6 SURGERY AND SURGICAL SUBSPECIALTIES

TRAUMA

The ABCDEs are the key to the initial management of patients with trauma. Always do them in order. For example, if the patient is bleeding to death and has a blocked airway, you may have to choose which issue to address first. The first priority is airway management.

A = Airway maintenance and cervical spine care. Provide, protect, and maintain an adequate airway at all times and assume a cervical injury is present while doing so (i.e., place cervical collar, and do not hyperextend neck) until it is excluded. If the patient can answer questions, the airway is fine. You can use an oropharyngeal or nasopharyngeal airway in uncomplicated cases and give supplemental oxygen. When the airway is blocked or if the Glasgow Coma Scale (GCS) score is less than 8, intubate. If intubation fails, do a cricothyroidotomy.

B = Breathing and ventilation. Assess chest wall, lung expansion, lung function, and ventilation. If the airway is open and patient is not breathing, intubate. If intubation fails, do a cricothyroidotomy. Look for acute problems such as flail chest, tension pneumothorax, or massive hemothorax.

C = Circulation and control of hemorrhage. If the patient seems hypovolemic (tachycardia, bleeding, weak pulse, paleness, diaphoresis, capillary refill >2 seconds), give intravenous (IV) fluids and/or blood products. The initial procedure is to start two large-bore catheters and give a bolus of 10–20 mL/kg (roughly 1 L) of lactated Ringer's solution (IV fluid of choice in trauma) and blood. Then reassess the patient for improvement. Repeat the bolus if necessary.

D = Disability and neurologic status. Check neurologic function (using the Glasgow Coma Scale). Rule out hypoxia, hypovolemia, and hypoglycemia before considering central nervous system injury.

E = Exposure/Environment. Remove the patient's clothing and "put a finger in every orifice" so that you do not miss any occult injuries. Look for hypothermia.

In general, all trauma patients should have chest and pelvic radiographs, with computed tomography (CT) scans used as needed for further evaluation or persistent symptoms despite negative radiographs.

Evaluate head and cervical spine trauma with *noncontrast CT* (better than magnetic resonance imaging [MRI] for intracranial hemorrhage and fractures).

Abdominal trauma

Blunt abdominal trauma: Initial findings determine a course of action (Table 6-1):

Table 6-1 Evaluation of Abdominal Trauma

STUDY	FEATURES	ADVANTAGES	DISADVANTAGES
Plain radiographs	Limited value in most abdominal traumas because CT provides better detail	Useful for projectile penetrating abdominal trauma (gunshot trajectory)	Not useful for blunt or nonprojectile penetrating abdominal trauma
Diagnostic peritoneal lavage	Generally not used anymore and replaced by FAST and CT scans	Takes little time to perform	Invasive, does not detect retroperitoneal bleed, does not detect organ damage
FAST scan	Gaining widespread use for initial evaluation of blunt trauma	Portable, rapid, noninvasive, useful for detecting hemoperitoneum	Does not detect organ damage, operator-dependent, may miss small bleeds (<100 mL)

Table 6-1 Evaluation of Abdominal Trauma—cont'd

STUDY	FEATURES	ADVANTAGES	DISADVANTAGES
CT scan	Commonly used if time permits	Provides details on hemorrhage and organ damage	Time constraints limit use in hemodynamically unstable patients
Laparotomy	Used for hemodynamically unstable patients who do not have time to undergo other studies	Rapid assessment and correction of abnormalities	Invasive, may not detect some organ injuries

- If the patient is awake and stable and your exam is benign, observe and repeat the abdominal exam later.
- If the patient has significant trauma and does not require urgent surgery, order a focused assessment with sonography for trauma (FAST) ultrasound or CT scan of the abdomen and pelvis with IV contrast after the patient is stabilized (diagnostic peritoneal lavage is no longer used). Do not send an unstable patient to the CT scanner.
- If the patient is hemodynamically unstable (from hypotension and/or shock that does not respond to a fluid challenge), proceed directly to laparotomy.

Penetrating abdominal trauma: Type of injury and initial findings determine the course of action:

- With a gunshot wound in an unstable patient, proceed directly to laparotomy.
- With a wound from a sharp instrument, management is more controversial. Either proceed directly to laparotomy (better choice if the patient is unstable) or do a CT scan. If the results are positive, consider laparotomy on the basis of the injury; if the results are negative, observe and repeat the exam later.

Chest trauma

Six thoracic injuries can be rapidly fatal and must be recognized immediately:

1. **Airway obstruction:** The patient has no audible breath sounds and cannot answer questions even though he or she may be awake and gurgling. Clear the airway if possible and treat with *endotracheal intubation.* If intubation fails, do a *cricothyroidotomy* (or tracheostomy in the operating room if there is time).
2. **Open pneumothorax:** An open defect in the chest wall causes poor ventilation and oxygenation. Treat with *endotracheal intubation, positive-pressure ventilation,* and *closure of the defect in the chest wall.* Gauze should be used and taped on only three sides to allow excessive pressure to escape so that you do not convert an open pneumothorax into a tension pneumothorax.
3. **Tension pneumothorax:** Usually seen after blunt trauma, tension pneumothorax occurs when air is forced into the pleural space and cannot escape. It collapses the affected lung and then *shifts the mediastinum and trachea to the opposite side* of the chest. The patient has *absent breath sounds* and a *hypertympanic or hyperresonant percussion sound* on the affected side. Impaired cardiac filling may result in hypotension and/or *distended neck veins.* Treat with *needle thoracentesis* (anterior second intercostal space usually preferred), followed by insertion of a chest/thoracostomy tube.
4. **Cardiac tamponade:** The classic history is one of penetrating trauma to the left chest. The patient has hypotension (due to impaired cardiac filling), *distended neck veins, muffled heart sounds, pulsus paradoxus* (exaggerated fall in blood pressure on inspiration), and *normal breath sounds.* Treat with *pericardiocentesis* if the patient is unstable: Put a catheter in the pericardial sac (via a subxiphoid approach) and aspirate the blood or fluid. If the patient is stable, you can do a Fast scan, an echocardiogram, or a chest CT scan to confirm the diagnosis.
5. **Massive hemothorax:** With loss of more than 1 L of blood into the thoracic cavity, the patient will have *decreased breath sounds* on the affected side, *dull note on percussion,* hypotension, *collapsed neck veins* (from blood leaving the vascular tree), and tachycardia. Placement of a chest tube allows the blood to come out. Give IV fluids and/or blood before you place the chest tube. If the bleeding stops after the initial outflow, order a radiograph and/or CT scan to check for remaining blood or pathology and treat supportively. Emergent thoracotomy is required if the bleeding does not stop or is massive.
6. **Flail chest:** When several adjacent ribs are broken in multiple places, the affected part of the chest wall can move paradoxically (inward during inspiration, outward during expiration) during respiration (Fig. 6-1). There is almost always an associated *pulmonary contusion,* which, combined with pain, may make respiration inadequate. When you are in doubt or if the patient is not doing well, *intubate* and give *positive-pressure ventilation.*

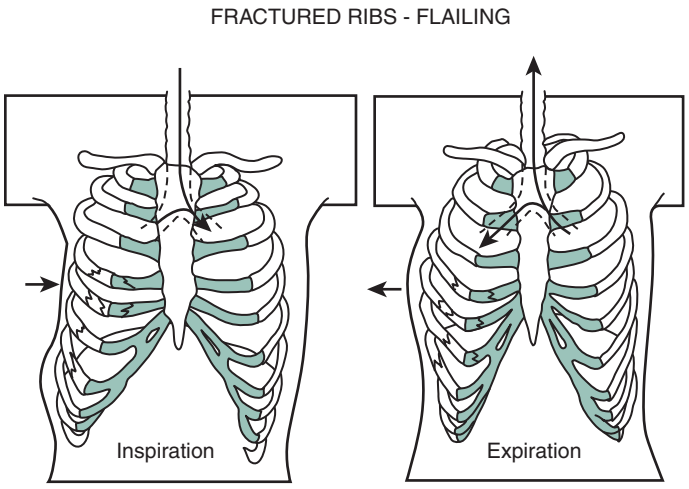

FIGURE 6-1 Paradoxical respiration: inward motion with inspiration; outward motion with expiration. *(From James EC, Corry RJ, Perry JF: Principles of Basic Surgical Practice. Philadelphia, Hanley & Belfus, 1987.)*

Other injuries

Thoracic aortic injury/rupture: The most common cause of immediate death after an automobile accident or fall from great height. Usually occurs just beyond the takeoff of the subclavian artery (aortic isthmus, where the aorta is tethered in place by the ligamentum arteriosum). Thoracic aortic laceration with a contained rupture (i.e., intact adventitia) can allow survival, but in many cases with rupture (50%), the patient dies within 24 hours of reaching the hospital. Thus, treat with immediate surgical management after diagnosing with chest CT with IV contrast. Without treatment survival beyond 4 months is less than 5%.

CASE SCENARIO: What is the classic chest x-ray finding with aortic laceration? Widened mediastinum. Order a CT chest with contrast (or CT angiogram) if you are suspicious (because of x-ray findings or due to trauma degree, location, and/or type). Treat with immediate surgical repair.

CASE SCENARIO: On which side does a traumatic diaphragm rupture usually occur? The left. The liver is believed to protect the right side. The classic findings consist of bowel sounds auscultated in the chest or seeing bowel loops in the thorax on chest radiograph. Fix surgically.

Head trauma: See "Neurosurgery" later.

Neck trauma: The neck is divided into three zones for trauma:

- **Zone I:** base of the neck (from 2 cm above the clavicles to the level of the clavicles)
- **Zone II:** midcervical region (2 cm above the clavicle to the angle of the mandible)
- **Zone III:** the angle of the mandible to the base of the skull

Management is dictated by location of the injury:

- With symptomatic zone I and III injuries, do a CT angiogram before going to the operating room in a stable patient.
- With zone II injuries, proceed to the operating room for surgical exploration in symptomatic patients; consider CT angiogram first if the patient is stable.
- In patients with obvious bleeding or a rapidly expanding hematoma, proceed directly to the operating room no matter where the injury is.

Choking victim: Leave choking patients alone if they are speaking, coughing, or breathing. If they stop doing all of these things, perform the Heimlich maneuver.

If a tooth is knocked out, put it back in place with no cleaning (or rinse it only with saline or milk) and stabilize as soon as possible. The sooner this is done, the better the prognosis for salvage of the tooth.

Burns

Burns may be electrical, chemical, or thermal. Initial management of all burns includes plenty of IV fluids (use lactated Ringer solution or normal saline if lactated Ringer is not a choice), removal of all clothes and other smoldering items on the body, copious irrigation of chemical burns, and, of course, the ABCs. You should have a low threshold for intubation; use 100% oxygen until significant carboxyhemoglobin from carbon monoxide inhalation is ruled out.

- **Electrical burns.** Because most of the destruction is internal, patients may have myoglobinuria, acidosis, and renal failure. Use aggressive IV fluids to prevent renal failure. The immediate,

life-threatening risk with electricity exposure and burns (including lightning and putting a finger in an electrical outlet) is cardiac arrhythmias. Order an electrocardiogram (ECG).

- **Chemical burns.** Alkali burns are worse than acidic burns because alkali penetrates more deeply. Treat all chemical burns with copious irrigation from the nearest water source.
- **Thermal burns.** Burned skin is much more prone to infection, usually by *Staphylococcus aureus* or *Pseudomonas* spp. Pseudomonal infection causes a *fruity smell* and/or *blue-green color.* Prophylactic antibiotics are given topically, not systemically. Severity is classified as shown Table 6-2.

Table 6-2 Burn Depth Classification

BURN DEPTH	APPEARANCE	SKIN LAYER(S)	SENSATION	HEALING
Superficial	Dry, pink or red	Epidermis	Painful	Days, keep clean
Superficial partial thickness	Moist, pink or red with blisters	Epidermis, less than ½ of dermis	Painful	7-14 days. Remove blister, apply antibiotic ointment, apply dressing
Deep partial thickness	Dry, white, brown, leathery, vesicles, blisters	Epidermis, more than ½ of dermis including nerves	Less painful than superficial burns	14-21 days. Keep clean and infection-free, apply antibiotic ointment, apply dressing, monitor fluid loss
Full thickness	Dry, brown, charred	Epidermis, full dermis including nerves	Dull	Excision, skin grafting, monitor fluid loss and metabolic effects

Many burns have a combination of degrees. Watch for compartment syndrome, which is treated with escharotomy.

CASE SCENARIO: What is the major difference in symptoms between superficial and deep burns? Superficial burns are painful, whereas deep burns are classically *painless* initially because of nerve damage.

CASE SCENARIO: What vaccine should burn victims receive? Tetanus.

Hypothermia and Hyperthermia

Hypothermia: Treatment varies depending on the degree of hypothermia and background health (Table 6-3).

Table 6-3 Overview of Hyperthermia

DEGREE	TEMPERATURE*	SYMPTOMS	WARMING THERAPY
Mild hypothermia	32.2-35°C	Arrhythmia, ataxia, shivering	Remove wet clothing, warm blankets
Moderate hypothermia	28-32.2°C	Decreased consciousness, decreased pulse and respiration, dysrhythmias, no shivering	Warm IV fluids, warm oxygen, warm bath water, extracorporeal blood warming with cardiopulmonary bypass
Severe hypothermia	<28°C	Absence of reflexes, no response to pain, risk of ventricular fibrillation	

*Measured core body temperatur.

