prognostic factor.

A. Hormone Receptors. Expression of **ERs** and **progesterone (PRs)** should be evaluated by immunohistochemistry. Intense ER and PR staining is a *good* prognostic factor.

TABLE 35-6 AJCC Anatomic Stage/Prognostic Groups[a,b]

Stage	T	N	M
0	Tis	N0	M0
IA	T1[a]	N0	M0
IB	T0	N1mi	M0

	$T1^b$	N1mi	M0
IIA	T0	$N1^b$	M0
	$T1^a$	$N1^b$	M0
	T2	N0	M0
IIB	T2	N1	M0
	T3	N0	M0
IIIA	T0	N2	M0
	$T1^a$	N2	M0
	T2	N2	M0
	T3	N1	M0
	T3	N2	M0
IIIB	T4	N0	M0
	T4	N1	M0
	T4	N2	M0
IIIC	Any T	N3	M0
IV	Any T	Any N	M1

- M0 includes M0(i+).
- The designation pM0 is not valid; any M0 should be clinical. If a patient presents with

M1 prior to neoadjuvant systemic therapy, the stage is considered Stage IV and remains Stage IV regardless of response to neoadjuvant therapy. Stage designation may be changed if postsurgical imaging studies reveal the presence of distant metastases, provided that the studies are carried out within 4 months of diagnosis in the absence of disease progression and provided that the patient has not received neoadjuvant therapy.

- Postneoadjuvant therapy is designated with ÒycÓ or ÒypÓ prefix. Of note, no stage group is assigned if there is a complete pathologic response (CR) to neoadjuvant therapy, for example, ypT0ypN0cM0.

[a]T1 includes T1mi.

[b]T0 and T1 tumors with nodal micrometastases only are excluded from Stage IIA and are classified Stage IB.

B. *Her2/neu (ERB2). Her2/neu* is a member of the epidermal growth factor receptor (EGFR) family and is involved in cell growth regulation. Overexpression due to gene amplification is seen in approximately 30% of patients with breast cancer. *Her2/neu* expression is measured by **immunohistochemistry** and, if equivocal, by **fluorescence** *in situ* **hybridization (FISH**). Overexpression of *Her2/neu* is a *poor* prognostic factor, as it results in an increased rate of metastasis, decreased time to recurrence, and decreased overall survival. Patients with Her2/neu-amplified (HER2+) tumors are treated with targeted monoclonal antibody therapies, such as **trastuzumab (Herceptin)** or **pertuzumab (Perjeta**). Pertuzumab, in combination with trastuzumab and docetaxel, has demonstrated significant efficacy in prolonging progression-free survival in metastatic HER2+ breast cancer (*Lancet Oncol.* 2013;14:461-471); its efficacy at treating other stages is the subject of several ongoing clinical trials.

C. Other **adverse tumor characteristics** include not expressing any tumor biomarkers (**Òtriple negativeÓ**), **lymphovascular invasion,** and other indicators of a high proliferative rate (>5% of cells in the **S phase** of mitosis or >20% **Ki-67).**

IV. DUCTAL CARCINOMA IN SITU.

Ductal carcinoma in situ is a lesion with malignant cells that have not penetrated the basement membrane of the mammary ducts.

A. DCIS is treated as a **malignancy** because DCIS has the potential to develop into invasive cancer.

- It is usually detected by mammography as **clustered pleomorphic calcifications (Fig. 35-4).**
- **Physical examination is normal in the majority of patients.**
- **It may advance in a segmental manner, with gaps between disease areas.**
- **It can be multifocal** (two or more lesions >5 mm apart within the same index quadrant) or **multicentric** (in different quadrants).

1. Histology

a. There are **five architectural subtypes:** papillary, micropapillary, solid, cribriform, and comedo (necrosis). Specimens are also grouped as *comedo* versus *noncomedo.*

b. The **high-grade subtype** is often associated with microinvasion, a higher proliferation rate, aneuploidy, gene amplification, and a higher local recurrence rate.

c. ER and PR expression levels should be obtained if hormone therapy is being considered.

2. Treatment

a. Surgical excision alone (via partial mastectomy) with **margins greater than 10 mm** is associated with a local recurrence rate of 14% at 12 years (*Am J Surg.* 2006;192:420-222). The addition of adjuvant radiation reduces the local recurrence rate to 2.5%. Approximately half of the recurrences present as invasive ductal carcinomas. **Surgical options** depend on the extent of disease, grade, margin status, multicentricity of disease, and patient age.

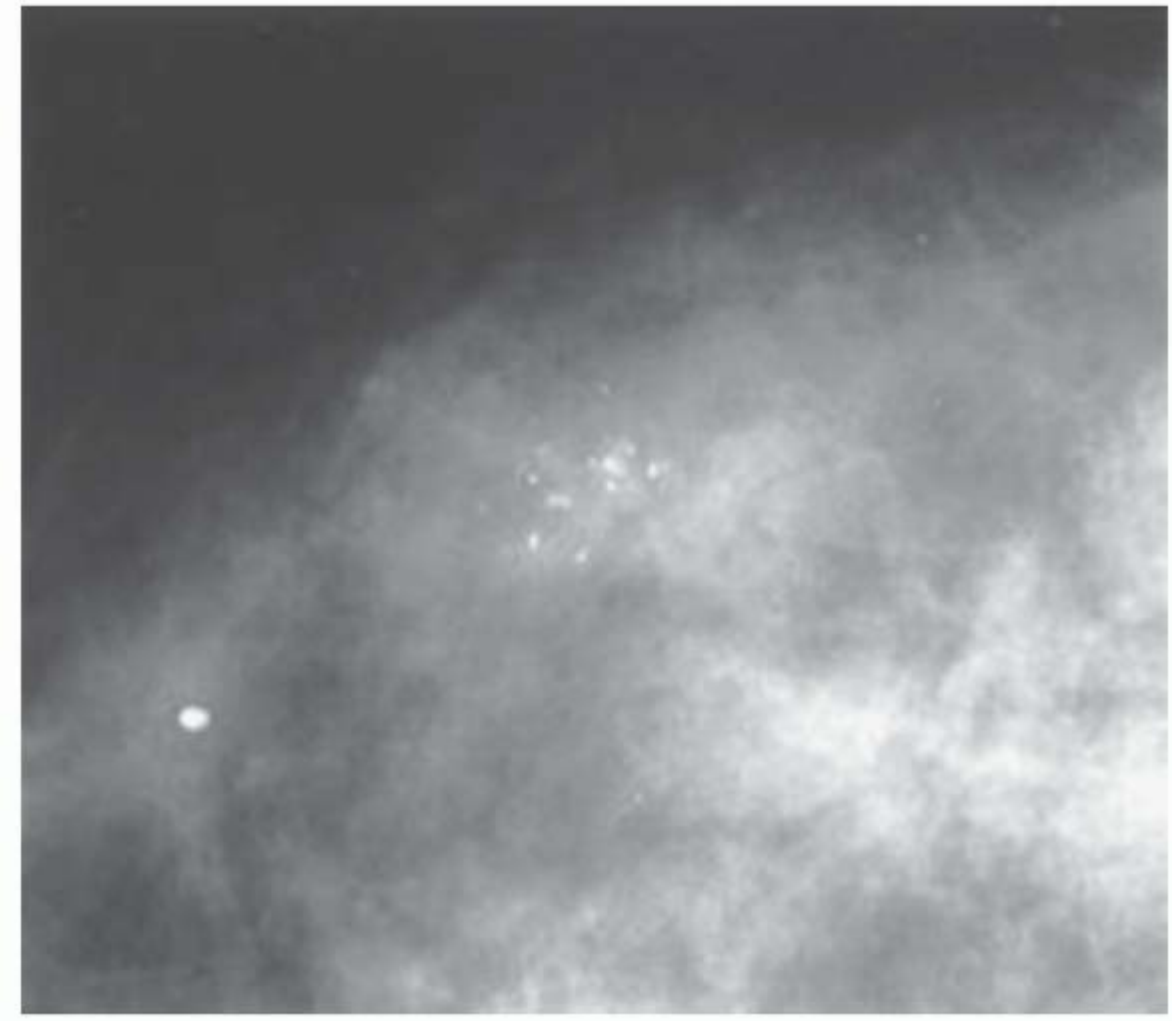


Figure 35-4 Mammogram of ductal carcinoma in situ (DCIS). (Reproduced with permission from Harris JR, Lippman ME, Osborne CK, et al., eds. *Diseases of the Breast.* Philadelphia, PA: Wolters Kluwer Health, 2014.)

(1) Partial mastectomy. For unicentric nonpalpable lesions, **needle localization** (sometimes with **bracketing**) is required to identify the area to be excised in most cases.

(2) Total (simple) mastectomy with or without immediate reconstruction is recommended for patients with multicentric lesions, extensive involvement of the breast (i.e., high tumor-to-breast-size ratio), or persistently positive margins with partial mastectomy.

b. Assessment of axillary lymph nodes. Axillary dissection is not performed for pure DCIS.

(1) Sentinel lymph node biopsy (**SLNB**) may be considered when there is a reasonable probability of finding invasive cancer on final pathologic examination (e.g., >4 cm, palpable, comedo subtype, or high grade).

(2) Some surgeons perform SLNB in all patients with DCIS undergoing mastectomy because

SLNB cannot be performed postmastectomy if an occult invasive cancer is found. This is an area of ongoing controversy and research.

(3) A positive sentinel node indicates invasive breast cancer and changes the stage of the disease. Historically, a **completion axillary dissection** was indicated for patients with a positive sentinel node, but the findings of the American College of Surgeons Oncology Group (ACOSOG) trial Z011, have contributed to a paradigm shift in the management of axillary disease (see later discussion in ÒManagement of the AxillaÓ).

c. Adjuvant therapy

(1) For pure DCIS, there is no added benefit from systemic chemotherapy because the disease is confined to the ducts of the breast. However, in those patients with **ER-positive (ER+) DCIS,** adjuvant **tamoxifen** can reduce the risk of breast cancer recurrence by 36% over 5 years and the risk of developing a new contralateral breast cancer *(*NSABP B-24 trial, *J Clin Oncol.* 2012;30:1268-1273) but confers no survival benefit. **Aromatase inhibitors** (e.g., **anastrozole, exemestane, letrozole**), which block the peripheral conversion of androgens into estrogens by inhibiting the enzyme aromatase but do not affect estrogen produced by the ovaries, are sometimes used as an alternative in postmenopausal patients.

(2) Adjuvant radiation should be given to patients with DCIS treated with partial mastectomy to **decrease the rate** of **local recurrence** (NSABP B-17 trial, *J Natl Cancer Inst.* 2011;103:478-488). This is especially true for younger women with close margins or large tumors. However, there is no survival benefit. For older patients with smaller, widely excised DCIS of low or intermediate grade, the benefit of radiation therapy is less clear and adjuvant radiation may not be necessary (*Ann Surg Oncol.* 2013;20:3175-3179).

d. The University of Southern California/Van Nuys Prognostic Index (Table 35-7) is a numerical algorithm used to determine which patients with DCIS are at greatest risk for recurrence and would therefore benefit from aggressive treatment. In order to

achieve a local recurrence rate of less than 20% at 12 years, **surgical excision** alone is recommended for all patients scoring 4, 5, or 6 and patients who score 7 but have margin widths ³3 mm; **surgical excision plus radiation therapy** for those who score 7 and have margins <3 mm, patients who score 8 and have margins ³3 mm, and for patients who score 9 and have margins >5 mm; and **mastectomy** for patients who score 8 and have margins <3 mm, who score 9 and have margins <5 mm, and for all patients who score 10, 11, or 12 (*J Natl Cancer Inst Monogr.* 2010;2010(41):193-196).

TABLE 35-7 University of Southern California/Van Nuys [a]

Prognostic Index Scoring System

	Score		
	1	**2**	**3**
Size (mm)	≤15	>15-40	≥41
Margins (mm)	≥10	1-9	<1
Histology	Grade 1/2 without necrosis	Grade 1/2 with necrosis	Grade 3 with or without necrosis
Age	>60	40-60	<40

[a]A score, ranging from 1 for lesions with the best prognosis to 3 for lesions with the worst prognosis, is given for each of the four prognostic predictors, thus establishing a range of 4 (best prognosis) to 12 (worst prognosis) for total scores.
Modified from Silverstein MJ, Lagios MD. Choosing Treatment for patients with ductal carcinoma in situ: fine tuning the University of Southern California/Van Nuys Prognostic Index. *J Natl Cancer Inst Monogr.* 2010;2010(41):193-196. PMID: 20956828.

V. INVASIVE BREAST CANCER

A. The most common **histology** identified includes *infiltrating ductal* (75% to 80%), *infiltrating lobular* (5% to 10%), *medullary* (5% to 7%), *mucinous* (3%), and *tubular* (1% to 2%).

B. Surgical options for **early-stage (T1-2, N1, or less clinical disease)** breast cancer:

1. Mastectomy with or without reconstruction.

a. Radical (Halsted) mastectomy involves total mastectomy, complete ALND (levels I, II, and III), removal of the pectoralis major and minor muscles, and removal of all overlying skin. This surgical approach is largely historical and is rarely, if ever, performed in modern practice.

b. Modified radical mastectomy (MRM) involves total mastectomy and ALND. It is indicated for patients with clinically positive lymph nodes or a positive (i.e., with macrometastases) axillary node based on previous SLNB or FNAB.

c. Total (simple) mastectomy with SLNB is for patients with a clinically negative axilla. A **skin-sparing mastectomy** (preserves skin envelope and inframammary ridge) may be performed with immediate reconstruction, resulting in improved cosmesis: The nippleÑareolar complex, a rim of periareolar breast skin, and any previous excisional biopsy or partial mastectomy scars are excised. **Nipple-sparing mastectomy,** in which all of the skin including the nippleÑareolar complex is left in place, may also be an option for select women. Patients must be counseled that the preserved nipple is often insensate and that nipple necrosis is not an infrequent complication of the procedure, occurring in an estimated 10% to 15% of patients (*Breast J.* 2014;20:69-73).

d. Immediate reconstruction at the time of mastectomy should be offered to eligible patients. Options include latissimus dorsi myocutaneous flaps, transverse rectus abdominis myocutaneous (TRAM) flaps, and inflatable tissue expanders followed by exchange for saline or silicone implants. 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.

e. Follow-up after mastectomy should involve physical examination every 3 to 6 months for 3 years, then every 6 to 12 months for the

next 2 years, and then annually (*J Clin Oncol* 2013; 31: 961-965). Mammography of the contralateral breast should continue yearly. Regular gynecologic follow-up is recommended for all women (*N.B.* tamoxifen increases risk of endometrial cancer).

2. Breast conservation therapy (BCT, partial mastectomy and SLNB [or ALND; see later discussion] followed by breast irradiation).

a. Several trials have demonstrated that BCT with adjuvant radiation therapy has similar survival and recurrence rates to those for MRM (*J Clin Oncol.* 1992;10:976-983).

b. Contraindications for BCT. Not every patient is a candidate for BCT. It is contraindicated in patients who may be unreliable with followup or with the radiation therapy treatments that take place 5 days a week for 5 to 6 weeks); when the extent of disease prevents adequate negative margins; when there is a high tumor-to-breast size ratio that prevents adequate resection without major deformity; with persistently positive margins on re-excision partial mastectomy; and with the inability to receive adjuvant radiation (e.g., prior radiation to the chest wall; first- and second-trimester pregnancy in which the delay of radiation to the postpartum state is inappropriate; collagen vascular diseases such as scleroderma).

c. For patients with large tumors who desire BCT, **neoadjuvant chemotherapy** and/or **neoadjuvant hormonal therapy** may be offered to attempt to reduce the size of the tumor to make BCT possible.

d. Partial mastectomy incisions should be planned so that they can be incorporated into a mastectomy incision should that prove necessary. Incisions for partial mastectomy and either

SLNB or ALND should be separate.

e. Adjuvant radiotherapy decreases the breast cancer recurrence rate from approximately 35% to less than 10% at 12 years and is a required component of BCT (*N Engl J Med.* 1995;333:1456-1456).

f. Followup after BCT. Physical examinations are the same as those for mastectomy (see earlier discussion). A posttreatment mammogram of the treated breast is performed no earlier than 6 months after completion of radiation therapy to establish a new baseline, after which annual bilateral mammograms can resume in conjunction with regular gynecologic followup (*J Clin Oncol.* 2013;31:961-965).

3. Management of the axilla. Approximately 30% of patients with clinically negative examinations will have positive lymph nodes in an **ALND** specimen. The presence and number of lymph nodes involved affect staging and thus prognosis. However, complications are not infrequent (see later discussion). Thus, **SLNB** was developed to provide sampling of the lymph nodes without subjecting patients to ALNDs.

a. The procedure requires a multidisciplinary approach, including nuclear medicine, pathology, and radiology.

(1) It involves injection of blue dye (either **Lymphazurin or methylene blue**) in the operating room and/or **technetium-labeled sulfur colloid** (in the nuclear medicine department, radiology suite, or sometimes by the surgeon). The combination of blue

dye and radioisotope provides higher node identification rates and increases the sensitivity of the procedure relative to using either agent alone. The goal is to identify the primary draining lymph node(s) in the axillary nodal basin.

(2) A variety of injection techniques are used: **Intraparenchymal** versus **intradermal** (intradermal methylene blue will cause skin necrosis at the injection site), **peritumoral** versus **periareolar.**

(3) The SLN is identified by its blue color, by high activity detected by a handheld gamma probe, and/or by a blue lymphatic seen to enter a nonblue node. Palpable nodes are also sentinel nodes even if not blue or radioactive.

(4) Twenty to 30% of the time more than one SLN is identified.

(5) Experienced surgeons (those who have performed at least 30 SLNBs with ALND for confirmation) can identify the SLN in greater than 90% of patients, accurately predicting the patients' remaining axillary lymph node status in greater than 97% of cases.

(6) Historically, **standard completion ALND was recommended if an SLNB was positive for metastasis greater than or equal to 0.2 mm; furthermore, isolated tumor cells were to be considered N0 disease and not important in the determination of**

therapeutic decisions. This remains the standard of care in many practices, but this surgical paradigm has recently been challenged by a number of studies, most significantly by the results of the **ACOSOG Z0011 trial.** This randomized trial compared the overall survival and local recurrence rates for patients with T1Ñ2 tumors and limited SLN metastatic disease who received BCT and systemic therapy and either had ALND or no further axillary procedures (*JAMA.* 2011;305:569-575.). There was no difference in the two groups, leading many to defer completion ALND for this subgroup of patients. All patients underwent lumpectomy, whole breast radiation, and systemic therapy; thus, the results cannot be generalized to all patients with a positive SLN. Nevertheless, this study contributed to growing concern that excess surgery is being performed for axillary disease. This concern has garnered further discussion following publication of the results of the **AMAROS trial,** a European randomized noninferiority trial that compared completion ALND to axillary radiation therapy for a positive SLN in patients with T1Ñ2 primary breast cancer and no palpable lymphadenopathy; rates of local recurrence were statistically equivalent and radiation was associated with less lymphedema than ALND (*Lancet Oncol.* 2014;15:1303-1310). Furthermore, the prognostic significance of **isolated tumor cells** has been brought into question by the **Dutch MIRROR cohort study,** which demonstrated an association between disease-free survival and isolated tumor cells in women who had been diagnosed at a young age and in women with triple-negative cancer who did

not undergo either ALND or axillary radiation (*N Engl J Med.* 2009;361:653-663). Finally, **the SOUND trial** is an ongoing clinical trial that is examining the extent to which SLNB can be obviated by sonographic staging of the axilla in a subset of patients (*Breast.* 2012;21:678-681; Fig. 35-5).

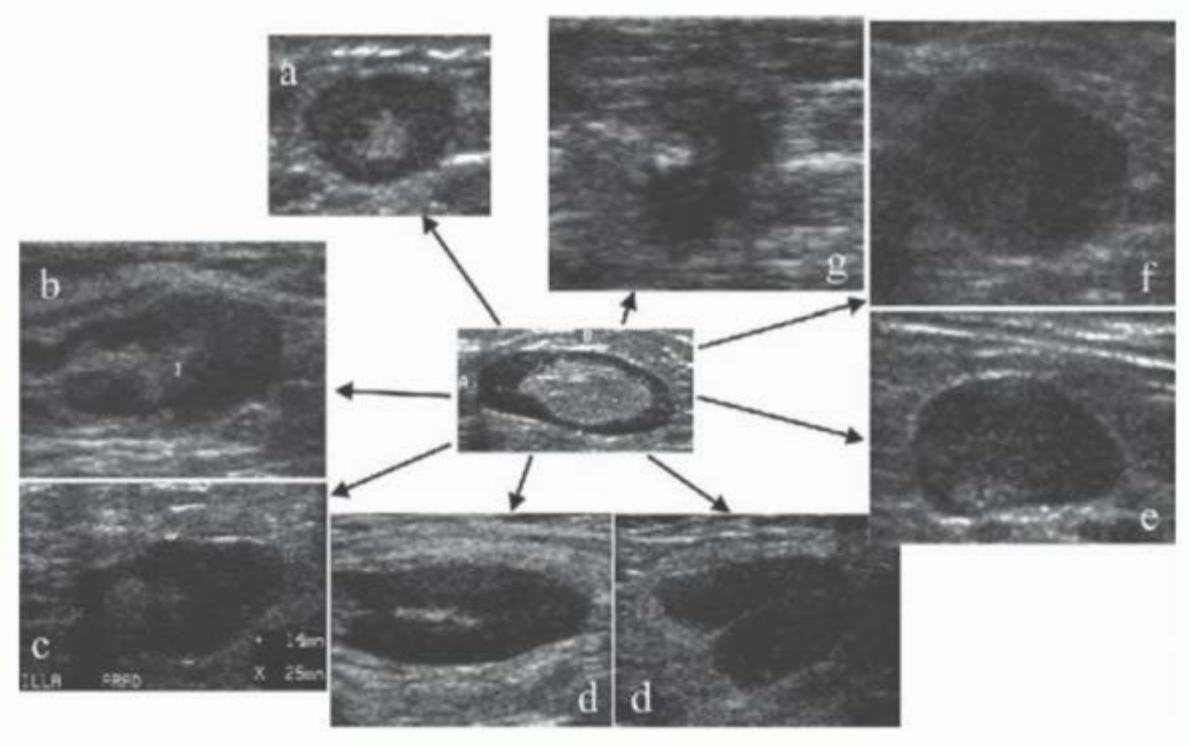


Figure 35-5 Range of abnormal sonographic lymph node appearances. (Reproduced with permission from Starvos TA. *Breast Ultrasound.* Philadelphia, PA: Lippincott Williams & Wilkins, 2003.)

b. ALND. Patients with **clinically positive lymph nodes** should undergo ALND for local control. It involves:

(1) Removal of **level I** and **level II nodes** and, *if grossly involved,* level III nodes. Motor and sensory nerves are preserved unless there is direct tumor involvement.

(2) An ALND should remove **10 or more nodes.** The number of nodes identified is often pathologist-dependent.

(3) Patients with **4 or more positive lymph nodes** should undergo **adjuvant radiation to the axilla.** Selective patients with 1 to 3 positive nodes may also benefit from radiation therapy to the axilla.

(4) The most frequent **postoperative complications** are wound infections and seromas. Persistent seroma may be treated with **repeated aspirations or reinsertion of a drain.** Other complications include pain and numbness in the axilla and upper arm, impaired shoulder mobility, and **lymphedema,** which occurs in approximately 10% to 40% of women undergoing axillary dissection; radiation to the axilla increases the risk of this complication. The most effective therapy is **early intervention with intense occupational therapy with massage;**

graded pneumatic compression devices and a **professionally fitted compression sleeve** can also provide relief and prevent worsening of lymphedema. Blood draws, blood pressure cuffs, and intravenous lines should be avoided in the affected arm, mainly to avoid infection. Infections of the hand or arm should be treated promptly and aggressively with antibiotics and arm elevation because infection can damage lymphatics further and cause irreversible lymphedema. Lymphedema increases the risk of developing **angiosarcoma.**

C. Adjuvant systemic therapy is given in appropriate patients after completion of surgery.

1. All node-positive patients should be considered for adjuvant chemotherapy.

a. Regimens are guided by the tumor biomarkers. Typical regimens consist of four to eight cycles of a combination of cyclophosphamide and an anthracycline followed by a taxane administered every 2 to 3 weeks.

b. Patients with **ER-positive tumors** receive **adjuvant hormonal therapy** for 5 to 10 years. **Tamoxifen** is given to premenopausal women, and **aromatase inhibitors** are given to postmenopausal women.

c. In postmenopausal women older than 70 years, chemotherapy is performed less frequently. In postmenopausal women with ER+ tumors, tamoxifen or an aromatase inhibitor is frequently the sole adjuvant medical therapy.

d. In patients with *Her2/neu*-**positive tumors,** polychemotherapy is combined with biologic therapy targeting the *Her2/neu* protein (see earlier discussion).

2. Node-negative patients may have increased disease-free survival from adjuvant chemotherapy and/or hormonal therapy. An individualized approach is crucial and requires thorough discussion with the patient regarding the risks of recurrence without adjuvant therapy, the cost and toxicities of treatment, and the expected benefit in risk reduction and survival.

a. Node-negative patients who are at **high risk** and benefit the most from adjuvant chemotherapy include those with tumors greater than 1 cm, high tumor grade, *Her2/neu* expression, aneuploidy, elevated Ki-67 expression, high percentage of cells in S phase, lymphovascular invasion, and ER/PR-negative tumors.

b. The NSABP B-20 trial and the International Breast Cancer Study Group trial IX showed that **polychemotherapy in combination with tamoxifen was superior to tamoxifen alone** in increasing disease-free and overall survival, especially in ER-negative patients, regardless of tumor size.

c. The St. Gallen Consensus Panel in 1997 suggested that patients who have node-negative disease and whose tumors are 1 cm or less and ER-positive may be spared adjuvant chemotherapy but still may benefit from adjuvant endocrine therapy.

d. The Web site *http://www.adjuvantonline.com* provides an online tool for physicians to use to

calculate the added benefit of hormonal and chemotherapeutic therapies.

D. Adjuvant Radiation

1. Indications for adjuvant radiation to the chest wall and axilla **after mastectomy** include T3 and T4 tumors, attachment to the pectoral fascia, positive surgical margins, skin involvement, involved internal mammary nodes, inadequate or no axillary dissection, four or more positive lymph nodes, and residual tumor on the axillary vein. Presence of one to three positive axillary nodes is a relative indication (*N Engl J Med.* 1997;337:949-955). (See earlier discussion of **radiation after BCT.**)

2. Complications. Radiation to the chest wall can cause skin changes. Infrequent complications include interstitial pneumonitis, spontaneous rib fracture, breast fibrosis, pericarditis, pleural effusion, and chest wall myositis. Radiation to the axilla can increase the incidence of lymphedema and axillary fibrosis.

E. Locally advanced breast cancer (LABC, i.e., T3, T4, N2, and/or N3 clinical disease)

1. Staging in LABC. Because up to 10% to 20% of these patients have distant metastasis at the time of presentation, all should receive a **bone scan** and **CT scan of the chest and abdomen** before treatment.

2. Noninflammatory LABC

a. Patients should receive **neoadjuvant chemotherapy** (often cyclophosphamide combined with an anthracycline [e.g., doxorubicin] and a taxane), followed by **surgery** and **radiation** as based on previously discussed criteria. Neoadjuvant chemotherapy also provides information regarding tumor response to treatment that may aid to guide further adjuvant therapy. Additional adjuvant chemotherapy is also necessary in select cases. SLNB may be used in selected patients with a clinically negative axilla, but the accuracy of this practice has been called into question by the results of **ACOSOG Z1071,** which demonstrated a false-negative rate of 12.6% in this subset of patients (*JAMA.* 2013;310:1455-1461). Work is ongoing to determine how the accuracy of axillary staging after neoadjuvant therapy might be improved.

3. Inflammatory LABC (T4d). Characterized by erythema, warmth, tenderness, and edema ([*peau d'orange*]; Fig. 35-6), it is often misdiagnosed initially as mastitis and represents 1% to 6% of all breast cancers. **Skin punch biopsy** confirms the diagnosis: In two-thirds of cases, tumor emboli are seen in dermal lymphatics. An **underlying mass** is present in 70% of cases. Associated **axillary adenopathy** occurs in 50% of cases. Approximately 30% of patients have distant metastasis at the time of diagnosis. Despite aggressive multimodal therapy, median survival is approximately 2 years, with a 5-year survival of only 5%.

4. Followup. Because of high risk for local and distant recurrence, patients should be examined every 3 months by all specialists involved in their care.

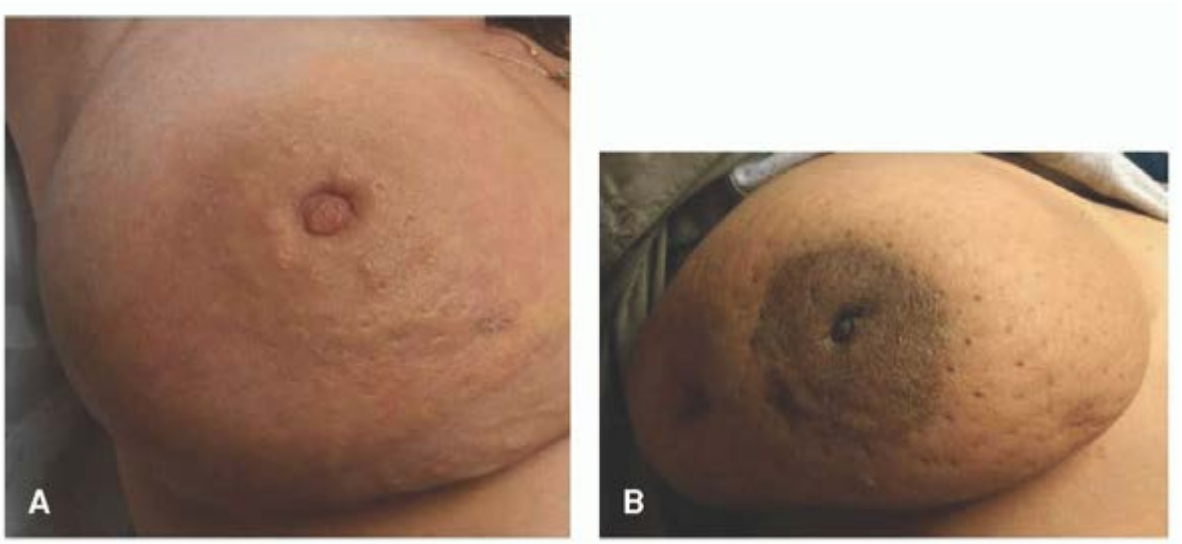


Figure 35-6 Classic clinical features of inflammatory breast cancer. (Reproduced with permission from Harris JR, Lippman ME, Osborne CK, et al, eds. *Diseases of the Breast.* Philadelphia, PA: Wolters Kluwer Health, 2014.)

F. Locoregional Recurrence. Patients with locoregional recurrence should have a **metastatic workup** to exclude visceral or bony disease and should be considered for systemic chemotherapy or hormonal therapy.

1. Recurrence in the breast after BCT requires total (simple) mastectomy. Provided margins are negative, survival is similar to that for patients who received mastectomy initially.

2. Recurrence in the axilla requires surgical resection followed by radiation to the axilla and systemic therapy.

3. Recurrence in the chest wall after mastectomy occurs in 4% to 5% of patients. One-third of these patients have distant metastases at the time of recurrence, and greater than 50% will have distant disease within 2 years. Multimodal therapy is essential. For an isolated local recurrence, excision followed by radiotherapy results in excellent local control. Rarely, patients require radical chest resection with myocutaneous flap closure.

SPECIAL CONSIDERATIONS

I. BREAST CONDITIONS DURING PREGNANCY

A. Bloody nipple discharge may occur in the second or third trimester. It results from epithelial proliferation under hormonal influences and usually resolves by 2 months postpartum. If it does not, standard evaluation of pathologic nipple discharge should be performed.

B. Breast masses occurring during pregnancy include **galactoceles, lactating adenoma, simple cysts, breast infarcts, fibroadenomas, *and* carcinoma.** Fibroadenomas may grow during pregnancy due to hormonal stimulation.

1. Masses should be evaluated by ultrasound, and a core needle biopsy should be performed for any suspicious lesion.

2. Mammography can be performed with uterine shielding but is rarely helpful due to increased breast density.

3. If a breast lesion is diagnosed as malignant, the patient should be given the **same surgical treatment** options, stage for stage, as a nonpregnant woman, and the **treatment should not be delayed** because of the pregnancy.

C. Breast cancer during pregnancy may be difficult to diagnose due to low levels of suspicion and increased breast nodularity and density.

1. It occurs in approximately 1 in 5,000 gestations and accounts for almost 3% of all breast cancers.

2. Workup is the same as in a nonpregnant woman. For advanced-stage disease, MRI scan or ultrasound may be used in lieu of CT scan for staging. Excisional biopsy can be safely performed under local anesthesia if there is some contraindication to the preferred core needle biopsy.

3. Therapeutic decisions are influenced by the clinical cancer stage and the trimester of pregnancy and must be **individualized.** The radiation component of BCT cannot be applied during pregnancy, and delaying radiation therapy is not ideal. For these reasons, BCT is usually not recommended to patients in their first or second trimester. **For patients in the third trimester, radiation can begin after delivery.** SLNB is starting to be used more frequently; the commonly used radioisotope is approved for use during pregnancy.

4. Chemotherapy may be given by the mid-second trimester.

II. PAGET DISEASE OF THE NIPPLE.

This is characterized by eczematoid changes of the nipple—areolar complex. It is almost always **accompanied by malignancy** and in 60% of cases is associated with a palpable mass. **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.** Burning, pruritus, and hypersensitivity may be prominent symptoms. Treatment should include **excision of the nipple—areolar complex** (i.e., a central lumpectomy) but is otherwise **dictated by the underlying malignancy.**

III. BREAST CANCER IN MEN.

This accounts for less than 1% of male cancers and less than 1% of all breast cancers. BRCA2 mutations are associated with approximately 4% to 6% of these cancers. **Mammography** can be helpful in distinguishing gynecomastia from malignancy. Eighty-five percent of malignancies are infiltrating ductal carcinoma. **MRM** was traditionally the surgical procedure of choice; however, SLNB has been shown to be effective in men. Thus, **total (simple) mastectomy with SLNB** is a valid option in men. **Adjuvant hormonal, chemotherapy, and radiation treatment criteria are the same as in women.** Overall **survival *per stage* is comparable to that observed in women,** although men tend to present in later stages.

IV. PHYLLODES TUMORS.

Phyllodes tumors account for 1% of breast neoplasms. They present as large, smooth, lobulated masses and may be difficult to distinguish from fibroadenomas on physical examination. FNAB cannot reliably diagnose these tumors; **at least a core needle biopsy is needed.** Ninety percent are benign; 10% are malignant, with biologic behavior similar

to that of sarcomas. Treatment is **wide local excision** to tumor-free margins or total mastectomy. **Axillary assessment is not needed** in clinically node-negative patients. Historically, there was no role for adjuvant radiation therapy, but a recent retrospective review demonstrated an association between receipt of **radiation** and improved local control (*Int J Radiat Oncol Biol Phys.* 2008;70:492-500). Tumors greater than 5 cm in diameter and with evidence of stromal overgrowth may benefit from adjuvant chemotherapy with **doxorubicin and ifosfamide** (*Cancer.* 2000;89:1502-1511). Patients should be followed with **semiannual physical examinations** and **annual mammograms and chest radiographs.**

CHAPTER 35: BREAST

Multiple Choice Questions

1. Which of the following patients should undergo radiation therapy as part of her breast cancer management?

a. A 25-year-old woman with 2 cm palpable left breast mass and history of non-Hodgkin lymphoma at 16

b. An 80-year-old woman with 6 mm of ductal carcinoma in situ (DCIS) in her right breast

c. A 32-year-old pregnant woman with T1 tumor and a suspicious lymph

node on axillary ultrasound at 33 weeks of gestation

d. A 41-year-old woman diagnosed with multicentric lobular carcinoma at 26 weeks of gestation

View Answer

2. Radiation therapy after partial mastectomy improves:

a. Rates of local recurrence

b. Overall survival

c. Risk of lymphedema after axillary lymph node dissection (ALND)

d. Breast-cancer-specific survival

View Answer

3. First-line pharmaceutical treatment for mastalgia is:

a. Oral vitamin E

b. Evening primrose oil

c. Topical vitamin E

d. Topical NSAIDs

View Answer

4. A 32-year-old breastfeeding woman who is 2 weeks postpartum presents to the emergency department with a 2-day history of warmth and erythema over the inferomedial right breast. She is afebrile and her skin is red but not edematous. Your next step in management is:

a. Incision and drainage (I&D)

b. Antibiotics, instruction to increase breastfeeding frequency

c. Antibiotics, instruction to cease breastfeeding

d. Skin punch biopsy

View Answer

5. Breast cancer in men:

a. Is associated with the BRCA2 mutation in about 5% of cases

b. Is more lethal when compared to stage-matched female controls

c. Is more likely to be infiltrating lobular carcinoma

d. Mandates radical mastectomy

View Answer

6. The most common cause of pathologic nipple discharge is:

a. DCIS

b. Lobular carcinoma in situ (LCIS)

c. Intraductal papilloma

d. Atypical ductal hyperplasia (ADH)

View Answer

7. A 75-year-old man presents with a chief complaint of bilateral breast enlargement over the past 4 months. His past medical history is significant for a history of congestive heart failure; atrial fibrillation, for which he takes digitalis; hypertension; benign prostatic hypertrophy, for which he takes finasteride; and stage 3 chronic kidney disease. Your next step in management is:

a. To perform a punch biopsy in the clinic

b. Order a stereotactic core needle biopsy

c. To call his primary care physician (PCP)

d. To inform him there is nothing to be done

View Answer

8. A 44-year-old G3P2 female whose mother was diagnosed with breast cancer at 65 comes to see you in clinic after being told that her most recent screening mammogram was read as BIRADS 3, probably benign. How do you counsel her?

a. Tell her she has nothing to worry about

b. Ask her to come back and see you in 6 months with repeat mammography

c. Start her on low-dose tamoxifen

d. Perform an ultrasound in clinic

View Answer

9. A 41-year-old female with a known history of lobular carcinoma in situ (LCIS) found on an excisional biopsy 4 months ago presents to your clinic after being referred to you by her primary care physician. She would like to undergo bilateral prophylactic mastectomy with reconstruction. Options for managing LCIS include:

a. Chemoprevention with an estrogen antagonist

b. Bilateral mastectomy

c. Close surveillance

d. All of the above

View Answer

10. Pertuzumab:

a. Is an aromatase inhibitor used in the treatment of ER+ breast cancer

b. Is associated with progression-free survival in women with metastatic HER2+ cancer when used with cyclophosphamide

c. Is associated with progression-free survival in women with metastatic HER2+ cancer when used with trastuzumab and docetaxel

d. Is still an experimental drug and not yet available in the United States (US)

View Answer

36

Skin and Soft-Tissue Tumors

Matthew S. Strand

Ryan C. Fields

DIAGNOSIS OF SKIN LESIONS AND SOFT-TISSUE MASSES

This chapter focuses on the diagnosis and management of benign and malignant cutaneous lesions and soft-tissue masses. While management of benign lesions is generally straightforward; management of malignant lesions, especially advanced forms, has become increasingly complex with the advent of new prognostic markers and therapeutic options. With more than 5 million Americans diagnosed with it annually, skin cancer represents the most common cancer in the United States, with its incidence exceeding that of lung, colon, breast, and prostate cancer combined (*Am J Prev Med.* 2014;S0749-3797(14):00510-00518). Fortunately, the vast majority of cases are curable. In contrast, soft-tissue sarcomas (STSs) are rare lesions that generally carry a poor prognosis. When a patient presents with a skin lesion or soft-tissue mass, a focused history and physical examination are crucial to derive the correct diagnosis. For the diagnosis of cutaneous lesions, biopsy remains the gold standard. For large or deep tumors, radiologic evaluation often precedes biopsy.

I. SKIN LESIONS

A. History. Pigmented lesions with a change in size, borders, and/or color are of concern for malignancy. Itching, bleeding, or ulceration should be assessed.

B. Physical Examination. The color, size, shape, borders, elevation, location, firmness, and surface characteristics should be noted. Photographs are helpful. Uniformly colored, small, round, circumscribed lesions are more likely to be benign. Irregularly colored, larger, asymmetric lesions with indistinct borders and ulceration are worrisome for malignancy. These features can be remembered using the mnemonic ABCDE: Asymmetry, (irregular) Borders, Color variation, Diameter, and Evolution (https://www.aad. org/spot-skin-cancer/understanding-skin-cancer/how-do-i-check-my-skin/what-to-look-for) (Fig. 36-1). In addition, melanoma can manifest as pigmented subungual or mucosal lesions.

C. Biopsy. Biopsy is warranted for lesions that have worrisome features or that change over time. Optimally, full-thickness tissue is obtained via punch or excisional biopsy. Biopsy should include the thickest portion of the lesion, avoiding areas of crusting, ulceration, or necrosis. Excisional biopsies of the extremity must be oriented parallel to the long axis to facilitate possible

need for subsequent definitive resection. Critical to the performance of the biopsy is the determination of the **Breslow depth;** hence superficial shave biopsies should be avoided while deep shave biopsies are usually adequate (*J Am Coll Surg.* 2011;212(4):454-460).

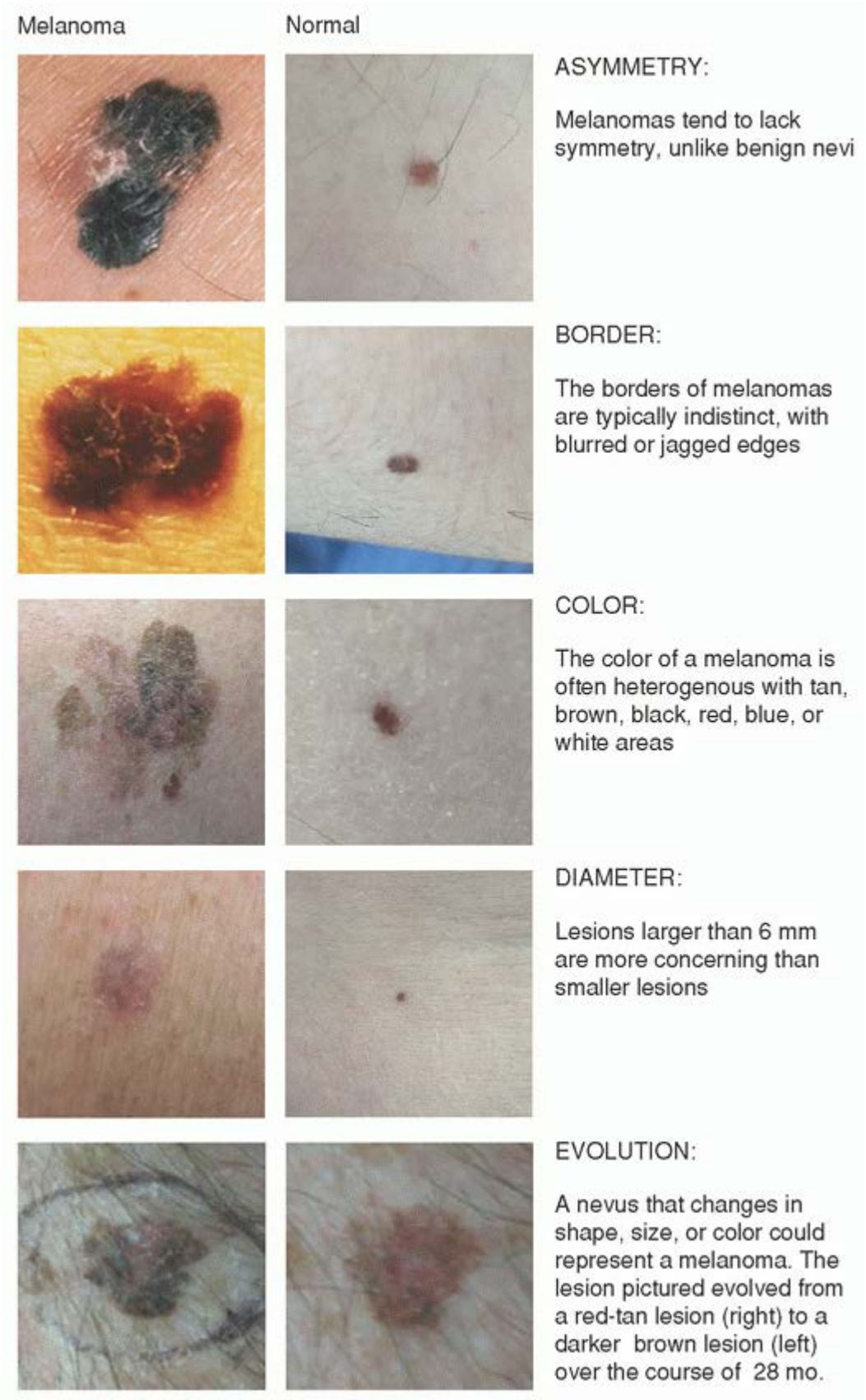


Figure 36-1 ABCDEs of Melanoma.

II. SOFT-TISSUE MASSES

A. History. A focused history includes descriptions of location, duration, change in size, and presence of associated symptoms. An enlarging, painless mass is the most common presentation. Pain is usually a late symptom. Lesions may be misdiagnosed as hematomas or muscle strain due to perceived antecedent trauma. Any symptom or perception of enlargement is concerning for malignancy.

B. Physical Examination. Key features are size, anatomic relationships with surrounding structures, borders, and mobility. A neurovascular examination of the affected area should be performed.

C. Radiologic Evaluation

1. Magnetic resonance (MR) scan is the best choice for imaging soft-tissue masses. T2-weighted images and gadolinium enhancement can help distinguish edematous normal tissue from tumors.

2. Computed tomography (CT) is used to assess the character and extent of larger, deeper tumors. CT-guided core-needle biopsy is useful for tumors with difficult surgical access. Chest CT should be obtained for patients with STSs for staging.

D. Biopsy

1. Excisional biopsy is performed for tumors that are probably benign or less than 3 cm in diameter. Typically, an elliptical incision is made around the tumor oriented parallel to the long axis of the limb and along skin lines of minimal tension. Complete excision with a thin margin of normal tissue followed by primary closure should be performed whenever possible.

2. Incisional biopsy is the gold standard for masses greater than 3 cm. An incision oriented parallel to the long axis of the extremity should be made such that the resulting scar can be excised at subsequent operation. Meticulous hemostasis to prevent hemorrhage from spreading tumor is critical. Drains should be avoided, but if needed, drain sites should be placed for excision at subsequent operation.

3. Core-needle biopsy provides a section of intact tissue for histologic analysis; it can provide the same information as an incisional biopsy if adequate. Indeterminate results should be confirmed by incisional or excisional biopsy.

4. Fine-needle aspiration (FNA) is the least invasive method of tissue diagnosis but is often the least informative. FNA can often determine the presence of malignancy and histologic type but often cannot determine grade. Indeterminate results should prompt definitive biopsy. FNA is the biopsy method of first choice in the head and neck.

BENIGN SKIN AND SOFT-TISSUE LESIONS

I. SEBORRHEIC KERATOSES.

Seborrheic keratoses are benign skin growths that characteristically appear in older people as multiple, raised, irregularly rounded lesions with a verrucous, friable, waxy surface, and variable pigmentation on the face, neck, or trunk. No treatment is indicated for most lesions. If treatment is desired, surgical excision, curettage followed by electrodesiccation, topical trichloroacetic acid, or cryotherapy with liquid nitrogen may be employed. The sudden development of multiple seborrheic keratoses in conjunction with acanthosis nigricansÑthe LeserÑTrelat signÑcan represent a paraneoplastic syndrome.

II. ACTINIC KERATOSES.

Actinic keratoses result from sun exposure and are found predominantly in elderly, fair-skinned patients. Lesions are small, usually multiple, flat-to-slightly elevated with a scaly surface ranging from red to yellowish brown to black. Unlike seborrheic keratoses, these lesions have malignant potential; up to 20% become squamous cell carcinoma. Benign-appearing actinic keratoses may be observed; treatments include excision, cryotherapy, dermabrasion, topical applications of imiquimod or 5-fluorouracil, and photodynamic therapy.

III. NEVI.

Junctional nevi are small (<6 mm), well-circumscribed, light brown or black macules found on any area of the body. Nevi rarely develop in people older than 40 years old, so any new lesion in this population should be considered a possible melanoma.

IV. CUTANEOUS CYSTS.

Cutaneous cysts may be of epidermal, dermal, or trichilemmal origin. Symptomatic or infected cysts should be removed or drained. Asymptomatic cysts may be removed for diagnosis, prevention of infection, or cosmesis. Excision should include the entire cyst, its lining, and any skin tract or drainage site to prevent recurrence.

V. NEUROFIBROMAS.

Neurofibromas are benign tumors seen most frequently in patients with neurofibromatosis. Neurofibromas are soft, pendulous, sometimes lobulated subcutaneous masses of variable size. Neurofibromas may be removed for pain, increases in size, or cosmesis.

VI. GANGLION CYSTS.

Ganglion cysts are subcutaneous cysts attached to the joint capsule or tendon sheath of the hands and wrists and are most common among young and middle-aged women. These lesions present as firm, round masses of the hand and wrist. To prevent recurrence, which is rare, the

capsular attachment and a small portion of the joint capsule should be removed.

VII. LIPOMAS.

Lipomas are benign tumors consisting of fat and are perhaps the most common human neoplasms. Malignant potential is rare; sarcomatous elements occur in less than 1%. They are soft, mobile subcutaneous masses of variable size. Asymptomatic small tumors can be observed, but large (>5 cm), symptomatic, or growing tumors are concerning and should be biopsied or removed. Lipomas must be excised cleanly at the first operation to prevent recurrence.

MALIGNANT LESIONS

I. DERMATOFIBROSARCOMA PROTUBERANS.

Dermatofibrosarcoma protuberans (DFSP) is a locally aggressive cutaneous sarcoma that rarely metastasizes but can recur locally. Because of the infiltrative nature of the tumor, excision should include 2 to 3 cm margins to achieve a high rate of R0 resection. Tumor depth for primary DFSP and margin positivity in recurrent DFSP are independent predictors for recurrence (*Ann Surg Oncol.* 2011;18(2):328-336). Radiation therapy can be employed for patients with microscopically positive margins but only one in three patients may benefit; therefore, the morbidity of radiation must be weighed against the possible need for additional resection. Mohs microsurgery can be employed to improve tissue conservation, cosmesis, and may actually improve recurrence rates (*Arch Dermatol.* 2012;148(9):1055).

II. DESMOID TUMORS.

Desmoid tumors are nonmetastasizing, locally aggressive tumors that arise from connective tissue. Excision with a margin of normal tissue should be performed. Local recurrences are common, requiring reexcision. Desmoids are radiosensitive, and radiation therapy can be used in the primary, adjuvant, or neoadjuvant setting. Anti-estrogen-receptor agents (e.g., Tamoxifen), nonsteroidal anti-inflammatory drugs (NSAIDs, e.g., sulindac), and a combination of both types of medications have been used with anecdotal success and may be used for recurrent or unresectable disease. Patients with a desmoid tumor require colonoscopy to exclude familial adenomatous polyposis (FAP; *Fam Cancer.* 2006;5:275).

III. MELANOMA.

The incidence of melanoma continues to rise at an epidemic rate. Melanoma represents the fifth-most common type of cancer in the United States (*CA Cancer J Clin.* 2014;64(1):9-29).

A. Lesions. Most pigmented lesions are benign, but approximately one-third of all melanomas arise from pigmented nevi. Amelanotic melanoma represents 2% of all melanomas.

1. Premalignant lesions

a. Dysplastic nevi have variegated color (tan to brown on a pink base), are large (5 to 12 mm), appear indistinct with irregular edges, and have macular and papular components. **Congenital nevi** (ÒbirthmarksÓ) are associated with an increased risk of melanoma developing from these lesions, particularly for giant congenital nevi, which have diameters greater than 20 cm and are sometimes hairy. Biopsied nevi that reveal moderate or severe dysplasia should be excised to negative margins.

2. Malignant lesions

a. While malignant melanoma was historically classified into histological subtypes (superficial spreading, nodular, lentigo maligna, and acral lentiginous), this convention is falling out of favor due to the recognition that all subtypes have similar outcomes when controlled for stage, Breslow depth, and presence of ulceration and mitoses (*J Clin Oncol.* 2009;27:6199-6206; *Ann Surg Oncol.* 2010;17:1475-1477; *Ann Surg Oncol.* 2010;17:2006-2014). Instead, new molecular

classification schemes including mutational analysis for genes such as BRAF, NRAS, c-Kit, and CDK4 are becoming relevant in the development of targeted therapies.

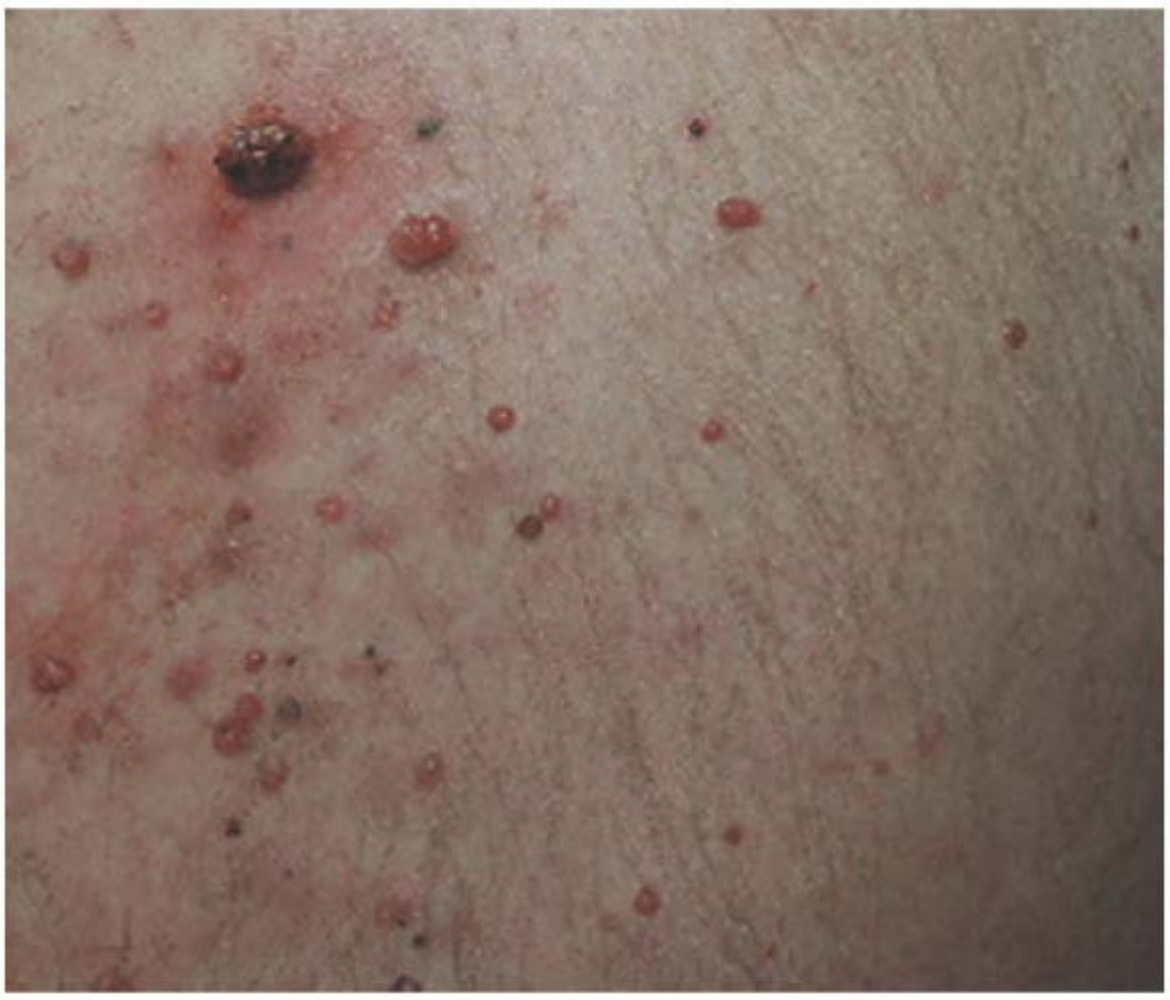


Figure 36-2 Cutaneous melanoma of the thigh with satellitosis.

b. In-transit metastases and **satellite lesions** (**Fig 36-2**) signify a poor prognosis with a high risk of local recurrence and distant metastasis. The distinction between the two is historical, as there is no difference in mechanism or outcome.

B. Risk Factors. A history for melanoma should include an assessment of risk factors and family history.

1. Each of the risk factors listed below carries at least a three-fold increase in the risk for melanoma; the presence of three or more risk factors carries approximately 20 times the risk (*Curr Probl Surg.* 2006;43:781).

a. Family or personal history of melanoma.

b. Blond or red hair.

c. Freckling of the upper back.

d. Three or more blistering sunburns before age 20.

e. Presence of actinic keratosis.

f. Blue, green, or gray eyes.

A recent meta-analysis demonstrated that individuals with at least one dysplastic nevus had a relative risk of 3.63 for melanoma compared to individuals who had none (*Cancer Prev Res.* 2010;3(2):233-245). Melanoma is familial in approximately 10% of cases, and in these cases, it is often associated with multiple atypical moles. **Familial atypical multiple-mole melanoma syndrome (FAMMM)** is a syndrome that causes a predisposition for melanoma. FAMMM is defined using the following criteria: (1) 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 with particular histopathologic features including architectural disorder with asymmetry, subepidermal fibroplasia, and lentiginous melanocytic hyperplasia with spindle or epithelial melanocyte nests. For patients who have a large number of moles, baseline photographs or computerized scanning are helpful. Patients with FAMMM should avoid sun exposure and undergo regular ophthalmic examinations due to the increased risk of ocular involvement. A proportion of those with FAMMM have germline mutations in p16, a cyclin-dependent kinase inhibitor, accounting for about 40% of melanoma-prone families (*J Med Genet.* 44:99-106).

C. Clinical Features. See ÒSkin Lesions, Physical ExaminationÓ above.

D. Staging and Prognosis. Tumor thickness is the most important factor in staging the tumor, both for overall survival and risk for nodal and distant metastasis (*J Clin Oncol.* 2001;19:3622). Tumors less than 1 mm thick have a 10-year survival of 92%, whereas lesions more than 4 mm thick have a 10-year survival of 50% (*J Clin Oncol.* 2009;27:6199). The **Breslow thickness,** a physical depth measurement of the primary tumor, is used to classify the tumor (ÒT ClassificationÓ in Table 36-1). Other than Breslow depth, ulceration, and number of mitoses are important histological characteristics that are negative prognostic factors. The American Joint Committee on Cancer (AJCC) TNM (tumor, node, metastasis) classification system (Tables 36-1 and 36-2) is the standard classification scheme. Other negative prognostic factors include older age, male gender, satellitosis, ulceration, and location on the *b*ack, posterolateral *a*rm, *n*eck, or *s*calp (**the BANS regions**). Regional node metastasis severely worsens prognosis (5-year survival, 40% to 70%) while distant metastases have a dismal prognosis (median survival 2 to 11 months).

E. Treatment (Fig 36-3)

1. Surgery

a. Wide local excision is the primary treatment for most melanomas and premalignant lesions. The surgical margin depends on the Breslow tumor thickness: Melanoma in situ (MIS) should be excised with 5 mm margins, thin melanomas (Breslow thickness <1 mm) should have a margin of 1 cm; lesions thicker than 1 mm should have a margin of at least 2 cm. A seminal trial addressed the efficacy of 2-cm versus 4-cm margins for Breslow thickness 1 to 4 mm (*Ann Surg.* 1993;218:262), demonstrating no significant difference in local recurrence rate, disease-free survival, or overall survival between the two groups at 10 years (*Ann Surg Oncol.* 2001;8:101). Adequacy of a 2-cm margin was confirmed in a randomized controlled trial of patients with thick (>2 mm) melanomas (*Lancet.* 2011;378(9803):1635). Wounds should be closed primarily, with flaps or skin grafts reserved for large defects. Mohs micrographic surgery can be employed where wide and deep excisions are difficult, such as the face, or for MIS, but recurrence rates are variable, ranging from 0% to 33% (*Int J Dermatol.* 2010;49:482). Randomized trials evaluating surgical margins for primary melanoma are tabulated below (Table 36-3; *Cochrane Database Syst Rev.* 2009;7(4):CD004835; *Lancet.* 2011;378(9803):1635).

TABLE 36-1 American Joint Committee on Cancer TNM (Tumor, Node, Metastasis) Definitions of Melanoma

T Classification	Ñ	Ñ

Tis	Melanoma in situ	Ñ
T1	²1.0 mm	a. Without ulceration and mitosis $<1/mm^2$
	Ñ	b. With ulceration or mitoses $^3 1/mm^2$
T2	1.01-2.0 mm	a. Without ulceration
	Ñ	b. With ulceration
T3	2.01-4.0 mm	a. Without ulceration
	Ñ	b. With ulceration
T4	>4.0 mm	a. Without ulceration
	Ñ	b. With ulceration
Regional Lymph Nodes (N)	Ñ	Ñ
N1	One lymph node	a. Micrometastasis[a]
	Ñ	b. Macrometastasis[b]
N2	2-3 lymph nodes	a. Micrometastasis[a]
	Ñ	b. Macrometastasis[b]
	Ñ	c. In-transit met(s)/satellite(s) without metastatic lymph node(s)

N3	³4 metastatic lymph nodes, matted lymph nodes, or in-transit met(s)/satellite(s) with metastatic lymph node(s)	
Distant Metastasis (M)	Ñ	Ñ
M1a	Distant skin, subcutaneous, or lymph node mets	Normal LDH
M1b	Lung mets	Normal LDH
M1c	All other visceral mets	
Any distant mets	Normal LDH	
Elevated LDH		

[a] Micrometastases are diagnosed after sentinel or completion lymphadenectomy (if performed).

[b] Macrometastases are defined as clinically detectable lymph node metastases confirmed by therapeutic lymphadenectomy or when any lymph node metastasis exhibits gross extracapsular extension.

LDH, lactic dehydrogenase; mets, metastases.

TABLE 36-2 American Joint Committee on Cancer Stage Groupings for Cutaneous Melanoma

Stage		Clinical Staging[a]		Pathologic Staging[b]		
0	Tis	N0	M0	Tis	N0	M0
IA	T1a	N0	M0	T1a	N0	M0
IB	T1b	N0	M0	T1b	N0	M0
	T2a	N0	M0	T2a	N0	M0
IIA	T2b	N0	M0	T2b	N0	M0
	T3a	N0	M0	T3a	N0	M0
IIB	T3b	N0	M0	T3b	N0	M0
	T4a	N0	M0	T4a	N0	M0
IIC	T4b	N0	M0	T4b	N0	M0
III[c]	Any T	³N1	M0	Ñ	Ñ	Ñ
IIIA	Ñ	Ñ	Ñ	T1-4a	N1a	M0
Ñ	Ñ	Ñ	Ñ	T1-4a	N2a	M0
IIIB	Ñ	Ñ	Ñ	T1-4b	N1a	M0
Ñ	Ñ	Ñ	Ñ	T1-4b	N2a	M0
Ñ	Ñ	Ñ	Ñ	T1-4a	N1b	M0

Ñ	Ñ	Ñ	Ñ	T1-4a	N2b	M0
Ñ	Ñ	Ñ	Ñ	T1-4a	N2c	M0
IIIC	Ñ	Ñ	Ñ	T1-4b	N1b	M0
Ñ	Ñ	Ñ	Ñ	T1-4b	N2b	M0
Ñ	Ñ	Ñ	Ñ	T1-4b	N2c	M0
Ñ	Ñ	Ñ	Ñ	Any T	N3	M0
IV	Any T	Any N	M1	Any T	Any N	M1

[a] Clinical staging includes microstaging of the primary melanoma and clinical/radiologic evaluation for metastases. By convention, it should be used after complete excision of the primary melanoma with clinical assessment for regional and distant metastases.

[b] Pathologic staging includes microstaging of the primary melanoma and pathologic information about the regional lymph nodes after partial or complete lymphadenectomy, except for pathologic stage 0 or stage Ia patients, who do not need pathologic evaluation of their lymph nodes.

[c] There are no stage III subgroups for clinical staging.

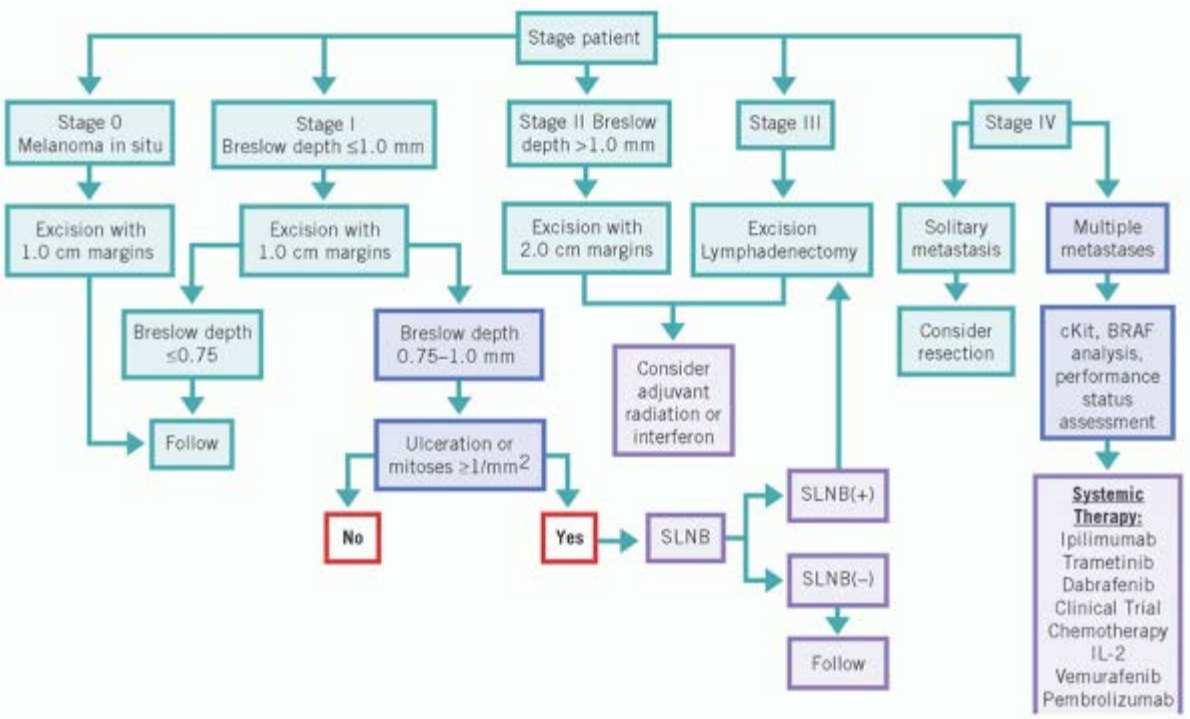


Figure 36-3 Therapeutic Decision Algorithm for Cutaneous Melanoma.

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b. Sentinel lymph node biopsy (SLNB). SLNB has greatly enhanced accurate staging of patients with melanoma and has replaced elective lymph node dissection (ELND), which is lymph-node dissection

P.678

performed in the absence of clinically positive nodes and which has not been associated with any survival benefit. This technique is based on the lymphatic drainage of melanomas to specific, initial lymph node(s), termed the *sentinel lymph node(s),* before further spread. The histology of the SLN is highly reflective of the rest of the nodal basinÑthe incidence of a positive nonsentinel node in the setting of a negative sentinel node is less than 1% (*Arch Surg.* 1992;127(4):392). A negative SLN obviates radical and morbid lymphadenectomy. The SLN can be accurately identified 96% of the time using radiolymphoscintigraphy and intraoperative dye injection with radioprobe guidance. SLNB is most beneficial for intermediate-thickness melanomas (Breslow thickness 1 to 4 mm) (*Ann Surg.* 2001;233:250). Data from the Multicenter Sentinel Lymphadenectomy Trial (MSLT)-I support the role of SLN and immediate (vs. delayed) complete lymphadenectomy if the SLN is positive. In MSLT-I, 1,269 patients with intermediate-thickness melanomas (1.2 to 3.5 mm) were randomized to either wide excision only followed by observation or to wide excision and SLNB. In the wide-excision plus observation-only group, complete lymphadenectomy was performed only when there was clinical evidence of nodal recurrence (delayed), whereas the SLNB group underwent a complete (immediate) lymphadenectomy if nodal micrometastases were

detected in any SLN. A 10-year disease-free survival rate was significantly higher in the SLNB group than in the observation group (71.3% vs. 64.7%, respectively; $p = 0.01$; *N Engl J Med.* 2014;370:599-609). The presence of metastatic disease within the SLN was found to be the most important prognostic factor for overall survival. The 5-year survival rate was 72.3% in patients with tumor-positive SLNs and 90.2% in those with tumor-negative SLNs. For thin melanomas (²1 mm thickness), the incidence of positive SLN is only 2% to 5% (*Surg Oncol Clin N Am.* 2007;16:35). Current National Comprehensive Cancer Network (NCCN) practice guidelines recommend that SLNB be considered for patients with high-risk stage IA melanoma and discussed and offered to patients with stage IBÑIIC melanomas (*NCCN Clinical Practice Guidelines in OncologyÑMelanoma.* 2015, v. 1). Melanomas <0.75 mm rarely have mitoses or ulceration and nodal metastases are exceedingly rare, hence SLNB is not recommended (see consensus guidelines of the American Society of Clinical Oncology and Society of Surgical Oncology; *J Clin Oncol.* 2012;30(23):2912-2918). MSLT-I also demonstrated that only 12% of sentinel node positive patients have additional nodal disease. On this basis, MSLT-II was designed to assess the need for therapeutic lymph node dissection in SLNB positive patients. In this trial, patients with positive SLNB were randomized to observation versus completion lymphadenectomy. Accrual has completed, but interim data are not expected until 2017.

TABLE 36-3 Randomized Controlled Trials and Melanoma Excision Margins

Author, Year	No. of Patients	Melanoma Thickness	Margins	Findings
Cascinelli et al., 1998	612	²2 mm	1 vs. 3 cm	1-cm margin is safe for melanomas <2 mm in thickness; melanomas >2 mm require margins ³2 cm
Cohn-Cedermark et al., 2000	989	>8 mm, ²2 mm	2 vs. 5 cm	2-cm margins are safe in this population; recurrence rate and survival were not different than 5-cm margins
Balch et al., 2000	486	1-4 mm	2 vs. 4 cm	2-cm margin is safe for melanomas >2 mm in thickness

Khayat et al., 2003	337	[2]2 mm	2 vs. 5 cm	5-cm margins offer no recurrence or survival benefit, 2-cm margins are adequate
Thomas et al., 2004	900	[3]2 mm	1 vs. 3 cm	3-cm margins reduce regional recurrence; 3-cm margins tended to favor survival, but this was not statistically significant
Gillgren et al., 2011	936	[3]2 mm	2 vs. 4 cm	2-cm margins are sufficient for patients with melanoma >2 mm thick

c. Therapeutic lymph node dissection should be performed for involved axillary and superficial inguinal lymph nodes unless

unresectable distant metastases are present. Therapeutic LND can achieve 5-year survival rates of 20% to 40% (*Ann Surg Oncol.* 1998;5:473; *Eur J Surg Oncol.* 2007;33(1):102-108). Surgical therapy of the inguinal region includes a superficial inguinal lymphadenectomy with extension to the deep ilioinguinal region if there is clinical, radiographic, or intraoperative evidence of lymph node involvement. Intraoperative pathologic analysis of clinically suspicious lymph nodes or Cloquet node may help determine the need for deep dissection.

d. Complications of melanoma excision include functional disability as well as complications potentially inherent to any surgery including infection and difficulty with wound healing. Local recurrence warrants reexcision, while lymphedema complicating a lymphadenectomy is managed with compression garments, exercise, and physiotherapy.

e. Resection of metastases. The surgical options for patients with metastatic melanoma can be divided into two categories: Curative or palliative. Curative-intent surgery for metastatic melanoma should carefully weigh the risks and benefits of surgery. Favorable factors include long disease-free intervals, fewer metastatic sites, and good functional status (*Cancer Treat Rev.* 2008;34(7):614-620). With the advent of more effective systemic therapy, surgery for selected, refractory metastatic disease is becoming more common.

2. Isolated limb perfusion (ILP) or isolated limb infusion (ILI) with melphalan, an alkylating agent, can be used for recurrent or in-transit extremity melanoma that is locally advanced and unresectable. ILP and ILI deliver high-dose regional chemotherapy to the affected

extremity while minimizing systemic toxicity. ILI is simpler and less toxic than ILP, but its response rates are lower (*J Am Coll Surg.* 2009;208(5):706). A review of ILP and ILI including the agents used, technique employed, and treatment algorithms was published by Tyler et al. in 2011 (*Surg Oncol Clin N Am.* 2011;20(1):79-103).

3. Immunotherapy. Ipilimumab, a monoclonal antibody against CTLA-4, and **pembrolizumab,** a monoclonal antibody against PD-1, have shown clinical efficacy in the treatment of melanoma and have been approved by the FDA. A placebo-controlled phase III trial of ipilimumab including 676 patients with metastatic melanoma showed a significant survival benefit. In this trial, patients received ipilimumab alone, ipilimumab plus glycoprotein 100 (gp100) vaccine, or gp100 vaccine alone. At 12 months, survival rates were 44%, 46%, and 25% respectively and at 24 months they were 22%, 24%, and 14% (*N Engl J Med.* 2010;363(8):711). An additional phase III trial was conducted comparing ipilimumab plus dacarbazine to placebo plus dacarbazine. Survival rates were significantly higher in the group receiving ipilimumab plus dacarbazine at 1-, 2-, and 3-year followup (47% vs. 36%, 29% vs. 18%, and 21% vs. 12%, respectively; *N Engl J Med.* 2011;364(26):2517). Randomized trials have also demonstrated the efficacy of pembrolizumab, especially in patients with

ipilimumab-refractory disease. A trial in which 540 ipilimumab-refractory patients were randomized to receive pembrolizumab 2 mg/kg, pembrolizumab10 mg/kg, and chemotherapy showed that pembrolizumab significantly reduced progression at 6 months compared to chemotherapy (*J Transl Med.* 2015;13:2062). Studies combining agents that target CTLA-4 and PD-1 or PDL-1 in conjunction with vaccines are currently ongoing and may improve therapeutic response.

Interferon-alpha (IFN-α) therapy is considered standard adjuvant therapy for patients with known nodal disease or at high risk for regional recurrence (primary melanoma >4 mm thick, ulceration, or elevated mitoses. Two randomized controlled trials have shown the benefit of IFN-α in select populations. Garbe et al. compared surgery alone to adjuvant INF-2α with or without dacarbazine. Overall survival in the IFN-2α group was significantly higher than in the surgery alone group at the 4-year mark (59 vs. 42%, P = 0.0045; *Ann Oncol.* 2008;19(6):1195-1201). Pegylated IFN-α2b adjuvant therapy versus surgery alone was compared in a phase III randomized trial of patients with resected stage III melanoma. At 4 years, patients in the IFN-α2b had improved recurrence-free survival (45.6% vs. 38.9%, P = 0.1); however, no survival benefit was seen (*Lancet.* 2008;372(9633):117-126). Combination therapy with ipilimumab as adjuvant therapy is currently ongoing in ECOG 1609.

4. Targeted molecular therapy. Testing for BRAF and NRAS mutations is now standard for patients with metastatic melanoma due to the advent of targeted therapies. BRAF and NRAS are proto-oncogenes that have been found to be activated about 40% and 15% of melanomas, respectively (*J Clin Oncol.* 2012;30(20):2522-2529). **Vemurafenib** and **dabrafenib** are small molecule inhibitors of BRAF that have shown clinical efficacy in patients with the V600 mutation in

BRAF. **Cobimetinib** and **Trametinib** are small molecule inhibitors of MEK, an effector just downstream of BRAF. Combination therapies with BRAF and MEK inhibitors have demonstrated improved efficacy over BRAF therapy alone for patients with V600 BRAF mutations in two recent randomized controlled trials (*N Engl J Med.* 2014;371(20):1877-1888; *N Engl J Med.* 2014;371(20):1867-1876). Another small molecule inhibitor, **imatinib** has also been efficacious in patients with a KIT mutation more prevalent in acral and mucosal melanomas. Generally these treatments are reserved as second-line therapy after immunotherapy but represent important advances in the application of genetic data to individualize care.

OTHER MALIGNANT SKIN TUMORS

I. BASAL CELL CARCINOMA.

Basal cell carcinoma is the most common skin cancer. They are slow-growing, may be large, disfiguring, and locally invasive, but rarely metastasize (<0.1%). Sun exposure is the most significant epidemiologic factor; consequently, this neoplasm is found most commonly on sun-exposed areas in fair-skinned patients older than 40 years.

A. Lesions. Lesions are flat but indurated with a smooth, whitish, waxy surface and indistinct borders. The noduloulcerative form is the most common and is characterized by shiny, translucent nodules with a central umbilication that often becomes ulcerated, with pearly, rolled, telangiectatic edges. The morpheaform variant is important to identify because it is more aggressive.

B. Treatment

1. Excisional biopsy is adequate for small tumors, with intraoperative frozen-section analysis (to confirm negative margins) and primary closure. Larger tumors may require incisional or punch biopsy followed by excision. A margin of 2 to 4 mm should be obtained, and positive margins should be reexcised.

2. Other options for treatment include Mohs micrographic surgery (useful for recurrent tumors or cosmetically sensitive areas), curettage with electrodessication, liquid nitrogen therapy, topical 5-FU or imiquimod (for small tumors), or radiation or photodynamic therapy for unresectable lesions or if treatment intent is palliative. The small molecule inhibitor **Vismodegib** is a relatively new therapy that has shown efficacy in patients with inoperable BCC (*N Engl J Med.* 2012;366(23):2171-2179). This has proven very useful in patients with Gorlin syndrome, a familial form of BCC.

II. SQUAMOUS CELL CARCINOMA.

Squamous cell carcinoma is the second-most common skin cancer. Sunlight is the major etiology, and elderly men with a history of chronic sun exposure are the most at risk. Squamous cell

carcinoma is usually found on sun-exposed areas and may develop from draining sinuses, radiation, chronic ulcers, and scars (Marjolin ulcer).

A. Lesions. Lesions are small, firm, erythematous plaques with a smooth or verrucous surface and indistinct margins with progression to raised, fixed, and ulcerated lesions. Most are preceded by actinic keratoses that progress into slow-growing, locally invasive lesions. Although rare, nodal metastases do occur more frequently than in basal cell carcinoma. Large and poorly differentiated tumors carry a higher risk and should be staged with imaging; SLNB in these cases should be considered.

B. Treatment mirrors that for basal cell carcinoma. Margins of 4 to 6 mm for cancers at low risk for invasion or spread while high-risk patients require larger margins. Risk factors associated with poor prognosis include size, a lesion's being recurrent, immunosuppression, rapid growth, neurologic symptoms, and pathologic factors. Mohs microsurgery is indicated for high-risk lesions or for cosmetically sensitive areas. Nonsurgical options are identical to those for basal cell carcinoma. Solitary metastases should be resected because of a relatively high cure rate.

III. MERKEL CELL CARCINOMA.

Merkel cell carcinoma is a rare, aggressive skin cancer. UV radiation, immunosuppression, and exposure to Merkel cell polyomavirus are predisposing factors. The cancer often recurs locally and

metastasizes to regional lymph nodes. Treatment mirrors melanoma, consisting of excision and SLNB, with completion lymphadenectomy reserved for clinical adenopathy or positive sentinel nodes. SLNB is positive in about 29% of patients with clinically localized disease (*Ann Surg Oncol.* 2011;18(9):2529). Because these lesions are highly radiosensitive, radiotherapy can be used for treatment of high-risk lesions or nodal basins or in patients who have unresectable disease or are otherwise not operative candidates. Interestingly, lymphovascular invasion, rather than nodal status, correlates better with poor prognosis (*Ann Surg.* 2011;254(3):465-473).

SOFT-TISSUE SARCOMAS

STSs represent a heterogeneous group of malignant tumors derived from mesodermal tissues. STSs are rare, constituting approximately 1% of adult malignant neoplasms. While most of these tumors occur de novo, some are seen as part of syndromesÑsuch as neurofibromatosis, Werner syndrome, or Li-Fraumeni syndromeÑthat predispose those with them to particular malignancies. Lymphedema and radiation have been shown to be causative in certain subtypes (*Am Surg.* 2006;72:665). Nodal metastases are rare in STS as dissemination usually occurs hematogenously. The epidemiology and natural history of this diverse group of malignancies have been reviewed in a series of 10,000 patients spanning 30 years at a single institution (*Ann Surg.* 2014;260(3):416-421).

I. LESIONS.

Sarcomas are classified by histologic cell type of origin and grade. Pleomorphic undifferentiated sarcoma (previously malignant fibrous histiocytoma, 40%) is most common, followed by liposarcoma (25%). Patients typically present with an asymptomatic mass that becomes visible or palpable. Retroperitoneal tumors can become massive before symptoms prompt presentation.

II. DIAGNOSIS.

Biopsy (usually core or incisional) is necessary for diagnosis. Care is needed to orient incisions to aid in definitive resection.

III. STAGING AND PROGNOSIS.

The AJCC staging system is based on tumor size and depth, nodal status, histologic grade, and metastasis (Table 36-4). Of these, tumor grade is the major prognostic factor and is based on mitotic index, nuclear morphology, and degree of anaplasia.

