

Selected Oncologic Emergencies

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PERSPECTIVE

Cancer remains the second leading cause of death in the United States. In 2007 alone, more than 1.4 million cases were diagnosed and 559,000 people died of cancer.¹ The most commonly diagnosed new cancer in men is prostate cancer and in women, breast cancer. The most common cause of cancer death for both sexes is lung and bronchial cancer.² Worldwide, the most commonly diagnosed new cancer in men is lung and bronchial cancer and in women breast cancer. Similarly, the most common cause of cancer death worldwide in men is lung and bronchial cancer and in women, breast cancer.³

Oncologic emergencies include fever and neutropenia, superior vena cava syndrome (SVCS), acute tumor lysis syndrome, hyperviscosity syndrome (HVS), hyperuricemia, hypercalcemia, neoplastic cardiac tamponade, spinal cord compression, and raised intracranial pressure (ICP). The accurate diagnosis and appropriate treatment of oncologic emergencies can improve the quality of life dramatically in patients with cancer. In addition, a reversible life-threatening emergency can occur in a patient with an underlying malignancy that is otherwise highly treatable or even curable, making identification and management of the oncologic emergency a potentially lifesaving action.

In 2003, it was estimated that the lifetime risk of developing cancer was 1:2 for men and 1:3 for women.⁴ Therefore, the emergency department (ED) physician should be well-versed in oncologic emergencies. Many factors can hinder the identification and management of oncologic emergencies in the ED (Box 121-1):

Changing trends in cancer that have produced an increased number of ED visits secondary to cancer and its complications include:

- More aggressive and broader use of chemotherapy regimens
- Increasing use of bone marrow transplantation
- More effective treatment options, increasing cure and survival rates
- Increased number of elderly patients receiving chemotherapy
- Increased survival for all cancers combined⁵

FEVER

Fever in the cancer patient can be caused by inflammation, transfusions, antineoplastics, antimicrobials, and tumor necro-

sis. Although fever can be secondary to malignancy with a significant tumor burden, most fevers (55–70%) occurring in cancer patients have an infectious origin. Neutropenia is defined as an absolute neutrophil count (ANC) fewer than 500 cells/mm³ or less than 1000 cells/mm³ with predicted decrease to less than 500 cells/mm³. ANC can be calculated as follows:

$$\begin{aligned} &(\text{Total white blood cell [WBC] count}) \\ &\times (\text{Percentage of neutrophils and bands})^6 \end{aligned}$$

The risk of infection and morbidity are increased with an ANC less than 1000/mm³, and substantially higher when counts are less than 100/mm³. In addition to the ANC, the risk of infection is related to the rate of development and the duration of neutropenia.

Fever in the neutropenic cancer patient should be considered a medical emergency. Prior to the era of empirical antibiotic therapy, infection accounted for almost 75% of mortality related to chemotherapy.⁶ Cancer patients with significant fever (defined by the Infectious Disease Society of America as a single oral temperature $\geq 38.3^\circ\text{C}$ [101°F] or an elevation of 38°C [100.4°F] for at least 1 hour) and a polymorphonuclear (PMN) leukocyte count less than 500/mm³ should be presumed to have an infectious origin. Antimicrobial therapy should be started immediately after appropriate cultures have been obtained.⁷

Clinical Features

Because fever is often the first and occasionally the only sign of infection in the neutropenic cancer patient, the emergency physician must take a careful and thorough history and perform a meticulous physical examination. In the absence of PMNs, traditional markers of inflammation such as erythema, warmth, and pyuria may be absent or minimal, making it essential to search for subtle signs of inflammation. On occasion, a neutropenic patient may not present with fever despite infection. This occurs more commonly in elderly patients and patients on corticosteroids.⁶

Many factors predispose the neutropenic patient to infection and sepsis, including prolonged bedrest, clinical deterioration, nutritional compromise, disruption of mucous membranes and skin barriers, and indwelling catheters. An undetected and untreated infection can be rapidly fatal in this population of patients. Therefore, broad-spectrum empirical antibiotic

BOX 121-1**FACTORS THAT CAN HINDER IDENTIFICATION AND MANAGEMENT OF ONCOLOGIC EMERGENCIES**

Patient or physician discomfort with the diagnosis of cancer
 A voluminous and frequently changing database of chemotherapeutic agents and complex classification systems
 Time constraints
 Lack of privacy
 Lack of an established physician-patient relationship
 Inappropriate or premature labeling of the cancer patient as "terminal"
 Failure to appreciate that effective treatments are available for oncologic emergencies and many of the cancers that cause them

BOX 121-2**FACTORS PREDISPOSING TO INFECTION AND SEPSIS**

Clinical debilitation, prolonged bedrest
 Nutritional compromise
 Disruption of mucous membranes and skin barriers
 Indwelling catheters
 Central nervous system dysfunction secondary to cancer, sedatives, opiates, or psychotropic medications

BOX 121-3**CURRENT RECOMMENDATIONS FOR ANTIMICROBIAL THERAPY OF FEVER IN NEUTROPENIC CANCER PATIENTS^{2,5,10}**

An antipseudomonal penicillin + an aminoglycoside ± vancomycin
 Ceftazidime ± an aminoglycoside
 Ceftazidime ± vancomycin
 Cefepime ± an aminoglycoside
 Cefepime ± vancomycin
 Imipenem/cilastatin
 Meropenem

therapy should be initiated promptly in all febrile, granulocytopenic patients (Box 121-2). Consulting with the oncologist will help categorize the risk for patients and aid selection of antibiotics based on local resistance patterns (Box 121-3).