In general, secure the airway, intubate if necessary, remove wet clothing, rewarm with blankets, and/or give warm IV fluids. The most important point is to monitor the ECG for arrhythmias, which are common with hypothermia. The rare but classic finding is the *J wave,* a small, positive deflection following the QRS complex (Fig. 6-2). Also monitor electrolytes, renal function, and acid-base status.

With frostnip (cold, painful areas of skin; mild injury) and frostbite (cold, anesthetic areas of skin; more severe injury), treat by rewarming affected areas with warm water (not scalding hot) and generalized warming (e.g., blankets).

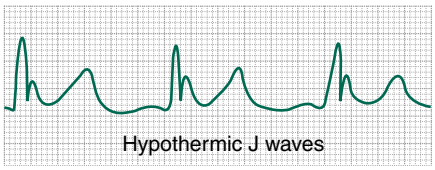

Figure 6-2 Hypothermic J waves. *(From Ferri FF: Practical Guide to the Care of the Medical Patient, 8th ed. Philadelphia, Mosby, 2011.)*

Hyperthermia may be due to heat exhaustion (38.7–40°C) and heat stroke (>40°C). Heat exhaustion symptoms include generalized malaise, weakness, cramps, nausea, and *sweating*. Heat stroke symptoms include neurologic abnormalities (seizures, psychosis, coma), dehydration, and the *absence of sweating*. Treat heat exhaustion with cooling in a dry area and oral replacement of fluids and salt. Treat heat stroke with airway support (ABCs), immediate cooling (wet blankets, ice packs, cold water), and IV fluids. The immediate threats to life are convulsions (treat with diazepam) and cardiovascular collapse. Rule out infection and other classic culprits:

1. Malignant hyperthermia: Look for succinylcholine or halothane exposure. Treat with supportive care and *dantrolene.*
2. Neuroleptic malignant syndrome: The classic patient is taking an antipsychotic. First, stop the medication. Second, treat with support (especially lots of IV fluids to prevent renal shutdown from rhabdomyolysis) and possibly dantrolene.

CASE SCENARIO: What lab value is markedly elevated in patients with neuroleptic malignant syndrome? The *creatine kinase (CK)* level, because of muscle breakdown.

3. Drug fever: idiosyncratic reaction to a medication that usually was started within the past few weeks.

Near drowning

Fresh water is said by some to be worse than salt water because fresh water, if aspirated, can cause hypervolemia, electrolyte disturbances, and hemolysis. Others think this distinction is nonsense. Intubate patients if they are unconscious, and monitor arterial blood gases if they are conscious. Patients who almost drown in cold water often do better than those who almost drown in warm water (due to decreased metabolic needs). Death usually results from hypoxia and/or cardiac arrest.

GENERAL SURGERY

Acute abdomen:

An inflamed peritoneum often buys the patient a laparotomy because it signifies a potentially life-threatening condition; important exceptions are pancreatitis, most cases of diverticulitis, and spontaneous bacterial peritonitis. The best physical confirmation of peritonitis is rebound tenderness and involuntary guarding/abdominal muscular rigidity. Voluntary guarding is a softer sign, as is tenderness to palpation; both are often present in benign diseases. When you are in doubt and the patient is stable, withhold narcotics, which mask symptoms, until you have a diagnosis. Do serial abdominal exams. Perform CT scan with oral and IV contrast. If the patient is unstable, proceed to laparoscopy/laparotomy.

Acute abdomen localization:

1. Right upper quadrant (RUQ): Think of gallbladder (cholecystitis), bile ducts (cholangitis), or liver (abscess).
2. Left upper quadrant (LUQ): Think of spleen (rupture with blunt trauma or rarely, abscess).
3. Right lower quadrant (RLQ): Think of appendix (appendicitis) or obstetric/gynecologic problem.
4. Left lower quadrant (LLQ): Think of sigmoid colon (diverticulitis) or obstetric/gynecologic problem.
5. Epigastric: Think of stomach (penetrating ulcer) or pancreas (pancreatitis).

Cholecystitis: The four Fs summarize the classic patient with *cholesterol* stones: fat, forty, fertile, female, and a fifth F, febrile, suggests cholecystitis, especially if gallstones are seen on ultrasound scan or the patient has a history of gallstones and/or gallstone-type symptoms (e.g., postprandial right upper quadrant [RUQ] colicky pain with bloating and/or nausea and vomiting). Look for the *Murphy sign*. Ultrasound imaging is the preferred initial test. Do a hepato-iminodiacetic acid (HIDA) scan if ultrasound findings are equivocal. Do a cholecystectomy for most patients. Remember that *pigment gallstones* are seen in patients with hemolytic anemias, not "5-F" patients.

CASE SCENARIO: What triad of findings is associated with cholangitis? *RUQ pain, fever* (usually with shaking chills), and *jaundice*. Patients often have a history of gallstones. Start antibiotics after getting blood cultures, and do a cholecystectomy once the patient is stable.

Splenic rupture: Patients have a history of blunt abdominal trauma, hypotension or tachycardia, shock, and *Kerr sign* (pain referred to the left shoulder). Ultrasound and CT can be used to evaluate the

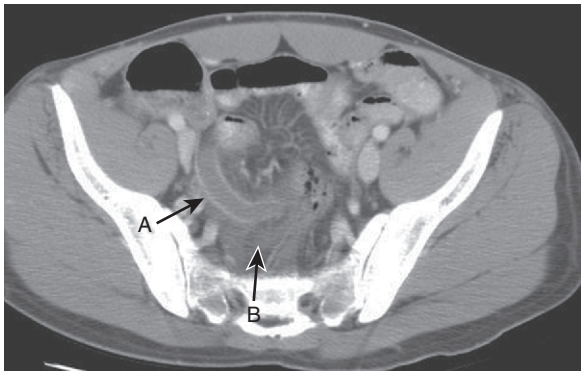

FIGURE 6-3 Appendicitis. Dilated thickened appendix (*A*), with adjacent hazy fat (**B**). *(From Rakel RE, Rakel D: Textbook of Family Medicine, 8th ed. Philadelphia, Saunders, 2011.)*

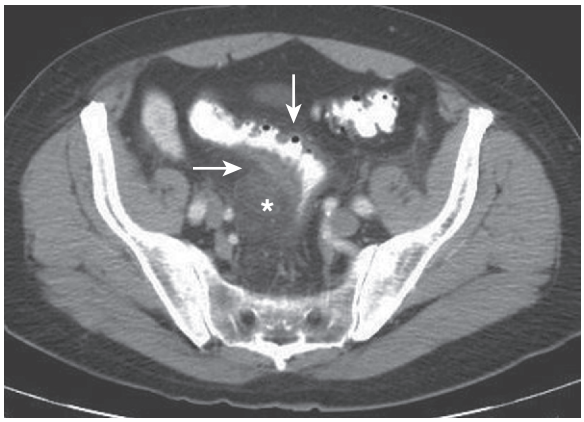

FIGURE 6-4 Diverticulitis. Computed tomography (CT) image demonstrates thickened wall of the sigmoid colon *(arrows)* with stranding in the adjacent fat (*). *(From McNally PR: GI/Liver Secrets Plus, 4th ed. Philadelphia, Mosby, 2010.)*

extent of injury. Consider splenectomy if the patient is unstable or if the spleen is extensively injured with continuous bleeding. Do not let patients with Epstein-Barr virus (EBV) infection play contact sports. Do not forget to immunize postsplenectomy patients (pneumococcal, meningococcal, *Haemophilus influenzae* type b vaccines).

Appendicitis: The incidence peaks in 10- to 30-year-olds. The classic history is crampy, poorly localized periumbilical pain followed by nausea and vomiting, then localization of pain to the RLQ and peritoneal signs with worsening of nausea and vomiting. Patients who are hungry and asking for food do not have appendicitis. Remember the Rovsing sign, as well as McBurney point tenderness. Use CT scan of the abdomen (or ultrasound in children or pregnant women) to help make the diagnosis when patients are stable (Fig. 6-3). Do an appendectomy even if the imaging is negative if the history and exam are indicative.

Diverticulitis: Localized LLQ pain in a patient older than 50 is diverticulitis unless you have a good reason to think otherwise. *CT scan* with oral, rectal, and/or IV contrast is the best test to confirm disease, rule out a complicating abscess, and exclude an alternative diagnosis (Fig. 6-4). Treat medically with broad-spectrum antibiotics (e.g., ciprofloxacin + metronidazole) and place the patient on NPO (nothing-by-mouth) status. If disease is recurrent or refractory to medical therapy, if there is an abscess, or if peritonitis is present, consider surgical resection. Diverticular abscesses can be treated with percutaneous drainage.

Acute pancreatitis: Look for epigastric pain in an alcohol abuser or a patient with a known history of gallstones. Pain may radiate to the back, and serum *amylase* and *lipase* are elevated (if these values are not given, order these tests). Other common symptoms include decreased bowel sounds, local ileus (sentinel loop of bowel on radiograph), nausea, vomiting, and anorexia. CT scan is the preferred imaging test (Fig. 6-5). Treat with pain medications, NPO status, nasogastric tube, IV fluids, and supportive care. Watch for complications of *pseudocyst* and *pancreatic abscess,* which may require surgical intervention.

CASE SCENARIO: A patient has a history of ulcers, epigastric pain, peritoneal signs, mildly elevated amylase, and normal lipase. A small amount of free air is noted under the diaphragm on abdominal radiograph. What is the likely diagnosis? Perforated peptic ulcer, which can cause elevated amylase (but lipase is typically normal).

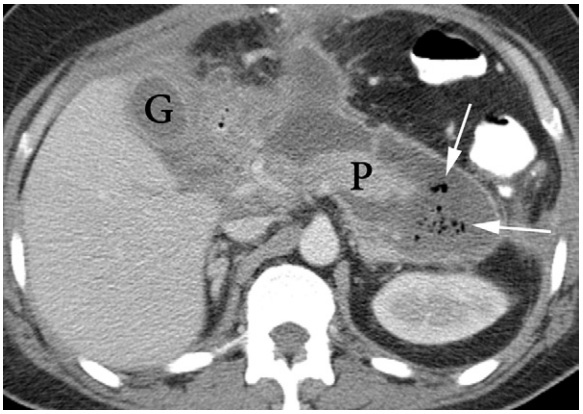

FIGURE 6-5 Acute necrotizing pancreatitis. On contrast-enhanced computed tomography scan, the pancreas (P) is surrounded by peripancreatic inflammation that contains bubbles of air *(arrows)* due to sterile necrosis. G, Gallbladder. *(From Feldman M, Friedman LS, Brandt LJ: Sleisenger and Fordtran's Gastrointestinal and Liver Disease, 8th ed. Philadelphia, Saunders, 2006.)*

Small bowel obstruction: Signs and symptoms include *bilious vomiting* (early symptom), *abdominal distention,* constipation, hyperactive bowel sounds (high-pitched, rushing sounds), and pain that is usually poorly localized. Radiograph shows multiple air-fluid levels in dilated small bowel loops. Patients often have a *history of previous surgery;* the most common cause of small bowel obstruction (SBO) in adults is *adhesions,* which usually develop from prior surgery. In children, think of *incarcerated inguinal hernia* or *Meckel diverticulum.* Start treatment with NPO status, nasogastric tube with suction, and IV fluids. Order CT scan with IV contrast to confirm the diagnosis and exclude a specific underlying disorder other than adhesions (e.g., hernia, tumor). If symptoms do not resolve or if the patient develops peritoneal signs, laparotomy is necessary to relieve the obstruction.

Large bowel obstruction: Signs and symptoms include gradually increasing abdominal pain, *abdominal distention,* constipation, and *feculent vomiting* (late symptom). This condition is seen more often in older patients as a result of *diverticulitis, colon cancer,* or *volvulus.* Treat early with NPO status and nasogastric tube. Sigmoid volvulus often can be decompressed with an endoscope. Other causes or refractory cases require surgery to relieve the obstruction. CT scan with IV contrast or barium enema can confirm the diagnosis and may suggest the etiology. In children, watch for *Hirschsprung disease.*

Hernias

The four common types (there are others) are treated with surgical repair if they are symptomatic. Now that bariatric surgery has become more common, internal hernias have also become more common:

1. **Indirect:** most common in both sexes and all age groups. The hernia sac travels through the inner and outer inguinal rings (protrusion begins lateral to the inferior epigastric vessels) and *into the scrotum (or labia)* due to a patent processus vaginalis (congenital defect).
2. **Direct:** the hernia (no sac) protrudes *medial to the inferior epigastric* vessels (and not into the scrotum or labia) due to weakness in the abdominal musculature (of Hesselbach triangle).
3. **Femoral:** more common in women. The hernia (no sac) goes through the femoral ring onto the *anterior thigh (located below the inguinal ring).* Femoral hernias are the most susceptible to incarceration and strangulation.
4. **Incisional:** after any wound (especially surgical), a hernia can occur through the site of the incision.