A. Staging includes physical examination and CT or MR scan to assess the size and extent of tumor. As hematogenous spread to the lungs is the most common form of metastasis, chest CT is required for grade II and III lesions. Abdominal CT scan is needed to evaluate retroperitoneal sarcomas, in which hepatic metastases are more common than in other STS.

B. Prognosis. Almost 80% of metastases are to the lungs and occur within 2 to 3 years of diagnosis. If pulmonary disease is resectable, overall survival is 30% at 3 years. In addition, tumor size, grade, tumor rupture during surgery, margins after resection, and anatomic location all influence local recurrence, overall survival, and tumor-free survival. Retroperitoneal and truncal STS have worse prognoses than extremity STS. While mortality from extremity STS is usually due to metastasis, retroperitoneal STS is usually fatal secondary to local recurrence.

TABLE 36-4 American Joint Committee on Cancer Staging System for Soft-Tissue Sarcoma

Tumor Grade (G)	Stage IA
GX: Grade cannot be assessed	G1/GX, T1a, N0, M0
G1: Well differentiated	G1/GX, T1b, N0, M0
G2: Moderately differentiated	**Stage IB**

G3: Poorly differentiated	G1/GX, T2a, N0, M0
G4: Undifferentiated	G1/GX, T2b, N0, M0
Primary Tumor (T)	**Stage IIA**
TX: Primary tumor cannot be assessed	G2-3, T1a, N0, M0
T0: No evidence of primary tumor	G2-3, T1b, N0, M0
T1: Tumor >5 cm in greatest dimension	**Stage IIB**
T1a: Superficial tumor[a]	G2, T2a, N0, M0
T1b: Deep tumor[a]	G2, T2b, N0, M0
T2: Tumor >5 cm in greatest dimension	**Stage III**
T2a: Superficial tumor[a]	G3, T2a, N0, M0
T2b: Deep tumor[a]	G3, T2b, N0, M0
Regional Lymph Nodes (N)	Any G, any T, N1, M0
NX: Regional lymph nodes cannot be assessed	**Stage IV**
N0: No regional lymph node metastasis	Any G, any T, any N, M1
N1: Regional lymph node metastasis	Ñ
Distant Metastasis (M)	Ñ
MX: Distant metastasis cannot be assessed	Ñ

M0: No distant metastasis	Ñ
M1: Distant metastasis	Ñ

[a] Superficial tumor is located exclusively above the superficial fascia without invasion of the fascia; deep tumor either is located exclusively beneath the superficial fascia or superficial to the fascia with invasion of or through the fascia or is located superficial and beneath the fascia. Retroperitoneal, mediastinal, and pelvic sarcomas are classified as deep tumors.

IV. TREATMENT

A. Surgical Resection. Smaller, grade I tumors can be excised with a minimum 1-cm margin and usually do not require adjuvant radiation. Larger tumors may benefit from a larger margin or from radiation to prevent recurrence. Grade II and III tumors, in general, require adjuvant radiation therapy to prevent recurrence. Local recurrence should be resected aggressively, and long-term followup is required. Amputation is rarely necessary.

Limb-sparing resection combined with radiation therapy offers rates of survival equivalent to those achieved with amputation (*Ann Surg.* 1982;196:305). Limb-sparing procedures have a psychological and functional advantage over amputations and are the procedures of choice for most tumors. Resection should include the area of previous incision. Intraoperative use of clips can aid in postoperative radiation therapy.

Retroperitoneal sarcomas are considerably more difficult to treat because the tumors often involve vital structures. Overall and disease free survival for retroperitoneal STS are 56.8% and 39.4%, respectively (*J Clin Oncol.* 2013;31(13):1649-1655). Organs associated with the tumor should be resected en bloc because 5-year recurrence rates are lower for en bloc resections than for cases of tumor resection alone (28% vs. 48%, respectively; *J Clin Oncol.* 2009;27:24). Postoperative irradiation may be used but is associated with

relatively high morbidity, often due to off-target effects. Preoperative irradiation therapy has several advantages including direct targeting of the tumor, reduced off-target effects, and conversion of tumors from unresectable to resectable.

Gastrointestinal stromal tumors (GISTs) are sarcomatous tumors of the GI tract, most commonly arising from the stomach. GISTs can present with acute or subacute GI bleeding, as vague abdominal pain, as an abdominal mass, or as an incidentaloma. These tumors are distinguished by expression of c-*kit* (CD117). Surgical resection with microscopically negative margins is standard treatment. Imatinib mesylate, a tyrosine kinase inhibitor, has been approved to treat patients with unresectable or metastatic GIST. Patients with GIST that is resistant to imatinib may respond to **sunitinib malate,** which has been approved as second-line treatment.

B. Radiation. For the majority of STS, surgery combined with radiation therapy is more effective than either therapy alone. Surgery alone may be appropriate for small (<5 cm), superficial lesions with favorable histologic subtype and grade. Otherwise, adjuvant radiotherapy is generally indicated to improve local control. Preoperative radiation can also be used for large, deep-seated tumors for which the surgeon anticipates a possible positive margin. Brachytherapy—that is, administration of radiation therapy via the implantation of radioactive beads—can also be used to improve localization to radiosensitive areas. Generally, radiotherapy is employed mostly

for extremity STS; radiotherapy for retroperitoneal STS is less effective and carries higher risk of off-target effects.

C. Chemotherapy. Several randomized, prospective trials have failed to show any improvement in survival with adjuvant chemotherapy for adult grade II or III sarcomas. The only drugs with response rates more than 20% are doxorubicin, epirubicin, and ifosfamide. Response rates are typically low (˜25%) although they are dependent on histologic subtype.

D. ILP and ILI. ILP and ILI with melphalan, as with melanoma, can be used to treat extremity STS, particularly in patients with either primary or recurrent unresectable disease. There is some suggestion of decreased local recurrence with definite downstaging of the tumor, but survival data are lacking (*Ann Surg Oncol.* 2007;14:230). ILP and ILI are limited to centers of expertise.

E. Other Therapies. Trabectedin (a small molecule inhibitor of DNA nucleotide excision repair) and **pazopanib** (an oral tyrosine kinase inhibitor) are new therapies with some efficacy in select histologic subtypes; both agents are currently being studied in clinical trials.

CHAPTER 36: SKIN AND SOFT-TISSUE TUMORS

Multiple Choice Questions

1. For which of the following patients is sentinel lymph node biopsy (SLNB) indicated?

a. A 55-year-old male with 0.6 mm thick melanoma of the back with no ulceration and 3 mitoses/mm^2

b. A 28-year-old female with melanoma in situ of the right back

c. A 66-year-old male with 1.2 mm thick acral melanoma of the left lower extremity with left inguinal adenopathy

d. A 54-year-old female with 3.2 mm thick right upper extremity melanoma with two pulmonary metastases

e. A 35-year-old male with 1.4 mm thick melanoma of the back with no ulceration or mitoses

View Answer

2. A 68-year-old male presents with a growing, smooth, hypopigmented 2-cm lesion on the right cheek that he says occasionally stings and bleeds. The best next step is:

a. Mohs microsurgery

b. Excisional biopsy

c. FNA

d. Punch biopsy

e. Shave biopsy

View Answer

3. A 38-year-old firefighter is referred to your clinic with a nonhealing wound that arose after sustaining a burn injury 4 months ago. His past medical history is significant for diabetes mellitus type II controlled on insulin; a recent HgbA1c is 6.8%. He smokes about 1 pack weekly. He is otherwise healthy. He denies any fever but complains of oozing from his lowerextremity wound. Examination of the left lower extremity reveals a 4-cm linear scar with a 1.5-cm central ulcer with serosanguineous drainage and mild local edema. Peripheral pulses are palpable. There is mild tenderness but no warmth or erythema. The best course of action is to:

a. Prescribe topical mupirocin

b. Increase insulin dose

c. Advise smoking cessation

d. Obtain ankle-brachial indices

e. Biopsy wound edge

View Answer

4. A 44-year-old male is referred to your clinic after punch biopsy of a lesion on his left shoulder revealed a 1.8-mm thick superficial spreading type melanoma. There is no clinically detectable lymphadenopathy. The most appropriate management is:

a. Wide local excision with 1-cm margins

b. Wide local excision with 2-cm margins

c. Wide local excision with 1-cm margins and sentinel lymph node biopsy

d. Wide local excision with 2-cm margins and sentinel lymph node biopsy

e. Wide local excision with 2-cm margins and left axillary lymph node dissection

View Answer

5. A 55-year-old-female presents to your clinic with a 4-mm brown, homogeneous, round macular lesion with slightly indistinct border located on the right posterior calf. She states it is new within the last year. The most appropriate management is:

a. Observation

b. Shave biopsy

c. Excision with 1-cm margins

d. Excisional biopsy

e. Topical 5-fluorouracil for 2 to 6 weeks

View Answer

6. A 46-year-old-male presents on referral from his primary physician to your clinic with a “lump” of the left posterior neck. He has noticed this since he was a child. He denies any associated symptoms or complaints. On examination, a 1-cm mobile, soft, round mass is palpable at the border of the left trapezius. Neurovascular examination is unremarkable. The most appropriate management is:

a. Observation

b. Core needle biopsy

c. Surgical excision

d. Incisional biopsy

e. CT of the head and neck

View Answer

7. Which of the following patients with melanoma is most appropriate for referral to radiation oncology?

a. A 63-year-old male with 1.1-mm thick melanoma of the back with ulceration and 3 mitoses/mm^2, with a negative sentinel lymph node

b. A 24-year-old female with melanoma in situ of the left calf, now status post Mohs microsurgical resection

c. A 66-year-old male with 1.9-mm thick acral melanoma of the left lower extremity with left inguinal adenopathy, status post left groin dissection with three positive lymph nodes

d. A 73-year-old female with 2.8-mm thick right thigh melanoma with two pulmonary metastases

e. A 35-year-old male with 1.4-mm thick melanoma of the back with no ulceration or mitoses, with a positive sentinel lymph node and negative completion lymph node dissection

View Answer

8. A 42-year-old-female presents with a 2 × 4 cm enlarging right upperextremity mass. She is right handed and has had problems dropping things recently. Physical examination reveals the mass without overlying skin changes. Grip strength is weak. The best management option is:

a. Refer for radiation therapy

b. Obtain MRI and chest CT

c. Perform incisional biopsy

d. Excise with wide margins and perform sentinel lymph node biopsy

e. Perform FNA

View Answer

9. A 63-year-old-female presents with a lump in her left axilla that she has noticed for the last 4 months. She denies any skin lesions, recent infections or fevers and physical examination is unremarkable except

for axillary adenopathy. Mammogram obtained 3 weeks ago is unremarkable. The most appropriate next step is:

a. Core needle biopsy

b. PET scan

c. Observe, followup in 1 month

d. 10-day course of Keflex

e. Radiation therapy

View Answer

10. Which of the following is the most important prognostic factor for softtissue sarcomas?

a. Histologic subtype

b. Age at presentation

c. Comorbidities

d. Necrosis

e. Grade

View Answer

11. A 73-year-old female was recently diagnosed with a left distal femoral malignant fibrous histiocytoma abutting the distal superficial femoral artery. The best next step is:

a. Wide local excision

b. Left above the knee amputation

c. Chest CT

d. Isolated limb perfusion

e. Radiation therapy

View Answer

12. A 44-year-old male is taken to the operating room for resection of a large retroperitoneal liposarcoma. He has no significant past medical history and preoperative laboratory studies were normal. After laparotomy, the tumor is discovered to encase the left renal artery and vein. The most appropriate course of action is:

a. Tumor debulking followed by adjuvant chemotherapy

b. Placement of clips, closure, and referral for radiation therapy

c. Closure and subsequent chemotherapy

d. Resection of the mass en bloc with the left kidney

e. Resection of the mass with a positive gross margin, percutaneous nephrostomy tube placement

View Answer

13. The worst prognostic factor in Merkel cell carcinoma is:

a. Age at diagnosis

b. Size of primary tumor

c. Elevated mitotic index

d. Presence of microscopic nodal disease

e. Lymphovascular invasion

View Answer

14. A 67-year-old male is diagnosed with a 2.2-cm basal cell carcinoma of the right shoulder. The next step is:

a. Chest CT

b. Resection with 0.4-cm margins

c. Mohs microsurgery

d. Resection with 1-cm margins, right axillary sentinel lymph node biopsy

e. Radiation therapy

View Answer

15. A 61-year-old male presents with a 6-cm squamous cell cancer of the left thigh arising from a chronic wound. He has no other medical comorbidities. Preoperative laboratory studies demonstrated LFT abnormalities and ultrasound reveals a single 4-cm hepatic lesion, which is confirmed by CT scan. Biopsy reveals squamous cell carcinoma without cirrhosis. A PET demonstrates avidity in the liver lesion and the left thigh and no other sites. Appropriate management consists of:

a. Resection of both lesions

b. Excision of the thigh lesion, radiation to the liver

c. Cryotherapy of the thigh lesion, radiation to the liver

d. Systemic chemotherapy

e. Topical imiquimod for the thigh lesion, resection of the liver lesion

View Answer

37

Fundamentals of Laparoscopic, Robotic and Endoscopic Surgery

Ahmed Zihni

Michael M. Awad

Minimally invasive surgical techniques have become a crucial component of surgical care. The goal of minimally invasive surgery is to perform safe surgical operations through much smaller incisions than traditional open surgery, or without incisions at all. A growing body of research demonstrates significant benefits associated with laparoscopic surgery compared to the open approach, including shorter length of hospital stay, less postoperative pain, and lower rates of postoperative wound occurrences. The use of robot-assisted laparoscopic surgery continues to expand in a variety of surgical fields. Surgeons continue to provide diagnostic endoscopy to a large segment of patients in the United States and have been instrumental in pioneering a variety of endoscopic surgical techniques. For these reasons, a familiarity with basic principles of laparoscopic, robotic, and endoscopic surgery is critical for surgical trainees.

A. Laparoscopy

1. Patient selection

Laparoscopy is used to perform a vast assortment of operations, and there are very few contraindications to its use. Absolute contraindications are limited to an inability to tolerate general anesthesia or laparotomy, hemodynamic instability, uncorrected coagulopathy, or unavailability of an experienced laparoscopic surgeon competent in the procedure being attempted. Relative contraindications include bowel obstruction, severe cardiopulmonary disease, peritonitis, and loss of domain. Preoperative assessment for laparoscopic procedures should proceed as it would for a comparable open procedure.

2. Basic principles

Laparoscopy is strictly defined as the use of an endoscope to explore the abdominal cavity. Laparoscopic surgery, as commonly defined among surgeons today, involves entry into the intraperitoneal cavity and establishment of pneumoperitoneum with CO_2 or, much less commonly, N_2O. Pneumoperitoneum creates working space for instruments that are inserted into the abdomen through plastic or metallic ports with valves that prevent loss of pneumoperitoneum. Visualization is achieved using a lighted rigid endoscope connected to display monitors in the operating room. Laparoscopic surgery is performed based on the principle of triangulation. The

camera port is placed centrally, with working ports to either side, allowing the long rigid instruments to converge at the surgical target

without colliding with the laparoscope or with one another. Laparoscopic surgery has several limitations. The surgical field is a three-dimensional space, but most laparoscopes project an image onto a traditional twodimensional monitor, creating limited depth perception for the operating surgeon. Most laparoscopic instruments can rotate around their long axis and some may have some degree of articulation at the instrument tip, but they have significantly limited dexterity compared to the human hand and wrist. In addition, laparoscopic instruments can transmit some haptic input through the instrument shaft and handle to the hand of the operator, but much of the haptic feedback that is essential to handling tissue in open surgery is lost during laparoscopic surgery.

3. Equipment and troubleshooting

a. Room setup and positioning. All necessary equipment should be in the operating room prior to patient arrival, including an insufflator and gas tank, light source, camera, laparoscope, monitors, laparoscopic ports and trocars, laparoscopic instrumentation, and instruments for open conversion if necessary. If intraoperative fluoroscopy or radiography is likely to be employed, the patient and equipment should be positioned to allow additional hardware to access the operating table. The patient, surgeon, and monitor should be positioned to place the operative field between the surgeon and the monitor. Patient position is dictated by the surgical procedure being undertaken. Most abdominal operations require the patient be placed in the supine position. This position is sometimes modified to split the legs and allow the surgeon to operate from between the legs to access the upper abdomen. Footboards, suction beanbags, and safety straps are often employed to secure the patient to the operating table, allowing for steep angles to be employed intraoperatively. Pelvic procedures in which a perineal approach may be required typically employ the lithotomy position. Lateral decubitus positioning is used to perform thoracoscopic surgery or laparoscopic retroperitoneal procedures. Monitors should be positioned to allow the operating surgeon to view them clearly without turning their head and with a 15-degree downward gaze, minimizing neck extension and postural fatigue. The operating table height and port placement should allow the operating surgeon to keep both arms at their sides with their elbows flexed to 90 to 120 degrees. A preoperative safety and equipment check is essential prior to any laparoscopic operation (Fig. 37-1).

b. Insufflation. A pressure-limited insufflator is typically used in laparoscopic surgery. This device controls the flow of CO_2 into the abdominal cavity. Most insufflators display the pressure in the system, which reflects the pressure in the target body cavity when the two are in continuity, as well as the flow rate of gas through the insufflator and the total volume of gas insufflated. The insufflator allows a target pressure to be selected and the rate of gas flow to be modulated. The minimum pressure that allows adequate working space should be employed. Loss of pneumoperitoneum or an increase in pressure can occur due to factors at the level of the gas

source, insufflator, tubing, ports, or change in patient sedation level (Fig. 37-1).

Problem	Cause	Solution
1. **Poor insufflation/ loss of pneumoperitoneum**	CO_2 tank empty	Change tank
	Accessory port stopcock(s) not properly adjusted	Inspect all accessory ports. Open or close stopcock(s) as needed
	Leak in sealing cap or stopcock	Change cap or cannula
	Excessive suctioning	Allow time to reinsufflate
	Loose connection of insufflator tubing at source or at port	Tighten connections
	Hasson stay sutures loose	Replace or secure sutures
	Tubing disconnection from insufflator	Connect tubing
	Flow rate set too low	Adjust flow rate
2. **Excessive pressure required for insufflation (initial or subsequent)**	Veress needle or cannula tip not in free peritoneal cavity	Reinsert needle or cannula
	Occlusion of tubing (kinking, table joint, etc.)	Inspect full length of tubing. Replace with proper size as necessary
	Port stopcock turned off	Fully open stopcock
	Patient is "light"	Give more muscle relaxant
	Cannula tip not in peritoneal space	Advance cannula under visual control
3. **Inadequate lighting (partial/ complete loss)**	Loose connection at source or scope	Adjust connector
	Light is on "manual-minimum"	Go to "automatic"
	Bulb is burned out	Replace bulb
	Fiber optics are damaged	Replace light cable
	Automatic iris adjusting to bright reflection from instrument	Re-position instruments, or switch to "manual"
	Monitor brightness turned down	Readjust setting
	Room brightness floods monitors	Dim room lights
4. **Lighting too bright**	Light is on "manual-maximum"	Go to "automatic"
	"Boost" on light source is activated	Deactivate "boost"
	Monitor brightness turned up	Readjust setting

Figure 37-1 Troubleshooting Algorithm for Laparoscopic Surgery. (Airan M (2012) Equipment Setup and Troubleshooting. In: Soper NJ, Scott-Connor CEH, eds. *The SAGES manual: Volume 1. Basic laparoscopy and endoscopy.* New York, NY: Springer, 21-43.) (*continued*)

Problem	Cause	Solution
5. No picture on monitor(s)	Camera control or other components (V.C.R., printer, light source, monitor) not "on" Cable connector between camera control unit and/or monitors not attached properly Cable between monitors not connected Input select button on monitor doesn't match "video in" choice	Make sure all power sources are plugged in and turned on Cable should run from "video out" on camera control unit to "video in" on primary monitor. Use compatible cables for camera unit and light source. Cable should run from "video out" on primary monitor to "video in" on secondary monitor Assure matching selections
6. Poor quality picture **a. fogging/haze** **b. flickering, electrical interference** **c. blurring, distortion**	Condensation on lens from cold scope entering warm abdomen Condensation on scope eyepiece, camera lens, coupler lens Moisture in camera cable connecting plug Poor cable shielding Insecure connection of video cable between monitors Incorrect focus Cracked lens, internal moisture Too grainy	Gently wipe lens on viscera; use anti-fog solution, or warm water Detach camera from scope (or camera from coupler), inspect and clean lens as needed Use suction or compressed air to dry out moisture (don't use cotton tip applicators on multi-pronged plug) Replace cables as necessary Move electrosurgical unit to different circuit or away from video equipment Reattach video cable at each monitor Adjust camera focus ring Inspect scope/camera, replace if needed Adjust enhancement and/or grain settings for units with this option
7. Inadequate suction/irrigation	Occlusion of tubing (kinking, blood clot, etc.) Occlusion of valves in suction/ irrigator device Not attached to wall suction Irrigation fluid container not pressurized	Inspect full length of tubing. If necessary, detach from instrument and flush tubing with sterile saline Detach tubing, flush device with sterile saline Inspect and secure suction and wall source connector Inspect compressed gas source, connector, pressure dial setting
8. Absent or "weak" cauterization	Patient not grounded properly Connection between electro-surgical unit and instrument loose Foot pedal or hand switch not connected to electrosurgical unit Wrong output selected Connected to the wrong socket on the electrosurgical unit Instrument insulation failure outside of surgeon's view	Assure adequate grounding pad contact Inspect both connecting points Make connection Correct output choice Check that cable is attached to endoscopic socket Use new instrument and inspect insulation

Figure 37-1 (*Continued*)

c. Imaging system. The typical laparoscopic imaging system consists of four basic components: A laparoscope, a light source, a camera and camera controller, and a monitor. Imaging difficulties can be caused by any component in the system (Fig. 37-1). The laparoscope is a rigid telescope that is inserted into the patient through a port site. It contains fiber optic elements that are connected to a high-energy light source by a light cable and provide illumination to the surgical field. Laparoscopes vary in diameter, with larger diameter scopes being able to provide greater illumination. Most abdominal surgery is performed using a 10-mm scope. Increasingly, 5-mm laparoscopes are coming into use for a variety of abdominal procedures. Laparoscopes can have flat tips (0-degree scopes) or angled tips (commonly 30 or 45 degrees). Angled scopes allow the field of view to be turned around the long axis of the scope. The laparoscope also contains optical components that transmit light into the camera, which captures and digitizes the image. The image resolution of laparoscopic cameras continues to increase, but must be coupled with a correspondingly high-resolution monitor.

d. Energy sources. A vast array of surgical instruments are available for use in laparoscopic operations. A full discussion of laparoscopic instrumentation is beyond the scope of this review; however a discussion of energy-based devices used in laparoscopic surgery is critical because of the particular importance of meticulous hemostasis in minimally invasive surgery, and the unique complications associated with laparoscopic energy devices.

(1) Monopolar energy devices use a current of electrons that originates at an electrode at the surgical site and travel to a dispersive electrode elsewhere on the patient's body, usually a pad on their leg or back. The density of current at the site of the surgical electrode generates heat, leading to tissue disruption. There are two commonly used modalities of monopolar energy. ÒCuttingÓ mode is a continuous high-frequency, low-voltage current that rapidly produces very high temperatures and tissue vaporization at the target. This allows the instrument to cut through tissue easily without achieving hemostasis, and is therefore rarely used in laparoscopic surgery where hemostasis is crucial for adequate visualization of tissue. ÒCoagulatingÓ mode is a pulsed low-frequency high-wavelength current that causes a slower, lower temperature heating of tissue that causes coagulative necrosis of tissue and hemostasis at the surgical site. There are unique risks associated with laparoscopic monopolar electrosurgery. Many monopolar devices do not have shielded instrument tips or require a connection to be made between two electrodes for the device to be activated, allowing for injuries to occur if the device is inadvertently activated while the instrument tip is out of the laparoscopic field of view. Also, current can pass through

breaks in the insulation, leading to injuries if points of insulation failure come into contact with tissue. Direct coupling is a phenomenon that occurs when current passes from a monopolar instrument through another conductive instrument or material. This is often used purposefully during surgery, but can also occur inadvertently when current is transmitted through metallic ports or other instruments to tissue. Capacitive coupling is a similar phenomenon that occurs when an active electrode passes through an uninsulated conductor, such as a plastic and metallic trocar, causing current to be stored and potentially discharged into tissue.

(2) Bipolar energy devices use a current that originates in a grounded energy source and travels between two electrodes that are contained within the working tip of the instrument. Because target tissue is contained between the two electrodes, there is very little thermal spread beyond this target. There is also a much lower risk of capacitive coupling associated with bipolar instruments. Bipolar devices are commonly used as vessel sealing devices, or to divide highly vascular tissue, because their ability to deliver a current between two instrument tines that are held together with pressure can effectively fuse the walls of vessels up to 7 mm in diameter.

(3) Argon beam coagulation is a form of monopolar electrosurgery in which a stream of ionized argon gas is emitted at the surgical site and completes the circuit between an active electrode and the distant dispersive electrode. A high current density flows through the argon gas, causing thermal damage to tissue contacted by the gas. This technology is very useful for achieving hemostasis across a large raw bleeding surface where bleeding would otherwise be difficult to control. Employing argon beam coagulation laparoscopically, however, can increase intra-abdominal pressure due to the additional gas being introduced to the abdomen.

(4) Ultrasonic energy devices employ an active blade that vibrates at very high frequency, generating thermal energy that can divide tissue that is compressed against the active blade by an inactive blade. They can be used to divide or dissect tissues, including vascularized tissue, and

can seal vessels up to 5 mm in diameter. These systems do not have any risk of direct or capacitive coupling. In addition, ultrasonic devices coagulate tissue at lower temperatures and generate much less thermal spread than monopolar devices. However, since the active blade of an ultrasonic device is heated through use, this thermal energy takes a few seconds to dissipate after the active blade is employed, and inadvertent injuries may occur if the active blade is brought into contact with tissue immediately after activation.

e. Abdominal access. Safe access to the abdomen is the crucial first step to any laparoscopic procedure. Two strategies to achieve access

to the abdomen are generally used: The ÒclosedÓ technique using a Veress needle, and the ÒopenÓ technique using a Hasson cannula.

(1) Closed Veress needle technique. The Veress needle is a 7 to 12 cm long 14-gauge needle attached to a valve and stopcock that allow gas to be insufflated through the needle. The needle has a sharp beveled tip with a spring-loaded retractable blunt tip that projects beyond the sharp tip and retracts when pressed against a surface. To achieve access to the abdomen using a Veress needle, the patient is first positioned appropriately and their abdomen prepped for surgery, and the site of Veress insertion selected. In a patient without prior abdominal surgery, a periumbilical insertion site is used. In patients with prior midline abdominal surgery, alternative sites on the abdomen remote from the prior incision may be used. The left upper quadrant and right lower quadrant are frequently used alternate sites of needle insertion and initial port placement. A stab incision is made at the site of Veress insertion. It is recommended to lift the abdominal wall away from the underlying viscera prior to needle insertion. The needle is then inserted into the abdomen, generally at an angle perpendicular to the abdominal wall, while held at the shaft rather than its hub. The needle encounters resistance followed by a sensation of give at two separate points, first as it crosses the midline fascia, then as it traverses the peritoneum and the blunt tip springs out of the needle. When performing Veress entry away from the midline, three ÒpopsÓ are typically encountered due to the presence of anterior and posterior fascial layers along with the peritoneum. Once the Veress needle has entered the abdomen, its position may be confirmed. First, a syringe containing saline is attached to the hub and aspiratedÑno blood or succus should be aspirated. Saline should easily instill into the abdomen without any return of saline on subsequent aspiration. After instilling 3 to 5 cc of saline, the syringe should be removed with the stopcock open and the saline remaining in the Veress needle should flow easily into the abdomen. This constitutes the ÒdropÓ test. The insufflation tubing is then connected to the hub and the gas flow initiated at 1 to 2 L/minute. Initial pressure should be no more than 10 mm Hg. Elevated initial pressure is caused by malposition of the Veress needle tip, either due placement of the needle in the preperitoneal space or due to the needle opening abutting a structure or sitting in a pocket with limited continuity with the rest of the abdomen. If this occurs, the needle may be gently rotated and slightly advanced or withdrawn. If the pressure does not quickly drop to the expected level, the needle should be withdrawn and another attempt made.

This may be safely repeated until an intra-abdominal position of the needle and normal insufflation are confirmed.

Insufflation of the abdomen proceeds at 1 to 2 L/minute for 1 minute; then the flow rate is increased to the maximal rate

the insufflator can support until the intra-abdominal pressure reaches the set target of 12 to 15 mm Hg. Once the abdomen is adequately insufflated, the Veress needle is removed, the skin is incised at the camera port site, and the first trocar is inserted. This can be done blindly, or under direct vision using an optical trocar. These trocars have a clear tip and allow a 0-degree scope to be inserted into the trocar. As it is pushed into the abdomen, the layers of the abdominal wall and intraperitoneal space can be visualized. Once the port is placed into the abdomen, the trocar is removed and the laparoscope inserted to the abdomen to confirm placement and perform an initial exploration. Particular attention is paid to the area directly below the initial port, where injury may have been caused by the Veress needle or the trocar. Afterward, subsequent ports may be placed with direct visualization using the camera. Ports should be placed 8 to 10 cm apart and adhere to the principle of triangulation described above.

(2) Open Hasson cannula technique. This technique involves entry into the abdomen at the initial trocar site under direct vision. This technique is particularly useful when safety concerns are present due to prior surgery and the possibility of viscera adherent to the abdominal wall. Some surgeons preferentially use an open access technique for all laparoscopic procedures. Open entry is usually performed at a periumbilical site which is used as the camera port during surgery. However, in patients with extensive prior midline abdominal surgery, an alternative site may be used, such as the left upper or right lower quadrants. After the skin is prepped, a 1 to 3 cm incision is made at the site of entry and carried down to the subcutaneous tissue using sharp dissection and electrocautery. Sweeping away subcutaneous fat using retractors, the fascia is exposed, pulled away from underlying viscera and incised. The peritoneum is then similarly elevated and opened. The abdominal cavity is then visualized and gently explored with a finger, and fascial sutures placed. Then the Hasson cannula is inserted into the intraperitoneal space. The Hasson cannula has a blunt obturator to prevent insertion trauma, a cone-shaped sleeve that sits in the fascial incision, and two struts that are fixed to the fascial stitches to hold the system in place. Some systems employ a balloon that sits over the intraperitoneal portion of the cannula and is inflated upon insertion, holding the system in place without fascial sutures when the sleeve is brought down into position. Once the cannula is positioned, the camera is inserted for an initial exploration of the abdomen and placement of subsequent ports as in the Veress technique.

f. Complications of abdominal access. A variety of complications can occur during abdominal access, particularly during blind portions such as Veress needle or blind trocar insertion. Visceral or vascular injury may occur during Veress needle insertion. If bowel contents or blood are aspirated during Veress insertion, the needle should be

withdrawn and reinserted and laparoscopic access should proceed. Upon entry to the abdomen, bowel or other injured organs should be examined and managed per established protocols for traumatic injury to intraperitoneal organs. In most cases, a Veress needle stick to intraperitoneal viscera does not require any intervention, but inadvertent intraluminal insufflation with the Veress needle can cause significant injury requiring bowel resection. The retroperitoneum should also be closely examined on abdominal exploration, particularly in situations where blood was encountered on initial Veress insertion. A stable hematoma in the mesentery or lateral retroperitoneum can usually be observed. However, central or rapidly expanding hematomas require laparotomy and exploration of the retroperitoneum.

Trocar insertion can cause injuries far more severe than Veress insertion. Bowel and visceral injuries should be considered penetrating traumas and managed accordingly, and any central or rapidly expanding retroperitoneal hematoma should be explored. If a rush of blood is encountered on trocar insertion, or open cannula placement, the surgeon must assume that a critical vascular injury has occurred and conversion to laparotomy must be performed immediately, without removing the trocar which may partially tamponade the injury.

Abdominal wall hemorrhage is a common complication of laparoscopic surgery that may be caused by injury to the epigastric arteries, veins, or their tributaries. These hemorrhages are often selflimited and cease with the tamponading effect of the port. If this fails to control the bleeding, control may be attempted using electrocautery or with a suture passing device.

g. Port removal and closure. At the conclusion of surgery, instruments and ports are removed under direct visualization, with the camera port being removed last, after the abdomen is desufflated. Fascial incisions 5 mm or smaller do not need to be closed. Ports of 10 mm or larger may be sutured to prevent hernia formation and incarceration of bowel if a bladed trocar was used or the site was dilated. Skin incisions are closed with subcuticular absorbable sutures, staples, or simply with skin tape or glue in the case of incisions 5 mm or smaller.

h. Physiologic effects of laparoscopy. Laparoscopic surgery requires the creation of pneumoperitoneum and a resultant increase in intra-abdominal pressure, which is associated with a variety of physiological effects

(1) Cardiovascular. Elevated intra-abdominal pressure leads to an increase in systemic vascular resistance due to compression of the aorta and visceral arteries, as well as the resultant activation of the renin-angiotensin-aldosterone axis. Inferior vena cava compression leads to reduced preload and lower cardiac output. Cephalad displacement of the diaphragm causes increased intrathoracic pressure, which leads to increased pulmonary vascular resistance.

(2) Respiratory. Increased intra-abdominal pressure limits diaphragmatic excursion, decreasing pulmonary compliance, and functional residual capacity, and increasing thoracic pressure. These changes are associated with the development of atelectasis. In addition, absorption of insufflated CO_2 can contribute to hypercarbia and acidosis.

(3) Renal. Increased intra-abdominal pressure leads to a reduced glomerular filtration rate due to reduced afferent arterial flow and elevated renal venous pressure.

(4) Neurologic. The increased intrathoracic and central venous pressure associated with pneumoperitoneum leads to a decrease in venous return from the brain. This leads to an increase in intracranial pressure and may contribute to temporary confusion on emergence from long laparoscopic operations.

4. Fundamentals of laparoscopic surgery. The Fundamentals of Laparoscopic Surgery (FLS) curriculum and examination are a validated program developed by the Society of American Gastrointestinal and Endoscopic Surgeons (SAGES) for the teaching and evaluation of didactic knowledge and technical skills associated with laparoscopic surgery. The program is composed of a web-based didactic curriculum, a written examination, and a skill-based examination.

The didactic portion of the FLS curriculum is a web-based series of modules that learners are able to navigate and study at their own pace. These modules cover basic preoperative considerations, intraoperative considerations, basic laparoscopic procedures, postoperative care and complications, and an explanation of the technical skills portion of the examination. After studying the didactic modules, the learner takes a 90-minute 75-question examination in a proctored setting, based on the content of the didactic modules.

The manual skills examination is a test of five tasks that are performed in a laparoscopic trainer box. These tasks are: Peg transfer, Pattern cutting, Endoloop, Extracorporeal stitch, and Intracorporeal stitch. Each task is scored for efficiency, and has a time limit to complete the task. In addition, each task is scored for performance, with specifically defined task errors leading to lower scores. Passing the FLS written and technical examinations is now a prerequisite for eligibility for board certification in General Surgery in the United States.

B. Robot-assisted Surgery. Robot-assisted laparoscopic surgery is used in a variety of surgical fields, particularly general surgery, urologic surgery, and gynecologic surgery, and continues to expand. Robotic surgery holds great promise in the advancement of minimally invasive surgery, but also has significant current limitations.

1. Equipment and basic principles. Strictly speaking, current ÒroboticÓ surgical systems are not robots, as the term implies some degree of automation and current robotic surgical platforms do not perform any autonomous functions. More accurately, the most common robotic surgical platform (daVinci Surgical System, Intuitive Surgical Inc.,

Sunnyvale, CA) can be described as a computer-assisted surgical system. This platform employs a Òmaster-slaveÓ configuration, in which the surgeon is seated at a console (surgeon cart) and manipulates hand-held controllers (masters), whose motions are mapped directly to the surgical instrument tip. The instruments are mounted to mechanical arms on a platform that is ÒdockedÓ to the patient at the laparoscopic port sites (patient cart). The laparoscope is also attached to an

arm of the patient cart and is controlled by the surgeon from the surgeon cart. Commonly used robotic surgical platforms employ a scope very similar to a traditional 10-mm high-definition laparoscope, but which captures two separate images that are projected to the binocular eyepieces the surgeon looks into at the surgeon cart, creating a three-dimensional display. In most cases, a nonrobotic assistant port is also placed, allowing an assistant surgeon to participate using traditional laparoscopic instruments and to assist with robotic instrument exchanges. Abdominal access for robot-assisted laparoscopic surgery is achieved in much the same way as in traditional laparoscopy. Triangulation of the surgical target remains the guiding principle for port placement, though robotic ports are generally arranged along a straight line, rather than the Òbaseball diamondÓ configuration that is commonly used in laparoscopic surgery, to facilitate the action of the robotic arms.

2. Advantages. Robot-assisted laparoscopic surgery has a variety of benefits for operating surgeons compared to laparoscopy. Robotic instruments are wristed, allowing for more degrees of freedom compared to laparoscopic instruments. In addition, the master-slave configuration allows the movements of the surgeon to be scaled down when they are converted to instrument actions, allowing for finer movements than laparoscopic instruments can achieve. Robot-assisted surgery has also been shown to generate less ergonomic stress on operating surgeons than laparoscopic surgery, both objectively and subjectively (*Surg Endosc.* 2014;28**:**3379-3384).

3. Limitations. Robot-assisted laparoscopic surgery has several limitations. The robotic platform requires docking of the patient cart to the laparoscopic ports, limiting surgery to one or two body quadrants. The newest iteration of the daVinci Surgical System (Xi) attempts to address this limitation by streamlining undocking and redocking with a different surgical target, but traditional laparoscopy still allows for greater freedom to explore the abdomen. In addition, the current surgeon cart provides minimal haptic feedback to the operating surgeon. This compels surgeons to increasingly rely on visual cues to safely manipulate tissue. Finally, robotic surgery carries additional expense compared to laparoscopic surgery, and this added expense has not always been shown to contribute to superior patient outcomes.

4. Fundamentals of Robotic Surgery. Fundamentals of Robotic Surgery (FRS) is an educational curriculum for training and assessing surgical learners in the use of robot-assisted surgery. This program has been developed by a group of experts in robotic surgery and surgical education, and is modeled on the FLS program, though its technical

component has yet to be implemented. The curriculum contains modules focusing on introducing learners to robotic surgical systems, learning about important psychomotor skills for robot-assisted surgery, and understanding the team-based approach and communication skills necessary to effectively perform robotic surgery.

D. Flexible Endoscopy and Endoscopic Surgery

1. Equipment and troubleshooting. Flexible endoscopy is performed using a flexible fiberoptic

endoscope, connected to a source of water or saline, a suction source, and an air or gas insufflator. Most modern endoscopes are operated with a CCD chip camera that captures an image from the endoscope tip, digitizes it, and projects it to a monitor. Commonly used endoscopes also have working channels that allow instrumentation to be passed through the scope to the target, where biopsies can be taken or interventions performed. The most commonly used endoscopes among surgeons are gastroscopes and colonoscopes. Gastroscopes are used for diagnostic esophagogastroduodenoscopy and have a typical working length of 92.5 to 110 cm and a thickness of 9.0 to 9.2 mm, and usually have a single working channel. Colonoscopes are longer (133 to 170 cm) and thicker (11.1 to 13.7 mm diameter) and also have a working channel. Diagnostic endoscopy is performed by inserting an endoscope into the gastrointestinal tract and meticulously examining the target mucosa while advancing or withdrawing the scope. Insufflation is used to dilate the gastrointestinal lumen to facilitate scope advancement or mucosal visualization. Irrigation is used to clean the tip of the endoscope or wash debris from segments of mucosa. All of these elements are subject to malfunction, and a systematic approach to troubleshooting is critical (Fig. 37-2).

2. Endoscopic surgery. A variety of graspers, electrosurgical hemostatic devices, biopsy snares, ablative technologies, stents, clips, and other devices are available for use in interventional endoscopy. A full description of interventional endoscopy is beyond the scope of this chapter, but this expansion of technology has made possible a variety of endoluminal therapies for disorders that would have required open or laparoscopic surgical interventions. These include palliative stenting for gastrointestinal cancers, endoluminal therapies for reflux, complications of bariatric surgery, and achalasia.

3. Fundamentals of endoscopic surgery. Fundamentals of Endoscopic Surgery (FES) is a didactic curriculum and technical exam developed by SAGES to train surgical learners in the theory and technical skills necessary to perform diagnostic and interventional endoscopy. The didactic portion is a web-based modular curriculum similar to the model established with FLS. Modules focus on technology, patient preparation, patient sedation, upper and lower diagnostic endoscopy, biopsy techniques, enteral access procedures, hemostatic techniques, and ERCP. A proctored written exam tests knowledge of these topics,

and is accompanied by a technical exam that assesses fundamental endoscopic skills in a simulator. Passage of the FES written and technical exam is a prerequisite for board eligibility in General Surgery in the United States beginning with graduates in 2018.

Problem	Check the Following
No light at distal end	1. Light source plugged in and turned on 2. Light source ignited 3. Not in "standby" mode 4. Lens at distal tip is dirty 5. Bulb burned out
Out of focus	1. Adjust focus ring 2. Fiberoptic scope—clean lens
No irrigation	1. Water bottle contains water 2. Water bottle connected to umbilical cord 3. Connection tight 4. Lid of water bottle screwed on tightly 5. Power turned on 6. Valve stuck or occluded
No insufflation	1. Umbilical cord firmly seated into light source and screwed in if necessary 2. Power turned on 3. Valve stuck or occluded
Clogged valve or nozzle	1. Take valve apart and clean 2. Flush channel of endoscope with cleaning solution, followed by clean water
Difficulty passing instrument	1. Check tip angulation; decrease angulation and try again 2. Ensure that the instrument is fully closed 3. Check size of instrument relative to instrument channel; try smaller diameter instrument

Figure 37-2 Troubleshooting for Endoscopy. (From Vitale GC, Davis BR (2012) Flexible Endoscopes: Characteristics, Troubleshooting, and Equipment Care. In: Soper NJ, Scott-Connor CEH, eds. *The SAGES manual: Volume 1. Basic laparoscopy and endoscopy.* New York, NY: Springer, 497-507.)

CHAPTER 37: FUNDAMENTALS OF LAPAROSCOPIC, ROBOTIC AND ENDOSCOPIC SURGERY

Multiple Choice Questions

1. Which of the following is an absolute contraindication to laparoscopic surgery?

a. Small bowel obstruction

b. Morbid obesity

c. Peritonitis caused by perforation

d. Uncontrolled coagulopathy

e. Hypothermia

View Answer

2. Intra-abdominal pressure during laparoscopic surgery should be closest to:

a. 2 mm **Hg**

b. 6 mm **Hg**

c. 12 mm **Hg**

d. 18 mm **Hg**

e. 25 mm **Hg**

View Answer

3. After insufflating the abdomen using a Veress needle, a trocar is inserted just inferior to the umbilicus with an immediate rush of bright red blood. The next step of management should be:

a. Keep the trocar in place and perform a laparotomy

b. Resume insufflation and insert the laparoscope to identify the injury

c. Apply manual pressure to the abdomen

d. Remove the trocar and perform a laparotomy

e. Abort the operation and proceed to interventional radiology to attempt angiographic localization and control of the injury

View Answer

4. Which of the following is a benefit of robot-assisted laparoscopic surgery compared to traditional laparoscopy?

a. Reduced operative time

b. Ergonomic advantages to the operating surgeon

c. Reduced blood loss

d. Improved haptic feedback compared to laparoscopic instruments

e. Expanded field of view compared to laparoscopic imaging systems

View Answer

5. Which of the following is a physiologic effect of pneumoperitoneum?

a. Increased preload

b. Metabolic alkalosis

c. Increased pulmonary compliance

d. Decreased intracranial pressure

e. Decreased glomerular filtration rate

View Answer

38
Hernias

Thomas J. Wade

L. Michael Brunt

I. INGUINAL HERNIA

A. Incidence. The true incidence and prevalence of inguinal hernia worldwide is unknown. Inguinal hernia formation is a by-product of genetic, environmental, and metabolic factors, combined with individual patient factors that can vary over time such as activity level, immune status, infection(s), medications, personal habits (e.g., smoking), and changes in body mass index (*Surg Clin North Am.* 2008;88:179-201). Laparoscopic studies have reported rates of contralateral defects as high as 22%, with 28% of these going on to become symptomatic during short-term followup. The male-to-female ratio is greater than 10:1. The lifetime prevalence is estimated to be 25% in men and 2% in women. Two-thirds of incident inguinal hernias are indirect whereas nearly two-thirds of recurrent hernias are direct. Approximately 10% of inguinal hernias will become incarcerated, and a portion of these may become strangulated. Recurrence rates after surgical repair are less than 1% in children and vary in adults related to the method of hernia repair.

B. Terminology and Anatomy

1. The inguinal canal (Fig. 38-1) is a tunnel that traverses the layers of the abdominal wall musculature, bounded on the lateral deep aspect by an opening in the transversalis fascia/transversus abdominis muscle (internal inguinal ring), and travels along the fused edges of the transversus abdominis/internal oblique/inguinal ligament and iliopubic tract posteriorly and layers of the external oblique musculature anteriorly, ending on the medial superficial aspect at an opening in the external oblique aponeurosis (external inguinal ring). The inguinal canal houses the spermatic cord (males) or the round ligament (females) and is subject to hernia formation due primarily to decreased mechanical integrity of the internal ring and/or transversalis fascia, allowing intra-abdominal contents to encroach into this space and form the characteristic bulge of a groin hernia.

2. Direct hernias occur as a result of weakness in the posterior wall of the inguinal canal, which is usually a result of attenuation of the transversalis fascia. The hernia sac protrudes through Hesselbach triangle, which is the space bounded by the inferior epigastric artery, the lateral edge of the rectus sheath, and the inguinal ligament.

3. Indirect hernias pass through the internal inguinal ring lateral to the inferior epigastric

vessels and Hesselbach triangle and follow the spermatic cord in males and the round ligament in females. During

dissection, an indirect hernia sac is typically found on the anteromedial aspect of the spermatic cord. Indirect hernias may become incarcerated at either the internal or external ring.

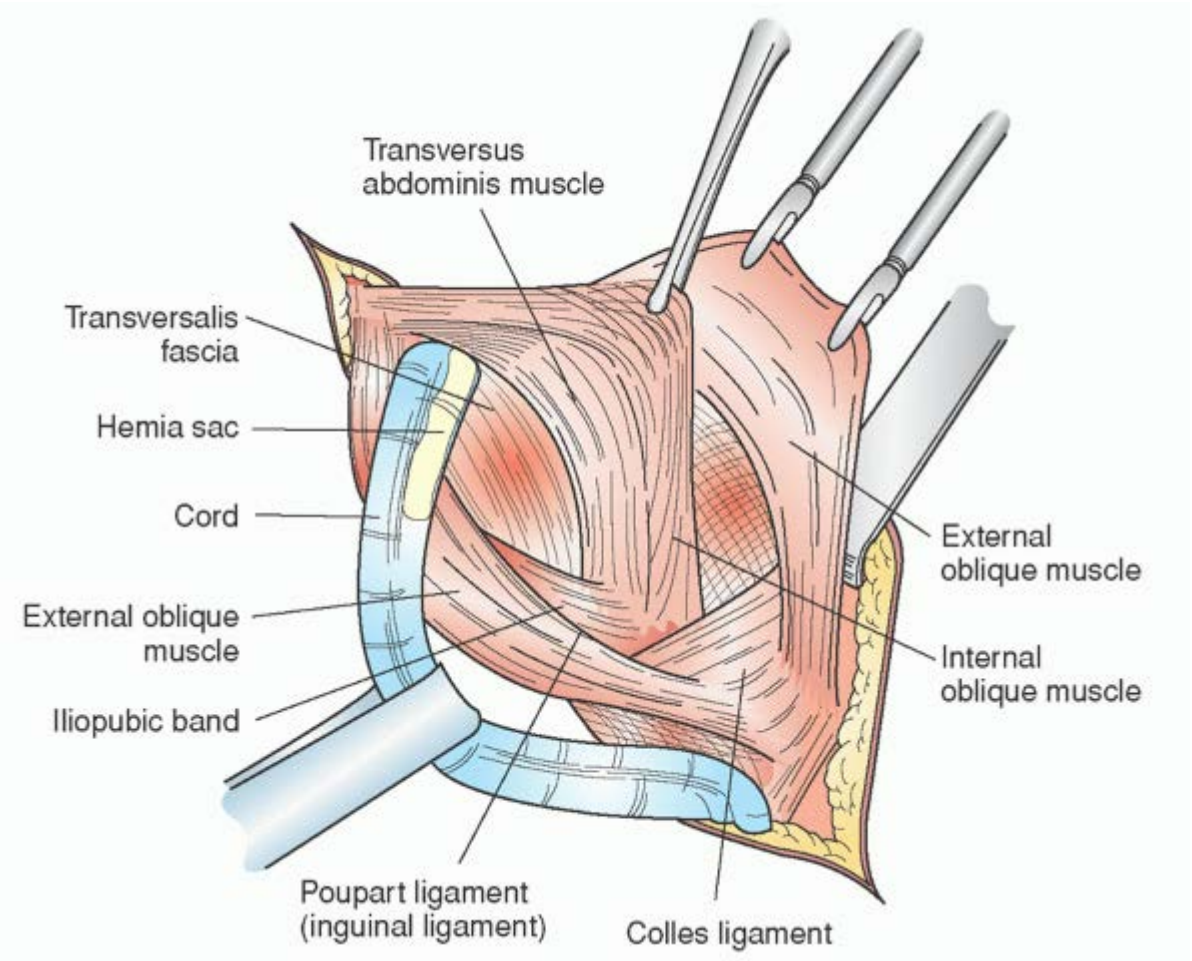

Figure 38-1 Anatomy of the right inguinal region.

4. In combined (pantaloon) hernias, direct and indirect hernias coexist.

5. Variants of inguinal hernias

a. Sliding hernia: (Usually indirect inguinal) denotes that a part of the wall of the hernia sac is formed by an intra-abdominal viscus (usually colon, sometimes bladder).

b. Richter hernia: A portion of (rather than the entire circumference) of the bowel wall is incarcerated.

c. LittrŽ hernia: Contains a Meckel diverticulum.

d. Amyand hernia: An inguinal hernia that contains the appendix.

6. Incarcerated inguinal hernias cannot be reduced into the abdominal cavity and may or may not be symptomatic. Strangulated hernias are incarcerated with vascular compromise of the herniated contents. Frequently, intense pain is caused by ischemia of the incarcerated segment.

C. Diagnosis

1. Clinical presentation

a. Most inguinal hernias present as an intermittent bulge that appears in the groin. In males, it may extend into the scrotal sac. Symptoms

are 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 and/or the presence of a bulge. In infants and children, a groin bulge is often noticed by caregivers during episodes of crying or defecation. Only in rare cases do patients present with bowel obstruction without the presence of a groin abnormality. All patients presenting with small bowel obstruction must be questioned carefully and examined for all types of hernia (i.e., inguinal, umbilical, incisional, obturator, etc.) as a possible etiology of obstruction.

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 as bulges with smooth, rounded surfaces that become more evident with straining. It may be necessary to invaginate the hemiscrotum to introduce an index finger through the external inguinal ring if the hernia is not apparent, but this maneuver is often uncomfortable for the patient and is unnecessary if an obvious bulge is present. It is difficult to determine whether the hernia is direct or indirect based solely on physical examination, although most hernias that extend into the scrotum are indirect. Incarcerated inguinal hernias present with pain, abdominal distention, nausea, and vomiting due to intestinal obstruction.

2. Radiographic evaluation. X-ray studies are rarely indicated. Ultrasonography or computed tomographic (CT) scan may occasionally be used to diagnose an occult groin hernia, particularly in the obese patient. Plain abdominal radiographs may verify intestinal obstruction in cases of incarceration.

D. Differential Diagnosis. Inguinal hernias should be distinguished from femoral hernias, which protrude below the inguinal ligament. Inguinal adenopathy, lipomas, hydrocele, epididymitis, testicular torsion, groin abscess, and vascular aneurysms/pseudoaneurysms all should be considered in the differential diagnosis.

E. Treatment

1. Preoperative evaluation and preparation. Most patients with inguinal hernias should be treated surgically although watchful waiting may be appropriate for individuals with asymptomatic hernias or for elderly patients with minimally symptomatic hernias (*J Am Coll Surg.* 2006;203:458-

468). Recently, Fitzgibbons et al. found that 68% of male patients who undergo watchful waiting will undergo repair within 10 years of presentation (*Ann Surg.* 2013; 258(3):508-515). Therefore, surgical repair should be considered in patients with asymptomatic or minimally symptomatic inguinal hernia. Associated conditions that lead to increased intra-abdominal pressure such as chronic cough, constipation, or bladder outlet obstruction should be evaluated and remedied to the extent possible before elective herniorrhaphy. In patients with symptoms of altered bowel habits (i.e., frequent straining/constipation),

one must assess the risk of underlying colorectal malignancy. In incarcerated hernias with intestinal obstruction and possible strangulation, broad-spectrum antibiotics should be given and nasogastric suction may be necessary. Correction of volume status and electrolyte abnormalities is also important when there is associated small bowel obstruction.

2. Reduction. In uncomplicated cases, the hernia should reduce with palpation over the inguinal canal while the patient is supine. If this does not occur, the physician should apply gentle pressure over the hernia with the concavity of the palm of his/her hand and fingers and exert a steady but gentle pressure as follows: Craniad and lateral for direct and indirect hernias, craniad and posterior for femoral hernias. If this is not successful, gentle traction over the mass with compression may allow bowel gas to leave the herniated segment, making the mass reducible. Sedation and Trendelenburg position may be required for reduction of an incarcerated hernia, but it may be difficult to distinguish between acute incarceration and strangulation, as the inguinal canal can become quite tender with or without ischemic contents. When an incarcerated hernia is reduced nonsurgically, the patient should be observed for the potential development of peritonitis caused by perforation or ischemic necrosis of a loop of strangulated bowel. Strong suspicion of strangulation (i.e., erythema over hernia site, pain out of proportion to examination) is a surgical emergency, and the patient should be taken expeditiously to the operating room.

3. Surgical treatment

a. Choice of anesthetic. Local anesthesia with sedation and monitored anesthesia care (MAC), which has several advantages over general or regional (spinal or epidural) anesthesia, is the preferred anesthetic for elective open repair for small- to moderate-sized hernias. This approach results in better postoperative analgesia, a shorter recovery room stay, and a negligible rate of postoperative urinary retention; it is also the lowest-risk anesthetic for patients with underlying cardiopulmonary disorders. Commonly, a mixture of a short-acting agent (lidocaine 1%) and longer-acting agent (bupivacaine 0.25% to 0.50%) is used. The dose limits for local anesthesia are 4.5 mg/kg of plain lidocaine or 7 mg/kg of lidocaine with 1:100,000 epinephrine and 2 mg/kg plain bupivacaine or 3 mg/kg bupivacaine with 1:100,000 epinephrine. Use of local anesthesia for herniorrhaphy in our hospital is routinely supplemented by MAC 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. In contrast, laparoscopic inguinal

hernia repair is performed under general anesthesia to facilitate tolerance of pneumoperitoneum.

b. Treatment of the hernia sac. For indirect hernias, the sac (peritonealized abdominal contents) is dissected from the spermatic cord and cremasteric fibers (Fig. 38-1). The sac can either be ligated deep into the internal ring with an absorbable suture after reduction of

herniated contents or invaginated back into the abdomen without ligation; the latter approach is thought to be associated with less postoperative pain because it avoids transection of innervated peritoneum (*Hernia.* 2014;18(2):199-204). 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 or torsion. Cord lipomas are frequently encountered during repair and should be excised or reduced into the preperitoneal space to avoid future confusion with a recurrent hernia. Sliding hernia sacs can usually be managed by reducing the sac and attached viscera without sac ligation. Direct sacs are usually too broadly based for ligation and do not need to be opened. The redundant attenuated tissue is inverted and if the defect is small, it may be closed with a few interrupted sutures before placement of mesh.

c. Primary tissue repairs without mesh were the mainstay of hernia surgery prior to the development of synthetic-mesh approaches. While primary repair avoids placement of foreign prosthetic material, disadvantages of this approach include higher recurrence rates (5% to 10% for primary repairs and 15% to 30% for repair of recurrent hernias) due to tension on the repair and a slower return to unrestricted physical activity. Although the vast majority of hernias are now treated with a tension-free mesh repair, a primary tissue repair can be considered in contaminated wounds (e.g., if strangulated bowel is resected), as placement of synthetic material would be contraindicated. The principal features of the more commonly performed tissue repairs are as follows:

(1) Bassini repair. The inferior arch of the transversalis fascia or conjoint tendon is approximated to the shelving portion of the inguinal ligament (iliopubic tract) with interrupted, nonabsorbable sutures. The Bassini repair has been used primarily for indirect hernias, including inguinal hernias in women.

(2) McVay repair. The transversalis fascia is sutured to Cooper ligament medial to the femoral vein and to the inguinal ligament at the level of, and lateral to, the femoral vein. This operation requires placement of a relaxing incision medially on the aponeuroses of the internal oblique muscle to avoid undue tension on the repair. Historically, this approach was used more commonly for direct hernias. The McVay repair closes the femoral space and therefore, unlike the Bassini repair, is also effective for femoral hernias.

(3) 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 (Thornhill, ON, Canada) with this repair has

P.710

been excellent, with recurrence rates of less than 1%, but higher recurrence rates have been reported in nonspecialized centers.

d. Open tension-free repairs. The most common mesh inguinal hernia repairs performed today are the tension-free mesh hernioplasty (**Lichtenstein repair**) and the **patch-and-plug** technique. In the Lichtenstein repair, a piece of polypropylene mesh measuring approximately 6 × 3 in is used to reconstruct the inguinal floor (Fig. 38-2). The mesh is sutured to the transversalis fascia and conjoint tendon medially and to the inguinal ligament laterally. 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. 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. The Prolene hernia system is another technique

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that involves the use of a bilayer mesh in which the posterior leaflet is placed in the preperitoneal space and the anterior leaflet is sutured to the same layers as that in the Lichtenstein repair. Note that it is important to make an attempt to identify the ilioinguinal nerve but it should not be dissected out from its sheath or exteriorized behind the external oblique aponeurosis as was done historically because of the risk of exposing the nerve to fibrosis around the mesh that could result in neuropathic groin pain. If, despite this, the nerve will potentially be exposed directly to the mesh, it may be preferable to resect it for 3 to 4 cm proximal to the internal ring and to allow the proximal end to retract into the muscle to minimize subsequent neuroma formation.

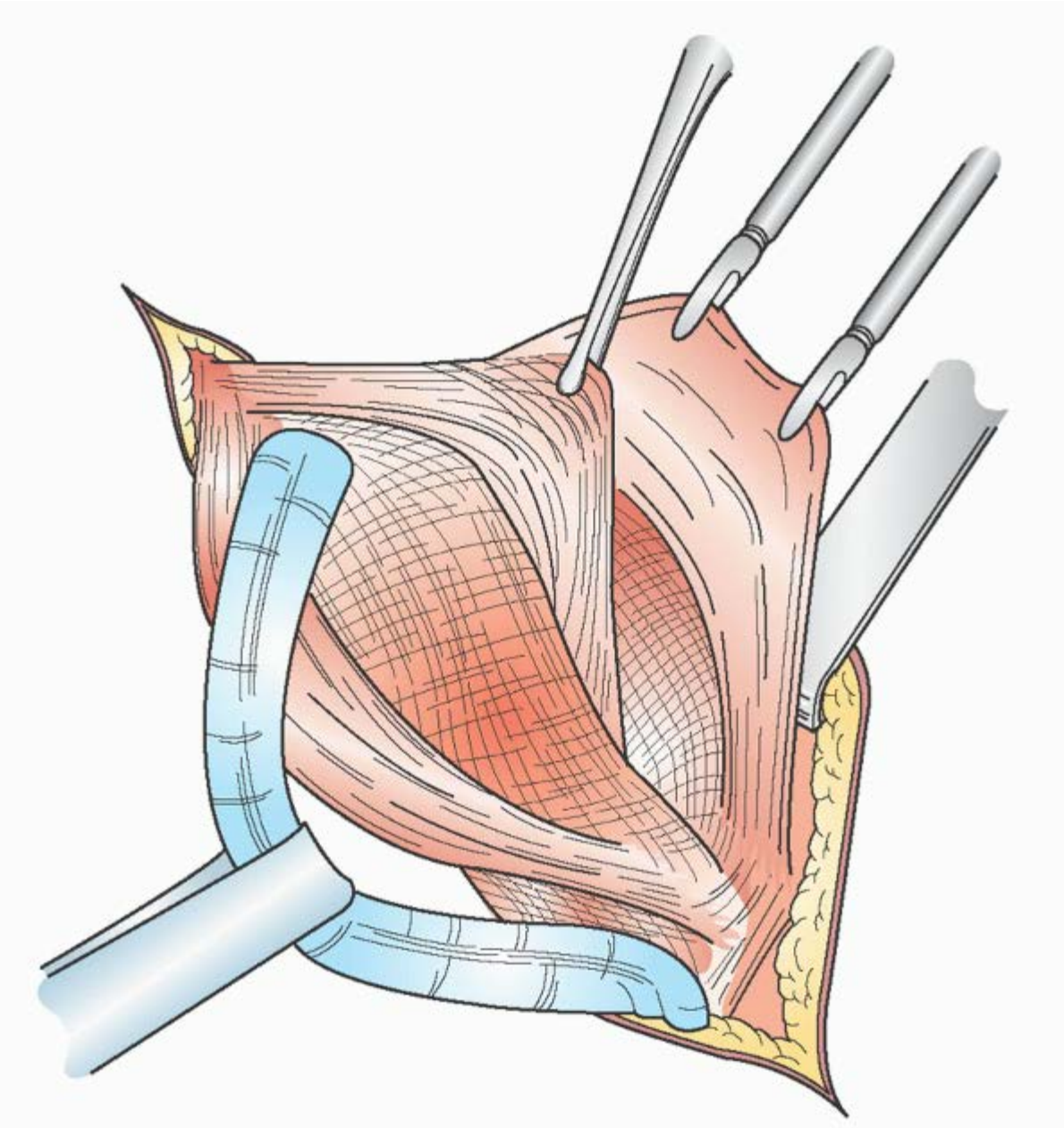


Figure 38-2 Lichtenstein tension-free hernia repair.

e. Laparoscopic inguinal hernia repair. The laparoscopic hernia repair is based on the technique of the late French-Algerian surgeon RenŽ 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. Laparoscopic hernia repair is typically advocated in the elective setting but is not generally used for patients presenting with signs and symptoms of incarceration or strangulation. Contraindications to the laparoscopic approach include inability to tolerate general anesthesia and/or pneumoperitoneum or the presence of a hernia with a significant scrotal component as it is more difficult to reduce laparoscopically. The laparoscopic approach is also relatively contraindicated in the patient who has previously undergone prostatectomy or other

lower midline abdominal surgery due to scarring in the preperitoneal space. There are two approaches to laparoscopic repair of inguinal hernias:

(1) Transabdominal preperitoneal (TAPP) repair. In the TAPP technique, the peritoneal space is entered by conventional means at the umbilicus, the peritoneum overlying the inguinal floor is dissected away as a flap, the hernia is reduced, mesh is fixed over the internal ring opening in the preperitoneal space, and the peritoneum is reapproximated. The advantages of the TAPP approach are that a large working space is retained, familiar anatomic landmarks are visible, and the contralateral groin can be examined for an occult hernia.

(2) Totally extraperitoneal repair (TEP). In the TEP technique, the preperitoneal space is developed with a dissecting balloon inserted between the posterior rectus sheath and the rectus abdominis and directed toward the pelvis inferior to the arcuate ligament (Fig. 38-3). The other ports are inserted into this preperitoneal space without entering the peritoneal cavity. The advantages of the TEP repair are that the peritoneum is not opened and, therefore, does not need to be closed and the operation is also typically faster to perform.

In either the TAPP or TEP technique, a large piece of mesh (6 × 4 in) is placed over the inguinal floor and fixed (1) superiorly to the posterior abdominal wall fascia on either side of

the inferior epigastric vessels, (2) inferiorly to Cooper ligament, (3) medially to the midline fascia, and (4) superolaterally to the fascia above the internal ring. Staples/tacks must not be placed inferomedial to the internal ring or inferior to the iliopubic tract because of the risk of injury to the external iliac vessels (triangle of doom) and ilioinguinal, genitofemoral, lateral femoral cutaneous, and femoral nerves (triangle of pain).

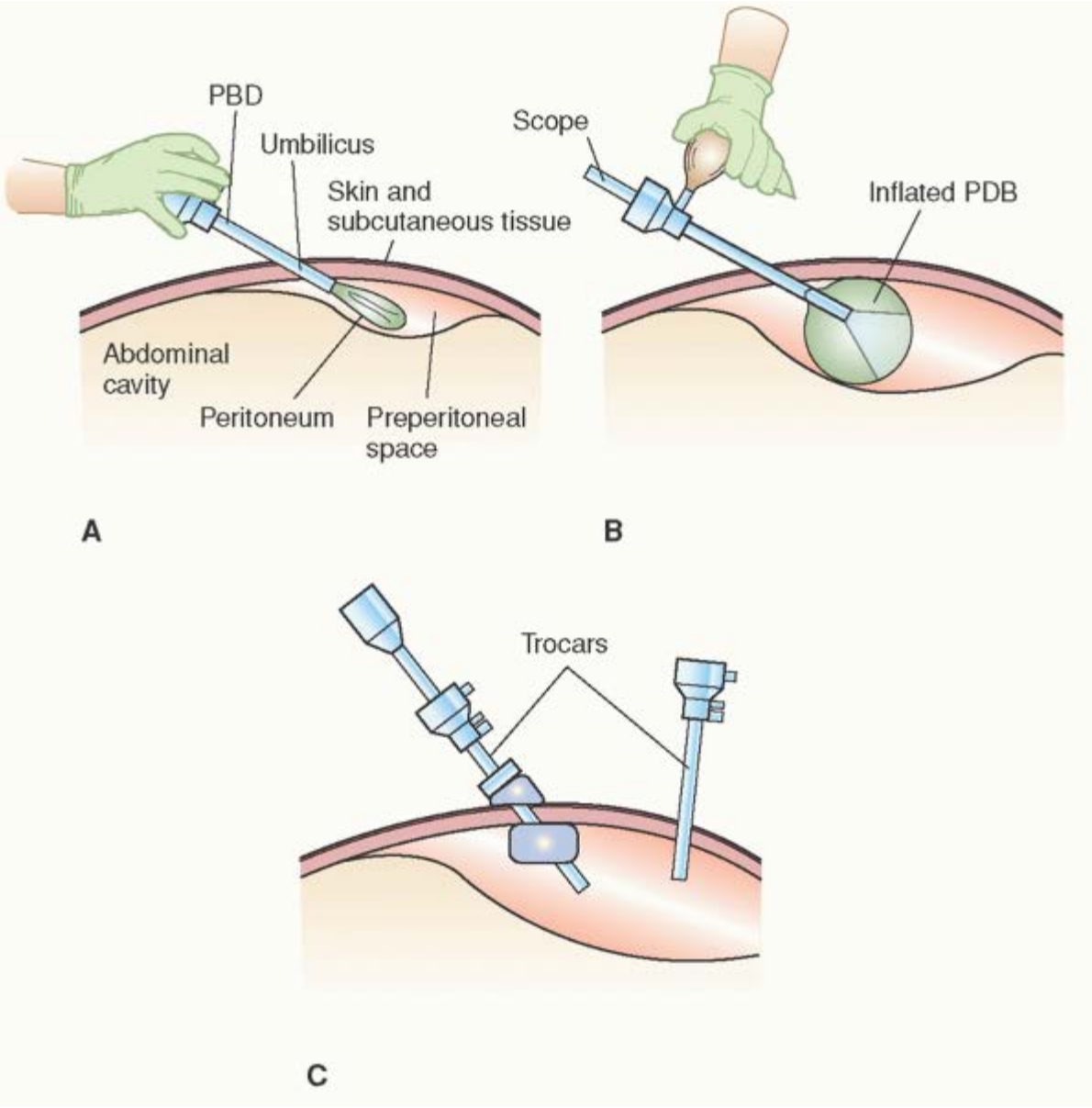


Figure 38-3 Laparoscopic total extraperitoneal approach with preperitoneal balloon dilation (PBD).

Studies comparing laparoscopic and open approaches to inguinal hernia repair have shown that laparoscopic repair is associated with less postoperative pain and faster recovery than open repair but that hospital costs have been higher for the laparoscopic technique. Operative times, complications, and recurrence rates (<3% for both laparoscopic and open repairs) have been similar. A randomized trial comparing open and laparoscopic

mesh inguinal hernia repairs at 14 Veterans Affairs (VA) institutions concluded that the open technique was superior to the laparoscopic technique for mesh repair of primary hernias due to decreased recurrence (4% vs. 10.1%) and complication rates (33.4% vs. 39%) (*N Engl J Med.*

2004;350:1819). However, this study has been criticized for both the lack of expertise in the laparoscopic group and the high rate of hernia recurrence in both groups. More recent randomized controlled trials, including the LEVEL trial of 660 patients randomized to Lichtenstein or TEP repair, concluded that laparoscopic repair was associated with earlier discharge from hospital, quicker return to normal activity and work, and significantly fewer postoperative complications than open inguinal hernia repair. Operating times were significantly longer for laparoscopic repairs ($p < 0.001$), but recurrence rate at mean followup of 49 months was similar ($p = 0.64$) (*Ann Surg.* 2010;251:819-824; *Int J Surg.* 2010;8:25-28).

Special circumstances in which laparoscopic repair may also be favored par 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 incisions, (3) in individuals with a unilateral hernia for whom a rapid recovery is critical (e.g., athletes and laborers), and (4) in obese patients.

f. Complications. Surgical complications include 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 less than 2%.

g. Recurrent inguinal hernias are more difficult to repair because scarring makes dissection difficult and because the hernia-producing disease process has continued to progress subsequent to the initial repair. Early recurrences within a few weeks or months of the initial repair suggest an inadequate initial repair and may reflect failure to identify an indirect hernia sac, whereas recurrence after 1 or more years suggests progression of the disease process that caused the initial hernia (e.g., increased intra-abdominal pressure, degeneration of tissues). Recurrences should generally be repaired because the defect usually is small with fixed edges that are prone to complications such as incarceration or strangulation. Repair after open inguinal hernia repair can be done by an anterior approach through the old operative field or by a posterior (open preperitoneal or laparoscopic) approach. Prosthetic mesh is used to reinforce attenuated tissues unless the operative field is contaminated.

F. Choice of Prosthetic Mesh in Inguinal Hernia Repairs. The choice of mesh for inguinal hernia repair is expanding rapidly as manufacturers compete to produce the ideal prosthetic material. The ideal material provides a

combination of adequate strength to prevent recurrence and flexibility to minimize chronic postoperative pain and/or the sensation of a foreign body. Examples of the three basic classes of synthetic meshes available to surgeons for use in inguinal hernia repair are summarized in Table 38-1; however, this is not an exhaustive list. Randomized clinical trials demonstrate that the use of lightweight polypropylene meshes for Lichtenstein hernia repair does not increase recurrence rates and is associated with less postoperative pain and discomfort (*Hernia.* 2010;14:253-258). These results support the use of lightweight mesh materials in inguinal hernia repair.

TABLE 38-1 Weight Classes of Mesh Used in Inguinal Hernia Repair

	Marlex[a] (Heavyweight)	Prolene Soft[b] (Midweight)	Ultrapro[b] (Lightweight)
Material	Polypropylene	Polypropylene	Polypropylene, poliglecaprone
Weight (g/m^2)	95	45	28
Pore size (mm)	0.6	2.4	4
Burst strength (newtons)	1,218	590	576
Stiffness (newtons/cm)	59.1	49.1	43.2

[a] Davol, Inc., Cranston, RI.

[b] Ethicon, Inc., Somerville, NJ.

Adapted from Cobb WS, Burns JM, Peindl RD, et al. Textile analysis of heavy weight, midweight, and light weight polypropylene mesh in a porcine ventral hernia model. *J Surg Res.* 2006;136(1):1-7.

II. FEMORAL HERNIAS

A. Incidence. Femoral hernias constitute between 2% and 4% of all groin hernias, with over 90% occurring in women. Approximately 25% of femoral hernias 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 ligament posteriorly.

C. Diagnosis

1. Clinical presentation

a. Symptoms. Patients may complain of an intermittent groin bulge or a groin mass that may be tender. Femoral hernias have a high

incidence of incarceration and 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 that for inguinal hernia.

c. 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 ligament repair (McVay) using 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.

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 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 HERNIA

A. Incidence. Of patients who present with acute intestinal obstruction, less 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. Internal hernia is also a common cause of small bowel obstruction after laparoscopic gastric bypass surgery, as the small bowel can herniate through a residual mesenteric defect. Adhesive bands from prior operations may also cause or contribute to mechanical obstruction.

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% to 16%. **Symptoms** usually are of intestinal obstruction without evidence of an external hernia. When there is intestinal obstruction or intestinal strangulation, the diagnosis is based on clinical rather than on laboratory findings.

2. Radiographic studies. Plain abdominal films may show dilated loops of bowel and air-fluid levels as one might see with small bowel obstruction. An abdominal CT scan is usually necessary to establish the diagnosis of an internal hernia preoperatively.

D. Differential diagnosis includes other causes of intestinal obstruction, such as adhesions, external hernia, malignancy, gallstone ileus, and intussusception.

E. Surgical Treatment. The diagnosis of internal hernia is often made at laparoscopy or laparotomy for small bowel obstruction. Intestinal loops proximal to the obstruction are dilated 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 second-look laparotomy in 24 to 48 hours may preserve small bowel length. The hernia defect should be closed primarily with nonabsorbable suture.

IV. ABDOMINAL WALL HERNIA

A. Incidence and Etiology

1. Incisional hernias occur at sites of previous incisions at which there has been a division of abdominal wall fascia. Contributing factors include obesity, wound infection, malnutrition,

smoking, and technical errors in wound closure. Hernias occur in up to 20% to 30% of patients undergoing abdominal operations and are most commonly seen with midline incisions. Most incisional hernias are now repaired with a mesh prosthetic via open or laparoscopic approach.

2. Umbilical hernias are congenital defects that may enlarge over time and become protuberant and symptomatic. They are more frequent in people of African ancestry. 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. Small umbilical hernias can be present for years without causing symptoms and may even go unnoticed. Over time, however, these hernias can enlarge and become incarcerated, usually with preperitoneal fat or omentum. Umbilical hernias greater than 3 cm should be repaired with a prosthetic mesh.

3. Epigastric hernias are hernias of the linea alba above the umbilicus. They occur more frequently in athletically active young men or women. When small or in obese individuals, epigastric hernias may be hard to palpate and difficult to diagnosis. 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 to 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: The **Petit (inferior lumbar)** triangle and the **GrynfelttÑLesshaft (superior lumbar)** triangle, although these hernias are quite rare. Petit hernias are located in an area limited posteriorly by the latissimus dorsi, anteriorly by the external oblique muscle, and inferiorly by the iliac crest; the floor is formed by the internal abdominal oblique muscle. GrynfelttÑLesshaft hernias are bordered superiorly by the 12th rib, medially by the quadratus lumborum muscle, and laterally by the internal abdominal oblique muscle, while the floor and roof of the triangle are formed by the transversalis fascia and the external abdominal oblique muscle, respectively.

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 with internal rotation of the thigh, known as the *HowshipÑRomberg 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 as well as lumbar and obturator hernias require the use of a prosthetic mesh because of their size and high recurrence rates after primary repair.

1. Prosthetic mesh in abdominal wall hernia repairs. The recurrence rate for ventral incisional hernia repair is 31% to 54% when primarily repaired. The technique of placing prosthetic biomaterials in the retrorectus preperitoneal space to repair ventral incisional hernias was popularized by Jean Rives and RenŽ Stoppa and has reduced the recurrence rate of these hernias to between 4% and 24%. Long-term followup of a randomized controlled trial showed that the use of mesh results in a lower recurrence rate and less abdominal pain and does not result in more complications than primary repair (*Hernia.* 2006;10:236-242). The choice of mesh for incisional hernia repair should be based on location and mesh properties. **Uncoated polypropylene mesh** should not be placed in an intraperitoneal position because it can form dense adhesions to the intestine and precipitate fistulization. For intraperitoneal placement, either microporous polytetrafluoroethylene (PTFE) mesh or a barrier-coated mesh should be used. **Microporous PTFE mesh** has a microporous architecture and hydrophobicity that prevent cellular penetration of intestine or abdominal viscera and may reduce the density of intraperitoneal adhesions. There are also several absorbable **barrier-coated meshes** with a polypropylene or polyester construction. Different types of mesh are summarized in Table 38-2. Currently, there are limited data to support the use of one product over another. What is clear, however, is that the use of mesh is superior to primary repair for incisional hernias.

2. Open repairs. The principles for ventral hernia repair include dissection and identification of all defects and repair with nonabsorbable sutures placed in healthy tissue. Incisional hernias should be repaired with mesh prosthesis that should be anchored by nonabsorbable sutures placed at least 3 cm beyond the margins of the defect. The placement of mesh in the retrorectus space, that is, **the RivesÑStoppa technique,** has the lowest reported recurrence rate for hernia repair. This technique includes closure of the posterior rectus sheath beneath the mesh, allowing for the use of uncoated mesh. The principles of this repair have been expanded to include transverse abdominis release for the repair of giant ventral/incisional hernias. The mesh utilized for these repairs 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, PTFE, Gore-Tex, and a composite mesh of polypropylene and PTFE. Several composite mesh products (Table 38-2), with absorbable barriers coating and external to polypropylene or polyester mesh cores, are available to minimize tissue attachment to intra-abdominal structures.

3. Laparoscopic repairs. The laparoscopic approach is another method for repair of incisional hernias. The repair involves placement of a mesh prosthesis to cover the hernia defect. This approach requires adhesiolysis of the entire prior incision, reduction of herniated abdominal

contents, and broad coverage with PTFE or a barrier-coated mesh. The mesh is anchored in place with sutures and tacks with a minimum of 4 cm overlap past the edge of the hernia defect on all sides. A pooled data analysis of 45 published series comparing open and laparoscopic ventral hernia repairs concluded that laparoscopic repair is associated with fewer wound-related (3.8% vs. 16.8%) and overall complications (22.7% vs. 41.7%) and has a lower rate of recurrence (4.3% vs. 12.1%) than open repairs (*Surg Endosc.* 2007;21:378-386). Contraindications to laparoscopic ventral hernia repair include inability to establish pneumoperitoneum safely, peritonitis or an acute abdomen with strangulated or infarcted bowel, or loss of abdominal domain.

TABLE 38-2 Commonly Used Biomaterials for Incisional Hernia Repair

	Product Trade Name	Manufacturer	Components
Absorbable barrier composite meshes	Sepramesh	Genzyme Corp., Cambridge, MA	Polypropylene mesh on one side, absorbable sodium hyaluronate/carboxymethylcellulose on the other side
	C-Qur	Atrium Medical, Hudson, NH	Lightweight polypropylene mesh (Prolite) coated with omega-3 fatty acid
	Parietex	Covidien-Medtronic, Minneapolis, MN	Polyester mesh with bovine type I collagen coating covered with absorbable PEG/glycerol layer
	Proceed	Ethicon, Inc., Somerville, NJ	Polypropylene mesh encapsulated with polydioxanone coated on one side with oxidized regenerated cellulose

	Physiomesh	Ethicon, Inc., Somerville, NJ	Polypropylene mesh laminated between two poligle-caprone-25 films with polydioxanone film
Nonabsorbable, barrier composite mesh	Bard Composix	C.R. Bard, Inc., Murray Hill, NJ	Macroporous bilayer mesh; polypropylene and microporous PTFE
	Gore-Tex Dual Mesh	W.L. Gore & Associates, Flagstaff, AZ	PTFE with different architecture on the peritoneal (intraabdominal) and parietal (abdominal wall) surfaces of the mesh
Bioremodelable materials (aka biologic meshes)	Surgisis	Cook Biotech, Inc., West Lafayette, IN	Acellular, extracellular matrix material derived from porcine small intestinal submucosa
	Alloderm	LifeCell Corp., Branchburg, NJ	Acellular dermal matrix harvested from cadaveric human dermis
	Flex HD	Musculoskeletal Transplant Foundation, Edison, NJ	Acellular dermal matrix harvested from cadaveric human dermis
	Strattice	LifeCell Corp., Branchburg, NJ	Acellular porcine dermal matrix
	Permacol	Tissue Science Laboratories, Covington, NJ	Acellular, cross-linked porcine dermal matrix
	Bio-A	W.L. Gore & Associates,	Biocompatible synthetic polymers

Flagstaff, AZ

PEG, polyethylene glycol; PTFE, polytetrafluoroethylene.

CHAPTER 38: HERNIAS

Multiple Choice Questions

1. Which of the following is true regarding inguinal hernias?

a. Inguinal hernia is more common in women than men.

b. Inguinal hernias are rarely bilateral.

c. Direct inguinal hernias are more common than indirect.

d. Recurrent hernias are more likely to be direct than indirect.

e. There is no difference in recurrence rate based on type of repair.