Diagnostic Strategies

While antibiotics are being started, the patient should have a complete blood count (CBC) with differential cell count, platelet count, prothrombin time, partial thromboplastin time, blood chemistries, urinalysis, and analysis of any accessible sites suggestive of infection. Two sets of blood culture specimens should be obtained for aerobic, anaerobic, and fungal growth. If an indwelling catheter is present, at least one set of blood culture specimens should be obtained from the device lumen as well as from a peripheral vein.⁷ Obtaining a routine chest radiograph is currently the standard of care at most institutions. However, studies show that a chest radiograph is not necessary in patients with no respiratory symptoms and a normal physical examination.^{7,8} Urine should be sent for culture even in the absence of pyuria. However, the use of sputum culture and Gram's stain, although still recommended, has become controversial because of inconsistencies in collec-

tion and preparation that have led to false-negative and false-positive results.

Some oncologists discourage rectal temperature readings for patients with neutropenia because of the risk of tearing the rectal mucosa and establishing a potential nidus for disseminated infection. However, this has not yet become the standard of care and may vary among institutions. An indwelling nasogastric tube predisposes the neutropenic patient to sinusitis. When imaging for suggested sinusitis is required, computed tomography (CT) rather than sinus films is preferred. A lumbar puncture, preceded by head CT, is indicated when symptoms point to the central nervous system (CNS). Some authorities have recommended surveillance cultures of the stool, nose, and throat. This recommendation is not universally accepted and is generally not indicated in patients with solid tumors. Despite an intensive and comprehensive evaluation, an infectious cause is initially substantiated in only 30% of febrile, granulocytopenic patients.⁹ Nuclear scans, gallium citrate, and indium III scans do not have a place in the emergency diagnosis and treatment of these patients but may be useful in the definitive evaluation.

Differential Considerations

Overall, approximately 85% of the initial pathogens are bacterial, and of these, 60 to 70% are gram-positive pathogens. Gram-negative bacilli, particularly *Pseudomonas aeruginosa*, were the most common pathogens until the 1980s. However, the administrations of prophylactic antibiotics primarily active against gram-negative pathogens during chemotherapy, the widespread use of indwelling venous catheters and newer chemotherapy regimens have lead to an increase in gram-positive pathogens.^{7,10}

Staphylococcus aureus, *Staphylococcus epidermidis*, and *Streptococcus epidermidis* are the predominant gram-positive organisms. Once believed to be a contaminant, *S. epidermidis* has arisen as a major pathogen and may be resistant to antistaphylococcal penicillins and cephalosporins. *Escherichia coli*, *Pseudomonas aeruginosa*, and *Klebsiella pneumoniae* remain the most common gram-negative pathogens.

Fungal, viral, and parasitic infections are also important primary and secondary complications. Fungal infections, especially with *Candida albicans*, can be a major problem in granulocytopenic febrile patients treated with broad-spectrum antibiotics for protracted periods. Although significant institutional variation has been noted, *Histoplasma*, *Cryptococcus*, *Aspergillus*, and *Phycomyces* are additional fungal pathogens encountered in the compromised host. In contrast to patients with acquired immunodeficiency syndrome (AIDS), parasitic infections are not a common source of infection in patients with solid tumors. *Pneumocystis jirovecii* (formerly *carinii*), however, may be seen when corticosteroid use or hematologic malignancy has resulted in lymphocyte dysfunction. Herpes simplex, herpes varicella zoster, and cytomegalovirus are common viral pathogens. The compromised host is at risk for infection from a large number of individual pathogenic agents, thus further complicating the diagnosis and treatment of these patients.^{6,11}

In an attempt to prevent these infections, oncologists may initiate antimicrobial prophylaxis with trimethoprim sulfamethoxazole (Bactrim) or quinolones for immunosuppressed patients prior to development of fever.⁹ Additionally, recombinant human granulocyte-stimulating colony-stimulating factor (G-CSF) and granulocyte macrophage CSF (GM-CSF) are used to stimulate rapid increase in granulocytes in neutropenic patients in an effort to decrease the duration and degree of neutropenia and immunosuppression.¹²

Occasionally fever is without a source and is believed to arise from the underlying disease. However, it is impossible to differentiate using clinical and demographic factors those patients with bacteremia-induced fever from those with unexplained fever. In addition, the absence of physical findings indicative of infection does not exclude a potentially life-threatening septic event because at least 50% of septic patients lack any distinct physical findings. Despite the potential for few physical findings, a meticulous physical examination should be conducted, including the fundi (looking for *Candida* endophthalmitis), rectum, perineum and groin (for perirectal abscess), skin and mucous membranes (for any lesions suggesting malignancy or cellulitis), axillae, and catheters.^{6,11}