There are two main complications associated with hernias:

- Incarceration is when herniated organs are trapped, cannot be reduced, and become swollen or edematous.
- Strangulation is when the entrapment/incarceration becomes so severe that the blood supply is cut off. *Strangulation can lead to necrosis and is a surgical emergency.* Patients may present with SBO symptoms and shock.

CASE SCENARIO: What is the most common cause of small bowel obstruction in a person who has never had surgery before? Incarcerated hernia (which is the second most common cause in patients with prior abdominal surgery, after adhesions).

Preoperative and postoperative points

1. Preoperatively, keep the patient on NPO status for at least 8 hours (when possible) to reduce the chance of aspiration.

2. Spirometry (and, of course, a good history) is the best preoperative test to order for assessment of pulmonary function. It measures forced vital capacity (FVC), forced expiratory volume in 1 second (FEV_1), FEV_1/FVC ratio (%), and maximum voluntary ventilation.
3. Use compressive/elastic stockings, early ambulation, and/or prophylactic-dose low-molecular-weight heparin to help prevent deep venous thrombosis and pulmonary embolism.
4. The most common cause of postoperative fever in the first 24 hours is *atelectasis* (usually low-grade fever). Prevent or treat with early ambulation, chest physiotherapy/percussion, incentive spirometry, and proper pain control. Both too much pain and too many narcotics increase the risk of atelectasis.
5. The mnemonic *"water, wind, walk, wound, and wonder drugs"* will help you recall the causes of postoperative fever: water, urinary tract infection; wind, atelectasis/pneumonia; walk, deep venous thrombosis; wound, surgical wound infection; wonder drugs, drug fever. If daily fever spikes occur, think about an intraabdominal abscess; consider a CT scan to locate the abscess. Abscesses often need surgical or CT-guided catheter drainage.
6. Fascial/wound dehiscence typically occurs 5–10 days postoperatively. Look for leakage of serosanguineous fluid from the wound (often after the patient coughs or strains), which is especially associated with wound infection. Treat with antibiotics (if secondary to infection) and reclosure of the incision.

EAR, NOSE, AND THROAT SURGERY

Infections

Rhinitis: edematous, vasodilated nasal mucosa and turbinates with clear nasal discharge. Causes include the following:

1. **Viral infection** (common cold): due to rhinovirus (most common), influenza virus, parainfluenza virus, adenovirus, or others. Treatment is symptomatic with short-term use of vasoconstrictors such as phenylephrine. Vasoconstrictors may cause rebound congestion, however.
2. **Allergy** (hay fever): associated with *seasonal* flare-ups, *boggy* and *bluish* turbinates, onset before the age of 20 years, *nasal polyps,* sneezing, pruritus, conjunctivitis, wheezing, asthma, eczema, family history, *eosinophils in nasal mucus,* and elevated serum immunoglobulin E (IgE) levels. Skin tests may identify an allergen. Treat with avoidance of any known antigen (e.g., pollen), antihistamines, immunotherapy, and/or *intranasal steroid spray* for severe symptoms. Desensitization is also an option.
3. **Bacterial infection:** typically due to streptococci, pneumococci, or staphylococci. Do a streptococcal throat culture. Treat with antibiotics if appropriate (sore throat, fever, tonsillar exudate).

Sinusitis is usually due to viral or bacterial causes (*Streptococcus pneumoniae, Haemophilus influenzae, Moraxella* spp., other streptococci, or staphylococci). Look for fever, *tenderness over the affected sinus, headache,* and *purulent nasal discharge* (yellow or green) generally for >10 days. The diagnosis is generally based on clinical signs. The gold standard for diagnosis is positive culture from the paranasal sinuses. Imaging helps support the diagnosis. A four-view sinus radiograph may show opacification or air-fluid levels. CT scan is more sensitive than radiographs (and is now the imaging modality of choice if imaging is necessary) and is used to evaluate chronic sinusitis or suspected extension of infection outside the sinus (in patients with high fever and chills) (Fig. 6-6). Acute viral sinusitis generally resolves in 2 weeks without antibiotics. For moderate or severe symptoms, treat with antibiotics (amoxicillin, macrolide, cephalosporin, or doxycycline) for 10–14 days. Surgical drainage is indicated for intracranial complications, frontal sinusitis, or sinusitis recalcitrant to medical therapy. Deviated nasal septum or other congenital defects causing recurrent sinusitis are treated with surgical correction.

Otitis externa (swimmer's ear): most commonly due to *Pseudomonas aeruginosa*. *Manipulation of the auricle produces pain* (this sign is not present in otitis media), the skin of the auditory canal is erythematous and swollen, and patients may have a foul-smelling discharge and conductive hearing loss. CT scan (rarely needed) may help define bone involvement and extent of disease. Treat with 2% acetic acid to inhibit growth, topical antibiotics (neomycin/polymyxin B), and steroids to help reduce swelling and inflammation.

CASE SCENARIO: What are the classic physical findings and bacterial cause for infectious myringitis? Otoscopy classically reveals vesicles on the tympanic membrane, and the classic cause is *Mycoplasma* spp. Other causes include *S. pneumoniae* and viruses. Treat with antibiotics.

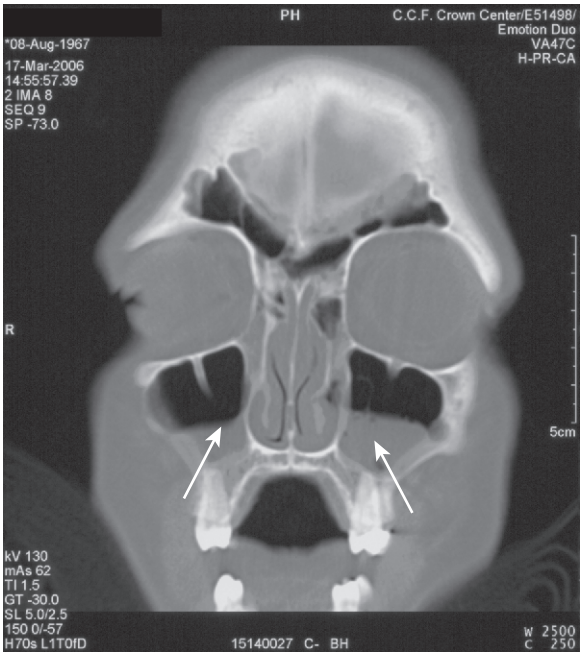

FIGURE 6-6 Computed tomography scan showing acute sinusitis. Note the fluid levels in the maxillary sinuses *(arrows). (From Cleveland Clinic: Current Clinical Medicine, 2nd ed. Philadelphia, Saunders, 2010.)*

Causes of hearing loss

1. **Aging** (presbycusis): most common cause of *sensorineural hearing* loss in adults; a normal part of aging, not a disease. Patients can use hearing aids, if necessary.
2. **Environmental noise:** Prolonged or intense loud noise can permanently affect hearing. Advise earplugs for occupationally exposed patients.
3. **Otosclerosis:** most common cause of progressive *conductive hearing loss* in adults. Otic bones become fixed together, impeding hearing. Treat with hearing aids or surgery.
4. **Meningitis or recurrent otitis media:** the classic causes in children. Screen for hearing loss after meningitis.
5. **Congenital hearing loss:** toxoplasmosis, other (congenital syphilis and viruses), rubella, cytomegalovirus, and herpes simplex virus (TORCH) infection or inherited disability.
6. **Ménière disease:** usually occurs in middle-aged patients. The cause is unknown. Look for severe *vertigo, tinnitus, fluctuating hearing loss, fullness in the ear.* Treat with diuretics (hydrochlorothiazide), prochlorperazine, antihistamines (e.g., *meclizine*), diuretics (hydrochlorothiazide), or surgery (for refractory cases).
7. **Drugs:** aminoglycosides, aspirin (overdose causes tinnitus), quinine, loop diuretics, cisplatin.
8. **Tumor:** usually a vestibular schwannoma (schwannoma of the eighth cranial nerve, associated with neurofibromatosis 2).
9. **Labyrinthitis:** may have a viral etiology or follow or extend from meningitis or otitis media. Viral etiology often causes sudden deafness that develops over a few hours. Hearing usually returns within 2 weeks, but loss may be permanent. No treatment has proved effective. Reassurance and bed rest are preferred. Antiemetic (phenergan), vestibular suppressant (meclizine), and empirical steroids (methylprednisone) often are used.

Causes of vertigo

1. **Ménière disease:** accompanied by tinnitus, hearing loss, and nausea/vomiting. See earlier discussion.
2. **Benign positional/paroxysmal vertigo:** *induced by certain head positions* and may be accompanied by nystagmus without hearing loss. Cases often resolve spontaneously; the only treatment generally necessary is to avoid the position that provokes symptoms.
3. **Acoustic schwannoma**
4. **Stroke**
5. **Infection**
6. **Multiple sclerosis:** a possible cause of any weird neurologic symptoms, usually in women of reproductive age.

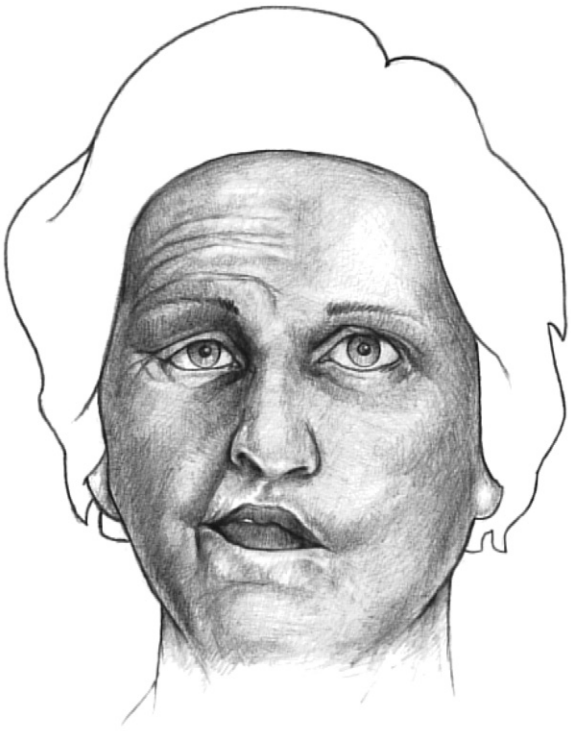

FIGURE 6-7 Bell palsy (facial nerve palsy). Note unwrinkled forehead, widely opened eyes (with weakness of eyelid color), flattening of the nasolabial fold, and a droop of the corner of the mouth. *(From Remmel KS, et al: Handbook of Symptom-Oriented Neurology, 3rd ed. St Louis, Mosby, 2002.)*

Causes of facial paralysis

Note *Perform CT or MRI if stroke, tumor, or fracture is suspected.*

1. **Stroke:** commonly associated with older age, other deficits, and stroke risk factors.
2. **Bell palsy:** acute facial (seventh) nerve palsy (Fig. 6-7). Characterized by sudden unilateral onset, usually after an upper respiratory infection. Peak incidence in persons older than 70 years and pregnant women. The cause is thought to be a reactivation of *herpes simplex* virus 1 in most cases. Patients may have *hyperacusis* (everything sounds loud because the stapedius muscle in the ear is paralyzed). In cases in which patients are unable to close the affected eye, use artificial tears and a patch to protect the eye. Glucocorticoids and antivirals have shown benefit. Most cases resolve spontaneously in about 1–3 months, but some patients have permanent sequelae. Remember that the patient will have a lower motor neuron deficit in this disorder, so the upper half of the affected side of the face is involved (whereas it should be spared with an upper motor neuron lesion such as stroke).
3. **Herpes zoster** (Ramsay Hunt syndrome): also causes ear pain. Look for *vesicles on the pinna and inside the ear.* Encephalitis or meningitis may be present.
4. **Lyme disease:** probably the most common cause of bilateral facial nerve palsy.
5. **Middle ear or mastoid infections/meningitis:** Look for other symptoms of the infection.
6. **Temporal bone fracture:** Patients may have Battle sign (bruising over the mastoid process), bleeding from the ear, and deafness.

Neck mass

75% benign in children, 75% malignant in patients older than 40 years. Causes include the following:

1. **Branchial cleft cysts:** seen in children; *lateral;* may become infected.
2. **Thyroglossal duct cysts:** seen in children; *midline;* elevates with tongue protrusion.
3. **Cystic hygroma:** lymphangioma seen in children, classically in patients with *Turner syndrome;* treat with surgical resection.
4. **Cervical lymphadenitis:** may occur in children or adults, usually as a result of streptococcal pharyngitis, Epstein-Barr virus (common in adolescents and young adults in their 20s), cat-scratch disease, or mycobacterial infection *(scrofula).*
5. **Neoplasm:** more common in adults than in children. The mass may be lymphadenopathy from primary (lymphoma) or metastatic neoplasm (usually a squamous cell carcinoma of the pharynx or larynx), or it may be the tumor itself.

CASE SCENARIO: What is the classic workup for an "unknown cancer" (unknown site of primary malignancy) found in the neck? Random biopsy of the nasopharynx, palatine tonsils, and the base of the tongue, as well as laryngoscopy, bronchoscopy, and esophagoscopy (with biopsies of any suspicious lesions)—the so-called triple endoscopy with triple biopsy. CT/MRI or positron emission tomography (PET) scan also may help to detect lesions not apparent on physical exam.