View Answer

2. An 83-year-old thin woman with no history of abdominal surgery presents with symptoms of a small bowel obstruction. On physical examination, she has pain with medial (internal) thigh rotation. There is no palpable hernia in the groin. What is the most likely diagnosis?

a. Femoral hernia

b. Inguinal hernia

c. Spigelian hernia

d. Obturator hernia

e. Adhesive small bowel obstruction

View Answer

3. A 35-year-old woman presents with a 1-day history of abdominal pain, distension, and nausea. Physical examination reveals temperature of 38.5°C, heart rate 115, abdominal distention, and a tender bulge in the right groin with erythema. What is the most appropriate next step in management?

a. Ultrasound of the right groin

b. CT scan of the abdominal/pelvis

c. Admission, NG tube decompression, IV fluid resuscitation

d. Laparoscopic inguinal hernia repair

e. Open inguinal hernia repair

View Answer

4. A 28-year-old active man with an athletic build is being evaluated for a 5-cm wide incisional hernia from a previous exploratory laparotomy for trauma. Which of the following repairs is preferred for this patient?

a. Open repair with intra-abdominal placement of synthetic mesh

b. Laparoscopic repair with synthetic mesh

c. Open repair with retrorectus placement of synthetic mesh

d. Laparoscopic repair with barrier-coated synthetic mesh

e. Open repair with retrorectus placement of biologic mesh

View Answer

5. A 70-year-old man with a history of congestive heart failure (CHF) and a prior open prostatectomy presents with a symptomatic but reducible right inguinal hernia. On examination, there is concern for a small asymptomatic left inguinal hernia. What is the most appropriate management strategy?

a. Bilateral open inguinal hernia repair without mesh

b. Laparoscopic repair of right inguinal hernia with mesh and evaluation of left side

c. Laparoscopic repair of right inguinal hernia and coverage of left inguinal floor

d. Open repair of right inguinal hernia with mesh and watchful waiting of left side

e. Watchful waiting of right inguinal hernia and potential left inguinal hernia

View Answer

39

Diseases of the Adrenal and Pituitary Gland and Hereditary Endocrine Syndromes

Kathryn J. Rowland

Jeffrey F. Moley

ADRENAL-PITUITARY AXIS

I. EMBRYOLOGY, ANATOMY, AND PHYSIOLOGY

A. Embryology. The **adrenal cortex** arises from the coelomic mesoderm around the fifth week of gestation. The **adrenal medulla** is populated by the neural crest cells originating from the neural ectoderm. The consequence of this migration is evident by the existence of **paragangliomas** (extra-adrenal pheochromocytomas) all along the paraspinal axis.

B. Anatomy. The adrenal glands are located in the retroperitoneum superior to the kidney and lateral to the vena cava (on the right) and aorta (on the left). This relationship is important in determining the vascular supply to the adrenals. Each adrenal is supplied by three arteries: **Superior adrenal artery** (arises from the inferior phrenic artery), **middle adrenal artery** (branch of the aorta), and **inferior adrenal artery** (branch of the renal artery). The right adrenal vein drains directly into the vena cava. The left adrenal vein drains into the left renal vein.

C. Physiology. The adrenal gland is histologically composed of four layers, each with their own biosynthetic products.

1. Adrenal cortex

a. Zona glomerulosa is responsible for mineralocorticoid production, of which **aldosterone** is the primary product. Aldosterone production is stimulated by angiotensin II and increased levels of serum potassium. Aldosterone acts to increase circulating blood volume by increasing sodium and chloride reabsorption in the distal tubule of the kidney.

b. Zona fasciculata produces the glucocorticoids of the adrenal glands, of which cortisol is the primary product. Cortisol production is stimulated by the release of **adrenocorticotropic hormone (ACTH**) by the anterior pituitary gland. ACTH itself is stimulated by the release of **corticotropin-releasing hormone (CRH)** by the hypothalamus. Glucocorticoids have extremely broad effects with the overall goal of inducing a catabolic state in the body in response to stress. Glucocorticoids increase blood glucose concentrations, stimulate lipolysis, enhance

adrenergic stimulation of the cardiovascular system, and reduce the inflammatory response of the immune system.

c. Zona reticularis produces the adrenal sex hormone androstenedione and DHEA. These hormones support the gonadal production of testosterone and estrogen.

2. The adrenal medulla produces the catecholamines **norepinephrine (noradrenaline)** and **epinephrine (adrenaline)** that act on peripheral α and β-adrenergic receptors. α Receptor stimulation produces peripheral vasoconstriction. β Simulation of the myocardium via β_1 receptors increases heart rate and contractility. Stimulation of peripheral β_2 receptors causes relaxation of smooth muscles.

II. BIOCHEMICAL EVALUATION AND IMAGING OF ADRENAL MASSES

A. Patients presenting with an adrenal mass should undergo a complete biochemical workup including (*Endocr Pract.* 2009;15:450-453):

1. Overnight dexamethasone suppression test with measurement of plasma cortisol to evaluate for a cortisol secreting mass.

2. Basic metabolic panel with measurement of serum potassium in hypertensive patients to evaluate for an aldosterone secreting mass.

3. Plasma **metanephrines** and **normetanephrines** to evaluate for a pheochromocytoma.

B. Malignant and benign adrenal masses can be distinguished on the basis of characteristic CT, MRI, and PET imaging (Table 39-1) (*Cancer Imaging.* 2010;10:102-113).

III. FUNCTIONAL ADRENAL MASSES

A. Cushing SyndromeÑHypercortisolism

1. The clinical manifestations of **Cushing 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.

2. Pathophysiology of excess circulating glucocorticoids.

a. Iatrogenic. Cushing syndrome is usually of iatrogenic etiology and secondary to the administration of exogenous glucocorticoids or ACTH.

b. Cushing disease. Hypersecretion of ACTH from the anterior pituitary gland (Cushing disease) is the most common pathologic cause (65% to 70% of cases) of endogenous hypercortisolism. The adrenal glands respond normally to the elevated ACTH, and the result is bilateral adrenal

hyperplasia. Excessive release of CRF by the hypothalamus is a rare cause of hypercortisolism.

c. Hypersecreting adrenal adenoma. Abnormal secretion of cortisol from a primary adrenal adenoma or carcinoma is the cause of hypercortisolism in 10% to 20% of cases. Primary adrenal neoplasms secrete corticosteroids independent of ACTH and usually result in suppressed plasma ACTH levels and atrophy of the adjacent and contralateral adrenocortical tissue.

TABLE 39-1 Imaging Characteristics of Adrenal Masses

Adrenal Mass	Imaging Characteristics
Adrenocortical adenoma	Unilateral, <4 cm in diameter Round, homogeneous density with smooth border Low attenuation on CT scan (<10 HU) Rapid, intense contrast enhancement followed by early contrast washout (absolute percent washout >60%, relative percent washout >40%) Microscopic fat with signal loss on opposed-phase MRI images
Myelolipoma	Macroscopic fat on CT Calcification Hyperintense signal on T1 MRI
Pheochromocytoma	Hypervascularity Rapid, intense contrast enhancement with variable delayed washout High T2 MRI signal intensity (light-bulb sign)
Adrenocortical carcinoma	Unilateral, >4 cm Irregular shape, heterogeneous, central tumor necrosis Calcification Increased attenuation on CT (>10 HU) Delay in contrast washout Elevated SUV on PET
Adrenal metastasis	Bilateral, irregular shape, heterogeneous

	Increased attenuation on CT (>10 HU) Delay in contrast washout Elevated SUV on PET

CT, computed tomography; HU, Hounsfield units; MRI, magnetic resonance image; PET, positron emission tomography; SUV, standardized uptake values.

d. Ectopic ACTH production. In approximately 15% of cases, Cushing syndrome is caused by ectopic secretion of ACTH or an ACTHlike 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 syndrome.

3. Diagnosis of Cushing syndrome is biochemical (Fig. 39-1). The goals are to first establish hypercortisolism and then identify the source.

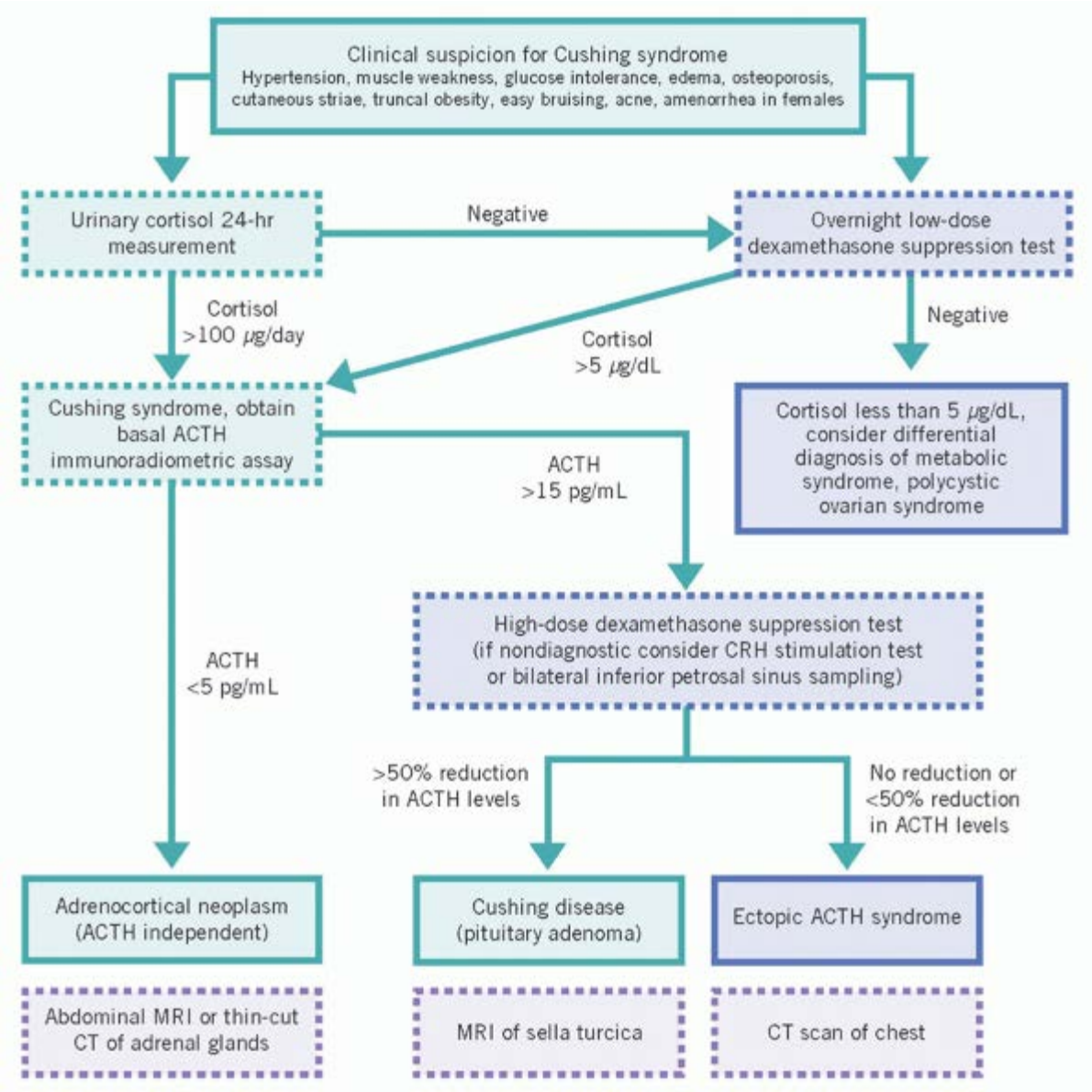


Figure 39-1 Diagnosis of Cushing syndrome is biochemical.

a. Establishing the presence of hypercortisolism:

(1) The best screening test for hypercortisolism is a **24-hour measurement of the urinary excretion of free cortisol.** Urinary excretion of more than 100 µg/day of free cortisol in two independent collections is virtually diagnostic of Cushing syndrome. Measurement of random plasma cortisol levels alone is not a reliable method of diagnosing Cushing syndrome due to overlap of the levels in normal and abnormal patients.

(2) A low-dose **overnight dexamethasone suppression test** (dexamethasone 1 mg orally is administered at 11 pm and plasma cortisol is measured at 8 am) is used to confirm Cushing syndrome, especially in obese or depressed patients who may have marginally elevated urinary cortisol levels. Patients with true hypercortisolism have lost normal adrenalÑpituitary 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 method of determining the cause of hypercortisolism. Suppression of the absolute level of ACTH to below 5 pg/mL is nearly diagnostic of adrenocortical neoplasms (ACTH-independent Cushing syndrome). ACTH levels in Cushing disease may range from the upper limits of to significantly above normal (15 pg/mL to 500 pg/mL). The 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 distinguish a pituitary source 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 orally every 6 hours for 48 hours) with a reduction in 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) CRH stimulation test may also be used to distinguish a pituitary source of ACTH from an ectopic source. Forty-five minutes following intravenous CRH administration, ACTH and cortisol levels are increased in patients with Cushing disease. Patients with a suppressed hypothalamicÑ pituitary axis (primary adrenal tumor, ectopic ACTH syndrome) usually do not have a compensatory rise in ACTH and cortisol levels in response to CRH.

c. Imaging tests are useful for identifying lesions suspected on the basis of biochemical testing.

(1) Patients with ACTH-independent hypercortisolism require thin-section CT scan or MRI scan of the adrenal gland, both of which identify adrenal abnormalities with more than 95% sensitivity.

(2) Gadolinium-enhanced MRI scan of the sella turcica is the best imaging test for pituitary adenomas suspected of causing ACTH-dependent hypercortisolism.

(3) Patients with ACTH-dependent hypercortisolism and either markedly elevated ACTH or a negative pituitary MRI scan should have CT scan of the chest to identify a tumor-producing ectopic ACTH.

(4) Bilateral inferior petrosal sinus sampling can delineate unclear cases of Cushing 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 of CRH. Levels of ACTH in the inferior petrosal sinus are compared to peripheral plasma ACTH levels: A basal ratio of ³2 or a ratio of ³3 after CRH administration is 100% sensitive and specific for pituitary adenoma.

4. Surgical treatment of Cushing syndrome involves removing the cause of cortisol excess.

a. Transsphenoidal resection of an ACTH-producing pituitary tumor is successful in 80% or more of cases of Cushing disease.

b. Treatment of ectopic ACTH syndrome involves resection of the primary lesion, if possible.

c. Primary adrenal causes of Cushing syndrome are treated by removal of the adrenal gland containing the tumor. All patients who undergo adrenalectomy for primary adrenal causes of Cushing syndrome require perioperative and postoperative glucocorticoid replacement because the pituitaryÑadrenal axis is suppressed. Recovery of the pituitaryÑadrenal axis may take as long as 6 to 18 months following unilateral adrenalectomy.

B. Conn SyndromeÑHyperaldosteronism

1. Primary hyperaldosteronism is a syndrome of hypertension and hypokalemia caused by hypersecretion of the mineralocorticoid aldosterone. Secondary aldosteronism is a physiologic response of the reninÑangiotensin system to renal artery stenosis, cirrhosis, congestive heart failure, and normal pregnancy. In these conditions, the adrenal gland functions normally.

2. Pathophysiology of primary hyperaldosteronism

a. 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.

b. Idiopathic bilateral adrenal hyperplasia (IHA) causes 30% to 40% of cases of primary aldosteronism.

c. Adrenocortical carcinoma and **autosomal dominant glucocorticoid-suppressible aldosteronism** are rare causes of primary aldosteronism.

3. Diagnosis

a. Given the prevalence of essential hypertension, it is not cost-effective to screen all adults with hypertension for primary hyperaldosteronism. Adults who should be evaluated include those with new onset severe hypertension at a young age and those whose blood pressure is labile or poorly controlled on three or more antihypertensives.

b. Laboratory diagnosis of primary aldosteronism begins with the demonstration of hypokalemia (<3.5 mEq/L), inappropriate kaliuresis (>30 mEq/day), and elevated aldosterone (>15 ng/dL) with normal cortisol. Aldosterone to renin ratio (ARR) is determined by the measurement of plasma renin activity (PRA) and plasma aldosterone concentration (PAC). This test is best performed upright, midmorning. An ARR of >20 is suggestive of primary hyperaldosteronism. Drugs that stimulate renin such as spironolactone, angiotensin-converting enzyme inhibitors (ACE inhibitors), angiotensin receptor antagonists (ARBs), or dihydropyridine calcium blockers (e.g., amlodipine) can produce false negatives. β-blockers, clonidine, and nonsteroidal anti-

in false positives (*Horm Metab Res.* 2012;44:170-176). Primary aldosteronism is confirmed by lack of aldosterone suppression (PAC >10 ng/dL) with salt loading. This is performed by having patients add a teaspoon of salt to each meal for 72 hours prior to PAC and PRA measurement. Alternatively, a 500 mL normal saline infusion can be administered.

c. 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 syndrome. Differentiation between APA and IHA is important because unilateral adenomas are treated by surgical excision, whereas bilateral hyperplasia is treated medically. Uncertainty regarding APA versus IHA after biochemical testing and noninvasive localization may be definitively resolved by **bilateral adrenal venous sampling** for aldosterone and cortisol. Simultaneous adrenal vein blood samples for aldosterone and cortisol are taken. The ratio of aldosterone to cortisol is greater than 4:1 for a diagnosis of aldosteronoma and less than 4:1 for a diagnosis of IHA.

4. Treatment. Surgical removal of an APA through a posterior or laparoscopic approach results in immediate cure or substantial improvement in hypertension and hypokalemia in more than 90% of patients with Conn syndrome. The patient should be treated with **spironolactone** (200 to 400 mg/day) preoperatively for 2 to 3 weeks to control blood pressure and to correct hypokalemia. Patients with IHA should be treated medically with spironolactone (200 to 400 mg/day). A potassium-sparing diuretic, such as **amiloride** (5 to 20 mg/day), and **calcium channel blockers** have also been used. Surgical excision rarely cures bilateral hyperplasia.

C. Pheochromocytoma

1. The clinical manifestations of pheochromocytoma include paroxysms of pounding frontal headache, diaphoresis, palpitations, flushing, or anxiety related to the excess sympathetic stimulation from catecholamines. The most common sign is episodic or sustained hypertension, but pheochromocytoma accounts for only 0.1% to 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).

2. Pathophysiology of pheochromocytoma (Table 39-2).

a. Pheochromocytomas are neoplasms derived from the chromaffin cells of the sympathoadrenal system that engage in unregulated, episodic oversecretion of catecholamines.

b. Approximately 80% to 85% of pheochromocytomas in adults arise in the adrenal medulla, whereas 10% to 15% arise in the extraadrenal chromaffin tissue, including the paravertebral ganglia, posterior mediastinum, organ of Zuckerkandl, and urinary bladder.

TABLE 39-2 Pheochromocytoma Rule of Tens

Ten Percent of Pheochromocytomas are:

Bilateral

Malignant

Extraadrenal

Familial

In children

c. Pheochromocytomas can occur in association with several hereditary syndromes, including MEN types 2A and 2B and von Hippel-Lindau syndrome. Tumors that arise in familial settings frequently are bilateral. Young patients with pheochromocytomas or patients with bilateral or extraadrenal pheochromocytomas should undergo genetic testing.

3. The biochemical diagnosis of pheochromocytoma is made by demonstrating elevated plasma metanephrines and/or normetanephrines or 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.

4. Radiographic tests are used to demonstrate the presence of an adrenal mass.

a. CT scanning is the imaging test of choice and identifies 90% to 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 compared with adenomas (Fig. 39-2).

b. Scintigraphic scanning after the administration of ^{131}I-meta-iodobenzylguanidine (MIBG) provides a functional and anatomic test of hyperfunctioning chromaffin tissue. MIBG scanning is very specific for both intra- and extra-adrenal pheochromocytomas.

5. The treatment of benign and malignant pheochromocytomas is surgical excision.

a. Preoperative preparation includes administration of an α-adrenergic blocker to control hypertension and to permit re-expansion of intravascular volume. Phenoxybenzamine, 10 mg orally twice a day, is initiated and increased to 20 to 40 mg orally twice a day until the desired effect or prohibitive side effects are encountered. Postural hypotension is expected and is the desired end point. β-Adrenergic blockade (e.g., propranolol) may be added if reflex tachycardia or arrhythmias develop but should only be initiated after complete α-adrenergic blockade. Patients with cardiopulmonary dysfunction may require a pulmonary artery catheter (SwanÑGanz)

perioperatively, and all patients should be monitored in the surgical intensive care unit in the immediate postoperative period.

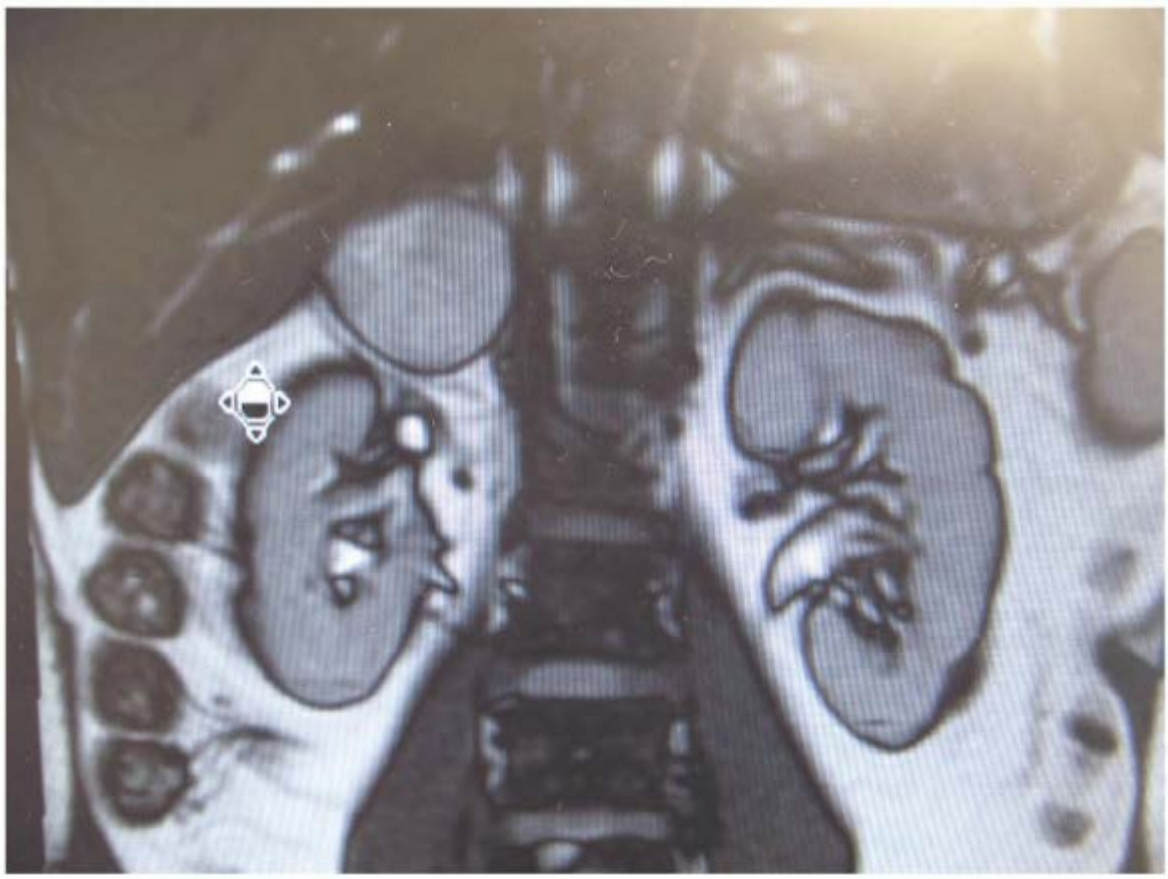


Figure 39-2 MRI T2-weighted image showing typical appearance for pheochromocytoma.

b. In patients with a sporadic, unilateral pheochromocytoma localized by preoperative imaging studies, adrenalectomy may be performed by an anterior or posterior open approach or, increasingly, by laparoscopic adrenalectomy. In patients with MEN type 2A or 2B and a unilateral pheochromocytoma, it is acceptable to remove only the involved gland (*Ann Surg.* 1993;217:595). The classic operative approach for familial pheochromocytomas is exploration of

adrenal glands, the preaortic and paravertebral areas, and the organ of Zuckerkandl through a midline or bilateral subcostal incision.

c. 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 to 10 µg/kg/minute) or phentolamine (5 mg).

d. Following pheochromocytoma excision, 10% to 15% of patients will develop recurrence. Long-term followup with measurement of plasma metanephrines and normetanephrines is necessary.

IV. NONFUNCTIONAL ADRENAL MASSES

A. Adrenocortical adenomas comprise the majority of incidentally discovered adrenal masses (incidentalomas). Incidental adrenal masses are detected

in 0.4% to 4.4% of abdominal CT scans obtained for other reasons. Adrenocortical adenomas are benign, and nonfunctioning, with the majority having no clinical significance.

1. Surgery should be performed in patients with nonfunctional adrenal adenomas greater than 6 cm in diameter given the increased probability of adrenocortical carcinoma in masses of increasing size. Strong consideration for removal of tumors between 4 and 6 cm should be based upon clinical scenario, patient age and comorbidities, and imaging characteristics. This recommendation is based upon the National Institutes of Health Consensus Statement (*NIH Consens State Sci Statements.* 2002;19:1-25). In contrast, the American Association of Clinical Endocrinologists and American Association of Endocrine Surgeons guidelines recommend resection of any mass greater than or equal to 4 cm (*Endocr Pract.* 2009;15:450-453). Tumors less than 4 cm should be monitored clinically and radiologically in 3 to 6 months followed by annually for 2 years. Any tumor that enlarges by more than 1 cm during the followup period should be removed. Biochemical testing should be performed annually for 5 years.

2. Either open or laparoscopic adrenalectomy is acceptable. Laparoscopic adrenalectomy has been associated with shorter hospitalization, faster recovery, and lower morbidity (*Br J Surg.* 2004;91:1259-1274). Its use is generally limited to malignant lesions less than 5 cm in diameter and benign-appearing lesions up to 10 cm in diameter. Both laparoscopic transperitoneal adrenalectomy and laparoscopic retroperitoneal adrenalectomy have been described with no difference in terms of operative time, blood loss, length of hospitalization, time to oral intake, morbidity, or mortality (*Surgery.* 2013;153:111-119).

B. Adrenal myelolipoma is a benign tumor composed of mature fat and hematopoietic elements.

1. Myelolipomas have characteristic macroscopic fat on CT imaging.

2. Myelolipomas may enlarge over time. Routine followup imaging is not necessary. Surgical excision is only indicated for masses causing local mass-effect symptoms. Spontaneous

retroperitoneal hemorrhage may occur with large masses. Approximately 4% of patients diagnosed with a myelolipoma will require adrenalectomy (*J Surg Oncol.* 2012;106:557-564).

C. Adrenocortical carcinoma is a rare but aggressive malignancy. Most patients with this cancer present with locally advanced disease.

1. Syndromes of adrenal hormone overproduction may include rapidly progressive hypercortisolism, hyperaldosteronism, or virilization (Fig. 39-3). Large (>6 cm) adrenal masses that extend to nearby structures on CT scanning likely represent carcinoma.

2. 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.

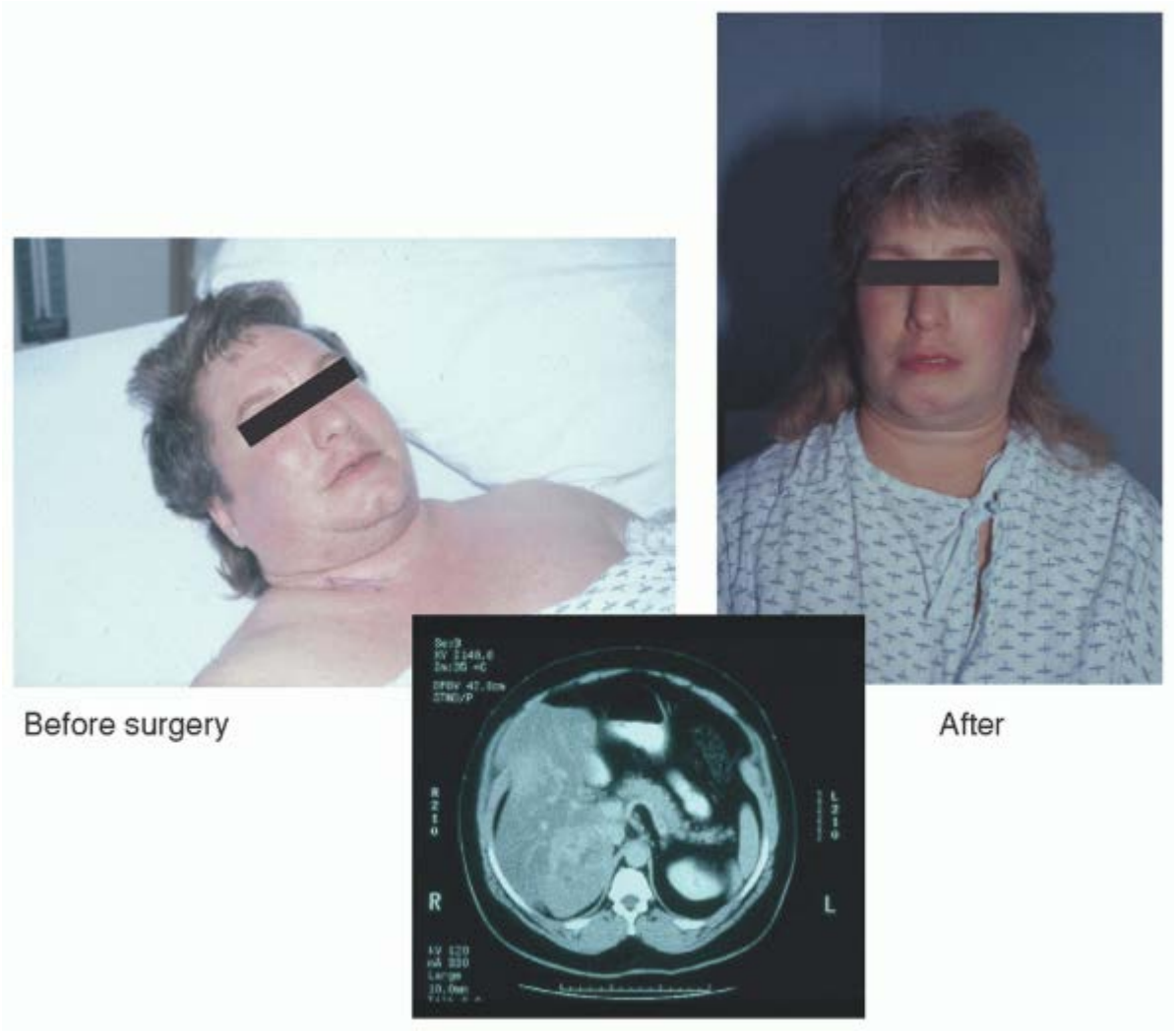


Figure 39-3 Adrenocortical carcinoma.

3. 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.

D. Adrenal metastases are the most common malignant lesions involving the adrenal gland. Frequently bilateral lesions are present.

1. Lung, breast, melanoma, colorectal, thyroid, and pancreatic cancer all metastasize to the adrenal glands. Renal cell carcinoma and hepatocellular carcinoma may also metastasize to the adrenals.

2. Diagnosis of metastatic disease can often be made from imaging and a history of cancer. Need for pathologic confirmation of metastatic disease is rare, but if necessary, biochemical testing for pheochromocytoma should be performed prior to biopsy.

3. Patients with bilateral metastatic disease should be evaluated for adrenal insufficiency.

4. Adrenalectomy for metastatic disease is rarely indicated but may be considered for an isolated adrenal lesion.

E. Collision tumor is a rare tumor that represents the coexistence of two histologically distinct but adjacent adrenal masses (*Cancer Imaging.* 2013;13:602-610).

1. Collision tumors may be composed of an adenoma with myelolipoma, adenoma with metastases, and adrenocortical carcinoma with myelolipoma.

2. Hemorrhage into an existing adrenal mass can mimic a collision tumor.

V. ACUTE ADRENAL INSUFFICIENCY

A. Acute adrenal insufficiency is an emergency and should be suspected in physiologically 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 occur in the postoperative setting following adrenal surgery or from autoimmune destruction of the adrenal cortex, adrenal hemorrhage (**Waterhouse-Friderichsen syndrome**), or, rarely, infiltration with metastatic carcinoma. The diagnosis and treatment of acute adrenal insufficiency in patients in septic shock is very controversial. Two prospective randomized trials have shown different effects in the use of hydrocortisone in patients with septic shock (*N Engl J Med.* 2003;348:727-734; *N Engl J Med.* 2008;358:111-124). Current Surviving Sepsis Guidelines call for the use of corticosteroids only in cases of septic shock where the blood pressure is not responsive to fluid administration or vasopressor therapy (*Crit Care Med.* 2013;41:580-637).

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. Diagnosis. A **rapid ACTH stimulation test** is used to test for adrenal insufficiency. **Cosyntropin** (i.e., synthetic ACTH, 250 µg) is administered intravenously, and plasma cortisol levels are measured on completion of the administration and then 30 and 60 minutes later. Normal peak cortisol response should exceed 20 µg/dL.

3. Treatment of adrenal crisis must be immediate and 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 of dexamethasone. Thereafter, 50 mg of hydrocortisone is administered intravenously every 8 hours and is tapered to standard replacement doses as the patient's condition stabilizes. Mineralocorticoid replacement is not required until intravenous fluids are discontinued and oral intake resumes.

4. Prevention. Patients who have known adrenal insufficiency or have received supraphysiologic 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 first postoperative 24 hours.

HEREDITARY ENDOCRINE TUMOR SYNDROMES (Table 39-3)

I. MULTIPLE ENDOCRINE NEOPLASIA TYPE I (MEN-1)

A. 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.* 1997;276:404-407). Genetic testing is available. Screening of affected family members should begin in their early teens, including yearly determinations of plasma calcium, glucose, gastrin, fasting insulin, vasoactive intestinal polypeptide (VIP), pancreatic polypeptide, prolactin, growth hormone, and β-human gonadotropin hormone levels.

1. Hyperparathyroidism. Because hyperparathyroidism is frequently the first detectable abnormality in patients with MEN-1, yearly calcium screening of asymptomatic kindred members is recommended. Patients with hyperparathyroidism and MEN-1 usually have generalized (fourgland) parathyroid enlargement. Surgery should consist of 3.5-gland parathyroidectomy or a total parathyroidectomy with autotransplantation of parathyroid tissue to the sternocleidomastoid

muscle or forearm. This method achieves cure in more than 90% of cases and results in hypoparathyroidism in less than 5%. Graft-dependent recurrent hyperparathyroidism, however, is seen in up to 50% of cases, and is managed by debulking of the autografted material (*Ann Surg.* 1980;192:451).

2. Pituitary tumors occur in up to 40% of MEN-1 patients and most commonly are benign prolactin-producing adenomas. Growth hormone-producing, ACTH-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.

TABLE 39-3 Hereditary Endocrine Tumor Syndromes

	MEN-1	MEN-2A	MEN-2B	FMTC
Gene mutation	MENIN	Ret	Ret	Ret
Endocrinopathy	HPT Pituitary Panc	MTC Pheo HPT	MTC Pheo	MTC

HPT, hyperparathyroidism; Panc, pancreatic islet cell tumor; MTC, medullary thyroid carcinoma; Pheo, pheochromocytoma.

3. Pancreatic islet cell tumors pose the most difficult clinical challenge and account for most of the morbidity and mortality of the syndrome. **Gastrinomas (i.e., ZollingerÑEllison Syndrome, ZES**) are most common, but VIP-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.

II. MULTIPLE ENDOCRINE NEOPLASIA TYPE 2 (MEN-2)

A. MEN2 is characterized by medullary thyroid carcinoma (MTC) and includes **MEN-2A, MEN-2B, and familial MTC (FMTC).** These autosomal-dominant syndromes are caused by gain-of-function mutations in the *RET* **proto-oncogene,** which encodes a transmembrane tyrosine kinase receptor. Mutations in *RET* lead to constitutive activation (tyrosine phosphorylation) of the RET protein, which drives tumorigenesis. Genetic testing should be performed on all suspected individuals. Because MTC occurs universally in all MEN-2 variants, thyroidectomy is indicated for all RET-mutation carriers. **Calcitonin** serves as a tumor marker for MTC and can be used to guide timing of thyroidectomy as well as postoperative monitoring for disease recurrence. When possible, prophylactic thyroidectomy should be performed prior to the presence of biochemical evidence of disease in order to reduce the risk of spread outside the thyroid that may lead to disease persistence or recurrence. Current guidelines call for prophylactic thyroidectomy in the first year of life for MEN-2B and/or RET codon 883, 918, or 922 mutation carriers; thyroidectomy before age 5 years in MEN-2A codon 611, 618, 620, or 634 mutation carriers; and thyroidectomy between ages 5 and 10 in MEN-2A codon 609, 768, 790, 791, 804, and 891 mutation carriers (*J Clin Endocrinol Metab.* 2001;86:5658-5671). A rising calcitonin level should prompt earlier intervention.

1. MEN-2A. All patients with MEN-2A will develop MTC, but the course of MTC is variable and can be predicted based upon codon mutation. Mutations in codon 611, 618, 620, 634 expose patients to an increasing, cumulative age-related risk of lymph node metastasis, starting from the mid-teens and reaching a greater than 40% cumulative risk by the age of 20. MEN-2A with mutations in codons 768, 790, 804, or 891 is less aggressive and often presents with MTC in the second or third decade of life. The penetrance of other features of the syndrome is variable. Pheochromocytomas arise in approximately 40% to 50% of patients, and hyperplasia of the parathyroid glands arises in approximately 25% to 35%. Patients with MEN-2A also develop gastrointestinal manifestations, including abdominal pain, distention, and

constipation as well as Hirschsprung disease (*Ann Surg.* 2002;235:648). On genetic analysis, patients with MEN-2A and Hirschsprung disease (MEN-2A-HD) share common mutations in either codon 609, 618, or 620 of exon 10 of the *RET* proto-oncogene. MTC generally occurs earlier than pheochromocytoma or hyperparathyroidism. Nonetheless, biochemical testing to exclude pheochromocytoma is mandatory in all MEN-2 and MTC patients prior to any elective surgical operation. Pheochromocytoma in children as young as age 8 has been reported (*J Surg Oncol.* 2013;108:203-206).

2. MEN-2B. MTC develops in all patients with MEN-2B and is particularly aggressive. MTC may be present at birth in these patients and invasive disease with lymph node metastasis occurs at an early age. Pheochromocytoma penetrance is variable. Patients also develop ganglioneuromatosis and a characteristic physical appearance, with hypergnathism of the midface, marfanoid body habitus, and multiple mucosal neuromas. MEN-2B patients may demonstrate multiple gastrointestinal symptoms and megacolon.

3. FMTC is characterized only by the hereditary development of MTC without other endocrinopathies. MTC is generally more indolent in these patients than in those with MEN-2A or -2B.

CHAPTER 39: DISEASES OF THE ADRENAL AND PITUITARY GLAND AND HEREDITARY ENDOCRINE SYNDROMES

Multiple Choice Questions

1. A 6-month-old female whose father has multiple endocrine neoplasia (MEN)-2B has tested positive for the RET proto-oncogene mutation. Calcitonin levels are undetectable. The patient should:

a. Have calcitonin levels closely monitored and undergo total thyroidectomy when calcitonin levels are greater than 20.

b. Undergo total thyroidectomy at age 5.

c. Undergo total thyroidectomy within the next several months.

d. Wait to undergo total thyroidectomy until over 1 year old to prevent permanent damage to the parathyroid glands.

e. Undergo total thyroidectomy at age 20.

View Answer

2. Which of the following is not consistent with the finding of a cortisol level less than 20 μg/dL on corticotropin stimulation testing?

a. Hypokalemia

b. Hypoglycemia

c. Hypotension

d. Fever

e. Emesis

View Answer

3. A 32-year-old male is incidentally found to have a 4 cm left adrenal mass on a CT scan performed following a motor vehicle accident. He is otherwise healthy. He undergoes a dexamethasone suppression test with normal suppression of cortisol levels. Plasma metanephrines and normetanephrines are within normal limits. What is the next step in the patient's management?

a. Undergo plasma aldosterone level testing.

b. Repeat CT scan in 6 months.

c. Perform laparoscopic left adrenalectomy.

d. Undergo MRI to further evaluate mass.

e. Undergo Ret proto-oncogene testing to rule out multiple endocrine neoplasia type 2.

View Answer

4. A 50-year-old male with persistent hypertension despite antihypertensive combination therapy with a beta blocker, calcium channel blocker, and ACE inhibitor presents with hypokalemia and elevations in plasma aldosterone levels. CT and MRI imaging demonstrate a 1-cm left-sided adrenal mass and a 6-cm right-sided adrenal mass, both benign appearing. Dexamethasone suppression testing and plasma metanephrines and normetanephrines are normal. The next step in management is:

a. Right adrenalectomy

b. Medical management alone with addition of spironolactone

c. Repeat imaging in 6 months

d. Bilateral adrenal vein sampling

e. Bilateral adrenalectomy

View Answer

5. A 65-year-old female with hypertension and coronary artery disease status post placement of drug eluting stent 3 weeks ago presents with an 8-cm right adrenal mass with macroscopic fat found incidentally on CT. Biochemical evaluation reveals a normal dexamethasone suppression test, normal plasma metanephrines and normetanephrines, and normal metabolic panel. Appropriate management of this patient includes:

a. Repeat CT imaging at 6 months

b. MRI

c. Immediate right adrenalectomy with patient on Plavix

d. Right adrenalectomy 1 year after coronary stent placement with patient off of Plavix

e. No further imaging or intervention

View Answer

40
Thyroid and Parathyroid Glands

Jennifer Yu

William E. Gillanders

THYROID

I. EMBRYOLOGY, ANATOMY, AND PHYSIOLOGY

A. Embryology. The thyroid gland develops from the endoderm of the primitive foregut and arises in the ventral pharynx near the base of the tongue, which ultimately becomes the foramen cecum. The thyroid then descends in the midline of the neck anterior to the hyoid bone and laryngeal cartilages. Congenital anomalies such as ectopic thyroid tissue or thyroglossal duct cysts are directly related to variations in this process. The parafollicular cells, or C cells, are derived from the neural crest and migrate to the thyroid to produce calcitonin.

B. Anatomy. The adult thyroid is a bilobar structure connected by an isthmus that lies anterior to the trachea. The thyroid gland's blood supply arises mainly from the superior and inferior thyroid arteries, which are branches of the external carotid artery and thyrocervical trunk, respectively. Important structures in close proximity to the thyroid include the external branch of the superior laryngeal nerve (SLN, located near the superior pole of the thyroid) and the recurrent laryngeal nerve (RLN, located just anterior or posterior to the inferior thyroid artery in the tracheoesophageal groove). Careful dissection around the inferior thyroid artery is necessary to avoid injury to the RLN.

C. Physiology. Thyrotropin-releasing hormone (TRH) secreted by the hypothalamus stimulates thyroid-stimulating hormone (TSH) secretion by the anterior pituitary gland. TSH then stimulates thyroid hormone secretion by the thyroid gland. The process of thyroid hormone synthesis begins when dietary iodide is ingested, actively transported into the thyroid, and oxidized by thyroid peroxidase (TPO) into iodine. Iodination of tyrosine residues in thyroglobulin creates monoiodotyrosine and diiodotyrosine. Coupling reactions of monoiodotyrosine and diiodotyrosine result in the formation of triiodothyronine (T_3) and thyroxine (T_4), both of which are bound to thyroglobulin and stored in thyroid follicles. When released into plasma, more than 99% of T_3 and T_4 is bound to carrier proteins, including thyroxine-binding globulin (TBG), thyroxine-binding prealbumin, and albumin. Only the unbound or ÒfreeÓ hormones are active (i.e., available to tissues), and T_4 is converted to T_3 by deiodinases in the peripheral tissues. Compared to T_3, T_4 has a 20-fold higher circulating concentration but is

3 to 5 times less potent. The half-life of T_4 (7 days) is significantly longer than the half-life of T_3 (1 day).

Assessment of thyroid function requires biochemical evaluation and interpretation in the context of clinical findings. Measurement of TSH (0.3 to 4.12 mIU/L) is the most useful biochemical test in the assessment of thyroid function. In most patients without hypothalamic or pituitary disease (rare), TSH and free T_4 (FT_4) vary inversely around a euthyroid state: Increased TSH and low FT_4 signify hypothyroidism, while suppressed TSH and high FT_4 suggest hyperthyroidism. Assessment of FT_4 (0.8 to 1.8 ng/dL) supports identified abnormalities in TSH and provides an index of severity of thyroid dysfunction. Total T_4 (5 to 12 µg/dL) is affected by changes in hormone production or binding and does not directly reflect the small FT_4 fraction.

Measurement of total T_3 (80 to 200 ng/dL) is unreliable as a test for hypothyroidism but is useful in the patient with suspected hyperthyroidism, suppressed TSH, and normal FT_4 in order to rule out T_3 thyrotoxicosis. A newer assay for free T_3 (2 to 3.5 pg/dL) may offer additional benefit in directly measuring the fraction of unbound active T_3. The American Thyroid Association (ATA) has published evidence-based guidelines on thyroid disease evaluation and recommends serum TSH as an initial screening test for thyroid dysfunction (*Thyroid.* 2012; 22(12):1200-1235).

II. BENIGN THYROID DISEASE

A. Thyroid Nodule. A solitary thyroid nodule is defined as a discrete lesion that is distinct from the surrounding thyroid parenchyma.

1. Epidemiology. Thyroid nodules are common: 4% to 7% of all adults have palpable thyroid nodules, with an even higher prevalence on ultrasound. Although commonly benign, up to 15% of thyroid nodules may harbor malignancy depending on patient factors such as age, sex, and history of radiation exposure; thus, diagnostic testing is recommended to separate patients with malignancy from the larger population with benign nodules (*N Engl J Med.* 2004;351(17):1764-1771). Revised management guidelines from the ATA Guidelines Task Force for the evaluation and treatment of thyroid nodules are expected in 2015 (http://www.thyroid.org/thyroid-guidelines/).

2. Clinical evaluation. A thorough history and physical examination are critical in the workup of a thyroid nodule. Risk of malignancy is higher at the extremes of age (especially in older men) and in those with a personal history of ionizing radiation exposure, a positive family history of thyroid malignancy, familial polyposis, or other endocrine diseases. Rapid nodule growth, pain, compressive symptoms, or hoarseness increase the likelihood of malignancy but are nonspecific. Physical findings of a solitary nodule with firm or irregular texture, fixation to surrounding structures, or associated enlarged cervical lymph nodes may also suggest malignancy. Generally,

only nodules >1 cm should undergo further investigation as these have a greater potential of being

clinically significant cancers. Figure 40-1 illustrates the algorithm for workup of a newly diagnosed thyroid nodule.

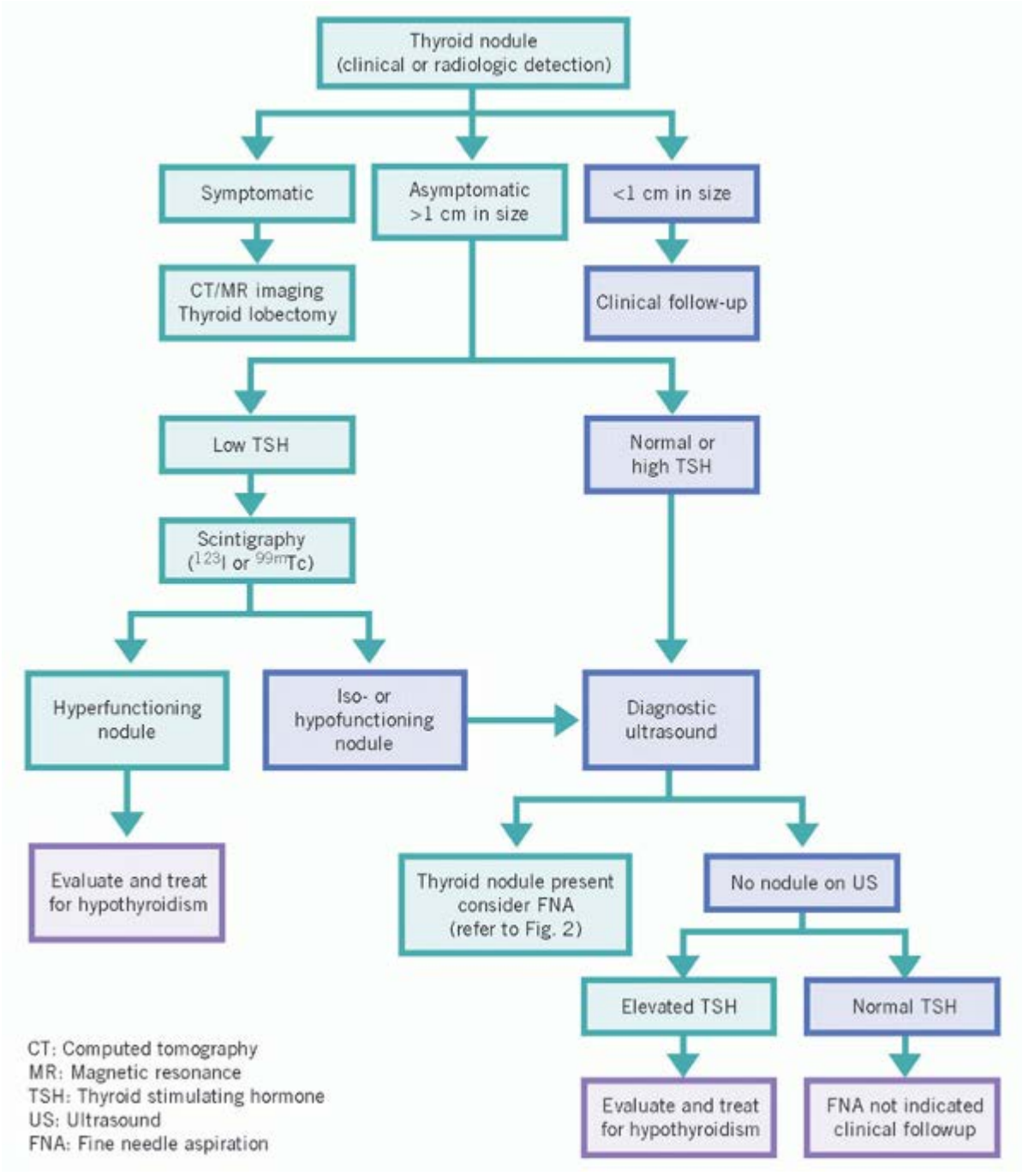


Figure 40-1 Thyroid nodule initial evaluation algorithm.

3. Biochemical evaluation. For patients with nodules >1 cm in size, a serum TSH level will

determine the subsequent diagnostic pathway. In patients with suppressed TSH, an Iofetamine (^{123}I) radionuclide scan should be performed. Malignancy is uncommon in hyperfunctioning nodules, that is, those that take up more tracer than surrounding normal thyroid, and no further workup is necessary in these lesions. However, patients with hypofunctioning nodules or with normal or high serum TSH should undergo diagnostic ultrasound.

4. Diagnostic imaging

a. Thyroid ultrasound. All patients with known or suspected thyroid nodules should undergo diagnostic ultrasonography, which can accurately determine gland volume and the number, character, and size of thyroid nodules. Ultrasound can also help guide fine needle aspiration (FNA) biopsy and cyst aspiration (*Radiol Clin North Am.* 2014;52(6):1283-1294). Purely cystic thyroid nodules have very low malignant potential, whereas nodules that are solid or hypoechoic and/or have an incomplete peripheral halo, irregular margins, and/or microcalcifications are more suspicious for malignancy. Importantly, FNA is not indicated for most nodules unless size and sonographic pattern criteria are satisfied. The ATA recommends FNA for nodules >1 cm with intermediate or high-suspicion sonographic patterns, nodules >1.5 cm with low-suspicion sonographic patterns, and nodules >2 cm with very low-suspicion sonographic patterns. Cervical lymph nodes should be evaluated concurrently, and FNA of suspicious lymph nodes and any associated thyroid nodule should be performed regardless of nodule size. For nodules that do not meet FNA criteria, repeat ultrasound is indicated in 6 to 12 months for high-suspicion sonographic patterns or 12 to 24 months for low-intermediate suspicion sonographic patterns. Table 40-1 summarizes common ultrasound characteristics of thyroid nodules and recommendations for management of each risk category.

b. Thyroid scan. Thyroid scans cannot differentiate benign from malignant lesions and are generally not recommended in the initial workup of a thyroid nodule. Following ultrasound, technetium (^{99m}Tc) pertechnetate or ^{123}I thyroid scanning can help distinguish solitary functioning nodules from multinodular goiter or Graves disease. Hypofunctioning areas (cyst, neoplasm, or suppressed tissue adjacent to autonomous nodules) are Òcold,Ó whereas areas of increased uptake are Òhot.Ó Cold nodules are more likely to be malignant than hot nodules, but this is an unreliable measure, as most cold nodules are benign. Whole body scanning at 4 to 24 hours after administration of ^{123}I or iodine-131 (^{131}I) is useful for identifying metastatic differentiated thyroid tumors or predicting a response to ^{131}I radioablation.

c. Other imaging studies. CT and MR imaging of the thyroid are generally reserved for assessing substernal or retrosternal masses, for staging known malignancy, or for evaluating local invasion that may change the operative approach (*Radiol Clin North Am.* 2015;53(1):145Ñ161). Routine use of preoperative fluorodeoxyglucose-positron emission tomography (FDG-PET) scan is not recommended.

5. Fine needle aspiration biopsy. FNA is an accurate and cost-effective diagnostic modality for evaluating thyroid nodules, can be performed at the bedside or under ultrasound guidance, and is safe to utilize during pregnancy. As shown in Table 40-2, the Bethesda System for Reporting Thyroid Cytopathology divides FNA results into six categories (*Thyroid.* 2009;19(11):1159-1166), and Figure 40-2 delineates the appropriate subsequent management for each type:

TABLE 40-1 Ultrasound Characteristics in Thyroid Nodule Evaluation

Ultrasound Category	Characteristics	Risk for Malignancy (%)	Recommendation
Benign	Purely cystic nodules (no Solid component)	<1	Clinical observation Aspiration if symptomatic
Very Low Suspicion	Spongiform or partially cystic nodules[a]	<3	³2 cm: FNA <2 cm: Clinical observation
Low Suspicion	Isoechoic or hyperechoic solid nodules[a] Partially cystic nodules with eccentric uniformly solid areas[a]	5-10	³1.5 cm: FNA <1.5 cm: Repeat US (12-24 mo)
Intermediate Suspicion	Solid hypoechoic nodules with smooth regular margins[a]	10-20	³1 cm: FNA <1 cm: Repeat US (12-24 mo)
High Suspicion	Solid hypoechoic nodules or partially cystic nodules with solid hypoechoic component	70-90	³1 cm: FNA <1 cm: (with no evidence of local invasion or distant

	AND ³1 risk feature: 1. Irregular margins (infiltrative, microlobulated) 2. Microcalcifications 3. Taller than wide shape 4. Disrupted rim calcifications with extrusion into soft tissue 5. Extrathyroidal extension	disease): FNA vs. repeat US (6-12 mo)

[a] Without microcalcifications, irregular margins, extrathyroidal extension, or taller than wide shape.
US, ultrasound; FNA, fine needle aspiration.

TABLE 40-2 The Bethesda System for Thyroid Cytopathology

Diagnostic Category	Risk for Malignancy (%)	Recommendation
Nondiagnostic or Unsatisfactory	1-4	Repeat FNA with ultrasound guidance
Benign	0-3	Clinical followup, repeat US (6-18 mo)
Atypia of Undetermined Significance Follicular Lesion of Undetermined Significance	5-15	Repeat FNA

Follicular Neoplasm Suspicious for Follicular Neoplasm	15-30	Thyroid lobectomy
Suspicious for Malignancy	60-75	Near-total thyroidectomy or thyroid lobectomy
Malignant	97-99	Near-total thyroidectomy

US, ultrasound; FNA, fine needle aspiration.
Adapted from Cibas ES, Ali SZ. The Bethesda System for Reporting Thyroid Cytopathology. *Thyroid* 2009;19(11):1159-1165.

a. Nondiagnostic cytology. Nondiagnostic cytology fails to meet the criteria for an adequate specimen, and repeat FNA should be performed under ultrasound guidance. Nodules that continue to yield nondiagnostic specimens require either close observation or surgical excision, particularly for lesions with high-suspicion sonographic patterns, growth during surveillance, or clinical risk factors for malignancy.

b. Benign cytology. Benign cytology has a low risk of malignancy (0% to 3%) and requires no further diagnostic testing. Patients can be followed clinically with a repeat ultrasound at 6 to 18 months.

c. Follicular lesion of undetermined significance. Cytology noting Òfollicular lesion of undetermined significanceÓ or Òatypia of undetermined significanceÓ carries a 5% to 10% risk of malignancy and repeat FNA after 3 months is recommended (*CytoJournal.* 2008;5:6). If repeat FNA also shows inconclusive cytology, either surgical excision or surveillance can be pursued based on risk factors, imaging, and patient preference. Evaluation of new molecular markers such as BRAF, RAS, RET/PTC, Pax8-PPARγ, or galectin-3 can be considered to improve diagnostic accuracy, though long-term outcome data is not yet available. Molecular assays designed to detect common somatic mutations are now commercially available to assist with clinical decision making, though testing should only be performed in CLIA/CAP-certified laboratories for quality assurance (*J Clin Endocrinol Metab.* 2011;96(11):3390-3397).

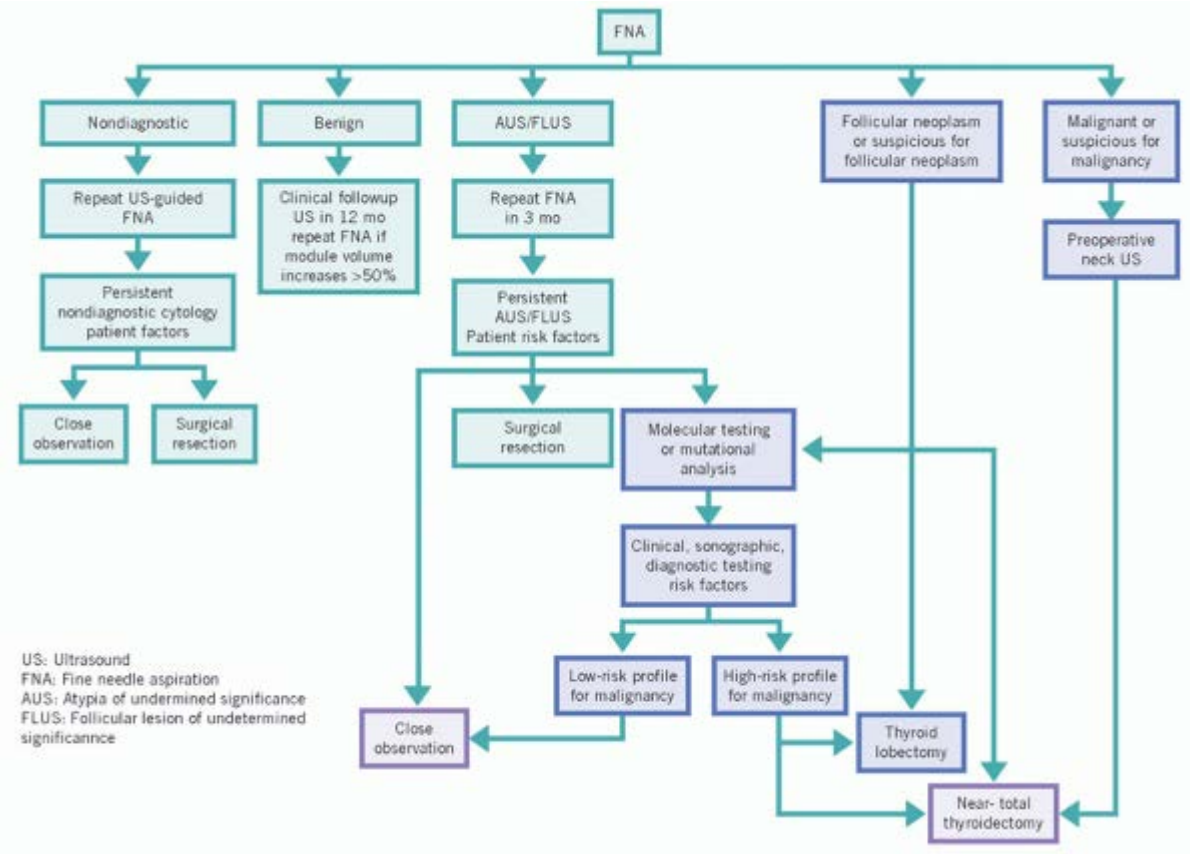


Figure 40-2 Thyroid nodule management following fine needle aspiration biopsy.

d. Suspicious for follicular neoplasm. Lesions reported as Òeither follicular or HŸrthle cell neoplasmsÓ (also referred to as ÒindeterminateÓ) or Òsuspicious for follicular neoplasmÓ carry a 20% to 30% risk of malignancy and are typically treated with surgical excision (thyroid lobectomy), though molecular testing may be used in this setting for additional diagnostic information before proceeding to surgery (*Diagn Cytopathol.* 2008;36(6):425-437). ^{123}I scintigraphy can also be considered if not yet performed. If final pathology reveals thyroid cancer, completion thyroidectomy should be considered depending on clinical features and patient factors (*Thyroid.* 2009;19(11):1167-1214).

e. Suspicious for malignancy or malignant. ÒSuspicious for malignancyÓ and ÒmalignantÓ designations carry risks of malignancy greater than 60% and should be treated with near-total thyroidectomy. Molecular analysis can also be used in these patients, but even a negative result may still carry a greater than 25% risk of cancer for lesions in these cytologic categories.

6. Operative strategies. Surgical intervention is indicated for nodules with malignant or indeterminate cytology and should also be considered for patients with symptomatic or cosmetically bothersome nodules. Nodules in children, in patients with history of radiation exposure, or in those with a family history of thyroid cancer are associated with an increased

suspicion for malignancy. Operative approach should be based upon imaging review and FNA results and frequently involves lobectomy versus near-total or total thyroidectomy.

B. Toxic Adenoma and Multinodular Goiter. Multinodular goiter is a common condition, and each individual nodule needs to be evaluated for possible biopsy. Indications for surgery include suspicious cytology as detailed above, nodule size, and compressive symptoms. Toxic adenoma and toxic multinodular goiter (Plummer disease) are most often secondary to autonomous function in one or several thyroid nodules which produce thyroid hormone independent of TSH stimulation. A radioactive ^{131}I scan is diagnostic with one or more ÒhotÓ areas and suppression of the rest of the gland. Either RAI ablation or surgical excision (lobectomy or subtotal thyroidectomy) can be implemented, though surgery is indicated for multiple large nodules, obstructive symptoms, thyrotoxicosis, or failure of RAI therapy.

C. Graves Disease

1. Epidemiology. Autoimmune diffuse toxic goiter (Graves disease) is the most common cause (60% to 80%) of hyperthyroidism, presenting up

to seven times more frequently in women and usually in the second to fourth decade.

2. Pathophysiology. Hyperthyroidism results from excess circulating levels of thyroid hormones and a resultant increase in catabolism and sympathetic activity. In Graves disease, this is due to constitutive activation of the TSH receptor by stimulating immunoglobulins thereby leading to increased hormone production. Diagnosis is made by history and physical examination, depressed TSH levels, and detection of anti-TSH-R antibodies, which are present in more than 90% of patients.

3. Clinical manifestations. Symptoms of hyperthyroidism include weight loss despite normal or increased appetite, heat intolerance, excessive perspiration, anxiety, irritability, palpitations, fatigue, and oligomenorrhea. Signs include goiter, sinus tachycardia or atrial fibrillation, tremor, hyperreflexia, fine or thinning hair, eyelid lag or retraction, thyroid bruit, muscle wasting, and proximal muscle weakness. Unique features of Graves disease include infiltrative ophthalmopathy and pretibial myxedema.

4. Medical therapy. Thioamide drugs, such as propylthiouracil (PTU) or methimazole, are used for antithyroid drug therapy and mainly prevent synthesis of thyroid hormones. However, long-term remission is achieved in less than 20% to 30% of patients. Methimazole is commonly recommended to prepare thyrotoxic patients for surgery or ablative therapy.

5. Radioactive iodine ablation therapy. RAI ablation is the treatment of choice for most patients with Graves disease. Given orally, the initial dose is approximately 75% effective after 8 to 12 weeks. A second dose at the same or higher dose is given in the 25% of patients who have persistent thyrotoxicosis 6 to 12 months later. Cure rates approach 90% by 1 year, and hypothyroidism will eventually develop in the majority of treated patients. Contraindications to

radiotherapy include pregnancy or lactation, newborns, patient refusal to comply with radiation safety guidelines, suspicion for or known thyroid cancer, and low RAI uptake (<20%).

6. Surgery. Thyroidectomy for Graves disease may be indicated for patients who refuse or have a contraindication to RAI or who have an obstructive goiter. Near-total or total thyroidectomy is recommended due to high recurrence rates in patients treated with subtotal thyroidectomy (8% to 15%) and a relatively high risk of occult malignancy (5% to 20%). Patients undergoing thyroidectomy as primary therapy should be treated at a high-volume thyroid surgery center with preoperative antithyroid drug therapy to render them euthyroid. Patients with recurrent hyperthyroidism after thyroidectomy should be considered for RAI treatment due to higher reoperative complication rates.

D. Nontoxic Goiter. Multinodular nonfunctioning goiter, typically secondary to iodine deficiency, can result in tracheal compression due to size or retrosternal extension. Subtotal or total thyroidectomy can be performed for compressive symptoms, suspicion of malignancy, or cosmetic distress.

E. Thyroiditis. Thyroiditis encompasses several autoimmune and inflammatory disorders characterized by inflammatory cell infiltration and subsequent fibrosis of the gland.

1. Hashimoto thyroiditis. Hashimoto thyroiditis is a chronic autoimmune disorder involving destructive lymphocytic infiltration of the thyroid and is the most common cause of hypothyroidism in the United States. Up to 10 times more common in women, more than 90% of affected patients have circulating anti-TPO and antithyroglobulin antibodies. Hypothyroidism generally has an insidious onset, and patients may initially be euthyroid and asymptomatic. Clinical features of hypothyroidism include cold intolerance, weight gain, constipation, edema, dry skin, weakness, somnolence, and menorrhagia. Diagnosis is made by an increased serum TSH level and decreased FT_4 levels. Thyroid hormone is the preferred replacement therapy for hypothyroid patients, and thyroidectomy is indicated for persistent compressive symptoms, a dominant nodule suspicious for malignancy, or cosmetic preference.

2. Other causes of thyroiditis. Other rare causes of thyroiditis include acute suppurative thyroiditis, subacute (de Quervain) thyroiditis, and Riedel thyroiditis. Acute suppurative thyroiditis is caused by pyogenic Streptococcus or Staphylococcus infection and requires antibiotic therapy with possible surgical drainage of abscesses. Subacute thyroiditis occurs more commonly in young women, often after a viral upper respiratory tract infection, and almost always remits spontaneously within a few weeks. Symptoms of fatigue, weakness, or jaw/ear pain from thyroid enlargement may be treated with nonsteroidal anti-inflammatory drugs or steroids. Finally, Riedel thyroiditis is an idiopathic progressive inflammatory condition of the entire thyroid gland, strap muscles, and other neck structures. Surgical excision may be required to exclude malignancy or relieve compressive symptoms.

III. THYROID CANCER.

Differentiated thyroid cancers (papillary, follicular, and HŸrthle cells) are relatively indolent cancers arising from follicular epithelial cells and are the most common types of thyroid malignancy. Overall, these cancers are rare in children, more common in females (2.5:1), and have a peak incidence in the fourth decade. Exposure to ionizing radiation is the best documented environmental factor, and prognosis depends on the patient's age as well as the extent and histologic subtype of the disease (85% to 90% of patients fall into a low-risk category with favorable prognosis). As of January 2015, the ATA Guidelines Task Force has issued draft versions of the new management guidelines for the workup and treatment of differentiated thyroid cancers (http://www.thyroid.org/thyroid-guidelines/). Table 40-3 reviews the major categories of thyroid malignancies with clinical characteristics and management recommendations for each type.

A. Papillary Thyroid Cancer

1. Epidemiology. Papillary thyroid carcinoma (PTC) represents 85% of thyroid carcinomas, is often multifocal, and frequently metastasizes to cervical lymph nodes. Occult, clinically insignificant foci of microscopic PTC are found in 5% to 30% of autopsies or in thyroidectomy specimens for benign diseases. Features deemed to be high risk and associated with worse prognosis include male gender, primary tumor size >4 cm, gross local invasion and extrathyroidal extension, age >45 years, certain histologic subtypes (specifically, the tall cell variant), lymphovascular invasion, or known metastatic disease (*Cancer Control.* 2011;18(2):96-103).

TABLE 40-3 Categories of Thyroid Malignancies

Category	Percent of All Thyroid Malignancies	Clinical Features	Management
Papillary carcinoma	70-80	Indolent, often multifocal Frequently metastatic to cervical lymph nodes	Surgery ± RAI ablation therapy ± thyroid hormone suppression therapy Followup: Serum thyroglobulin, neck ultrasound

Follicular carcinoma	10-15	More common in women, age ³30 yrs Hematogenous spread to bone, lung, liver	
Medullary carcinoma	5-10	Sporadic (75%) vs. familial (25%) Familial forms: MEN syndromes type 2A and 2B Frequent early nodal metastasis Elevated calcitonin and CEA levels	Genetic testing (RET proto-oncogene) for all patients Screening for pheochromocytoma Total thyroidectomy + lymph node dissection
Anaplastic carcinoma	1-2	Frequently presents at advanced stage May be highly symptomatic with local invasion Nearly 100% diseasespecific mortality	Palliative surgery Chemotherapy ± external beam radiation
Lymphoma	2-8	Often non-Hodgkin type Associated with Hashimoto thyroiditis	External beam radiation vs. chemotherapy

MEN, multiple endocrine neoplasia; RAI, radioactive iodine.

2. Staging. Despite being an indolent cancer, cervical lymph node metastases are found in 20% to 50% of patients at the time of diagnosis. Therefore, it is reasonable to consider preoperative neck ultrasound with FNA of suspicious nodes >8 to 10 mm prior to surgical intervention. Alternatively, ultrasound on the day of surgery can be used to mark suspicious nodes which are sent intraoperatively for frozen section, followed by compartmental resection if malignancy is

detected.

3. Operative strategies

a. Total thyroidectomy. Total thyroidectomy is recommended in several conditions: Tumors greater than 4 cm, presence of bilateral nodules, regional or metastatic disease, personal history of head/neck radiation exposure, or first-degree relatives with PTC.

b. Thyroid lobectomy. Thyroid lobectomy is appropriate for low-risk patients with small (<1 cm), intrathyroidal, unifocal tumors without evidence of regional or metastatic disease. For similar patients with tumor size 1 to 4 cm, either thyroid lobectomy or total thyroidectomy can be considered.

c. Lymph node dissection. Lymph node dissection of the central neck compartment (level VI) and/or the ipsilateral lateral neck compartment (levels II, III, IV) should be performed in all patients with biopsy-proven nodal disease. Furthermore, prophylactic central neck dissection should be considered for high-risk patients (e.g., large tumors, bilateral tumors, radiation exposure) even in the setting of clinically uninvolved lymph nodes, since up to 90% of patients with PTC will have micrometastases at the time of surgery.

4. Complications

a. Hemorrhage. Hemorrhage is a rare but serious complication (0.3% to 1%) of thyroidectomy that usually occurs within 6 hours of surgery. Management typically requires airway control by endotracheal intubation but may necessitate immediate opening of the incision and/or an emergent surgical airway before return to the operating room for wound irrigation and control of bleeding.

b. Hypocalcemia. Transient hypocalcemia may occur 24 to 48 hours after total or near-total thyroidectomy. We start patients routinely on oral calcium carbonate (1 gram TID) for 2 weeks after total thyroidectomy. If symptoms occur, additional oral calcium and calcitriol (0.25 µg/day) can be implemented, or if severe, intravenous replacement is achieved using six ampules of 10% calcium gluconate (93 mg elemental calcium in 10 mL) mixed in 500 mL of 5% dextrose in water (D5W) at a goal infusion of elemental calcium at 0.5 to 1.5 mg/kg/hour. Permanent hypoparathyroidism

is uncommon after total thyroidectomy. Normal parathyroid tissue removed or devascularized during surgery should be minced into 1 mm fragments and autotransplanted into individual muscle pockets in the sternocleidomastoid muscle to minimize risk of postoperative hypoparathyroidism (*Ann Surg.* 1996;223(5):472-478).

c. Recurrent laryngeal nerve injury. RLN injury is a significant complication of thyroidectomy that should rarely occur (<1%). Unilateral RLN injury causes hoarseness, which often presents immediately but may appear days to weeks after surgery, and bilateral injury compromises the airway, usually manifesting immediately postoperatively and potentially requiring tracheostomy.

Reoperative neck surgery or thyroidectomy for extensive goiter; Graves disease; or fixed, locally invasive cancers carries additional risk of RLN injury. Intentional (as with locally invasive cancer) or inadvertent RLN transection can be repaired primarily or with a nerve graft, and primary repair has been associated with improved phonation in small studies. Transient RLN injury can also occur following thyroidectomy but usually resolves over a period of 1 to 6 weeks. If a permanent injury develops, a cord medialization procedure should be considered.

5. Radioactive iodine ablation. RAI ablation therapy is recommended for all patients with primary tumor size >4 cm, gross local invasion, and for selected patients with tumor size 1 to 4 cm and high-risk features such as age >45 years, certain histologic subtypes, extrathyroidal extension, lymphovascular invasion, or known metastatic disease. Ablation is performed with 30 to 150 mCi of ^{131}I approximately 2 to 4 weeks after total thyroidectomy once the patient is hypothyroid (i.e., TSH >30 mU/mL on no replacement of T_4) and may be repeated at 6 to 12 months if residual disease is detected on followup surveillance.

6. Thyroid hormone suppression therapy. Thyroid hormone suppression therapy should be considered after total or near-total thyroidectomy or RAI ablation and functions to suppress TSH levels by negative feedback mechanisms. Thyroid hormone suppression therapy with initial TSH suppression below 0.1 mU/L and lifelong TSH suppression at or below the lower limit of normal (0.1 to 0.5 mU/L) decreases recurrences and may improve survival. Oral levothyroxine is started at a dose of 1.4 µg/kg/day. Adequacy of thyroid hormone replacement is assessed by measuring TSH and FT_4 6 to 12 weeks after initiating therapy. Dose adjustments should be conservative (12.5 to 25 µg increments) and not more frequent than monthly in the absence of symptoms.

7. Followup. Long-term followup for differentiated thyroid cancer requires monitoring of serum thyroglobulin levels every 6 to 12 months and periodic neck ultrasound in patients who underwent less than total thyroidectomy or did not undergo RAI ablation. For low- and intermediate-risk patients undergoing RAI ablation, if posttherapy whole-body RAI scans (WBS) do not reveal uptake outside the thyroid bed, subsequent WBS are not indicated in the setting of undetectable thyroglobulin levels and negative cervical ultrasound. Diagnostic WBS should be used in patients at increased risk of persistent disease.

B. Follicular Thyroid Cancer. Follicular thyroid carcinoma (FTC, 10% of thyroid carcinomas) is rare before age 30 years, is three times more common in women than in men, and has a slightly worse prognosis than PTC. Unlike PTC, FTC tends to spread hematogenously to bone, lung, or liver. Small (<1 cm), unilateral lesions with limited invasion of the tumor capsule may be treated with thyroid lobectomy, whereas tumors >1 cm, multicentric tumors, and tumors with more extensive capsular and vascular invasion or distant metastases are treated with total thyroidectomy. RAI ablation is indicated after total thyroidectomy, followed by lifelong TSH suppression with thyroid hormone suppression therapy.

C. Medullary Thyroid Cancer

1. Epidemiology, pathophysiology, and clinical manifestations. Medullary thyroid carcinoma (MTC) arises from the thyroid parafollicular C cells and accounts for 5% to 10% of all thyroid cancers in the United States. MTC may occur sporadically (75%) or may be associated with a familial syndrome, either alone or as a component of multiple endocrine neoplasia (MEN) syndromes type 2A or 2B (25%). Sporadic MTC generally presents as a firm, palpable, unilateral nodule with or without involved cervical lymph nodes, whereas hereditary MTC more often develops as bilateral, multifocal tumors with diagnosis on the basis of genetic or biochemical screening. MTC spreads early to cervical lymph nodes and may metastasize to liver, lungs, or bone. All patients with MTC should undergo genetic testing for germline mutations in the RET proto-oncogene to exclude familial medullary thyroid carcinoma (FMTC) or MEN2 syndromes. Similar to other thyroid malignancies, diagnosis is made by FNA of suspicious thyroid nodules. More than 50% of patients presenting with a palpable primary tumor will already have nodal metastases and elevated basal serum calcitonin levels (>20 to 100 pg/mL). Calcitonin and CEA levels are important tumor markers for MTC and correlate strongly with extent of disease. Preoperative neck ultrasound is critical for identification of regional metastases and surgical planning, and screening for pheochromocytoma with serum or urine metanephrines and catecholamines should be considered in all patients undergoing surgery for MTC. (For more information on MEN, see Chapter 39: Diseases of the Adrenal and Pituitary Gland and Hereditary Endocrine Syndromes.)

2. Operative strategies. Total thyroidectomy alone is only indicated for MEN 2 patients who have thyroid nodules <5 mm and calcitonin levels <40 pg/mL. Otherwise, treatment of both hereditary and sporadic MTC requires at least total thyroidectomy with central neck compartment (Level VI) lymph node dissection, and additional dissection of ipsilateral lateral compartment nodes should be performed in patients with palpable primary tumors. Surgical management of residual or recurrent disease remains the standard of care based upon patient clinical factors.

3. Medical therapy. MTC does not concentrate radioactive iodine, and postoperative RAI treatment is not recommended in the absence of

concurrent differentiated thyroid cancer. Adjuvant external beam radiation and systemic chemotherapy options have not shown significant benefit. However, clinical trials using tyrosine kinase inhibitors targeting the RET receptor have demonstrated improvement in progression-free survival, and two agents (vandetanib and cabozantinib) have been approved by the FDA. The ATA Guidelines Task Force issued a newly revised statement for the management of medullary thyroid cancer in 2015 (http://www.thyroid.org/thyroid-guidelines/).

D. Anaplastic Thyroid Cancer. Undifferentiated or anaplastic thyroid carcinoma (1% to 2% of thyroid cancers) carries an extremely poor prognosis with nearly 100% disease-specific mortality. Disease usually presents as a fixed, sometimes painful goiter in patients older than 50 years, and over 90% of patients have regional or distant disease at the time of diagnosis. Invasion of local

structures can preclude resection, and local symptoms such as dysphagia, respiratory compromise, or hoarseness may occur due to RLN involvement. External beam radiation or chemotherapy may provide limited palliation.

E. Primary Thyroid Lymphoma. Primary malignant lymphoma of the thyroid is usually non-Hodgkin type and is frequently associated with Hashimoto thyroiditis. Surgical resection is usually not indicated following diagnosis, and radiation or chemotherapy regimens are effective.

PARATHYROID

I. EMBRYOLOGY, ANATOMY, AND PHYSIOLOGY

A. Embryology. The inferior and superior parathyroid glands are derived from the endoderm of the third and fourth pharyngeal pouches, respectively. The inferior parathyroids are intimately associated with the thymus, which also develops from the third pharyngeal pouch, and ectopic inferior glands can be found anywhere along the tract of descent by the thymus into the chest that becomes the thyrothymic ligament. The superior glands have a limited descent from the neck and are much less variable in position.

B. Anatomy. Typically, the inferior parathyroid glands are found inferior to the inferior thyroid artery and anterior to the RLN. The superior glands are usually found at the posterolateral aspect of the superior thyroid lobe, posterior to the RLN, and superior to the inferior thyroid artery, which is the main blood supply for all of the parathyroids. Because the embryologic path of descent of the inferior parathyroid crosses that of the superior glands, the glands can rarely be found at the same level, above or below the crossing of the inferior thyroid artery and RLN.

C. Physiology. Serum calcium levels are maintained within normal range (8.2 to 10.2 mg/dL) by the interplay of parathyroid hormone (PTH) and vitamin D. Upon stimulation, chief cells of the parathyroid glands secrete PTH that (1) stimulates calcium and phosphate release from bone, (2) increases calcium and inhibits phosphate reabsorption in the kidneys,

and (3) enhances intestinal absorption of calcium through increased renal activation of vitamin D. Vitamin D is initially absorbed through the small intestine, undergoes hydroxylation in the liver to $25(OH)D_3$, and then undergoes a second hydroxylation in the kidney under the influence of PTH to its active form, $1,25(OH)D_3$. While vitamin D stimulates bone and intestinal calcium resorption, calcitonin antagonizes the effect of PTH in the bones and kidneys.

II. BENIGN PARATHYROID DISEASE

A. Primary Hyperparathyroidism

1. Epidemiology. Primary hyperparathyroidism (HPT) has an incidence of 0.25 to 1 per 1,000 in the United States and is especially common in postmenopausal women. It most often occurs sporadically but can be inherited alone or as a component of familial endocrinopathies, including

MEN types 1 and 2A.

2. Clinical manifestations. Common clinical findings associated with HPT include nephrolithiasis, osteoporosis, hypertension, and emotional disturbances. Patients may also have subtle symptoms such as muscle weakness, polyuria, anorexia, fatigue, bone/joint pain, poor sleep, reflux, and nausea.

3. Biochemical evaluation. Diagnosis of primary HPT typically requires documentation of hypercalcemia (serum calcium >10.5 mg/dL) and an elevated PTH level. The assay of choice for PTH is the highly sensitive and specific intact PTH immunoassay, and ionized calcium is a more sensitive test of physiologically active calcium. In addition, multiple biochemical abnormalities may be present concurrently with hypercalcemia and require correction prior to surgical intervention. Metabolic acidosis and hypophosphatemia are more commonly associated with primary hyperparathyroidism due to increased urinary excretion of bicarbonate and phosphate. Patients are also more likely to present with hyperchloremia due to the increased urinary excretion of bicarbonate. Hypomagnesemia can occur in 5% to 10% of patients with primary hyperparathyroidism. Serum alkaline phosphatase levels are often elevated in patients with bone disease from hyperparathyroidism due to an increase in osteoclastic bone resorption.