Management

In the initial evaluation and management of the febrile cancer patient, one must take into account the particular underlying malignancy, prior use of antimicrobial therapy, and how the degree of treatment has affected the host's immunologic compromise. For example, in acute leukemia normal circulating neutrophils and monocytes are largely replaced by blast cells, which do not function well in the phagocytizing and killing of bacterial and fungal agents. Chemotherapeutic agents and irradiation exacerbate or potentiate the underlying defect in already compromised host defenses. Corticosteroids impair granulocyte and mononuclear cell mobilization in leukemic patients. Patients with severely compromised host defenses and those in whom fever is accompanied by an increase in respiratory rate; change in mental status, agitation, or apprehensiveness; and hemodynamic instability should be urgently treated.

The optimal antimicrobial regimen should be synergistic, broad-spectrum, and bactericidal with a low potential for toxicity and chosen for efficacy against the most likely causes of systemic and rapidly progressing infection: *S. aureus*, *S. epidermidis*, *E. coli*, *P. aeruginosa*, and *Klebsiella* species. Traditionally, a two-drug regimen was selected because historical studies from the 1980s found that patients with gram-negative bacteremia had a higher survival rate when the isolate was sensitive to and treated with two antibiotics, compared with when the isolate was sensitive to only one of two antibiotics in the combined regimen.

In the past 10 years there has been significant advances in the antimicrobial armamentarium with development of broad-spectrum single agents such as the carbapenems (imipenem/cilastin, meropenem) and the third- to fourth-generation cephalosporins (ceftazidime, cefipime). These agents when investigated as monotherapy for granulocytopenic, febrile patients have been found to be as effective as a dual-drug combination of an antipseudomonal penicillin (ticarcillin, carbenicillin, or piperacillin) plus an aminoglycoside (gentamycin or tobramycin) in clinical trials. Amikacin is generally reserved as a second-line aminoglycoside for isolates that demonstrate aminoglycoside resistance.^{7,10,11}

Local resistance patterns should be used to guide treatment. Patients are risk-stratified and then treated accordingly. Patients with fever who appear in good condition are considered low risk. Patients with fever who have severe neutropenia, appear ill, and who are expected to have a protracted course, are high risk.¹³

Use of initial empirical vancomycin is included as first-line therapy at institutions where the incidence of methicillin-resistant *S. aureus* has been significant. Vancomycin should be included empirically in selected patients with:

- Clinically suspected catheter infections
- Known colonization with penicillin- and cephalosporin-resistant pneumococci or methicillin-resistant *S. aureus*

- Positive blood cultures for gram-positive bacteria before final identification and susceptibility testing
- Hypotension or other evidence of cardiovascular impairment

Patients should be admitted to an isolation room if possible, but rapid movement out of a congested waiting room into a private space is the higher priority. Hand washing and reverse isolation techniques should be used.

Current recommendations for antimicrobial therapy of fever in neutropenic cancer patients include the following:^{2,5,8-10}

- An antipseudomonal penicillin + an aminoglycoside ± vancomycin
- Ceftazidime ± an aminoglycoside
- Ceftazidime ± vancomycin
- Cefipime ± vancomycin
- Imipenem/cilastin
- Meropenem

■ SUPERIOR VENA CAVA SYNDROME

Epidemiology

SVCS is an acute or subacute process caused by the obstruction of blood flow through the superior vena cava (SVC) secondary to compression, infiltration, or thrombosis. Malignancy, most commonly lung cancer, is the most common cause of SVCS and currently accounts for 60 to 85 % of cases of SVCS.¹⁴ In fact, SVCS is often the initial presenting sign of the tumor.¹⁵

In recent years, benign causes for SVCS have gradually increased due to increasing use of intravascular devices.¹⁴ Other common nonmalignant causes include goiter, pericardial constriction, primary thrombosis, idiopathic sclerosing aortitis, tuberculous mediastinitis, fibrosing mediastinitis (histoplasmosis and methysergide treatment), arteriosclerotic or (rarely) luetic aneurysm, nephritic syndrome, and indwelling central venous catheters.^{16,17} In contrast to the adult population SVCS in pediatric patients is most often iatrogenic secondary to indwelling catheters, ventriculoperitoneal shunts, and complications of cardiovascular surgical procedures.