Parotid swelling: Classically due to mumps. The best treatment for mumps and the complication of infertility is prevention through immunization. Provide supportive care for acute cases. Parotid swelling also may be due to alcoholism, human immunodeficiency virus (HIV) infection–related, neoplasm (the most common is benign pleomorphic adenoma), Sjögren syndrome, or sarcoidosis.

After a nasal fracture (seen on radiograph or CT scan), rule out a septal hematoma, which must be evacuated to prevent pressure-induced septal necrosis.

NEUROSURGERY

Intracranial bleed

Whenever an intracranial bleed is suspected, order a *CT scan without contrast.* Blood appears white and may cause a shift of midline structures to the opposite side (Fig. 6-8). Causes include the following:

1. **Subdural hematoma,** which is due to bleeding from veins that bridge the cortex and dural sinuses. On radiographs the hematoma is *crescent-shaped.* Subdural hematomas are common in alcoholics and after head trauma. They may manifest immediately after trauma or as long as 1–2 months later. If the question gives a history of head trauma, always consider the diagnosis of a subdural hematoma. Treat with surgical evacuation if significant or progressive symptoms are present.
2. **Epidural hematoma** is due to bleeding from meningeal arteries (classically, the middle meningeal artery). On CT scan the hematoma is *biconvex.* Almost all epidural hematomas are associated with a *temporal bone skull fracture,* and roughly 50% of patients develop an *ipsilateral "blown" pupil.* The classic history is one of head trauma with loss of consciousness, followed by a *lucid interval* of minutes to hours and then neurologic deterioration. Treat with surgical evacuation.
3. **Subarachnoid hemorrhage** is due to blood between the arachnoid and pia mater. The most common cause is *trauma,* followed by *ruptured intracranial* (typically berry) *aneurysm.* Blood can be seen in the cerebral cisterns, ventricles, and sulci. The classic patient has an aneurysm rupture and presents with the *"worst headache of my life,"* although many die before they reach the hospital or may be unconscious. If awake, patients have *signs of meningitis* (Kernig and Brudzinski signs) without significant fever. Remember the association between *polycystic kidney disease* and berry aneurysms. CT scan is the test of choice to diagnose subarachnoid hemorrhage. Lumbar puncture will demonstrate *grossly bloody cerebrospinal fluid* (but is not necessary if the CT scan is positive). Treat with support, anticonvulsants, and observation. Perform CT angiography or magnetic resonance angiography (MRA) to look for aneurysms and arteriovenous malformations (AVMs), which are usually treated with surgical clipping and ligation. Conventional catheter-based angiography may be necessary to diagnose more subtle aneurysms if MRI is negative, and some aneurysms can be treated using endovascular coils rather than open surgery.
4. **Intraparenchymal hemorrhage** describes bleeding directly into the brain parenchyma. The most common cause is hypertension; other causes include AVMs, coagulopathies, tumor, and trauma. Two-thirds of hypertensive bleeds occur in the basal ganglia, and patients often present in a coma; the other common locations for hypertensive bleeds are the brainstem and cerebellum. Awake patients may have contralateral hemiplegia and hemisensory deficits. Blood (white) is seen in the brain parenchyma and also may be seen in the ventricles. Surgery is reserved for large bleeds that are accessible.

Intraparenchymal bleeds from trauma are most commonly in the inferior frontal lobes and anteroinferior temporal lobes, where the brain rubs on the irregular surface of the skull base.

After a history of trauma, a dilated, unreactive pupil (i.e., "blown" pupil) on only one side most likely represents impingement of the ipsilateral third cranial nerve and impending uncal herniation due to increased intracranial pressure. Of the different intracranial bleeds, this is most commonly seen with epidural bleeds.

CASE SCENARIO: Why should you not do a lumbar puncture in the setting of trauma or evidence of increased intracranial pressure? You may cause uncal herniation and death. First

do a CT scan without contrast. If it is negative and the diagnosis remains unclear, then consider lumbar puncture (rarely indicated to detect blood products).

Fractures and trauma

Basilar (skull base) fractures are usually only treated when contaminated or persistent bleeding or cerebrospinal leaking occurs and have four classic signs:

1. Raccoon eyes: periorbital ecchymosis
2. Battle sign: postauricular ecchymosis
3. Hemotympanum: blood behind the eardrum
4. Cerebrospinal fluid otorrhea/rhinorrhea: clear fluid drains from ears or nose

Skull fractures of the calvaria (top of skull) are seen on CT scan (test of choice), generally as a linear or depressed fracture. Surgical repair is done only for contaminated fractures (cleaning and débridement), impingement on the brain parenchyma, or an open fracture with cerebrospinal fluid leak. Otherwise, such fractures can be observed and generally heal on their own.

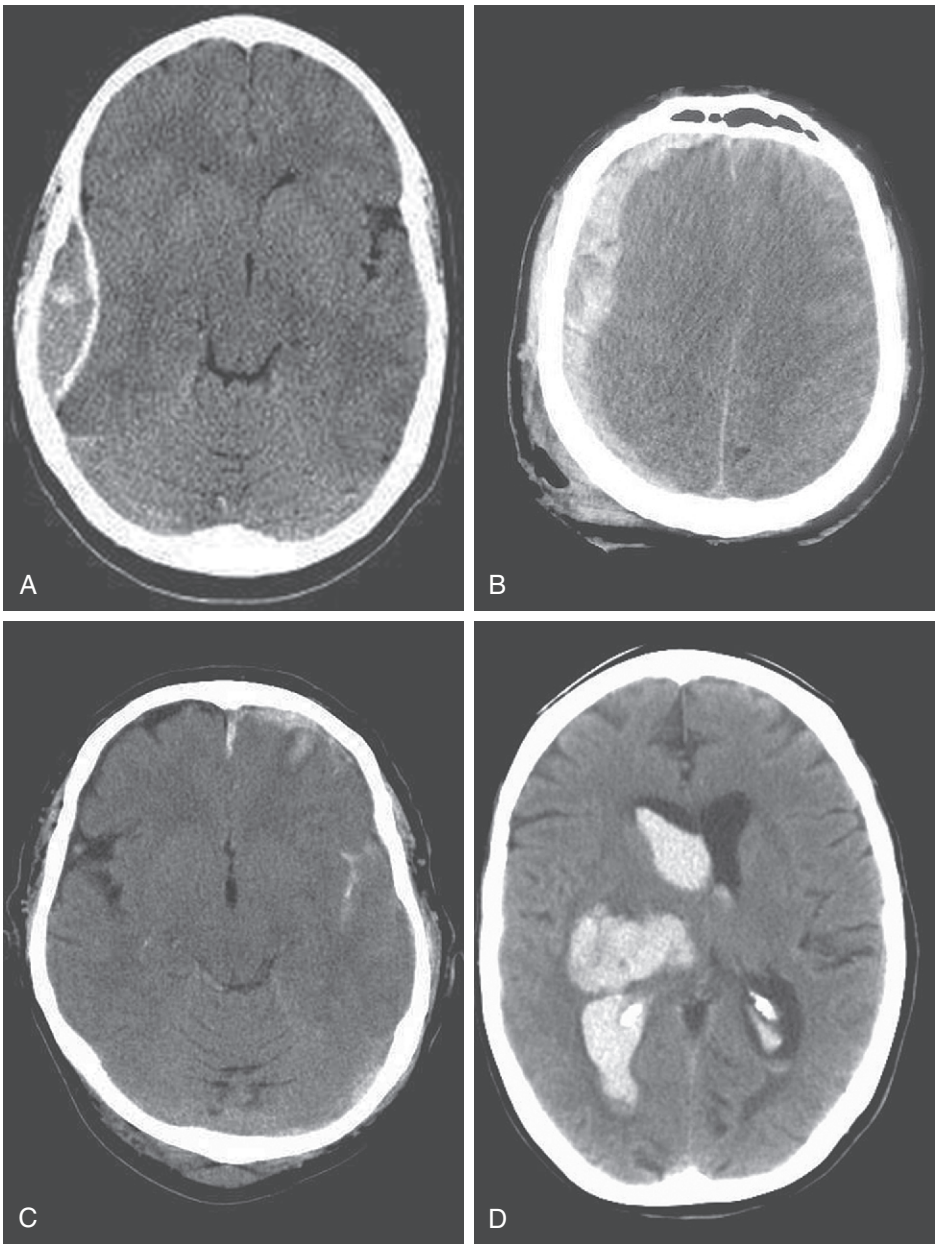

FIGURE 6-8 Computed tomography scans of intracranial hemorrhage. **A,** Epidural hematoma; **B,** subdural hematoma; **C,** subarachnoid hematoma; **D,** intracerebral hematoma. *(From DeLee JC, Drez D, Miller MD: DeLee and Drez's Orthopaedic Sports Medicine, 3rd ed. Philadelphia, Saunders, 2009.)*

Head trauma also may cause cerebral contusion or shear injury (i.e., *diffuse axonal injury*) of the brain parenchyma, both of which may not show up on a CT scan but may cause temporary or permanent neurologic deficits. They can be detected with MRI, but there is no treatment. MRI is used only for prognostic information in this setting.

Spinal cord trauma often manifests with "spinal shock" (loss of reflexes and motor function, hypotension). Order standard trauma radiographs (cervical spine, thorax, pelvis), as well as additional CT scans based on physical exam. Give IV corticosteroids immediately, which may improve outcome. Surgery is done for incomplete neurologic injury (with some residual function) with external bony compression of the cord (e.g., subluxation, bone chip).

Miscellaneous

Increased intracranial pressure (ICP), also known as intracranial hypertension (normal ICP = 5–15 mm Hg), should be suspected in patients with *bilaterally dilated and fixed pupils*. Other signs and symptoms include headache, *papilledema,* nausea and vomiting, and *mental status changes.* Look also for the classic *Cushing triad* (increasing blood pressure, bradycardia, and respiratory irregularity), which indicates very high ICP. The first step is to put the patient in reverse Trendelenburg position (head up) and intubate. Once intubated, the patient can be hyperventilated to lower the ICP rapidly. Hyperventilation decreases intracranial blood volume by causing cerebral vasoconstriction. If this maneuver does not lower ICP, *mannitol* diuresis can be tried to lessen cerebral edema. Furosemide is also used, but it is less effective. Decompressive craniotomy (burr holes) is a last resort. Prophylactic anticonvulsants are controversial.

CASE SCENARIO: How should hypertension be treated in the setting of increased intracranial pressure? It generally should not be treated! Cerebral perfusion pressure = blood pressure – ICP. The body, therefore, reflexively causes hypertension in the setting of increased ICP to maintain cerebral perfusion.

Subacute spinal cord compression is often due to metastatic cancer but also may result from a primary neoplasm, subdural, or epidural abscess or hematoma (especially after a lumbar tap or epidural/spinal anesthesia in patients with a bleeding disorder or taking anticoagulation). Patients present with local spinal pain (especially with bone metastases) and neurologic deficits below the lesion (e.g., hyperreflexia, Babinski sign, weakness, sensory loss). The first step is to give high-dose IV *corticosteroids.* Then order MRI of the appropriate spinal level. Give radiotherapy if radiosensitive metastases are present. Alternatively, surgical decompression can be done for radioresistant tumors. For hematoma or subdural/epidural abscess (seen especially in diabetics, usually due to *S. aureus*), surgery is indicated for decompression and drainage.

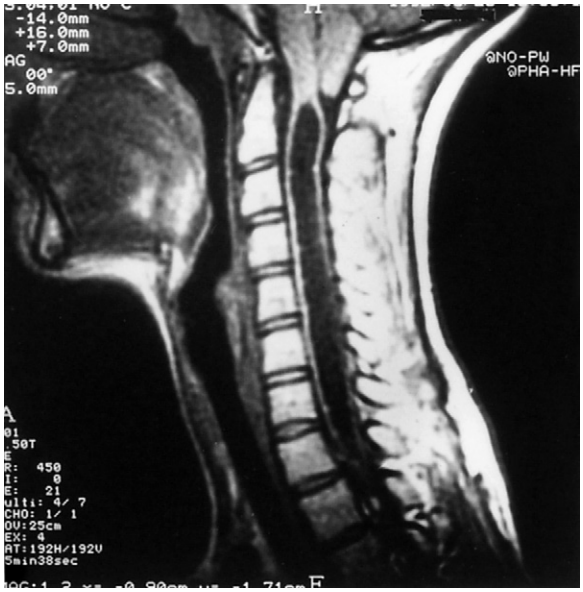

FIGURE 6-9 Magnetic resonance image demonstrates a large syringomyelic cavity in the cervical cord. *(From Bradley WG, Daroff RB, Fenichel G, Jankovic J: Neurology in Clinical Practice, 5th ed. Philadelphia, Butterworth-Heinemann, 2008.)*

Syringomyelia is a central pathologic cavitation of the spinal cord, usually in the cervical or upper thoracic region. It may be idiopathic, the result of trauma, or congenital cranial base malformations (e.g., *Arnold-Chiari malformation* or Dandy-Walker syndrome). The classic presentation is a *bilateral loss of pain and temperature sensation below the lesion in the distribution of a "cape"* secondary to involvement of the lateral spinothalamic tracts. The cavitation in the cord gradually widens to involve other tracts, causing motor and sensory deficits. MRI is the diagnostic study of choice (Fig. 6-9), and treatment is typically surgical (creation of a shunt).