4. Differential diagnosis. Hypercalcemia can be due to a variety of causes (e.g., malignancy, Paget disease, sarcoidosis, and milk-alkali syndrome), which are typically associated with low PTH levels. Familial hypocalciuric hypercalcemia (FHH) commonly presents with mild hypercalcemia and low urine calcium, with elevated PTH in 15% to 20% of patients. Caused by loss-of-function mutations in renal and parathyroid calcium-sensing receptors, FHH patients have loss of feedback inhibition of PTH secretion and inadequate clearance of calcium in the urine. FHH can be distinguished from HPT by a 24-hour measurement of urine calcium or by measuring the renal calcium/creatinine clearance ratio. A ratio less than 0.01 suggests FHH while the ratio seen in HPT is usually much higher. Parathyroidectomy is ineffective and not indicated for FHH.

5. Preoperative localization studies. Parathyroid imaging has no role in the diagnosis of HPT but is critical in operative planning to facilitate limited neck exploration. Technetium-99 sestamibi scintigraphy has historically been utilized to localize hyperfunctioning parathyroid tissue, though newer modalities of scintigraphy in combination with single photon emission computed tomography (SPECT), SPECT with computed tomography (SPECT/CT), and four-dimensional CT have demonstrated higher sensitivity and positive predictive values (*J Nucl Med.* 2007;48(7):1084-1089). Ultrasonography with color Doppler examination complements the sestamibi scan and can assist in precise localization of adenomas, assessment of concomitant thyroid pathology, or FNA of equivocal lesions.

6. Indications for surgery. Parathyroidectomy is indicated for patients with classic symptoms of primary HPT (i.e., nephrolithiasis, pathologic fracture, neuromuscular disturbances, and hypercalcemic crisis). Management of asymptomatic patients is more controversial, but recent

guidelines from an expert consensus panel recommend parathyroidectomy for those meeting one of the following criteria: (1) Age less than 50 years; (2) unable to participate in appropriate followup; (3) serum calcium level >1 mg/dL above normal range; (4) urine calcium >400 mg per 24 hours; (5) creatinine clearance <60 mL/min; or (6) complications of primary HPT (*J Clin Endocrinol Metab.* 2014;99(10):3561-3569). If patients do not meet one of these criteria, surgery is not required, but remains an option. Many patients choose surgery if they have significant nonspecific symptoms. Nephrolithiasis, bone disease, and neuromuscular symptoms are improved following surgery more often than renal insufficiency, hypertension, and psychiatric manifestations.

7. Operative strategies

a. Minimally invasive parathyroidectomy. Preoperative localization studies used with rapid intraoperative PTH measurement now enable several minimally invasive techniques, including open, radioguided, video-assisted, and endoscopic methods. Since the majority (>85%) of patients with primary HPT have a single parathyroid adenoma, successful preoperative localization allows for directed unilateral neck exploration while normal parathyroids do not need to be identified. The rapid intraoperative PTH assay allows surgeons to verify the adequacy of resection: following adenoma removal; a 50% decrease in PTH levels at 10 minutes is highly indicative of cure.

b. Conventional neck exploration. Historically, bilateral neck exploration and identification of all four parathyroids has been the cornerstone of surgical management in HPT, with resultant normocalcemia in more than 95% of patients. If an abnormally enlarged parathyroid or all four parathyroids cannot be found, exploration for ectopic or supernumerary glands should be performed. Ectopic superior glands may be found posterior and deep to the thyroid, in the tracheoesophageal groove, or between the carotid artery and the esophagus. Ectopic inferior glands are most likely found embedded in the thymus in the anterior mediastinum. Occasionally, multiple

parathyroid adenomas are found and should be removed, leaving at least one normal parathyroid behind. Four-gland parathyroid hyperplasia is rare, and acceptable management options include total parathyroidectomy with parathyroid autotransplantation or 3.5-gland parathyroidectomy. A clear factual operative note detailing the identification and position of each gland is essential.

c. Parathyroid autotransplantation. Total parathyroidectomy with heterotopic parathyroid autotransplantation should be considered in patients with renal failure and secondary hyperparathyroidism, four-gland parathyroid hyperplasia, and those undergoing neck re-exploration in which the adenoma is the only remaining parathyroid gland. The sternocleidomastoid or the brachioradialis muscles of the patient's non-dominant forearm are common sites for autotransplantation. Parathyroid autotransplantation into the forearm is advantageous if recurrent HPT is a possibility (e.g., MEN type 1 or 2A) because the transplanted hyperfunctioning parathyroid tissue can easily be localized and excised under local anesthesia

with sedation.

Freshly removed parathyroid tissue is finely minced (approximately 1 × 1 × 2 mm) and placed in sterile iced saline. Separate intramuscular beds are created by spreading the fibers of the brachioradialis or the sternocleidomastoid with a fine forceps. Four to five pieces of parathyroid tissue are placed in each site for a total transplant volume of approximately 100 mg. Nonabsorbable suture is used to close the beds and to mark the site of transplanted tissue. Transplanted parathyroid tissue begins to function within 14 to 21 days of surgery.

Cryopreservation of parathyroid glands is performed in all patients who are at risk for permanent hypoparathyroidism after repeat exploration. Approximately 200 mg of minced parathyroid tissue is frozen in vials containing autologous serum, dimethyl sulfoxide, and cell culture media. Cryopreserved tissue can be used for autotransplantation in patients with failure of the initial graft. Viable cryopreservation and subsequent thawing must be performed in a Food and Drug Administration (FDA)-approved facility.

8. Management of postoperative hypocalcemia

a. Clinical manifestations. Transient hypocalcemia commonly occurs after total thyroidectomy or parathyroidectomy and requires treatment if it is severe (total serum calcium <7.5 mg/dL) or if the patient is symptomatic. Symptoms may involve numbness/paresthesias in the distal extremities, perioral numbness, or hyperactive tendon reflexes. Chvostek sign (twitching of the facial muscles with tapping over the facial nerve anterior to the ear) indicates relative hypocalcemia, but it is present in up to 15% of the normal population and does not necessarily require calcium replacement.

b. Oral calcium supplementation. Hypocalcemic patients may require postoperative supplementation for 6 to 8 weeks and are given oral calcium carbonate (500 to 1,000 mg TID) and calcitriol (0.25 µg/day).

c. Intravenous calcium supplementation. Intravenous administration of calcium gluconate or calcium chloride may be necessary in persistently symptomatic patients or in emergent situations such as hypocalcemic tetany. Ten to 20 mL of 10% calcium gluconate are given intravenously over 10 minutes and may be repeated every 15 to 20 minutes as required until symptom resolution. Subsequently, a continuous infusion of calcium gluconate in D5W is initiated at 0.5 to 1.5 mg/kg/hour with correction of any concurrent hypomagnesemia.

B. Secondary and Tertiary Hyperparathyroidism

1. Biochemical evaluation. Most commonly due to chronic renal failure, secondary HPT manifests as increased PTH levels in response to hypocalcemia. Decreased serum calcium levels are a terminal feature of kidney dysfunction, which becomes evident through phosphate retention, decreased vitamin D activation, and poor calcium absorption. Intestinal malabsorption of calcium or vitamin D can also result in elevated PTH levels and secondary HPT. Thus, patients

with secondary HPT have high PTH levels and low calcium levels. Tertiary hyperparathyroidism can be seen in patients who have undergone a kidney transplant for renal failure. Typically, parathyroid gland function returns to normal within one year after kidney transplant, but in patients with tertiary HPT, the parathyroid glands fail to respond to normal signals for PTH secretion and regulation of calcium homeostasis. In these patients, both the PTH and calcium levels are high.

2. Medical management. Hypercalcemia from secondary and tertiary HPT is treated initially with dietary phosphate restriction, phosphate binders, and vitamin D supplementation. Cinacalcet, a calcimimetic, is also commonly used. Although it does not impact mortality, it may decrease the need for parathyroidectomy.

3. Operative strategies. Patients with medically unresponsive, symptomatic HPT (e.g., bone pain, osteopenia, ectopic calcification, or pruritus) may undergo total parathyroidectomy with autotransplantation or subtotal parathyroidectomy. Controversy exists regarding type of surgery and appropriate postoperative PTH level to prevent adynamic bone disease.

C. Recurrent or Persistent Hyperparathyroidism

1. Biochemical evaluation. In all cases of persistent hypercalcemia, the diagnosis of HPT should be confirmed. In addition to calcium and intact PTH levels, a 24-hour urine calcium should be obtained to rule out FHH. Factors associated with recurrent or persistent disease include failed preoperative localization studies, multiple gland disease, ectopic or supernumerary glands, malignancy, and surgeon inexperience.

2. Preoperative localization. Preoperative localization is mandatory in patients being considered for reoperative parathyroidectomy and may include ^{99m}Tc-sestamibi scintigraphy with SPECT/CT, ultrasound with FNA, or 4D CT scan. Approximately 70% to 80% of patients undergoing reexploration have a missed gland that is accessible through a

cervical incision. Noninvasive imaging is successful in gland localization in 25% to 75% of cases. For patients with negative or discordant noninvasive studies, selective venous sampling with rapid PTH assessment can be considered.

3. Operative strategy. The goal of reexploration is to perform an orderly search based on information from the initial operation and from preoperative localization studies. Reoperative parathyroid surgery carries a substantially higher risk of RLN injury and of hypocalcemia due to postoperative scarring and disruption of normal tissue planes.

a. Missed parathyroid glands can be found in either normal anatomic location or in ectopic sites. They may occasionally be intrathyroidal (especially in patients with multinodular goiter), and thyroid lobectomy can be performed if an exhaustive search fails to identify a parathyroid adenoma. If four normal glands have been located, a supernumerary gland is likely responsible. Intraoperative ultrasound and/or venous sampling from the right and left internal jugular veins

can sometimes be useful in localizing adenomas.

b. Mediastinal adenomas within the thymus are managed by resecting the cranial portion via gentle traction on the thyrothymic ligament or by a complete transcervical thymectomy using a specialized substernal retractor (*Ann Surg.* 1991;214:555). Median sternotomy is associated with higher morbidity and increased postoperative pain, and the possibility that this procedure may be required should be discussed with the patient preoperatively.

III. PARATHYROID CANCER

A. Clinical Manifestations. Parathyroid cancer is a rare disease, accounting for <1% of patients with primary HPT. Most (>90%) of parathyroid carcinomas are biochemically functional. Approximately 50% of patients have a palpable neck mass, and serum calcium levels may exceed 15 mg/dL. Ultrasound and ^{99m}Tc sestamibi imaging can help localize disease, but diagnosis depends on histologic findings of vascular or capsular invasion, metastases, or gross invasion of local structures.

B. Medical Management of Hypercalcemic Crisis. Patients with parathyroid cancer and some patients with benign HPT may develop hypercalcemic crisis with serum calcium levels of 16 to 20 mg/dL and azotemia. Symptoms of this acute, sometimes fatal illness, include profound muscular weakness, nausea and vomiting, drowsiness, and confusion. Ultimate treatment is parathyroidectomy, but volume and electrolyte abnormalities should be addressed first. First-line therapy involves intravenous infusion of 0.9% sodium chloride at 300 to 500 mL/hour to restore intravascular volume and to promote renal excretion of calcium. After urinary output exceeds 100 mL/hour, furosemide (80 to 100 mg intravenously every 2 to 6 hours) may be given to promote further renal sodium and calcium excretion, though consequent hypokalemia and hypomagnesemia should be corrected. Thiazide diuretics impair calcium excretion and should be avoided. If diuresis alone is unsuccessful in lowering the serum calcium,

other agents may be used, including the bisphosphonates pamidronate and etidronate, mithramycin, and salmon calcitonin. Orthophosphate, gallium nitrate, and glucocorticoids also have calcium-lowering effects.

C. Operative Strategy. Surgical treatment of parathyroid cancer entails radical local excision of the tumor, surrounding soft tissue, lymph nodes, and ipsilateral thyroid lobe. Reoperation is indicated for local recurrence or to control malignant hypercalcemia.

CHAPTER 40: THYROID AND PARATHYROID GLANDS

Multiple Choice Questions

1. A 56-year-old man presents to clinic for evaluation of a small right neck mass. He has no significant past medical history and denies any

history of smoking. The mass has been slowly enlarging over the last 2 years but is not painful. The patient is normotensive with a negative review of systems, and he denies any dyspnea, choking sensations, or hoarseness. What is the first diagnostic study that should be performed in the workup of this mass?

a. Ultrasonography of the thyroid

b. Serum thyroid stimulating hormone (TSH) level

c. Fine-needle aspiration (FNA)

d. Computed tomography (CT) scan of the neck and chest

e. Thyroid scintigraphy

View Answer

2. A 64-year-old woman presents to the emergency department with vague abdominal pain, nausea, confusion, and muscle weakness. An EKG shows a short QT interval. A serum calcium level is 15.2 mg/dL. What etiology does this suggest?

a. Thiazide use

b. Secondary hyperparathyroidism

c. Parathyroid carcinoma

d. Single parathyroid adenoma

e. Factitious hypercalcemia

View Answer

3. Following total thyroidectomy, a 50-year-old male presents for his 1 year followup visit. He is currently on daily levothyroxine therapy. The best method to monitor the adequacy of replacement therapy is:

a. Radioactive iodine (RAI) uptake

b. Thyroglobulin

c. Triiodothyronine resin uptake (RT_3U)

d. Serum TSH level

e. Total thyroxine level (total T_4)

View Answer

4. A 72-year-old woman with recently diagnosed primary hyperparathyroidism presents for surgical evaluation. Her serum calcium level is found to be 13.6 mg/dL. Other biochemical abnormalities

that may accompany hypercalcemia in patients with primary hyperparathyroidism include which of the following?

a. Metabolic alkalosis

b. Hyperphosphatemia

c. Hypochloremia

d. Hypermagnesemia

e. Elevated alkaline phosphatase

View Answer

5. A 63-year-old patient with primary hyperparathyroidism and nonlocalizing studies undergoes neck exploration. He is found to have normal right and left superior parathyroids and a normal left inferior parathyroid gland.

Of the following, which is the most likely location for the missing right inferior parathyroid?

a. Tracheoesophageal groove

b. Right thyroid lobe

c. Superior thymus

d. Posterior mediastinum

e. Pharyngeal mucosa

View Answer

6. Which of the following patients with thyroid gland enlargement is LEAST likely to have a diagnosis of thyroid cancer?

a. A 5-year-old boy with two family members with medullary thyroid carcinoma

b. A 75-year-old man with a solitary nodule and hoarseness

c. A 56-year-old woman with a solitary nodule and a history of radiation therapy to the neck

d. A 43-year-old woman with a multinodular goiter and tremor

e. A 14-year-old girl with an asymptomatic solitary nodule

View Answer

7. A 47-year-old woman presents to clinic for evaluation of weight gain, thinning hair, constant fatigue, constipation and muscle weakness over the past year. She denies any prior history of thyroid disorders and

currently takes no medications. Serum TSH level is elevated at greater than 30 mIU/L. What is the most likely cause of the patient's symptoms?

a. Thyroid adenoma

b. Self-administration of thyroid hormone

c. Papillary thyroid carcinoma

d. Radioactive iodine administration

e. Hashimoto thyroiditis

View Answer

8. A 65-year-old female presents for her first postoperative visit following total thyroidectomy for papillary thyroid carcinoma. Which of the following features on the pathology report may indicate tumor aggressiveness and poor outcome?

a. Tumor size larger than 4 cm

b. Macroscopic lymph node metastasis

c. Local invasion through capsule into surrounding soft tissue

d. Poorly differentiated histologic grade

e. All of the above

View Answer

9. A young family brings a 1-month-old female into your clinic for evaluation after referral from their pediatrician. The child's father underwent total thyroidectomy in infancy and multiple family members have required adrenalectomy for pheochromocytomas. Which of the following is the most sensitive method for detecting medullary thyroid carcinoma in patients with multiple endocrine neoplasia (MEN) type 2 syndromes?

a. Genetic evaluation

b. Plasma carcinoembryonic antigen level

c. Plasma catecholamine level

d. Serum thyroglobulin level

e. Serum calcium level

View Answer

10. A 34-year-old female presents to clinic for evaluation of a large

thyroid goiter. She complains of an intermittent tremor, increased appetite, palpitations, and increased perspiration. What other features may also be present on history and physical examination?

a. Exophthalmos and pretibial myxedema

b. Suppressed thyroid stimulating hormone level

c. Cold intolerance and somnolence

d. Answers A and B only

e. All of the above

View Answer

41

Otolaryngology for the General Surgeon

Lauren T. Roland

John S. Schneider

I. THE NECK

A. Anatomy and Physiology

1. The **cervical fascia** provides planes for passage of infection, hemorrhage, and surgical dissection (Fig. 41-1).

2. The **lymphatic system** of the neck is divided into six levels. Level I contains the submental and submandibular lymph nodes, level II through IV parallel the jugular vein, level V consists of the posterior triangle, and level VI is the central compartment. These levels are important for predicting spread of cancer from the head and neck, as well as determining the extent of surgery necessary during a neck dissection (Fig. 41-2).

B. Neck Masses. History for a neck mass should focus on duration, location, and symptoms (pain, fevers, weight loss, dysphagia, voice changes, otalgia), past medical history, and social history (tobacco and alcohol use, travel history, animal exposures, sick contacts). Differential diagnosis is strongly influenced by age. Neck masses in adults are presumed malignant until proven otherwise. In contrast, neck masses in children are usually inflammatory or congenital, and neoplasms are rare.

1. Adult neck masses. Adult neck masses are commonly metastatic squamous cell carcinoma (SCC) from a primary tumor of the oral cavity, pharynx, or larynx. See Figure 41-3 for an algorithm describing workup of a neck mass in an adult.

2. Pediatric neck masses. Children often have palpable lymph nodes; however, large, persistent masses should be investigated through ultrasound (US), as this is safe and noninvasive. CT should be reserved for deep neck space infections. Additional studies include white blood cell count (WBC) with differential and specific serologic tests for infectious etiologies. See Figure 41-4 for an algorithm describing workup of a neck mass in children.

C. Congenital Neck Lesions. Congenital masses may swell during an upper respiratory infection (URI). The acute infection should be treated with antibiotics. If necessary, needle aspiration may be performed for decompression.

1. Branchial cleft anomalies. These congenital masses may result in cysts, sinuses, or fistulae. The most common anomaly is of the **second branchial cleft,** which presents as a nontender,

fluctuant mass anterior to the sternocleidomastoid muscle, with a deep tract that travels between the

internal and external carotid arteries to the tonsillar fossa. **First branchial cleft** anomalies present near the angle of the mandible or around the ear and may be associated with the facial nerve. **Third branchial cleft** anomalies present as lower neck masses with tracts that end in the thyrohyoid membrane or pyriform sinus.

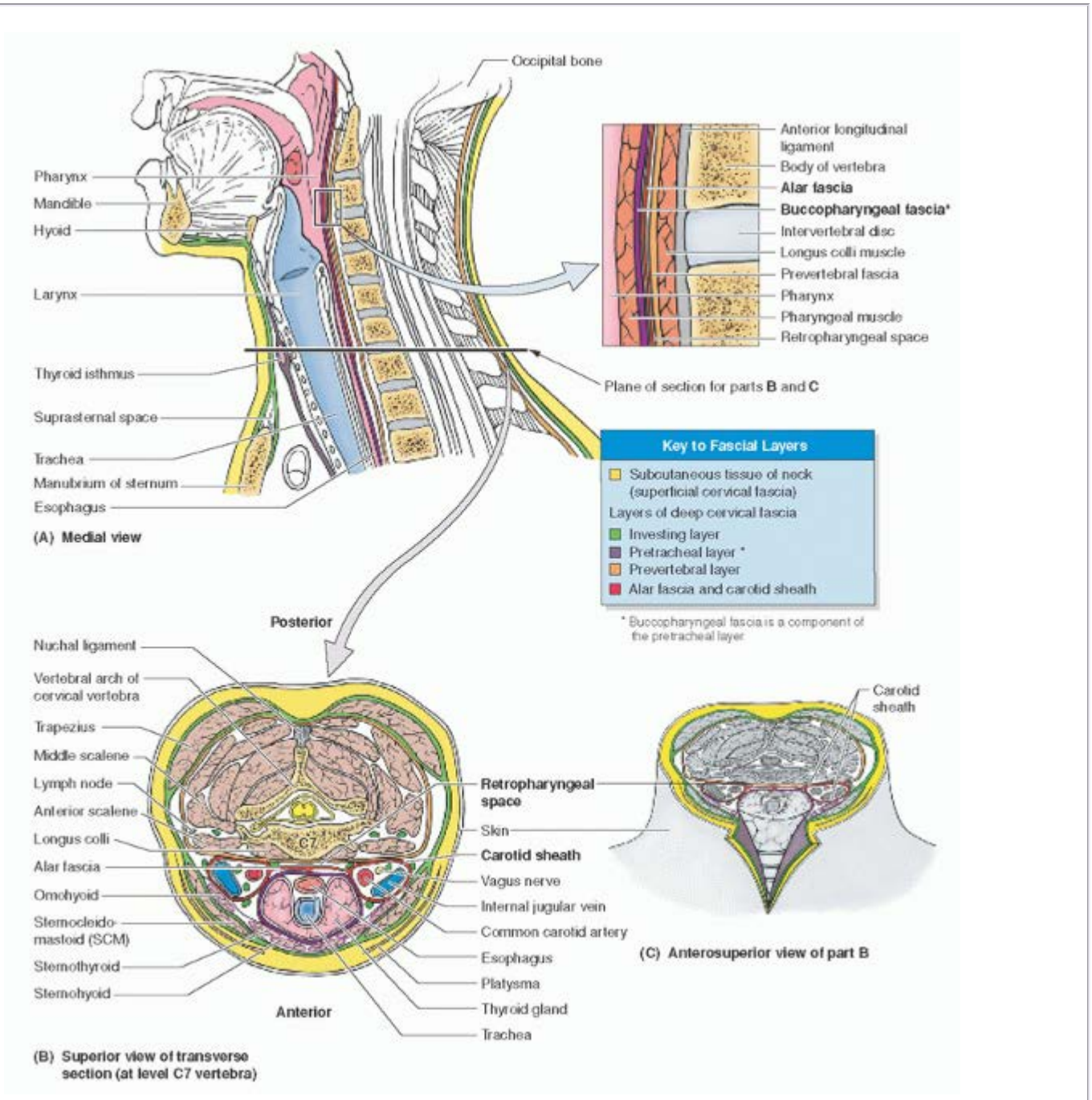


Figure 41-1 Fascial layers of the neck. (From Moore KL, Dalley AF, Agur AM. *Clinically oriented anatomy.* 6th ed. Baltimore, MD: Wolters Kluwer Health, 2010.)

2. Thyroglossal duct cysts. These masses are due to lack of obliteration of the thyroglossal duct after descent of the thyroid from the base of the tongue during embryologic development. Due to attachment to the tongue, these midline masses often move with tongue protrusion. Before surgery, the physician should perform an US to ensure that the mass is not the sole thyroid tissue, as removal would cause hypothyroidism. The

definitive treatment is the **Sistrunk procedure** and involves removal of the tissue as well as the midline portion of the hyoid bone that the embryologic tissue passes through.

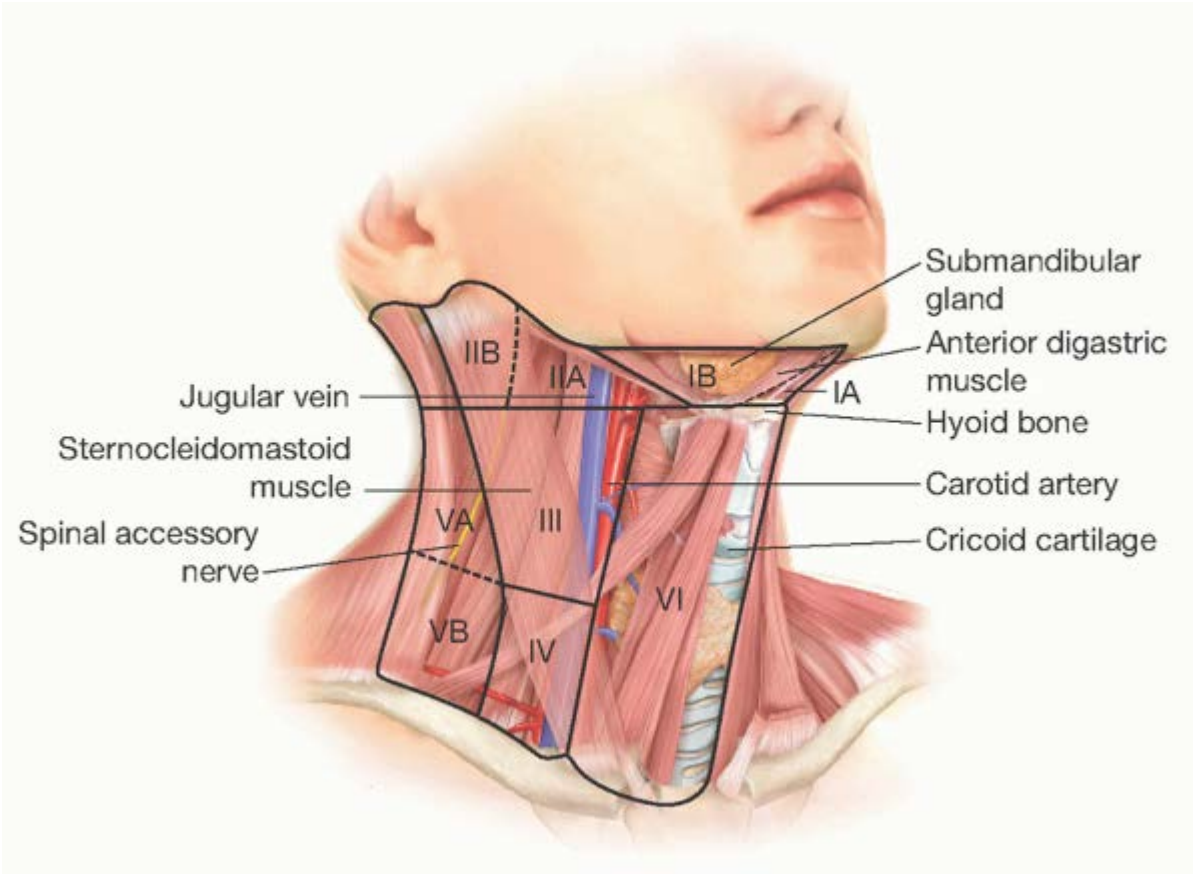


Figure 41-2 Neck Node Levels. **IA:** Submental, **IB:** Submandibular, **II:** Upper jugular, **III:** Middle jugular, **IV:** Lower jugular, **V:** Posterior triangle, **VI:** Anterior compartment. (From Mulholland, MW, ed. *Operative techniques in surgery.* Philadelphia, PA: Wolters Kluwer Health, 2015.)

3. Hemangioma. This lesion presents as a reddish-bluish compressible mass in infancy. Hemangiomas typically grow rapidly during the first year of life and then slowly involute; the majority do not require treatment. However, cervical hemangiomas may be associated with subglottic hemangiomas or airway masses which may be symptomatic and require emergent

attention. Propranolol, steroids, laser therapy, and surgical resection are treatment options.

4. Lymphatic malformations. These lesions are soft, doughy, compressible lesions. Treatments include sclerotherapy and surgery.

5. Other congenital masses include **laryngoceles, dermoid cysts, teratomas, plunging ranulas,** and **thymic cysts.**

D. Infectious/Inflammatory Disorders

1. Suppurative bacterial lymphadenitis. This condition is common in children and is usually the result of *S. aureus* or group A streptococcal infections. Treatment includes incision and drainage (I&D) and intravenous (IV) antibiotics.

2. Acute mononucleosis. Mononucleosis is caused by EpsteinÑBarr (EBV) infection and commonly causes lymphadenopathy. It is associated with fevers, tonsillitis, and hepatosplenomegaly. Treatment is supportive.

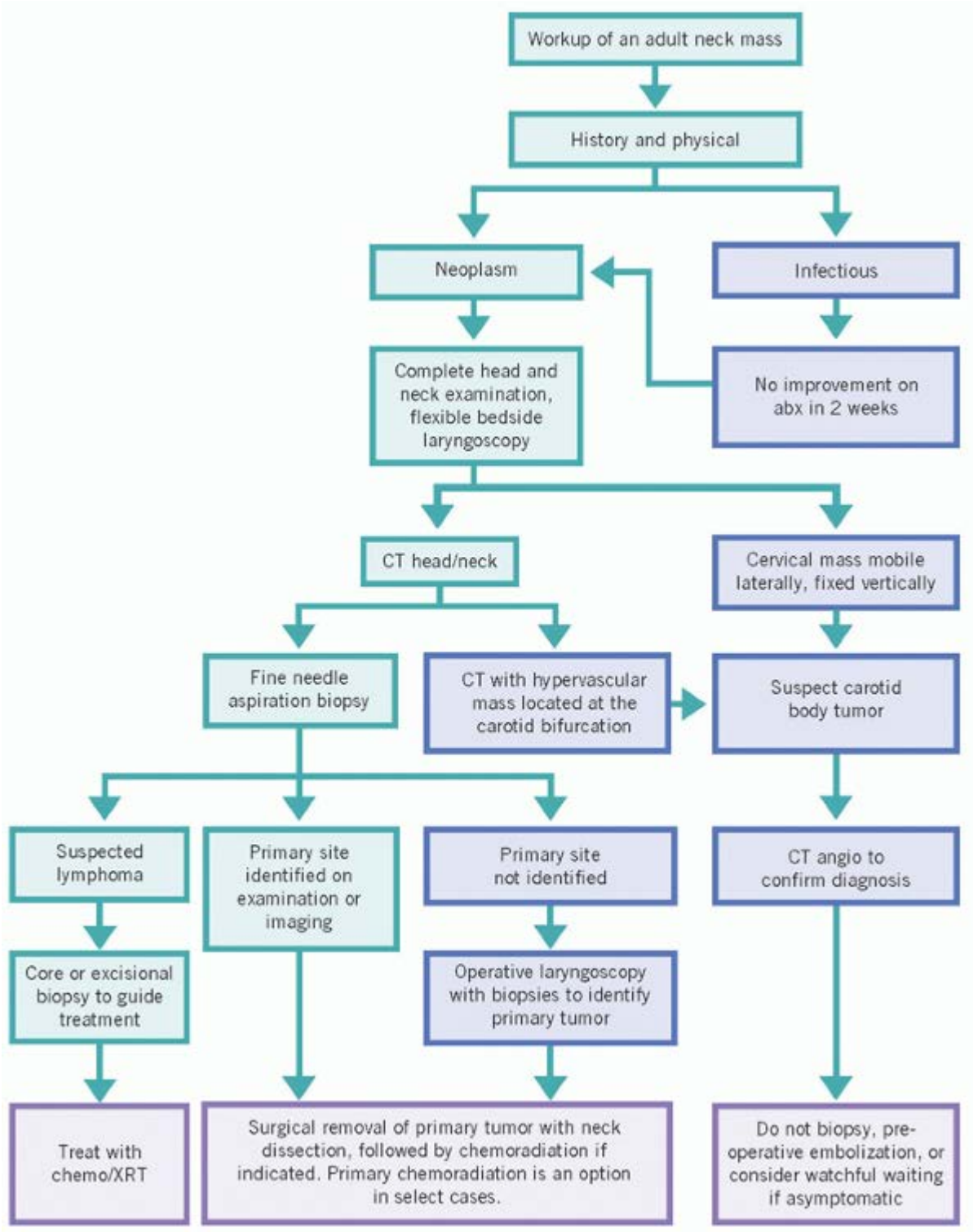


Figure 41-3 Workup of adult neck mass.

3. Deep neck space infections. Neck abscesses present with neck swelling, tenderness, fevers, and dysphagia/odynophagia. Again, treatment is I&D and IV antibiotics. A particularly dangerous infection is cellulitis of the submandibular and submental spaces called **Ludwig angina.** The swelling from this infection can cause retrusion of the tongue and airway compromise.

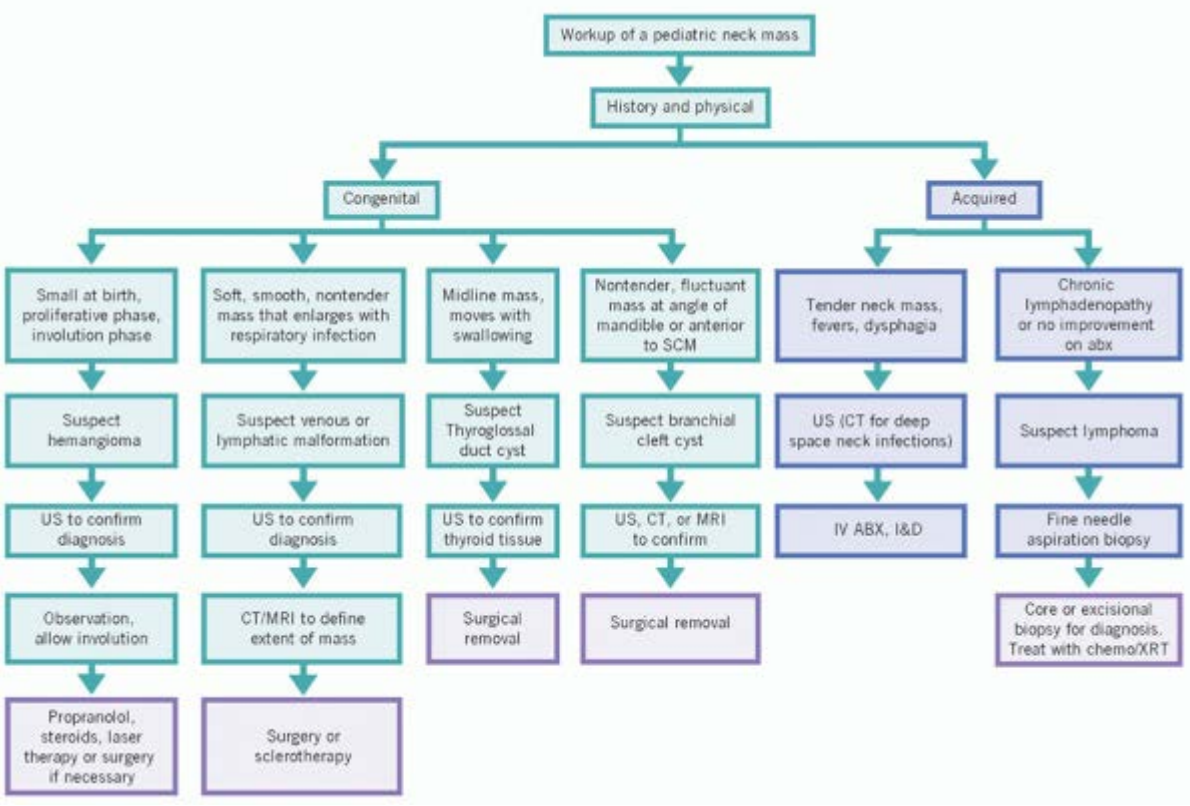


Figure 41-4 Workup of pediatric neck mass.

E. Neoplasm

1. Benign

a. Paragangliomas. These tumors arise from paraganglionic cells of the autonomic nervous system. Paragangliomas can be familial and 40% of patients have a genetic mutation (*Nat Rev Endocrinol.* 2015;11:101-111). The most common paragangliomas are **carotid body tumors.** These tumors can be monitored over time if asymptomatic, and definitive management includes preoperative embolization followed by surgical removal.

(1) Complications and management. Carotid body tumors can be challenging to remove surgically. Preoperative embolization can help with blood loss. Complications include significant intraoperative blood loss, cerebrovascular insults (stroke), need for reconstruction of the carotid artery, and injury to the vagus nerve.

b. Other benign tumors include **lipomas, schwannomas, infiltrative fibromatosis, neurofibromas,** and **salivary gland neoplasms.**

2. Malignant. The most common malignant neck mass in adults is metastatic SCC, and in children, lymphoma. Location of the mass is suggestive of primary site, based on patterns of lymphatic drainage.

a. SCC of aerodigestive mucosa often metastasizes to the neck. The neck is treated with lymphadenectomy, with possible adjuvant radiation and chemotherapy, often with combined modalities. Neck dissections are termed *therapeutic* for clinically palpable metastases or *elective* in the absence of clinical lymphadenopathy. *Radical* neck dissection includes resection of all lymph nodes, the sternocleidomastoid muscle, internal jugular vein, and spinal accessory nerve (cranial nerve [CN] XI). *Modified radical* neck dissection reduces morbidity by sparing one or more of these structures. *Selective* neck dissection removes only nodal groups at greatest probability for containing metastases for a particular primary site.

(1) A special diagnostic dilemma is cervical SCC with unknown primary. Patients should undergo operative panendoscopy with biopsies of the nasopharynx as well as palatine and/or lingual tonsillectomy. PET/CT may also detect a primary source.

(2) Complications and management. Complications of neck dissection include potential injury to major nerves and vessels of the neck. The internal jugular vein and carotid artery are both exposed during this surgery. Patients are counseled regarding the risk of injury and paralysis to nerves including the marginal mandibular nerve and the spinal accessory nerve during the careful removal of affected lymph nodes. In the event of recognized injury to a CN, immediate repair can be performed. Postoperative weakness of CN XI often improves with physical therapy.

b. Thyroid carcinoma (see **Chapter 40**, **Thyroid and Parathyroid Glands,** for more in-depth discussion of thyroid malignancy). Thyroid nodules are very common but malignancy is rare (*Cancer Imaging.*

2011;11:209-223). The most common thyroid cancer is **papillary carcinoma,** followed by **follicular** and **medullary** thyroid cancer. Rare pathologies include lymphoma, **metastatic** lesions, and **anaplastic** carcinoma. Nodules larger than 1 cm, with suspicious US findings, should undergo US-guided fine needle aspiration (FNA) biopsy. Total thyroidectomy is indicated for biopsies diagnostic of malignancy and lobectomy may be performed for low-risk, small tumors. Neck dissection is also considered for cervical metastases. Unresectable or anaplastic disease may undergo external beam radiation therapy.

(1) Complications and management. A potential complication of thyroid surgery is injury to the nearby **recurrent laryngeal nerve**s, which innervate the vocal cords. Injury to a single nerve can cause hoarseness and dysphagia, whereas a bilateral injury can lead to airway compromise. Injuries, including transient palsies and permanent injury, occur at a rate of about 3% to 4% per nerve (*J Surg Res.* 2014;188:152-161). (See below under The Larynx > Neuromuscular disorders > vocal cord paralysis, section II.D.I, for treatment options.)

c. Lymphoma. The majority of head-and-neck lymphomas present in cervical lymph nodes.

Patients may have **B symptoms** (fevers, night sweats, weight loss). Surgery aids in diagnosis and is not curative. FNA biopsy provides cytologic material, but tissue samples obtained from core or excisional biopsy are often required for architectural detail, flow cytometry, and immunophenotyping. Treatment is chemotherapy and radiation.

II. THE LARYNX

A. Anatomy and Physiology

1. The **larynx** is divided into the supraglottis (which includes the epiglottis, arytenoid cartilages, false vocal cords/folds, and ventricles), the glottis (true vocal cords/folds), and subglottis (extending from the true vocal cords inferiorly to the cricoid cartilage). See Figure 41-5 for more detailed laryngeal anatomy.

2. The **recurrent laryngeal nerve** (CN X) provides sensory innervation to most of the laryngeal mucosa and motor innervation to all of the intrinsic laryngeal muscles, except for the cricothyroid muscle, which is innervated by the **superior laryngeal nerve** (CN X). Most importantly, the recurrent laryngeal nerves provide innervation to the vocal cords, allowing for movement during speech and swallowing.

B. Congenital Disorders

1. Laryngomalacia. This disorder presents with inspiratory stridor in an infant; awake flexible fiberoptic laryngoscopy demonstrates prolapse of the arytenoid mucosa into the airway on inspiration, shortened aryepiglottic folds, and often an omega-shaped epiglottis likely due to neuromuscular hypotonia. A surgical intervention called **supraglottoplasty** is indicated for difficulty feeding and failure to thrive, apnea, or cyanosis.

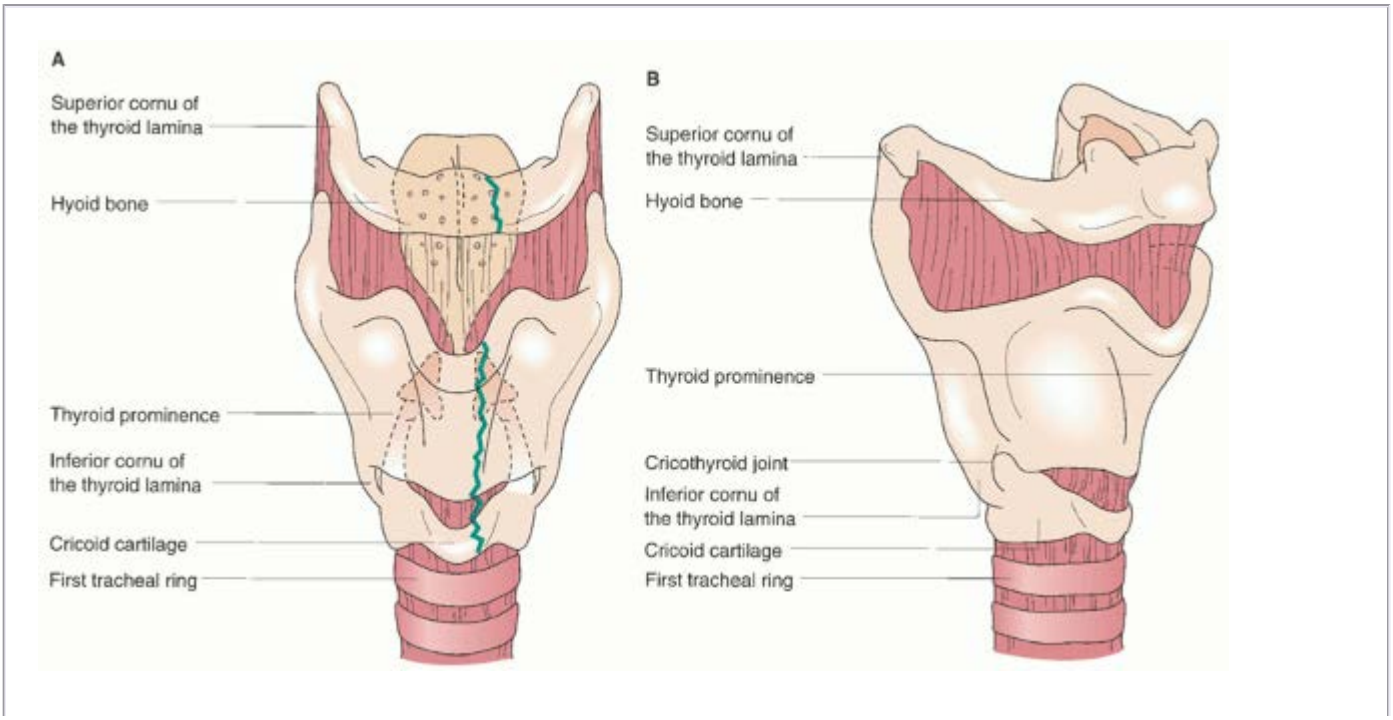


Figure 41-5 A, B: External Larynx Anatomy. (From Mulholland, MW et al., eds. *Greenfield's surgery: scientific principles and practice.* Philadelphia, PA: Wolters Kluwer Health, 2011.)

2. Vocal cord paralysis. Vocal cord paralysis causes inspiratory or biphasic stridor, aspiration, and weak cry. Etiologies include birth trauma, neurologic disease, and iatrogenic injury. Diagnosis is made by awake flexible laryngoscopy. Most noniatrogenic unilateral paralysis resolves spontaneously within the first year of life. Bilateral cord paralysis often requires tracheotomy.

3. Subglottic stenosis. Congenital subglottic stenosis is due to abnormally formed cricoid cartilage. Acquired stenosis is usually due to intubation. If stenosis is mild, patients may improve as they grow. Otherwise, surgical laryngotracheal reconstruction or cricotracheal resection may be necessary.

C. Infectious/Inflammatory Disorders

1. Viral croup. Viral laryngotracheitis is glottic and subglottic inflammation from parainfluenza virus. Patients present with a barking cough, hoarseness, and inspiratory stridor. Lateral airway x-ray may show the **steeple sign** from subglottic edema. Treatment includes humidified air, glucocorticoids for moderate to severe croup, racemic epinephrine, and heliox.

2. Epiglottitis. This infection is usually caused by *H. influenzae* type B. Patients present with fever, muffled voice, drooling, and stridor. Treatment is urgent airway management and IV antibiotics.

D. Neuromuscular Disorders

1. Vocal cord paralysis. This disorder occurs secondary to recurrent laryngeal nerve injury, often due to surgery, neoplasm, or trauma to the neck or thorax. Iatrogenic injuries during surgery should be repaired by primary epineural anastomosis or cable grafting. Treatment for unilateral paralysis consists of speech therapy and observation, as recovery often occurs over several months. **Temporary vocal cord medialization** via injection of a resorbable material (e.g., hyaluronic acid, Gelfoam) helps prevent aspiration and improves voice during nerve recovery, with effects lasting up to 6 months. If significant problems persist, **thyroplasty and laryngeal reinnervation** are other surgical options. Bilateral paralysis can cause stridor from airway obstruction and is treated with **arytenoidectomy, cordectomy,** or **tracheostomy.**

E. Neoplasm

1. Benign. Recurrent respiratory papillomatosis. RRP is characterized by bulky papillomas, caused by **HPV 6 and 11** on the larynx and tracheobronchial tree, causing hoarseness and airway obstruction. Treatment is repeated excision.

2. Malignant. Laryngeal malignancies are most commonly **SCC.** Patients present with

hoarseness, dyspnea, stridor, dysphagia, and neck mass (from metastases). Patients are treated with a combination of surgery (either total laryngectomy or partial laryngectomy ± neck dissection), chemotherapy, and radiation, depending on stage and location (glottic, supraglottic, subglottic) of the tumor.

F. Trauma

1. Blunt or penetrating **laryngeal trauma** requires rapid airway assessment and management, often requiring intubation or awake tracheostomy. Workup involves fiberoptic laryngoscopy, CT, and operative endoscopy. Laryngeal hematomas and small lacerations are managed conservatively with airway observation and humidified air. Displaced fractures and laryngeal instability require urgent tracheostomy followed by open reduction and internal fixation.

III. ORAL CAVITY AND PHARYNX

A. Anatomy and Physiology

1. The oral cavity. The oral cavity extends from the vermillion border of the lips anteriorly to the circumvallate papillae and junction of the hard and soft palate posteriorly. The pharynx is divided into the nasopharynx, oropharynx, and hypopharynx.

B. Congenital Disorders

1. PierreÑRobin sequence. This congenital disorder is characterized by micrognathia, glossoptosis, and a U-shaped cleft palate. Treatment may be needed for airway obstruction or feeding difficulties and includes prone positioning, glossopexy, mandibular advancement, and tracheostomy.

2. Cleft lip and palate. Clefts are caused by an embryologic failure of midface fusion. In addition to cosmetic and feeding issues, cleft palate causes abnormal insertion of the tensor veli palatini muscle, resulting in eustachian tube dysfunction and recurrent ear infections.

C. Infectious/Inflammatory Disorders

1. Tonsillopharyngitis. This common childhood infection is usually viral; bacterial infections are typically caused by group A *β*-hemolytic streptococci. Current guidelines recommend tonsillectomy (± adenoidectomy) in children with 7 or more episodes in a year, or 5 episodes per year for 2 years, or 3 episodes per year for 3 years (*Otolaryngol Head Neck Surg.* 2011;144:S1-S30).

a. Complications and management. Postoperative tonsillectomy bleeding occurs in up to about 10% of cases (*J Laryngol Otol.* 2004;118:937Ñ 940). These events are usually within 2 weeks of surgery and require close monitoring; they may even necessitate a second surgical intervention.

2. Peritonsillar abscess (PTA). A PTA is a collection of purulence between the tonsil bed and capsule. Patients have often had a recent episode of tonsillitis and present with fever, pain,

trismus, and a muffled Òhot potato voice.Ó Examination reveals a bulging, erythematous soft palate with deviation of the uvula. Treatment is needle aspiration or I&D and antibiotics.

D. Neoplasms

1. Benign

a. Ameloblastoma. This benign tumor arises from odontogenic epithelium, most frequently occurring in the mandible. It often requires segmental mandibulectomy with reconstruction.

b. Premalignant lesions include **leukoplakia** (white hyperkeratotic patches) and **erythroplakia** (velvet-red patches).

2. Malignant

a. SCC is the most common neoplasm of the head and neck, and can arise in the oral cavity or pharynx and metastasize to the neck. Tobacco and alcohol use increase the risk for SCC, and recently HPV genotypes 16 and 18 have been found to increase the risk of SCC in young adults without a smoking or alcohol history. Staging follows the TNM (primary Tumor, regional Nodal metastases, distant Metastasis) site-specific guidelines from the American Joint Committee on Cancer. Treatment of SCC is complex and based on location, nodal involvement, local invasion, and metastasis. Treatment options include a combination of surgery (either open, robotic or transoral laser microsurgery), chemotherapy, and radiation.

b. Other oral cavity and oropharyngeal cancers include **minor salivary gland carcinomas, verrucous carcinoma, lymphoma, mucosal melanoma,** and **Kaposi sarcoma.**

E. Trauma

1. Mandible fractures occur most commonly at the angle and parasymphysis, as well as at the condylar neck. Fractures present with dental malocclusion, halitosis, and pain with crepitus while chewing or on manipulation. Panorex radiographs are usually sufficient to diagnose and visualize them postreduction; however, high-resolution maxillofacial CT may be more sensitive. Fractures can be treated by closed reduction and external fixation (Òwiring the jaw shutÓ) or by open reduction and internal fixation with lag screws and/or plates. Complications include wound infection, malocclusion, nonunion, tooth loss, temporomandibular joint ankylosis, and paresthesias.

IV. THE SALIVARY GLANDS

A. Anatomy and Physiology

1. There are three pairs of major **salivary glands** (parotid, submandibular, and sublingual) and many minor salivary glands in the mucosa of the oral cavity, oropharynx, and nasopharynx.

2. The **parotid gland,** which lies over the masseter muscle, is the largest salivary gland. The

facial nerve travels through this gland, dividing it into superficial and deep lobes. CN IX provides parasympathetic innervation, which helps to regulate saliva flow.

3. The **submandibular gland** is inferomedial to the mandible and the **sublingual gland** lies beneath the floor of the mouth mucosa. Secretomotor innervation to these glands is provided by the chorda tympani nerve (CN VII).

B. Inflammatory Diseases

1. Acute sialadenitis. This infectious process usually involves the parotid gland, presenting as a tender, preauricular swelling with purulence expressible from the parotid duct (Stensen duct). It occurs as a result

of retrograde bacterial contamination from the oral cavity due to stasis of saliva or stones. Treatment is hydration, warm compresses, massage, antibiotics, and sialogogues to stimulate saliva flow.

2. Sialolithiasis. Ductal calculi, or stones, are most commonly found in the submandibular glands. The obstruction causes swelling and pain. Stones are removed transorally using probing instruments, via open excision, or minimally invasively via sialoendoscopy.

C. Neoplasms. Salivary neoplasms are most frequent in the parotid gland (70%), but most parotid tumors are benign. A larger percentage of submandibular tumors are malignant (50% malignant), and sublingual tumors have a high likelihood of malignancy (*Sem Rad Oncol.* 2012;22:245-253).

1. Benign. The most common neoplasm is **pleomorphic adenoma,** followed by **Warthin tumor.** These tumors grow slowly, are painless, and usually occur in the parotid gland. Treatment is excision with a cuff of normal parotid tissue, sometimes necessitating superficial parotidectomy, with facial nerve preservation.

2. Malignancy. The most common malignancy is **mucoepidermoid carcinoma,** followed by **adenoid cystic carcinoma.** Other types include acinic cell carcinoma, adenocarcinoma, and primary SCC. Treatment is parotidectomy, with facial nerve sacrifice if involved in tumor, possible neck dissection, and possible adjuvant radiation therapy.

a. Complications and management. An important potential risk of **parotid surgery** is injury to the facial nerve. The **facial nerve** divides the superficial and deep parotid lobes, and is at risk during surgery, requiring intraoperative facial nerve monitoring. Iatrogenic nerve injury can be repaired primarily if recognized. In the event that the facial nerve is sacrificed due to tumor involvement, postoperative care to protect the exposed eye (eye patch, drops for moisture, gold weight implant in eyelid), and cosmetic surgery to improve facial symmetry may be indicated in the future.

V. THE EAR

A. Anatomy and Physiology

1. External ear. The auricle (pinna) is composed of elastic cartilage and channels sound waves to the external auditory canal.

2. Middle ear. The middle ear is a mucosa-lined sinus in the temporal bone containing the ossicular chain, which consists of the malleus, incus, and stapes.

3. Inner ear. The end-organs of hearing and balance are surrounded by thick bone, the otic capsule. The cochlea is a snail-shaped structure containing the organ of Corti. The vestibular system consists of three semicircular canals, which sense angular acceleration, and the saccule and utricle, which sense linear acceleration.

4. The **facial nerve** (CN VII) travels a complex path through the temporal bone, where it is vulnerable to trauma. It innervates the facial musculature, stapedius muscle, and taste sensation for the anterior two-thirds of

the tongue via the chorda tympani. It then exits the skull base to enter the parotid gland and innervate the muscles of facial expression.

B. Infectious/Inflammatory Disorders

1. Otitis externa, or ÒSwimmer's ear,Ó is inflammation of the external auditory canal causing severe ear pain, drainage, pruritus, canal swelling, and conductive hearing loss (CHL).

2. Acute otitis media (AOM) is acute inflammation of the middle ear, usually of infectious etiology. Tympanostomy tubes are recommended for recurrent infections: Three episodes in 6 months, or four in a year (*Curr Opin Otolaryngol Head Neck Surg.* 2008;16:406-411).

3. Otitis media with effusion (OME) is fluid present in the middle ear without acute infection. Patients present with hearing loss, aural fullness/pressure, and a dull, gray, or yellow tympanic membrane (TM) with reduced mobility. Tympanostomy tube placement is indicated for bilateral OME for greater than 3 months, unilateral OME for more than 6 months, and in children with hearing loss and concerns for speech/language delay (*Curr Opin Otolaryngol Head Neck Surg.* 2008;16:406-411). Adults with persistent unilateral OME should undergo evaluation of the nasopharynx for masses causing eustachian tube obstruction.

4. Mastoiditis. This infectious process presents with fevers, otalgia, postauricular tenderness and swelling, and a proptotic pinna. CT can help demonstrate the severity of infection, which can include subperiosteal abscess and intracranial complications. Treatment includes IV antibiotics, tympanostomy, and/or mastoidectomy.

a. Potential complications. Tympanomastoidectomy, which is performed for mastoiditis, requires exposure of the middle ear very close to the pathway of the facial nerve. Facial nerve injury is rare, and facial nerve monitoring is routinely used. Inadvertent entry into the tegmen tympani bone, which separates the mastoid from the middle cranial fossa, can lead to

cerebrospinal fluid (CSF) leaks or meningitis.

C. Hearing loss can be conductive, sensorineural, or mixed.

1. CHL is caused by pathology of the external auditory canal, TM, and middle ear, resulting in attenuation of sound energy delivered to the inner ear.

2. Sensorineural hearing loss (SNHL) involves the cochlea or auditory neural pathway. Congenital SNHL occurs in 1 in 1,000 newborns, with approximately 50% from genetic etiology (*AJNR Am J Neuroradiol.* 2012;33:211-217).

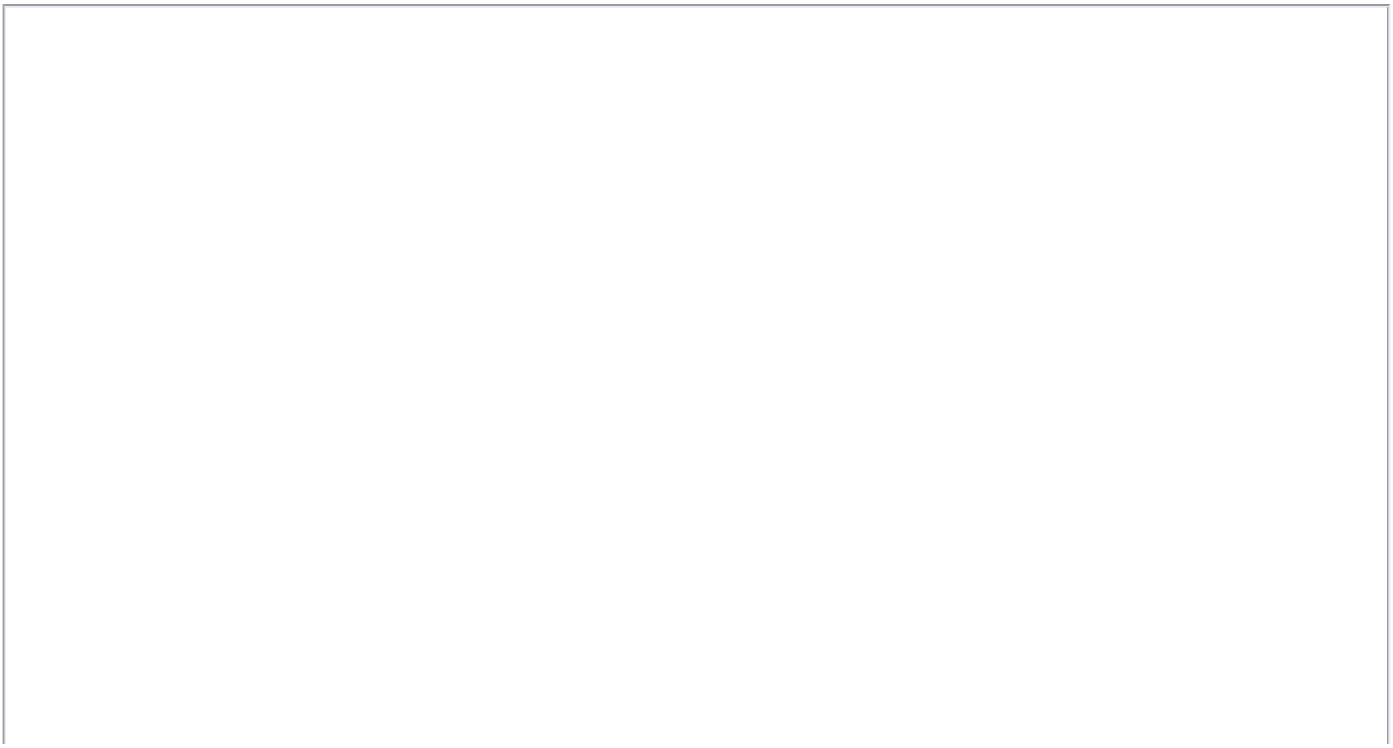
3. Treatment of hearing loss

a. Hearing loss is commonly treated with hearing aids. Idiopathic sudden SNHL can be treated with high-dose systemic or intratympanic steroids.

b. Profoundly deaf individuals who do not benefit from hearing aids are candidates for cochlear implant (CI), in which an electrode array is placed into the cochlea.

D. Vestibular schwannomas, also known as acoustic neuromas, are benign neoplasms arising from CN VIII. Patients can present with hearing loss, vertigo, and/or facial nerve paralysis. Workup includes auditory brainstem response (ABR) and MRI, and definitive treatment includes surgical excision, usually requiring a craniotomy. Many are slow-growing and may be closely monitored, particularly in patients not suitable for surgery.

E. Vertigo. True vertigo originates from the inner ear and is described as a sensation of spinning or moving. Diagnosis is made by history, physical examination, and vestibular testing. Please see Figure 41-6 for an algorithm describing the workup and management of vertigo.



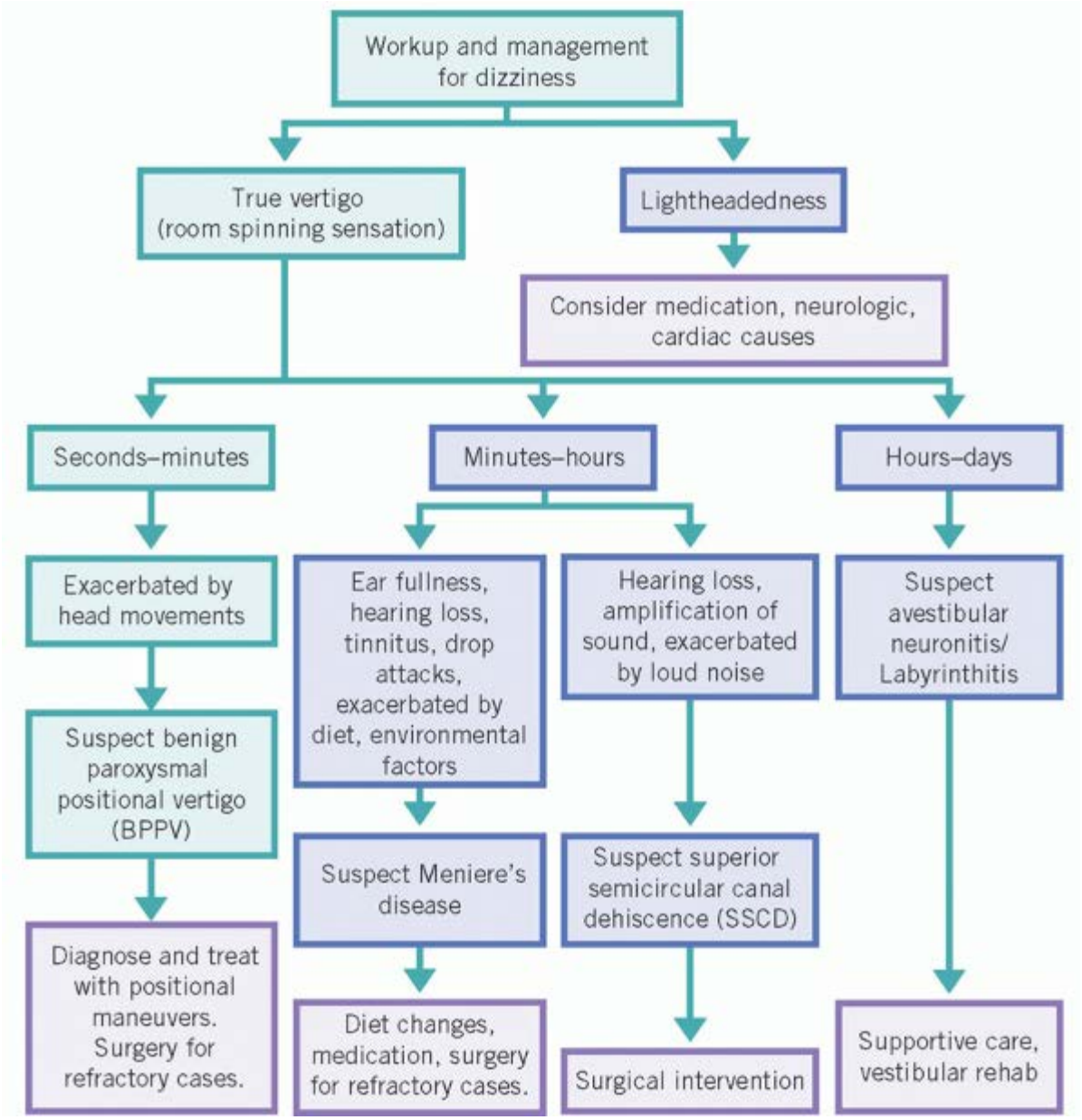


Figure 41-6 Algorithm for workup of dizziness.

VI. THE NOSE AND PARANASAL SINUSES

A. Anatomy and Physiology

1. The nose and septum are composed of bone superiorly and posteriorly and cartilage anteriorly. The turbinates are mucosa-covered bony prominences from the lateral nasal cavity that humidify, warm, and filter inhaled air. The nasopharynx contains the eustachian tube orifices bilaterally, and the adenoid pad centrally, which involute in late childhood.

2. The paranasal sinuses are pneumatized cavities in the skull named for the bone in which they

lie (frontal, sphenoid, ethmoid, or maxillary). They reduce the weight of the skull, contribute to the resonance of a person's voice, and cushion the cranial contents against trauma. They are lined with ciliated respiratory epithelium.

B. Infectious/Inflammatory Disorders

1. Acute rhinosinusitis. The etiology of this process may be infectious or allergic. Although most acute rhinosinusitis is viral, bacterial superinfection may occur, typically with *S. pneumoniae, H. influenzae, M. catarrhalis,* or *S. aureus.* Treatment consists of saline nasal irrigations to improve mucociliary clearance, mucolytics to thin secretions, hydration, and antibiotics for bacterial sinusitis (first-line agent is amoxicillin). Nasal topical steroids reduce symptom duration. Topical decongestion (e.g., oxymetazoline) aids in symptomatic relief, but use *should not exceed 3 days,* as it may result in rebound congestion, that is, *rhinitis medicamentosa.* Intraorbital or intracranial infectious complications may rarely occur.

2. Chronic rhinosinusitis is diagnosed when the signs and symptoms of rhinosinusitis persist for more than 12 weeks. CT may show mucosal thickening, sinus opacification, obstruction of the osteomeatal complex, and/or anatomic/bony abnormalities. Treatment typically includes a combination of antibiotics, nasal saline irrigation, and topical nasal and/or oral steroids. Patients refractory to medical therapy benefit from **functional endoscopic sinus surgery (FESS).** The objectives of FESS are to reestablish the patency of the sinus ostia, ventilate the sinuses, and selectively remove diseased mucosa or polyps.

3. Fungal sinusitis

a. Noninvasive fungal sinusitis, typically associated with *Aspergillus* species, includes **mycetoma** (fungus ball) and allergic fungal sinusitis. It is treated with steroids, saline irrigations, and FESS.

b. Acute invasive fungal sinusitis is an aggressive, potentially fatal angioinvasion of fungal species that results in tissue death and is seen in immunocompromised patients. Common pathogens include *Aspergillus* in neutropenic patients, *Zygomycetes* in diabetic patients as well as *Rhizopus* and *Mucor* species. Management is IV antifungal therapy and surgical debridement of necrotic tissue.

C. Nasal Airway Obstruction

1. Adenoid hypertrophy. The adenoids are foci of lymphoid tissue present in the posterior nasopharynx that hypertrophy during childhood

and then atrophy with age. Adenoid hypertrophy can cause nasal obstruction, snoring, sleep-disordered breathing, and recurrent AOM.

2. Nasal polyposis. Nasal polyps are inflammatory, edematous, hyperplastic regions of nasal mucosa that often obstruct sinus drainage. Nasal polyps are usually treated with nasal or systemic steroids and surgical debulking.

3. Nasal septal deviation. Deviation of the septum results from nasal trauma or differential growth. Septal deviation can be corrected in conjunction with FESS if nasal obstruction and sinus disease are present.

D. Epistaxis. Nose bleeds are caused by trauma, neoplasm, environmental irritants, rhinitis, coagulopathies, and granulomatous diseases. Patients should be instructed to pinch the cartilaginous nose and lean forward to avoid swallowing blood. A vasoconstrictor such as oxymetazoline (Afrin) spray should be used and bleeding can be cauterized with silver nitrate or electrocautery if necessary. The nose can also be packed using epistaxis balloons, gauze, or absorbable hemostatic agents. Arterial embolization is reserved for refractory cases. Preventative measures involve moisturization via nasal saline spray, Vaseline application, and humidification of air.

E. Neoplasms

1. Benign

a. Inverted papilloma. The most common benign lesion is the inverted papilloma, a wart-like growth usually arising from the lateral nasal wall. It has a 5% to 10% incidence of malignant transformation into SCC (*Laryngoscope.* 2014;124:1981-1982). Wide local excision is necessary to prevent recurrence, which is very common.

b. Juvenile nasopharyngeal angiofibromas. These vascular tumors usually present in adolescent boys as nasal obstruction and recurrent epistaxis. Treatment is surgical excision (either open or endoscopic), with preoperative embolization to reduce blood loss. Bleeding and recurrence are both complications to be discussed with patients (*Laryngoscope.* 2013;123:859-869).

2. Malignant

a. Nasopharyngeal SCC. These tumors are most common in Asia and Africa, where it is often associated with EBV. Treatment is chemotherapy and radiation, with surgical resection reserved for residual disease.

CHAPTER 41: OTOLARYNGOLOGY FOR THE GENERAL SURGEON

Multiple Choice Questions

1. A 5-year-old, otherwise healthy male presents to clinic with a neck mass that appears to move vertically when he swallows. The mass is nontender and midline. What important diagnostic test/study should be performed before surgical intervention?

a. Fine needle aspiration biopsy

b. MRI of the head and neck

c. Ultrasound of the neck

d. CT of the neck and chest

e. EBV testing for mononucleosis

View Answer

2. A 45-year-old woman is found to have papillary thyroid cancer and undergoes a total thyroidectomy. Immediately following surgery in PACU, the patient complains of difficulty breathing. On examination, she is noted to have stridor and desaturations. An awake scope examination is performed in PACU, and it appears that both vocal cords are paralyzed. Which of the following options below would provide the best care for this patient?

a. Speech therapy

b. Oxygen via nasal cannula

c. Temporary vocal cord injection

d. Thyroplasty

e. Tracheostomy

View Answer

3. A 12-year-old female with a history of asthma presents to clinic with large bulky persistent cervical lymph nodes, despite a 2-week course of antibiotics. A fine needle aspiration (FNA) biopsy is suspicious for lymphoma. What is the neck best step in management and treatment for this patient?

a. Bilateral radical neck dissection

b. Selective neck dissection of affected nodes

c. Radiation without chemotherapy

d. Core or excisional biopsy for flow cytometry, to direct further treatment

e. Fine needle aspiration of a second lymph node to verify diagnosis

View Answer

4. A 40-year-old female, who was previously healthy, presents to clinic with a slowly growing, painless cheek mass, lying over her masseter muscle. You plan to perform a fine needle aspiration biopsy. What is the most common diagnosis for the parotid mass described here?

a. Adenoid cystic carcinoma

b. Adenocarcinoma

c. Pleomorphic adenoma

d. Squamous cell carcinoma

e. Warthin tumor

View Answer

5. A 25-year-old female presents with a slowly growing deep neck mass. She reports that she has been having episodes of lightheadedness with fainting and she remembers a family member having neck surgery in the past. The radiologist reviews her CT and notifies you that the mass appears to be splaying the internal and external carotid arteries, and you are considering a diagnosis of carotid body tumor. What is your next step in management?

a. Fine needle aspiration biopsy

b. Core or open biopsy to confirm diagnosis

c. Watch and wait

d. Surgical resection

e. Angiography with preoperative embolization

View Answer

6. A 50-year-old male with a history of intermittent smoking over the past 10 years and arthritis presents to clinic with a right-sided neck mass. Examination reveals asymmetry of his tonsils with bulkiness of the right tonsil. You suspect squamous cell carcinoma, as you know this commonly metastasizes to the neck. What is the next best step in management?

a. Core or open biopsy to confirm diagnosis

b. Watch and wait

c. Removal of right tonsil, and selective right neck dissection

d. Fine needle aspiration biopsy of the right neck node

e. Chemoradiation

View Answer

42
Plastic and Hand Surgery

Gwendolyn Hoben

Kamlesh B. Patel

Plastic surgery has no defined anatomic territory and thus is built on principles and techniques rather than specific procedures. In this chapter, we discuss topics pertinent to the general surgeon.

BASIC TECHNIQUES AND PRINCIPLES

I. THE RECONSTRUCTIVE LADDER.

When planning reconstruction, the simplest approach is often the best, but it is critical to know when a more complex solution is appropriate.

A. Healing by Secondary Intention. Dressings should be utilized to maintain a clean protected wound. Contraindications include exposed critical structures, prolonged (>3 weeks) anticipated period of healing, undesirable aesthetic consequences. **Negative pressure wound therapy (NPWT)** can be used to accelerate the healing process; contraindications include the presence of malignancy, ischemic wounds, infected wounds, or inadequately debrided tissue beds.

B. Primary closure will often have best aesthetic result but care must be taken to eliminate tension and prevent displacement of neighboring structures (e.g., lower eyelid) or necrosis of the skin flaps.

C. Skin grafting requires a healthy, vascularized, uninfected wound bed and an available donor site. Bare tendon, desiccated bone or cartilage, radiation-damaged tissue, or infected wounds will not support skin graft survival. **NPWT** may be used to promote granulation tissue and create a graft-able wound.

D. Local tissue transfers of skin, fascia, and muscle may be used in regions with nearby healthy tissue.

E. Distant tissue transfers move healthy tissue into the wound bed while leaving it attached to its native blood supply and requires subsequent division of the pedicle. These are rarely used as they require long-term immobilization and multiple procedures.

F. Free tissue transfer requires microvascular anastomosis but may allow single-stage wound closure and often an acceptable aesthetic outcome.

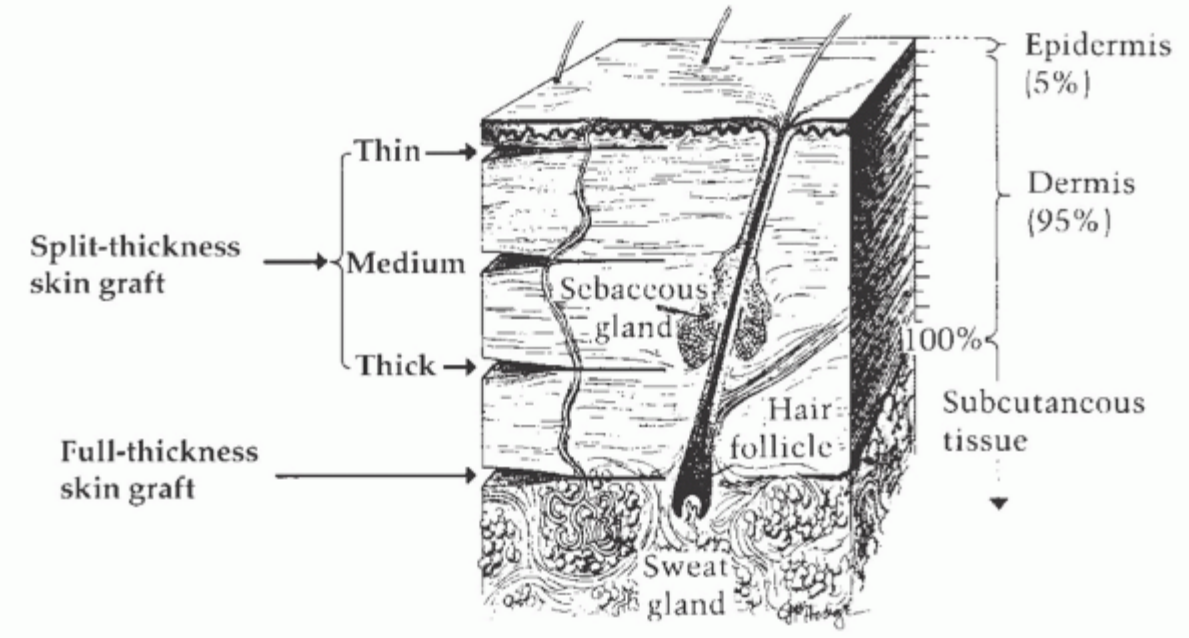


Figure 42-1 Skin Graft Thickness. (From Thorne CH, et al., eds. *Grabb and Smith's Plastic Surgery,* 7th ed. Philadelphia, PA: Wolters Kluwer Health, 2014.)

II. TYPES OF GRAFTS

A. Skin Grafts (Fig. 42-1)

1. Split-thickness grafts (STSGs) include epidermis and a portion of dermis.

a. 20% primary contracture, 40% secondary contracture (thicker grafts will have reduced contracture)

b. Donor sites: Thigh, buttock, and scalp

(1) Heals by secondary intention

(2) Use a moist occlusive dressing to reduce pain and facilitate healing

c. Meshing can be used to increase coverage surface area and reduce risk of fluid accumulation (a ratio of 1.5:1 is commonly used)

d. Relative contraindications: Covering a joint, aesthetically demanding location

2. Full-thickness grafts include epidermis and dermis.

a. >40% primary contraction, ÷0% secondary contraction

b. Donor sites: Groin, postauricular, supraclavicular, abdomen

(1) Heal by primary closure or split thickness grafting

c. Critical to de-fat the graft to allow for optimal take

d. Cutting slits in graft to allow fluid egress and bolstering may reduce the risk of graft loss

3. Graft healing

a. Days 1 and 2: **Imbibition** (diffusion of nutrients from the wound bed)

b. Days 3 and 5: **Inosculation** (graft and wound bed capillaries begin to align)

c. Day 4: Graft well anchored by collagen (earliest time at which bolster should be removed)

d. >5 days: Graft revascularized by capillary ingrowth

e. 6 weeks: 90% of maximal tensile strength

4. Graft failures are most often the result of hematoma, seroma, infection, or shearing.

B. Other Graft Sources

1. Tendon grafts. Palmaris longus, plantaris tendons.

2. Bone grafts

a. Cancellous: Iliac crest

b. Cortical: Ribs, outer table of cranium, fibula

3. Cartilage grafts. Costal cartilage, concha of ear, nasal septum.

4. Nerve grafts. Sural nerve, lateral or medial antebrachial cutaneous nerves. Cadaveric allograft also available.

5. Fat grafts. Liposuction of abdomen, thigh, buttocks.

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 are used to cover adjacent defects.

a. Blood supply from the dermal and subdermal plexus

b. Length-to-width ratio (usually 3:1), varies by anatomic region (e.g., the face has a ratio of up to 5:1).

c. Flaps that rotate about a pivot point include **rotation flaps** (Fig. 42-2) and **transposition flaps** (Fig. 42-3).

(1) The effective length shortens through the arc of rotation

(2) The further from the base of the flap, the greater the risk of necrosis

(3) More complex rotation flaps: **Bilobed flaps** (Fig. 42-4) and **rhomboid flaps** (Fig. 42-5).

d. Advancement of skin directly into a defect without rotation

(1) Simple advancement

(2) V-Y advancement (Fig. 42-6)

(3) Bipedicle advancement flap.

2. Axial cutaneous flaps contain a single dominant arteriovenous system, allowing for a potentially greater length-to-width ratio.

a. Peninsular flap. Skin and vessels are moved together as a unit.

b. Island flaps. Skin is divided from surrounding tissue and maintained on an isolated, intact vascular pedicle.

c. Free flaps. Vascular pedicle is isolated and divided.

B. Specialized Flaps

1. Fascial/fasciocutaneous flaps are used when thin, well-vascularized coverage is needed (e.g., dorsum of the hand or foot).

a. Fascia: Temporoparietal fascia.

b. Fasciocutaneous flaps: Radial forearm, anterolateral thigh, lateral arm, and groin flaps.

2. Vascularized bone flaps are used for critical bony defects >8 cm.

a. Donors: Fibula, scapular spine, iliac (with overlying internal oblique muscle), and rib (with pectoralis major or intercostal muscle).

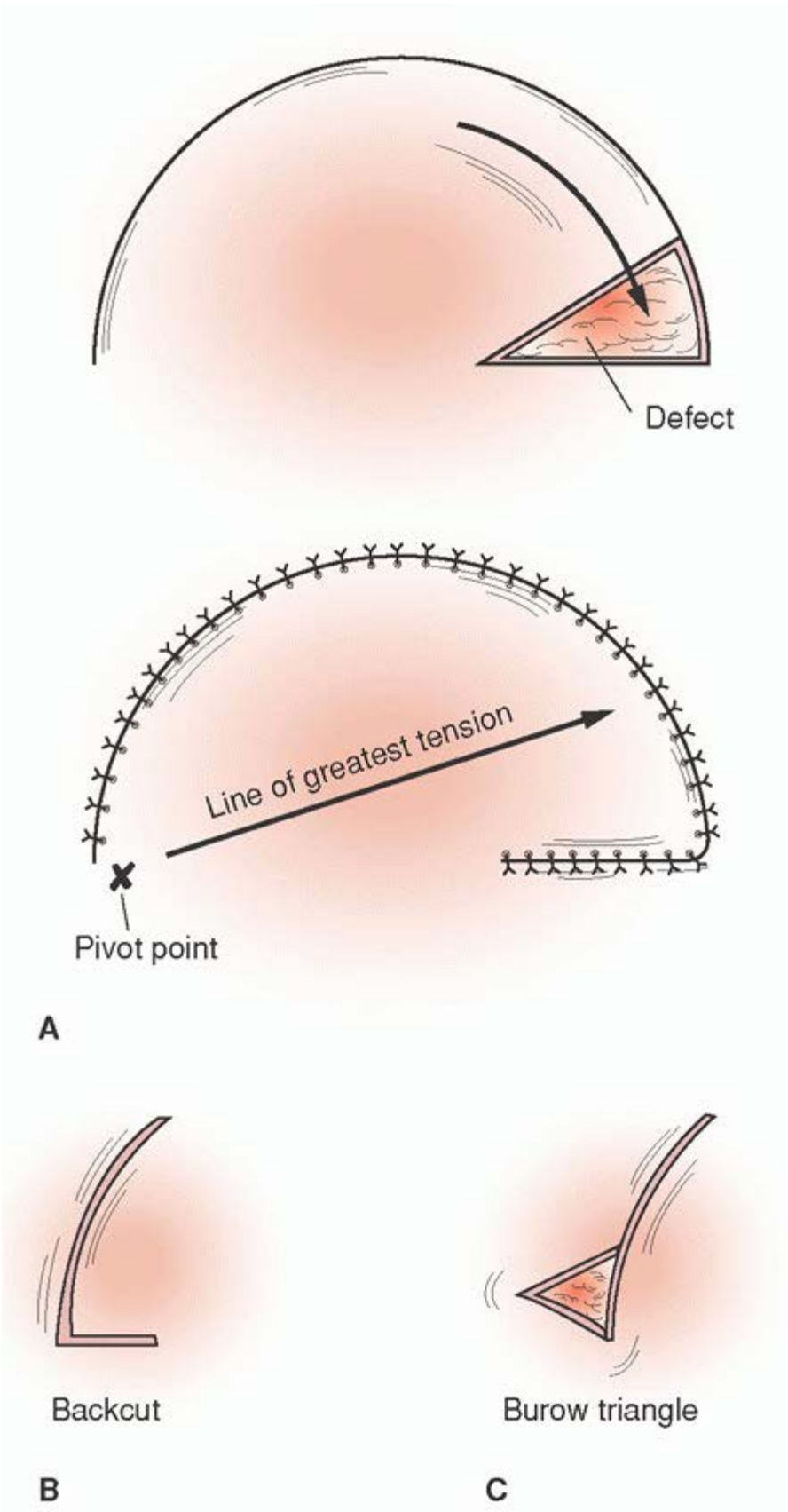


Figure 42-2 Rotation flap. **A:** The edge of the flap is four to five times the length of the base of the defect triangle. **B, C:** A backcut or Burow triangle can be useful if the flap is under tension.