Clinical Features

Knowledge of the unique anatomic relationship of the SVC in the anterior superior mediastinum is crucial to understanding the clinical presentation of SVC obstruction. The SVC is easily compressed by any of its bounding contiguous structures (trachea, heart, aorta, azygos vein, and paratracheal and bronchial lymph nodes). This compression can produce a constellation of symptoms that reveal the exact site of the pathophysiologic process (Fig. 121-1). The SVC arises from the innominate veins, which in turn arise from the internal jugular and subclavian veins. The azygos vein, the last main auxiliary vessel of the SVC, drains blood from the chest wall. As a consequence of this anatomic relationship, if the SVC is blocked above or at the entrance of the azygos, blood may bypass and decompress the obstruction through the chest wall collateral vessels and rejoin the SVC via the azygos. If the obstruction falls below or at the entrance of the azygos, blood must traverse in a retrograde manner down the azygos and other chest wall veins to reach the drainage area of the inferior vena cava and subsequently cause more prominent symptoms.¹⁸ The severity of the syndrome is also related to the rate at which complete obstruction occurs. The more gradual the onset of obstruction, the longer the time for development of collateralization with less severe symptomatology.²

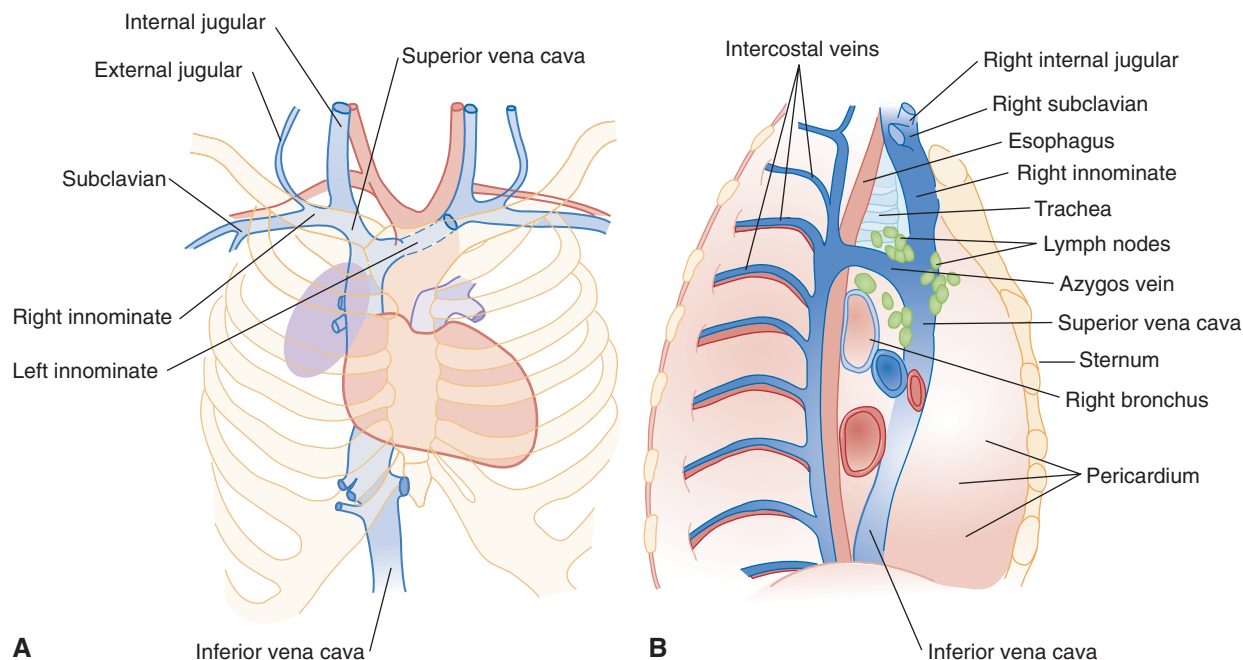


Figure 121-1. Frontal (A) and sagittal (B) sections of the thorax showing the relationship of the azygos vein to the superior vena cava (SVC), coalescence of innominates to form the SVC at the right second rib, and encasement of the SVC by nodal structures. Shaded area indicates classical site of obstruction. (From Lokich JL, Goodman R: Superior vena cava syndrome. *JAMA* 231:58, 1975.)

Because the clinical features of the SVCs are characterized by venous hypertension within the area ordinarily drained by the SVC, many of the findings are more noticeably evident in the recumbent or stooped-over position.

Early signs may include periorbital edema, conjunctival suffusion, and facial swelling, which will be most evident in the early morning hours and subside by midmorning. The most common symptom associated with SVCS is dyspnea with swelling of the face, trunk, and upper extremities observed in approximately 40% of patients. Cough, dysphagia, and chest pain are less commonly reported, each occurring in approximately 20% of patients. With increasing impedance to blood flow, the full-blown syndrome begins to manifest itself with thoracic and neck vein distention (67% and 59%, respectively), facial edema (56%), tachypnea (40%), tightness of the shirt collar (the Stoke sign), plethora of the face, edema of the upper extremities, and cyanosis.^{18,19}

Early reports of severe SVCS or prolonged and severe SVCS were believed to lead to irreversible thrombosis and death.^{20,21} Important concepts have changed since these early reports. Little evidence in the current literature substantiates the notion of untreated SVC obstruction as life-threatening except when it occurs with tracheal compression. Survival in patients with SVCS depends mainly on the course of the underlying disease.^{16,18}

SVCS can occur in conjunction with spinal cord compression (Rubin's syndrome). Venous obstruction usually develops before the spinal cord compression, which is localized in most instances to the low cervical or upper thoracic spinal cord. This syndrome is most commonly found with malignancies of lymphoma and lung cancer. Patients with venous obstruction and back pain should be evaluated with magnetic resonance imaging (MRI) of the vertebral spine.