CASE SCENARIO: In what condition of the elderly is the classic triad of ataxia, dementia, and urinary incontinence seen? Normal-pressure hydrocephalus, which is a type of communicating hydrocephalus that sometimes requires treatment with ventricular shunt tube placement.

OPHTHALMOLOGY

Conjunctivitis

Conjunctivitis causes conjunctival vessel hyperemia and eye irritation. If vision loss occurs, think of more serious conditions. Classic causes are listed below Table 6-4.

Table 6-4 Overview of Conjunctivitis

ETIOLOGIC CATEGORY	UNIQUE SIGNS/SYMPTOMS	TREATMENT
Allergic	Itching; bilateral, seasonal, long duration	Topical antihistamine (e.g., cetirizine) or mast cell inhibitors (cromolyn)
Viral (especially adenovirus)	Preauricular adenopathy; highly contagious (history of infected contacts); one eye affected and then the other, clear, watery discharge	Supportive treatment; hand washing (prevents spread)
Bacterial	Purulent discharge; look for its presentation in a neonate	Topical antibiotics (e.g., fluoroquinolone); *Chlamydia* or gonorrheal infections necessitate systemic antibiotics as well

Glaucoma

Glaucoma is best thought of as ocular hypertension with corresponding optic nerve damage and loss of visual field. Two types exist:

1. **Open-angle glaucoma** accounts for 90% of the cases of glaucoma. Risk factors include increased intraocular pressure, thin central corneal thickness, positive family history, increased age, and African-American ancestry. It is *painless* and has no acute attacks. The only signs are elevation of intraocular pressure (usually 20–30 mm Hg), a *gradually progressive visual field loss* (starts in the periphery), and *optic nerve changes* (increased cup-to-disc ratio on funduscopic exam, with possible disc hemorrhage). Treat with several different types of medications, including β-blockers (timolol), prostaglandins (latanoprost), acetazolamide, and mannitol, and/or surgery.
2. **Closed-angle glaucoma** can be either chronic or acute. Acute angle closure is the rare type that everyone worries about. It manifests with *sudden ocular pain,* seeing *haloes around lights, red eye,* very high intraocular pressure (>30 mm Hg), nausea and vomiting, headache, sudden decrease in vision, and a *fixed, middilated pupil.* Treat immediately with IV mannitol or topical pressure-lowering drops (pilocarpine, β-blockers, acetazolamide) to break the attack. Then use laser or surgery to prevent further attacks *(peripheral iridotomy).* Very rarely, anticholinergic medications can cause an attack of closed-angle glaucoma in a susceptible, previously untreated patient. Medications do not cause attacks in patients with open-angle glaucoma or surgically treated closed-angle glaucoma.

Steroids, whether topical or systemic, used long term can cause glaucoma and cataracts. Topical steroids can worsen fungal infections. For board purposes, do not give steroid eye drops. Refer the patient to an ophthalmologist if you think that it is indicated.

CASE SCENARIO: A patient complains of eye pain and has a branching dendritiform ulcer over his cornea with terminal bulbs that stain green with fluorescein. What condition does the patient have? Herpes simplex keratitis. Refer to an ophthalmologist promptly for antiviral treatment (e.g., oral acyclovir, topical ganciclovir, topical trifluridine).

Vision Loss

Sudden unilateral, *painless* vision loss includes the following differential diagnoses:

1. **Central retinal artery occlusion.** The funduscopic appearance is classic, with retinal whitening and a cherry-red spot in the macula (Fig. 6-10). The most common cause is emboli (from carotid plaque or heart); treatment is generally supportive unless the cause is temporal arteritis (in which case you should give high-dose corticosteroids immediately to decrease risk of vision loss in the other eye).
2. **Central retinal vein occlusion.** The funduscopic appearance is classic "blood and thunder," with tortuous veins and retinal hemorrhages (Fig. 6-11). No satisfactory treatment is available. The most common causes are hypertension, diabetes, glaucoma, and increased blood viscosity (e.g., leukemia). Complications (vision loss, glaucoma) are related to ischemia and neovascularization.
3. **Retinal detachment.** The history usually includes seeing *"floaters"* and *flashes of light.* Patients often describe the presentation as a *"curtain or veil coming down in front of my eye."* This history should prompt immediate referral to an ophthalmologist, as prompt surgery (reattachment of the retina) may save the patient's vision.
4. **Vitreous hemorrhage.** The most common cause is bleeding from areas of neovascularization, classically in *diabetics.* The condition sometimes resolves or may improve after surgical vitrectomy.
5. **Optic neuritis/papillitis.** This condition takes at least a few hours to develop and is usually painful, but it may occur quickly and without pain. Sometimes symptoms are bilateral. Presentation in a 20–40-year-old woman should raise suspicion for multiple sclerosis, particularly if there is history of prior neurologic deficits (or Lyme disease with the appropriate history). Worry about a tumor in male patients with bilateral optic nerve edema and signs of intracranial hypertension or other neurologic deficits. Disc margins are typically blurred on funduscopic exam, as in papilledema.
6. **Stroke or transient ischemic attack:** see Table 6-5 for visual pathway lesions.

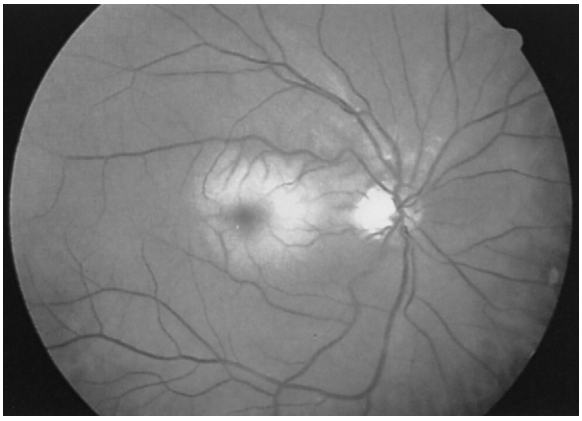

FIGURE 6-10 Central retinal artery occlusion. Note the cherry-red spot in the center of the macula, with surrounding whitening of the retina. *(From Bradley WG, Daroff RB, Fenichel G, Jankovic J: Neurology in Clinical Practice, 5th ed. Philadelphia, Butterworth-Heinemann, 2008.)*

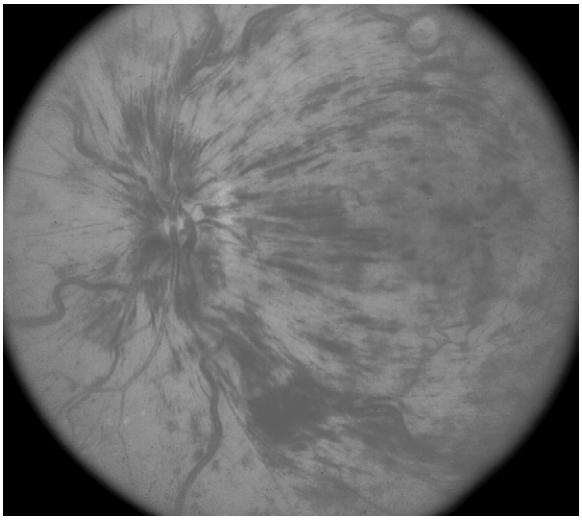

FIGURE 6-11 Central retinal vein occlusion. Note the dramatic retinal hemorrhages in all four quadrants. The veins are dilated and tortuous. The optic disc is blurred with blood from peripapillary hemorrhage. *(From Palay DA, Krachmer JH: Primary Care Ophthalmology, 2nd ed. Philadelphia, Mosby, 2005.)*

Table 6-5 Visual Pathway Lesions

VISUAL FIELD DEFECT	LOCATION OF LESION
Right anopsia (monocular blindness)	Right optic nerve
Bitemporal hemianopsia	Optic chiasm (classically due to pituitary tumor)
Left homonymous hemianopsia	Right optic tract
Left upper quadrant anopsia	Right optic radiations in the right temporal lobe
Left lower quadrant anopsia	Right optic radiations in the right parietal lobe
Left homonymous hemianopsia with macular sparing	Right occipital lobe (from posterior cerebral artery occlusion)

Sudden unilateral, *painful vision loss*:

1. **Trauma:** The history gives it away. Encourage use of goggles or safety glasses during athletics and work. With chemical burns to the eye (acid or alkaline), the key to management is *copious irrigation with the closest source of water* (tap water is fine). Check pH again after irrigation. The longer you wait, the worse the prognosis; do not get additional history in this instance. *Alkali burns have a worse prognosis* because they tend to penetrate more deeply into the eye.
2. **Closed-angle glaucoma:** See previous discussion for presenting signs and symptoms and treatment.
3. **Optic neuritis:** Usually painful, as described earlier.
4. **Migraine headache:** Common! Look for nausea, vomiting, and aura.

Sudden bilateral loss of vision is rare, but consider the following possible causes:

1. **Toxins:** The classic example is *methanol poisoning,* usually seen in alcoholics.
2. **Exposure to ultraviolet light** can cause keratitis (corneal inflammation) with resultant pain, foreign body sensation, red eyes, tearing, and decreased vision (usually some vision remains). Patients have a history of *welding, using a tanning bed or sunlamp,* or *snow-skiing* ("snow-blindness"). Treat with a topical antibiotic, possibly also with an anticholinergic (cycloplegic) agent to reduce pain.
3. **Conversion reaction/hysteria/nonorganic**

Gradual-onset loss of vision, unilateral or bilateral, has a longer list for the differential diagnosis but is more common than sudden-onset vision loss:

1. **Cataracts** are the most common cause of a painless, slowly progressive loss of vision. Often bilateral, but one side may be worse than the other. Look for an opacified lens and the patient complaining of *"looking through a dirty windshield"* (Fig. 6-12). Treatment is surgical.
2. **Open-angle glaucoma:** See earlier discussion for specifics. Screen people older than 40, especially if they are Black or have a positive family history. This is the most common cause of irreversible blindness in African-Americans.
3. **Macular degeneration** is the most common cause of blindness in adults older than 60 years of age. Blindness is often bilateral, but one side may be worse than the other. The appearance of the fundus (the yellow-white deposits of drusen in the macular area) makes the diagnosis. No good treatment is available for the most common (dry-type) form, but high doses of vitamins A, C, and E and the minerals zinc and copper may delay progression. The less common wet-type form (10% of cases) of macular degeneration can be treated with antiangiogenic therapies.

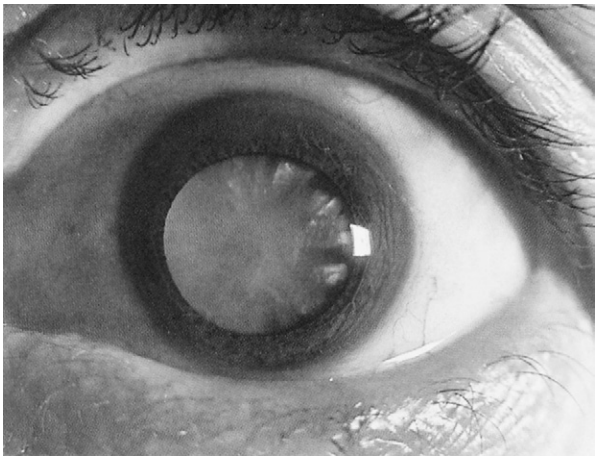

Figure 6-12 "Senile"-type cataract, which can occur at an earlier age in diabetic patients than in the normal population. *(From Forbes CD, Jackson WF: Endocrine, metabolic, and nutritional disorders. In Forbes CD, Jackson WF [eds]: Color Atlas and Text of Clinical Medicine. St. Louis, Mosby, 1993, pp 303-352.)*

FIGURE 6-13 Third cranial nerve palsy of the left eye. *(From Kliegman RM, Stanton BMD, St. Geme J, et al: Nelson Textbook of Pediatrics, 19th ed. Philadelphia, Saunders, 2011.)*

4. **Diabetes** is the most common cause of blindness in adults overall. Retinal/fundus changes include *dot-blot hemorrhages, cotton-wool spots, microaneurysms,* and *neovascularization.* Proliferative diabetic retinopathy (with neovascularization) is treated by a laser applied to the periphery of the whole retina (*panretinal photocoagulation*). Focal laser treatment is often done for diabetic macular edema (the laser is applied only to the affected area).
5. **Uveitis:** Look for association with autoimmune-type diseases. Screen children with *juvenile rheumatoid arthritis* regularly to detect uveitis. The usual treatment is topical steroids (treated by an ophthalmologist).
6. **Papilledema** by definition is optic nerve edema due to increased intracranial pressure (e.g., brain tumor, idiopathic intracranial hypertension [IIH]/*pseudotumor cerebri*).
7. **Optic neuritis** classically results from autoimmune-type conditions (most often multiple sclerosis), infections (viral, Lyme disease), or drugs (*ethambutol*).
8. **Infection of the cornea** (herpes keratitis, corneal ulcer, especially with contact lens wear) or retina (cytomegalovirus retinitis in AIDS), or orbital cellulitis.
9. **Direct insult to brain:** stroke, tumor, meningitis (see Table 6-5 for visual pathway information).
10. **Presbyopia:** between ages 40 and 50 years, the lens gradually *loses its ability to accommodate.* People need bifocals or reading glasses for near vision. This is a normal part of aging, not a disease.