3. Functional muscle may be transferred with its accompanying dominant nerve.

a. Donors. Gracilis, latissimus dorsi.

IV. TISSUE EXPANSION.

Tissue expansion uses an inflatable silicone balloon to serially expand surrounding skin. During expansion, the dermis thins, collagen re-aligns, and the epidermis thickens. Common indications include burn alopecia, congenital nevi, and postmastectomy breast reconstruction.

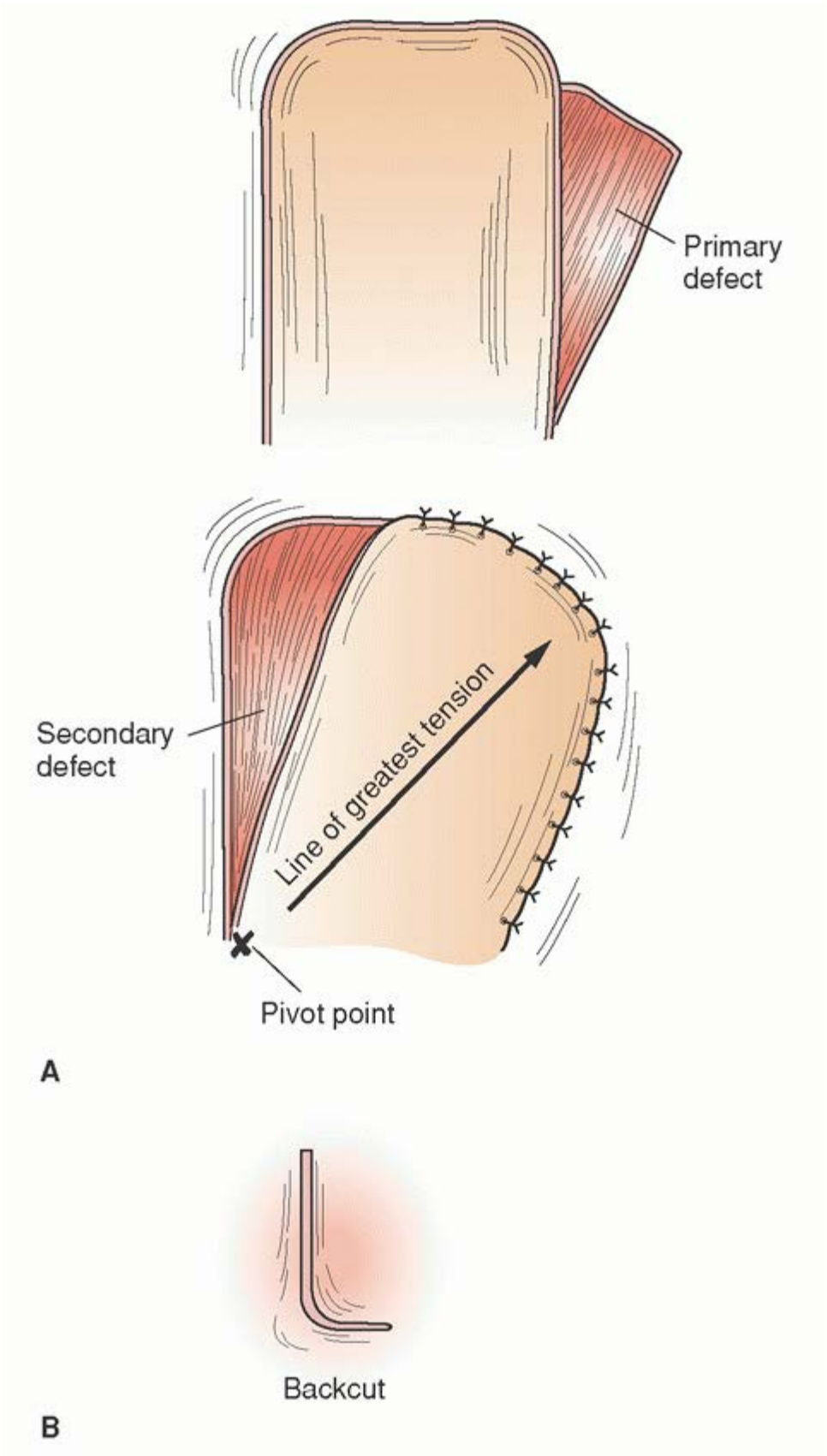


Figure 42-3 A: Transposition flap (more complex rotation flap that creates a defect that must be closed). The secondary defect is typically covered with a skin graft. **B:** A backcut may be added to reduce tension at the pivot point.

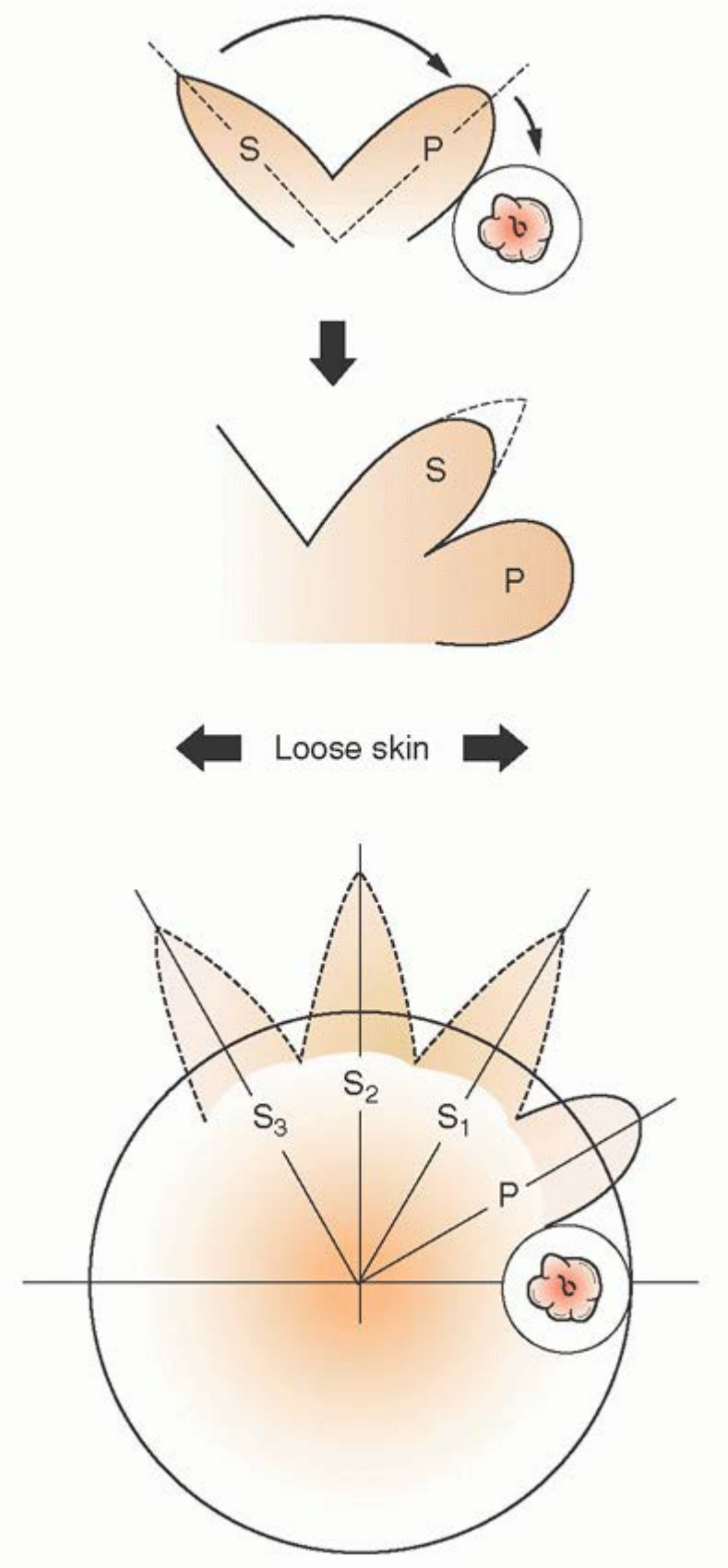


Figure 42-4 Bilobed flap. After the lesion is excised, the primary flap (P) is transposed into the initial defect, and the secondary flap (S) is moved to the site vacated by the primary flap. The bed of the secondary flap is then closed primarily. The primary flap is slightly narrower than the initial 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 (S_1, S_2, S_3).

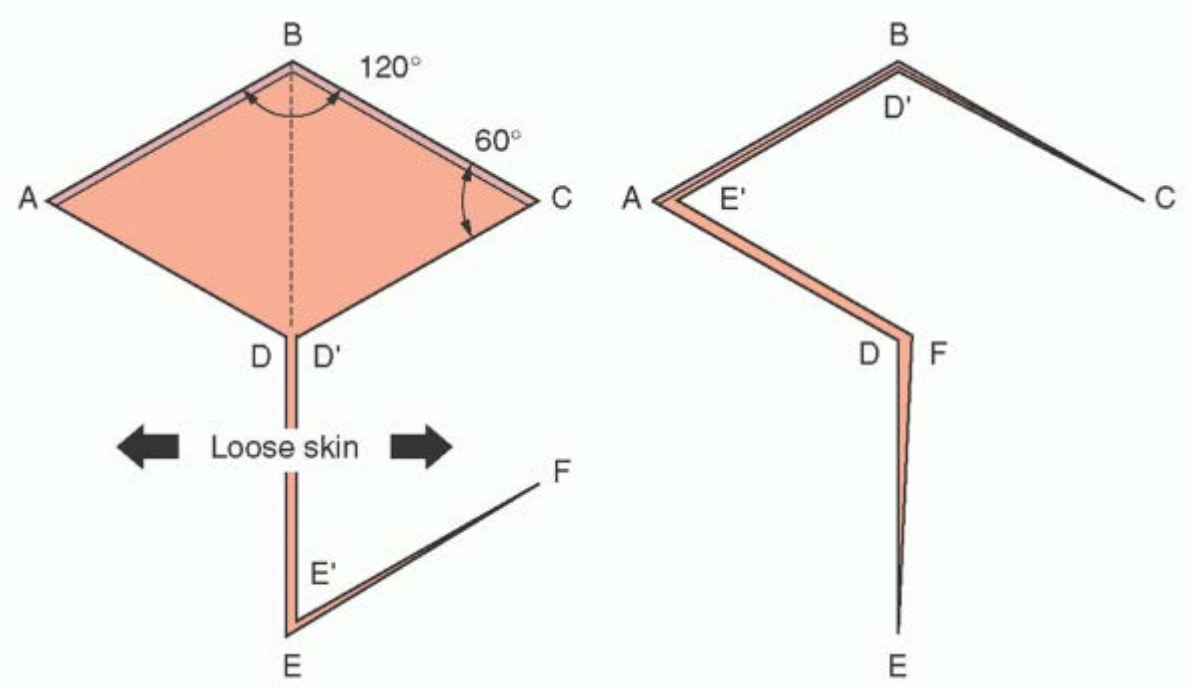


Figure 42-5 Rhomboid or Limberg. The rhomboid defect must have 60- and 120-degree angles so that the length of the short diagonal is the same as the length of the sides. The short diagonal is extended by its own length to point E. The line EF is parallel to CD, and they are equal in length. There are 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. The greatest tension will be between points F and D.

A. Advantages. Low donor-site morbidity, provision of donor tissue of similar color, texture, thickness, and sensation to the recipient tissue (like for like).

B. Disadvantages. Staged technique, there is a visible deformity during the period of expansion, frequent visits for expansion, and a relatively high rate of complications (infection, extrusion).

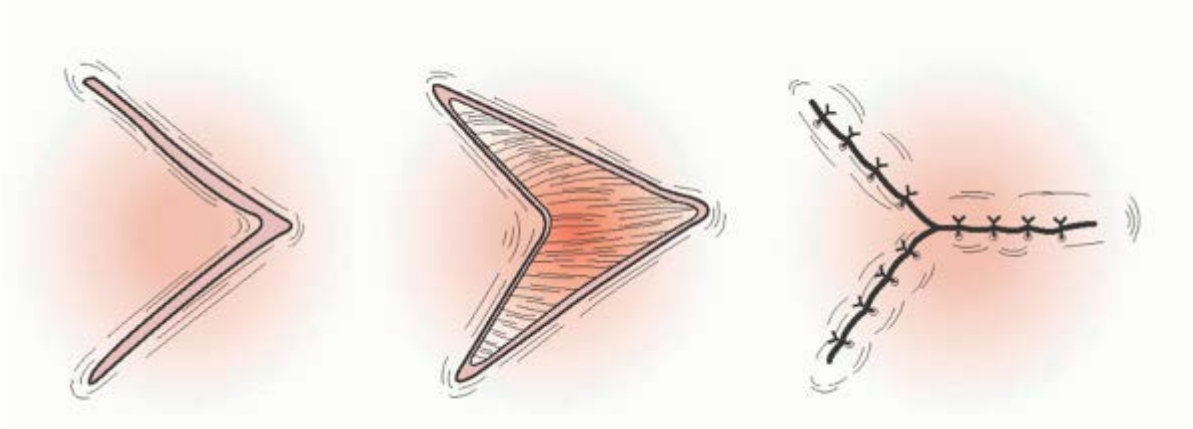


Figure 42-6 V-Y advancement. The skin to the sides of the V is advanced.

C. Technique Pearls

1. Goal of expanded surface area is 130% of the defect size.

2. Expander placement should be through an incision at the junction of the defect and the area of proposed expansion, placed perpendicular to the direction of expansion.

3. Ports

a. External ports: Minimize anxiety and pain during filling, especially in the pediatric population.

b. Internal ports: Associated with reduced infection risk, commonly used when overlying skin is insensate.

4. The expansion phase begins 2 to 3 weeks after expander placement.

a. Inflate weekly/bi-weekly using saline and sterile technique.

b. Amount infused depends on patient comfort, skin tension, and blanching of overlying skin.

c. Waiting 2 to 3 weeks after the desired volume is achieved allows the expanded skin to soften and decreases the contraction at the time of flap transposition.

ACUTE INJURIES

I. FACIAL TRAUMA

A. Examination Pearls

1. C-spine. 10% of facial trauma patients have associated c-spine injury.

2. Oral examination. Malocclusion or limited mouth opening can indicate fractures; assess for loose teeth that are at risk for aspiration.

3. Facial nerve. Assess the five branches (temporal, zygomatic, buccal, marginal mandibular, cervical) by having the patient raise the eyebrows, squeeze the eyelids shut, smile, and frown, noting any paresis or asymmetry. Facial nerve injuries located lateral to the lateral canthus should be explored and repaired within 72 hours; more medial injuries can be managed expectantly as the facial nerve is highly arborized in the medial face.

4. Ocular examination

a. Assess extra-ocular movements; limited up-gaze may **indicate inferior rectus entrapment** in an orbital fracture (acute indication for surgery).

b. Orbital compartment syndrome. Results from retrobulbar hematoma and requires emergent lateral cantholysis. Look for proptosis, acute vision loss, and severe eye pain.

5. Nasal examination. Examine the nasal septum with an otoscope to assess for septal hematoma. Acute evacuation of the hematoma will prevent permanent deformity.

B. Imaging

1. CT with <3 mm cuts through the facial bones is a fast, sensitive, and specific means of determining the location and orientation of facial fractures.

2. Panorex radiograph is useful in the setting of isolated mandible fractures or dental injuries.

C. Facial Hemorrhage. Nasal hemorrhage will generally cease with packing. Massive hemorrhage that does not stop with appropriate pressure is likely a branch of the external carotid and will require embolization.

D. Soft-tissue Repair

1. Local anesthesia may be used to block facial nerve branches

2. Copiously irrigate, debride devitalized tissue

3. Deep dermal closure: Interrupted resorbable suture

4. Epidermal closure: Running suture, generally better scarring with permanent monofilament

E. Fractures

1. Most can be repaired electively within 1 to 2 weeks

2. Exceptions: Airway compromise, ocular muscle entrapment

3. Indications for fixation

a. Mandible. Malocclusion

b. Orbital. Persistent diplopia, enophthalmos, extraocular muscle entrapment, hypoglobus

c. Midface. Cosmetic deformity, trismus/malocclusion

d. Nasal fracture. Cosmetic deformity, septal hematoma, airway obstruction

II. HAND TRAUMA

A. Assessment Pearls

1. History. Hand dominance and patient occupation

2. Examination

a. Inspect the resting position of the patient's hand looking for swelling or asymmetry compared with the contralateral hand.

b. Vascular assessment. Color, temperature, capillary refill, and the presence of pulses at the wrist and individual digits (palpable or Doppler)

(1) Allen test. Verifies integrity of the palmar arches. Have the patient make a fist, manually occlude the radial and ulnar arteries by applying pressure at the wrist, have the patient open their hand and observe the color. Remove pressure from the radial artery: Color will return to the hand if the superficial arch is intact. Repeat with the ulnar artery to assess the deep arch.

(2) Control bleeding with direct pressure. Tourniquets should be reserved for life-threatening exsanguinations only and should be placed as distally as possible.

c. Motor examination

(1) 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.

(2) Flexor digitorum superficialis (FDS) is tested by stabilizing all other fingers in full extension and asking the patient to flex the finger being tested at the PIP joint.

(3) Extensor tendons. Place hand on a flat surface, palm down. Have patient lift each finger off the surface individually.

TABLE 42-1 Unambiguous Tests of Hand Nerve Function

Test	Radial Nerve	Median Nerve	Ulnar Nerve

Sensory	Dorsum first web	Index fingertip	Little fingertip
Extrinsic motor	Extend wrist	FDP index	FDP small
Intrinsic motor	None	Abduct thumb perpendicular to palm	Cross long finger over index (interossei)

FDP, flexor digitorum profundus.

d. Sensory testing includes gross examination of the ulnar, radial, and median nerves (Table 42-1).

(1) Two-point discrimination. Test on the palmar aspect of both the radial and ulnar sides of the digits and compare to the uninjured hand. A normal examination is feeling two points ²5 mm apart.

(2) Strauch 10-10 test. The patient rates the level of light touch sensation in an injured area on a scale of 0 to 10 (0 = no sensation, 10 = normal sensation) as compared to an uninjured area. (Note: The original scale was 1 to 10, but we find patients better understand 0 = no sensation.)

e. Skeletal examination involves palpating for any tenderness, soft-tissue swelling, or deformity of the bones. Joint integrity is assessed by gently stressing the ligaments and noting any instability, crepitus, or pain.

3. Diagnostic radiology

a. If **fracture/dislocation** suspected, obtain at least three views (posteroanterior, lateral, oblique) including the joint above and below the suspected fracture/dislocation.

b. Penetrating trauma or **abscess.** Use plain films to assess for retained foreign bodies such as needles.

B. Fractures

1. Principles of management

a. The description of the fracture pattern must include: The bone(s) involved, open versus closed, simple versus comminuted, displaced versus nondisplaced, transverse versus oblique versus spiral, angulation, or rotation of the distal fragment, and intra-articular versus extra-articular

b. Postreduction radiographs should be done for all fractures after splinting or casting.

c. Splinting the fracture

(1) Use plaster or fiberglass with appropriate padding; in the acute situation, the splint should not be circumferential to allow for swelling.

(2) Positioning: Ò**Intrinsic-plus**Ó position may be used for almost all hand injuries. It places ligaments in their longest position. The interphalangeal (IP) joints are in full extension, the metacarpophalangeal (MCP) joints are at 60 to 90 degrees of flexion, and the wrist is in 20 to 30 degrees of extension. The thumb should be abducted with the MCP extended.

(3) The splint should stabilize the joints above and below the fracture while avoiding immobilization of the remainder of the patient's hand and wrist.

d. Indications for operative intervention. Reduction is not maintained after closed reduction, contaminated open fractures, associated soft-tissue injuries, malalignment (uncorrected rotated, angulated, or shortened deformities of the digit), and articular incongruity >1 mm.

e. Operative repair

(1) Closed-reduction, percutaneous pinning (CRPP) is used when a fracture is closed but reduction is inadequate or unstable. Reduction is performed under anesthesia with the aid of fluoroscopy. Kirschner wires (K-wires) are placed to hold the reduction.

(2) Open reduction-internal fixation (ORIF). An incision is made over the fracture and plates/screws are used to hold the reduction. Although more invasive, fixation is rigid and allows for early active motion and passive range of motion, thus reducing stiffness.

(3) External fixation is used when significant contamination or soft tissue loss does not allow for safe placement of permanent hardware.

C. Dislocations and ligament injuries should be assessed by stressing the periarticular structures and ranging the joint. If instability is demonstrated, operative management should be considered. A stable joint is managed with protective splinting and early range-of-motion exercise. Perform neurovascular exam before and after manipulation.

D. Tendon Injuries

1. Assessment. Look for a change in the resting tone of the digits, which normally lie in a radial-to-ulnar cascade of flexion. Assess both flexor tendons and extensor tendons separately. If movement against resistance elicits pain, suspect a partial laceration.

2. Flexor tendons are frequently lacerated during everyday activities.

a. Flexor tendon zones (Fig. 42-7)

(1) Zone I. At the DIP level, distal to the FDS insertion.

(2) Zone II. From proximal A1 pulley (MCP joint) to FDS insertion. Injuries in this zone may affect both FDS and FDP.

(3) Zone III. From distal transverse carpal ligament (carpal tunnel) to A1 pulley. Look for associated palmar arch or median/ulnar nerve injury.

(4) Zone IV. Within the carpal tunnel, look for median nerve injury.

(5) Zone V. Proximal to the carpal tunnel.

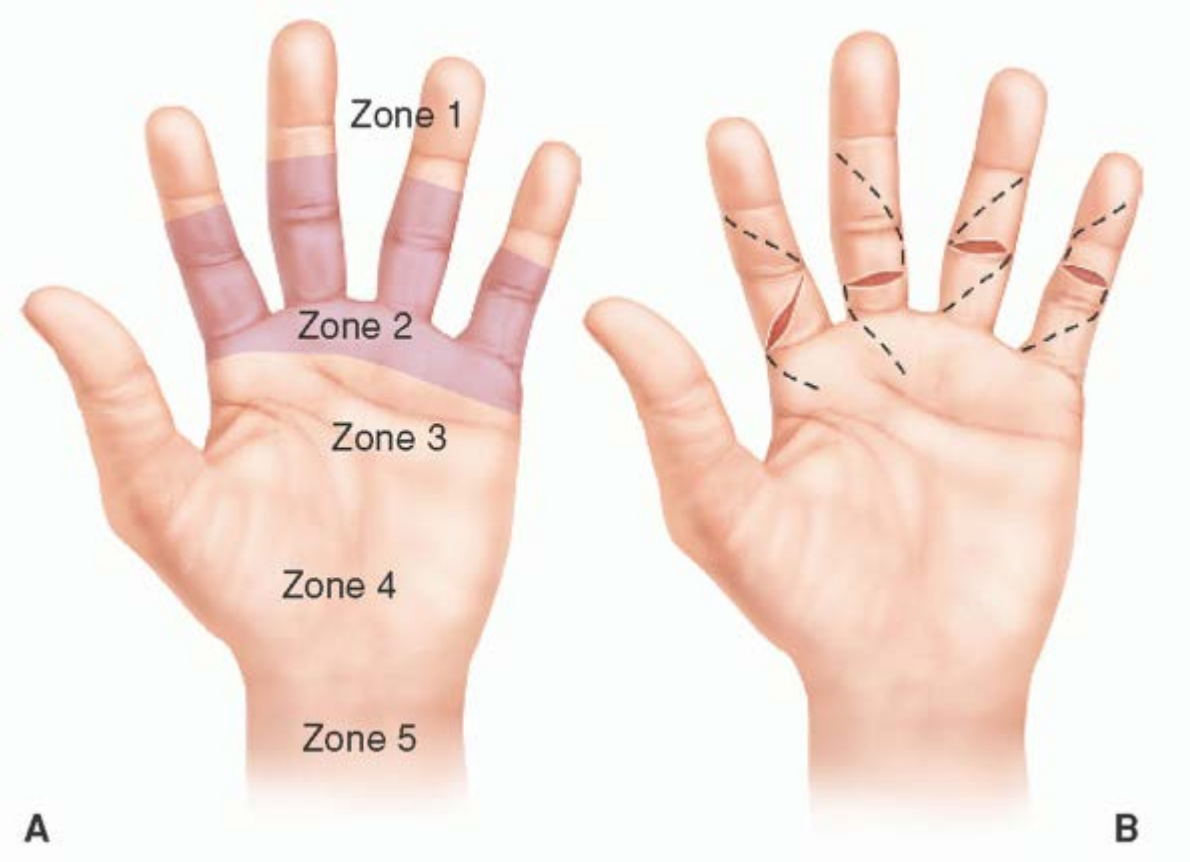


Figure 42-7 Zones of flexor tendon injury. **A:** Distal to the flexor superficialis insertion (zone 1), within the digital sheath of the flexor superficialis and profundus (zone 2), palm (zone 3), within carpal tunnel (zone 4), and in the forearm proximal to the carpal tunnel (zone 5). In general, flexor tendons repaired in zones 1, 3, 4, and 5 have a better prognosis than those in zone 2, known as Òno man's land.Ó **B:** Brunner zigzag extensions to optimize exposure of the proximal and distal ends of the flexor tendon. (From Thorne CH, et al., eds. *Grabb and Smith's Plastic Surgery,* 7th ed. Philadelphia, PA: Wolters Kluwer Health, 2014.)

b. Acute management. The proximal end will commonly retract preventing acute repair. Wound should be irrigated and closed with dorsal splinting with 20 to 30 degrees of wrist flexion,

90-degree MCP flexion, and IP joints in extension. Repair within 10 days to avoid the need for tendon grafting.

c. Definitive repair involves a core, locking suture and an epitendinous repair. Injuries in zone II to IV have a worse prognosis because of scarring and reduced tendon glide within the sheath.

3. Extensor tendon injuries result from lacerations and closed axial loading of the digits.

a. Extensor tendon zones

(1) Zone I. Over the DIP joint. Rupture of the terminal tendon secondary to forced flexion results in **Mallet finger,** or inability to extend the DIP.

(2) Zone II. Over the middle phalanx.

(3) Zone III. Over the PIP joint. May injure the central slip and/or lateral bands and if untreated, can result in a **boutonnière deformity** (PIP flexion and DIP hyperextension).

(4) Zone IV. Over the proximal phalanx. The lacerations are often partial because of the width of the tendon at this level.

(5) Zone V. Over the MCP joint. Carefully explore to assess for sagittal band injury at this level as this must be repaired to prevent tendon subluxation. These injuries can occur secondary to delivering a punch to the mouth (see Section **Local infections** > **Human bites/fight bites).**

b. Management. Can be repaired acutely as the tendons generally do not retract as much as flexors secondary to the juncturae tendinum, which are the narrow, fibrous bands of tissue that pass obliquely between the diverging tendons of the extensor digitorum on the dorsal hand. Mallet injuries can be treated in a closed fashion with extension splinting of the DIP for 6 weeks.

4. Local infections

a. Management. Most finger infections heal well if all the purulence is drained and the wound is irrigated 2 to 3 times daily with soapy water and allowed to heal from the inside out. Splinting for comfort and elevation to reduce edema are important adjuncts.

(1) Antibiotics may be needed if there is associated cellulitis, in the case of human/animal bites, and if there is involvement of the tendon sheath or joint space.

(2) Bites and joint injuries may require multiple washouts in the OR.

(3) Always obtain plain films prior to exploration to assess for foreign bodies (i.e., needles).

b. Paronychia is a localized infection of the skin and lateral nail fold, often due to nail biting or foreign-body penetration, such as a needlestick injury. Treatment requires incision and drainage (I&D), with removal of the nail when the infection extends deep to the nail plate. **Chronic paronychia** is sometimes associated with underlying osteomyelitis or fungal organisms (Fig. 42-

8).

c. Felon is a local infection of the finger pulp commonly due to a puncture wound. Adequate drainage requires dividing all involved septae.

d. Cellulitis in the hand usually arises secondary to a laceration, abrasion, or other soft-tissue injury. Management involves draining an abscess, if present, and treating with antibiotics.

e. In **animal bites,** 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.

f. Human bites/fight bites are most commonly associated with a punch to the mouth. Aggressive wound exploration, generally in the operating room, must be undertaken as the skin will retract proximally and cover a deeper wound; 75% of fight bites result in bone/tendon/cartilage injury. Treat with intravenous (IV) antibiotics acutely. May require multiple washouts.

E. Surgical Emergencies

1. Compartment syndrome in the hand and forearm results from increased pressure within an osseofascial space, leading to decreased

perfusion pressure. Left untreated, it will result in muscle and nerve ischemia and necrosis.

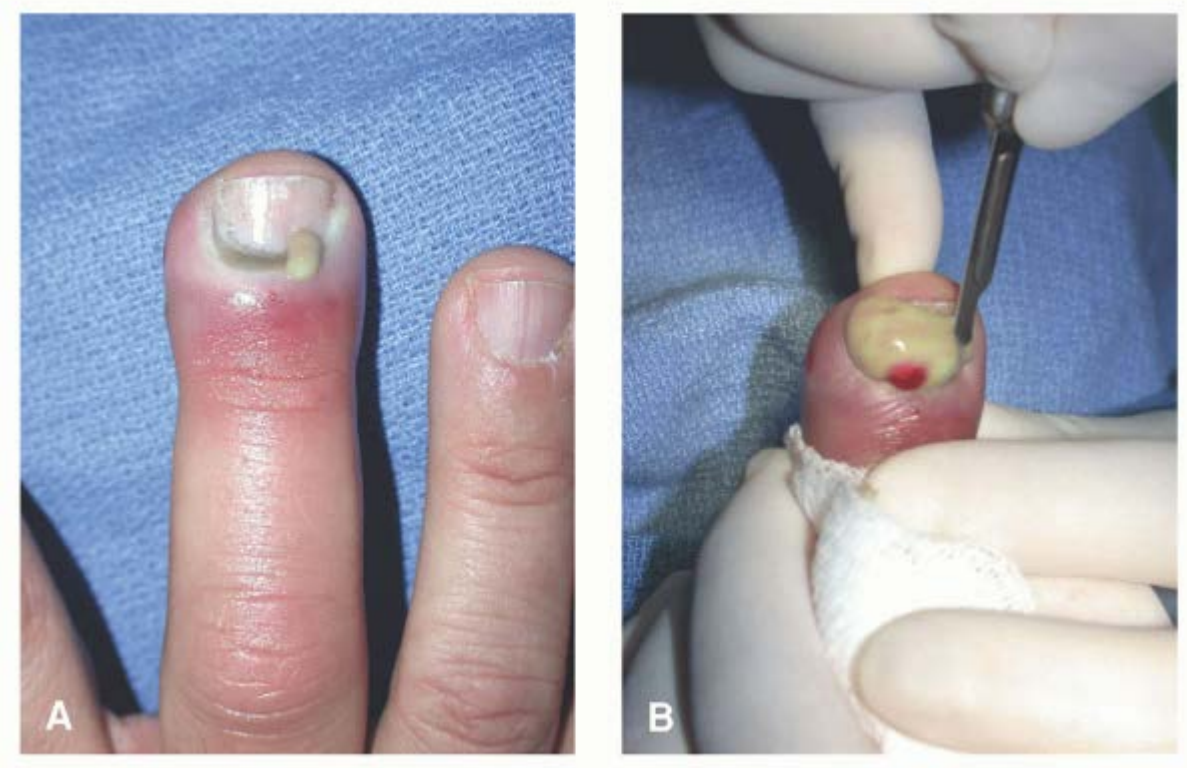


Figure 42-8 Acute paronychia. **A:** As seen in the emergency room. **B:** Incision is made

through the most fluctuant region. (From Thorne CH, et al., eds. *Grabb and Smith's Plastic Surgery,* 7th ed. Philadelphia, PA: Wolters Kluwer Health, 2014.)

a. Etiology. Fractures that cause bleeding, crush and vascular injuries, circumferential burns, bleeding dyscrasias, reperfusion after ischemia, or tight dressings.

b. Diagnosis. It is critical to have a **high index of suspicion** based on the type of injury. Pain with passive stretch of the compartment musculature and paresthesias are early indicators and should be followed up aggressively. Paralysis and pulselessness occur later once the damage is likely irreversible. Measurement with a pressure monitor of a **compartment pressure >30 mm Hg** confirms diagnosis.

c. Treatment of incipient compartment syndrome involves close observation, frequent examinations, removal of tight casts and dressings, and elevation of the extremity above the level of the heart. Acute suspected compartment syndrome requires urgent fasciotomies within 6 hours to prevent irreversible muscle ischemia.

2. Suppurative tenosynovitis is infection of the flexor tendon sheath, usually due to a puncture wound to the volar aspect of the digit or palm.

a. Diagnosis: Cardinal signs of Kanavel

(1) Finger held in flexion

(2) Fusiform swelling of the finger

(3) Tenderness along the tendon sheath

(4) Pain on passive extension

b. Management involves urgent I&D in the operating room and IV antibiotics.

3. 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.

4. Necrotizing infections threaten both limb and life. Aggressive and repeated surgical debridement, broad spectrum antibiotics, and supportive management are the mainstays of treatment.

5. High-pressure injection injuries result from grease or paint injected at up to 10,000 lb/in^2. Although the external wounds are often small and unassuming, deep-tissue injury can be severe. Management involves urgent, thorough debridement, irrigation, decompression, systemic antibiotics, and splinting.

GENERAL RECONSTRUCTIVE PLASTIC SURGERY

I. SCALP, CALVARIAL, AND FOREHEAD RECONSTRUCTION

A. Scalp Layers. Skin, subcutaneous tissue, galea aponeurotica, loose areolar tissue, and pericranium.

B. Scalp Lacerations

1. Often associated with blunt head trauma: Assess for associated skull, cervical spine, or intracranial injuries.

2. The rich blood supply to the scalp can produce significant blood loss so expedient management is critical. Hemostasis is achieved with closure of the galea and skin following thorough irrigation and debridement of devitalized tissue. Stapled closure of the hair-bearing scalp is expedient and results in reduced trauma to hair follicles compared to sutures.

C. Partial-thickness scalp loss from avulsion usually occurs at the subaponeurotic layer. Large avulsions may be skin grafted acutely. Definitive management often requires tissue expander placement to replace hair-bearing scalp.

D. 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.

2. Medium-sized defects (3 to 10 cm) can be covered with a skin graft or skin substitute (e.g., Integra) by burring the bone down to the vascular diploic space or using a scalp flap combined with skin grafting of the donor pericranium. Tissue expansion can later be used to replace hair-bearing scalp.

3. Large defects (>10 cm) often require free tissue transfer with omentum or latissimus dorsi (Fig. 42-9).

II. TRUNK

A. Breast

1. Postmastectomy breast reconstruction is an important part of recovery for many women and can lead to a significant improvement in body image (*J Natl Cancer Inst.* 2000;92:1422-1429).

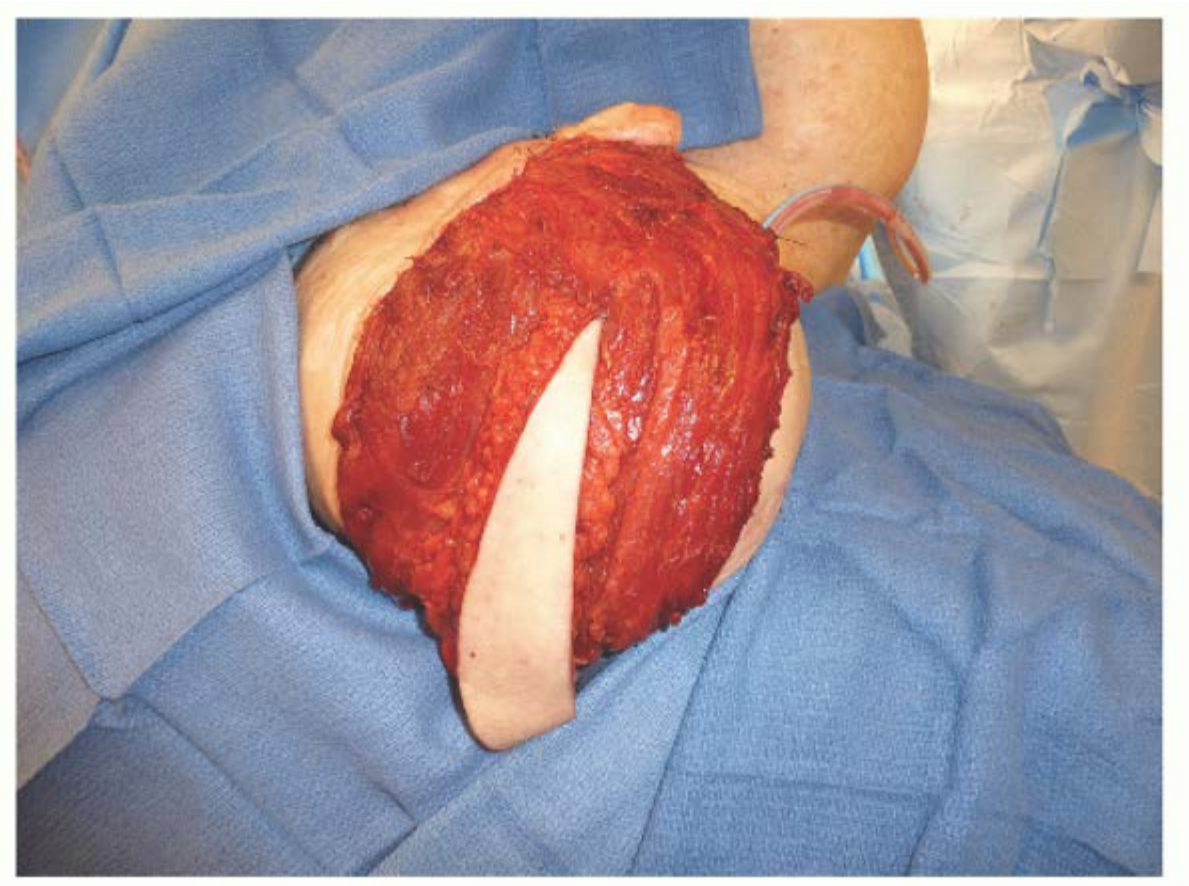


Figure 42-9 Myocutaneous latissimus free flap for scalp coverage. Although skin grafting of the muscle is still required, the skin island provides extra coverage and an area for improved monitoring postoperatively. (From Thorne CH, et al., eds. *Grabb and Smith's Plastic Surgery,* 7th ed. Philadelphia, PA: Wolters Kluwer Health, 2014.)

a. The aims of reconstruction are to create symmetric breast mounds and, if desired, a new nippleÑareola complex. 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.

b. Implant-based reconstruction is the most common form of breast reconstruction. Tissue expansion is generally required because the mastectomy flaps are insufficient for the desired size of the breast. A **tissue expander** is placed at the time of mastectomy and serial expansions performed until the desired size is reached. The expander is then replaced with a silicone gel or saline-filled permanent implant.

(1) Advantages: Minimal additional operative time, fewer additional scars, and a shorter recovery.

(2) Disadvantages: The risks of permanent implants (rupture, infection) and the inability to

reproduce certain natural contours.

(3) Anaplastic large cell lymphoma (ALCL) is an extremely rare malignancy associated with implants (<1/1,000,000). Most commonly found in association with a late-onset seroma. Diagnosis is via seroma fluid cytology.

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.**

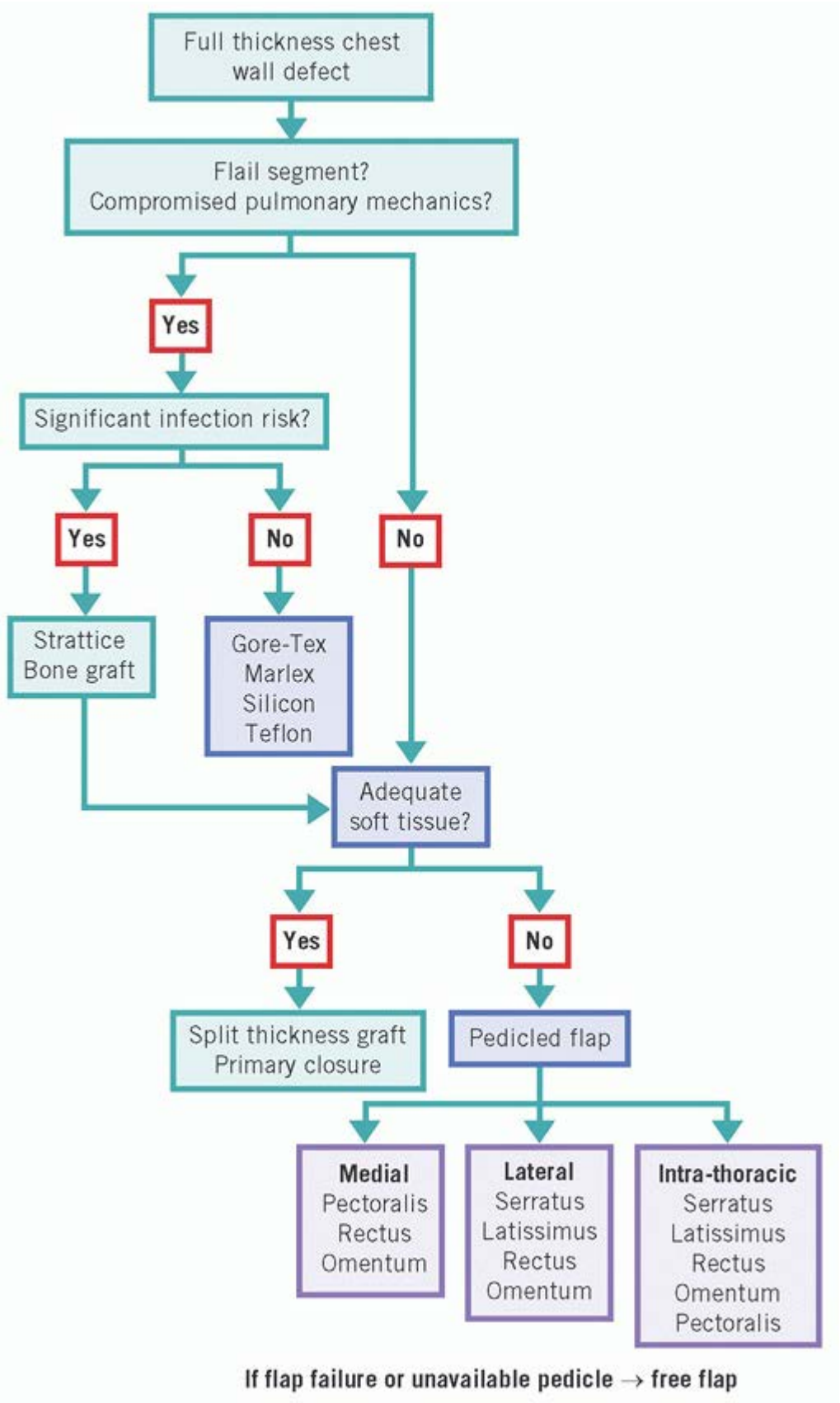


Figure 42-10 Chest wall reconstruction algorithm.

(1) Advantages: A more natural appearance for some patients, permanent reconstruction without the need for future procedures, and fewer complications with subsequent radiation therapy.

(2) Disadvantages: A relatively long procedure, additional scars, and potential donor-site morbidity.

d. Reconstruction of the nipple-areola complex is most commonly done via local flaps. Tattooing can be used to recreate an areola.

e. Symmetry procedures may be performed concomitantly or subsequently and include modification of the inframammary fold, removal of dog ears, liposuction of flaps, or reduction mammoplasty or mastopexy of the contralateral side. Symmetry procedures are almost always covered by insurance.

B. Chest Wall Reconstruction

1. Indications

a. Obliteration of dead space (e.g., closure of Clagett window, sternotomy dehiscence, obliteration of pocket from left ventricular assist device [LVAD])

b. Provision of vascularized tissue around an intrathoracic anastomosis

c. Restore chest wall integrity

2. Preoperative assessment

a. Available vascular pedicles must be assessed. Prior use of the internal mammary artery for a bypass precludes using an ipsilateral rectus abdominis flap or pectoralis turnover flap.

b. The chest wall defect must be completely clear of neoplasm, radiation damage, and infected tissue/hardware (i.e., sternotomy wires). NPWT can be used to bridge to definitive coverage.

3. Flaps are chosen based on location.

4. Skeletal stabilization is required if more than four rib segments or 5 cm of chest wall are missing. This can be achieved using autologous or prosthetic material.

C. Abdominal Wall Reconstruction

1. Goals: Fascial and cutaneous coverage to recreate a competent abdominal wall

2. Primary closure of fascial defects represents the best approach and can be assisted by sliding myofascial advancement flaps. Lateral release of the external oblique fascia, or Ò**component separation,**Ó is ideal for midline musculofascial defects >3 cm. Using bilateral relaxing incisions and release, a total of 10, 18, and 6 to 10 cm of advancement may be obtained in the upper, middle, and lower thirds of the abdomen, respectively (*Plast Recon Surg.* 1990;86:519). The anterior sheath of one or both rectus muscles can be divided and turned over to provide additional fascia for closure.

3. Mesh reinforcement/bridging can be used to strengthen a fascial closure or provide closure when component separation is inadequate. Synthetic meshes have the advantage of being resistant to stretch and are generally associated with lower recurrence rates. Biologic meshes should

be considered in the case of contaminated abdominal closure because they will revascularize. However, most available biologic meshes stretch significantly.

4. Skin coverage. STSGs can be used over vascularized tissue. Coverage of synthetic mesh can be achieved using abdominal advancement flaps or grafting of muscle flaps.

5. More challenging cases may require myofascial or myocutaneous flaps harvested from the anterolateral thigh, tensor fascia lata, or rectus femoris.

D. Pressure Sores

1. The **etiology and staging criteria** of pressure ulcers are described in **Chapter 9**, **Wound Care.** Stage I/II ulcers can generally be managed with wound care, while stage III/IV ulcers may benefit from definitive coverage.

2. A pressure sore is considered ready for definitive coverage when it is clean and infection-free, all devitalized tissue has been debrided, and associated osteomyelitis has been treated. It is critical that the patient is nutritionally optimized, any underlying conditions predisposing to pressure sores (i.e., spasticity) have been addressed, and that the patient has appropriate resources to prevent further ulcers or recurrence (i.e., pressure-relieving mattress, position-change regimen, etc.). **Diverting colostomy,** as part of the treatment strategy is controversial; in select patients, laparoscopic colostomy has been shown to reduce recurrence (*Dis Colon Rectum.* 2003;46:1525).

3. Commonly utilized flaps. Gluteus maximus, tensor fascia lata, hamstring, or gracilis-based rotation or advancement flaps.

4. Recurrence is high even under ideal circumstances.

III. 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. 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.

B. Open tibia 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 42-2).

2. Gustilo types IIIb and IIIc frequently require flap coverage of exposed bone, most commonly with a pedicled gastrocnemius or soleus flap; for more distal wounds, consider a perforator or free flap.

C. Limb salvage reconstruction for neoplasm may require replacement of large segments of bone, nerve, or vessels. **Skeletal replacement** can be

accomplished using an endoprosthesis, allogenic bone transplant, or vascularized free bone (fibula) transfer.

TABLE 42-2 Gustilo Open Fracture Classification

Classification	Characteristics
I	Clean wound <1 cm long
II	Laceration >1 cm long with extensive soft-tissue damage
III	Extensive soft-tissue laceration, damage, or loss; open segmental fracture; or traumatic amputation
IIIa	Adequate periosteal cover of the bone despite extensive soft-tissue damage; high-energy trauma with small wound or crushing component
IIIb	Extensive soft-tissue loss with periosteal stripping and bone exposure requiring soft-tissue flap closure; usually associated with massive contamination
IIIc	Vascular injury requiring repair

Adapted from Gustilo RB, Anderson JT. Prevention of infection in the treatment of one thousand and twenty-five open fractures of long bones: retrospective and prospective analysis. *J Bone Joint Surg Am.* 1976;58A:453.

D. Foot wound coverage may be necessary due to trauma, neoplasm, or ulceration. The etiology of ulcers (e.g., arterial insufficiency requiring extremity revascularization in the case of ischemic ulcers) should be addressed prior to coverage.

1. Plantar surface. Optimal coverage of the plantar surface provides a durable, sensate platform; options include dermal substitutes, rotation of instep-tissue for small wounds, or free flaps.

2. Dorsal foot. Coverage should be thin to allow for standard footwear. In the presence of peritenon, a skin graft can be used; otherwise, consider a dermal substitute or thin fascial free flaps (temporoparietal, parascapular, or radial forearm) covered by skin grafts.

IV. PERIPHERAL NERVE

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. Evaluation of motor nerve function is standardized (Table 42-3).

2. Diagnostic studies

a. Nerve conduction studies (NCS) characterizes the conduction of large-diameter, myelinated nerves and can help determine 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.

TABLE 42-3 Classification of Motor Function

Grade	Motor Function
M0	No contraction
M1	Perceptible contraction in proximal muscles
M2	Perceptible contraction in proximal and distal muscles
M3	All important muscles powerful enough to act against gravity

M4	Muscles act against strong resistance; some independent movement possible
M5	Normal strength and function

Adapted from Mackinnon SE, Dellon AL. *Surgery of the Peripheral Nerve.* New York, NY: Thieme, 1988:118.

b. Electromyography (EMG) samples the action potentials from muscle fibers. Motor unit potentials (MUPs) can indicate early reinnervation, while fibrillations may represent denervation.

3. Nerve injuries are classified according to severity (Table 42-4).

B. Management of Acute Injuries

1. Sharp transection (trauma, iatrogenic). Nerve should be repaired urgently.

a. Debride injured nerve to healthy fascicles.

b. If possible, a tension-free primary repair should be performed.

c. If gap is too large for tension free repair, consider grafting.

2. Acute postoperative nerve deficit unrelated to location of surgery

a. Commonly **due to patient positioning or traction,** especially at anatomically restrictive sites (e.g., the ulnar nerve at the elbow or the common peroneal nerve at the knee).

b. Usually a first-, second-, or third-degree injury, and full recovery can be expected in most cases.

c. If no improvement by 6 weeks, obtain baseline NCS and EMG. If there is no evidence of return of function at 3 months, obtain repeat studies.

(1) If MUPs are present but there are reduced conduction velocities at known compression points, decompression of the nerve will aid recovery.

(2) If there are no MUPS on EMG, the nerve should be explored and repaired.

3. Loss of nerve function after gunshot or open blunt trauma is usually the result of first- or second-degree injury.

TABLE 42-4 Classification of Nerve Injuries

Sunderland[a]	Seddon[b]	Structure Injured	Prognosis
First degree	Neurapraxia	Schwann cell (demyelination)	Complete recovery within 12 wks
Second degree	Axonotmesis	Axon (Wallerian degeneration)	Complete recovery regeneration 1 mm/day
Third degree		Endoneurium	Incomplete recovery
Fourth degree		Perineurium	No recovery
Fifth degree	Neurotmesis	Epineurium	No recovery
Sixth degree		Mixed injury, neuroma incontinuity[c]	Unpredictable recovery

[a] Sunderland S. A classification of peripheral nerve injuries producing loss of function. *Brain.* 1951;74:491.

[b] Seddon HJ. Three types of nerve injury. *Brain.* 1943;66:237.

[c] Mackinnon SE. New direction in peripheral nerve surgery. *Ann Plast Surg.* 1989;22(3):257-273.

a. Treat as a closed injury, observing for improvement and obtaining EMG and NCS at 6 weeks and 3 months, as needed.

b. If the nerve is visible or the wound is explored for other reasons (e.g., vascular repair), the nerve is explored. If the nerve is in continuity, manage as one would 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).

4. Nerve deficit from compartment syndrome is treated by emergent fasciotomy. If decompressed early (within 6 hours), there is usually a rapid return of function.

C. Compression neuropathy due to compression or repetitive trauma is a common clinical problem. Typically involved nerves include the median nerve at the wrist (**carpal tunnel syndrome**), the ulnar nerve at the elbow or wrist (**cubital tunnel syndrome**), the anterior or

posterior interosseous nerves in the forearm, the brachial plexus at the thoracic outlet (**neurogenic thoracic outlet syndrome**), the common peroneal nerve at the knee, and the posterior tibial nerve at the ankle (**tarsal tunnel syndrome).**

1. Initial management is usually physical therapy, behavior modification, and splinting to avoid repetitive compression for at least 6 weeks.

2. If nonsurgical intervention fails, open decompression and transposition is indicated.

AESTHETIC SURGERY PEARLS FOR THE GENERAL SURGEON

I. AUGMENTATION MAMMOPLASTY

A. Implants

1. Placement. Subglandular, submuscular or a combination (*dual plane)* via periareolar, transaxillary, or inframammary fold incisions

2. Monitoring

a. Cancer screening. Use **standard mammography plus Eklund views** (pushes implant against the chest wall and breast tissue is pulled forward)

b. Rupture. Saline implant rupture presents as a deflated breast. Silicone implants must be **monitored by MRI** with an initial study 3 years after placement and every 2 years thereafter.

B. Fat transfer may be used in large volume to recreate a whole breast or to fill the upper pole following implant placement, most commonly in the case of postmastectomy reconstruction.

1. Fat necrosis on standard mammography requires careful examination to determine if further investigation to rule out cancer is needed. Most commonly appear as scattered microcalcifications on mammography or oil cysts on gross inspection. Generally, it is a late finding occurring months to years postprocedure.

II. ABDOMINOPLASTY

A. Involves elevation of large abdominal flaps extending from umbilicus to xiphoid, transposition of the umbilicus, with or without rectus plication. Blood supply to the flaps comes from lateral intercostals, subcostals, and lumbar vessels. Large abdominal scars (e.g., subcostal incision after open cholecystectomy) can result in flap necrosis.

B. Risk of **pulmonary embolism** is between 1/300 and 1/1000, with the highest risk period being the first postoperative week. Accordingly, patients are commonly discharged on 5 to 10 days of anticoagulation therapy.

III. POSTBARIATRIC BODY CONTOURING

A. Generally **not covered by insurance.**

B. Timing. Weight should be stable for at least 3 to 4 months; generally, this occurs 12 to 18 months after a bariatric operation. The lower the BMI, the lower the risk of complications and the better the cosmetic outcome.

C. Patients commonly have nutritional deficiencies that must be corrected before further surgery: The most common is low iron, but it is also worthwhile to check levels of cyanocobalamin (vitamin B_{12}), calcium, folate, and albumin.

CHAPTER 42: PLASTIC AND HAND SURGERY

Multiple Choice Questions

1. An 83-year-old man undergoes excision of a basal cell carcinoma of the cheek. The defect is 3 × 3 cm with subcutaneous fat exposed at the base. Reconstruction with skin grafting is planned. Which of the following will minimize long-term graft contracture?

a. Split thickness skin grafting with meshing

b. Split thickness skin grafting without meshing

c. Full thickness skin grafting

d. Cultured epidermal autografting

e. A skin graft is inappropriate in this situation

View Answer

2. A 23-year-old man is brought to the trauma bay by emergency services following a motor vehicle accident. He has multiple forehead lacerations, bilateral orbital ecchymoses and reports he cannot see out of his right eye. What is the first step in management?

a. Maxillofacial CT

b. Intraocular examination

c. Assessment of pupillary response

d. Completion of the primary survey

e. Immediate lateral cantholysis

View Answer

3. Following an altercation at a bar, a 20-year-old male presents with a cut over the third metacarpal head on the dorsum of his right hand. On physical examination he has full range of motion but reports pain on

extending his middle finger. Radiographs show no fracture. To minimize this patient's risk of metacarpal joint destruction, which of the following is most appropriate?

a. Wound exploration and wash-out in the operating room

b. Irrigate wound thoroughly and allow to heal by secondary intention

c. Begin oral antibiotics immediately

d. Instruct the patient in range of motion exercises

e. Obtain an MRI of the hand

View Answer

4. A 46-year-old female is brought to the emergency room by her husband. He reports she had an abdominoplasty at an ambulatory surgery center 3 days ago. The patient appears confused and lethargic. On examination, she is hypotensive and tachycardic and pulse oximetry on room air is 98%. Her abdomen is tender, incisions are intact, and dressings are clean. Her drains contain sanguineous fluid and a few clots. The husband is not sure when the drains were last emptied. Following initial resuscitation and stabilization, which of the following studies is most appropriate?

a. Spiral CT of the chest

b. Type and cross

c. Lower-extremity duplex ultrasound

d. Chest x-ray

e. Urine drug screen

View Answer

5. A polytrauma patient has a thigh wound measuring 15 × 23 cm. There is heavy dirt and gravel contamination and frayed tendon and muscle at the wound base. An external fixator has been placed on the associated femur fracture. Following the initial wash-out and debridement, what is the most appropriate therapy?

a. “Wet to dry” wound care

b. Negative pressure wound therapy

c. Split thickness skin grafting

d. Rotation flap coverage

e. Free flap

View Answer

6. A 56-year-old male with a history of bladder cancer and pelvic radiation, was found to have metastases in the previous incision, abdominal wall, and bladder dome. Urology has planned full-thickness abdominal wall resection and radical cystectomy. The abdominal wall defect is 12 × 12 cm in the lower abdomen and includes portions of the bilateral rectus abdominis. Which of the following options will provide the best abdominal wall reconstruction?

a. Vicryl mesh

b. Component separation

c. Component separation with split thickness skin graft

d. Pedicled anterolateral thigh fasciocutaneous flaps

e. Free latissimus dorsi muscle flap with split thickness skin graft

View Answer

7. A 76-year-old male underwent coronary artery bypass surgery using the left internal mammary artery and left saphenous vein for grafts. His course was complicated by mediastinitis and sternal dehiscence with a resultant 3 × 7 cm central chest defect with exposed sternum. Following multiple debridements and negative pressure wound therapy, wound cultures are negative. Which of the following is most appropriate for coverage?

a. Skin graft

b. Left pedicled rectus abdominis flap with skin graft

c. Left turn-over pectoralis flap with skin graft

d. Pedicled omentum with skin graft

e. Free anterolateral thigh flap

View Answer

8. A 20-year-old suffered multiple gunshot wounds to the chest, abdomen, and right arm. He is stabilized and able to participate in an upperextremity examination 1 week following presentation. On examination, he is unable to extend his wrist, fingers, and thumb. What is the most appropriate management of this patient?

a. Obtain electromyography and nerve conduction studies, explore nerve if evidence of denervation

b. Obtain baseline electromyography and nerve conduction studies at 6 weeks, repeat at 3 months, and explore nerve if no evidence of recovery

c. Observation, explore nerve if no recovery at 6 months

d. Explore and graft nerve injury as soon as patient is stable for surgery

e. Immediate tendon transfers for elbow and wrist extension

View Answer

9. A 33-year-old paraplegic woman in an assisted living facility is found to have a stage IV ischial pressure ulcer. There is a hydrocolloid dressing in place, the wound base appears cleans, and the surrounding skin is clean and intact. Which of the following is a contraindication to flap coverage?

a. Osteomyelitis of the ischium

b. Fecal incontinence

c. Negative pressure wound therapy

d. Low serum Fe

e. Baclofen treatment for spasticity

View Answer

10. A 42-year-old women who had augmentation mammoplasty with silicone implants and fat transfer 6 months ago comes to the office complaining of right breast pain. Mammography shows linear calcifications in the lower outer quadrant of the right breast and lipid cysts in the upper poles of both breasts. What is the next most appropriate step in management?

a. Reassurance

b. MRI

c. Repeat mammography in 1 year

d. Core needle biopsy of bilateral upper outer quadrants

e. Core needle biopsy of the right breast lower outer quadrant

View Answer

43

Urology for the General Surgeon

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The discipline of **urologic surgery** encompasses the management of benign and malignant conditions of the genitourinary system including the kidneys, ureters, bladder, urethra, and the male external genitalia.

I. HEMATURIA.

Hematuria or blood in the urine, warrants a complete urologic workup. **Gross hematuria** is visibly bloody urine, whereas **microscopic hematuria** is defined as *3 or more* red blood cells per high-power field. Common etiologies include: Urinary tract infection (UTI), stones, benign prostatic hypertrophy, malignancy, and recent trauma or instrumentation.

A. Evaluation for hematuria (gross or microscopic) consists of the following:

1. Urinalysis and **urine culture** from a *freshly voided* mid-stream specimen. **Cytology** should also be obtained for *gross hematuria.*

2. Computed tomography (CT) urogram: Consisting of a three-phase CT of the abdomen/pelvis without contrast, with contrast, and with delayed contrast imagesÑis the preferred imaging modality. However, *magnetic resonance (MR) urogram* and *renal ultrasound with retrograde pyelograms* are adequate in appropriately selected patients.

3. Cystoscopy is required for a complete evaluation of the urethra and bladder as imaging can miss small lesions or strictures.

4. If the etiology of hematuria remains unclear, repeat a UA in 1 year.

a. For recurrent *gross* hematuria: Annual complete workup.

b. For recurrent *microscopic* hematuria, repeat workup Q3Ñ5 years.

B. Treatment of Symptomatic Gross Hematuria

1. Patients in **urinary retention due to clots** require urgent urologic consultation and a 22F to 24F three-way Foley catheter. The catheter should be manually irrigated and aspirated with sterile saline or water until the bladder is clot-free. **Continuous bladder irrigation** (CBI) *should not be initiated* until the bladder is clot free.

2. Persistent gross hematuria from a lower urinary tract source (bladder, prostate, urethra) despite conservative measures requires operative management via *cystoscopy and fulguration* of the bleeding source.

3. Persistent hematuria from an upper urinary tract source (kidney, ureter) may be due to a **hemorrhagic renal mass, angiomyolipoma, arteriovenous fistula, or renal trauma.** These patients may require surgical intervention by urology or angiography ± embolization by interventional radiology.

TABLE 43-1 Bosniak Classification System of Renal Cystic Masses

Bosniak Category	Characteristics	Risk of Malignancy	Management
I	Simple cyst. Thin wall, no septa. Low attenuation, no enhancement.	0%	None
II	Few thin septa or fine calcifications. Can be high attenuation (if <3 cm) but no enhancement.	5-18.5%[a]	None
IIF	Multiple thin septa, minimal wall thickening or thick calcifications. Can be high attenuation (>3 cm) but no enhancement.	15-25%	Surveillance imaging Q6-12 mo
III	Walls or septa with measurable enhancement	33+%	Resection/ablation
IV	Contains enhancing soft tissue components	92.5%	Resection/ablation

[a] Note: There is limited data on Category II cysts as most studies combine II and IIF

cysts. Category II cysts (excluding IIF) are thought to be essentially benign.
Adapted from Whelan TF. Guidelines on the management of renal cyst disease. *Can Urol Assoc J* 2010;4(2):98-99. PMID: 20368890.

II. DISEASES OF THE KIDNEY

A. Renal cysts occur in approximately 50% of persons older than 50 years, and the vast majority are benign. Renal cysts can be diagnosed and evaluated with CT, MRI, or ultrasound (Table 43-1).

B. The majority (80%) of **solid renal masses** are malignant. The historical triad of **flank pain, hematuria, and flank mass** occurs uncommonly. **Renal cell carcinoma** is the most common type of renal cancer. Other conditions in the differential for solid renal masses include transitional cell cancer, oncocytoma (usually benign), lymphoma, and metastatic tumors (lung, breast, gastrointestinal, prostate, pancreas, and melanoma).

1. Evaluation of a renal mass:

a. Laboratory tests: Complete blood count, renal panel, and liver function tests. Lactate dehydrogenase (LDH) may be prognostic in metastatic disease.

b. Abdominal Imaging with both noncontrast and contrast phases (*Renal Protocol CT/MR*) to **assess for enhancement. Ultrasonography** can determine whether a mass is cystic or solid.

c. Chest x-ray is required to rule out metastasis (chest CT can be performed if there is high suspicion of chest metastasis or if CXR is positive).

d. *Bone scan* is indicated in patients with elevated alkaline phosphatase or bone-related complaints. *Head CT* is indicated in patients with neurologic symptoms.

e. The role of percutaneous **renal mass biopsy** has expanded in recent times, with diagnostic accuracy greater than 90% while complication rate is less than 5% (*J Urol.* 2008;179:20-27). It is most often recommended for patients with significant surgical comorbidities.

2. Paraneoplastic syndromes occur in 10% to 40% of renal cell carcinomas.

a. Renin overproduction can present as hypertension.

b. *Stauffer syndrome* is benign elevation of liver enzymes which usually resolves after tumor removal.

c. Hypercalcemia can be caused by the production of **parathyroid hormone-like protein (PTHrP)** produced by the tumor.

d. Erythrocytosis may result from overproduction of erythropoietin.

C. Management of Renal Masses

1. The **management of solid renal masses depends on the tumor stage** (Table 43-2).

a. T1 and T2 lesions. Nephron-sparing surgery via *Partial Nephrectomy* **is preferred** whenever possible; otherwise, **radical nephrectomy is considered the standard of care.**

b. T3 and T4 lesions mandate radical nephrectomy.

2. Metastatic renal cell carcinoma is resistant to radiation and chemotherapy. It is unclear how beneficial cytoreductive nephrectomy is in the modern era; studies are pending. Targeted therapy with agents such as *tyrosine kinase inhibitors,* **VEGF inhibitors,** as well as **IL-2 immunotherapy** has shown survival benefit in some patients (*BMC Cancer.* 2009;9:34).

III. DISEASES OF THE URETER

A. Hydronephrosis is commonly caused by **ureteral obstruction,** which can be intrinsic or extrinsic in origin.

1. Common *intrinsic* causes include stones, ureteropelvic junction obstruction, stricture, and urothelial carcinoma. Common *extrinsic* causes include compression from a crossing vessel, pregnancy, abdominal mass, or retroperitoneal fibrosis.

2. Short-term management includes **placement of a ureteral stent** or **percutaneous nephrostomy tube** to relieve obstruction. Long-term management is based on the etiology.

3. The best **imaging** modality depends on the suspected etiology. Ultrasound and noncontrast CT are best to evaluate urolithiasis (see below). Intrinsic compression is best defined by CT/MR urogram. CT/MR with contrast can distinguish most forms of extrinsic compression.

TABLE 43-2 AJCC 2010 TNM Staging for Renal Cell Carcinoma

Primary Tumor (T)

Tx	Primary tumor cannot be assessed
T0	No evidence of primary tumor
T1	Tumor 7 cm or less in greatest dimension, limited to kidney

T1a	Tumor 4 cm or less in greatest dimension, limited to kidney
T1b	Tumor greater than 4 cm but not larger than 7 cm and limited to kidney
T2	Tumor greater than 7 cm in greatest dimension and limited to kidney
T2a	Tumor greater than 7 cm but less than 10 cm and limited to kidney
T2b	Tumor greater than 10 cm and limited to kidney
T3	Tumor extends into the major veins or perinephric tissues but not into the ipsilateral adrenal or beyond Gerota fascia
T3a	Tumor grossly extends into the renal vein or its segmental branches or tumor invades perirenal and/or renal sinus fat but not beyond Gerota fascia
T3b	Tumor grossly extends into the vena cava below the diaphragm
T3c	Tumor grossly extends into the vena cava above the diaphragm or invades the wall of the IVC
T4	Tumor invades beyond Gerota fascia

Regional Lymph NodesÑClinical Stage (N)

Nx	Regional lymph nodes cannot be assessed
N0	No regional lymph node metastasis
N1	Metastasis in regional node(s)

Distant Metastasis (M)

M0	No distant metastasis

M1 Distant metastasis

4. To determine the *functional significance* of chronic obstruction, **diuretic renal scintigraphy** (furosemide renal scan) may be performed. This study can estimate the relative function and excretion of each kidney.

B. Urolithiasis (ureteral calculi) typically presents as the acute onset of severe, intermittent flank pain often associated with nausea and vomiting. Patients may present with hematuria, but this is nonspecific. Patients should always be assessed for signs of infection including history of fever because UTI and

obstructive uropathy can quickly progress to sepsis, which requires urgent intervention.

1. Epidemiology. Stone formation commonly occurs between the **third and fifth decades** and tends to have a **male** predominance.

2. There are numerous **risk factors** for stone formation. Common modifiable factors include **low fluid intake** and diets that **contain high animal protein, sodium, or oxalate.** Medical conditions such as **obesity, diabetes, inflammatory bowel disease, gout, hyperparathyroidism,** and **type I renal tubular acidosis (RTA)** increase urolithiasis risk. Various medications and high dose vitamins C or D can favor stone formation (*Lancet.* 1997;349:1294-1295).

3. Evaluation of urolithiasis.

a. In the *acute* setting: **Urinalysis, urine culture,** and **a renal panel** should be performed.

b. Noncontrast CT is the most accurate study for nephrolithiasis. Though CT has superior sensitivity to ultrasound (88+% vs. 57%), recent data have shown that ultrasound may be a reasonable and safe option as an initial diagnostic study, providing lower cost and radiation exposure without significantly affecting clinical outcome (*N Engl J Med.* 2014;371:1100-1110).

c. Abdominal x-ray (KUB) is useful to monitor for stone passage of radiopaque stones and to assess whether the stone is amenable to **extracorporeal shock therapy (ESWL).**

4. Management. Many patients can be managed as an outpatient with close urologic followup. Patients with intractable pain, nausea, or emesis not adequately controlled by oral medication require **hospital admission.** Urgent **surgical intervention** is indicated if there are signs of infection, significant acute kidney injury, an obstructed solitary kidney, or bilateral obstruction.

a. Medical expulsive therapy. Over **70% of stones <5 mm and up to 40% of stones <10 mm will pass spontaneously.** Stone passage can be aided with **narcotics** and **daily alpha blocker therapy (tamsulosin),** which has been shown to improve stone passage rates by up to 20%. Urine should be strained for stones. Spontaneous stone passage may take up to 3 to 4 weeks (*J Urol.* 1997;178:2418-2434).

b. Surgical treatment. In the acute setting, patients meeting surgical criteria can be managed by **ureteral stenting or percutaneous nephrostomy tube placement.** If patients remain symptomatic, have failure of stone passage within 2 to 3 weeks, or have large stones, surgical options include shock wave lithotripsy (SWL), ureteroscopy ± laser lithotripsy, and **percutaneous nephrolithotomy (PCNL).**

C. Ureteropelvic Junction Obstruction (UPJO)

1. UPJO etiologies include a congenital aperistaltic or stenotic segment of proximal ureter, extrinsic compression from crossing vessels, benign polyps, and scarring.

2. Patients may present at any age. Common symptoms are **flank pain** (which may be intermittent), nausea/vomiting, and pyelonephritis.

3. CT/MR with contrast and **diuretic renal scintigraphy** help to determine the site and *functional significance* of the obstruction.

4. Treatment is based on severity of symptoms and renal function of the affected side. It can consist of **observation, endopyelotomy** of the strictured segment via ureteroscopy, or **pyeloplasty** (surgical reconstruction of the affected segment).

IV. DISEASES OF THE URINARY BLADDER

A. Bladder cancer is found in up to 5% of patients with microscopic hematuria.

1. Urothelial cell carcinoma (UCC) accounts for more than 90% of bladder tumors in the United States; squamous cell carcinoma and adenocarcinoma are rarer. Bladder cancer is linked strongly to **smoking,** as well as to **textile dyes, cyclophosphamide, chronic indwelling catheters, chronic parasitic infection** (*Schistosoma haematobium*), **and radiation exposure.**

2. UCC is categorized as superficial or invasive. Staging is outlined in Table 43-3. Evaluation for UCC requires upper tract imaging via CT/MR urogram, cystoscopy, and urine cytology.

a. Superficial tumors (CIS, Ta, T1) do not invade the muscular bladder wall. These tumors can be staged and treated with **transurethral resection (TUR).** Between 40% and 80% of superficial tumors recur within 1 year; thus, diligent followup is necessary. Recurrent tumors are treated with TUR and **intravesical therapy (bacillus CalmetteÑGuŽrin** or **mitomycin C).**

b. Muscle-invasive UCC (stage ³ T2) is treated with radical cystectomy and urinary diversion. This involves **radical cystoprostatectomy** (removal of bladder, prostate, and possibly urethra) in males and **anterior exenteration** (removal of bladder, uterus, cervix, and vaginal anterior wall) in females.

(1) Metastatic evaluation includes **chest x-ray/CT, CT urogram, and liver function tests.** Despite aggressive management, 5-year survival ranges from 15% to 63% based on stage (*National Cancer Institute SEER database*).

c. Urinary diversion (Table 43-4**)** typically consists of a reservoir made from detubularized ileum or colon; stomach is rarely used. Diversion may be *continent* (requiring Valsalva or catheterization to empty) or *incontinent* (drains continuously). Common chronic derangements from the use of intestine include metabolic acidosis, B_{12} deficiency, and bone loss.

d. Locally advanced or metastatic bladder cancer is treated with **chemotherapy.** Neoadjuvant and adjuvant chemotherapy have been shown to benefit patients with invasive UCC who undergo surgery.

V. DISEASES OF THE PROSTATE

A. Prostate cancer is the most common malignancy in American men and the second leading cause of cancer death. Prostate cancer rarely causes symptoms until it becomes locally advanced or metastatic. Risk factors for prostate cancer include African-American race, family history, and advanced age.

TABLE 43-3 AJCC 2010 TNM Staging for Urothelial Cell Carcinoma

Primary Tumor (T)

Tx	Primary tumor cannot be assessed
T0	No evidence of primary tumor
Ta	Noninvasive papillary carcinoma
Tis	Carcinoma in situ; ÒFlat TumorÓ

T1	Tumor invades the subepithelial connective tissue
T2	Tumor invades the muscularis propria
pT2a	Tumor invades superficial muscularis propria
pT2b	Tumor invades deep muscularis propria
T3	Tumor invades perivesical tissue
pT3a	Microscopic invasion
pT3b	Macroscopic invasion
T4	Tumor invades any of the following: prostatic stroma, seminal vesicles, uterus, vagina, pelvic/abdominal wall

Regional Lymph NodesÑClinical Stage (N)

Nx	Regional lymph nodes cannot be assessed
N0	No regional lymph node metastasis
N1	Single regional lymph node in the true pelvis
N2	Multiple regional lymph nodes in the true pelvis
N3	Lymph node metastasis to the common iliac lymph nodes

Distant Metastasis (M)

M0	No distant metastasis
M1	Distant metastasis

TABLE 43-4 Common Types of Urinary Diversion

Type	Name	Outlet
Noncontinent	Ileal Conduit	Abdominal stoma empties into urostomy bag
Continent	Indiana/Miami Pouch Neobladder	Continent catheterizable channel made of the ileocecal valve and terminal ileum Orthotopic anastomosis to the native urethra

1. Screening includes **digital rectal examination (DRE)** and measurement of **serum prostate-specific antigen (PSA**).

a. DRE and PSA provide a better sensitivity for screening than either alone.

b. PSA screening remains controversial based on contradictory conclusions from two large, prospective randomized trials (*N Engl J Med.* 2009;360:1310-1319; *N Engl J Med.* 2009;360:1320-1328). Current American Urological Association (AUA) guidelines (2013) recommend discussing screening with patients. **If elected, screening should begin at age 55 and occur every 1 or 2 years until age 70.**

c. Abnormalities in DRE or PSA should be evaluated by a urologist for consideration of a **transrectal ultrasound (TRUS) with needle biopsy of the prostate.**

2. Staging of prostate cancer (Table 43-5) is performed for men with highrisk disease and consists of an **abdomen/pelvis CT/MRI** and a **bone scan.**

3. Treatment options for men with organ-confined prostate cancer include active **surveillance, radical prostatectomy,** and **radiation therapy.** Choice of treatment modality is a complex decision tailored to the patient based on multiple factors.

a. Recurrent or **metastatic disease** is treated with **androgen deprivation therapy,** medications which inhibit testosterone precursors or receptors, and/or **chemotherapy.**

B. Acute bacterial prostatitis presents with signs and symptoms of acute illness and UTI; many patients have **fevers, malaise, and significant voiding complaints.** Prostate exam will reveal a tender enlarged prostate. Prostatic massage should not be performed to avoid urosepsis/bacteremia. Empiric **antibiotics** should be started immediately. If no improvement, pelvic imaging to **rule out abscess** should be obtained. Other forms of prostatitis include chronic bacterial prostatitis, chronic pelvic pain syndrome, and asymptomatic prostatitis.

C. Benign prostatic hyperplasia (BPH) can manifest with both irritative and obstructive **lower urinary tract symptoms** (**LUTS)** including weak stream, frequency/urgency, incontinence, bladder outlet obstruction, and nocturia.

1. Objective evidence of **bladder outlet obstruction** includes decreased urinary flow rate, increased postvoid residual, and urinary retention. Bladder stones, hematuria, recurrent UTI, and renal failure can also occur.

a. Postobstructive diuresis (polyuria and natriuresis) can occur after chronic urinary obstruction is acutely relieved via catheterization. Rarely this can last >48 hours and cause severe electrolyte abnormalities. Patients should be monitored closely with vital signs, serial laboratory tests, and fluid replacement as needed.

TABLE 43-5 AJCC 2010 TNM Staging for Prostate Carcinoma

Primary Tumor (T)

Tx	Primary tumor cannot be assessed
T0	No evidence of primary tumor
T1	Clinically inapparent tumor neither palpable nor visible by imaging
T1a	Tumor incidental histologic finding in 5% or less of tissue resected
T1b	Tumor incidental histologic finding in more than 5% of tissue resected

T1c	Tumor identified by needle biopsy via screening (PSA, DRE)
T2	Tumor confined to the prostate
T2a	Tumor involves one-half of one lobe or less
T2b	Tumor involves more than one-half of one lobe, but not both lobes
T2c	Tumor involves both lobes
T3	Tumor extends through the prostatic capsule
T3a	Extracapsular extension
T3b	Seminal vesicle invasion
T4	Tumor is fixed or invades adjacent structures other than the SVs, such as external sphincter, rectum, bladder, levator muscles, and/or pelvic wall

Regional Lymph NodesÑClinical Stage (N)

Nx	Regional lymph nodes cannot be assessed
N0	No regional lymph node metastasis
N1	Metastasis in regional node(s)

Distant Metastasis (M)

M0	No distant metastasis
M1	Distant metastasis
M1a	Nonregional lymph node(s)

M1b Bone(s)

M1c Other site(s) with or without bone disease

2. Treatment

a. Observation is best suited for minimally symptomatic patients.

b. Medical treatment includes long-acting **selective** α-**blockers** (e.g., tamsulosin) or **5α-reductase inhibitors** (e.g., finasteride). Combination therapy with an α-blocker + a 5α-reductase inhibitor is used for moderate to severe BPH.

3. Surgical therapy is indicated in patients who have failed medical therapy or have severe symptoms. The gold standard is **transurethral resection of the prostate (TURP),** but transurethral photoselective vaporization of the prostate **(GreenLight PVP)** offers similar functional results with less morbidity (*J Urol.* 2015;193:570-578).

VI. DISEASES OF THE PENIS

A. Priapism is a persistent penile erection that continues beyond, or is unrelated to, sexual stimulation. Ischemic priapism lasting >4 hours is an **emergency** (Table 43-6).

TABLE 43-6 Ischemic versus Nonischemic Forms of Priapism

	Ischemic Priapism	Nonischemic Priapism
Emergency?	**YES**	No
Pain	Painful	Nonpainful
Rigidity	Rigid corpora, flaccid glans	Partially rigid phallus

Pathogenesis	Decreased venous outflow	Increased arterial inflow
Etiologies	i. Hematologic abnormalities, such as **sickle cell disease;** ii. **Drugs,** particularly trazodone, cocaine, and erectile dysfunction medications; iii. Invasive neoplasm	i. Arterial fistula related to perineal/genital trauma; ii. Neurogenic
First-line Treatment	Irrigation, aspiration, phenylephrine	None, most resolve with observation
Prognosis	**Erectile dysfunction** from fibrosis/scarring of the corpora can occur without prompt treatment	Good, but selective arterial embolization can be used for refractory/recurrent cases

1. Treatment

a. First-line treatment involves **corporal irrigation and aspiration** of old blood via a 14G to 18G needle

b. Intracorporal injection of an **α-adrenergic agent** (phenylephrine, 250 to 500 µg/mL) administered every 2 to 5 minutes until detumescence is achieved. Patients should be monitored for hypertension and reflex bradycardia during treatment.

c. For patients with sickle cell disease, treatment involves aggressive hydration, supplemental oxygen, and blood transfusion if anemic.

d. Surgical cavernosal-venous shunting should be considered if the above methods fail. A penile prosthesis can be placed at the same time, if desired.

B. Paraphimosis is a *urologic emergency* in which the foreskin becomes constricted below the glans, resulting in progressive penile edema, pain, and potentially penile necrosis. It may occur following Foley catheter placement in an uncircumscribed if the foreskin is not returned to its anatomical position.

1. Immediate **manual reduction** should be performed: Edema should be reduced by applying a firm grip to the distal penis squeezing for several minutes and the glans pushed down, within the foreskin. If this is unsuccessful, a Urology consult should be obtained urgently.