Ancillary Evaluation

The clinical diagnosis of SVC obstruction is mimicked by a few other clinical entities—most noteworthy of which are peri-

cardial tamponade and heart failure, which can usually be excluded by physical examination, and pericardial effusion, which can be excluded by two-dimensional echocardiography. Because SVCS does not usually represent an immediately life-threatening oncologic emergency, once the clinical diagnosis is entertained, a tissue biopsy specimen should be obtained promptly. Although supportive therapy may be instituted to alleviate symptoms, definitive therapy should await the determination of histologic diagnosis because malignancy is known to be the cause of 60 to 85 % of reported cases. The chest film reveals a mass in nearly 10% of patients. When superior mediastinal mass is present, 75% are on the right side, and in approximately 50% of patients the masses are combined with pulmonary lesions or hilar adenopathy.

Pleural effusion is an associated finding in approximately 20 to 25% of patients and is customarily found in the right hemithorax.¹⁴ Morbidity secondary to excessive bleeding from puncture sites has been reported rarely with venous access procedures. IV injections may be less reliable because of slowing of drug distribution. Low flow rates may result in local irritation with thrombosis or phlebitis. Venous access is preferable on the contralateral side to the obstruction.

Venography is relatively contraindicated because of its concomitant bleeding complications. Invasive diagnostic procedures, including bronchoscopy, mediastinoscopy, scalene node biopsy, and limited thoracotomy are commonly used to establish the diagnosis and extent of the disease. Once SVC obstruction is suggested, the appropriate consulting services should be contacted and plans for prompt diagnosis undertaken.^{19,21}

Management

Historically, emergent radiation therapy was the treatment for SVCS. Currently, this is only recommended emergently for patients who present with stridor due to central airway obstruction or severe laryngeal edema. Current management uses chemotherapy because of the increased incidence of tumor

sensitivity to newer antineoplastic agents in an attempt to relieve the obstruction.¹⁵ However, temporizing measures that would alleviate symptoms related to vascular compression should be rapidly instituted.

Elevation of the head of the bed has been shown to be an effective immediate measure. Diuretics have been used with transient symptomatic relief, although they must be used judiciously because they can result in hypovolemia and further slowing of blood flow. Steroids have been shown to be of limited effectiveness but may be useful in the presence of respiratory compromise in reversing symptomatology.¹⁵ Current management approaches include percutaneous transluminal stent placement or bypass surgery.^{15,17}

The prognosis for patients treated for SVCS depends on the tumor type, with better survival rates in patients with lymphoma than with bronchogenic carcinoma. The overall survival is approximately 25% at 1 year and 10% at 30 months after treatment.

■ ACUTE TUMOR LYSIS SYNDROME

Acute tumor lysis syndrome (TLS) refers to the constellation of metabolic disturbances that occur as a result of ongoing cell death in a rapidly growing tumor. It also occurs frequently within a few hours to a few days after the initiation of chemotherapy or radiation therapy to treat bulky and treatment-responsive tumors.

This syndrome is most commonly seen after chemotherapy of hematologic malignancies, including acute leukemias and high-grade non-Hodgkin's lymphomas, particularly Burkitt's lymphoma, in which the growth fraction often exceeds 90%.²²⁻²⁴ With advances in the effectiveness of chemotherapy it has also been described following treatment of solid tumors such as small-cell lung carcinoma and germ cell tumors.

The risk of acute TLS increases with the bulk of the tumor, with the presence of hyperuricemia or with renal impairment before antineoplastic therapy (Box 121-4). Large numbers of neoplastic cells are killed rapidly, leading to release of intracellular ions and metabolic by-products into the systemic circulation. A correlation between a very high level of blood lactate dehydrogenase and the development of TLS has been observed.^{22,24}

Biochemical hallmarks of this syndrome include hyperuricemia (DNA [nucleic acids-purine] breakdown), hyperkalemia (cytosol breakdown), and hyperphosphatemia (protein breakdown). Hypocalcemia develops secondary to hyperphosphatemia. Acute renal failure; cardiac dysrhythmias, neuromuscular symptoms, and sudden death from hyperkalemia or hypocalcemia; and lactic acidosis and metabolic acidosis from acute renal failure may ensue. Early aggressive treatment is crucial.

Clinical Features

Symptoms are related to the underlying malignancy and hyperuricemia, hyperkalemia, hyperphosphatemia, and hypo-

calcemia. Hyperuricemia with resultant urate nephropathy is the most commonly recognized metabolic cause of renal insufficiency.²⁴

The kidney provides the primary mechanism for excretion of uric acid, potassium, and phosphate. Rapid proliferation of tumor cells may exceed the removal rate of the respective substances, resulting in increased levels. In fact, increased quantities of these substances have been observed in patients undergoing rapid lysis of chemosensitive tumors.