Miscellaneous conditions

Effects of hypertension on the fundus include arteriolar narrowing, copper/silver wiring, cotton-wool spots, and optic nerve edema (with severe hypertension).

Hordeolum (stye) is a painful, red lump near the lid margin. Treat with warm compresses, lid scrubs.

Chalazion is a chronic, painless lump near the lid margin. Treat with warm compresses and lid scrubs; if this approach fails, treat with steroid injection or incision and drainage.

Ophthalmic herpes zoster infection should be suspected with involvement of the tip of the nose (Hutchinson sign) and/or medial eyelid with a typical zoster dermatomal pattern. Check the cornea with fluorescein dye. Treat with oral acyclovir, topical ganciclovir, or topical trifluridine and ophthalmologic referral.

Ophthalmologic cranial nerve (CN) palsies are usually due to *vascular complications of diabetes or hypertension* and resolve gradually in 3–6 months. In patients younger than 40, those without diabetes or hypertension, patients with multiple cranial nerve palsies, other neurologic deficits or severe pain, and patients who fail to improve within 8 weeks, order MRI and MRA of the brain. Look for a tumor or aneurysm in this setting.

1. **Oculomotor (CN3):** Eye is *"down and out"* and cannot do anything but move laterally (Fig. 6-13). Patients can have complete ptosis (lower eye lid) on the same side.

CASE SCENARIO: How can the pupil help you to decide between a serious and a benign cause of a third cranial nerve palsy? If the palsy is due to hypertension or diabetes, the pupil is usually normal. A "blown" (dilated, nonreactive) pupil is a medical emergency. The most likely cause is an aneurysm or tumor. Order MRI and MRA of the head.

2. **Trochlear (CN4):** When the gaze is medial, the patient *cannot look down* (vertical diplopia).
3. **Abducens (CN6):** The patient *cannot look laterally* with the affected eye (horizontal diplopia) (Fig. 6-14).

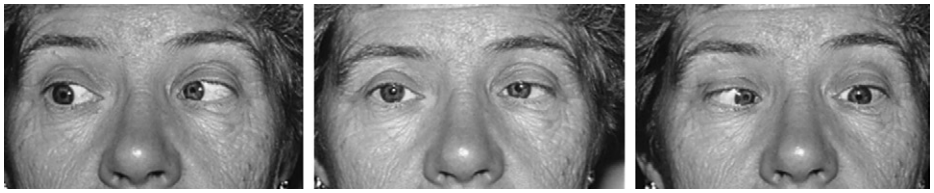

FIGURE 6-14 Left sixth cranial nerve paresis. Note the poor movement of the left eye in left gaze. *(From Palay DA, Krachmer JH: Primary Care Ophthalmology, 2nd ed. Philadelphia, Mosby, 2005.)*

4. **CN5 and CN7** palsies also affect the eye due to corneal drying (loss of corneal blink reflex). Use artificial tears/ointment and address the underlying cause, if possible.

ORTHOPEDIC SURGERY

Fractures

With any fracture, do a neurologic and vascular exam distal to the fracture site to determine whether there is any compromise of nerves or blood vessels (either may be an emergency). With a suspected or obvious fracture, get *two x-ray views* (usually anteroposterior and lateral) of the site, and include the joints above and below the suspected fracture site.

When a fracture is suspected clinically (severe pain, point tenderness, swelling) but the radiographs are negative, *treat conservatively as if the patient has a fracture.* Radiographs can be negative at first with smaller, nondisplaced fractures. Put the limb in a splint or even a cast, and tell the patient not to use it (no weight bearing) if symptoms are significant. If the suspected fracture is in the leg, the patient can use crutches. Repeat the radiograph in 1 week; evidence of a fracture is usually apparent by this time.

CASE SCENARIO: What fractures are associated with the highest mortality rate? Pelvic fractures. Most pelvic fractures occur in elderly people who fall down and have many coexisting health problems. Young people, in whom pelvic fractures usually are due to severe trauma, may bleed to death. If a patient is unstable, consider heroic measures such as military antishock trousers and external fixator. Consider bladder or urethral injury from pelvic fractures in the setting of hematuria or blood at the meatus. Cystogram or retrograde urethrogram can be performed for further evaluation (see urology section later).

In an open (compound) fracture, the skin is broken over the fracture site. In a closed fracture, the skin is intact. For open fractures, give broad-spectrum antibiotics, do surgical débridement, give tetanus vaccine, lavage fresh wounds (<8 hours old), and do an *open reduction with internal fixation* (i.e., cut open the skin in the operating room to align the fracture fragments under more direct visualization). The main risk in open fractures is infection. Closed fractures often can be treated with *closed reduction and casting* (i.e., pull on the limb to align the fracture fragments without cutting open the skin).

Compartment syndrome usually occurs after fracture, crush injury, burn, or other trauma or as a reperfusion injury (e.g., after revascularization procedure). The most common site is in the *calf.* Symptoms and signs include *pain at rest; pain on passive movement* (out of proportion to the injury); *paresthesias;* cyanosis or pallor; a *firm-feeling muscle compartment;* hypesthesia or numbness (decreased sensation and two-point discrimination); paralysis (late, ominous sign); and *elevated compartment pressure* (>30–40 mm Hg). The diagnosis is usually made clinically without the need to measure compartment pressure, though this is fairly accurate and confirmatory. Compartment syndrome is an emergency, and quick action can save an otherwise doomed limb. *Pulses are usually palpable* (or detectable with Doppler ultrasound) at the time of diagnosis. Treatment is prompt *fasciotomy* (incising the fascial compartment relieves the pressure). Untreated, this condition progresses to permanent nerve damage and muscle necrosis.

The classic clinical scenarios for compartment syndrome include supracondylar elbow fracture in children, proximal or midshaft tibial fractures, electrical burns, arterial or venous disruption, and revascularization procedures.

Reasons to do an open surgical reduction (closed reduction should be done for all other fractures) are intraarticular fractures or articular surface malalignment, open (compound) fractures, nonunion or failed closed reduction, compromise of blood supply or nerves (Table 6-6), multiple trauma (to allow mobilization at earliest possible point), and extremity function requiring perfect reduction (e.g., professional athlete).

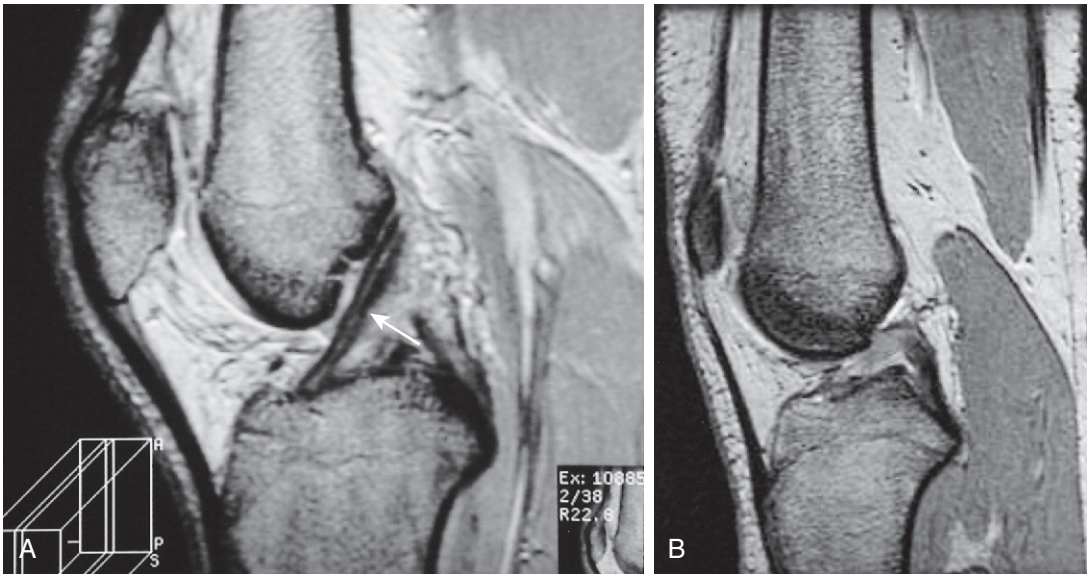

FIGURE 6-15 **A,** Normal anterior cruciate ligament (ACL) composed of separate anteromedial and posterolateral bundles. **B,** Sagittal image through the intercondylar region fails to demonstrate any normal anterior cruciate ligament indicating a chronic complete ACL rupture. *(From Adam A, Dixon AK, Grainger RG, Allison DJ: Grainger & Allison's Diagnostic Radiology, 5th ed. Philadelphia, Churchill Livingstone, 2008.)*

TABLE 6-6 Nerves Commonly Involved in Traumatic Injury

NERVE	MOTOR	SENSORY	WHEN CLINICALLY DAMAGED
Radial	Wrist extension	Back of forearm, back of hand (first 3 digits)	Humeral fracture (wrist-drop)
Ulnar	Finger abduction	Front and back of last 2 fingers on hand	Elbow dislocation (claw-hand)
Median	Pronation, thumb opposition	Palmar surface of hand (first 3 digits)	Carpal tunnel syndrome, humeral fracture
Axillary	Abduction/lateral rotation	Lateral shoulder	Upper humeral dislocation/fracture
Peroneal	Dorsiflexion/eversion	Dorsal foot and lateral leg	Knee dislocation (foot-drop)

Ligament injuries in the knee

Ligament injuries in the knee commonly cause pain, joint effusions, instability of the joint, and history of the joint's *"popping," "buckling," or "locking up."*

1. **Anterior cruciate ligament (ACL).** ACL tears are the most common. Perform the *anterior drawer test.* The knee is placed in 90 degrees of flexion and pulled forward (like opening a drawer). If the tibia pulls forward more than normal (e.g., more than the unaffected side), the test is positive and you have an ACL tear (Fig. 6-15).
2. **Posterior cruciate ligament (PCL).** Perform the *posterior drawer test.* Push the tibia back with the knee in 90 degrees of flexion. If the tibia pushes back more than normal, the test is positive and a PCL tear is present (Fig. 6-16).
3. **Medial collateral ligament (MCL).** Perform the *abduction or valgus stress test.* With the knee in 30 degrees of flexion, abduct the ankle while holding the knee. If the knee joint abducts to an abnormal degree, the test is positive and a medical compartment injury is present (Fig. 6-17).
4. **Lateral collateral ligament.** Perform the *adduction* or *varus stress test.* Adduct the ankle while holding the knee. If the knee joint adducts to an abnormal degree, the test is positive and lateral compartment injury is present.

- MRI or arthroscopy can be used to look for other injuries or confirm a diagnosis in doubt.
- Treatment may be nonsurgical (older patient, nonathlete, minor injury) or surgical (young patient, athlete, severe injury).

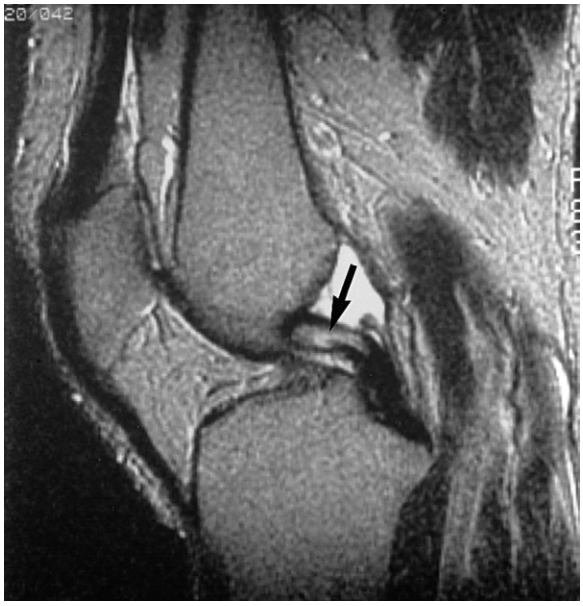

FIGURE 6-16 Posterior cruciate ligament tear. Sagittal T_2-weighted image shows abnormal bright signal *(arrow)* within normally dark posterior cruciate ligament. Fluid is also seen around proximal extent of partially torn posterior cruciate ligament. *(From Canale ST, Beaty JH: Campbell's Operative Orthopaedics, 11th ed. Philadelphia, Mosby, 2008.)*

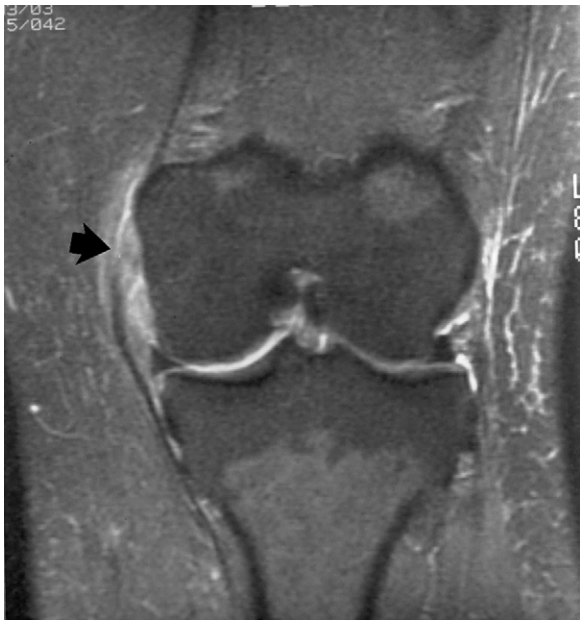

FIGURE 6-17 Medial collateral ligament tear. Complete disruption of proximal medial collateral ligament *(arrow)* is shown in coronal fat-suppressed, proton density–weighted image. *(From Canale ST, Beaty JH: Campbell's Operative Orthopaedics, 11th ed. Philadelphia, Mosby, 2008.)*

CASE SCENARIO: What is the "unhappy triad" knee injury? Damage to *ACL, MCL, and medial meniscus*. Classically this triad occurs when an extended knee joint is hit from the side and the knee is pushed medially while the foot is planted.