C. Phimosis is typically a benign condition where the foreskin is unable to be fully retracted over the glans. Many patients are asymptomatic but phimosis can result in glans/foreskin infections or urinary retention (rare).

VII. DISEASES OF THE SCROTUM AND TESTICLES

A. Testicular torsion is the rotation of the testicle on its vascular pedicle, resulting in ischemia. This is a true **emergency** because testicular viability depends on detorsion within a few hours.

1. History. Acute onset of severe testicular pain and swelling often associated with abdominal pain, nausea, and/or vomiting.

2. Examination reveals an extremely tender, swollen testicle which may be high riding in the scrotum with a transverse lie. The cremasteric reflex is usually absent on the affected side. Normal urinalysis and the absence of leukocytosis may help to rule out epididymitis.

3. Testicular torsion is a **clinical diagnosis** and treatment should not be delayed to obtain imaging. However, *Doppler ultrasound* can help to confirm or exclude the diagnosis with reported sensitivity and specificity >95%.

4. Immediate **scrotal exploration and bilateral orchiopexy** is required if torsion is suspected. Manual detorsion of the testicle may be attempted; however, bilateral orchiopexy is still necessary.

B. Torsion of testicular/epididymal appendage (appendix testis) presents with mild to moderate testicular pain and onset over 12 to 24 hours without abdominal symptoms. On examination, the testicle has a normal

position but tenderness in the superior testicle and/or epididymis. The testicle may be mildly swollen due to reactive inflammation. There may be a palpable Òpea-likeÓ nodule on the superior testicle or epididymis, which can sometimes be visible through the scrotal skin (Òblue dotÓ sign). Diagnosis can be confirmed with ultrasound. Treatment consists of **NSAIDs, light physical activity and scrotal support** until resolution over 7 to 14 days.

C. Epididymitis usually presents with a 1- to 2-day onset of unilateral testicular pain and swelling sometimes associated with dysuria, urethral discharge, or LUTS.

1. Findings include a painful, indurated epididymis, and pyuria. Urinalysis, urine culture, and CBC are obtained. When clinically indicated, urethral swabs for gonococci and chlamydiae are performed.

2. If sexually transmitted infection (STI) is unlikely (e.g., if >35 years), treat empirically with an **oral fluoroquinolone,** then a **culture-specific antibiotic for 2 weeks. NSAIDs** can reduce inflammation and provide symptomatic relief. For moderate to severe cases with fever and/or leukocytosis, *ultrasonography* can be useful to rule out abscess formation and assess testicular

perfusion. Broad-spectrum antibiotics may be required.

D. Fournier gangrene is a severe *necrotizing fasciitis* involving the subcutaneous tissue of the genitals and perineum. This is an **emergency** as mortality ranges from 20% to 30% in most series. Diabetic, alcoholic, and other immunocompromised patients are more susceptible to this condition.

1. Examination reveals **painful edema and erythema** of the skin of the scrotum, phallus, and perineal area. This may progress rapidly to frank necrosis with crepitus and malodor.

2. Evaluation should include a CBC, electrolytes, urinalysis, urine, and blood cultures. A **CT scan** can be obtained to evaluate for extension of infection and **presence of subcutaneous gas** but should **never delay operative** treatment.

3. Broad-spectrum antibiotics that are active against mixed organisms (aerobic and anaerobic, gram positive and negative) should be started immediately.

4. Wide surgical debridement is urgently required, with aggressive postoperative support. *Orchiectomy is rarely indicated* because the testicles are usually uninvolved. The wound should be left open and initially managed with wet to dry dressing changes. Multiple debridements may be necessary. Wound closure often is an extensive process and may involve skin grafts.

E. Nonacute Scrotal Masses

1. Hydroceles are fluid collections around the testicle that **transilluminate** and are usually asymptomatic. **Ultrasound** is recommended to rule out testicular malignancy or other scrotal pathology. If hydroceles do become symptomatic, they can be surgically repaired. **Reducible hydroceles** indicate an open peritoneal communication.

2. Varicoceles are abnormally dilated testicular veins. On examination, they feel like a Òbag of wormsÓ and increase in severity with Valsalva. *Right-sided varicoceles* **are rare and should be evaluated with retroperitoneal imaging to rule out malignancy.** Varicoceles are the **most common surgically correctable cause of male infertility;** nevertheless, most men with varicoceles remain fertile, asymptomatic, and never require treatment. Varicocele repair results in improved semen quality in approximately 70% of patients.

F. Testicular tumors are the most common solid tumors in males aged 15 to 35 years. The estimated lifetime risk for testicular malignancy is <1 in 300. Owing to improved multimodality therapy, overall 5-year survival for testis cancer is 95%. Risk factors associated with testicular tumors include cryptorchidism, HIV infection, gonadal dysgenesis, and infertility.

1. The typical clinical finding is **a painless testicular mass,** although onethird of patients may present with pain. Scrotal ultrasound is mandatory. α-Fetoprotein (AFP), β-human chorionic gonadotropin (βhCG), and LDH are **serum tumor markers** that help to identify and stage the tumor. The markers are also used to monitor the effectiveness of therapy and to screen for

recurrence.

2. Testicular tumor staging (Table 43-7) consists of a chest x-ray and CT/MRI of the abdomen/pelvis.

3. Initial therapy for all testicular tumors is **radical inguinal orchiectomy.**

a. Seminomas constitute 60% to 65% of germ-cell tumors, are sensitive to chemotherapy and radiation, and have the best prognosis. Low-stage seminomas are often closely observed after orchiectomy. Advanced disease is usually treated with chemotherapy.

b. Nonseminomatous germ cell tumors include embryonal carcinoma, teratoma, choriocarcinoma, and yolk sac elements. These are more likely to present with advanced disease. Patients may require **chemotherapy** and/or **retroperitoneal lymph node dissection.**

c. Nongerm cell tumors are rare and include **Leydig and Sertoli cell tumors** (90% benign) and lymphoma (common in men >50 years).

VIII. GENITOURINARY TRAUMA

A. Renal Trauma

1. Evaluation (Figure 43-1)

2. Grading of renal Injury (Fig. 43-2, Table 43-8).

3. Management:

a. Grade I to IV Injuries can usually be **observed** with serial CBCs, bedrest until hematuria resolves, **repeat imaging in 36 to 72 hours (Grade III to IV injuries),** and long-term **blood pressure checks.**

b. Vascular Grade IV or **Grade V Injuries** may require renal **embolization** or renal **exploration with possible nephrectomy.**

c. Absolute indications for intervention include hemodynamic instability, persistent hemorrhage from renal injury, expanding or pulsatile perirenal mass, or renal pedicle avulsion.

TABLE 43-7 AJCC 2010 TNM Staging for Testes Carcinoma

Primary Tumor (T)

pTx	Primary tumor cannot be assessed

pT0	No evidence of primary tumor
pTis	Intratubular germ cell neoplasia
pT1	Tumor limited to testis and epididymis without vascular/lymphatic invasion; tumor may invade the tunica albuginea but not the tunica vaginalis
pT2	Tumor limited to testis and epididymis with vascular/lymphatic invasion, or tumor extending through into the tunica vaginalis
pT3	Tumor invades the spermatic cord with or without vascular/lymphatic invasion
pT4	Tumor invades the scrotum with or without vascular/lymphatic invasion

Regional Lymph NodesÑClinical Stage (N)

Nx	Regional lymph nodes cannot be assessed
N0	No regional lymph node metastasis
N1	Metastasis with a lymph node mass 2 cm or less in greatest dimension, or multiple nodes, none greater than 2 cm in greatest dimension
N2	Lymph node mass >2 cm but <5 cm or multiple nodes with one mass >2 cm, but none >5 cm
N3	Lymph node mass >5 cm in greatest dimension

Distant Metastasis (M)

M0	No distant metastasis
M1	Distant metastasis

M1a Non-regional nodal or pulmonary metastasis

M1b Distant metastasis other than to non-regional nodes and lung

Serum Tumor Markers (S)

S0 Marker study levels within normal limits

S1 LDH × normal and hCG <5,000 and AFP <1,000

S2 LDH 1.5 to 10× normal or hCG 5-50,000 or AFP 1,000-10,000

S3 LDH > 10× normal or hCG >50,000, or AFP >10,000

Sx Marker studies not available or not performed

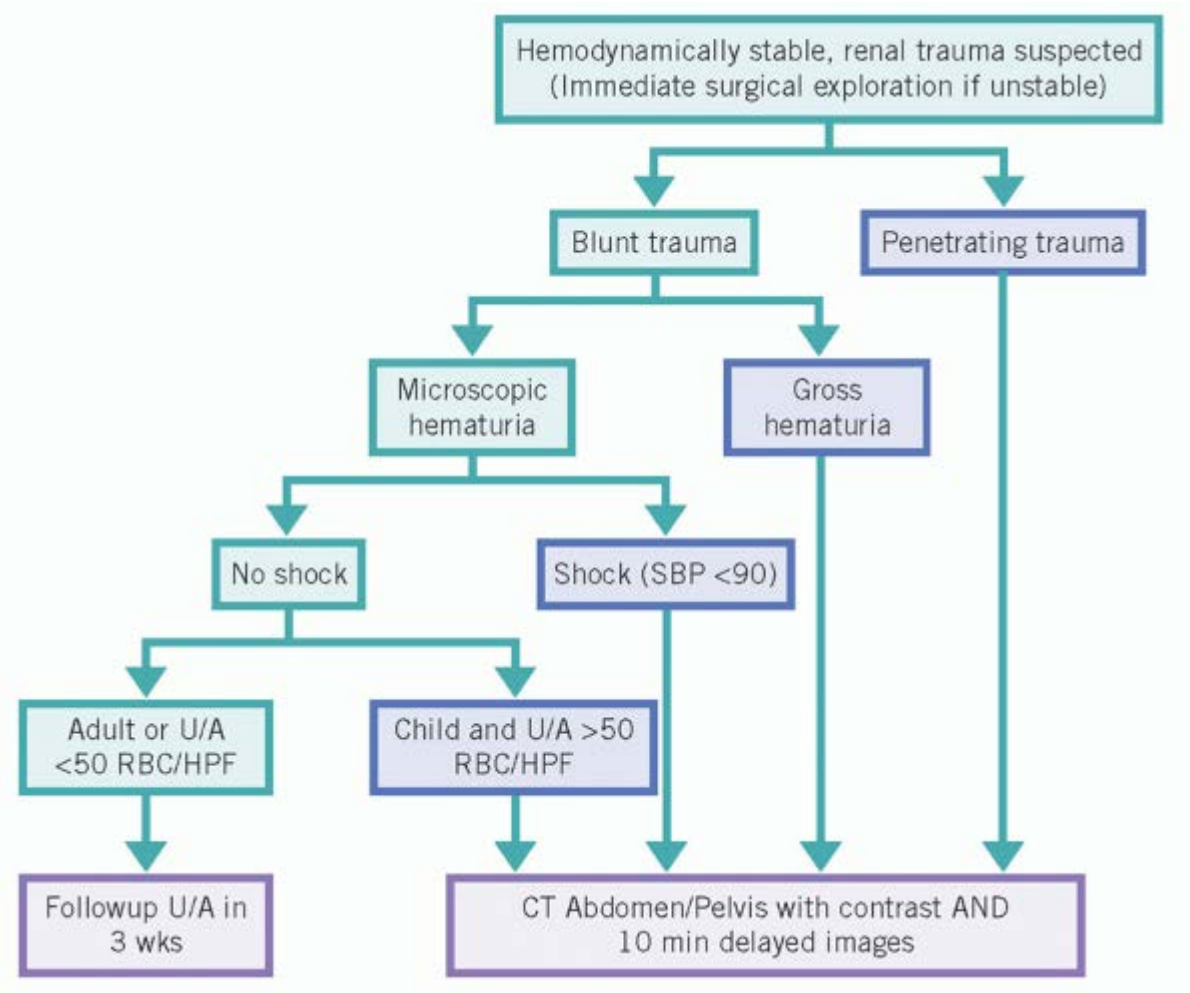

Figure 43-1 Algorithm for the Management of Suspected Renal Trauma. (Adapted from Wieder, J. *Pocket guide to urology.* 4th ed.)

B. Ureteral injuries are associated with penetrating and multiorgan trauma. A high index of suspicion is often necessary to make the diagnosis, and many ureteral injuries have a delayed presentation. Up to 30% of patients do not have hematuria.

1. Radiographic findings include **extravasation,** lack of contrast in the distal ureter, proximal dilation, and deviation of the ureter. *Delayed CT images* are necessary to assess ureteral integrity.

2. Adequately visualizing the ureter during laparotomy is important for diagnosing ureteral injury; **IV injection of indigo carmine or methylene blue,** which are excreted in the urine, may help.

3. Most ureteral injuries (minor extravasation or ureteral damage without extravasation) can be managed with **ureteral stent placement.**

4. Management of more severe injuries may require **ureteroureterostomy** or **ureteral**

reimplantation, depending on location.

C. Bladder Injuries

1. The majority of bladder injuries present with **gross hematuria.** *CT cystogram* is necessary in any patient with gross hematuria and pelvic fracture and should be considered for (i) patients with recent pelvic trauma and hematuria or suprapubic pain or (ii) pelvic fracture without hematuria.

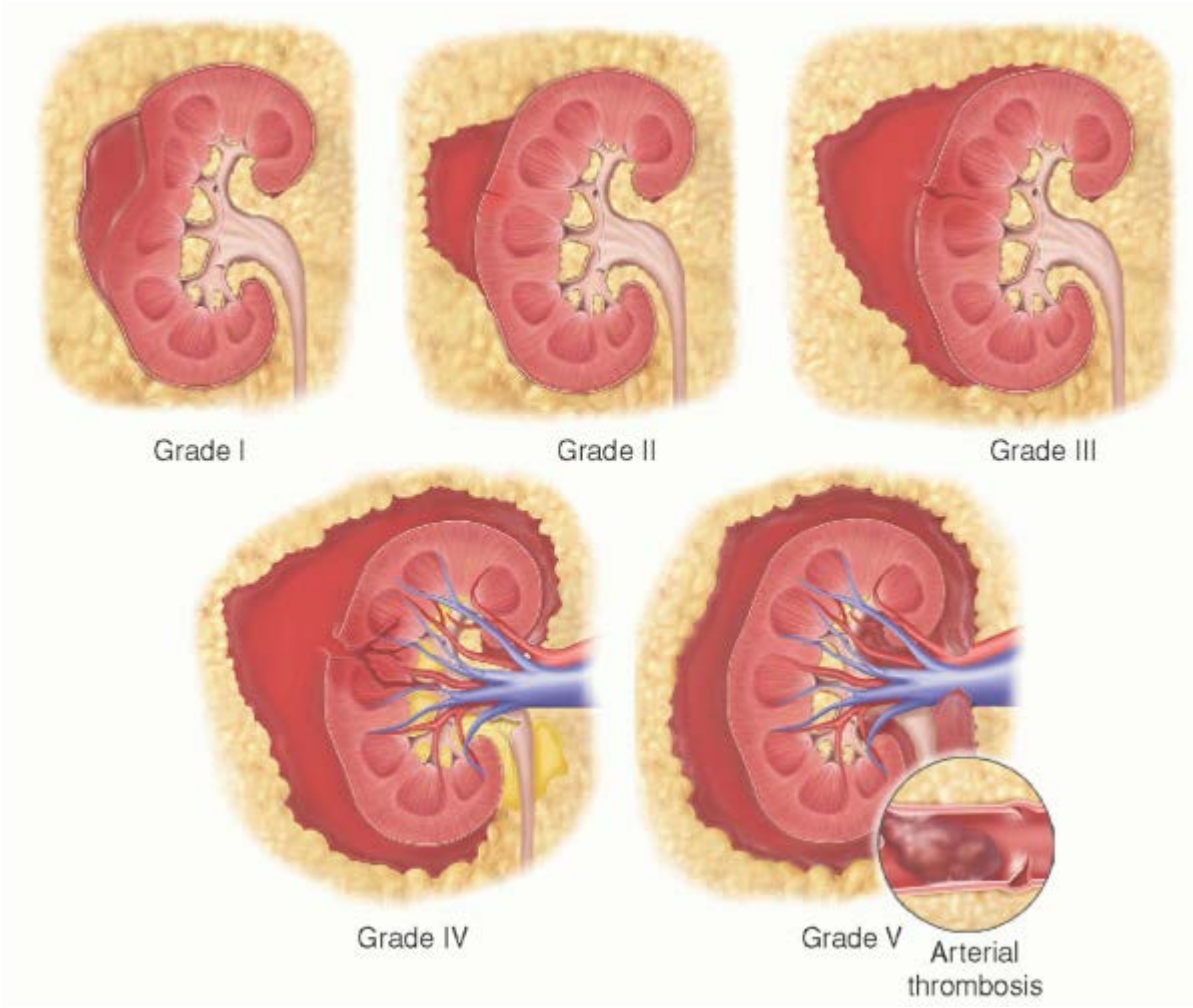

Figure 43-2 American Association for the Surgery of Trauma Grading. (From Britt LD, et al., eds. *Acute Care Surgery.* Philadelphia, PA: Wolters Kluwer Health, 2012.)

2. CT cystogram is the most sensitive imaging modality for bladder injury. The bladder should be filled retrograde by gravity via a catheter. Filling with excreted contrast does not constitute an adequate study.

3. Treatment

a. Patients with **intraperitoneal extravasation** of contrast require **surgical exploration and repair** of the bladder.

b. Patients with **extraperitoneal extravasation** of contrast can be managed nonoperatively initially with **catheter drainage** for 10 to

14 days. A cystogram should be performed prior to catheter removal with postdrainage films.

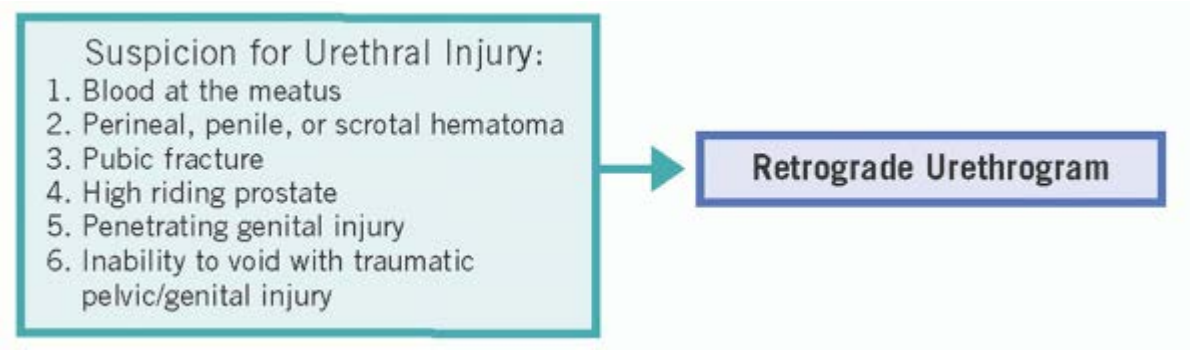

Figure 43-3 Workup for suspected urethral injury. (Adapted from Wieder, J. *Pocket guide to urology.* 4th ed.)

TABLE 43-8 American Association for the Surgery of Trauma Grading

Grade	Type	Description
I	Contusion	No laceration
	Hematoma	Subcapsular, nonexpanding
II	Hematoma	Nonexpanding perirenal hematoma
	Laceration	<1 cm parenchymal depth of renal cortex without urinary

		extravasation
III	Laceration	>1 cm parenchymal depth of renal cortex without collecting system rupture or urinary extravasation
IV	Laceration Vascular	Parenchymal laceration extending through renal cortex, medulla, and collecting system Main renal artery or vein injury with contained hemorrhage
V	Laceration Vascular	Completely shattered kidney Avulsion of renal hilum, devascularizing the kidney

D. Urethral Injuries

1. Posterior urethral injuries involve the **prostatic and membranous urethra** (from the bladder neck to the external sphincter).

a. Acute management involves urologic consultation with careful urethral catheterization.

b. If **catheterization is unsuccessful, recommend suprapubic catheter placement with attempt at cystoscopic primary realignment within 72 hours.**

c. Surgical repair of a posterior urethral injury is *not recommended* **in the acute setting** as it is complicated by higher rates of impotence, incontinence, and stricture (*Urol Clin North Am.* 2006;33:87-95).

d. Delayed repair in 3 to 6 months is recommended.

2. Anterior urethral injuries include injuries to the **bulbous and penile urethra distal to external sphincter.** Examination may show diffuse penile ecchymosis or a Ò**butterflyÓ pattern of perineal ecchymosis.** The 2014 AUA Urotrauma guidelines recommend **immediate surgical repair** for anterior injuries unless the patient is unstable or there is concern about tissue viability.

E. Penile Trauma

1. Penile fracture occurs when excessive bending force is applied to the erect penis resulting in a tear of the tunica albuginea. Patients describe an auditory ÒpopÓ heard during intercourse followed by rapid detumescence and swelling of the penis/scrotum. Inability to void or blood at the meatus indicates concomitant urethral injury (20% of cases).

a. Examination demonstrates edema and ecchymosis/hematoma of the penis (Òeggplant deformityÓ) and/or perineum.

b. Imaging is not indicated, unless urethral injury is suspected.

c. Early surgical exploration (<36 hours) with repair is the standard of care and is associated with better outcomes than delayed repair.

2. Serious blunt or penetrating trauma with injury to the corpus cavernosum requires surgical exploration and repair. It is necessary to rule out urethral injury.

F. Testicular Injury

1. Surgical exploration is required for all penetrating scrotal trauma deep to the dartos fascia or if there is concern for **testicular rupture.**

2. Ultrasonography can help to diagnose traumatic testicular injury with a 100% sensitivity and 93.5% specificity (*J Urol.* 2006;175:175-178).

3. Traumatic testicular repair consists of **hematoma evacuation, debridement of the necrotic tubules,** and **closure of the tunica albuginea.** Orchiectomy is *rarely indicated.*

G. Scrotal avulsion with skin loss should be copiously irrigated and debrided. Clean wounds may be closed in layers, whereas grossly contaminated wounds should be left open to heal by secondary intention.

CHAPTER 43: UROLOGY FOR THE GENERAL SURGEON

Multiple Choice Questions

1. A 72-year-old male presents to you with a complaint of mild right lower back pain and one episode of blood in his urine 2 weeks ago. He denies voiding symptoms. He quit smoking 5 years ago. His creatinine is 0.8. What are the next step(s) in management?

a. Urine culture, noncontrast (stone protocol) CT abdomen/pelvis

b. Urine culture, urine cytology, renal/bladder ultrasound

c. Renal/bladder ultrasound, cystoscopy with bilateral retrograde pyelograms

d. Urine culture, urine cytology, CT urogram, cystoscopy

e. No workup needed unless he has another episode of hematuria

View Answer

2. A 45-year-old woman with a history of diverticulitis comes to the Emergency Room (ER) with a 2-day history of left lower-quadrant pain and vomiting. She has mild leukocytosis with a white blood cell (WBC) count of 15. Her creatinine is mildly elevated to 1.3, consistent with

dehydration. In the ER, she develops a fever of 38.5°C. Urinalysis shows positive leukocyte esterase but no nitrites. A CT scan reveals a 6-mm left ureteral stone with mild to moderate hydronephrosis. What are the next best step(s) in management?

a. Urine culture, antibiotics, IV fluids

b. Urine culture and stat Urology consult for left ureteral stent placement

c. Bowel rest (make NPO) and IV fluids

d. Urine culture, antibiotics, and Urology consult for a possible left ureteroscopy

e. Discharge home with oral antibiotics and followup with Urology

View Answer

3. A 62-year-old male presents with urinary retention and perirectal pain. He has had recent low-grade fevers, urinary urgency, and increasing difficulties voiding until he was unable to void at all this morning. Digital rectal exam reveals a swollen, boggy prostate that is tender on examination. What is the likely diagnosis?

a. Benign prostatic hyperplasia (BPH)

b. Urinary tract infection

c. Bacterial prostatitis

d. Prostatic abscess

View Answer

4. Which of the following statements about priapism management is FALSE?

a. Any form of priapism constitutes a urologic emergency

b. First-line treatment for priapism caused by Sickle Cell Disease is supplemental oxygen and hydration

c. Delay in priapism treatment may result in permanent erectile dysfunction

d. Ischemic priapism is treated with intracavernosal injections of phenylephrine

e. Ischemic priapism is typically caused by medications or illegal drug use

View Answer

5. A 35-year-old obese male presents to the Emergency Room with concern for perirectal abscess. He recently developed increasing

perianal pain with fevers. He has had perianal abscesses before which required incision and drainage. On examination he has a 1.5-cm palpable perianal fluctuant collection consistent with an abscess. The fluid appears to track up the perineum. The perineum and inferior scrotum are moist, erythematous, and edematous. His WBC is 18.5. A CT of the abdomen/pelvis shows a perianal abscess tracking to the perineum with a few flecks of gas and edema of the scrotal wall. What is the most appropriate management at this time?

a. IV antibiotics and admission for observation

b. Bedside I&D of the perirectal abscess with packing

c. Bedside I&D of the perirectal abscess with antibiotics for scrotal cellulitis

d. Admission with IV antibiotics and abscess I&D in the OR in the morning

e. Immediate surgical exploration and debridement

View Answer

6. TRUE or FALSE: Most traumatic renal injuries do not require any surgical intervention.

View Answer

7. A 25-year-old male was in an ATV accident and presents with a shattered pelvis with bruising extending to his perineum. In the ER, he is unable to void. There is no blood at the meatus. What are the appropriate next steps in management?

a. Carefully place a Foley catheter. If there is hematuria, obtain a CT urogram.

b. Obtain a retrograde urethrogram and CT urogram.

c. Obtain a retrograde urethrogram; if negative, place a Foley and obtain a CT cystogram.

d. Place a suprapubic tube and obtain a cystogram.

View Answer

44

Obstetrics and Gynecology for the General Surgeon

Ivy Wilkinson-Ryan

Andrea R. Hagemann

I. OBSTETRIC AND GYNECOLOGIC DISORDERS.

Vaginal Bleeding (Fig. 44-1, Table 44-1). Gather a thorough history including pattern and intensity of bleeding and date of last menstrual period. Obtain urine beta-human chorionic gonadotropin (β-hCG) level, complete blood count (CBC), coagulation studies and blood type. Physical examination should include a speculum and bimanual pelvic exam. Pelvic and transvaginal ultrasound (U/S) is the most sensitive imaging modality for pelvic organs in the pregnant and non-pregnant patient.

A. Obstetric Etiologies. Forty percent of all pregnancies are associated with vaginal bleeding and approximately half of these result in spontaneous abortions (SABs).

First trimester: SABs, postcoital bleeding, ectopic pregnancy (see Section IIA Nonobstetric surgery in the pregnant patient, Perioperative considerations), lower genital tract lesions/lacerations, and expulsion of a molar pregnancy.

Third trimester: Placenta previa, placental abruption, vasa previa, preterm labor, and lower genital tract lesions/lacerations.

1. Terminology

a. Threatened abortion: Any vaginal bleeding <20 weeks of gestation without expulsion of products of contraception (POCs); cervix closed

b. Missed abortion: Nonviable gestation <20 weeks of gestation with retention of POCs; cervix closed

c. Inevitable abortion: Cervical dilation with or without ruptured membranes

d. Incomplete abortion: Partial passage of POCs; cervix open

e. Complete abortion: Expulsion of all POCs; cervix closed

f. Septic abortion: Retained infected POC

2. Transvaginal ultrasound

a. Intrauterine gestation seen with β-hCG >2,000 mIU/mL

b. Cardiac activity seen with β-hCG >10,000 mIU/mL

3. Treatment

a. Threatened abortion

(1) Viable: Expectant management

(2) Indeterminate viability: Repeat U/S in 7 days, repeat β-hCG in 48 hours.

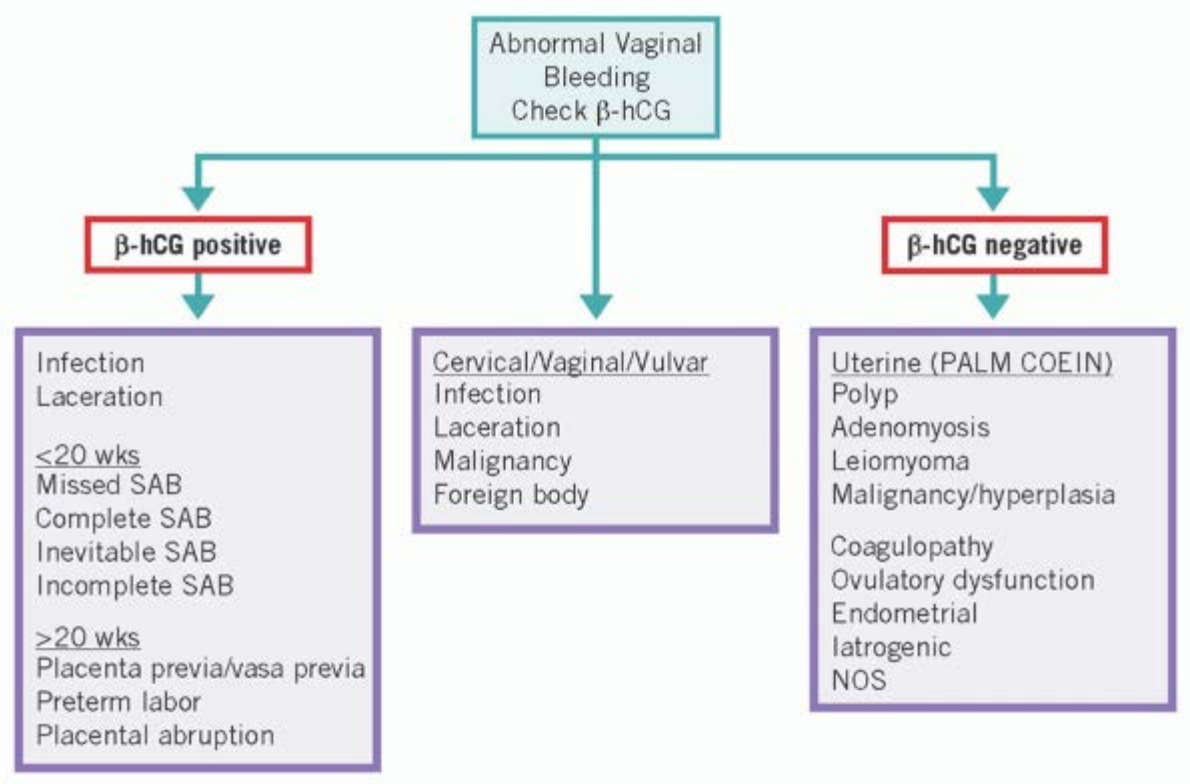

Figure 44-1 Management of Vaginal Bleeding.

b. Missed / Inevitable / Incomplete abortion. Expectant management, surgical management (Dilation and Evacuation [D&E]), or medical therapy (Misoprostol 400 µg vaginally q4h × 4 doses) are acceptable. Give bleeding precautions (return to emergency room (ER) or call a physician for bleeding soaking 1 pad/hour) and infection precautions (return to ER or call a physician for temperature of 101¡F).

c. Complete abortion. If hemodynamically stable, expectant management is appropriate.

d. RhoGAM (50 µg intramuscular [IM]) to any pregnant patient with vaginal bleeding who is Rh negative with a negative antibody screen.

B. Nonobstetric Etiologies of Vaginal Bleeding. See Figure 44-1 for diagnostic workup and Table 44-1 for treatment options.

C. Risks and Complications of D&E

1. Uterine perforation: Perform laparoscopy/cystoscopy if concerned for bowel/bladder injury. Manage expectantly if hemodynamically stable and no injury to surrounding organs.

2. Endometritis: If unrelated to gonorrhea/chlamydia, Doxycycline 100 mg BID × 7 days. If high level of suspicion for gonorrhea/chlamydia infection, use pelvic inflammatory disease (PID) treatment regimen.

II. NONOBSTETRIC SURGERY IN THE PREGNANT PATIENT.

If nonemergent, it is **safest to proceed with surgery in the second trimester.** Surgery in the first trimester carries a risk of spontaneous abortion. Surgery in the third trimester carries a risk of inducing preterm labor and injury to the enlarging uterus. Risk of pregnancy interruption (any trimester) from nonobstetric

surgery in the pregnant patient is approximately 3% to 11%. Pregnancy is not a contraindication to indicated/emergent surgery (*Clin Obstet Gynecol.* 2009;52:586-596). Once the need for surgery has been identified, communication with the obstetrics and anesthesia teams is critical. The age and weight threshold for viability is controversial and may differ between institutions. Traditionally, 24 weeks or 500 g is used as the cut-off for fetal viability. However, some institutions are offering interventions after 23 weeks or 400 g.

TABLE 44-1 Nonobstetric Causes of Vaginal Bleeding

Differential Diagnosis	Laboratory Data	Signs and Symptoms	Treatment
Menses	CBC count, urine hCG	Cyclic bleeding every 21-35 days	Iron therapy if indicated
Dysfunctional uterine bleeding	CBC count, urine hCG, endometrial biopsy if >45	Non-cyclic bleeding; may have associated	Hormonal therapy if patient is hemodynamically stable; if unstable,

	years old, or any age with risk factors	dysmenorrhea, fatigue, or dizziness	transfuse as needed, IV estrogen or high-dose OCPs
gonorrhea/*Chlamydia* cervicitis	Cervical culture, wet prep	Purulent vaginal discharge, possible spotting	Ceftriaxone, 125 mg IM × 1; azithromycin, 1 g PO × 1
Trichomonas vaginitis	Wet prep	Yellow-green frothy vaginal discharge, possible spotting	Metronidazole, 500 mg PO bid × 7 days or 2 g PO × 1 (if pregnant, defer until second trimester)
Sexual trauma	Rape kit	Vaginal bleeding and/or discharge	Emergency contraception, prophylactic treatment for STDs; if laceration, pack vagina, possible surgical repair

bid, twice daily; CBC, complete blood cell; hCG, human chorionic gonadotropin; OCPs, oral contraceptive pills; STDs, sexually transmitted diseases; IM, intramuscular; IV, intravenous; PO, oral.

A. Perioperative Considerations

1. Increased risk for aspiration results from upward displacement of the stomach and the inhibitory effects of progesterone on gastrointestinal

motility during pregnancy. Nonparticulate antacids should be given prior to induction of anesthesia (*J Clin Anesth.* 2006;18:60-66).

2. Vena cava compression: Maintain left lateral positioning in third trimester to decrease IVC compression and maintain adequate maternal circulation and placental blood supply (*J Clin Anesth.* 2006;18:60-66).

3. Fetal monitoring: If previable (<23 weeks, <400 g), Doptone maternal abdomen prior to and following the procedure. If pregnancy is viable, intraoperative continuous fetal monitoring and uterine contraction monitoring is recommended if the following conditions are met: (1) Monitoring is compatible with patient position and planned procedure, (2) there are appropriate staff in the operating room (OR) to interpret fetal monitoring, and (3) there are appropriate staff immediately available to perform an emergency cesarean section. If intraoperative continuous monitoring is not feasible, monitor fetal heart rate and uterine contractions pre- and postoperatively. (ACOG Committee Opinion Number 474, Feb 2011, http://www.acog.org/-/media/Committee-Opinions/Committee-on-Obstetric-Practice/co474.pdf?dmc = 1&ts = 20141013T1551357449. *Clin Obstet Gynecol.* 2009;52:586-96, PMID: 20393411.)

4. Antenatal steroids: If gestation is between 23 and 34 weeks, start antenatal steroids (Betamethasone 12 mg IM q24h × 2 doses) prior to proceeding to OR to decrease risk of respiratory distress syndrome, intraventricular hemorrhage, necrotizing enterocolitis, neonatal infection, and neonatal mortality (*NIH Consensus Statement.* 1994;12:1-24).

B. Laparoscopy in Pregnancy. Based on the Society of American Gastrointestinal Endoscopic Surgeons (SAGES) Guidelines of Laparoscopic Surgery During Pregnancy (*Surg Endosc.* 1998;12:189), laparoscopy is safe and has similar advantages over open surgery as in the nonpregnant patient. Initial trocar placement is best accomplished by an open Hasson technique and/or left upper quadrant entry. Avoid Veress needle entry. Carbon dioxide pneumoperitoneum up to 15 mm Hg is safe and is unlikely to result in fetal hypoxia or acidosis. Avoid cervical or uterine manipulation.

III. GYNECOLOGIC SURGICAL ANATOMY

A. Uterus and Cervix. The primary blood supply to the uterus and cervix comes from the uterine branch of the hypogastric artery. After passing through the cardinal ligament the uterine artery has ascending and descending branches (Fig. 44-2). The uterine suspensory ligaments include the uterosacral ligaments, cardinal ligaments, round ligaments, and utero-ovarian ligaments. The ureter travels through the cardinal ligament passing under the uterine artery and vein within the ureteric tunnel. Hypogastric or uterine artery ligation either surgically or by interventional radiology (IR) can be used to treat uncontrolled uterine hemorrhage. The uterus can be amputated from the cervix and the cervix can be left *in situ,* for instance in a supracervical hysterectomy, or the cervix can be removed with the uterus in the case of a total abdominal hysterectomy.

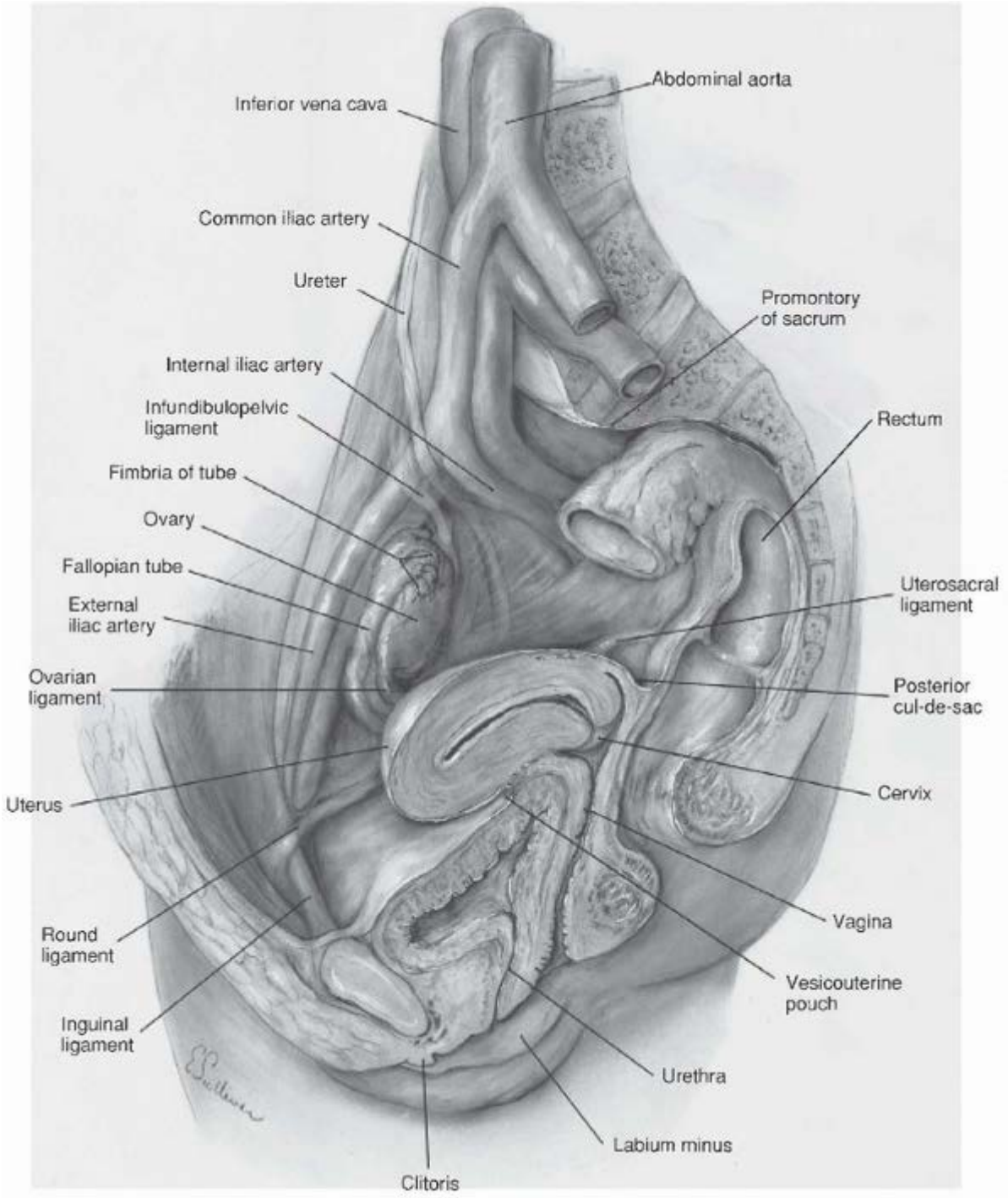

Figure 44-2 Pelvic viscera. (From John A Rock, Howard W Jones, eds. *Te Linde's Operative Gynecology,* 10th ed. Philadelphia, PA: Wolters Kluwer Health, 2008.)

B. Ovary. The ovary receives its primary blood supply from the gonadal arteries, which branch off the aorta inferior to the celiac plexus and renal vessels and superior to the inferior mesenteric artery. The left ovarian vein drains into the left renal vein. The right ovarian vein empties into the vena cava. The gonadal vessels travel retroperitoneally within the infundibulopelvic ligament. The infundibulopelvic ligament and gonadal vessels pass over the ureter approximately at the

bifurcation of the iliac vessel into external and internal branches as the ureter crosses the pelvic brim and descends into the pelvis. The ovary attaches to the uterus via the utero-ovarian ligament.

C. Bladder. In order to safely perform a hysterectomy, the bladder must be dissected off the uterus and cervix. This is done by incising the vesicouterine peritoneum down to the pubocervical fascia and opening the vesicovaginal space. Fat and vascular tissue stays with the bladder during this dissection. If the plane is obscured by prior cesarean section, the plane can often be established distally and then developed in a retrograde manner to avoid bladder injury.

D. Rectum. The posterior cul-de-sac can be obliterated by adhesion, inflammatory diseases, and malignancy. In these circumstances, the rectovaginal septum can be developed by incising the posterior uterus just caudad to the attachment of the uterosacral ligament to the uterus. The plane can be dissected bluntly, thus dropping the rectum away from the uterus, cervix, and vagina to isolate the structures.

E. Pelvic Avascular Spaces (Fig. 44-3). Developing the avascular spaces allows for identification of the major vessels and ureter, palpation of the parametrial tissue (an important step in evaluation of pelvic masses and gynecologic malignancies), and separation of pelvic organs.

1. Prevesical (also called **space of Retzius):** Lies between posterior pubic bone and anterior wall of the bladder.

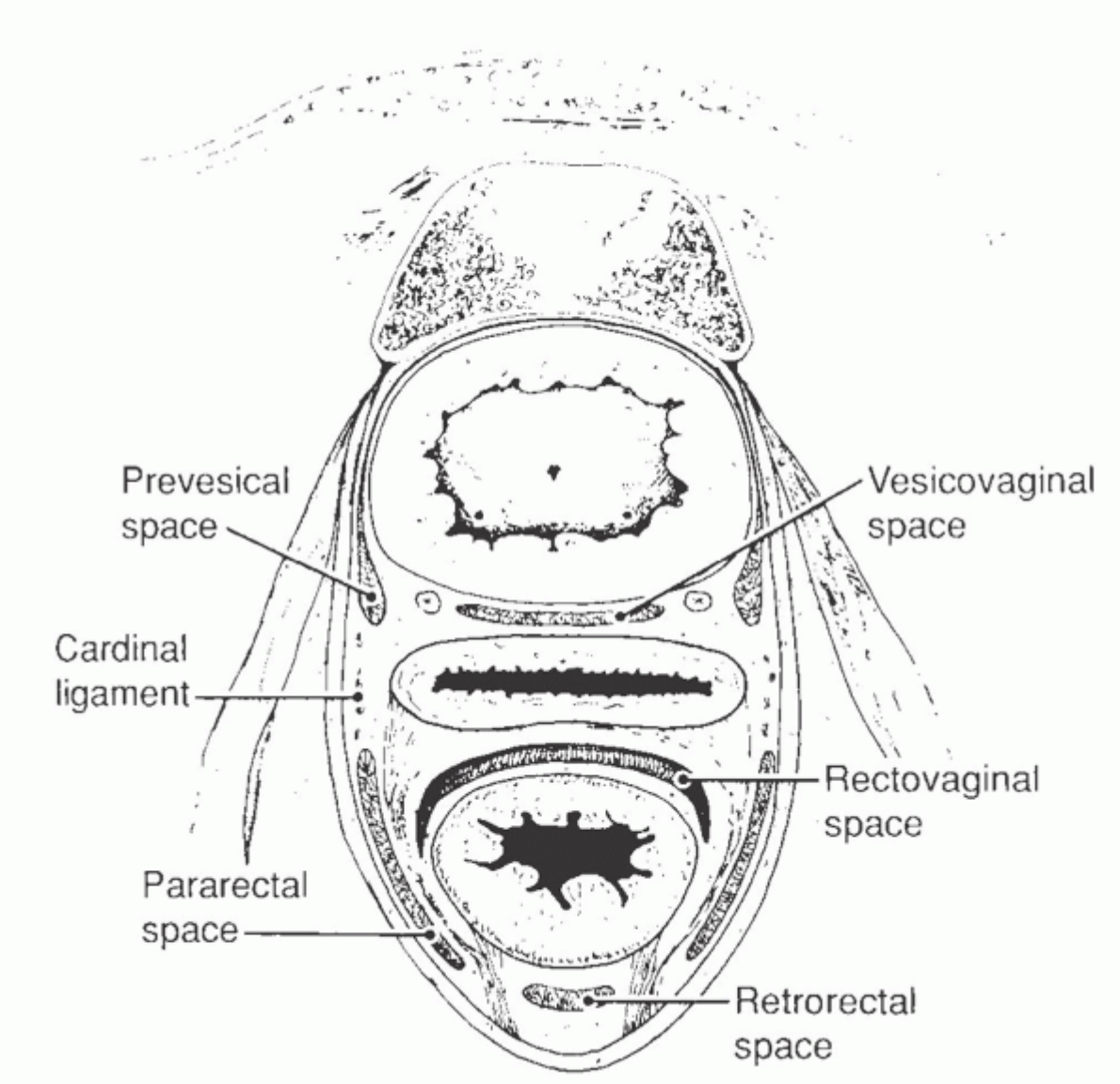

Figure 44-3 Cross-section of the pelvis showing avascular planes. (From John A Rock, Howard W Jones, eds. *Te Linde's Operative Gynecology,* 10th ed. Philadelphia, PA: Wolters Kluwer Health, 2008.)

2. Vesicovaginal space: Lies between vagina posteriorly and bladder anteriorly. Laterally defined by bladder pillars.

3. Paravesical space: Bordered by bladder pillars medially and pelvic sidewall/obturator internus laterally.

4. Pararectal space: Identified between the ureter and the hypogastric artery.

5. Rectovaginal space: Potential space between the rectum and vagina. Laterally defined by the rectal pillars/uterosacral ligaments.

IV. GYNECOLOGIC MALIGNANCIES.

Over 94,000 new cases of gynecologic malignancy were diagnosed in the United States in 2014 (*CA Cancer J Clin.* 2014;64:9-29). A brief overview of vulvar, cervical, endometrial, and ovarian cancers is presented with emphasis on diagnosis and initial management. If a gynecologic malignancy is suspected, all effort should be made to obtain an intraoperative consult with a gynecologic oncologist.

A. Vulvar Carcinoma

1. Epidemiology. There were an estimated 4,850 new cases and 1,030 deaths in the United States in 2014. The mean age at diagnosis is 68 years old. Vulvar cancer is associated with HPV in 60% of cases. Other risk factors for vulvar cancer include cigarette smoking, vulvar and cervical dysplasia, vulvar dystrophy, and immunodeficiency.

2. Presentation and clinical features. The most common symptom is itching, but lesions can be pruritic, painful, or asymptomatic. Vulvar examination typically yields an ulcerated, hyper- or hypopigmented, or exophytic lesion. A biopsy should be taken any time such a lesion is noted on the vulva.

3. Standard of care. See Table 44-2 for treatment by stage. Vulvar cancer is staged using both clinical and surgical assessment. Size of the lesion and regional or distant metastasis must be assessed. Radical vulvectomy is defined as complete resection of the lesion with at least 1 cm margins and deep excision to the inferior fascia of the urogenital diaphragm (coplanar with the fascia lata and fascia over the pubic symphysis). Adjuvant radiation is reserved for cancers confined to the vulva at high risk for recurrence or tumors that metastasize to the inguinofemoral lymph nodes (Fig. 44-4).

4. What to do if a lesion is identified pre- or intraoperatively. As above, it is essential to obtain a biopsy for pathologic diagnosis. A 3 to 5 mm Keyes punch biopsy is usually adequate.

B. Cervical Carcinoma

1. Epidemiology. There were an estimated 12,360 new cases and 4,020 deaths in the United States in 2014. The mean age of diagnosis is 48 years old. HPV is detected in over 99% of cervical cancer cases. Other risk factors for cervical cancer include cigarette smoking, immunosuppression, history of multiple sexual partners, early sexual debut (i.e., onset of sexual activity), history of sexually transmitted infections (STIs), and vulvar or vaginal dysplasia.

TABLE 44-2 Vulvar Cancer Staging

TNM	FIGO	Definition	Treatment
T1	I	Tumor confined to the vulva	
T1a	IA	Lesions ²2 cm in size with stromal invasion ²1 mm	Radical excision
T1b	IB	Lesions >2 cm in size or with stromal invasion >1 mm	Radical excision and inguinofemoral lymphadenectomy Ipsilateral lymphadenectomy if >2 cm from midline Bilateral lymphadenectomy if <2 cm from midline Resection of distal 1 cm or urethra for periurethral lesions Sentinel lymph node resection acceptable for palpably negative inguinal lymph nodes
T2	II	Tumor of any size, extending to adjacent perineal structures (lower one-third urethra, lower one-third vagina, anus) with negative nodes	Stage IIÑIVA radical excision and bilateral inguinofemoral lymphadenectomy if resection is feasible Primary radiation therapy for unresectable lesions
T3	III	Tumor of any size with or without extension to adjacent perineal structures (lower onethird urethra, lower one-third vagina, anus) with positive inguinofemoral lymph nodes	

T3a	IIIA(i)	With 1 lymph node metastasis (³5 mm)	
	IIIA(ii)	1-2 lymph node metastasis(es) (<5 mm)	
T3b	IIIB(i)	With ³2 lymph node metastasis (³5 mm)	
	IIIB(ii)	³3 lymph node metastasis(es) (<5 mm)	
T3c	IIIC	With positive lymph nodes with extracapsular spread	
T4	IVA(i)	Invades two-third upper urethral, two-thirds upper vagina, bladder/rectal mucosa, or fixed to pelvic bone, or	
	IVA(ii)	Fixed or ulcerated inguinofemoral lymph nodes	
M1	IVB	Distant metastasis including pelvic nodes	Primary radiation if all disease within treatment field Systemic chemotherapy (taxane + platinum) if not a candidate for primary radiation, with radiation for symptom control only

FIGO, International Federation of Gynecology and Obstetrics; TNM, tumor, node, metastasis. Used with the permission of the American Joint Committee on Cancer (AJCC), Chicago, Illinois. The original source for this material is the AJCC Cancer Staging Manual, Seventh Edition (2010) published by Springer Science and Business Media LLC,

www.springer.com.

2. Presentation and clinical features. Irregular or postcoital vaginal bleeding and malodorous, watery discharge are the most common symptoms. Advanced stage disease may present with leg pain (sciatic nerve involvement), flank pain (ureteral obstruction), renal failure, or rectal bleeding from a pelvic mass.

3. Standard of care. See Table 44-3 for treatment by stage. **Cervical cancer is clinically, not surgically, staged.** The World Health Organization (WHO) recognizes a standard worldwide staging system for cervical cancer that includes a pelvic examination, x-ray, intravenous pyelogram, endoscopy (hysteroscopy, cystoscopy, proctoscopy), and cervical biopsy. A speculum should be placed prior to a bimanual exam to obtain visualization and approximate measurements of the tumor. Rectovaginal examination is essential to characterize parametrial involvement. In the

United States, a positron emission tomography (PET) scan, magnetic resonance imaging (MRI), or computerized tomography (CT) is typically performed to assess for pelvic or para-aortic lymphadenopathy and guide treatment planning, but this does not change clinical stage as it is not available worldwide.

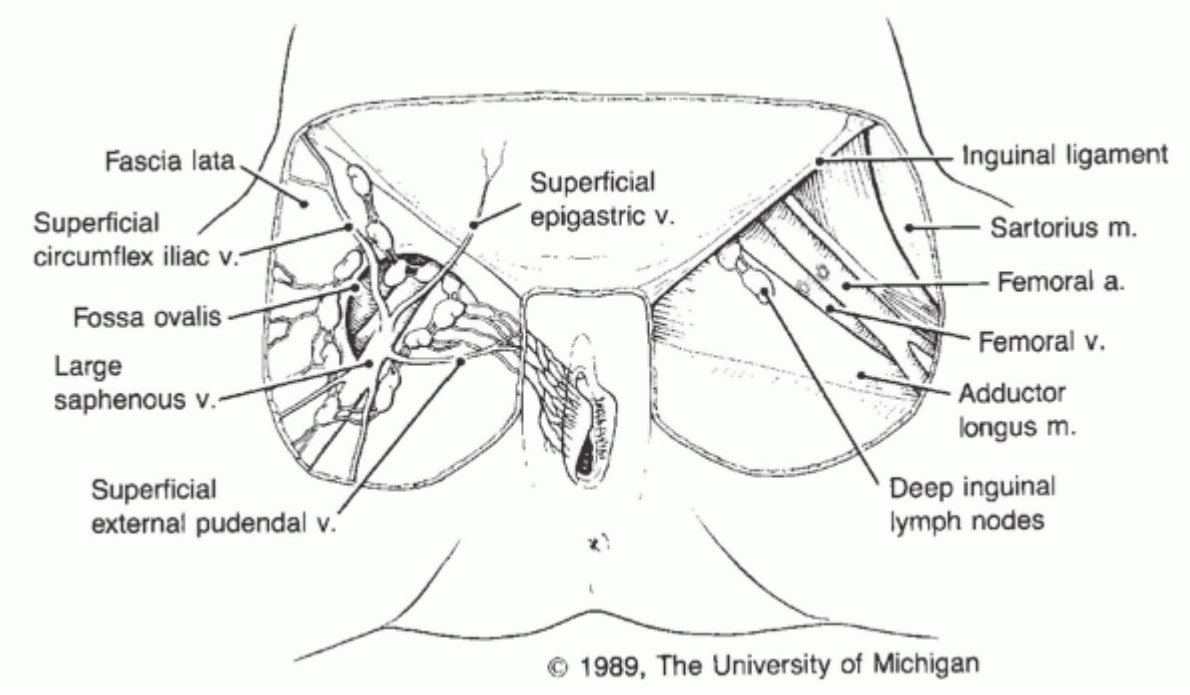

Figure 44-4 Lymphatic drainage of the vulva and femoral triangle. (From John A Rock, Howard W Jones, eds. *Te Linde's Operative Gynecology,* 10th ed. Philadelphia, PA: Wolters

Kluwer Health, 2008.)

Early-stage cervical cancers (Stage IA1-IB1) can often be cured by surgical management alone. For patients with early-stage disease desiring fertility, a *loop electrosurgical excision procedure* (LEEP) or cone biopsy with negative margins (Stage IA1) or a radical cervicectomy (also known as trachelectomy; Stage IB1) may be recommended. While surgery is an option for bulkier early-stage cancers (Stage IB2-IIA), the benefits must be weighed against the potential risks of vesicovaginal or rectovaginal fistula formation from subsequent radiation; chemoradiation without surgery is often a reasonable option. Locally advanced cervical cancers (Stage IIB-IVA) should be treated with radiation and sensitizing cisplatin. Cervical cancers with distant spread can be treated with combination taxane and platinum chemotherapy in combination with anti-VEGF-A monoclonal antibody bevacizumab (Avastin, Genentech/Roche). In the case of distant spread, radiation is used for palliation of bleeding or potentially for isolated supraclavicular lymphadenopathy. Pelvic exenteration with removal of the bladder and rectum is reserved for recurrent cervical cancer localized to the pelvis.

4. What to do if suspected pre- or intraoperatively. Obtain a biopsy. Perform a speculum examination and use a Tischler biopsy forceps to obtain a biopsy of cervix or malignant-appearing tissue. Do not attempt to resect bulky cervical cancer.

TABLE 44-3 Cervical Cancer Staging

TNM	FIGO	Definition	Treatment
T1	I	Cervical carcinoma confined to the cervix (disregard extension to the corpus).	
T1a	IA	Preclinical invasive carcinoma, diagnosed by microscopy only. Deepest invasions ≤5 mm and largest extension ≤7 mm	
T1a1	IA1	Microscopic stromal invasion ≤3 mm in depth and extension ≤7 mm	Simple hysterectomy LEEP or cone biopsy (margins negative) if

			fertility desired
T1a2	IA2	Tumor with stromal invasion between 3 and 5 mm in depth and extension <7 mm	Radical hysterectomy and pelvic lymphadenectomy
T1b	IB	Clinically visible tumor confined to the cervix but larger than IA2	
T1b1	IB1	Clinical lesions ²4 cm in size	Radical hysterectomy and pelvic lymphadenectomy or primary radiation
T1b2	IB2	Clinical lesions >4 cm in size	Stage 1B2-IIA primary radiation with sensitizing cisplatin or radical hysterectomy with pelvic lymphadenectomy
T2	II	Invades beyond the cervix but not to the pelvic side wall or the lower one-third of the vagina	
T2a	IIA	Tumor without parametrial involvement	
T2a1	IIA1	Tumor ²4 cm	
T2a2	IIA2	Tumor >4 cm	
T2b	IIB	Tumor with parametrial involvement	Stage IIBÑIVA Primary radiation with sensitizing cisplatin
T3	III	Extends to the pelvic side wall and/or involves the lower one-third of the vagina and/or causes	

		hydronephrosis or nonfunctioning kidney	
T3a	IIIA	Invades lower one-third of the vagina with no extension to the pelvic side wall	
T3b	IIIB	Extends to the pelvic side wall and/or causes hydronephrosis or a nonfunctioning kidney	
T4	IVA	Invades mucosa of the bladder/rectum and/or extends beyond the true pelvis	
M1	IVB	Distant metastasis	Taxane, Platinum, Bevacizumab with or without pelvic radiation for symptom control

5. Uncontrolled vaginal bleeding. Heavy vaginal bleeding from cervical cancer can occasionally be encountered in the emergency department. Place a tight vaginal packing and a transurethral Foley catheter. Acetone-soaked gauze is the most effective packing for vessel sclerosis and control of hemorrhage from necrotic tumor. If ineffective at controlling bleeding, IR embolization may be necessary. Prompt consultation with radiation oncology is also indicated once a biopsy has confirmed invasive cancer.

C. Endometrial Cancers

1. Epidemiology. Endometrial carcinoma is the most common gynecologic cancer in the United States with 52,630 new cases and 8,590 deaths in 2014. The average age of diagnosis is 61 years old. Risk factors

include excess/unopposed estrogen (as seen with obesity, medication, anovulation, early menarche, and late menopause), age, and Lynch Syndrome. Over 80% of endometrial cancers are **endometrioid adenocarcinomas,** and they are subdivided into grades 1, 2, and 3 based on the degree of glandular formation. Less common histologies include *clear cell, serous,* and *poorly differentiated.* More recently, endometrial cancers have been divided into ÒType 1Ó and ÒType 2Ó cancers. **Type 1 cancers** are thought to be related to excess estrogen and typically have endometrioid histology with a grade of 1 and 2. **Type 2 cancers** include grade 3 endometrioid, clear cell, serous, or poorly differentiated adenocarcinomas, all of which are high-grade and more aggressive and less related to excess estrogen than their more common Type 1 counterparts. **Uterine carcinosarcomas** also arise from the endometrium and are considered a poorly differentiated endometrial carcinoma. They are aggressive, having features of endometrial carcinoma as well as sarcomatous overgrowth, and are also known as malignant mixed MŸllerian tumors (MMMT). **Sarcomas** are malignancies that arise in the connective tissue/stroma (**stromal sarcomas**) or myometrium (**leiomyosarcomas**) of the uterus. They make up 4% of all uterine malignancies and are staged separately from endometrial cancers. These tumors are managed surgically, with or without adjuvant therapy.

2. Presentation and clinical features. The most common symptom is abnormal vaginal bleeding, often in a postmenopausal patient.

3. Standard of care. Endometrial cancers are staged surgically with a total abdominal hysterectomy, bilateral salpingo-oophorectomy, and bilateral pelvic and paraaortic lymphadenectomy (Fig. 44-5). Omental biopsy should be performed for high-grade cancers. The role of lymphadenectomy is evolving, especially for lower grade and earlier stage cancers. Adjuvant or postoperative treatment recommendations are outlined in Table 44-4 by stage.

4. What to do if suspected pre- or intraoperatively. If suspected preoperatively, obtain an endometrial biopsy using either a curette or a biopsy Pipelle. Endometrial biopsy should be performed in a any woman with postmenopausal vaginal bleeding, any woman >45 years old with abnormal uterine bleeding, or women of any age with abnormal bleeding and risk factors such as obesity, polycystic ovarian syndrome, or unopposed estrogen. If suspected intraoperatively because of uterine surface disease, obtain a biopsy and send for frozen pathology. If positive for adenocarcinoma of MŸllerian origin, consult a gynecologic oncologist if available. If unavailable, proceed with total abdominal hysterectomy, bilateral salpingo-oophorectomy, and staging lymphadenectomy or close the abdomen and promptly refer to a gynecologic oncologist.

D. Ovarian Carcinoma

1. Epidemiology. Ovarian cancer is the most deadly gynecologic malignancy in the United States with 21,980 new cases and 14,270 deaths in 2014. The average age at diagnosis is 63 years old. Risk factors include age, *BRCA1* or *BRCA2* mutation, early menarche, late menopause, nulliparity, endometriosis, and infertility. Efforts to decrease lifetime ovulation (birth control or

pregnancy) decrease the risk for ovarian cancer.

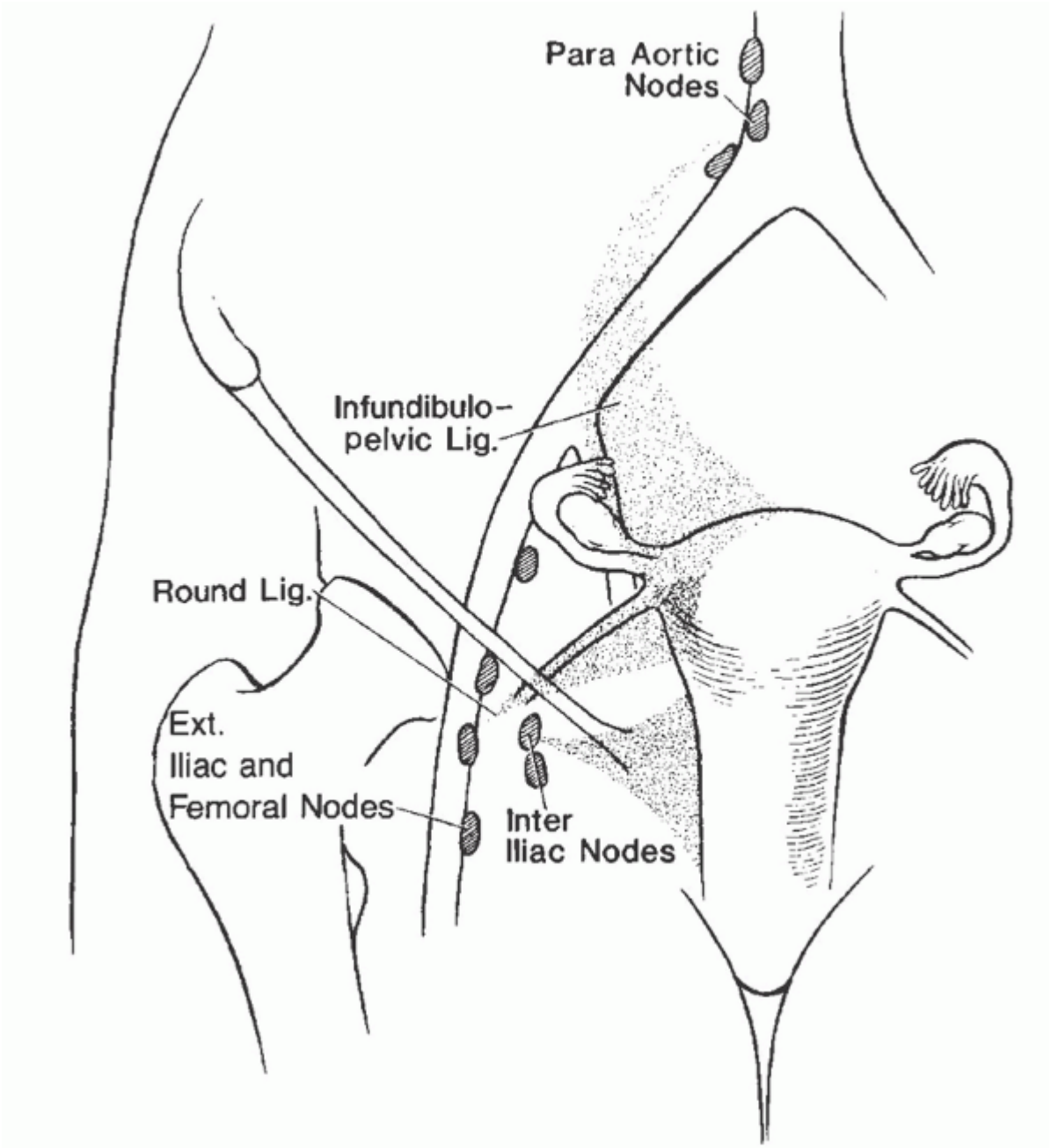

Figure 44-5 Lymphatic pathways of tumor spread of endometrial carcinoma to pelvic and extrapelvic nodes. (From John A Rock, Howard W Jones, eds. *Te Linde's Operative Gynecology,* 10th ed. Philadelphia, PA: Wolters Kluwer Health, 2008.)

TABLE 44-4 Endometrial Cancer Staging

TNM	FIGO	Definition	Adjuvant Treatment
T1	I	Tumor confined to the corpus uteri	
T1a	IA	Invades one-half or less of the myometrium	Observation for most Type 1 carcinomas Consider chemotherapy (platinum + taxane) with radiation for Type 2 carcinomas
T1b	IB	Invades more than one-half of the myometrium	Vaginal Brachytherapy or Pelvic Radiation Consider chemotherapy (platinum + taxane) with radiation for Type 2 carcinomas
T2	II	Invades cervical stroma but does not extend beyond the uterus	Pelvic radiation Platinum and taxane chemotherapy with radiation for Type 2 carcinomas
T3	III	Local and/or regional spread as specified	Radiation of involved field (pelvic +/- paraaortic) and chemotherapy (platinum + taxane)
T3a	IIIA	Involves the serosa and/or adnexa	
T3b	IIIB	Vaginal and/or parametrial involvement	
N1	IIIC	Metastasis to the pelvic and/or para-aortic lymph nodes	

	IIIC1	Positive pelvic nodes	
	IIIC2	Positive paraaortic lymph nodes with or without positive pelvic lymph nodes	
T4	IVA	Invades the bladder and/or bowel mucosa	Chemotherapy (platinum + taxane) with radiation depending on pattern of spread
M1	IVB	Distant metastasis including intraabdominal and/or inguinal lymph nodes	Chemotherapy (platinum + taxane)

2. Presentation and clinical features. Early-stage disease is generally asymptomatic. Patients experience pelvic pain or pressure, nausea, early satiety, weight loss, and bloating with increasing tumor burden (*Cancer.* 2011;117:4414-4423).

3. Standard of care. Ovarian cancer is surgically staged with a total abdominal hysterectomy, bilateral salpingo-oophorectomy, bilateral pelvic and paraaortic lymphadenectomy, peritoneal biopsies, and omental biopsy. If bulky disease is identified outside of the pelvis, the goal of the surgery is maximal cytoreduction of all gross disease. This can require bowel resection, diaphragm resection, liver resection, splenectomy, peritoneal stripping, and ablation or coagulation of tumor deposits using argon beam or plasma jet. Adjuvant treatment is listed by stage in Table 44-5. **Fallopian tube and peritoneal carcinomas** are staged and treated similarly to ovarian carcinoma. Neoadjuvant chemotherapy can be given to reduce tumor bulk, followed by interval cytoreduction and continued chemotherapy.

TABLE 44-5 Ovarian Cancer Staging

TNM	FIGO	Definition	Adjuvant Treatment
T1	I	Tumor limited to one or both ovaries	
T1a	IA	Limited to one ovary; capsule intact, no tumor on ovarian surface, no malignant cells in ascites or peritoneal washings	Carboplatin and Paclitaxel for high grade or clear cell carcinomas
T1b	IB	Limited to both ovaries; capsule intact, no tumor on ovarian surface, no malignant cells in ascites or peritoneal washings	Carboplatin and Paclitaxel for high grade or clear cell carcinomas
T1c	IC	Limited to one or both ovaries with any of the following: capsule ruptured, tumor on ovarian surface, malignant cells in ascites, or peritoneal washings	Carboplatin and Paclitaxel
T2	II	Tumor involves one or both ovaries with pelvic extension	Combination intravenous and intraperitoneal platinum and taxane chemotherapy
T2a	IIA	Extension and/or implants on uterus and/or tubes; no malignant cells in ascites or peritoneal washings	
T2b	IIB	Extension to other pelvic tissues; no malignant cells in ascites or peritoneal washings	
T2c	IIC	Pelvic extension with malignant cells in ascites or peritoneal washings	

T3	III	Tumor involves one or both ovaries with microscopically confirmed peritoneal metastasis outside the pelvis and/or regional lymph node metastasis	Combination intravenous and intraperitoneal platinum and taxane chemotherapy
T3a	IIIA	Microscopic peritoneal metastasis beyond the pelvis	
T3b	IIIB	Macroscopic peritoneal metastasis beyond the pelvis ²2 cm	
T3c	IIIC	Peritoneal metastasis beyond the pelvis >2 cm and/or regional lymph node involvement	
M1	IV	Distant metastasis (excludes peritoneal metastasis)	Carboplatin and paclitaxel

FIGO, International Federation of Gynecology and Obstetrics; TNM, tumor, node, metastasis.

Treatment of **sex cord stromal tumors** and **germ cell tumors of the ovary** are outside of the scope of this chapter.

4. What to do if suspected pre- or intraoperatively. If suspected preoperatively, obtain pelvic U/S and serum CA-125 level. If U/S is suspicious, a CT scan of the abdomen/pelvis is indicated to assess disease spread, along with a CXR or chest CT to evaluate the lungs. Consult a gynecologic oncologist if possible. Do not proceed with nonemergent surgery until this consultation is complete if suspicion for ovarian malignancy is high. If detected intraoperatively, obtain a tissue biopsy and send for frozen section. If positive for MŸllerian malignancy, obtain an intraoperative gynecologic oncology consult. If unavailable, bilateral salpingo-oophorectomy in a

postmenopausal patient with or without hysterectomy is reasonable if within the operating surgeon's scope of practice.

CHAPTER 44: OBSTETRICS AND GYNECOLOGY FOR THE GENERAL SURGEON

Multiple Choice Questions

1. Which of the following procedures is included in an ovarian cancer staging procedure?

a. Small-bowel resection

b. Splenectomy

c. Diaphragm stripping

d. Omentectomy

View Answer

2. What is the most appropriate next step in an asymptomatic 52-year-old woman with a suspected adnexal mass on clinical examination?

a. CT chest/abdomen/pelvis

b. Pelvic and transvaginal ultrasound

c. Observation

d. Exploratory laparotomy

View Answer

3. Which of the following genetic syndromes is most associated with endometrial cancer?

a. Lynch Syndrome (also call hereditary nonpolyposis colorectal cancer)

b. BRCA 1

c. BRCA 2

d. Multiple Endocrine Neoplasia 1 (MEN1)

View Answer

4. Which of the following patients needs an endometrial biopsy?

a. 35 yo with BMI 42 kg/m^2 and regular menses every 29 days

b. 35 yo with BMI 42 kg/m^2 and daily vaginal bleeding for 3 months

c. 35 yo with BMI 21 kg/m^2 and heavy periods every 29 days

d. 35 yo with BMI 21 kg/m^2 with vaginal bleeding at 23 weeks of

gestation

View Answer

5. If feasible, what is ideal positioning for a gravid patient undergoing surgery?

a. Supine with a left lateral tilt

b. Dorsal lithotomy

c. Prone

d. Supine in Trendelenburg

View Answer

6. Which of the following is the most appropriate treatment for stage IIIB cervical cancer with no distant metastatic disease?

a. Radical surgical resection

b. Simple hysterectomy followed by radiation

c. Radiation with sensitizing cisplatin

d. Chemotherapy with cisplatin, taxane, and bevacizumab

View Answer

7. Which of the following scenarios describes an incomplete abortion?

a. Vaginal bleeding, cervix closed, gestational sac appropriate for estimated gestational age identified on transvaginal U/S

b. Vaginal bleeding, cervix open, thickened endometrial strip with heterogeneous echogenicity and no viable intrauterine pregnancy on transvaginal U/S

c. Lower abdominal cramping, cervix closed, fetal pole identified measuring 8 weeks with no heart beat on transvaginal U/S

d. Vaginal bleeding, cervix closed, no identifiable intrauterine pregnancy and normal appearing endometrium on transvaginal U/S with downtrending β-hCG

View Answer

45

Biostatistics for the General Surgeon

Leisha C. Elmore

Graham A. Colditz

Evidence-based medicine aims to use information gleaned from the existing and ever-expanding body of published research to inform clinical practice. Whether contributing to the literature via the conduct and publication of original research, critically evaluating existing data to incorporate changes into day-to-day healthcare delivery, or evaluating practice patterns to improve patient outcomes at a systems level, a basic knowledge of biostatistics is important for the general surgeon.

I. STUDY DESIGN.

Study design can be stratified into two separate categories based on the aims of inquiry: Observational and experimental. In an **observational study,** participants are not assigned to treatment or control groups. Inferences are made by surveillance of a group of participants. Observational studies can determine associations between exposures and outcomes, but causation cannot be established. In contrast, in **experimental studies,** investigators assign participants to treatment and nontreatment groups and observe a specified effect. In a properly crafted prospective trial, experimental studies can establish a causal relationship between exposure and outcome (Fig. 45-1).

A. Observational Studies

1. Case-control study. In this study design, participants are selected based on the *presence or absence of the outcome* of interest. In order to determine the relationship of exposure to outcome, a group of individuals with the disease (cases) are compared to a group of individuals without the disease (controls). Evaluation of exposures in both cases and controls is conducted to determine systematic differences between the groups. A case-control study can be a quick and relatively inexpensive first step in evaluating whether an exposure is associated with a given disease. Additionally, this study design is useful for investigation of rare diseases. This approach can also be used to sample cases and controls from a much larger randomized trial or cohort study in which biomarkers or other expensive laboratory measures are being considered for additional analysis (Fig. 45-2).

2. Cohort study. In cohort studies, individuals are divided into groups based on *exposure* and followed over time to document incidence of disease or the development of the outcome of

interest. At the outset of the study, the study participants must be free of the outcome of interest or disease process. As such, cohort studies require that the study population be followed for a long period of time in order to evaluate whether the outcome has developed. Cohort studies can be retrospective or prospective (Fig. 45-3).

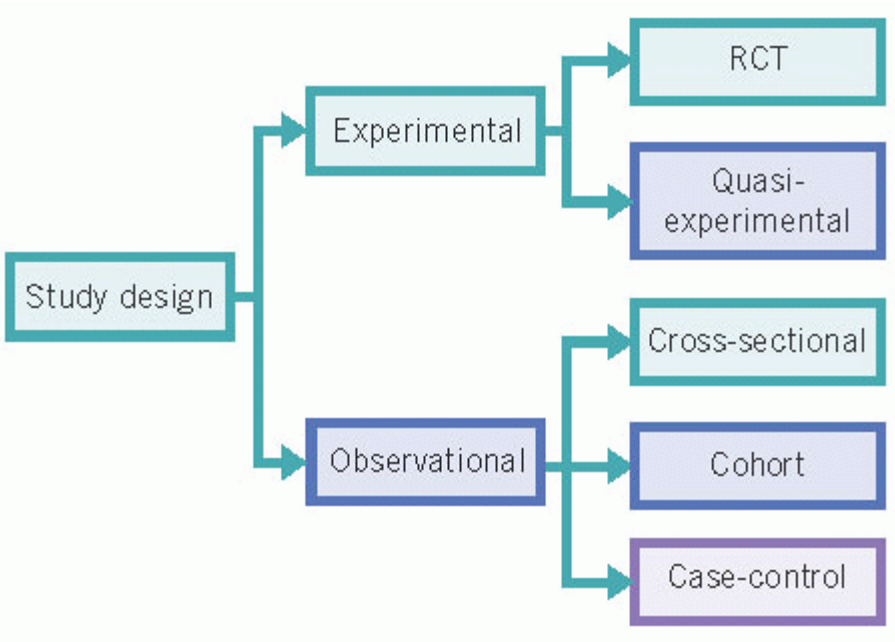

Figure 45-1 Summary of study designs.

a. In a **prospective** cohort design, the study population is identified at the outset of the trial and followed for a period of time sufficient for the outcome of interest to develop in a portion of the population. This trial design is limited by the intensive time requirement for data collection, as many of the trials require that participants be followed for many years to monitor the development of a specified outcome or disease process.

b. In a **retrospective** cohort design, historical data are used to compare exposed and nonexposed individuals. The outcome of interest is evaluated at the study's onset and historical data from a preexisting population are used to evaluate exposure status. This methodology allows for quicker completion of studies but is limited by the posthoc definition of exposure and by having to rely on the quality of the historical database.

3. Cross-sectional studies provide information on a population during a single period of observation. Exposure and outcomes are measured simultaneously in a defined study population. The benefit of this method of study is that it is easy to conduct, low-cost, and a large volume of information can be obtained quickly. This study method is useful for describing populations, identifying risk factors, quantifying the magnitude of health problems, and generating hypotheses

about exposures and disease outcomes.

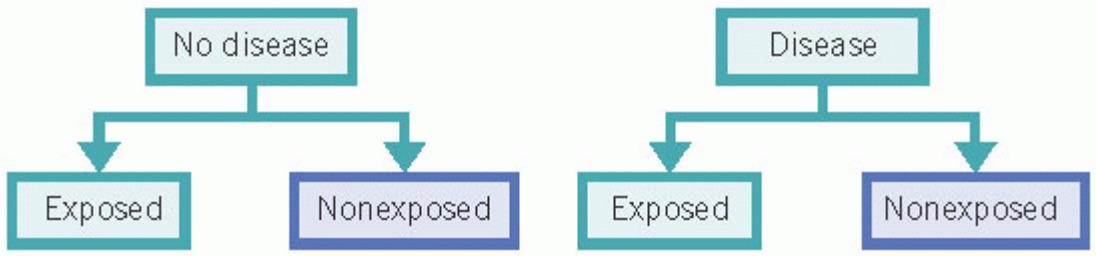

Figure 45-2 Design of a case-control study.

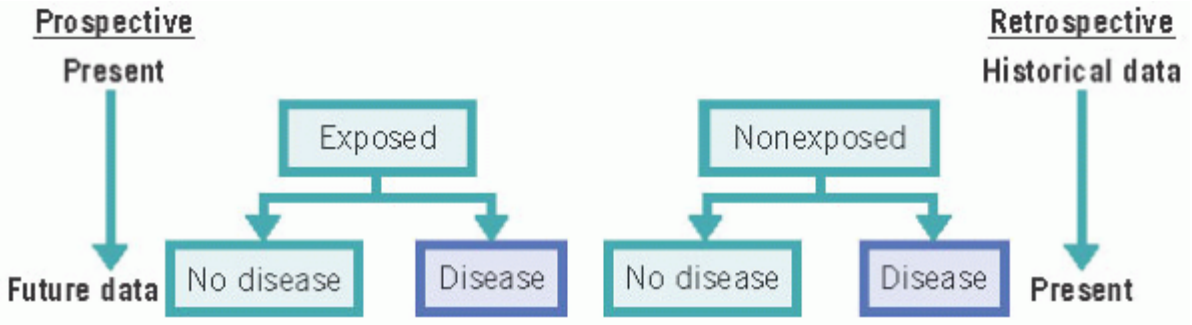

Figure 45-3 Design of a prospective and a retrospective cohort studies.

4. Case series are conducted and published on a small group of participants. These studies document the natural course of a disease and the associated treatment and outcome. These reports are typically used to document rare disease processes or outcomes and are hypothesis-generating in nature. Including all cases treated in a defined period is necessary to reduce bias.

B. Experimental Studies

1. Randomized controlled trials (RCT). In an RCT, patients are randomly assigned to *intervention* and *control* groups or arms. This trial design is the gold standard for clinical research.

a. Randomization is a key element in this study design. The random allocation of participants facilitates unbiased distribution to the study arms of both known/measured and unknown/unmeasured participant **covariates,** that is, observed, immutable characteristics that may or may not be associated with the outcome of interest and that may bias an observer's interpretation of the relationship between the intervention being studied and the outcome to be observed.

b. Blinding is the concealment of participant allocation to treatment and control groups. It can be utilized by the trial researchers with respect to both the investigators and the participants. From the patient perspective, blinding means that participants do not know if they are in the treatment or control group. Investigators can also be blinded so they are not aware of patient assignment. When neither investigators nor participants are informed of participants' allocation status, an experiment is considered **double-blinded.**

2. Quasi-experimental studies. This study design is similar to the RCT in that patients are assigned to treatment and control groups and observed for a specific outcome. The distinguishing characteristic is that patients are not randomly assigned to treatment groups. This design is used when randomization is impractical or unethical.