The integrity of renal function is a critical factor in determining the degree of metabolic derangements. In patients with preexisting renal insufficiency, the metabolic derangements of acute tumor lysis are more likely to be severe. However, even when renal function appears normal at the start of treatment, the rapid lysis of certain tumors may overwhelm the excretory capacity of the kidney. Similar to hyperuricemia, hyperphosphatemia may also cause renal failure. A possible mechanism is precipitation of calcium phosphate within the kidney.²⁴

Hyperkalemia, along with a contributing hypocalcemia, may result in life-threatening ventricular dysrhythmias.²² Hypocalcemia may also cause neuromuscular instability with muscle cramps and occasionally tetany. Confusion and convulsions also have been described in case reports.²⁴

Management

In approaching a patient with potential TLS, it is "easier to stay out of trouble than get out of trouble."²⁴ The main principles of TLS management are (1) identification of high-risk patients with initiation of preventive therapy and (2) early recognition of metabolic and renal complications with prompt supportive care, including hemodialysis. Most of the complications can be readily managed when they are recognized early; however, delay in recognition and initiation of treatment of TLS can be life-threatening.²⁵

Chemotherapy should be delayed, if possible, until metabolic disturbances, especially prerenal azotemia and hyperuricemia are corrected. Initial management is aimed at the control of preexisting hyperuricemia with hydration, allopurinol, and alkalinization of the urine to a pH greater than 7. Diuretics are added if necessary, and frequent monitoring of electrolytes, calcium, and phosphorus is essential.

Hydration

Volume depletion is a major risk factor for TLS and must be corrected vigorously. Rapid intravenous (IV) hydration is the single most important intervention. Hydration not only helps correct electrolyte disturbances by diluting extracellular fluid, but it also increases intravascular volume. Increased volume enhances renal blood flow, glomerular filtration rate, and urine volume, which consequently decreases the concentration of solutes in the distal nephron and medullary microcirculation. Continuous infusion rates as high as 4 to 5 L/day yielding urine volumes of at least 2 to 3 L/day should be given unless the patient's cardiovascular status indicates impending volume overload. Ideally, IV hydration in high risk patients should begin 24 to 48 hours prior to initiation of cancer therapy and continue for 48 to 72 hours after completion of chemotherapy.²⁵

Hyperuricemia

Allopurinol is a xanthine oxidase inhibitor and is given to reduce the conversion of nucleic acid by-products to uric acid in order to prevent urate nephropathy and subsequent oliguric

BOX 121-4 RISK FACTORS FOR ACUTE TUMOR LYSIS SYNDROME

- Increased lactate dehydrogenase levels (>1500 U/L)
- Advanced disease with abdominal involvement
- Preexisting renal dysfunction
- Post-treatment renal failure
- Acidic urine
- Concentrated urine
- Preexisting volume depletion
- Young age

BOX 121-5 CRITERIA FOR INSTITUTING HEMODIALYSIS

Serum potassium 6 mEq (6 mmol/L)
 Serum uric acid 10 mg/dL (590 mol/L)
 Serum creatinine 10 mg/dL (880 mol/L)
 Serum phosphorus 10 mg/dL (phosphate 3.2 mmol/L) or rapidly rising
 To reduce volume overload
 Symptomatic hypocalcemia

renal failure. Because allopurinol inhibits the synthesis of uric acid but has no effect on preexisting uric acid, uric acid levels usually do not fall until after 48 to 72 hours of treatment. Allopurinol usually is given orally between 300 and 600 mg/day for prophylaxis and 600 to 900 mg/day for treatment of TLS.²⁵

Rasburicase (recombinant urate oxidase) is a newer therapy that can be used when the uric acid levels cannot be lowered sufficiently by standard approaches. This modality of treatment is rarely initiated by emergency physicians. Rasburicase is useful in cases of hyperuricemia. Humans don't express urate oxidase; urate oxidase catalyses the conversion of poorly soluble uric acid to soluble allantoin. By converting uric acid to water-soluble metabolites, it effectively and rapidly decreases plasma and urinary uric acid levels. Unlike allopurinol, rasburicase does not increase the excretion of xanthine and other purine metabolites; therefore, it does not increase tubule crystallization of these compounds, thereby decreasing the risk of urate nephropathy.²⁵

Most articles agree that it is wise to alkalinize the urine as a prophylactic measure against hyperuricemia, but caution is advised should hyperphosphatemia and hypocalcemia develop. In patients with concomitant hyperphosphatemia, alkalinization favors precipitation of calcium/phosphate complexes in the renal tubules. Furthermore, alkali therapy may aggravate manifestations of hypocalcemia such as tetany.^{22,26} Although alkalinization increases the solubility of uric acid, the primary means of uric acid control is hydration and diuresis to maintain adequate urinary flow.^{24,26}

The use of furosemide or mannitol for osmotic diuresis has not proven to be beneficial as front-line therapy. In fact, these modalities may contribute to uric acid or calcium phosphate precipitation in renal tubules in a volume-contracted patient. Instead, diuretics should be reserved for well-hydrated patients with insufficient diuresis, and furosemide alone should be considered for the normovolemic patient with hyperkalemia or for the patient with evidence of fluid overload.

If TLS develops and it is refractory to the previously mentioned treatments, hemodialysis should be considered as a potentially lifesaving measure. This therapy is effective in lowering uric acid, potassium, and phosphate levels, as well as in controlling uremic symptoms. See the suggested criteria for instituting hemodialysis in [Box 121-5](#).