Pain in the anatomic snuffbox after trauma (e.g., fall on an outstretched hand, especially in young adults) is usually a scaphoid bone fracture.

After a fall on an outstretched hand, the most likely fracture in older adults is a Colles fracture (distal end of radius).

Disk herniation

Lumbar disk herniation is a common correctable cause of low back pain. Look for sciatica (not just back pain) with the straight leg raise test. The most common site is the L5-1 disk; the second most common site is the L4-L5 disk.

1. **L5-S1 disk herniation** usually affects the S1 nerve root: decreased ankle jerk, weakness of plantar flexors in the foot, pain from the midgluteal area to the posterior calf (i.e., sciatica).

2. **L4-L5 disk herniation** usually affects the L5 nerve root: decreased biceps femoris reflex, weakness of foot extensors, and pain in the hip or groin.
   - ❍ Diagnosis is confirmed with MRI or CT myelogram.
   - ❍ Conservative treatment works in 90% of cases and includes rest and analgesics, followed by physical therapy. Surgical treatment (diskectomy) is an option if conservative treatment fails.
   - ❍ Cervical disk disease (classic symptoms include neck pain and cervical radiculopathy) is less common than lumbar disk diseases. Diagnosis can be confirmed with MRI. The C6-C7 disk is most commonly affected, typically with C7 nerve root involvement. Look for *decreased triceps reflex/strength* and *weakness of forearm extension.*

Miscellaneous conditions

Spinal stenosis is another cause of back pain that usually manifests in the elderly and is due to degenerative changes in the spine. Patients may complain of pain with activity that is relieved by rest (sometimes called *"neurogenic" claudication*). Diagnosis is confirmed with MRI or CT. Treatment is conservative with physical therapy and nonsteroidal antiinflammatory drugs (NSAIDs). Surgery (spinal decompression with laminectomy) is reserved for cases that fail conservative management.

Charcot joints and neuropathic joints are most commonly seen in *diabetes* and sometimes by other conditions causing peripheral neuropathy (e.g., tertiary syphilis). Lack of proprioception causes gradual arthritis/arthropathy and joint deformity. Order radiographs for any (even minor) trauma in neuropathic patients who may not feel even a severe fracture. MRI may be necessary for evaluation secondary to complex joint derangement on radiograph.

With a posterior knee dislocation, worry about vascular injury. Perform CT or magnetic resonance angiography (MRA) if pulses are asymmetric.

The most common type of bone tumor is metastatic (most commonly from the breast, lung, or prostate).

The most common cause of a pathologic fracture is *osteoporosis* (especially in elderly, thin women).

The most common pathogenic organism in osteomyelitis is *S. aureus,* but think of gram-negative organisms in immunocompromised patients and IV drug abusers, as well as *Salmonella* spp. in sickle cell disease. Aspirate the bone and do a Gram stain and culture and sensitivity of the sample, as well as blood cultures and complete blood cell count with differential if you are suspicious.

Septic arthritis is also most commonly due to *S. aureus,* but in a sexually active younger adult, suspect *Neisseria gonorrhoeae.* Aspirate the joint (arthrocentesis) and do a Gram stain and culture and sensitivity testing of the joint fluid, as well as blood cultures, complete blood cell count with differential, and urethral cultures when appropriate if you are suspicious.

UROLOGY

Testicular torsion versus epididymitis (Table 6-7)

Table 6-7 Overview of Testicular Torsion versus Epididymitis

	TESTICULAR TORSION	EPIDIDYMITIS
Age	<30 yr (classically, adolescent)	>30 yr
Appearance	Testis may be elevated into inguinal canal; swelling	Swollen testis, overlying erythema, positive urinalysis, urethral discharge/urethritis, prostatitis
Prehn sign	Pain stays the same or worsens	Pain decreases with testicular elevation
Ultrasound findings	No testicular blood flow	Normal testicular blood flow
Treatment	Immediate surgery to salvage the testicle; orchioplexy for both testes	Antibiotics*

*In men <50 yr, commonly due to chlamydial infection or gonorrhea; treat accordingly. In men >50 yr, commonly due to urinary tract infection; treat with trimethoprim-sulfamethoxazole or ciprofloxacin.

Testicular cancer

Testicular cancer usually manifests as a *painless mass in a young man* (age 20–40 years). The main risk factor is *cryptorchidism* (40-fold higher risk). Roughly 90% are germ cell tumors; the most common type is *seminoma.* Testicular cancer is generally treated with orchiectomy and radiation; if the disease is widespread, use chemotherapy. α-Fetoprotein is a tumor marker for yolk sac tumors, and human chorionic gonadotropin is a marker for choriocarcinoma. Leydig cell tumors may secrete androgens and leading

onset of precocious puberty. The first site of metastasis is often *retroperitoneal lymph nodes,* as the testicular veins (and accompanying lymphatics) drain to the inferior vena cava (right) or renal vein (left).

Mumps

Remember mumps as a cause of orchitis (painful, swollen testis, usually unilateral, in a postpubertal male). The best treatment is *prophylaxis* (immunization). Orchitis rarely causes sterility, because it is usually unilateral.

Benign prostatic hyperplasia

Symptoms include urinary hesitancy, intermittency, terminal dribbling, decreased size and force of stream, sensation of incomplete emptying, nocturia, urgency, dysuria, and frequency of urination. Benign prostatic hyperplasia (BPH) may result in *urinary retention, urinary tract infections, hydronephrosis,* and even permanent kidney damage and/or failure in severe cases. PSA is elevated in 30–50% of patients. Drug therapy for BPH is started when the patient becomes symptomatic and includes α_1-blockade (e.g., *tamsulosin, prazosin*) and antiandrogens (e.g., *finasteride*). Saw palmetto is a dietary supplement that may help relieve symptoms. Transurethral resection of the prostate (TURP) is used for more advanced cases, especially those associated with recurrent urinary tract infections, acute urinary retention, and hydronephrosis or kidney damage from reflux. Prostatectomy may also be used, but it carries a higher risk of morbidity and is usually not the preferred treatment.

With acute urinary retention (pain, palpation of full bladder on abdominal exam, history of BPH, no urination in past 24 hours), the first step is to *empty the bladder.* If you cannot pass a regular Foley catheter, use a firm-tipped catheter (coudé catheter) or do a suprapubic tap/place a *suprapubic Foley catheter.* Then address the underlying cause.

Erectile dysfunction

Erectile dysfunction (impotence) is most commonly caused by *vascular disease.* Medications are also a common culprit (especially *antihypertensives* and *antidepressants*). Diabetes can be a vascular (increased atherosclerosis) or neurogenic cause of erectile dysfunction. Patients undergoing dialysis and patients with spinal cord injury also commonly have erectile dysfunction. Remember "point and shoot": Parasympathetic nerves mediate erection, sympathetic nerves mediate ejaculation.

History often gives you a clue if the cause of erectile dysfunction is psychogenic. Look for *selective dysfunction* (e.g., the patient has normal erections when masturbating, but not with his wife) and stress, anxiety, or fear.

CASE SCENARIO: What does a normal pattern of nocturnal erections mean in a patient with erectile dysfunction? This finding essentially rules out a physical cause for the erectile dysfunction.

In all trauma patients, look for signs of *urethral injury* (high-riding ballottable prostate, blood at the urethral meatus, severe pelvic fracture, scrotal or perineal ecchymosis) before trying to pass a Foley catheter. If any of these signs are present, do not try to pass a Foley catheter until you have ruled out a urethral injury, which is a contraindication to a Foley catheter.

CASE SCENARIO: What test should be ordered in the setting of possible urethral injury? A *retrograde urethrogram*. A contrast agent is injected backward through the urethra to look for a leak or tear.

Hydrocele versus varicocele

Hydrocele represents a remnant of the processus vaginalis and *transilluminates.* It generally causes no symptoms and requires no treatment. A varicocele is a *dilatation of the pampiniform venous plexus* (described as a "bag of worms" on physical exam, usually on the *left*), does not transilluminate, *disappears in the supine position,* and may be a cause of *male infertility* or pain (in which case it is surgically treated). Diagnosis of either can be confirmed using ultrasound, which can also help exclude other causes of palpable mass.

Renal stones

Renal stones (nephrolithiasis): The risk is increased with dehydration. Patients present with *severe, intermittent, colicky, unilateral flank and/or groin pain,* and, in most cases, nausea and vomiting. Patients

with "renal colic" classically cannot get comfortable and often move about while trying to, whereas patients with peritonitis often lie still. Look for *hematuria* on urinalysis; 85% of stones show up on abdominal radiograph, but CT scan of the abdomen without contrast (or IV pyelogram) is generally performed to confirm the presence of a stone. Most cases are idiopathic and should be treated with hydration and pain control (to see if the stone will pass). If the stone does not pass, the patient needs shock wave lithotripsy or surgery (preferably endoscopic). Whenever possible, check stone composition, which can give a clue to the cause:

1. **Calcium stones** (75%): usually idiopathic but look for *hypercalcemia* (typically due to hyperparathyroidism or malignancy) or history of small bowel bypass (calcium oxalate stones).
2. **Struvite/magnesium-ammonium-phosphate stones** (10%): due to urinary tract infection (more common in women) with ammonia-producing bugs (*Proteus* spp., staphylococci). Look for staghorn calculi, which are large stones that fill up the renal pelvis and/or collecting system and form a "cast" of these structures.
3. **Uric acid stones** (10%): due to *hyperuricemia* and therefore associated with gout and leukemia treatment (allopurinol and IV fluids are often given before chemotherapy in leukemia to prevent stone formation). Uric acid stones usually dissolve with *alkalinization of the urine.*
4. **Cystine stones:** nearly diagnostic of *cystinuria*/aminoaciduria. Alkalinization of urine (i.e., getting to a pH of >7.5 with the use of penicillamine) is useful for patients with recurrent cystine stones.

CASE SCENARIO: Which type of stone classically cannot be seen on radiographs? Uric acid stones. These can be seen on CT scans, however.

CASE SCENARIO: A 32-year-old man has acute flank pain. An IV pyelogram reveals a 2-mm stone in the right ureter. How should you manage this patient? IV fluids and pain control. Let the patient pass the stone on his own. Do not be overly aggressive, because most small stones (<4–5 mm) pass spontaneously within 48 hours.

TRANSPLANT MEDICINE (Tables 6-8 and 6-9.)

Table 6-8 Indications for Organ Transplantation

ORGAN	MAJOR DISEASES
Heart	Coronary artery disease; cardiomyopathy (dilated, restrictive, hypertrophic); valvular heart disease; congenital heart disease
Lung	Congenital disease, emphysema, COPD, cystic fibrosis, idiopathic pulmonary fibrosis, primary pulmonary hypertension, α_1-antitrypsin deficiency
Liver	Chronic hepatitis B or C infection, alcohol-related liver disease, cryptogenic cirrhosis including NASH,* cholestatic liver disease, hepatocellular carcinoma, metabolic diseases, acute liver failure
Kidney	Glomerular diseases, diabetes mellitus, polycystic kidney disease, hypertensive nephrosclerosis, tubular and interstitial diseases, congenital and metabolic diseases
Pancreas	Diabetes mellitus, pancreatic cancer
Small bowel	Intestinal failure in which TPN can no longer be maintained

COPD, Chronic obstructive pulmonary disease; *NASH,* nonalcoholic steatohepatitis; *TPN,* total parenteral nutrition.

Table 6-9 Immunosuppressive Agents Following Transplantation

CLASS OF DRUG	MECHANISM OF ACTION	SIDE EFFECTS
Calcineurin inhibitors (cyclosporine, tacrolimus)	Inhibition of cytokine production from T cells	Nephrotoxicity, dyslipidemia, hypertension, hirsutism, hyperkalemia, gingival hyperplasia
Corticosteroids	Inhibition of cytokine production from T cells and antigen-presenting cells	Hypertension, mental status changes, dyslipidemia, impaired wound healing, hyperglycemia, Cushing syndrome, myopathy, osteoporosis, fluid retention
Antimetabolites (azathioprine, mycophenolate)	Antagonizes purine metabolism and/or synthesis	Nausea, vomiting, diarrhea, cytopenias, pancreatitis

Table 6-9 Immunosuppressive Agents Following Transplantation—cont'd

CLASS OF DRUG	MECHANISM OF ACTION	SIDE EFFECTS
Antibody induction (antithymocyte globulin, OKT-3, basiliximab, alemtuzumab)	Depletes and/or modulates T-cell function	Cytokine release syndrome, abdominal pain, cytopenias, dyspnea, hypertension, sepsis
Sirolimus	Blocks T- and B-cell activation by cytokines	Cytopenias, dyslipidemia, interstitial lung disease, peripheral edema, impaired wound healing

Renal transplantation

Renal transplantation is an option for many patients with end-stage renal disease. Living, related donors are best (siblings or parents), especially when human leukocyte antigen (HLA)-similar, but cadaveric kidneys are more common because of availability. Before the transplant, perform ABO blood typing and lymphocytotoxic (HLA) cross-matching.