C. Systematic review and meta-analysis are methods to critically appraise existing published literature and develop a consensus amongst the data. The terms are often used interchangeably, but they are distinct entities. A **systematic review** is a qualitative literature review. It is conducted by establishing a clear clinical question, completing an exhaustive search of published data, identifying relevant studies, and analyzing the data.

Analysis is largely qualitative but may have quantitative components. A quantitative statistical analysis of results gleaned from a literature search of published data is referred to as a **meta-analysis;** furthermore, if individual patient data are abstracted from these results, they can be analyzed in **a pooled analysis,** which reduces heterogeneity among studies by using a more uniform classification of participant covariates.

II. DATA ANALYSIS

A. Basic Statistical Principles

1. The **null hypothesis** (H_0) is the premise that there is no difference between the control group and the treatment group. In the simplest terms, the aim of the typical study is to reject the null hypothesis and demonstrate a statistically significant difference between the control and treatment groups.

2. p-value *(p)* is defined as the probability of rejecting the null when the null hypothesis is in fact true. In the typical clinical study, authors refer to a finding as statistically significant when $p < 0.05$.

3. A **confidence interval (CI)** indicates the precision of a study measure. It is expressed as a lower limit (A) and an upper limit (B) bounded by parentheses (A,B). When the *p*-value is set at 0.05, the CI is 95%, indicating that if a study were repeated multiple times, the true sample parameter or measure would fall within the CI 95% of the time.

B. Estimating Risk

1. Prevalence is the proportion of individuals in a given population with a specific disease process or risk factor at a particular point in time.

2. Incidence is the number of *new cases* developing in a population over a discrete period of time.

3. Relative risk (RR) is the measure of association for *cohort studies.* It is calculated by evaluating the incidence of a disease in those who were exposed to a certain risk factor and comparing it to the incidence of that disease in those who were not exposed to the same risk factor.

a. Interpreting relative risk

(1) If RR = 1, the risk of developing the outcome is the same for individuals with and without the risk factor, that is, exposed and unexposed individuals.

(2) If RR > 1, the risk of developing the outcome is greater among the exposed individuals.

(3) If RR <1, the risk of developing the outcome is less in the exposed group.

4. Odds ratio (OR) is the measure of association for *case-control studies.* This ratio compares the likelihood that people with the outcome of interest might have been exposed to a particular risk factor to the likelihood that people who did NOT have the outcome of interest were exposed. The odds ratio represents a snapshot of cases and controls and does not evaluate the incidence of disease over time as seen in a cohort study.

TABLE 45-1 Sample 2 × 2 Table

	Outcome (Disease)	No Outcome (No Disease)
Exposed	A	B

Unexposed	C	D

a. Interpreting odds ratios

(1) If OR =1, there is no difference in exposure between cases and controls.

(2) If OR > 1, those with disease are more likely to have been exposed.

(3) If OR <1, those with disease are less likely to have been exposed.

5. Using contingency tables (2 × 2 tables) to calculate relative risk and odds ratios

a. A **contingency table** organizes data according to outcome and exposure status (Table 45-1).

b. Calculating relative risk

$$\text{Relative risk} = \frac{\text{Incidence of disease in exposed population}}{\text{Incidence of disease in unexposed population}}$$

$$= \frac{A/(A+B)}{C/(C+D)}$$

c. Calculating odds ratios

$$\text{Odds ratio} = \frac{\text{Odds that a case was exposed}}{\text{Odds that a control was exposed}}$$

$$= \frac{A/C}{B/D}$$

C. Evaluating Screening and Diagnostic Tests. Sensitivity and **specificity** evaluate the accuracy of a screening or diagnostic tool from the perspective of the *disease status.* The **predictive value** of a tool describes its performance from the perspective of the *test result.*

1. Sensitivity (i.e., the true positive rate) is the ability of a test to correctly identify those with disease. This is the probability of having a positive test result provided that the disease is present. A high sensitivity means that there will be a low Òfalse negativeÓ rate.

2. Specificity (i.e., the true negative rate) is the ability to correctly identify individuals without disease. In other words, this is the probability of having a negative test result if disease is not present. A high specificity means that there will be a low Òfalse positiveÓ rate.

3. Positive-predictive value (PPV) is the probability of having a disease given a test result is positive.

4. Negative-predictive value (NPV) is the probability of not having a disease given a negative test result.

TABLE 45-2 Calculating Sensitivity, Specificity, PPV, and NPV Using a 2 × 2 Table

	Disease Present	Disease Absent	
Positive test	True positive (A)	False positive (B)	PPV = A/(A + B)
Negative test	False negative (C) Sensitivity = A/(A + C)	True negative (D) Specificity = D/(B + D)	NPV = D/(C + D)

PPV, positive predictive value; NPV, negative predictive value.

5. It is worth noting that while sensitivity and specificity are not affected by the prevalence of disease, the predictive value of a test is related to the prevalence of a disease process. As the prevalence of a disease process decreases, the positive predictive value also decreases and the negative predictive value increases (Table 45-2).

D. Commonly Used Statistical Tests and Analytic Approaches

1. t-tests allow for comparison of a continuous, normally distributed variable of interest in *two* groups. For example, a t-test could be used to compare hospital lengths of stay after laparoscopic versus open colectomy.

2. Analysis of variance (ANOVA) allows for comparison of a continuous, normally distributed variable of interest in *more than two* groups. Thus, ANOVA could be used to compare hospital lengths of stay after laparoscopic versus open versus robotic colectomy.

3. The **Chi-Square test** is used to analyze categorical data and contingency tables.

4. Survival analyses allow for modeling and comparison of differences in *time-to-event* between groups. Time-to-event is the duration of time between entry into a study or diagnosis of disease and a specified endpoint, which in medicine is often death or recurrence of disease. A patient is *censored* if her time-to-event is unknown either because the patient or her information cannot be accounted for (e.g., she becomes lost to follow-up) or because the study ends before

the patient experiences the event.

a. The **KaplanÑMeier** method involves comparing the survival curves of two or more populations.

b. Cox proportional-hazards regression allows for the comparison of the effect of several covariates on survival in a given population. It assumes the relationships between the covariates and the effects of the covariates upon survival do not vary over time.

III. EVALUATING STUDY QUALITY.

Study quality is assessed based on self-reported design details. Guidelines such as CONSORT for randomized trials, STROBE for observational studies, and COREQ for qualitative research have improved the consistency of reporting.

A. Internal Validity

1. Power is defined as the probability of rejecting a null hypothesis that is false. In other words, it is the ability of a statistical test to correctly detect a difference between the experimental and control groups. Power is affected by the significance level of the test, or *p*-value (typically set at 0.05), and the sample size.

2. A **Type I error** is also known as a Òfalse positive.Ó This occurs when the null hypothesis is true but is falsely rejected.

3. A **Type II error** is known as a Òfalse negative.Ó In this scenario, the null hypothesis is false but is incorrectly accepted. In other words, a difference between the experimental and control groups is not detected even though the difference actually exists. It is important to note that as type I error decreases, type II error increases and conversely, as type II error decreases, type I error increases. In order to minimize both error types, sample size must be increased (Table 45-3).

4. Bias is the existence of systematic differences between experimental groups that affect the observed causal relationship between exposure and disease.

a. Selection bias occurs when there are systematic differences in participants' and nonparticipants' covariates, often as a result of procedures or criteria used to include or exclude participants.

b. Measurement or misclassification bias occurs when there is an error in how the exposure status of participants or the recording of outcomes is coded. Measurement error may be introduced through variation in laboratory assays or recall of exposure in epidemiologic studies.

c. Recall bias is an error that results from the incomplete or inaccurate recollection of past events by study participants in a way that is systematically different between participants in different arms of a study.

d. Interviewer bias occurs when interviewers are not blinded to exposure or disease status and frame questions and record responses differently based on participant status.

e. The **Hawthorne effect** occurs when individuals modify their behavior because they are aware they are under observation.

TABLE 45-3 Assessing Internal Validity: Power and Error

		Null Hypothesis	
		True	**False**
Decision	*Accept*	Correct decision 1-α	Type II error (β)
	Reject	Type I error (α)	Correct decision Power = 1-β

f. Attrition bias results when participants are lost to follow-up. These individuals may be systematically different from those that continue to participate in a trial.

g. Lead-time bias occurs when a disease is detected earlier due to the implementation of screening protocols in one group. This early detection falsely lengthens survival or time-to-event.

5. Confounding may occur when a factor (i.e., covariate) is associated with both the exposure and outcome. This covariate, also known as a **confounder,** interferes with or drives the relationship between the exposure and outcome. This factor or covariate must be addressed with randomization or statistical analysis to prevent erroneous or inaccurate conclusions.

B. External validity refers to the ability to generalize the results of a study to the greater population. In order to improve the external validity or generalizability of the data, the experimental population must be similar to the reference population from which the study population was extracted. More extensive reporting of patient selection processes, study participants' demographic information, and providers' professional and personal characteristics assists with the extrapolation of study conclusions to other clinical settings and patient populations.

IV. CLINICAL PRACTICE GUIDELINES.

With the wealth of published data, evaluating the integrity of research findings and determining best practices to implement can be challenging for the clinician. Researchers have developed a hierarchical grading system to assess the quality of data and clinical recommendations. While there are many published grading systems, the United States Preventive Services Task Force (USPSTF) has developed one of the most widely accepted systems (see Tables 45-4 and 45-5).

TABLE 45-4 USPSTF Quality of Data Classification

Level of Evidence	Source of Evidence
I	Evidence from systematic review of randomized controlled trials
IIa	Evidence from controlled trials without randomization
IIb	Evidence from cohort or case-control studies
IIc	Evidence from multiple time series or historic controls
III	Expert opinion based on clinical experience

USPTF, United States Preventive Services Task Force.

TABLE 45-5 USPSTF Clinical Practice Recommendations

Grade of Evidence	Recommendations for Practice
A	High certainty that the net benefit is substantial. Offer or provide this service.

B	High certainty that the net benefit is moderate to substantial. Offer or provide this service.
C	Moderate certainty that the net benefit is small. Provide this service based on individual patient preference and professional judgment.
D	Moderate to high certainty that this service has no benefit or the harms outweigh the benefit. Discourage use of this service.
I	Insufficient evidence to assess benefits and harms of service.

USPTF, United States Preventive Services Task Force. These systems for synthesis of evidence have now been more formally accepted by many professional societies. The Institute of Medicine has published a set of guiding principles addressing the methods for developing these types of practice guidelines in order to bring a more consistent approach to their development across the overlapping sectors in healthcare delivery.

Additional References

Detterbeck F, Gettinger S, Socinski M. Lung neoplasms. In: Norton J, Barie P, Bollinger R, et al., eds. *Surgery: Basic Science and Clinical Evidence.* 2nd ed. New York, NY: Springer; 2008:1491-1523.

Gordis L. *Epidemiology.* Philadelphia, PA: Saunders Elsevier; 2009.

Web Sites

The equator site maintains the reporting guidelines accepted across many biomedical journals for clinical studies: **http://www.equator-network.org/toolkits/authors/**

Chapter 45: Biostatistics for the General Surgeon

Multiple Choice Questions

1. A study was conducted to determine the impact of sodium-

bicarbonate preprocedural hydration on the incidence of contrast-induced nephropathy. Which of the following analytic techniques would be best suited to analyze this data?

a. ANO VA

b. Cox proportional-hazards analysis

c. Chi-square test

d. t-test

View Answer

2. A colorectal surgeon wants to investigate the role of Alvimopan (Entereg), a peripherally acting μ-opioid antagonist, on the number of days until return of bowel function after open colectomy. Which of the following statistical methods would be used to evaluate the impact of this drug?

a. Wilcoxon rank sum test

b. t-test

c. ANO VA

d. Chi-square test

View Answer

3. As the prevalence of a disease increases, which of the following is true?

a. Positive predictive decreases

b. Positive predictive value does not change

c. Sensitivity increases

d. Sensitivity does not change

View Answer

4. Pseudomyxoma peritonei is a form of cancer that produces mucinous ascites and is most commonly secondary to a primary tumor of the appendix. Researchers are interested in conducting a study to determine factors associated with the development of pseudomyxoma peritonei in patients with appendiceal cancer. Which of the following study designs is most appropriate to investigate this clinical question?

a. Case-Control Study

b. Cohort Study

c. Randomized Controlled Trial

d. Cross-Sectional Study

View Answer

The following scenario applies to questions 5 to 7:

Researchers are developing a new diagnostic test to identify patients with lung cancer. A total of 150 patients with lung cancer are tested and 300 patients without lung cancer are included for study. A total of 125 of the patients with lung cancer and 20 of the patients without lung cancer receive positive tests results.

5. What is the sensitivity and specificity of the diagnostic test, respectively?

a. 93% and **83%**

b. 86% and **83%**

c. 83% and **93%**

d. 92% and **86%**

View Answer

6. What are the positive predictive value and the appropriate interpretation of the result?

a. Given a positive test results, the likelihood of having lung cancer is 86%.

b. Given a positive test results, the likelihood of having lung cancer is 83%.

c. In patients with lung cancer, the probability of a positive test is 86%.

d. In patients with lung cancer, the probability of a positive test is 83%.

View Answer

7. What is the false positive rate?

a. 17%

b. 13%

c. 7%

d. 8%

View Answer

46

Patient Safety and Quality Improvement in Surgery

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I. PATIENT SAFETY

A. Introduction: What is Patient Safety?

1. Institute of Medicine (IOM): To Err is Human (National Academies Press (US); 2000. Bookshelf ID: NBK225179)

a. In 1999, the IOM reviewed medical errors in the United States and estimated that up to 98,000 patients die in hospitals each year due to **preventable medical errors.**

b. The majority of errors do not result from individual recklessness, but rather from issues within hospital systems and processes that create an environment prone or vulnerable to error. For example, the storage of adult and pediatric doses of heparin next to each other in similar containers could lead to improper dosing.

c. Medical errors are defined as failure of planned action to be completed as intended or the use of a wrong plan to achieve an aim.

d. The Swiss Cheese Model of Accident Causation (Fig. 46-1):

(1) Psychologist James Reason suggested the existence of **latent errors,** defects in the layers of organization of systems that could lead to active errors that then can cause harm. (Cambridge University Press; 1990. ISBN: 05214194)

(2) In this model, processes are represented by slices of cheese; each slice represents a different process stage or aspect, and holes represent weaknesses in each layer/process step that could lead to failures. Weaknesses are either latent errors present in organizational influences (e.g., inadequate institutional structure to promote safety training), improper levels of process supervision, conditions predisposing to errors, or active errors consisting of unsafe acts. Systems fail to prevent hazards from propagating to actual losses/errors when the ÒholesÓ (potential for error) in all layers (process steps) align. *(Nat Rev Urol.* 2013;10(3):161-173.)

e. Examples: Wrong-site surgery, falls, adverse drug events, improper transfusions

f. Types of error

(1) Diagnostic

(2) Treatment

(3) Preventative (e.g., inadequate follow-up or failure to provide prophylactic treatment)

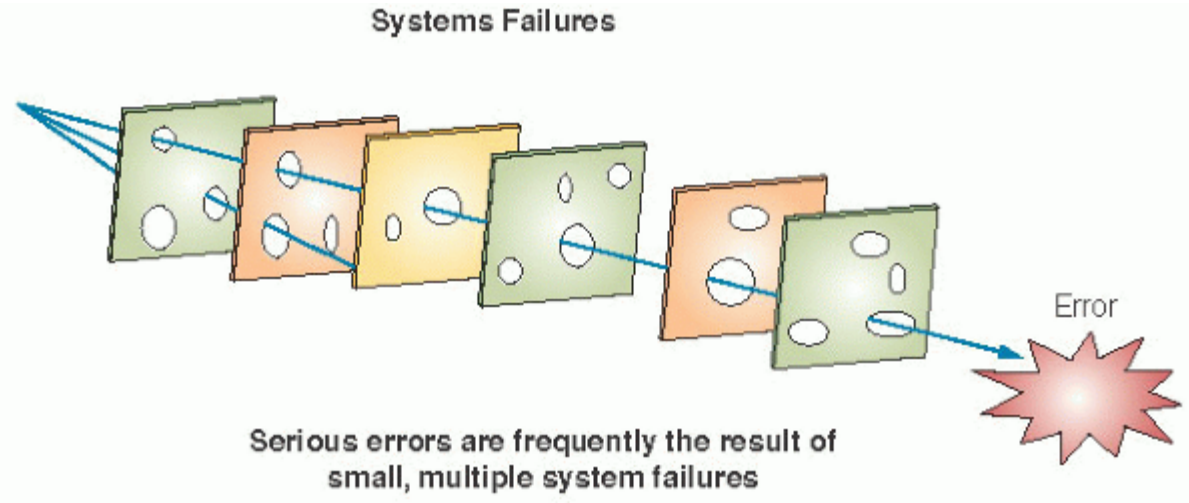

Figure 46-1 Swiss Cheese Model of Accident Causation. (From Mulholland MW, Lillemoe KD, Doherty GM, et al., eds. *Greenfield's surgery: scientific principles and practice.* 5th ed. Philadelphia, PA: Wolters Kluwer Health, 2010.)

(4) Commission and OmissionÑ**Errors of commission** result from Òdoing the wrong thingÓ while **errors of omission** result from Ònot doing the right thing.Ó Errors of omission are more difficult to detect and are likely more common (e.g., failure to provide/perform diagnostic, therapeutic, or preventative measures that could improve care)

(5) Other (e.g., communication or equipment failure)

2. Normalization of deviance in healthcare

a. Medical personnel repetitively and often progressively violate standard care practices until the deviant practice becomes the new norm, creating conditions that can cause harm to patients *(Bus Horiz.* 2010;53(2):139). An example of this phenomenon is clinicians' not washing or sanitizing their hands properly and routinely before seeing patients.

B. Detecting and Reporting Medical Errors

1. Errors represent opportunities to improve processes in medical practice, but they must be recognized and addressed in a systematic fashion in order for organizations to appropriately and

consistently benefit from error reporting.

2. ÒNear missesÓ are events in which an error occurs but does not result in an adverse event. These are also important to identify as opportunities for improvement in patient safety.

3. Many medical institutions use **voluntary, often anonymous, reporting systems** (e.g., paper- or computer-based error reports, hotlines) open to anyone working at the institution. The reports are then analyzed by hospital risk management personnel or a patient safety and/or quality improvement department (Institute for Healthcare Improvement, http://www.ihi.org, 2015).

a. Only 10% to 20% of medical errors are caught using such a system due to lack of error recognition, fear of punishment, difficulty in

using reporting systems, and perceptions that error-reporting is punitive.

b. In addition to hospital-based systems, reporting systems for medical errors can be in place at the regional, state, or national levels.

4. Human factors in medical errors (Institute for Healthcare Improvement, http://www.ihi.org, 2015)

a. Human factors engineering is a field of science that takes into consideration human characteristics within processes. A multidisciplinary approach (physiology, anatomy, ergonomics, and psychology) is used to understand how humans perform under varying conditions.

b. Human factors that can potentially increase the risk of a medical error include

(1) Pre-existing **psychological and/or physiological states** including stress, fatigue, and boredom

(2) Decision-making factors including memory, reasoning, and judgment

(3) Action factors including communication skills, influence, and technique to perform a specific action

c. Example: Pattern Matching during ReadingÑHumans visually read the beginning and ending letters of each word. When looking at ÒDo___mine,Ó some individuals automatically think of ÒDopamine,Ó others think of ÒDobutamineÓ; as a result, these two medications could be inadvertently substituted.

5. Root cause analysis (RCA)

a. A method to **retrospectively review medical errors** to find latent errors within a system with the overall **goal of preventing these errors from recurring**

b. Often initiated after an adverse event or a Ònear missÓ

c. A misnomer since multiple Òroot causesÓ are often found with each analysis

d. RCA is conducted in a **team-based fashion** with goals to identify strategies to improve processes and not to blame particular individuals

(1) The team generally consists of 4 to 6 members from all aspects of care within a health system and members from a Risk Management or Safety/Quality Office. Although practices vary between institutions, generally personnel directly involved in the evaluated incident are excluded from the RCA team in order to minimize bias.

e. RCA steps:

(1) Define the problem: Gather all information relevant to the incident using chart review, debriefing of involved individuals, and development of a detailed timeline

(2) Develop a cause-and-effect chart: Often referred to as a **Fishbone or Ishikawa Diagram** (see Fig. 46-3), this will help organize possible cause-and-effect relationships that might have contributed to the investigated error. Ask Òwhy?Ó five times for each possible cause identified to help identify a root cause.

(3) Identify possible solutions: As a group, find possible systemic fixes to prevent the error from occurring again, categorize the fixes as high or low impact, and assess the resource requirements for each fix. In this manner, tasks with the highest impact but lowest resource utilization can be the focus for the short term, whereas resource-intensive tasks can be considered after more discussion and research.

(4) Implementation and review: Initiate any solutions found to be appropriate from the previous step and intermittently review the effectiveness of new processes.

C. Communication and Handoffs

1. Communicating adverse events

a. Adverse event: An injury or suboptimal outcome caused by medical management rather than the patient's underlying disease. *(Lippincotts Case Manag.* 2006;11(4):193-194.)

b. Healthcare workers might avoid reporting adverse events due to fear of censure, colleague disapproval, negative publicity, or anger from patients and/or employers; threat of medical malpractice; and the effort required to report.

c. Important steps that should be taken following an adverse event:

(1) First and foremost **address medical needs** of the patient.

(2) Communicate what is known about the adverse event to the patient or patient representative with a statement of empathy.

(3) Notify departments, entities, specific people about the adverse event per facility policies in your institution.

(4) Document adverse event and communication following the adverse event with the patient and other entities in the medical record.

d. An apology to the patient by the physician following a medical error can decrease blame, anger, and the risk of litigation and may also increase trust in the doctorÑpatient relationship. Despite this evidence, some institutions view apologies to patients as risky and an admission of guilt that could subsequently increase the risk of litigation; therefore, it is important to understand your institution's specific guidance on apologizing to patients. *(Clin Orthop Relat Res.* 2009;467(2):376-382.)

2. Patient health education

a. The American Hospital Association adopted the **Patients' Bill of Rights** in 1973, stating that patients have the right to relevant, current, and understandable information of their diagnosis, treatment, and prognosis.

b. Evidence shows that following **informed consent,** a significant number of surgical patients do not adequately understand all aspects of the planned surgical procedure. *(Am J Surg.* 2009;198(3):420-435.)

c. Effective communication of health information to the patient is essential to respect patient autonomy, manage patient expectations following a surgical procedure, and improve patient satisfaction.

d. The informed consent process can be personalized and made more patient-centered through use of **the Request for Treatment Form;** following the consent process, the patient completes the form in his or her own words at home, and then returns the form to the physician so that the patient's understanding of the planned treatment can be confirmed and any questions or misconceptions can be answered or clarified. *(Ann R Coll Surg Engl.* 2010;92(2):93-100.)

3. Handoffs in surgical residency

a. Resident duty-hour restrictions established in 2010 have resulted in a greater number of **handoffs** between clinical providers. *(N Engl J Med.* 2010;363(2):e3.)

b. Handoffs, if conducted improperly, can lead to an increase in adverse events, worse patient outcomes, longer hospital stays, and increased admissions to the intensive care unit. *(Am J Med.* 2012;125(1):104-110.)

c. Communication breakdown during handoffs is one of the most common factors recognized as causing errors that lead to adverse events. *(Surgery.* 2003;133(6):614-621.)

d. Due to the unique needs of different care teams and surgical subspecialties, a universal standardized handoff tool might not be feasible.

e. Handoffs can occur between two individuals but are often most efficient if carried out as a

group with the supervision of senior residents, fellows, or faculty.

f. Mnemonics and tools have been created to help standardize handoffs and thereby prevent the omission of the most important information:

(1) ÒSHOUTÓÑSick or not, History, Objective data, Upcoming plan/disposition, To do. *(Practical QI: The Basics of Quality Improvement.* American College of Surgeons; 2014.)

(2) ÒSBARÓÑSituation, Background, Assessment, Recommendation. *(Jt Comm J Qual Patient Saf.* 2006;32(3):167-175.)

(3) ÒI-PASSÓÑIllness severity, Patient summary, Action list, Situational awareness/contingency plan. *(Pediatrics.* 2012;129(2):201-204.)

D. Culture of Safety

1. Healthcare organizations establish processes that focus on the continuing improvement of reliability and safety in caring for patients.

a. The following aspects must be addressed by organizations to attain a **culture of safety,** which requires **psychological safety** in which employees understand that their concerns will be addressed, **active leadership** that encourages open expression of concerns from healthcare workers, **transparency** in addressing patient safety issues, and **fairness** in dealing with medical errors in which people are not blamed for systems-based errors. (Institute for Healthcare Improvement, http://www.ihi.org, 2015)

b. Discussing patient safety can be difficult in healthcare settings due to the hierarchical nature of medicine and the relative ease of staying quiet rather than voicing concerns about patient safety issues.

c. Surveys of U.S. hospitals show that a strong culture of safety is promoted by organizations that value teamwork, whereas weak safety culture is associated with organizations focused on hierarchy. *(Health Care Manage Rev.* 2009;34(4):300-311.) Organizations with a strong culture of safety reported better performance on patient safety indicators. *(Health Care Manage Rev.* 2009;34(4):300-311.)

2. Physician peer support (second victim)

a. Following an adverse event, patients and their families receive support from most medical teams, but physicians involved in the event are rarely treated in the same manner. Physicians involved in adverse events report anxiety about future errors, loss of confidence, sleep difficulties, reduced job satisfaction, and increase in job-related stress. *(Jt Comm J Qual Patient Saf.* 2007;33(8):467-476.)

b. A physician involved with and affected by an adverse event, is the **Òsecond victim.Ó** With appropriate institutional support, he or she will pass through the following phases en route to

recovery: Accident response, managing intrusive reflections, restoring personal integrity, enduring the review of the case by the institution, getting proper emotional support from peers, and moving on from the adverse event. *(Qual Saf Health Care.* 2009;18(5):325-330.)

3. Professionalism

a. American College of Surgeons (ACS) Code of Professional Conduct notes that professionalism Òserves as the basis of the social contract between medicine and the society that it serves.Ó (American College of Surgeons, https://www.facs.org/about-acs/statements/stonprin, 2014)

b. The ACS Code of Professional Conduct defines responsibilities related to patient safety including

(1) Full disclosure adverse events and medical errors

(2) Participation in self-regulation by setting, maintaining, and enforcing practice standards

(3) Improvement of care by evaluating its processes and outcomes

II. QUALITY IMPROVEMENT

A. Introduction

1. Quality improvement (QI) consists of systematic and continuous improvements in healthcare settings that can be measured, especially in reference to the health status of targeted patient groups (Health Resource and Services Administration, http://www.hrsa.gov/quality/toolbox/methodology/qualityimprovement/, 2015)

2. In QI, the focus is typically on improvement of systems instead of individual performance

3. IOM: Crossing the Quality Chasm (National Academies Press (US); 2001. Bookshelf ID: NBK222271)

a. Following IOM's 1999 report, titled *To Err is Human,* on errors in the medical system, *Crossing the Quality Chasm* was published in 2001 as a shared vision of six aims to help healthcare be more:

(1) SafeÑavoid injuries

(2) EffectiveÑprovide care based on scientific evidence

(3) Patient-centeredÑprovide care respectful to patient values

(4) TimelyÑreduce wait-time and harmful delays

(5) EfficientÑavoid waste of equipment, ideas, and energy

(6) EquitableÑprovide care that does not vary in quality because of personal characteristics,

geographic location, or socioeconomic status

b. To help achieve the six aims, ten rules were created to help redesign better quality healthcare systems:

(1) Care should be **provided when patients need and want it** utilizing many forms including in-person visits, telephone calls, and Internet communication.

(2) Care should be **customized** to patient needs and values.

(3) Patients should have the amount of **control** they want over healthcare decisions.

(4) Patients should have **full access to their medical information.**

(5) Clinical decision-making should be **evidence-based.**

(6) Safety should be addressed as a system property.

(7) Healthcare systems should be made **transparent** including public reporting on safety, evidence-based practice, and patient satisfaction.

(8) Systems should **anticipate patient needs** instead of react to events.

(9) Patient time and resources **should not be wasted.**

(10) Cooperation among clinicians should be a priority to ensure coordination of care.

B. Systems and Methods of Improvement

1. The Model for Improvement (see Fig. 46-2)

a. Combines addressing three questions with a **Plan-Do-Study-Act (PDSA) Cycle** plan to address quality improvement

b. Questions to guide improvement:

(1) What are we trying to accomplish?

(2) How will we know a change is an improvement?

(3) What change can we make that will result in improvement?

c. PDSA Cycle

(1) Four-phase cycle to develop, test, and implement changes in healthcare

(2) Works best when used to quickly create and test changes on a small scale before broad implementation

2. Quality improvement basic tools

a. Cause-and-effect ÒFishboneÓ diagram (Fig. 46-3)

(1) Helps visually identify many possible causesÑgrouped into common categoriesÑthat might be

contributing to a particular problem

b. Checklists

(1) Checklists such as the **World Health Organization Surgical Safety Checklis**t (see Fig. 46-4) can help standardize medical

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processes, improve medical team communication, and reduce complications. *(N Engl J Med.* 2009;360(5):491-499.)

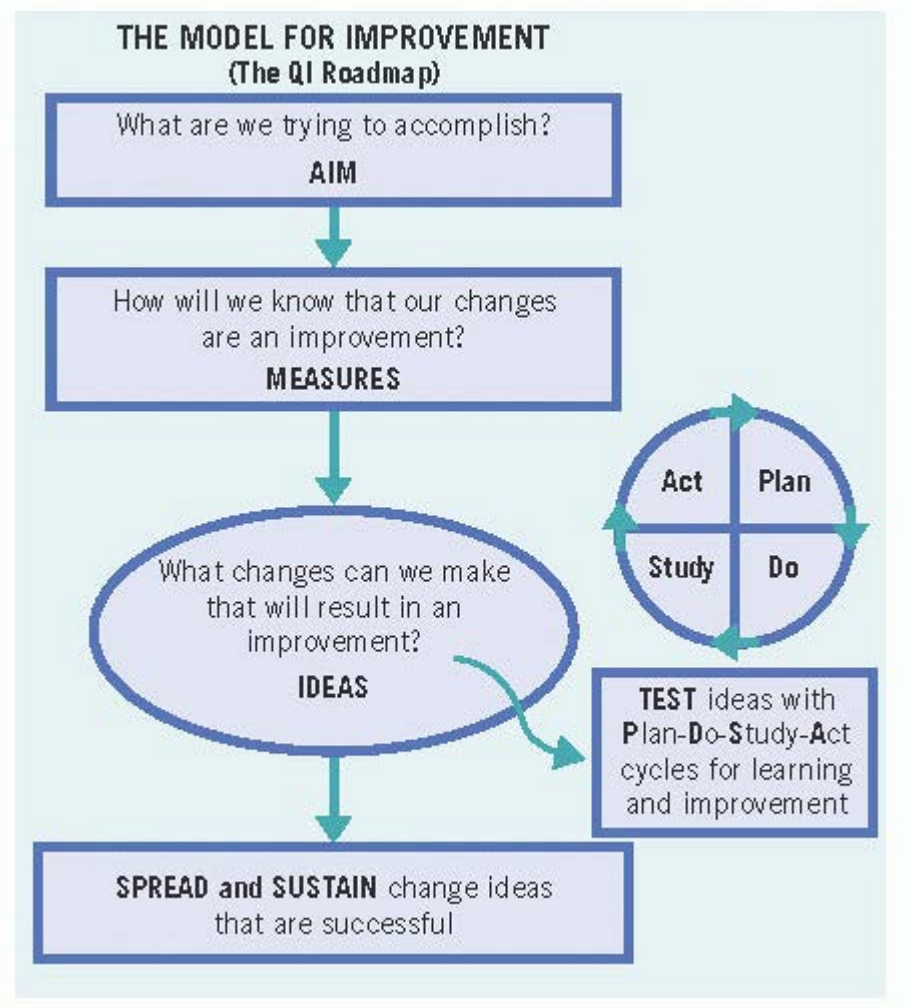

Figure 46-2 Model for Improvement. (From *J Public Health Manag Pract.* 2012;18(1):19-26, with permission.)

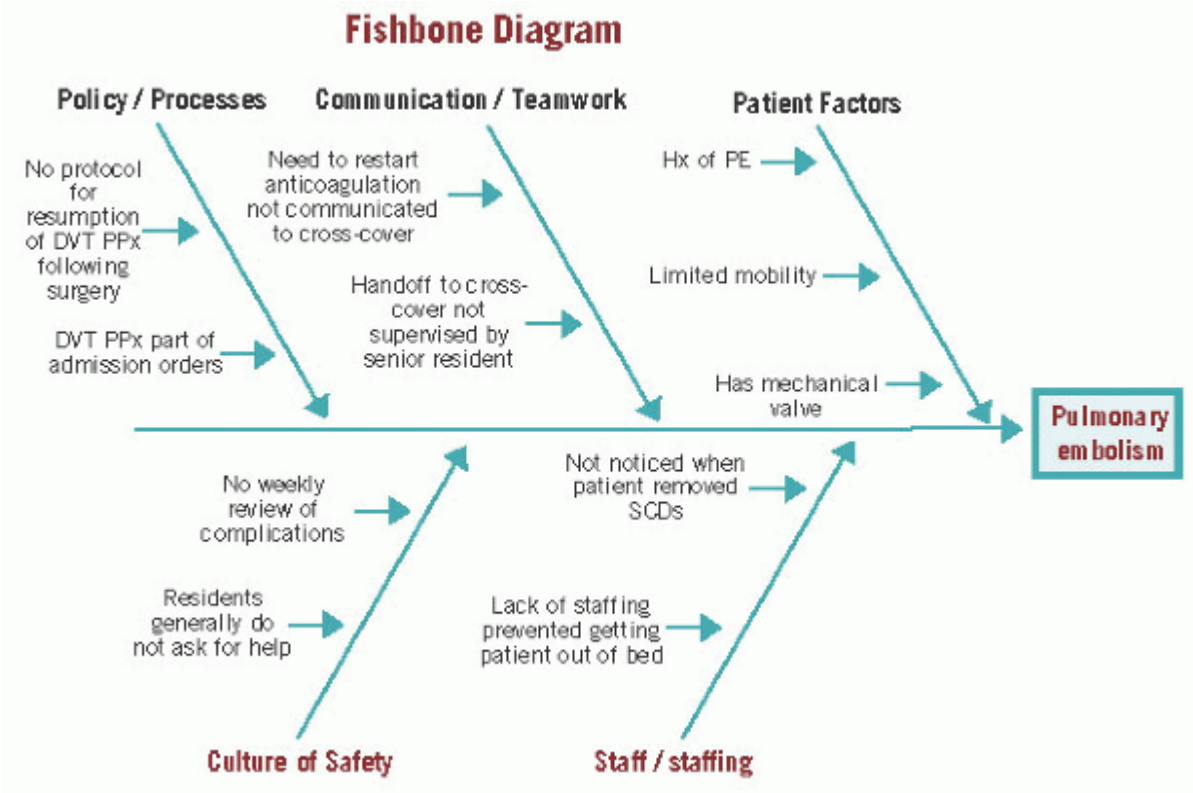

Figure 46-3 Example of possible Fishbone Diagram identifying factors related to a problem outcome (pulmonary embolism). DVT, deep vein thrombosis; PE, pulmonary embolism; PPx, prophylaxis; SCDs, sequential compression devices.

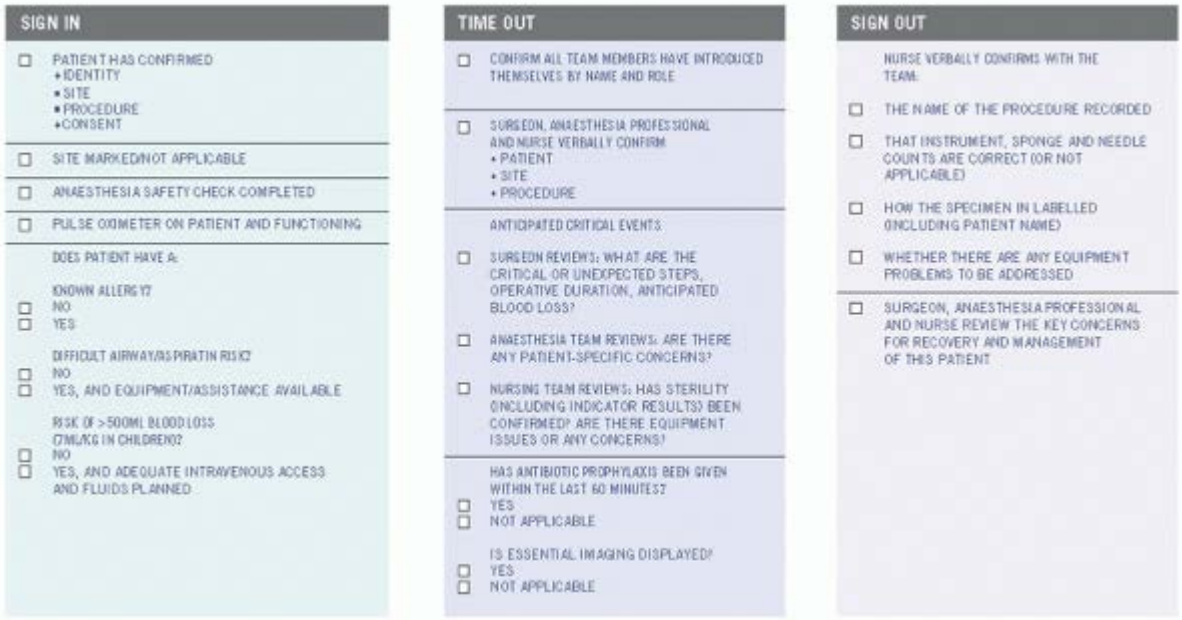

Figure 46-4 World Health Organization Surgical Safety Checklist. (World Alliance for Patient

Safety, World Health Organization; 2008.)

c. Pareto charts

(1) A combination of a bar graph and a line graph in which the bars represent frequency of an event or cost with largest bars (most significant factors) on the left side of the graph and the line represents cumulative number of events (Tague NR. *The Quality Toolbox.* 2nd ed. ASQ Quality Press; 2010)

(2) Helpful to display the most significant factors or causes of a problem when there are many

d. Run charts

(1) A graph that shows a measured value plotted sequentially in time (Institute for Healthcare Improvement, http://www.ihi.org, 2015)

(2) Useful for displaying the measured effect of a quality intervention

(3) A median should be calculated from measured values before the onset of the intervention and plotted as a horizontal ÒbaselineÓ from which changes can be measured to gauge success of the intervention

3. Morbidity and mortality conference

a. One of the first forums to review medical errors, it is a format widely used in current surgical training and practice.

b. The following practices can help improve the effectiveness of the conference *(Practical QI: The Basics of Quality Improvement.* American College of Surgeons; 2014):

(1) Should be held regularly, **at least once a month**

(2) Must have **high level of attendance/engagement** from faculty and residents

(3) A list of potential **cases for discussion are selected prior** to the conference

(4) Method of case reporting should be as complete and accurate as possible. *Hospital datasets* that track outcomes often provide more complete case lists than resident-reported case lists

4. Six Sigma and Lean

a. Two frameworks for quality improvement focused on specific concepts to improve quality

b. Six Sigma, born out of Motorola in 1986, focuses on decreasing process errors to six standard deviations (i.e., sigma or σ) below the mean. However, most medical processes have error rates that average 3 to 4 sigma around the mean. Six Sigma consists of five steps *(Milbank Q.* 1998;76(4):565-591, 510):

(1) DefineÑthe problem in detail

(2) MeasureÑerrors (per million in the original system)

(3) AnalyzeÑprocess measures to determine the conditions under which errors occur

(4) ImproveÑdesign changes aimed at decreasing errors and measure outcomes

(5) ControlÑtake steps to sustain improvements

c. Lean was created by Toyota in the 1970s as a method of quality improvement that centered around maximizing value from the customer's perspective and minimizing waste.

(1) Types of waste: Overproduction, waiting, excess transportation, processing, excess inventory, unnecessary movement, and defective products

(2) Value stream mapping: Processes are mapped as a series of steps for which efficiency is defined as time during each step in which value is added for the customer (or patient) compared to time spent without any value added

(3) 5 SÑsystem of steps essential to the Lean process. Below are the English counter-parts to Japanese words for each step:

(a) SortÑkeep only essential tools in the workplace

(b) StraightenÑarrange workplace for ease of use

(c) SweepÑmaintain a clean work environment

(d) StandardizeÑestablish best practices for each process

(e) SustainÑcommit former steps to habit

5. National Surgical Quality Improvement Program (NSQIP)

a. Developed by the American College of Surgeons, NSQIP uses trained surgical clinical reviewers to collect data from patient charts on preoperative risk factors, intraoperative variables, and 30-day postoperative morbidity and mortality. (American College of Surgeons, http://site.acsnsqip.org/, 2015)

b. NSQIP evaluations are **risk-adjusted** to account for patient characteristics and **case-mix-adjusted** to account for complexity of surgeries completed at a single center to allow for comparison of outcomes across different medical centers.

c. Using reports of comparative performance, opportunities to improve outcomes at participating institutions are identified and can be targeted with best practice guidelines and directed efforts to improve quality of surgical care.

C. High-Reliability Organizations (HRO)

1. An organization that succeeds in avoiding errors where they are expected due to risk factors and the complexity of the system. They exhibit five characteristics that help avoid errors and deal with unexpected situations *(Qual Saf Health Care.* 2004;13 Suppl 2:ii39-44):

a. Preoccupation with failure

b. Reluctance to simplify interpretations

c. Sensitivity to operations

d. Commitment to resilience

e. Deference to expertise

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Chapter 46: Patient Safety and Quality Improvement in Surgery

Multiple Choice Questions

1. Which of the following tools is best described as a graph that shows a measured value plotted sequentially in time that is useful for displaying the measured effect of a quality intervention?

a. Pareto chart

b. Run chart

c. Scatter plot

d. Histogram

e. Fishbone diagram

View Answer

2. Value-stream mapping, a method to calculate the efficiency or value added to the customer/patient for each step in a system process, is a component of which of the following systems of quality improvement?

a. Six Sigma

b. National Surgery Quality Improvement Program

c. Lean

d. Model for Improvement

e. High-reliability organizations

View Answer

3. While driving to work, a physician notes that the majority of drivers around her are consistently driving above the speed limit. The concept in patient safety that describes this behavior of routinely violating standard practices is:

a. Human factors engineering

b. The “second victim” phenomenon

c. Errors of omission

d. Normalization of deviance

e. Preventive error

View Answer

4. In the Model for Improvement, three questions about a quality issue must be addressed (What are we trying to accomplish? How will we know a change is an improvement? What change can we make that will result in improvement?) and which of the following must also be utilized?

a. Plan-Do-Study-Act Cycle

b. Fishbone Diagram

c. Surgical Safety Checklist

d. Morbidity and Mortality Conference

e. Swiss Cheese Model of Accident Causation

View Answer

5. Which of the following represents a team-based approach to retrospective review after a medical error or “near miss” event to identify latent errors within a system?

a. Swiss Cheese Model of Accident Causation

b. Human Factors Engineering

c. Lean

d. Six Sigma

e. Root Cause Analysis

View Answer

Answer Key

CHAPTER 1

1. Answer: c. Per Table 1-1, patients with a history of TIA are at elevated risk for MACE. Patients with diabetes controlled with oral agents, mild renal insufficiency, and controlled hypertension are not in this elevated risk group.

2. Answer: b. Please see Table 1-2 for details regarding assessment of functional status.

3. Answer: d. Please see Table 1-3. To reduce SSI risk, hair removal should be performed with clippers. Glucose control goal should be <180 mg/dL. Core body temperature should be kept above 36 degrees.

4. Answer: c. Please see Table 1-5. Protein C deficiency is considered a high-risk hypercoagulable state.

5. Answer: b. Please see Table 1-5. All mitral prostheses are considered high risk for thromboembolism. Bileaflet aortic valves are at lower risk.

6. Answer: c. Please see Figure 1-1. The patient in scenario C has several risk factors and has claudication that limits the ability to perform exercise stress testing. The patient in A requires emergent surgery. The patients in B and D, despite risk factors, have no functional impairment requiring testing.

7. Answer: c. Please see Section I.B.7.a. Coumadin should be stopped 5 days prior to elective surgery. Coumadin causes the production of inactive clotting factors. Once the drug is withheld it can take up to 5 days for the liver to generate sufficient functional clotting factors.

8. Answer: b. The patient in B is anuric with fluid overload that appears to be leading to a requirement of mechanical ventilatory support. This is an indication for hemodialysis to remove fluid and improve pulmonary function. Mild-to-moderate metabolic derangements such as in A and C can be treated with measures less invasive than hemodialysis. Creatinine level (D) is not an indication for hemodialysis per se.

9. Answer: b. Please see Section I.B.5.b.5. Foley catheters should be removed on postoperative day 1 unless there is concern for urinary retention, urinary tract injury, or renal insufficiency/oliguria.

CHAPTER 2

1. Answer: a. In the oliguric patient, the fractional excretion of sodium helps to distinguish between prerenal and intrinsic renal causes of renal failure. A FENa of <1% is consistent with a

prerenal cause of renal failure.

2. Answer: b. Acute delirium is best treated with sleep hygiene and minimizing mood-altering medications. For patients with acute, hyperactive delirium, haloperidol can be used to prevent self-injurious behavior. Quetiapine has been demonstrated to improve delirium and can help restore day/night sleep wake cycling. Benzodiazepines can exacerbate delirium and should be strictly avoided.

3. Answer: g. Oliguria and tachycardia in the early postoperative period is a red flag for possible bleeding. This patient is in stage II shock with oliguria and mild tachycardia. The initial workup and treatment are to restore intravascular volume with a bolus of crystalloid and check a hematocrit to assess for bleeding. This patient may need transfer to the intensive care unit, but the first step is to give the fluid bolus and assess for bleeding. Prior to returning to the operating room, the patient should receive resuscitation and the cause of his oliguria need to be established. Although he could be bleeding, he may also be under-resuscitated.

4. Answer: d. The Surgical Care Improvement Project recommends a maximum of 24 hours of perioperative antibiotic coverage for routine general surgery procedures.

5. Answer: b. Antibiotic impregnated central venous catheters are not part of the Surgical Care Improvement Project guidelines for the prevention of perioperative infections. The CDC does recommend considering antibiotic impregnated catheters as an alternative method of decreasing hospital-acquired infections.

6. Answer: d. The first step in evaluating this agitated patient is to check her vital signs and pulse oximetry. It is more likely a deficit in executive function than a focal neural deficit, therefore a to c should be considered after the vital signs, but at this time e is not indicated.

7. Answer: b. In addition to sleep hygiene, constant reorientation, and exterior views, for recalcitrant delirium, Quetiapine 25 to 50 mg can be effective adjunctive therapy. Haldol 1 to 5 mg can be used for hyperactive delirium.

8. Answer: c. This patient is most likely suffering from alcohol withdrawal, given his time point of 3 days (72 hours) since his last drink, his hallucinations, and autonomic instability (diaphoresis and tachycardia) he most likely has delirium tremens. Alcoholic seizures usually occur before 72 hours, benzodiazepines are used to treat alcohol withdrawal and an overdose would not produce these symptoms, delirium's symptoms are not listed.

9. Answer: e. While a cause for hypovolemia, sepsis typically is not on the differential until after postoperative day 2, unless the patient has recently been operated on, or was septic prior to the operation.

10. Answer: c. Classically most postoperative MI's occur on postoperative day 2. Any new or

worsening chest pain deserves a full myocardial infarction workup.

11. Answer: a. Since his oxygen saturation is low, he should be placed on supplemental oxygen and monitored for the need for intubation. With his surgery 2 days prior he may now be mobilizing his third space fluids and furosemide will aid in diuresis of excess fluid

12. Answer: b. The patient who recently started eating after an operation is elderly and had two bouts of emesis putting him at high risk for a gastric aspiration. Frequently an immediate postaspiration chest x-ray will not show significant changes. Eventually, it will show diffuse interstitial infiltrates in the affected lobe/s.

13. Answer: d. Patients with inability to void after 6 hours, especially after a hernia repair, should undergo a bladder scan. If there is significant volume they can be straight catheterized or have a Foley inserted. If there is no volume in the bladder, a Foley can be inserted to monitor urinary output, or a fluid challenge and brief waiting period can be tried, knowing that if the patient fails the fluid challenge they will need a Foley catheter.

14. Answer: d. The patient's low normal urinary output and his FeNa less than 1% both indicate prerenal failure. The next step would be a fluid challenge and close monitoring.

15. Answer: e. In the first 24 hours after surgery nausea is typically attributed to anesthesia and other medications. After that time, ileus must be considered. A complete ileus often leads to distended small bowel and stomach which require decompression to avoid aspiration or emesis, and to encourage return of bowel function.

CHAPTER 3

1. Answer: b. (Section 1.B). Carbohydrate digestion is initiated by the action of salivary amylase, and salivary and pancreatic enzymes cleave starches into oligosaccharides. Ketone bodies form when dietary carbohydrates are not ingested after starvation. Cholecystokinin is stimulated by fat in the duodenum. Muscle breakdown occurs when energy needs are unmet by nutrition, and glycogen is depleted during starvation and stress.

2. Answer: d. (Section Stress Metabolism, A). After 12 hours carbohydrate stores are exhausted and fat and protein are broken down for calories.

3. Answer: a. (Section III.C). Nonstressed adults typically require 0.8 to 1.2 g/kg/day of protein, those who are critically ill need 1.2 to 1.5 g/kg/day. Burn, septic, and obese patients need 1.5 to 2 g/kg/day.

4. Answer: b. (Section II.B). Clinical signs of inflammation include fever, hypothermia, tachycardia, and hyperglycemia.

5. Answer: e. (Section III.B). BMI can be used to estimate caloric requirements in hospitalized patients. It is calculated by the following equations: Weight (kg)/m^2. Daily calorie estimates are

below:

BMI	Daily Energy Needs (kcal/kg/day)
<15	35-40
15-19	30-35
20-25	20-25
26-29	15-17
>29	15

6. Answer: b. (Section Nutrition Administration, C diet selection, 2 surgery-specific diets). Patients that undergo a pancreaticoduodenectomy are at a higher risk of developing dumping syndrome due to the reduction of the reservoir capacity of the stomach. When undigested, hyperosmolar food reaches the jejunum, fluid shifts into the intestine to equalize osmotic pressure lead to nausea, diaphoresis, tachycardia, bloating, diarrhea, and abdominal cramping. Patients should have small frequent meals that limit beverages, liquids, and simple carbohydrates and sugars.

7. Answer: d. (Section Routes of Nutritional Support, A). Enteral feedings may be contraindicated in patients with an intestinal obstruction, upper GI bleeding, severe diarrhea, intractable vomiting, enterocolitis, high output enterocutaneous fistula, and severe inflammatory disease. In this patient, he does have enteral access with an NGT, and none of the other choices are contraindications.

8. Answer: d. (Section Routes of Nutritional Support, A Enteral, 5 complications). Patients who suffer a CVA who have dysphagia and are unable to eat will need feeding access. Many skilled nursing facilities require a more permanent feeding tube than a small bowel feeding tube. In this patient with a large hiatal hernia with intermittent large volume emesis, a functional gastric outlet obstruction should be considered. Feeding via a nasogastric or gastrostomy tube can lead to high residuals, and he is at a higher aspiration risk after his stroke. It is unlikely if he has failed his swallow study that he will improve enough quickly to take in adequate calories. Therefore, a jejunostomy tube is the best option for long-term feeding in this patient.

9. Answer: a. (Section Routes of Nutritional Support, A Enteral, 5 complications). Refeeding syndrome is a potentially lethal complication in patients who are severely malnourished. Alterations in phosphate, potassium, and magnesium can be seen which can lead to harmful

effects on the cardiac, respiratory, hepatic, neuromuscular and hematologic systems.

10. Answer: b. (Section Routes of Nutritional Support, B Parenteral Nutrition, 2 TPN). TPN

solutions are generally administered as a 3-in-1 admixture of protein, carbohydrates, and fat. Typical dosing may be based using the patient's BMI to estimate the total daily caloric requirement.

BMI	Daily Energy Needs (kcal/kg/day)
<15	35-40
15-19	30-35
20-25	20-25
26-29	15-17
>29	15

Patients who are not stressed typically need 0.75 g/kg/IBW of protein per day. Patients who are critically ill without renal or hepatic dysfunction need 1 to 1.5 g/kg/IBW of protein per day.

11. Answer: c. (Section Routes of Nutritional Support, B Parenteral Nutrition, E Complications with TPN). Metabolic complications with TPN include electrolyte abnormalities and glucose homeostasis. While it was previously thought that strict maintenance of serum glucose levels below 110mg/dL improves mortality, it was shown in the NICESUGAR study that intensive glucose control actually increased mortality. Therefore blood glucose levels should be kept below 180 mg/dL.

12. Answer: e. (Table 3-2 Minerals). Zinc deficiency can lead to impaired wound healing, in addition to alopecia, hypogonadism, olfactory and gustatory dysfunction, and acrodermatitis. Manganese deficiency leads to dermatitis, weight loss, nausea, vomiting, and coagulopathy. Fluorine deficiency leads to dental caries, selenium deficiency leads to cardiomyopathy and copper deficiency leads to hypochromic microcytic anemia, neutropenia, bone demineralization, and diarrhea.

13. Answer: a. (Nutrition Administration, A Diet selection, 2 Surgery Specific diets, C Low-fiber diet). A low-fiber diet <10 g/day is recommended for patients going through an acute phase of IBD. A regular diet will likely contain too much fiber for this patient. A high-fat, high-protein diet is recommended for patients with high catabolism such as burn patients. She should be able to take an oral diet and not require tube feedings. A dumping syndrome diet is recommended for patients who underwent a procedure that reduced the reservoir

capacity of the stomach, compromised the pyloric sphincter or altered secretion of GI hormones such as a standard Whipple procedure, partial or total gastrectomy or antrectomy, esophagectomy or pyloromyotomy.

14. Answer: c. (Routes of Nutritional Support, B Parenteral Nutrition, 2 TPN, C Additives). Electrolytes such as potassium should be adjusted daily in the TPN. Vitamin K is added in a standard dose as are H_2-receptor antagonists, copper, and zinc.

15. Answer: d. (Routes of Nutritional Support, A Enteral, 5 Complications, F diarrhea). Diarrhea occurs in 10% to 20% of patients. After an infectious source is ruled out, an antidiarrheal medication may be started. Antibiotics will not be useful in this patient as *Clostridium difficile* was ruled out. A more concentrated tube feeding formula will cause increased diarrhea, a decreased concentration formula may be beneficial. Adding soluble fiber is an option, but should be used with caution within 7 days of a bowel resection as this patient had. Parenteral nutrition should be started only if the patient is unable to tolerate enteral feeds.

CHAPTER 4

1. Answer: e. Although the infusion of hypertonic saline solutions may be valuable in head injured patients or in patients suffering traumatic shock through the reduction of inflammatory mediators, no clinical reduction in mortality has ever been shown by the infusion of hypertonic solutions.

2. Answer: c. In a septic patient requiring high volume fluid resuscitation, albumin should be administered if the patient continues to have a low MAP after large volume crystalloid administration.

3. Answer: b. This patient has hypervolemic hyponatremia associated with congestive heart failure. Initial management includes fluid restriction to 1 L of free water per day.

4. Answer: a. Diabetes insipidus is characterized by polydipsia, polyuria, and hypotonic urine. A urine specific gravity less than 1.005 is diagnostic of diabetes insipidus.

5. Answer: b. Hyperkalemia should first prompt an EKG to look for peaked T waves, followed by a confirmatory whole blood level. Insulin and glucose are then administered. Dialysis is last in the algorithm for refractory hyperkalemia.

6. Answer: d. The most common clinical manifestation of hypomagnesemia is QT prolongation and associated ventricular arrhythmias.

7. Answer: e. The most common causes of metabolic alkalosis in postsurgical patients are dehydration from inadequate fluid resuscitation, followed by acid losses from nasogastric suction.

8. Answer: b. Bicarbonate therapy is indicated as treatment for refractory acidemia when underlying causes of acidosis have been addressed.

9. Answer: c. This patient has severe hypocalcemia. Immediate intravenous calcium therapy is indicated because of the severity of her symptoms.

10. Answer: b. Rapid correction of free water deficit can lead to cerebral edema. In order to

avoid this complication, half of the free water deficit should be corrected in the first 24 hours, and the remaining deficit should be corrected in the next 2 to 3 days.

CHAPTER 5

1. Answer: c. The patient has developed HIT type II. HIT type II is a severe immune-mediated syndrome caused by heparin-dependent antiplatelet antibodies and develops 5 to 10 days following heparin exposure. The patient needs anticoagulation because of the atrial fibrillation and new DVT; however, this should be achieved with a nonheparin anticoagulant such as bivalirudin. Transfusion of platelets is contraindicated since it will actually worsen the thrombosis.

2. Answer: d. DIC is often marked by low levels of fibrinogen. Patients present with prolonged INRs and PTTs, along with decreased fibrinogen levels. Cryoprecipitate is often used to help correct fibrinogen deficiency in DIC or as second line therapy in vWD. Transfusions of all other products are inappropriate in this case.

3. Answer: c. Antifactor VIII antibodies may develop in hemophiliacs in response to prior factor VIII infusion. Hemophilia A rarely presents with spontaneous bleeding. Since hemophilia A is characterized by decreased factor VIII levels, there is no need for transfusion of factor IX prior to surgery. Hemophilia A is an X-linked recessive disorder and is therefore found mostly in males. Platelet membrane receptors are normal.

4. Answer: e. Active menses in females is not considered an absolute contraindication. All other options are. For a list of both absolute and relative contraindications, see Table 5-4.

5. Answer: e. Von Willebrand disease is the most common inherited bleeding disorder. It is characterized by low levels of vWF (type 1) or dysfunctional vWF (types 2 and 3). Type 1 is treated with administration of DDAVP which causes endothelial release of vWF, increasing intravascular levels. Types 2 and 3 require exogenous functional vWF which can be found in cryoprecipitate.

6. Answer: b. Factor XIIIa is involved in the cross-linking of fibrin which helps to stabilize newly formed clots. It has no role in the activation of platelets. Christmas disease, otherwise known as Hemophilia B,

is marked by a deficiency in factor IX, not XIIIa. The prothrombinase assembly involves factors Va, Xa and calcium.

7. Answer: a. Prekallikrein is involved in the intrinsic clotting cascade and its deficiency causes an elevation in the PTT but does not actually cause a hypercoagulability. Protein C, Protein S, Plasminogen, and Antithrombin III are all natural anticoagulants, and deficiencies in these cause hypercoagulable states.

8. Answer: a. Antithrombin III is a major inhibitor of thrombin and Xa. Heparin, an anticoagulant, binds to antithrombin III and increases its activity. Argatroban is a direct thrombin

inhibitor while fondaparinux is an indirect factor Xa inhibitor. Antithrombin III is secreted by the liver, and its synthesis is not inhibited by warfarin.

9. Answer: a. The prothrombinase complex is composed of factor Xa and factor Va bound to a negatively charged phospholipid membrane with calcium. Heparin, an inhibitor of factor Xa and thrombin, works by activating antithrombin III. Therefore the prothrombinase complex is inhibited by heparin. Argatroban directly inhibits thrombin which is not involved. Ionized calcium is required for phospholipid binding. Clopidogrel and aspirin both decrease platelet binding by binding to the ADP receptor and inhibiting cyclooxygenase respectively.

10. Answer: e. Cryoprecipitate contains factors VIII, XII, and vWF. It is used in patients with DIC, dysfunctional vWF, and hemophilia A. FFP contains factors II, V, VII, IX, X, and XI.

CHAPTER 6

1. Answer: d. This patient has developed acute signs of local anesthetic systemic toxicity (LAST), presumably from accidental intravascular injection of local anesthetic. The priorities for management of LAST include airway management, seizure suppression, management of cardiac arrhythmias, and lipid emulsion therapy. Benzodiazepines are the preferred antiseizure medications in LAST, as propofol is a cardiovascular depressant that may exacerbate hemodynamic instability and its lipid content is too low to provide benefit (a). Hypotension and cardiovascular collapse should be treated with modified ACLS protocols, including the avoidance of vasopressin (b) and the use of smaller doses of epinephrine (<1 µg/kg) (c). High doses of epinephrine can impair resuscitation in LAST and reduce the efficacy of lipid rescue. Twenty percent lipid emulsion therapy is the preferred antidote for local anesthetic toxicity, and although the timing of infusion is controversial, it should be implemented when the clinical severity and rate of progression of symptoms suggest a high likelihood of progression to severe toxicity and cardiovascular compromise (d). Prolonged monitoring for >12 hours after any signs of LAST is recommended, since cardiovascular depression can persist or recur after treatment (e).

(See American Society of Regional Anesthesia Checklist for Treatment of LAST. *Reg Anesth Pain Med.* 2012;37(1):16-8; PMID: 22189574)

2. Answer: b. The minimum requirements for patient monitoring during anesthesia are defined by the American Society of Anesthesiologists ÒStandards for Basic Anesthesia MonitoringÓ and apply to all general anesthetics, regional anesthetics, and monitored anesthesia care. The physiologic variables that should be monitored include the patient's oxygenation (by oxygen analysis of inspired gas and pulse oximetry), ventilation (preferably by continuous end-tidal CO_2 analysis) (e), circulation (by continuous pulse, blood pressure, and ECG monitoring) (a, d), and temperature (c). During regional or local anesthesia without an endotracheal tube or supraglottic airway, ventilation should be assessed by noninvasive end-tidal CO_2 monitoring or, if not feasible, by continuous observation of qualitative clinical signs. Cerebral oximetry may reflect cerebral

tissue oxygenation more accurately than pulse oximetry and is gaining more widespread use in major cardiac and vascular surgery, however its use remains controversial and is not included in the standards for basic anesthesia monitoring (b). (See American Society of Anesthesiologists Standards for Basic Anesthesia Monitoring, July 2011; http://www.asahq.org/÷/media/Sites/ASAHQ/ Files/Public/Resources/standards-guidelines/standards-for-basicanesthetic-monitoring.pdf)

3. Answer: d. Cervical blockade is used to achieve anesthesia of the neck and was historically used primarily for carotid endarterectomy, although its use is now falling out of favor (a). Interscalene (b), supraclavicular (c), and infraclavicular (d) blockade will all provide anesthesia of the upper arm and are acceptable techniques for humerus fixation. However, of these the infraclavicular approach carries the lowest risk of ipsilateral phrenic nerve palsy and is therefore preferred to minimize the risk of pulmonary complications in patients with COPD. Axillary blockade (e) provides reliable anesthesia only for procedures below the elbow.

4. Answer: b. The diaphragm (b) exhibits the most rapid recovery from neuromuscular blockade. Recovery of upper airway, pharyngeal, and flexor hallucis muscles generally parallels that of the adductor pollicis, which can be used for twitch monitoring as an indicator of extubating conditions. Both the muscles of respiration and the muscles that protect the airway must recover in order to extubate the patient safely.

5. Answer: c. The patient is exhibiting signs concerning for malignant hyperthermia (MH), which is most often triggered by the administration of volatile anesthetics (e.g., sevoflurane) or succinylcholine and is characterized by hyperthermia, tachycardia, tachypnea, hypertension, acidosis, and skeletal muscle rigidity. The first step in management for an acute MH event is the immediate cessation of triggering agents (c). Other management steps that should be rapidly performed include calling for an MH cart, administration of dantrolene 1 to 2.5 mg/kg IV

(a), and hyperventilation with 100% oxygen to flush volatile anesthetics and lower end-tidal CO_2 (b). Monitoring for hyperkalemia and associated electrocardiogram changes is important (d). A family history (e) of malignant hyperthermia greatly increases the risk of an MH event, and this history should be explicitly sought during preoperative evaluation and the anesthetic plan tailored accordingly. (See Emergency treatment of an acute MH event, Malignant Hyperthermia Association of the United States (www.mhaus.org))

6. Answer: a. Intraoperative awareness is the unintended consciousness and recall of intraoperative events, which may result in long-term psychological sequelae for the patient. Risk factors for awareness include use of neuromuscular blockade (a), use of total intravenous anesthesia (as opposed to inhalational anesthetics [b]), and inadequate anesthetic dosing due to a variety of technical, surgical, or patient-related factors. Patient-related factors increase resistance to anesthetics and therefore increase the risk of awareness include pyrexia, hyperthyroidism (e), obesity, anxiety, younger age (c), emergency surgery (d), and chronic

exposure to tobacco, alcohol, recreational drugs, or anesthetic agents.

7. Answer: b. Contraindications to the use of succinylcholine for neuromuscular blockade include patients with severe burns (a), muscular dystrophy (c), hyperkalemia (d), and a family or personal history of malignant hyperthermia (e). Succinylcholine does have a more rapid onset of action (30 to 60 seconds) than any of the nondepolarizing neuromuscular blocking agents (90 seconds for high-dose rocuronium), and is therefore the preferred agent for rapid sequence (b) and emergent intubation in the absence of contraindications. The short duration of action of succinylcholine (5 to 10 minutes) is also of benefit in the case of blunt head injury or some elective surgeries, when the ability to complete a neurologic examination soon after induction is desired.

CHAPTER 7

1. Answer: d. This patient likely had a PA rupture due to balloon inflation. This patient should have the side of the PA catheter in the dependent position and an urgent thoracic surgical consult should be obtained.

2. Answer: b. The sedating medication and sedation goal should be decided upon and communicated to the bedside nurse who will titrate the sedative to reach the desired goal. Sedation should be minimal to keep the patient comfortable and interrupted for a daily sedation vacation. A BIS of 40 to 60 is the goal for a patient receiving a neuromuscular blockade. Propofol leads to hypotension due to increased venous capacitance and decreased preload. Ketamine is often used in patients with depressed cardiac function due to its lack of cardiac depression.

3. Answer: a. This patient has a TI fistula with a Òherald bleedÓ the day prior. The treatment is as described, and must be done urgently if the patient is to survive.

4. Answer: d. APRV or BiLevel is an advanced ventilatory mode. It uses an inverse I:E ratio so that there is more time spent at the pressure high to increase the mean airway pressure without increasing the peak. Ventilation occurs during spontaneous breathing over the pressure high and during the pressure release to pressure low.

5. Answer: c. In a patient with a poorly developed tract after tracheostomy placement, if it is inadvertently removed, the patient should be intubated from above. Once an airway is secured, the tracheostomy can be replaced in a more controlled setting. The blind replacement of a tracheostomy tube can result in placement in the pretracheal space and potentially the patient's demise.

6. Answer: e. The findings listed above are consistent with cardiogenic shock.

7. Answer: e. The current guidelines suggest that steroids should be given to septic patients who do not respond to volume or vasoactive medications. A cortisol level does not need to be checked, they should be given hydrocortisone 50 mg IV Q6 hours. The data seems to show a

quicker duration of sepsis, however, the impact on survival is less clear.

8. Answer: b. This patient most likely has a tension pneumothorax and should be treated with needle decompression and tube thoracostomy.

9. Answer: a. Stress ulcer prophylaxis should be administered selectively in the ICU to patients with a high risk because its use does increase the risk of *C. difficile* infection.

10. Answer: c. Information from the TRICC trial illustrates the futility of blood transfusion when unnecessary. A restrictive transfusion protocol would necessitate transfusion only if the hemoglobin is <7 mg/dL, unless the patient has had a recent cardiac event.

CHAPTER 8

1. Answer: c. (Assessment and Management of Burns, Section B.1). As with any trauma, airway is the first priority. Although the patient is maintaining his oxygen saturation, he is tachypneic and showing signs of smoke inhalation with facial burns and hoarseness. He should be intubated before proceeding to the rest of the primary and secondary survey.

2. Answer: b. (Burn-specific Secondary Survey, Table 8-1). First-degree burns are red but have no blisters. Second-degree burns have blisters and are painful. Third- and fourth-degree burns are insensate.

3. Answer: b. (Burn-specific Secondary Survey, Section G.2). The Òrule of 9Ós helps determine the percent body surface area. The anterior surface of each leg is 9%, and the anterior torso is 18%. Note, children have a different percentage distribution.

4. Answer: e. (Management, Section A.7). Pseudomonas and fungi are the most common causes of burn wound sepsis

5. Answer: a. (Management, Section 9.2). Mafenide acetate can lead to a metabolic acidosis due to carbonic anhydrase inhibition.

6. Answer: b. (Burn Mechanisms: Special Considerations, Section B). Patients with electrical burns are at risk for renal failure due to rhabdomyolysis. The release of myoglobin from injured cells can lead to precipitation in the renal tubules.

7. Answer: e. (Management). Although burn patients are at risk for infection and sepsis, there is no role for prophylactic antibiotics in the management of burns.

CHAPTER 9

1. Answer: e. (Acute Wound Healing, Section C.3). Even well-healed wounds never reach the original strength of uninjured tissue.

2. Answer: a. (Chronic Wound Healing, Leg Ulcers, Section B.1). Wounds that are not infected do not require antibiotics. It is important to establish if there is any arterial insufficiency that is

impeding adequate healing. This patient is showing signs of rest pain and tissue loss, suggestive of an arterial inflow problem that must be addressed before debridement in the setting of a noninfected wound.

3. Answer: e. (Chronic Wound Healing, Pressure Ulcers, Section C.1). Refer to Table 9-1 for pressure ulcer staging. The stage of the ulcer is known until the eschar is incised and the depth of necrosis determined.

4. Answer: b. (Chronic Wound Healing, Pressure Ulcers, Section C.3). In a noninfected wound, there is no reason to use Dakin's or hydrogen peroxide as they impede wound healing. The patient is at high risk for recurrence due to being bedridden and should not get a musculocutaneous flap. Normal saline damp to dry dressing changes continue to debride tissue with every dressing change.

5. Answer: c. (Types of Wound Closure, Section B). This wound will heal by secondary intention. The wound is left open and will heal by contraction and epithelization.

6. Answer: e. (Open Wound Care Options, Section L). NPWT have multiple benefits and increases wound healing by decreasing edema and increasing capillary growth, thereby increasing granulation tissue. The VAC also protects the wound from any external contaminants.

7. Answer: d. (Open Wound Care Options, Section L). Contraindications for negative pressure dressings include infected wounds, wounds with exposed blood vessels, and wounds with cancer.

CHAPTER 10

1. Answer: c. Using the Canadian Head CT rules, this patient qualifies for an NCCT. He has a battle sign (redness behind the ears), which is a sign of basal skull fracture. Other factors which are concerning in this patient is the mechanism of injury and the bout of emesis. Observing the patient in the ED for 4 hours and admitting the patient for observation are not advocated due to the higher risk of an intracranial injury in this patient. The mechanism of injury does not warrant a CTA of the head or neck for initial evaluation of the patient.

2. Answer: d. The blown pupil should cause one to be concerned for increased intracranial pressure. Initiating emergent protocols to reverse the ongoing process is critical. The patient is already intubated, hence hyperventilating the patient, elevating the head of the bed, and bolusing Mannitol are actions that could be quickly performed. Delaying therapy for imaging results or consultation of another service should be avoided. There is no indication for steroid therapy in this clinical scenario.

3. Answer: c. Using the Denver Screening Criteria this patient qualifies for a CTA of the neck. He has a significant mechanism, basilar skull, facial, and cervical spine fractures. The patient is at risk for a concurrent spine injury; however, a CT scan is the preferred imaging modality. Irrigation and repair of his wound would be needed, but this is not emergent. Diagnosing and treating a

BCVI reduces the morbidity of this patient population. A chest tube may be needed in this patient in the future, but the current size of his pneumothorax does not warrant an intervention. Repeat NCCT and c-spine CT should not be done routinely and should only be considered on patients in which the OSH films are not available or are not adequate.

4. Answer: b. Based on the New Orleans criteria and the Canadian CT head rule the patient will need a CT scan of the c-spine to rule out a potential injury. He will also need to be reassessed when sober. However, a cervical collar should be placed initially to prevent the propagation of any cervical spine injury. An MRI is not the preferred screening modality.

5. Answer: d. The above patient has a possible airway injury (inaudible voice) and impending airway loss (expanding hematoma). The patient is also hemodynamically unstable. The ABC's should be performed before proceeding with any intervention.

CHAPTER 11

1. Answer: d. The patient is suffering from a tension pneumothorax. This is a life-threatening condition which leads to obstructive shock. Signs

and symptoms include absent breath sounds on the affected side, tracheal deviation to the contralateral side, hypotension, tachycardia, hypoxia, and mediastinal shift to the opposite side. Tension pneumothorax should be diagnosed clinically. Initial emergent treatment is needle decompression with a 14-gauge angiocatheter placed in the second intercostal space in the midclavicular line. Thoracostomy tube placement follows initial emergent needle decompression.

2. Answer: b. While the majority of hemopneumothorax in trauma can be managed by simple thoracostomy tube placement, according to trauma.org, emergent thoracotomy in the operating room following thoracic trauma should be undertaken if initial chest tube output is 1,500 mL or 200 mL/hour of blood is evacuated over the following few hours.

3. Answer: c. Resuscitative thoracotomy is performed in the emergency department in certain instances of thoracic trauma. The steps include left-sided thoracotomy in the fifth intercostal space, dissection and division of the inferior pulmonary ligament, incision in the anterior pericardium with subsequent evacuation of clot and blood, repair of any cardiac injury, and (in some cases) cross-clamp of the thoracic aorta.

4. Answer: a. According to the EAST guidelines, the initial diagnostic test to evaluate for BCI is an EKG. See Table 11-2 for EAST recommendations.

5. Answer: c. Aerodigestive injury can be difficult to diagnose, but can represent significant morbidity in thoracic trauma. A high index of suspicion for injury to the trachea or esophagus is essential for prompt diagnosis and management. Initial testing includes bronchoscopy and esophagoscopy or esophagography. While CXR is usually performed early in any trauma work-up and can reveal mediastinal gas or pleural fluid, sensitivity for aerodigestive injury is low and direct evaluation of the trachea and esophagus are required to make the diagnosis.

6. Answer: d. Tension pneumothorax, cardiac tamponade, and pulmonary embolism are all capable of causing obstructive shock. While the etiologies and treatments of these potentially life-threatening conditions are often quite different, each can potentially cause obstruction of cardiac outflow with subsequent hemodynamic collapse. Because of this, these three disorders have been lumped into their own separate category of shock.

CHAPTER 12

1. Answer: b. This patient has an extraperitoneal rectal injury. Although older studies advocated diversion, distal rectal washout, and presacral drainage (the so-called Ò3 DsÓ), further research demonstrated this resulted in overall worse outcomes and no improvement in the rate of pelvic sepsis. However, diversion is still mandatory.

2. Answer: c. This patient has a duodenal hematoma and cannot tolerate oral intake. Given the self-limited nature of most duodenal hematomas, generally nonoperative management with nasogastric drainage (if needed) and TPN is recommended, awaiting resolution of the hematoma.

3. Answer: a. This patient is in severe shock. As all trauma patients are assumed to be in hemorrhagic shock until proven otherwise, you must find the source of bleeding causing his shock. Given that he has no obvious signs of external bleeding, his chest and pelvis x-rays are normal, and he has no obvious extremity fractures, the most likely source of bleeding is in his abdomen. The patient is too unstable for transport to the CT scanner, thus FAST is the best choice. DPL would be a reasonable option if FAST was not available or the FAST was negative.

4. Answer: d. This patient suffered a flank GSW and is hemodynamically normal without peritonitis. Given that the majority of flank GSWs do not penetrate the peritoneal cavity, the next best step is to obtain a CT scan to delineate the bullet's trajectory. If the bullet clearly did not violate the peritoneum, he can be safely discharged. Thus, neither immediate laparotomy nor serial abdominal examinations are indicated.

5. Answer: b. This patient has a Grade IV blunt liver injury. Most blunt hepatic trauma can be managed nonoperatively. In fact, operating on hemodynamically normal patients can result in disruption of the clot around the liver and the release of the natural tamponade of a closed abdomen, worsening the degree of hemorrhage. However given the severity of injury on imaging, the patient should be admitted to the ICU for close observation. Evidence of contrast extravasation (i.e., a ÒblushÓ) should trigger referral to IR for angiography and possible embolization. Ongoing transfusion requirements or the development of hemodynamic instability of peritonitis is an indication for immediate laparotomy.

6. Answer: d. Given the patient's profound hypothermia, acidosis, and coagulability, and that the bleeding is temporarily controlled, the operation should be aborted and the patient taken to the ICU for further resuscitation. More formal attempts at controlling bleeding at this time are

likely to be futile.

7. Answer: a. This patient is in hemorrhagic shock and needs to be given blood products as soon as possible. Given that she is a female of child-bearing age, she should be given uncrossmatched Type O negative blood until crossmatched blood is available in order to prevent the development of Rh sensitization.

8. Answer: b. After splenectomy, patients are susceptible to infections by encapsulated organisms, thus B is correct.

9. Answer: c. This patient is hypotensive and has an open-book pelvic fracture. Placing a binder will help stop bleeding from the pelvic

fracture. The other options may be necessary later, but a binder should be the first step.

10. Answer: e. This patient has a <50% circumferential injury to the colon within minimal contamination and is hemodynamically normal. Closure of the defect alone is sufficient.

CHAPTER 13

1. Answer: c. To adequately decompress the deep posterior compartment, the surgeon must visualize and incise the fascia over the entire length of the compartment. To accomplish this proximally, the soleus origin must be partly taken down to gain access to the proximal portion of the deep posterior compartment.

2. Answer: b. Using pressure measurements for diagnosis of compartment syndrome has been done for decades, but benchmark pressures were challenged by a group in Scotland establishing the Òdelta 30Ó criteria, by which the compartment pressure is compared to the current diastolic blood pressure. A difference of less than 30 mm Hg is diagnostic, and follow-up studies confirmed no sequelae of missed compartment syndrome of the leg in patients with tibia fracture.

3. Answer: c. Patterns of knee injury, including tibial plateau fractures, with knee dislocation are commonly associated with popliteal artery injury (up to 30%) because of bony displacement of the tibia relative to the femur and the tethering effect of the adductor hiatus and the soleal arch.

4. Answer: c. Initial volume containment of the retroperitoneum can be accomplished very quickly with a sheet or binder following evaluation of the abdomen. A study of practice guidelines in Australia demonstrated a decrease in mortality comparing pre- and postimplementation, with a goal of abdominal ÒclearanceÓ and noninvasive pelvic binding within 15 minutes.

5. Answer: d. Polytrauma patients with femur fractures, especially those with thoracic injury, are predisposed to a second-hit phenomenon that can result in ARDS following intramedullary nail fixation of the femur in an under-resuscitated state. To accomplish the goal of femur fracture stabilization within 24 hours of injury, in the setting of ongoing resuscitation requirements, external fixation (Òdamage control orthopedicsÓ) has been recommended to control pain, allow for comfortable positioning changes, decrease the risk of fat embolism, and avoid the

complications associated with instrumenting the medullary canal.

6. Answer: c. Most missed injuries occur in the distal extremities and are more common in patients who have higher ISSs, are intubated, are taken directly from the ER to the OR, or have altered mental

status. A formal tertiary survey has been demonstrated to reduce missed injuries.

7. Answer: b. The tibia pilon (from the French for pestle or hammer) fracture is usually the result of a fall from height or an MVC with axial load through the hindfoot resulting in energy absorbed through the distal end of the tibia and the soft tissues of the distal leg.

8. Answer: c. Two-stage treatment of tibia pilon fractures was demonstrated in the late 1990s to decrease soft tissue compromise (leading to infection) that had been associated with open approaches to reduction and fixation in the preceding decades through soft tissues that were severely traumatized and swollen in the acute phase of injury.

9. Answer: c. Reduction and stabilization of femur fractures decreases the risk of malunion, fat embolism, blood loss at the fracture, and nonunion. Stabilization of these fractures within 24 hours was demonstrated to decrease ICU length of stay, and possibly the complication of ARDS. However, more recent publications have demonstrated an increased risk of ARDS for ÒborderlineÓ patients who are under-resuscitated prior to intramedullary fixation of femur fractures.

10. Answer: c. Loss of wrist and digit extension due to an injury to the radial nerve is commonly associated with humerus shaft fractures. While radial nerve palsy is common with humerus shaft fractures, neurapraxia (stretch but no transection of the nerve) is the most common nerve injury. Most neurapraxia will recover. Transection of the nerve that would benefit from early surgical intervention is, most commonly, the result of a high energy injury resulting in an open fracture or a direct laceration of the nerve from a stab wound.

CHAPTER 14

1. Answer: c. Given this patient's age, diabetes, and compromised renal function, his likelihood of needing dialysis access in the future is high. Therefore, it would be prudent to avoid the subclavian access sites to minimize the risk central stenosis and loss of potential sites for AV fistula. A femoral CVAD would not be ideal in this patient because his habitus would make maintaining a sterile dressing difficult and his diabetes increases his risk of infection.

2. Answer: d. This patient will likely require cardiopulmonary resuscitation. A femoral CVAD does not interfere with the other procedures of cardiopulmonary resuscitation and is therefore preferred approach when there is no injury to the vena cava.

3. Answer: a. Catheters placed during a medical emergency or code where sterile technique cannot be assured should be replaced within 48 hours of insertion to minimize the risk of CRBSI.

4. Answer: c. In a patient with an ileus, a midline approach (A) would place the patient at a greater risk for intestinal puncture. The upper quadrants are not advisable for abdominal paracentesis due to the risk of puncturing the liver or the transverse colon. The safest place for an abdominal paracentesis in this patient would be a lower quadrant approach.