The prognosis is good in the absence of renal failure. If renal failure exists and hemodialysis of 5 to 7 days is necessary, the prognosis is grave. With aggressive management, the incidence of renal and metabolic complications of cytoreductive therapy may be decreased.

■ HYPERVISCOSITY SYNDROME

Hyperviscosity syndrome (HVS) refers to the clinical sequelae of increased blood viscosity. Viscosity is the resistance that a liquid exhibits to the flow of one layer over another. Excessive elevations in certain paraproteins (circulating immunoglobu-

lins) or cellular blood components (leukocytosis, erythrocytosis, and thrombocytosis) can result in elevated serum viscosity and the development of significant sludging, decreased perfusion of the microcirculation, and vascular stasis.

The outcome of these pathophysiologic events leads to the development of HVS, which requires urgent medical therapy to forestall or reverse the effects of sludging in the microcirculation of the CNS, visual system, and cardiopulmonary system.²⁷

Pathophysiology

HVS is most commonly associated with plasma cell dyscrasias (the paraproteinemias) and is due to the large size of the excess immunoglobulin M (IgM) paraproteins in these disorders. Waldenström's macroglobulinemia is the most common cause and accounts for about 85 to 90% of cases of HVS. Less frequently, the disease can occur in multiple myeloma (especially with myeloma proteins of the IgA and IgG₃ types). Other causes include cryoglobulinemia, a benign hyperglobulinemia of the IgM-IgG type, and leukemias.²⁷⁻²⁹

The blastic phase of chronic myelogenous leukemia, chronic granulocytic leukemia, and the blast cell crisis of acute lymphoblastic and nonlymphoblastic leukemias also commonly cause HVS.^{27,28} Other more benign causes include leukemoid reaction, polycythemia vera, and the accumulation of abnormal hemoglobins in sickle cell disease. The incidence of HVS in Waldenström's macroglobulinemia is found to be approximately 20%, in IgG myeloma approximately 4.2%, and in IgA myeloma as high as 25%.²⁸

The inherent physiochemical properties of the dysproteinemias along with extremely high concentrations of these proteins seem to predispose to the development of hyperviscosity. Paradoxically, HSV also has been reported in κ -light-chain disease owing to a greater tendency to form unstable, highly polymerized circulating aggregates. The etiologic factor most responsible for HVS in the leukemias appears to be leukocytosis with WBC counts in excess of 100,000, usually accompanied by blast forms exceeding 100,000 in the peripheral smear. The clinical manifestations of HVS become most apparent when the serum viscosity relative to water is greater than 4 to 5, normal serum viscosity relative to water being 1.4 to 1.8.²⁷⁻²⁹

Clinical Features

A symptomatic triad of mucosal bleeding, visual disturbances, and neurologic manifestations is a classic presentation of HSV. Visual disturbances and, on occasion, visual loss may occur with retinopathy characterized by venous engorgement (e.g., "sausage-link" or "boxcar" segmentation), which is also seen in the bulbar conjunctiva, microaneurysms, hemorrhages, exudates, and occasionally papilledema. Persistent bleeding diatheses from mucosal surfaces, especially nasal mucosa, the gastrointestinal (GI) tract, and sites of minor surgery or trauma, even in the face of a normal platelet count, are common. Other clinical findings encompass myriad neurologic disturbances, including headache, dizziness, jacksonian and generalized seizures, somnolence, lethargy, coma, auditory disturbances (including hearing loss), and hypotension. Constitutional symptoms of fatigue, anorexia, and weight loss that are non-specific early on are commonly associated with the underlying malignancy or with numerous electrolyte disturbances related to the underlying malignant process. Cardiopulmonary findings, including acute respiratory failure and hypoxemia, congestive heart failure, myocardial infarction, and valvular abnormalities have all been reported. Renal insufficiency and

failure may be a complication of the syndrome and will exacerbate existing clinical findings secondary to the expanded plasma volume.^{27,28}

The laboratory evaluation of the patient with suggested HVS should include a coagulation study and renal, electrolyte, and differential white count profiles. Serum and urine protein electrophoresis should be done in all cases of suggested dysproteinemias; the diagnosis is supported by a large spike on the serum electrophoresis. A clue to the presence of hyperviscosity may be the inability of the laboratory to perform chemical tests on the blood because of the serum stasis and increased viscosity that jams analyzers. In multiple myeloma significant hypercalcemia may also occur, and with high M-protein fractions a factitious hyponatremia may be present. The diagnosis may be also entertained when a patient is brought to the emergency room in a stupor or coma and anemia and rouleaux formation are found on the peripheral smear.³⁰

Because HVS is often a presenting characteristic of dysproteinemias and leukemias with blastic transformation and because a history of previously documented disease is often absent, this syndrome should be considered in patients with unexplained somnolence and coma.