- A transplanted kidney is placed in the iliac fossa (for easy biopsy access in case of a problem, as well as for technical reasons). Usually the recipient's kidneys are left in place to reduce morbidity.
- Unacceptable kidney donors: newborns, people older than 70, and people with a history of certain infections (e.g., AIDS, hepatitis), any disease with possible renal involvement (e.g., diabetes, hypertension, lupus erythematosus), or malignancy.

Transplant rejection

1. **Hyperacute rejection:** Occurs within minutes to hours and is due to *preformed cytotoxic antibodies* against donor kidney (occurs with ABO mismatch and other preformed antibodies). Classic description: Surgery is completed, vascular clamps are released, and the kidney quickly turns bluish-black. Treat by removing the kidney. Repeat transplant procedure new kidney.
2. **Acute rejection:** *T cell–mediated* rejection that presents within *days to weeks* with fever, oliguria, weight gain, tenderness and enlargement of the graft, hypertension, and/or renal function lab derangement. Treat by increasing steroids or using antithymocyte globulin (ATG) or other immunosuppressants. Accelerated rejection occurs over the *first few days* and is felt to reflect *reactivation of previously sensitized T cells.*
3. **Chronic rejection:** Occurs over *months to years* and is believed to be *T cell– and/or antibody-mediated.* Late cause of renal deterioration presenting with *gradual decline in kidney function, proteinuria, and hypertension.* Treatment is supportive and not effective, but the graft may survive for several years before it gives out completely. If possible, retransplant with a new kidney.

- Follow serum creatinine to assess asymptomatic rejection.
- Cyclosporine causes nephrotoxicity, which can be difficult to distinguish from graft rejection clinically. When in doubt, drug levels are checked and a biopsy and/or ultrasound scan of the graft should be done. Practically speaking, if you increase the immunosuppressive dose, acute rejection should decrease, whereas cyclosporine toxicity will stay the same or get worse.
- The risks of immunosuppression include infection (with common and strange organisms seen in patients with AIDS) and cancer (especially lymphomas).

VASCULAR SURGERY

Carotid artery stenosis

The classic presentation is that of a transient ischemic attack (TIA), typically *amaurosis fugax* or sudden onset of transient, unilateral blindness, sometimes described as a "shade being pulled over one eye." Patients may have a *carotid bruit.* If a bruit is heard or the patient has a TIA, ultrasound imaging of the carotid arteries should be done to determine whether carotid stenosis is present. Cerebral angiography is the gold standard, but it is invasive, costly, and associated with complications.

CASE SCENARIO: What is the best treatment for a patient with a TIA and carotid stenosis with <50% blockage? Aspirin and medical management of atherosclerosis risk factors.

The management of carotid stenosis depends on degree of stenosis, whether the patient is symptomatic, gender, and age (Table 6-10). In asymptomatic patients, carotid endarterectomy (CEA) is recommended in men younger than age 75 if the extent of blockage is >60%. CEA is not recommended for

asymptomatic women. For symptomatic patients, CEA is recommended if blockage due to stenosis is >50% in men and >70% in women.

Table 6-10 Carotid Stenosis Management

	DEGREE OF STENOSIS		
CLINICAL CATEGORY	**<50%**	**50-69%**	**70-99%**
Asymptomatic	Medical management	Men: CEA if stenosis >60% and age <75 yr; otherwise, medical management Women: medical management	Men <75 yr: CEA Women: medical management
Symptomatic	Medical management	Men: CEA Women: medical management	Men: CEA Women: CEA

Data from Ferri FF: Ferri's Clinical Advisor 2012. Philadelphia, Mosby, 2012.
CEA, Carotid endarterectomy.

- Patients should not undergo CEA after a stroke that leaves them severely disabled because they will receive no benefit; the damage is already done. Nor should patients undergo CEA during a TIA or stroke in evolution; CEA is an elective, not emergent, procedure.
- Carotid stenosis and peripheral vascular disease (PVD) are generalized markers for atherosclerosis. Almost all patients have significant coronary artery disease (CAD). In fact, perioperative myocardial infarction (MI) is the most common cause of death in patients undergoing vascular surgery. Make sure to evaluate and medically manage risk factors for atherosclerosis in all "vasculopaths."

Aortic abnormalities

Abdominal aortic aneurysm: Look for a *pulsatile abdominal mass*, which may cause abdominal pain. If pain is present, suspect possible rupture of the AAA, although an unruptured AAA may cause some pain. CT scan is usually used for initial evaluation. Management of AAA depends on the size.

- AAA <4 cm: monitor by ultrasound imaging or CT every 2–3 years
- AAA 4–5.4 cm: monitor by ultrasound or CT every 6–12 months
- AAA ≥5.5 cm surgical repair should be performed. Some patients with AAA <5.5 cm may also benefit if it is rapidly enlarging. Surgical correction is generally advised, with either stent-graft placement or open surgical repair.

CASE SCENARIO: What should you do with a patient who has a pulsatile abdominal mass and hypotension? Prepare the patient for emergent laparotomy (get CT scan or US to confirm the diagnosis if patient is stable). This combination of findings suggests a ruptured AAA (mortality rate = roughly 90%).

Aortic dissection: Aortic wall splits and blood dissects in between layers of the media in the arterial wall. Classically causes a "tearing" or "ripping" type of chest pain that may radiate to the back and is generally seen in the setting of *hypertension* (e.g., whether essential or induced by cocaine) or *Marfan syndrome*. When this entity is suspected clinically, a CT scan of the chest (and possibly abdomen and pelvis) with IV contrast should be ordered. Treatment depends on the clinical classification. A dissection involving the ascending aorta and/or aortic arch (Stanford type A and DeBakey types I and II) is treated with immediate surgery (≤5% of patients survive 1 year without surgery). A dissection that spares the ascending aorta and arch (Stanford type B and DeBakey type III) typically begins just beyond the origin of the left subclavian artery in the isthmus of the aorta/proximal descending thoracic aorta and extends over a variable distance (may stop in the thoracic aorta or extend into the abdominal aorta and its branches). These types of dissections that spare the aortic arch and ascending aorta are managed medically with antihypertensives (>70% of patients survive more than 1 year without surgery), in the absence of any signs of impending rupture or end-organ ischemia from vascular compromise (which would indicate the need for surgical intervention). An aortic dissection may or may not be associated with an aneurysm (the term "dissecting aneurysm" is often misused, because many aortic dissections do not have aneurysmal dilatation associated with them).

Miscellaneous conditions

Claudication: Pain in the lower extremity (usually) brought on by exercise and relieved by rest. Claudication is an indicator of severe atherosclerotic disease. Associated physical findings include *cyanosis*

(with dependent rubor); atrophic changes (*thickened nails, loss of hair, shiny skin*); *decreased temperature;* and *decreased (or absent) distal pulses.* The best treatment is conservative (smoking cessation, exercise, control of cholesterol, diabetes, and hypertension). β-Blockers may theoretically worsen claudication (due to β_2 receptor blockade), but affected patients (who almost always have associated coronary artery disease) may benefit from the cardioprotection of β-blockers due to prior myocardial infarction.

- If claudication progresses to rest pain (forefoot pain, generally at night, relieved by hanging the foot over the edge of the bed) or the patient cannot continue current lifestyle or work obligations, advise a revascularization procedure (angioplasty and/or bypass graft).
- Severe pain in the foot of sudden onset with no previous history of foot pain, trauma, or any associated chronic physical findings is generally more serious and may represent an embolus. Look for atrial fibrillation.

CASE SCENARIO: What is Leriche syndrome? Claudication in the buttocks, buttock atrophy, and impotence in men. It is a classic marker for aortoiliac occlusive disease. Patients usually benefit from an aortoiliac bypass graft.

Chronic mesenteric ischemia: The classic patient has a long history of postprandial abdominal pain (i.e., *"intestinal angina"*), which causes a "fear" of food that results in weight loss. This is a difficult diagnosis because the disorder classically manifests in patients older than 50 who have other problems that may cause similar symptoms (e.g., ulcers, pancreatic or stomach cancer). Look for a history of extensive atherosclerosis (previous MI or stroke, known coronary artery disease or peripheral vascular disease with several risk factors), *abdominal bruit,* and no jaundice (the presence of jaundice should steer you toward pancreatic cancer). Most patients get a CT scan of the abdomen, which is negative for tumor and will demonstrate atherosclerotic disease. The diagnosis is confirmed with CT/MRA. Patients should be treated surgically with revascularization (angioplasty with stent and/or surgical bypass) because of the risks of bowel infarction and malnutrition.

After penetrating trauma in an extremity (or iatrogenic damage), patients may develop an arteriovenous fistula. An important clue to this entity is a *bruit* over the area or a *palpable pulsatile mass* (pseudoaneurysm). Diagnosis can be confirmed with ultrasound or MRA/CT. Arteriovenous fistulas can be left alone if they are small, but surgical correction is often necessary.

Venous insufficiency: Generally refers to the lower extremities. Look for a history of deep venous thrombosis; chronic swelling in the extremity; pain, fatigability, and heaviness, all of which are *relieved by elevating the leg;* and/or *varicose veins.* Patients may have increased skin pigmentation around the ankles with skin breakdown and ulceration (venous stasis ulcer, classically over the *medial malleolus*) (Fig. 6-18). Ultrasound can help define whether the disease involves the superficial or the deep venous system (or both). The initial treatment is conservative: elastic compression stockings, elevation with minimal standing, and treatment of any ulcers with cleansing, wet-to-dry dressings, and antibiotics (if cellulitis is present).

Superficial thrombophlebitis: Patients have *localized leg pain with superficial, cordlike induration, reddish discoloration,* and mild fever. This should not be confused with deep venous thrombosis, because superficial thrombophlebitis generally does not cause pulmonary emboli and patients do not need anticoagulation. Many patients have associated varicose veins. Diagnosis can be confirmed with ultrasound imaging, if necessary. Medical therapy is used if the pain is mild or the patient does not want surgery; use *NSAIDs.* The pain generally subsides in a few days on its own. Surgical thrombectomy under local anesthesia is necessary for more severe cases that fail to respond to nonsurgical treatment.

Subclavian steal syndrome: Usually due to left subclavian artery obstruction/stenosis proximal to the left vertebral artery origin (85% of cases are left-sided). To get blood to an exercising arm, blood is "stolen" from the vertebrobasilar system. Blood flows retrograde through the vertebral artery into the distal subclavian artery instead of forward into the brainstem. The patient develops *central nervous system symptoms* (syncope, vertigo, confusion, ataxia, dysarthria) and unilateral *upper extremity claudication.* Treat with surgical bypass and/or stent placement.

Thoracic outlet syndrome: May be due to a cervical rib (a normal variant) or muscular hypertrophy that compromises subclavian vessel blood flow. Patients have unilateral *upper extremity claudication.* Confirm the diagnosis with angiography. Treat with surgery (e.g., rib resection).

CASE SCENARIO: What is the easy way to tell the difference between thoracic outlet syndrome and subclavian steal syndrome using only the patient history? Subclavian steal causes central nervous system symptoms; thoracic outlet syndrome does not. Both can cause unilateral upper extremity claudication.

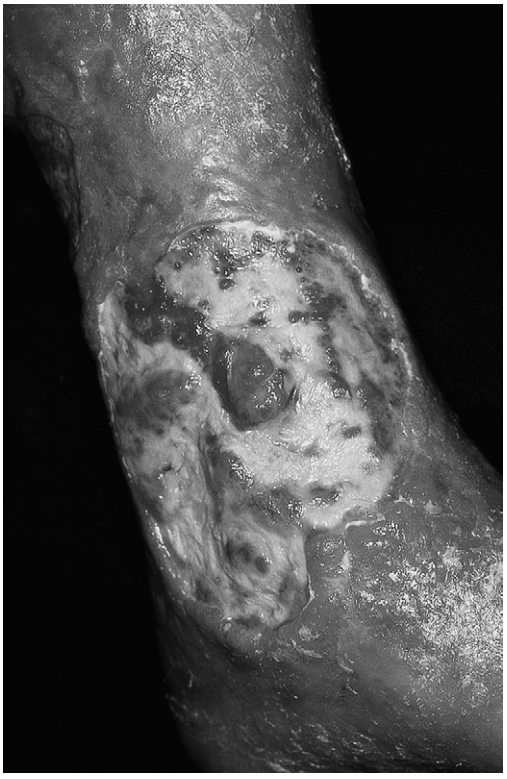

FIGURE 6-18 Venous stasis ulcer. Increased skin thickening and pigmentation with skin breakdown and ulceration are typically seen over the medial or lateral malleolus. The lesion is typically not nearly as painful as it looks and can be asymptomatic. *(From Bolognia JL, Jorizzo JL, Rapini RP, et al: Dermatology, 2nd ed. Philadelphia, Mosby, 2008.)*