5. Answer: c. 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, with the vein lying 1 to 2 cm medial to this point. In a code situation, it is unlikely that you will have time to utilize ultrasound guidance and waiting for spontaneous return of circulation would delay central access unnecessarily.

6. Answer: b. Catheters larger than 7F should be removed in a setting in which operative repair of the arteriotomy can be achieved.

7. Answer: d. While answers A, B, and C have relative contraindications to placement of a right subclavian CVAD, the patient described in D has symptoms of a right upper extremity DVT. Venous thrombosis is an absolute contraindication to catheter placement at the affected site.

CHAPTER 15

1. Answer: e. The initial pain in acute appendicitis is poorly localized to the periumbilical region, which is visceral in nature. Once the inflammation involved the parietal peritoneum, the pain is better localized to the right lower quadrant.

2. Answer: b. Abdominal pain followed by loss of consciousness suggests an intra-abdominal catastrophe with associated sepsis and/or shock. The first step is resuscitation, basic labs, and a quick abdominal film to rule out a perforated viscus. Induction of anesthesia of an unresponsive patient with sepsis or shock will often precipitate cardiovascular collapse.

3. Answer: c. This patient may have acute appendicitis. An ultrasound is the best next step in the diagnostic workup of a pregnant patient with right lower quadrant pain, when appendicitis is suspected. If the appendix cannot be visualized on ultrasound, an MRI can be performed.

4. Answer: b. Appendectomy is the treatment of choice for acute appendicitis. The risk of fetal loss is as high as 35% if there is progression to perforation. Therefore, urgent appendectomy (laparoscopic or open) is needed.

5. Answer: e. This patient has sigmoid volvulus without evidence of necrotic bowel. The first step is endoscopic reduction followed by elective sigmoidectomy after bowel preparation.

6. Answer: c. This patient has sigmoid volvulus without evidence of necrotic bowel. The first step is endoscopic reduction followed by elective sigmoidectomy after bowel preparation.

7. Answer: a. This patient likely has an acute abdomen with peritonitis. A radiographic

abdominal obstructive series with a chest x-ray is a fast way to determine if the patient has a perforated viscus, and should be the first diagnostic test. If there is intraperitoneal free air, the patient needs an urgent operation.

8. Answer: c. This patient has a ruptured abdominal aortic aneurysm, which carries an extremely high mortality. Time is of the essence. Given that the patient is hypotensive and acidotic, urgent repair is indicated. There is no time for imaging.

CHAPTER 16

1. Answer: d. A type III hiatal hernia is more common than a pure type II and involves the herniation of both the greater curvature of the stomach and the GE junction into the chest.

2. Answer: c. Frequently with type III hiatal hernias the esophagus is shortened since the GE junction and the greater curvature of the stomach have herniated into the chest. In order to perform the repair it may be necessary to perform a lengthening procedure so that the repair may sit in the abdominal cavity without tension.

3. Answer: a. Characteristics of a manometrically abnormal LES are (1) a resting pressure less than 6 mm Hg, (2) an overall length less than 2 cm, and (3) an abdominal length less than 1 cm. A patient with one or more of these abnormal values has a 90% probability of having reflux.

4. Answer: a. The first line of therapy for mild to moderate GERD is lifestyle adjustments including losing weight, stopping smoking, reducing consumption of alcohol, peppermint, caffeine, and avoiding lying down after meals.

5. Answer: e. The Toupet fundoplication is the preferred posterior fundoplication for GERD patients with abnormal esophageal motility due to a lower incidence of postoperative dysphagia.

6. Answer: c. 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 wrap. The ability to belch is preserved, thereby avoiding gas-bloat syndrome.

7. Answer: e. While all of the options are potential symptoms of achalasia, virtually all patients will experience progressive dysphagia.

8. Answer: a. Due to the patient's comorbidities, he may be a candidate for peroral endoscopic myotomy, a less invasive approach than a

modified Heller procedure and more effective than a pure anti-reflux (Toupet) procedure. He does not need a gastrostomy tube since achalasia improves with myotomy in ÷90% of patients (*Adv Surg.* 2014;48:27-41).

9. Answer: c. Nutcracker esophagus is 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.

10. Answer: d. Zenker is the most common type of symptomatic diverticulum. A hypertensive upper esophageal sphincter (UES) or uncoordinated pharyngeal contraction and opening of the UES results in increased pharyngeal intraluminal pressure. Herniation of only the mucosa and submucosa results in this false diverticulum.

11. Answer: d. About 75% of esophageal perforations occur secondary to instrumentation injuries. These are most likely to occur at anatomic sites of narrowing of the esophagus (e.g., at the cricopharyngeus and GE junction).

12. Answer: a. With low-grade dysplasia, the patient requires frequent follow-up with surveillance esophagoscopy and biopsy. If the patient has been on medical therapy it should continue. If the patient is asymptomatic they should start medical therapy once a biopsy shows low-grade dysplasia. The c, d, and e treatments are for high-grade dysplasia or adenocarcinoma.

13. Answer: b. Early detection offers the best opportunity to improve survival after resection, which is 20% at 5 years for all patients with cancer but far higher in those detected by surveillance and screening.

14. Answer: a. Risk factors for esophageal adenocarcinoma include white race, GERD, Barrett esophagus, obesity, and cigarette smoking. Risk factors for squamous cell carcinoma include African American race, alcohol and cigarette use, achalasia, caustic esophageal injury, and geographic locations of China, South Africa, France, and Japan.

15. Answer: b. Intramural tumors, like leiomyomas, are typically asymptomatic, but can produce dysphagia or chest pain if large enough. Intramural tumors usually can be enucleated from the esophageal muscular wall without entering the mucosa. Intraluminal tumors, like polyps, cause esophageal obstruction, and patients present with dysphagia, vomiting, and aspiration. Intraluminal tumors can usually be removed endoscopically.

CHAPTER 17

1. Answer: d. Patients with significant gastric losses, such with prolonged vomiting or nasogastric tube suction, experience a hypochloremic, hypokalemic metabolic alkalosis.

2. Answer: e. Low-grade mucosal associated lymphoid tissue lymphomas of the stomach are thought to arise as a result of *H. pylori* infection. First-line treatment of this disease begins with *H. pylori* eradication, which will often result in cure.

3. Answer: b. Bleeding duodenal ulcers are usually located on the posterior wall. In the hemodynamically unstable patient, unable to tolerate endoscopy, duodenotomy with three-point ligation is the treatment of choice. Vagotomy has largely been abandoned due to the added morbidity with the high efficacy of proton pump inhibitors.

4. Answer: c. Gleevec is first-line therapy for metastatic or recurrent gastrointestinal stromal tumors.

5. Answer: d. A minimum of 15 lymph nodes should be resected during gastric cancer lymphadenectomy for adequate staging and possibly therapeutic control.

CHAPTER 18

1. Answer: b. The indications for bariatric surgery include:

- BMI index of 40 or greater
- BMI index of 35 or greater with one or more weight-related comorbidities
- BMI index of 30 to 34.9 with poorly controlled diabetes or metabolic syndrome

2. Answer: a. Intravenous fluid resuscitation and prompt surgical intervention is warranted in an unstable patient with imaging evidence of obstruction. Observation is inappropriate in this patient. Further imaging is unnecessary and will likely delay delivery of appropriate care.

3. Answer: e. Persistent nausea, vomiting, regurgitation following LAGB should be treated with immediate removal of the fluid from the adjustable band as these symptoms suggest that the band is too tight. Imaging can be used to assess band positioningÑabdominal x-rays and esophagram with Gastrografin provide the most useful information. If plain films are not helpful or the patient's symptoms appear unrelated to the adjustable band, a CT scan may be obtained. Nasogastric tube placement is not indicated at this point.

4. Answer: d. In asymptomatic incisional hernias, repair should be deferred until weight loss has stabilized (typically around 12 to 18 months postoperatively) and nutritional status is optimized (to allow for good healing). If patient is symptomatic, or presents with evidence of incarceration or strangulation, prompt surgical management is warranted. Decreasing the frequency of exercise is not necessary and may impair weight loss.

5. Answer: e. This patient has a marginal ulcer, a complication noted in approximately 16% of patients undergoing RYGB. Medical therapy

with proton pump inhibitors and sucralfate is employed. Surgical therapy is rarely necessary since medical therapy is usually effective.

CHAPTER 19

1. Answer: c. The most common cause of intestinal obstruction is adhesions from prior surgery. In children and patients without previous surgery, hernias are the most common cause.

2. Answer: b. This patient has peritonitis with signs of systemic inflammatory syndrome and is likely septic from ischemic bowel. He needs an urgent exploration.

3. Answer: a. This patient has an incarcerated inguinal hernia with resulting intestinal obstruction. Given that there are no obvious signs of strangulation, this patient should undergo attempted reduction followed by hernia repair, preferably during the current hospital admission.

4. Answer: d. This patient has an enterocutaneous fistula, and is 2 weeks out from her operation. An operation at this point would not be advisable, and ideally should be delayed 4 to 6 months if spontaneous closure does not occur.

5. Answer: a. This patient has metastatic gastrointestinal stromal tumor (GIST). Imatinib (Gleevec) has been showed to improve survival.

6. Answer: c. Octreotide is a somatostatin analogue that is useful for controlling the symptoms of neuroendocrine tumors.

7. Answer: b. Patients with short bowel syndrome requiring long-term TPN can have numerous complications, such as central venous catheter blood stream infections, liver failure, and central venous stenosis.

CHAPTER 20

1. Answer: c. This patient has an urgent indication for operative repair given the incarcerated bowel in a hernia. Medical optimization would play a larger role in an elective setting. In patients with platelets >50,000/uL, invasive procedures are safe from spontaneous bleeding events. After successful repair, attention must be paid to minimizing the risk of postoperative ascites leak, which can be associated with wound complications, sepsis, and death. Paracentesis prior to operative repair may achieve this end temporarily, but is no more effective than fluid removal intraoperatively. A drain should be placed intraperitoneally to allow periodic drainage of ascitic fluid to promote wound healing. There is no role for antibiotics or conservative management in this patient, even if she is a cirrhotic.

2. Answer: d. In this patient with likely variceal bleeding, the first step is to insure he is resuscitated and monitored in a higher level of care.

Transfusions may be required with active bleeding, however transfusions for hemoglobin >7 g/dL is not necessary. A nasogastric tube may be placed if the patient continues to have hematemesis; however gastric lavage would likely confirm what is already suspected about the upper gastrointestinal source. Propranolol is best initiated as a prophylaxis and has no role in an acute setting. Instead, vasoactive substances such as octreotide or vasopressin are required. Endoscopy is the mainstay treatment for acute variceal bleeding since it is diagnostic and therapeutic. TIPS may be considered if there is recurrence of variceal bleeding after endoscopy.

3. Answer: e. This patient is a favorable candidate for surgical resection of her HCC. Biopsies are not required if the imaging findings are characteristics of HCC. Although liver transplantation has better outcomes than surgical resection, in particular with lower recurrence risks, this patient does not have evidence of portal hypertension or significant liver dysfunction. Thus, there is a

benefit to early resection as opposed to permitting disease progression while on the transplant list. Radiofrequency ablation is useful in patients that are not operative candidates, but in tumors larger than 3 to 4 cm, there is a high risk of recurrence. In performing the resection, a nonanatomic resection with adequate margins of 1 to 2 cm may leave behind larger volumes of parenchyma than a formal anatomic resection in an already diseased liver.

4. Answer: b. Surgical resection is not advised when there is more than a solitary lesion. TACE, RFA, and sorafenib therapy can all be used to bridge to a liver transplant.

5. Answer: a. A hepatic adenoma >5 cm in size should be excised because of its bleeding risk and malignant potential. Smaller adenomas may be observed, especially after cessation of contraceptive use when spontaneous regression is possible. All the other lesions described, including FNH, a hepatic cyst, hemangioma and bile duct hamartomas may be observed if not symptomatic.

CHAPTER 21

1. Answer: c. This patient meets the diagnostic criteria for mild acute cholecystitis according to the Tokyo guidelines, although he does not have an impressive CT scan. In approximately 40% of patients, CT shows gallbladder distension, and in 50%, there is mild pericholecystic fat stranding. Other findings include gallbladder wall edema, pericholecystic fluid or abscess. Although ultrasound is more specific and sensitive as a diagnostic test than CT, it is not uncommon to find the CT ordered first in the emergency room in the middle of the night prior to surgical consultation. Given the imaging findings, symptoms, and laboratory data, there is high suspicion that this patient has acute cholecystitis, and thus further imaging studies are not necessary,

although commonly an additional test may be ordered if the reads are equivocal. Nevertheless, initial management is to begin antibiotics and fluid resuscitation. Laparoscopic cholecystectomy in this patient is not an emergent indication, and can typically wait until there is more staff in the hospital for a safer procedure. Finally, although the patient is male and elderly, factors portending a difficult cholecystectomy, there is no other patient factor (e.g., symptom duration >72 hours, comorbidities and vital sign abnormalities) that preclude early cholecystectomy.

2. Answer: d. This patient likely has biliary pancreatitis. Although she has a mild bilirubinemia, the absence of CBD dilation or stone on imaging and a relative low bilirubin level (<1.8 mg/dL) do not warrant an extra procedure or diagnostic test such as ERCP or MRCP to investigate for choledocholithiasis. Present studies favor interval LC with IOC as opposed to ERCP for this patient with intermediate risk for choledocholithiasis. The best treatment is thus offered during the same admission after resolution of abdominal symptoms and laboratory abnormalities. Given the likelihood of recurrence and the morbidity of recurrent pancreatitis, the patient should not be discharged home without definitive therapy. Although she is obese and recently had a C-section, these findings are not contraindications to laparoscopic surgery.

3. Answer: a. This patient has signs and symptoms concerning for ascending cholangitis, although he was treated for concern of acalculous cholecystitis as well. In this elderly male with jaundice but without signs of cholelithiasis, there should be suspicion of a biliary tract neoplasm causing obstruction. The workup for hilar cholangiocarcinoma includes obtaining tumor markers such as CEA and CA19-9, MRCP to determine the relationship of the lesion to the biliary anatomy, as well as endoscopy with cytologic brushings. Nevertheless, in this patient who would likely be a poor operative candidate and has a potentially life-threatening problem, a decompressive PTC would be the next best treatment for his ascending cholangitis.

4. Answer: e. In this patient with a T1a mass, cholecystectomy with a negative cystic margin should result in less than 5% risk of recurrence. Further interventions such as re-resection of the gallbladder fossa, serial imaging, and laboratory value checks have not been validated in the literature as additional steps in management.

5. Answer: b. In a suspected bile duct injury during cholecystectomy, an intraoperative cholangiogram is the next best step to facilitate further operative planning. Because the surgeon is already asking for assistance regarding this difficult gallbladder, it would be unreasonable to ask him to convert the operation to an open procedure and attempt any kind of repair. On the other end of the spectrum, simply transferring the patient to a specialist before establishing that there is in fact an injury is premature. Finally, a suspected bile duct injury should

ideally be treated in an early rather than delayed fashion. Because missed injuries can become severe complications, the patient should not be discharged home before an injury is ruled out.

CHAPTER 22

1. Answer: a. Age greater than 55 is associated with increased mortality according to Ranson criteria for nongallstone pancreatitis.

2. Answer: b. All patients with pancreatitis and no clear etiology should undergo a right upper quadrant ultrasound to evaluate for gallstones. If gallstones are present, she should undergo a cholecystectomy prior to discharge.

3. Answer: c. This patient has an asymptomatic side branch IPMN. He can be observed with serial imaging, since he has no mention of worrisome features.

4. Answer: a. This patient has a mucinous cystic neoplasm (MCN) of the pancreas. All MCNs should be resected given that they are considered premalignant, with the potential to become cancer.

5. Answer: c. New onset of blood in the drain of patient 2 weeks after a pancreaticoduodenectomy is evidence of a pseudoaneurysm until proven otherwise. The best course of action is an angiogram with embolization, if a pseudoaneurysm is identified.

6. Answer: b. One must maintain a high level of suspicion for cancer in any patient who

presents with jaundice and a pancreatic head mass. Patients with resectable pancreatic cancer in the pancreatic head who are good surgical candidates should undergo pancreaticoduodenectomy.

CHAPTER 23

1. Answer: d. The first-line therapy for TTP is plasmapheresis. Plasmapheresis has improved initial response (47% vs. 25%) & 6-month survival (78% vs. 63%) compared with plasma infusion (*N Eng J Med.* 1991:325:393-397). Second-line medical therapy includes rituximab, cyclosporin, and increased frequency of plasmapheresis. Splenectomy in patients who do not respond to medical management has limited utility and has only shown benefit in the setting of continued plasmapheresis. ADAMT-13, a von Willebrand factor cleaving protein, is often severely deficient and levels have been shown to increase following splenectomy.

2. Answer: d. The majority of splenic abscesses arise from seeding of the spleen by a distant site, most commonly endocarditis and urinary tract infections. Abdominal CT and/or ultrasound imaging are the diagnostic modalities of choice. CT images reveal a low intensity lesion that does not enhance with contrast. Staphylococcus and streptococcus account for the most commonly identified organisms.

Fungal infections are rare, and may resolve with antifungal treatment alone. Percutaneous drainage may be used in select cases, however splenectomy and appropriate antibiotic therapy is definitive treatment.

3. Answer: d. Overwhelming postsplenectomy sepsis is relatively rare, however can be rapidly fatal if it does develop. OPSS is uncommon following splenectomy for trauma. *S. pneumoniae,* followed by *H. influenzae* type B, meningococcus, and group A streptococcus account for the majority of cases. Initial antibiotic therapy should include vancomycin and a third generation cephalosporin such as ceftriaxone or cefotaxime. Children under 5 years of age and the immunocompromised are at particular risk, likely because they produce an insufficient immune response to the pneumococcal vaccination. Recommendations for children include prophylaxis with oral penicillin daily until age 5 and/or until 1 year following splenectomy.

4. Answer: d. ITP is the most common reason for elective splenectomy followed by hereditary spherocytosis, hemolytic anemia, and TTP. Trauma is the most common reason for splenectomy overall. Previously splenectomy for staging of Hodgkin lymphoma had been a common reason for elective splenectomy.

5. Answer: e. Splenic artery aneurysms are usually found incidentally. In a woman of child bearing age, splenic aneurysm >2 cm should be addressed due to the high maternal and fetal mortality associated with rupture during gestation. For aneurysms in the proximal and middle third of the splenic artery exclusion by proximal and distal ligation may be performed. Splenic perfusion occurs through collaterals in the short gastrics. For distal aneurysms resection with splenectomy is the curative.

6. Answer: b. Patients undergoing splenectomy are at increased risk for thrombotic complications, particularly portal vein thrombosis. The etiology is a multifactorial combination of thrombocytosis, alterations in platelet function, and decreased velocity in the splenic vein remnant. Symptoms include low-grade fever, abdominal pain, leukocytosis, and thrombocytosis. CT of the abdomen with contrast is the diagnostic modality of choice, followed by prompt treatment with systemic anticoagulation. Splenomegaly >30 cm and myeloproliferative disorders are the two main risk factors for portal vein thrombosis.

7. Answer: e. Approximately 80% of accessory spleens are found in the splenic hilum. Other locations include the gastrocolic ligament, tail of the pancreas, omentum, stomach, and mesentery. Identification of an accessory spleen is critical, particularly in the setting of hematologic indications, as retained accessory spleen is associated with recurrence.

8. Answer: a. For patients undergoing elective splenectomy pneumococcal vaccination should be planned for 2 to 3 weeks prior to the operation.

Vaccination less than 14 days preoperatively is associated with an increased requirement for repeat vaccination. Patients undergoing emergent splenectomy for trauma should receive vaccine 14 days postoperatively.

9. Answer: a. A meta-analysis of 2,940 patients across 51 studies found that laparoscopic splenectomy had significantly fewer complications compared with open (15.5% vs. 26.6%). Laparoscopic operation was associated with significantly less pulmonary, infectious, and wound complications as well as shorter length of stay. Laparoscopic splenectomy was found to have an increased risk of bleeding when conversion to open procedures was included (5% vs. 3%). There was no significant difference in overall mortality between laparoscopic (0.6%) or open (1.1%) cases.

10. Answer: e. Vaccination with pneumococcal vaccine should occur 2 to 3 weeks prior or 2 weeks after splenectomy. If patient has not had *H. influenzae* type B vaccine or meningococcal vaccine this should also be administered (if older than 2 years of age). Influenza vaccine is recommended annually for asplenic patients as it increases susceptibility to bacterial infections.

CHAPTER 24

1. Answer: a. The Rome criteria for the diagnosis of constipation are as follows:

(1) Straining during at least 25% of defecations.

(2) Lumpy or hard stools in at least 25% of defecations.

(3) Sensation of incomplete evacuation for at least 25% of defecations.

(4) Sensation of anorectal obstruction/blockage for at least 25% of defecations.

(5) Manual maneuvers to facilitate at least 25% of defecations (e.g., digital evacuation, support

of the pelvic floor).

(6) Fewer than three defecations per week.

2. Answer: c. Neostigmine has potent cholinergic properties that can lead to bradyarrhythmias requiring atropine.

3. Answer: b. It is imperative to ensure the patient is adequately resuscitated or continues to receive appropriate resuscitation as the workup for a source is undertaken.

4. Answer: d. The initial treatment of sigmoid volvulus is endoscopic decompression; however, the risk of recurrence is 40% and mortality of emergent sigmoidectomy is much higher than elective sigmoidectomy, so all patients should undergo sigmoidectomy if medically able to decrease risk of mortality.

5. Answer: a. Diverticulosis is the most common cause of LGIB in the United States.

6. Answer: b. Hinchey IV diverticulitis is characterized by fecal peritonitis.

7. Answer: c. Distinguishing between ulcerative colitis and Crohn colitis can be difficult. Ulcerative colitis is characterized by mucosal disease that always begins in the rectum and extends proximally without skip lesions. Crohn colitis or disease causes full thickness disease, can involve any part of the GI tract and commonly has ÒskipÓ lesions or areas of disease interspersed between normal areas. Crohn disease also commonly causes fistulae and includes perianal disease which distinguish Crohn's from ulcerative colitis. Pyoderma gangrenosum or other systemic manifestations of IBD can be associated with either Crohn disease or ulcerative colitis.

8. Answer: b. Total abdominal colectomy with end ileostomy or total proctocolectomy are required for the treatment of ulcerative colitis as leaving any colon in situ involves risking recurrence of disease and potential for dysplasia or malignancy. Segmental colectomy has been shown to be associated with poor outcomes.

9. Answer: a. It is important to remember that the IMA is ligated in an open AAA repair and covered by the endograft in an EVAAR, so in this patient your primary concern should be ischemic colitis. To confirm this diagnosis, the patient should undergo flexible sigmoidoscopy to diagnose and characterize the extent of disease. CT scan may show colitis, but does not definitively diagnose ischemic colitis. Barium enema is contraindicated.

10. Answer: d. Hyperplastic polyps are 10 times more common than adenomatous polyps and are benign. Large polyps (>1 cm) or right side hyperplastic polyps may be a marker of increased risk for adenomatous polyps; however, so it is important to consider this when performing endoscopy.

11. Answer: c. The Kudo classification SM3 is an independent risk factor for lymph node metastasis in malignant polyps and therefore patients require surgical resection for treatment.

12. Answer: c. The current recommendations state that screening colonoscopy should be initiated 10 years prior to the earliest age relative with colon cancer, so the age of the patient's father at time of diagnosis is the most important factor to consider.

13. Answer: b. While it is true that patients with stage II disease receive only marginal benefit from receiving adjuvant therapy, this patient did not receive an adequate lymph node harvest, so there is the potential for inappropriate downstaging. Giving this information, the patient should be counseled that adjuvant chemotherapy may be beneficial given his or her inadequate staging. The current recommendations are that all patients with stage III disease receive chemotherapy.

14. Answer: a. To adequately stage rectal cancer a chest x-ray or CT is obtained to confirm the lack of lung metastasis, abdominal CT to

confirm lack of liver metastasis and pelvic MRI or transrectal ultrasound (TRUS) to assess lymph nodes and the level of tumor invasion as this will determine whether the patient is a candidate for transanal resection, surgery alone or if there is need for preoperative chemoradiation therapy.

15. Answer: b. The two most important principles of resection for rectal cancer limiting recurrence are to ensure a complete and intact total mesorectal excision and high ligation of the arterial pedicle (IMA) to perform a complete lymphadenectomy. Resection of Denonvilliers fascia is not necessary, resection of the hypogastric nerves could cause issues with continence and sexual function, and as long as a 1 cm distal margin is insured, patients can undergo low anterior resection with coloproctostomy or coloanal anastomosis.

CHAPTER 25

1. Answer: d. The patient described has a Grade IV hemorrhoid (see Table 25-1) that would be treated with excisional hemorrhoidectomy. Although all of the answer choices are complications of excisional hemorrhoidectomy, urinary retention is the most common. To attempt to prevent urinary retention, perioperative intravenous fluids should be limited.

2. Answer: b. Division of the internal sphincter using electrocautery and placement of seton encircling the external sphincter. The patient described has an intersphincteric fistula. Younger patients may tolerate transection of internal and external sphincters without loss of anal continence; however, this technique risks incontinence in older patients and women. The seton permits resolution of the infection over a minimum period of 6 weeks. A second-stage procedure can then be performed including use of fibrin glue or an anal advancement flap.

3. Answer: a. 2 cm because there is no correlation between local recurrence and the extent of distal margin when it is greater than 2 cm.

4. Answer: b. The increase in pressure during voluntary contraction (squeeze pressure) is due to the external sphincter. The internal sphincter (involuntary) is innervated by the autonomic nervous system and accounts for 80% of resting pressure. The rectal-anal inhibitory reflex is

defined by distal rectal distention causing involuntary relaxation of the internal sphincter.

5. Answer: c. Primary chemoradiation therapy. Treatment of anal squamous cell carcinoma involves chemoradiation according to the Nigro protocol: 3,000-cGy external-beam radiation, mitomycin C, and 5-fluorouracil. Surgical treatment is reserved only for locally persistent or recurrent disease.

6. Answer: e. Wide local excision is the most appropriate treatment in noninvasive disease. Paget disease is an intraepithelial adenocarcinoma,

most commonly seen in elderly patients. Any patient with suspected Paget disease should also undergo colonoscopic evaluation to ensure that the biopsy results are not due to the inferior spread of a rectal signet-ring cell cancer, as 50% of patients may have a coexisting visceral carcinoma. Treatment of noninvasive Paget disease is with wide local excision of all lesions.

7. Answer: c. Ninety percent of patients heal with medical treatment, including fiber, sitz baths, and topical nifedipine ointment. Ninety percent of anal fissures occur posteriorly, and an external skin tag or Òsentinel pileÓ may also be present, but there is not typically an internal hemorrhoid present. If surgery is required, lateral internal sphincterotomy is 95% successful with recurrence and minor incontinence occurring in fewer than 10%.

CHAPTER 26

1. Answer: c. In general, ischemic strokes are more prevalent among men than among women, although women tend to account for a higher percentage of stroke deaths. This is likely attributed to their greater longevity, compared to men. The risk of stroke increases with advancing age. Atherosclerosis increases with age, subsequently increasing the risk of myocardial infarction and ischemic stroke. For each decade after age 55 years, the risk of stroke approximately doubles. For individuals greater than 80 years of age the prevalence of stroke is 27%; for those 60 to 79 years of age, the prevalence is 13%. Population studies estimate that the incidence of stroke is nearly three times higher among African-American individuals, and nearly two times higher among Hispanic individuals. The relationship between hypertension and stroke is well established. Observational studies indicate that the risk of stroke death doubles with each 20 mm Hg incremental increase above a systolic blood pressure of 115 mm Hg. Smoking similarly increases the risk of stroke, and overall has a relative risk of 1.9 among all smokers. Interestingly, former smokers continue to have an increased risk of stroke despite cessation, and second-hand smoke exposure nearly doubles the risk of stroke.

2. Answer: d. In 1995, the Asymptomatic Carotid Atherosclerosis Study (ACAS) demonstrated that asymptomatic patients with at least 60% carotid stenosis, whose general health is suitable for elective surgery, have a significantly lower 5-year risk of ipsilateral stroke if carotid endarterectomy can be performed with less than 3% perioperative morbidity.

3. Answer: d. In 1991, the North American Symptomatic Carotid Endarterectomy Trial (NASCET)

demonstrated that symptomatic patients with at least 70% carotid stenosis have a significantly lower 2-year risk of ipsilateral stroke if carotid endarterectomy is performed. The absolute risk reduction is 17%, when compared to

patients that received medical therapy. The Asymptomatic Carotid Atherosclerosis Study (ACAS) demonstrated that the absolute risk reduction in stroke is 6% over 5 years among asymptomatic patients if carotid endarterectomy is performed. The European Carotid Study Trial (ECST) revealed that among symptomatic patients with 80% to 99% stenosis (equivalent to 60% to 99% by NASCET criteria), the 3-year absolute risk reduction for stroke is approximately 13% after carotid endarterectomy (20% among controls and 7% among CEA patients).

4. Answer: b. Cranial nerve dysfunction is the most common neurologic complication of carotid endarterectomy, and exceeds the risk of perioperative stroke. The incidence of postoperative cranial nerve dysfunction ranges from 5% to 20% in most respective series, and was 4.7% among patients that underwent carotid endarterectomy during the CREST study. The majority of these cranial injuries had no significant impact on patients, and seldom represented permanent nerve injuries. Most cranial nerve injuries are transient and resolve within a few weeks to months after carotid endarterectomy. There is considerable variability in the reported incidence of cranial nerve injury, and discrepancy as to which nerve is most commonly injured. Many series, however, suggest that the hypoglossal nerve is the most commonly injured nerve. It is important to identify this nerve during carotid exposure, particularly before clamping the internal carotid artery. The position of the hypoglossal nerve is quite variable; a safe approach is to follow the ansa cervicalis cephalad to its junction with the hypoglossal nerve, and to avoid dissecting tissue along the anterior border of the ansa until the hypoglossal is first identified. The reported incidence of hypoglossal nerve injury ranges from 4% to 17%.

5. Answer: a. During carotid exposure, the facial vein is identified coursing medially from the internal jugular vein. The vein is ligated and divided to facilitate exposure of the underlying carotid sheath and the bifurcation of the artery. Frequently, the vein has multiple branches that need to be ligated. The superior thyroid artery (rather than the vein) is identified medially at the carotid bifurcation or proximal external carotid artery. This vessel is controlled with a tie or vessel loop, and should not be ligated. The anterior jugular and subclavian veins are not often encountered during carotid exposure.

6. Answer: d. The internal carotid artery has no *extra* cranial branches. The ophthalmic artery represents the first branch of the internal carotid artery (ICA), and branches just after the ICA emerges from the cavernous sinus. This artery is of particular importance when it becomes temporarily occluded, giving rise to the syndrome of amaurosis fugax (i.e., transient monocular vision loss). The superior thyroid and lingual arteries are branches of the external carotid artery. The inferior thyroid artery is a branch of the subclavian artery, and not often encountered during carotid exposure.

7. Answer: a. Among the responses above, only the superior thyroid and lingual arteries are branches of the external carotid artery. If the surgeon were to dissect cephalad along the external carotid artery, the following arterial branches would be encountered (in order): Superior thyroid, ascending pharyngeal, lingual, facial, occipital, posterior auricular, maxillary, and superficial temporal. The superior thyroid artery is identified medially at the carotid bifurcation or proximal external carotid artery. This vessel may be controlled with a tie or vessel loop, and should be preserved during carotid exposure.

8. Answer: e. According to the North American Symptomatic Carotid Endarterectomy Trial (NASCET), a symptomatic patient with at least 70% carotid stenosis has a significantly lower 2-year risk of ipsilateral stroke after carotid endarterectomy (CEA). Long-term follow-up data from NASCET demonstrated that symptomatic patients with 50% to 69% carotid stenosis also have a significantly lower 5-year risk of ipsilateral stroke after CEA, although the absolute risk reduction is only 7%. The patient above should be offered CEA if she is a suitable candidate for elective surgery, and if the specialist or surgeon performing carotid endarterectomy has perioperative stroke and death rates below 6%.

9. Answer: b. Carotid stenting is reserved for high-risk patients, with severe cardiac disease, or adverse neck conditions that increase the complexity of carotid endarterectomy. The latter includes a history of prior ipsilateral neck surgery or neck radiation, contralateral vocal cord paralysis, or a surgically inaccessible lesion that extends caudally near the clavicle or cephalad to the C2 vertebral body. There are conflicting data in the literature demonstrating an increased risk of stroke in patients with contralateral carotid occlusion. Current observational studies do not support contralateral carotid occlusion in the absence of other criteria as an absolute indication for carotid stenting.

10. Answer: c. Bradycardia and hypotension can manifest during carotid stenting, and frequently occur during predilatation or after stent deployment. The mechanism is due to stretching of the carotid bulb, which can cause vagal stimulation, resulting in severe bradycardia and hypotension. Preventive measures consist of fluid administration in the preoperative holding area, intraprocedural atropine administration, and infusion of vasopressors. Temporary pacemakers are indicated only in the presence of pre-existing dysrhythmias. It is also critical that the interventionist or surgeon inform the anesthesia provider when balloon dilatation and stent deployment are performed.

CHAPTER 27

1. Answer: a. Of the listed risk factors, only DM has not been associated with development or enlargement of AAA.

2. Answer: 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.

3. Answer: d. Asymptomatic AAAs <5.5 cm in males and <5.0 cm in females can safely be observed.

4. Answer: d. Cutaneous ischemia following AAA repair can be observed in the setting of adequate perfusion.

5. Answer: c. Transmural necrosis of the sigmoid colon is a feared complication of AAA repair, and requires emergent resection of the involved segment to prevent perforation and peritoneal contamination.

6. Answer: c. All of the above are supplied by branches of the hypogastrics with the exception of small bowel.

7. Answer: a. Surgical repair of ruptured AAAs can be marked by massive fluid resuscitation. In some instances, abdominal compartment syndrome (ACS) may develop with the triad of distended abdomen, high peak airway pressures/elevated bladder pressures, and abdominal distention. These patients require decompression via laparotomy in the operating room or the ICU.

8. Answer: c. The aorta is composed of 3 layers; the intima (lined with endothelium), the adventia (composed of smooth muscle and ECM proteins), and the adventia (composed of loose connective tissue and fibroblasts). All three layers are involved in aneurysmal degeneration, with the majority occurring in the media.

9. Answer: c. Of the above, only an occluded left hypogastric artery does not preclude endograft placement.

10. Answer: c. Type I endoleaks are due to inadequate seal of the proximal or distal components, and are usually treated as soon as identified. Type II endoleaks are due to collateral circulation and may be watched if there is no sac expansion. Type III endoleaks are due to inadequate seal between components, including fractures. Type IV is due to graft porosity. There is no Type IV endoleak.

11. Answer: d. The EVAR1 and DREAM (EVAR 1; *Lancet.* 2004;364:843-848; DREAM; *NEJM.* 2005;352:2398-2405) studies demonstrated short-term reductions in perioperative morbidity and mortality, and duration of hospitalization. However, DREAM and UK EVAR (DREAM; *NEJM.* 2010;362:1881-1889; UK EVAR; *NEJM.* 2010;362:1863-1871) did not show a reduction in long-term mortality on long-term follow-up.

12. Answer: d. An asymptomatic TAA can be watched if less than 6 cm. Open repair is done via a left thoracotomy, often with use of aortofemoral bypass. Thoracic endovascular repair of aortic aneurysm

(TEVAR) is a viable option, and this patient should be evaluated for candidacy.

13. Answer: b. Rapid surgical repair of Type A dissections has significantly decreased the mortality of this condition. At this time there are no endovascular options for treatment of this condition.

14. Answer: a. This patient has an uncomplicated, chronic (greater than 14 days duration) Type B dissection. β-blockade forms the basis of blood pressure management in these patients. The INSTEAD-XL trial (INSTEAD-XL, *Circ Cardiovasc Interv.* 2013;6:407-416. PMID 23922146) showed improved mortality at 5 years in those patients treated with endovascular coverage of the intimal tear.

15. Answer: e. Renovascular HTN should be distinguished from other forms of HTN prior to surgical intervention. Of the above, age of onset in the middle ages (25 to 55) suggests some other underlying cause.

16. Answer: d. Nonocclusive mesenteric ischemia (NOMI) is characterized by intestinal ischemia in the absence of thromboembolic occlusion. The patients are marked by a low-output cardiac state, and imaging often reveals minimally diseased vessels. The mortality rate is high, and patients benefit from optimization of their hemodynamics rather than surgical intervention.

CHAPTER 28

1. Answer: b. Palpable pulses in the contralateral/unaffected limb with no prior history of claudication confirms absence of significant underlying atherosclerotic disease thus suggesting an embolic etiology.

2. Answer: b. Nonoperative management with risk factor modification and structured exercise is always the initial management for claudication symptoms. Lifestyle limiting claudication despite conservative management is an indication for an operative intervention. The Society of Vascular Surgeons also has practice guidelines for atherosclerotic occlusive disease of the lower extremities (http://www. vascularweb.org).

3. Answer: c. Acute lower extremity compartment syndrome should always be suspected and aggressively managed with adequate 4-compartment fasciotomies when duration of ischemia exceeds 6 hours.

4. Answer: a. Statins are helpful in reducing adverse cardiovascular events in patients with peripheral atherosclerosis in whom coronary atherosclerosis often coexists (*Lancet.* 2002; 360(9326)). Small studies have also shown to have a positive effect of statins on pain free walking distance in patients with intermittent claudication (*Am J Med.* 2003;114(5), *Circulation.* 2003;108(12)).

5. Answer: c. Traumatic joint dislocation can cause mechanical compression or a temporary spasm of an adjacent artery which is completely

reversible by reduction of the involved joint. This should always be attempted prior to any

operative intervention for an abnormal distal vascular examination.

CHAPTER 29

1. Answer: d. The diagnosis of DVT based on physical findings is inaccurate, rather a high index of suspicion along with risk factor identification results in appropriate referral for venous duplex. In fact, 80% of patients with a DVT have at least one identifiable risk factor. IVF filters may be indicated in certain patients with contraindications to anticoagulation but are certainly not indicated in all patients with a DVT. Finally, DVT is a common complication of orthopedic surgery.

2. Answer: a. This patient has C_5 E_s A_s P_r classification of her lower extremity venous disease. Her ulcer is healed, which is C_5. It is secondary to venous reflux diagnosed on the duplex. Her GSV was involved which is a superficial lower extremity vein. Finally, the pathology as stated in the body of the text is reflux, no mention was made of obstructive pathology.

3. Answer: c. LDUH reduces the risk of VTE by 50% to 70% *(N Engl J Med.* 1988;318:18) and does not require laboratory monitoring.

4. Answer: d. Current practice as mandated by the CHEST Guidelines, 9th Edition recommend that proximal DVT provoked by surgery should be treated with a lovenox bridge to Coumadin therapy for a 3-month duration.

5. Answer: e. No surgical procedure is without complications and IVC filter placement is no exception. While many view this relatively simple procedure as benign, some of its complications include the very thing it is trying to prevent. All of the listed complications are possible after IVC filter placement.

6. Answer: c. Current recommendations for an asymptomatic below-theknee DVT are for follow-up ultrasound to detect for evolution or resolution of the clot. If the clot has not propagated within the 2-week period it is unlikely to do so later and no treatment is recommended.

CHAPTER 30

1. Answer: d. 200 to 500 mL/minute is necessary for adequate hemodialysis.

2. Answer: c. Pseudointimal hyperplasia in a graft or neointimal hyperplasia in a native fistula are the most common causes of dysfunction. Hemodynamically significant stenoses can lead to thrombosis. Evaluation with Duplex ultrasound and fistulogram aid in diagnosis, and intervention with angioplasty or surgery may be required.

3. Answer: a. Anticipation of the need for dialysis allows for adequate surgical planning. When the serum creatinine reaches 4mg/dL or the creatinine clearance is less than 30 mL/minute, dialysis access planning is indicated. These guidelines should be considered in light of the rate of worsening renal function as well.

4. Answer: d. Catheter-associated peritonitis is a serious complication of PD requiring removal of the catheter if antibiotic therapy is unsuccessful.

5. Answer: b. Initial empiric antibiotics for catheter-associated peritonitis should cover Gram-positive (vancomycin or first-generation cephalosporin) and Gram-negative (third-generation cephalosporin or aminoglycoside) bacteria.

6. Answer: c. Juxta-anastomotic stenosis occurs secondary to shear stress on the vessel walls adjacent to the anastomosis (between 2 and 4 cm) proximal on the arterial inflow side of the anastomosis and 2 to 4 cm distal on the venous outflow side of the anastomosis.

7. Answer: d. For ease of access, AV fistulae may require superficialization if the conduit is greater than 5 mm deep following maturation.

8. Answer: b. Autogenous refers to native tissue. Synthetic grafts are nonautogenous. The graft serves as the conduit or needle access site (NAS).

9. Answer: b. Banding of the access decreases the diameter of the anastomosis, thereby reducing the flow and decreasing the severity of steal.

10. Answer: b. The risk of infection is much greater in catheters than permanent accesses like AVF and AVG. Tunneled dialysis catheters have a reduced risk of infection compared to nontunneled dialysis catheters because of fibroblast ingrowth forming a microbial barrier around the catheter within the pocket.

CHAPTER 31

1. Answer: e. The evaluation of potential living donors includes assessment of their overall health, co-morbid conditions and psychosocial influences. Compatibility with their intended recipient is determined though ABO blood typing and HLA histocompatibility. Donors who are not compatible with their intended recipient may still donate through paired exchange and ABO-incompatible protocols.

2. Answer: c. ÒHepatic artery thrombosis in the early posttransplantation period may lead to fever, hemodynamic instability, and rapid deterioration, 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 ductsÕ main vascular supply. Acute thrombosis may be treated

by attempted thrombectomy; however, this is usually unsuccessful and retransplantation is needed.Ó The gold standard treatment is to relist this patient for a new liver allograft.

3. Answer: b. *Transplantation for hepatic malignancy.* Cirrhosis is a risk factor for hepatocellular carcinoma (HCC). Given that most patients who develop HCC die from their underlying cirrhosis rather than from metastatic disease, it was reasoned that transplantation may be a potentially curative approach to the primary tumor as well as the underlying pathology. The **Milan Criteria** are outcome driven and establish guidelines for considering OLT in patients who present with

early stage I or II HCC and underlying cirrhosis. Given the concern for HCC progression while awaiting transplantation, candidates receive MELD exception points beyond what is calculated from their cirrhosis.

4. Answer: a. Venous hypertension can be caused by the presence or development of outflow vein obstruction. It manifests as swelling, skin discoloration, and hyperpigmentation in the access limb. Management consists of fistulogram and correction of stenosis through either balloon dilatation and/or stent placement.

5. Answer: b. Most patients are type I diabetics with concomitant nephropathy who are evaluated for a pancreas transplant in conjunction with kidney transplantation. Ninety-five percent of all pancreas transplants are performed in conjunction with a kidney transplant. Long-term survival of an SPK recipient is similar to that of a diabetic with ESRD receiving a living donor kidney transplant. Whole organ pancreas transplantation represents the only therapeutic option for long-term insulin independence.

6. Answer: d. 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 to 48 hours postoperatively. Persistent transaminitis should prompt a liver ultrasound to assess vessel patency and flow.

7. Answer: b. 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. A renal scan demonstrates radioisotope outside the urinary tract. Urine leaks are treated by placing a bladder catheter to reduce intravesical pressure and subsequent surgical exploration.

8. Answer: e. Approved indications for intestinal transplant include the life-threatening complications associated with PN therapy.

9. Answer: d. Panel reactive antibodies (PRA) help to predict the likelihood of 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 with which the patient's sera react is the PRA. Patients who have been exposed to other HLAs via blood transfusion, pregnancy, or prior transplantation will have higher PRAs.

CHAPTER 32

1. Answer: d. Initial blood transfusion should be in the volume of 10 mL/kg. Her initial fluid boluses should also have been in a volume of 10 mL/kg, with up to 20 mL/kg up to two times acceptable.

2. Answer: b. Type II choledochal cyst is an isolated cyst arising from the common bile duct and is typically repaired via hepaticojejunostomy.

3. Answer: c. The injury described on imaging represents a Grade II splenic injury. Per the recommendations of the Liver/Spleen Trauma Study Group of the American Pediatric Surgical Association (Stylianos et al., 2000), the patient should be hospitalized for 3 days and be restricted to 4 weeks of light activity. Answer choices B, D, and E represent appropriate management choices for Grade I, III, and IV splenic injuries in the hemodynamically stable patient. Choice A is not part of the guidelines.

4. Answer: c. Plain abdominal films can aid in the diagnosis of tracheoesophageal fistula. Coiling of the orogastric tube in the upper chest is sufficient for a presumptive diagnosis of esophageal atresia. Plain abdominal film could aid in the determination of whether or not there is an associated tracheoesophageal fistula. The presence of air within the bowel would suggest that there is a communication between the trachea and the distal esophagus. Other additional imaging modalities are not essential in the absence of other examination findings. Plain radiographs are sufficient for operative planning.

5. Answer: c. Proximal esophageal atresia and distal tracheoesophageal fistula are the most common type of tracheoesophageal malformation, accounting for 80% to 90% of cases.

6. Answer: b. Gastroschisis is believed to be the result of an intrauterine vascular insult that creates a defect in the abdominal wall through which bowel herniates. In contrast to omphalocele (described by choices A, C, and D), gastroschisis usually involves a smaller defect, has a much less frequent association with anomalies, and is not covered by a peritoneal sac.

7. Answer: c. Clinical examination and imaging are concerning for a delayed presentation of a congenital diaphragmatic hernia. Emergent thoracotomy is not advisable in this situation as the patient is not stable. Additionally, bedside echocardiogram is insufficient therapy for a hypoxic patient but likely should be done eventually for cyanotic workup and preoperative evaluations. Finally, ECMO is a

useful, but it is a last-step effort only after the patient's clinical criteria are determined to meet institutional ECMO standards, which should always include attempts to stabilize on conventional or other ventilators as well as ruling out irreversible conditions such as irreparable cardiac abnormalities. Potential for volvulus of herniated intestines should be investigated but only after the patient's acute respiratory condition is improved.

8. Answer: d. Meissner plexus resides in the submucosal plane, while the Auerbach plexus is between the longitudinal and circular muscular layers.

9. Answer: c. This description is concerning for a mucosal perforation as a result of pyloromyotomy. Although an incomplete or stenotic myotomy is also a postoperative concern, these patients would not demonstrate abdominal distention to the degree that perforated patients would. Mucosal perforation can be managed safely in the stable pediatric patient by bowel rest and decompression.

10. Answer: d. Pexying the cecum does not have any benefit in a Ladd procedure. Appendectomy is performed to eliminate any future appendicitis from arising in a nonanatomical position.

CHAPTER 33

1. Answer: b. In a patient with a low cardiac index after open heart surgery, it is important to rule out hemorrhage and tamponade. Transfusion would not be the correct answer since there is no evidence of hemorrhage. Other causes of low cardiac index after open heart surgery are hypovolemia and LV dysfunction. Since the CVP is elevated in this scenario, the preload is adequate, and one can increase the cardiac index by increasing the contractility (i.e., adding Dobutamine) or reducing afterload (i.e., adding a vasodilator such as nitroglycerin). An intra-aortic balloon pump is reserved for patients who are in cardiogenic shock refractory to inotropes or volume.

2. Answer: e. Since the patient is hemodynamically stable, there is no indication for reexploration in the ICU or operating room. Indiscriminately transfusing platelets or FFP in patients who are hemodynamically stable is also not recommended. Patients who have just returned from the operating room may be cold and have inadequate reversal of their heparin. Therefore, warming the patient and giving protamine is the best first step in management of this patient.

3. Answer: b. The indications for IABP are preoperative low-cardiac-output states, preoperative unstable angina refractory to medical therapy, intraoperative weaning from cardiopulmonary bypass after inotropic agents alone are maximized, and postoperative low cardiac output states. In the case of aortic insufficiency, a balloon pump is contraindicated because the balloon inflates during diastole, which increases the

regurgitant volume, and thereby exacerbating the deleterious effects of the aortic insufficiency.

4. Answer: d. Current guidelines recommend that CABG be performed to improve survival in patients with significant (³50% stenosis) left main coronary artery stenosis, in patients with significant (³70% stenosis) in three major coronary arteries or in the proximal LAD plus one other major artery, in patients with significant stenosis in two major coronary arteries with severe or extensive myocardial ischemia or target vessels supplying a large area of viable myocardium, in patients with LV dysfunction (ejection fraction 35% to 50%) and significant stenosis when viable myocardium is present in the area of intended revascularization, in patients with significant stenosis in the proximal LAD and evidence of extensive ischemia, in patients with complex three-vessel CAD with or without involvement of the LAD who are good candidates for surgery, and in in patients with multivessel CAD with diabetes mellitus. Contraindications include porcelain aorta, no adequate conduits or targets, and unacceptably high perioperative risk.

5. Answer: d. Relative indications for cardiac transplant include refractory cardiogenic shock,

instability in fluid balance or renal function despite optimal medical therapy, severe persistent angina not amenable to revascularization, markedly reduced exercise capacity (peak VO_2 <10 to 14 mL/kg/minute), and recurrent refractory ventricular arrhythmias. Relative contraindications to transplantation include age older than 65 years, irreversible pulmonary hypertension, active infection or malignancy, recent pulmonary embolus, excessive comorbidity (renal dysfunction, hepatic dysfunction, systemic disease such as amyloidosis, significant peripheral vascular disease, active peptic ulcer disease, uncontrolled diabetes mellitus, morbid obesity), mental illness, active substance abuse, inadequate social support, or psychosocial instability.

CHAPTER 34

1. Answer: b. The patient has no respiratory distress, has a job that is not high risk, is a first time pneumothorax, and is small.

2. Answer: b. This patient is developing a tension pneumothorax, likely from a ruptured bleb in the setting of positive pressure ventilation. The immediate next step is needle decompression, followed by further evaluation and chest tube placement.

3. Answer: a. This patient has a transudative effusion, so his pleural fluid should have <0.5 pleural protein to serum protein, <0.6 pleural LDH to serum LDH, and pleural LDH <2/3 upper limit of normal (upper limit is 200 to 300).

4. Answer: d. This anterior mediastinal mass is likely a nonseminomatous germ cell tumor, with elevation of both AFP and β-HCG. Treatment

is with platinum-based chemotherapy followed by resection of the remaining masses.

5. Answer: b. Given the size, it is concerning for malignancy. Thus further workup with PET scan is indicated to help determine malignant nature and any regional or distant spread. Navigational bronchoscopy can be challenging to reach and biopsy peripheral lesions. A biopsy in this scenario may be obtained by a CT-guided biopsy or a wedge followed by a lobectomy if cancerous, depending on institutional preferences.

6. Answer: a. The patient needs a mediastinoscopy or EBUS to confirm N2 disease. If the N2 level nodes are positive, then the patient has stage IIIA cancer, which should be managed initially with neoadjuvant therapy.

7. Answer: c. See Figure 34-7 for preoperative lung function assessment.

CHAPTER 35

1. Answer: c. Radiation therapy is contraindicated in patients who have received past chest wall radiation (A) or in patients in the second or third trimester of pregnancy (D). Radiation therapy is of minimal utility in the management of a small focus of DCIS in an elderly woman (B). However, a woman in the late stages of pregnancy with a small tumor and possible lymph node

involvement could receive and would benefit from *postpartum* radiation therapy both as part of breast conservation treatment (BCT) and axillary treatment without undue delay.

2. Answer: a. Radiation therapy is NOT associated with improved overall or breast-cancer-specific survival and increases the risk of lymphedema after ALND.

3. Answer: d.

4. Answer: b. First-line treatment for likely lactation mastitis is antibiotics and increased frequency of breastfeeding. If her symptoms fail to resolve, inflammatory breast cancer must be excluded.

5. Answer: a. BRCA2 mutations are associated with approximately 4% to 6% of male breast cancers. Eighty-five percent of malignancies are infiltrating ductal carcinoma. MRM was traditionally the surgical procedure of choice; however, SLNB has been shown to be effective in men. Thus, total (simple) mastectomy with SLNB is a valid option in men. Adjuvant hormonal, chemotherapy, and radiation treatment criteria are the same as in women. Overall survival *per stage* is comparable to that observed in women, although men tend to present at later stages.

6. Answer: c.

7. Answer: c. The patient likely has a combination of senescent and drug-related gynecomastia. Renal failure may also be contributing to the problem. The treating physician should contact his PCP to discuss potential changes to his medications.

8. Answer: b. Follow-up for BIRADS 3 lesions is 4 to 6 months with repeat imaging.

9. Answer: d.

10. Answer: c. Pertuzumab was approved for use in the United States by the Food and Drug Administration (FDA) in 2011. When administered with trastuzumab (another anti-Her2 monoclonal antibody) and docetaxel (a taxane), pertuzumab has been associated with progression-free survival in patients with metastatic HER2+ breast cancer.

CHAPTER 36

1. Answer: e. Melanomas less than 0.75 mm regardless of histology do not require SLNB (choice A), nor does melanoma in situ (choice B). Regional spread (choice C) warrants lymph node dissection while SLNB has no role in metastatic disease (choice D). See section Melanoma > Treatment > Sentinel Lymph Node Biopsy or NCCN Clinical Practice Guidelines in Oncology Ð Melanoma, Version 1.2015.

2. Answer: d. Mohs microsurgery may be appropriate, but not until after diagnosis (choice A). Excisional biopsy is not indicated on cosmetically sensitive areas such as the face (choice B). FNA is reserved for soft-tissue masses, not cutaneous lesions (choice C). Shave biopsy is not the preferred method since depth of the lesion is difficult to assess and leads to an inferior cosmetic

result (choice E). See section Skin Lesions > Biopsy.

3. Answer: e. This patient has a chronic nonhealing wound that is concerning for the development of cancerÑlikely a Marjolin ulcer. There are no signs or symptoms of infection so topical antibiotics are not indicated (choice A). The patient's diabetes is well controlled and his smoking is minimal, so they are likely not contributing significantly to his failure to heal (choices B and C). Ankle-brachial indices are likely to be unhelpful in this young patient with palpable pulses (choice D). See section Other Malignant Skin Lesions > Squamous Cell Carcinoma.

4. Answer: d. 1 cm margins are insufficient for melanomas >1 mm in thickness (choices A and C). SLNB is indicated for all lesions >1 mm in thickness in the absence of metastases or clinical adenopathy, hence choice B is incorrect. Axillary lymph node dissection is not indicated at this juncture as he does not have documented adenopathy (choice E). See Therapeutic Decision Algorithm for Cutaneous

Melanoma, or NCCN Clinical Practice Guidelines in Oncology Ð Melanoma, Version 1.2015.

5. Answer: d. Observation (choice A) is not appropriate as new nevi after 40 years of age must be considered potentially malignant until proven otherwise. Shave biopsy is suboptimal due to difficulty in assessing depth (choice B). Margins should be dictated by pathology instead of arbitrarily chosen (choice C). A diagnosis is needed before topical therapy can be instituted (choice E). See section Benign Lesions > Nevi.

6. Answer: a. This lesion is small and has benign characteristics including mobility, soft texture, and perhaps most importantly, the lesion has not grown over time. This presentation is most consistent with lipoma. Given these characteristics and the fact that the patient is asymptomatic, this can be safely observed. See section Benign Lesions > Lipoma.

7. Answer: c. Radiation therapy may be indicated for locoregional control in patients with regional disease. It is not indicated for primary melanoma alone, micrometastatic disease limited to a single sentinel node, or widely metastatic disease (choices A, B, D, E).

8. Answer: b. The patient's presentation is concerning for soft-tissue sarcoma with likely neurovascular involvement. MRI is the preferred imaging modality for primary disease while CT chest is indicated for staging. A diagnosis must be made prior to intervention (choices A and D). While biopsy will be needed, the preferred method is core tissue biopsy, hence choices C and E are incorrect.

9. Answer: a. This patient has adenopathy of unknown origin, most consistent with occult or regressed melanoma given normal mammograms. PET scan will not reveal a diagnosis which is what this patient needs (choice B). Core needle biopsy is warranted to obtain a diagnosis. Observation is not appropriate in a patient with likely malignancy (choice C). The presentation is not consistent with an infectious etiology (choice D) and a diagnosis is needed before treatment can be considered (choice E).

10. Answer: e. Grade has been found to be the most important prognostic factor in soft-tissue sarcoma.

11. Answer: c. Patients with soft-tissue sarcoma should be staged before undergoing therapy (choices A, B, D, E).

12. Answer: d. En bloc resection of a soft-tissue sarcoma with involved organs greatly enhances survival. This patient has no history of renal failure and has a normal creatinine, so en bloc resection is indicated. Chemotherapy is relatively ineffective and gross positive margins worsen survival, hence answers A, B, C and E are incorrect.

13. Answer: e. Lymphovascular invasion has been shown to portend lower survival than any other prognostic factor in Merkel cell carcinoma.

14. Answer: b. Chest CT is not indicated as basal cell carcinomas are locally invasive but do not metastasize (choice A). Mohs surgery is costly and time-consuming and reserved for areas where tissue conservation is important (choice C). Since basal cell carcinomas do not metastasize, lymph node biopsies are not indicated (choice D). Radiation therapy can be used if lesions are unresectable or treatment is palliative, however this lesion is amenable to surgical therapy and should be excised.

15. Answer: a. Squamous cell carcinomas and solitary metastases should be resected when possible due to a high chance of cure. Radiation is usually reserved for patients who are not candidates for surgery (choices B and C). Likewise, systemic and topical therapies are not preferred in this scenario (choices D and E.)

CHAPTER 37

1. Answer: d. Uncontrolled coagulopathy is an absolute contraindication to laparoscopic surgery.

2. Answer: c. Pneumoperitoneum should be maintained at 12 to 15 mm Hg.

3. Answer: a. Major intravascular injury, though rare, is a devastating complication of laparoscopic surgery. The highest risk is during obtaining intra-abdominal access.

4. Answer: b. Robot-assisted laparoscopic surgery is associated with improved ergonomics compared to laparoscopic surgery alone.

5. Answer: e. Pneumoperitoneum is associated with decreased GFR; however, there is no evidence of kidney injury as result of this transient physiologic change.

CHAPTER 38

1. Answer: d. Inguinal hernia is a common surgical problem. The ratio of the incidence of inguinal hernia in men to women is 10:1. Bilateral inguinal hernia is common, present in nearly one quarter of patients. Indirect inguinal hernias are more common, but recurrent hernias are

more likely to be direct. Several methods of inguinal hernia repair have been described, with the mesh-based Lichtenstein repair commonly reported to have lower recurrence rate than tissue-based repair techniques.

2. Answer: d. Small bowel obstruction is a common surgical problem. Adhesive small bowel obstruction is the most common etiology, however not in a patient without previous surgery. The presence of pain

with medial thigh rotation is known as the Howship-Romberg sign and is consistent with an obturator hernia. Femoral, inguinal, and Spigelian hernia etiology for small bowel obstruction would include palpable masses in the appropriate locations: below the inguinal ligament, along the inguinal canal, or in the semilunar line, respectively.

3. Answer: e. The patient presented here has signs and symptoms of a strangulated inguinal hernia with fever, tachycardia, tenderness, and skin changes. Additionally, imaging is not indicated as it will delay but not change management. Admission and NG decompression is not indicated in the setting of potential strangulation. Open hernia repair is preferred to laparoscopic as it will allow tissue-based hernia repair and possible bowel resection in the setting of potential ischemic or necrotic bowel.

4. Answer: c. A number of surgical approaches are available for incisional hernia repair. For a young and active patient, reapproximation of the midline with retrorectus placement of synthetic mesh will offer the lowest risk of recurrence with the most favorable functional outcome. Laparoscopic placement of a barrier-coated mesh is also a reasonable option, but it does not offer the benefits of midline approximation or of a functional abdominal wall, important considerations in a physically active individual. Intra-abdominal placement of noncoated mesh is contraindicated due to adhesive complications. Biologic mesh is not indicated in this clean surgical field.

5. Answer: d. The patient's CHF and history of open prostatectomy both favor open over laparoscopic inguinal hernia repair. Open repair with mesh is favored over open primary repair due to a lower recurrence rate. Watchful waiting has been advocated for asymptomatic hernias; however, this patient is symptomatic on presentation and would benefit from repair.

CHAPTER 39

1. Answer: c. MEN-2B patients should undergo prophylactic thyroidectomy within the first year of life. MTC is particularly aggressive in these patients and may be present at birth. Surgery should not be delayed if these patients have a normal calcitonin level as medullary thyroid carcinoma has been found in patients with a normal calcitonin level.

2. Answer: a. A cortisol level less than 20 µg/dL on corticotropin stimulation testing is consistent with adrenal insufficiency. Acute adrenal insufficiency may manifest as nausea, emesis, fever, hypotension, lethargy, hypoglycemia, azotemia, hyponatremia, and hyperkalemia. Immediate treatment with administration of intravenous steroids and hydration with intravenous fluids is

critical and should be initiated prior to laboratory confirmation of adrenal insufficiency.

3. Answer: c. A healthy, young patient with a nonfunctional adrenal mass between 4 and 6 cm should undergo laparoscopic unilateral adrenalectomy. Plasma aldosterone testing is not necessary if the patient is not hypertensive. MRI to further evaluate the mass may be warranted in a patient with a mass less than 4 cm or in a patient who is a poor operative candidate secondary to advanced age, medical comorbidities, or with a strong desire to avoid surgery. In such patients, observation with repeat imaging would be a reasonable option if the mass appears benign on MRI. Routine Ret proto-oncogene testing should not be performed in patient with a nonfunctional adrenal mass. Young patients with pheochromocytomas or patients with extra-adrenal pheochromocytomas should undergo genetic testing to detect mutations in the Ret gene (multiple endocrine neoplasia), VHL gene (von Hippel-Lindau disease), and succinate dehydrogenase genes (Cowden syndrome).

4. Answer: d. Persistent hypertension on a multidrug antihypertensive regimen with elevations in plasma aldosterone levels is consistent with an aldosterone-producing adenoma. Given CT and MRI findings of bilateral adrenal masses, it is unclear from imaging as to the location of the aldosterone-producing adenoma. Bilateral adrenal vein sampling will help to isolate the side of the aldosterone-producing adenoma and direct operative management. An aldosterone-producing adenoma should be surgically resected, but avoiding bilateral adrenalectomy, if possible, is ideal given the morbidity of permanent adrenal insufficiency. Spironolactone is used in the preoperative period for control of hypertension and to correct hypokalemia. Long-term medical management alone is indicated for bilateral adrenal hyperplasia, as surgical resection is rarely curative.

5. Answer: e. Macroscopic fat on CT scan is the characteristic finding of myelolipoma. Myelolipomas are benign lesions that do not require repeat imaging. If identified clearly as a myelolipoma on imaging, resection of masses greater than 6 cm is not necessary as there is no malignant potential. Surgery is reserved for symptomatic masses resulting in abdominal pain or local obstructive effects. Large masses may be resected to prevent spontaneous retroperitoneal hemorrhage.

CHAPTER 40

1. Answer: b. Careful history and physical are critical in the evaluation of a thyroid mass or nodule; subsequently, noninvasive laboratory testing should include a serum TSH level to ascertain the patient's thyroid status. Patients with hyperthyroidism generally do not require fine-needle aspiration, while those who are euthyroid or hypothyroid (elevated TSH) should undergo diagnostic ultrasound with fine-needle aspiration if a nodule is seen.

2. Answer: c. Parathyroid carcinoma is more likely to present with hypercalcemia greater than expected for other causes of hyperparathyroidism.

Serum calcium levels tend to be 3 to 4 mg/dL higher than the upper limit of normal versus 1 to 2 mg/dL as seen in other etiologies, and patients with parathyroid carcinoma are also more likely to be symptomatic from hypercalcemia. Arrhythmias are common, and presenting symptoms may include fatigue, weakness, nausea, polyuria, polydipsia, weight loss, anorexia, and vomiting.

3. Answer: d. Levothyroxine is used for treatment of hypothyroidism, both from natural disease and following total thyroidectomy, and for the suppression of TSH. Monitoring of the levothyroxine dosage should be done by checking the serum TSH level, which reflects the effect of thyroid hormone on the peripheral tissues and is the most sensitive test of the adequacy of therapy. RAI uptake measures percent uptake of administered iodine-131 and approximates the affinity of thyroid tissue for iodine. Total T_4 levels are affected by changes in hormone binding and do not directly reflect the free T_4 fraction. Thyroglobulin is synthesized by both normal and malignant cells and is used as a tumor marker in patients who have had a thyroidectomy for thyroid cancer. T_3 (triiodothyronine) resin uptake measures binding of radioactive-labeled T_3 to thyroid-binding globulin (TBG) and is an indirect measurement of TBG. Neither thyroglobulin nor T_3 uptake accurately measures the adequacy of levothyroxine therapy. Direct measurements of serum TSH or free T_4 levels are the most accurate methods for evaluating thyroid status.

4. Answer: e. Multiple biochemical abnormalities may be present concurrently with hypercalcemia and require correction prior to surgical intervention. Metabolic acidosis and hypophosphatemia are more commonly associated with primary hyperparathyroidism due to increased urinary excretion of bicarbonate and phosphate. Patients are also more likely to present with hyperchloremia due to the increased urinary excretion of bicarbonate. Hypomagnesemia can occur in 5% to 10% of patients with primary hyperparathyroidism. Serum alkaline phosphatase levels are often elevated in patients with bone disease from hyperparathyroidism due to an increase in osteoclastic bone resorption.

5. Answer: c. Abnormalities of descent may lead to ectopic gland positions. The superior parathyroids are more likely than the inferior glands to be found in the tracheoesophageal groove, posterior mediastinum, or in an intrathyroidal position. The inferior parathyroids may often be found near the thyrothymic ligament and are not uncommonly found embedded in the thymus itself due to the embryologic association of these structures.

6. Answer: d. Though thyroid nodules are very common, thyroid cancer is rare, and certain patient features may prompt the physician to have additional concern for malignancy. Nodules at the extremes of age, in patients with a history of radiation exposure, and in patients with family history concerning for possible syndromic association are

more likely to be malignant than those that occur in the third through seventh decade without other warning signs in the patient history. A solitary nodule is also more likely to be malignant than multinodular disease, and malignancy is more common in men than in women after age 60.

7. Answer: e. Hashimoto thyroiditis, a chronic autoimmune disorder, is the most common cause of hypothyroidism in patients without history of thyroid surgery or RAI therapy and occurs more frequently in women than men. Symptoms may include a large goiter, cold intolerance, weight gain, constipation, edema, menorrhagia, or muscle weakness. Serum TSH levels will be elevated while free T_4 levels will be decreased. Papillary thyroid carcinoma has not been associated with the development of hypothyroidism, and thyroid adenoma and self-administration of thyroid hormone are more likely to be associated with hyperthyroidism.

8. Answer: e. Representing the majority of all thyroid cancers, papillary thyroid cancer generally has an excellent prognosis and good longterm survival with timely surgical intervention. However, some tumors may demonstrate more aggressive features, including poorly differentiated histology, size greater than 4 cm, local invasion, and bulky nodal disease. Male gender and age greater than 45 are also poor prognostic factors.

9. Answer: a. Medullary thyroid carcinoma may occur sporadically or as part of multiple endocrine neoplasia (MEN) syndromes type 2A or 2B. Given the strong family history, the patient should undergo workup and preoperative evaluation for total thyroidectomy. The patient should be tested for germline mutations in the RET proto-oncogene, and the family should be referred for genetic counseling. Biochemical testing involves checking the plasma calcitonin level, which can be measured either in a basal state or in a stimulated state. Plasma CEA levels may also be elevated in patients with advanced or metastatic medullary thyroid carcinoma, but it is not as sensitive for detecting early disease as plasma calcitonin. Plasma catecholamine and serum calcium levels should also be checked in the biochemical evaluation of these patients, as these may be associated with pheochromocytomas or hyperparathyroidism, but these tests do not have any relationship with the presence of medullary thyroid carcinoma.

10. Answer: d. Graves disease is an autoimmune disorder and the most common cause of hyperthyroidism. It often demonstrates a genetic predisposition and a higher incidence among women, and the disease is caused by constitutive activation of the TSH receptor due to stimulating immunoglobulins. Increased thyroid hormone production occurs, with resultant decreased TSH production due to negative feedback. Common signs and symptoms include weight loss despite normal or increased appetite, heat intolerance, anxiety, irritability,

palpitations, heart arrhythmias, tremor, hyperreflexia, and muscle weakness. Pathognomonic features include exophthalmos and pretibial myxedema.

CHAPTER 41

1. Answer: c. The mass described here is a thyroglossal duct cyst which is due to lack of obliteration of the thyroglossal duct after descent of the thyroid from the base of the tongue. The mass moves with tongue movement due to its attachment to the tongue. It is important to perform an US of the neck to ensure that the mass is not the patient's sole thyroid tissue, as surgical removal would cause iatrogenic hypothyroidism.

2. Answer: e. The situation described here is bilateral vocal cord paralysis following iatrogenic injury from total thyroidectomy. Speech therapy, vocal cord injections, and thyroplasty are appropriate for unilateral injuries. Tracheostomy will protect the airway.

3. Answer: d. Core or excisional biopsy is needed for immunophenotyping and flow cytometry to direct treatment.

4. Answer: c. Parotid tumors are most commonly benign, and the most common benign tumor of the parotid gland is pleomorphic adenoma. The indicated treatment is removal of the mass with a cuff of normal tissue, which can usually be accomplished with a superficial parotidectomy.

5. Answer: e. This patient has a symptomatic carotid body tumor, therefore watching and waiting (which is appropriate for asymptomatic slow growing tumors) is not a good option. Biopsy is not recommended for these vascular tumors. Angiography with embolization is recommended before surgery.

6. Answer: d. The next step in management is fine-needle aspiration biopsy. Open/excisional biopsy of cervical metastatic SCC is not recommended since it is associated with increased risk of distant metastases and late locoregional recurrence.

CHAPTER 42

1. Answer: c. A full-thickness skin graft includes both the epidermis and dermis. As such, the elastin fibers in the dermis recoil, resulting in up to 40% contracture immediately following harvest from the donor site. However, there is minimal contracture once the graft is inset. Split-thickness skin grafts, regardless of meshing, do not contract greatly initially, but contract up to 40% during the healing process, likely due to the action of myofibroblasts.

2. Answer: d. Although this patient's presentation is concerning for a retrobulbar hematoma, the first step in management with any trauma

patient is completion of the primary and secondary surveys. There is a high incidence of c-spine injury and intracranial injuries in facial trauma patients.

3. Answer: a. This patient presents with a classic Òfight bite.Ó These wounds have a high incidence of tendon, cartilage, and bony injury. In this case, the report of pain with extension is suspicious for a partial tendon laceration. However, the most feared outcome is rampant infection secondary to human oral flora in the wound. The presentation is often innocuous, but 75% of these wounds have deeper injuries to the cartilage, bone, or tendon. These wounds should be explored and washed out in the operating room and often require multiple wash-outs. Intravenous antibiotics are also an important part of initial management.

4. Answer: b. This patient's presentation and examination is most concerning for postoperative hemorrhage. Abdominoplasty is associated with the highest rate of venous thromboembolism

among elective cosmetic surgeries and most patients are discharged on enoxaparin or other anticoagulant therapies, increasing their risk for postoperative hemorrhage. Drain output can be misleading because clots in the tube will reduce output, although clots in the drain bulb should raise suspicion for sanguineous output. There is a large volume of dead space beneath the abdominoplasty flaps, allowing for a large volume of hemorrhage. Although the index of suspicion for pulmonary embolism following abdominoplasty should always be high, this presentation is more consistent with hemorrhage.

5. Answer: a. Heavily contaminated soft tissue wounds often need to be washed out and debrided multiple times before the infection risk is sufficiently reduced to allow for definitive coverage. Heavy contamination is also a contraindication to negative pressure wound therapy. In this case, a wet to dry dressing will help clean the wound until it is sufficiently clean for coverage.

6. Answer: d. The described defect requires both fascial and cutaneous components in the setting of a previously irradiated abdomen. Resection of the rectus abdominis precludes successful component separation. The pedicled anterolateral thigh flap provides the necessary coverage without the need for position changes, microsurgery, and skin grafting, as would be needed with the latissimus free flap.

7. Answer: d. The critical component of this scenario is recognizing that the left internal mammary artery is the vascular pedicle for options (b) and (c). A skin graft would not take over exposed sternum and a free flap is rarely necessary in the chest. Other reasonable options would be a right pedicled rectus flap (right internal mammary artery), pectoralis advancement flap (based on thoracoacromial pedicle), or right turn-over pectoralis flap (right internal mammary artery).

8. Answer: b. Nerve palsies secondary to gunshot wounds, in the absence of vascular injury or observation of the nerve in the wound, should be treated as closed injuries. There are no useful findings on electromyography and nerve conduction studies in the acute period, so it is best to wait until 6 weeks to obtain baseline studies and follow-up in 3 months. If there is functional recovery, continued observation and hand therapy is warranted. If there is no functional recovery, repeat studies are obtained to determine if operative intervention is indicated.

9. Answer: a. Osteomyelitis must be treated prior to definitive coverage; a bone biopsy showing more than 10 organisms per gram of tissue is predictive of flap failure. As long as surrounding skin is clean and the wound can be adequately protected, fecal incontinence is not a contraindication. Negative pressure wound therapy can be adequate treatment for stage I/II ulcers, but is unlikely to result in full healing of a stage IV ulcer. Secondary to a chronic inflammatory state, serum iron is low in most patients with pressure sores and cannot be reversed with supplementation. Untreated spasticity would be a contraindication for flap coverage; baclofen is a standard therapy.

10. Answer: e. Fat grafting can result in fat necrosis-related changes on mammography. The

most common changes are lipid cysts and scattered microcalcifications. In the case of augmentation mammoplasty, fat grafting is most commonly to the upper pole. A finding of bilateral oil cysts is consistent with fat grafting to bilateral upper poles. However, this patient also has pain and a unilateral finding of linear calcifications, findings concerning for malignancy; a core needle biopsy should be obtained.

CHAPTER 43

1. Answer: d. A complete hematuria workup should be performed with any history of hematuria, even if it has resolved. A noncontrast CT or renal/bladder ultrasound would be appropriate imaging modalities for a patient with confirmed kidney stones, but are inadequate for diagnostic hematuria workup. Patients with renal failure or an IV contrast allergy require renal ultrasound, cystoscopy, and retrograde pyelograms (performed in the OR) as well as a urine culture and cytology.

2. Answer: b. A patient with an obstructing stone and any signs of infection (fevers, chills, leukocytosis, urinalysis concerning for urinary tract infection [UTI]) is at high risk for developing urosepsis and needs an urgent Urology consult for either a ureteral stent or percutaneous nephrostomy tube to relieve the obstruction. Any patient with an obstructing stone should have a urinalysis with microscopic analysis and a urine culture sent immediately before starting

antibiotics. In a patient with a stone and fever, it is appropriate to start an empiric antibiotic, usually a fluoroquinolone or third- or fourth-generation cephalosporin but this does not replace obtaining an urgent consult.

3. Answer: c. Urinary retention can be caused by any of the above. BPH alone does not present with systemic symptoms like fevers. The prostatic examination is typically normal with a urinary tract infection. Prostatic abscess typically presents with high fevers, leukocytosis, and significant pain. A periprostatic fluid collection may be palpable. While this clinical scenario does not rule out a prostatic abscess, the most likely diagnosis is bacterial prostatitis which is treated with empiric antibiotics.

4. Answer: a. Ischemic priapism is a urologic emergency and can result in permanent erectile dysfunction if untreated. Nonischemic or Òhigh flowÓ priapism is usually caused by increased arterial flow due to a traumatic fistula; it is not painful, and the penis is only semirigid on examination. Ischemic priapism is treated initially with irrigation with normal saline, aspiration, and intracavernosal phenylephrine injections. It is most commonly caused by medications (including trazodone and treatments for erectile dysfunction) or use of illegal drugs (e.g., cocaine) that affect vascular contractility.

5. Answer: e. Fournier gangrene is a necrotizing groin infection which initially presents with erythema and edema of the penis, scrotum, and/or perineum. It may originate from a scrotal, perineal, or perianal abscess. If left untreated, it progresses rapidly to frank necrosis with

significant morbidity and mortality risk. It is a clinical diagnosis, but the presence of gas/free air in the subcutaneous groin tissues along with the above examination findings should prompt immediate exploration with debridement. Patients often also have fevers and leukocytosis.

6. Answer: TRUE. All Grade I to III renal injuries and most Grade IV renal injuries may be safely observed with serial CBCs, bedrest until hematuria resolves, and repeat imaging in 48 to 72 hours. Exploration with possible nephrectomy is required when there's hemodynamic instability, an injury to the major renal vessels, and/or persistent hemorrhage. Urinary diversion, usually via a ureteral stent, is required if there is significant injury to the renal pelvis and/or proximal ureter with disruption of contrast excretion down the ureter.

7. Answer: c. Suspect bladder and/or urethral injury in any patient with pelvic or pubic fractures, particularly if they have urinary retention, hematuria, perineal or penile bruising. Blood at the meatus is a sensitive sign for urethral injury, but its absence does not rule it out. Catheterization should never be attempted without a retrograde urethrogram if there is any concern for urethral injury. If the RUG is

negative, a Foley catheter can be safely placed. CT cystogram is the most sensitive test for bladder injury. Passive bladder filling via a CT Urogram is not adequate and a standard fluoroscopic cystogram may miss subtle or posterior injuries. Placing a suprapubic tube may be appropriate if there is significant urethral injury but is unnecessarily morbid as initial management.

CHAPTER 44

1. Answer: d. Surgical staging for ovarian cancer involves collecting pelvic washings, total abdominal hysterectomy, bilateral salpingo-oophorectomy, pelvic and paraaortic lymphadenectomy, peritoneal biopsies, and omentectomy. If bulky disease is present in the intraperitoneal cavity, it should be removed to accomplish a debulking surgery to no gross residual disease. This can require splenectomy, bowel or bowel mesentery resection, diaphragm and peritoneal resection or stripping, and argon beam or plasma jet coagulation of tumor deposits. These procedures are not standard staging procedures.

2. Answer: b. Ultrasound is the best first-line imaging to evaluate the pelvic anatomy and differentiate physiologic adnexal structures from abnormal or potentially malignant masses. CT and MRI can be helpful in further classifying pelvic masses once the presence of a mass is established with ultrasound.

3. Answer: a. Women with Lynch Syndrome have a 15% to 66% lifetime risk of endometrial cancer depending on the mutation. There is some preliminary data to suggest that women with BRCA mutations may be at increased risk for endometrial cancer but not to the degree of women with Lynch Syndrome.

4. Answer: b. Women with postmenopausal bleeding, women >45 yo with abnormal bleeding,

or women of any age with risk factors for endometrial cancer and abnormal bleeding are recommended to undergo an endometrial biopsy. In this case, the patient has abnormal bleeding (daily) and a risk factor (obesity).

5. Answer: a. The gravid uterus can compress the inferior vena cava resulting in decreased blood return to the mother and poor placental perfusion resulting in maternal hypotension and fetal hypoxia.

6. Answer: c. The standard of care for stage IIIB cervical cancer is radiation therapy with sensitizing cisplatin. Stage IIIB cervical cancer by definition extends to the sidewall or obstructs ureteral flow and is therefore not resectable without significant morbidity.

7. Answer: b. An incomplete abortion is an ongoing spontaneous abortion such that the cervix is open and there continue to be products of conception within the endometrial canal. The cervix is closed in a missed abortion, inevitable abortion, and complete abortion.

CHAPTER 45

1. Answer: c. Chi-square test is used for categorical or nominal data. In this instance, patients either received prehydration with sodium bicarbonate or they did not. The outcome represents the presence or absence of contrast-induced nephropathy. This data can be summarized in a 2×2 contingency table.

2. Answer: c. A t-test allows for comparison of means between two separate treatment groups. If more than two treatment groups were compared, ANOVA would be the statistical test of choice. In order to use a t-test, data must be normally distributed.

3. Answer: d. While positive and negative predictive values are affected by the prevalence of a disease, sensitivity and specificity are not. As the prevalence of a disease increases, the positive predictive value increases and negative predictive value decreases.

4. Answer: a. Case-control studies are useful for rare diseases, as is the case with appendiceal cancer and pseudomyxoma peritonei. In the above question, the cases would be presented by patients with pseudomyxoma peritonei from appendiceal cancer and the controls would be patients with appendiceal cancer alone. Exposures in both groups could be evaluated to determine factors associated with the development of pseudomyxoma peritonei.

5. The correct answer for no. 5 is C, for no. 6 is A, and for no. 7 is C. The false positive rate is equal to the false positives divided by the sum of the false positive patients and the true negative patients.

CHAPTER 46

1. Answer: b. Run charts display a measured value plotted sequentially in time. They are particularly useful for measuring the effect of a quality improvement intervention by measuring

the change in the measured value following the intervention.

2. Answer: c. Value stream mapping is a component unique to the Lean method of quality improvement that in which processes are mapped as a series of steps for which efficiency is defined as time during each step in which value is added for the customer (or patient) compared to time spent without any value added

3. Answer: d. Normalization of deviance takes place when people repetitively and often progressively violate standard practices until the deviant practice becomes the new norm and creates conditions that are prone to error.

4. Answer: a. PDSA cycles describe a four-phase cycle to develop, test, and implement changes in health care, best used to test small changes before broad implementation. In combination with a

series of three questions, the PDSA cycle constitutes the Model for Improvement.

5. Answer: e. Root cause analysis is a retrospective team-based approach to delve deeper into a medical error or Ònear missÓ event to identify possible latent errors within a system. It is accomplished through defining the problem at hand, creating a detailed timeline of events, exploring possible causes to find their source, brainstorming possible solutions, and then implementing and intermittently reviewing these solutions to measure effect of changes made through the process.