Management

Emergency leukapheresis or plasmapheresis is the definitive treatment. Temporizing measures provided by the emergency physician should focus on adequate rehydration and diuresis. An immediate temporizing measure in a patient with frank coma and an established paraproteinemia is a two-unit phlebotomy with replacement of the patient's RBCs with physiologic saline.²⁷⁻³⁰ After plasmapheresis or leukapheresis has adequately alleviated the clinical findings, chemotherapeutic modalities can be used.

■ HYPERURICEMIA

Hyperuricemia is a serious and well-known consequence of certain malignant disorders, which, if recognized early, can result in a significant decrease in morbidity for the cancer patient. The major source is cell breakdown, and its major excretory pathway is via the kidneys.

Pathophysiology

The pathogenesis of hyperuricemia results from either the increased production or decreased excretion of uric acid, or both. Increased production of uric acid commonly results from accelerated generation of uric acid through purine metabolism as a result of rapid dissolution of neoplastic tissues (cell death) following chemotherapy or radiation therapy of undifferentiated lymphomas or lymphoblastic lymphomas and rapid cell proliferation and turnover with acute lymphoblastic leukemias.

In addition, hyperuricemia may be seen with multiple myeloma and occasionally with disseminated metastatic carcinoma. With massive release of precursors, uric acid levels rise precipitously and may reach levels as high as 15 to 20 mg/dL. As a result, uric acid crystals form in the highly concentrated and acidified urine of the distal tubules, intrarenal obstruction follows, and acute renal failure ensues.^{24,31}

Chronic, moderately elevated levels of the serum uric acid may result in renal colic, obstructive uropathy, or chronic renal failure. Either uric acid renal calculi or interstitial deposits of sodium urate may develop. This situation is associated with neoplastic overproduction of uric acid precursors. Polycythemia vera, myeloid metaplasia, mast cell disease, and chronic

granulocytic leukemia are often associated with this type of hyperuricemia.

Decreased excretion may be a result of underlying renal insufficiency or as a consequence of precipitation of urates in the renal tubules, parenchyma, or ureters with subsequent development of renal insufficiency and further reduction in excretion of uric acid. Three types of renal diseases are attributable to hyperuricemia: acute hyperuricemic nephropathy, uric acid nephrolithiasis, and gouty nephropathy.

Clinical Features

Hyperuricemia can occur with or without symptoms. Symptoms may be associated with the underlying malignancy. Hyperuricemia precipitated or aggravated by therapy of these diseases may occur as an isolated metabolic disturbance or may be accompanied by other manifestations of the TLS (see previous discussion on TLS). If an underlying neoplastic disease has been diagnosed, the possibility of hyperuricemia should be investigated before, during, and after treatment with chemotherapy or radiation. The hyperuricemia should be treated to prevent renal damage. In patients with urate stones and hyperuricemia, examination of the peripheral blood may provide evidence of an underlying myeloproliferative disorder. Acute oliguria following chemotherapy or radiation therapy suggests the diagnosis of hyperuricemia, and the uric acid level in the blood often far exceeds that associated with acute renal failure.

A number of benign diseases are associated with hyperuricemia that may coexist with neoplasia. These include hereditary gout, hyperparathyroidism, psoriasis, sarcoidosis, and renal failure of any cause. The long-term administration of certain drugs may lead to elevation of the serum uric acid level. Various diuretics, including thiazides and furosemide, are important examples.^{24,32} From a therapeutic standpoint, however, the finding of hyperuricemia obviates the importance of the primary cause; the therapy is the same.

Management

When possible, hyperuricemia should be treated before chemotherapy or radiation therapy, especially with bulky tumors or if the serum uric acid level is borderline or increased. If a uric acid elevation of more than 9 mg/dL is found, allopurinol, fluids, and alkalinization of the urine should be initiated. If possible, this regimen should be started a day or two before the initiation of chemotherapy or radiation treatment.

Patients with histories of gouty arthritis should also receive colchicine (0.6 mg orally twice a day) to avoid the acute attacks that can be associated with allopurinol administration. Patients should be kept well hydrated. In patients with acute distal tubular uric acid obstruction, treatment includes the administration of allopurinol, together with the fluid and electrolyte management used in other forms of acute renal failure.

If hyperuricemia is secondary to malignancy, cytolytic therapy should be stopped. Allopurinol in dosages of 300 to 600 mg/day usually causes a decrease in the serum uric acid level in approximately 3 days, so its administration should be started 2 or 3 days before cytolytic therapy, if time permits. Hydration is vital in maintaining a urine output above 2 L/day. Again, rasburicase (recombinant urate oxidase) is a newer therapy that can be used when the uric acid levels cannot be lowered sufficiently by standard approaches (see previous discussion on TLS).

Alkalinization to keep the urine pH above 7 can be accomplished by administering sodium bicarbonate (9–12 g/day). Diuretics are to be used as needed. Acetazolamide (Diamox)

in doses of 1 g/day usually alkalinizes the urine temporarily until allopurinol becomes effective. If oliguria occurs, IV mannitol may be started with 12.5 g of a 20% solution given over 3 minutes to keep urine output more than 250 mL/hr. The dose of mannitol is limited to 100 g/24 hours to avoid clinical features resembling water intoxication. If these measures fail, peritoneal dialysis or hemodialysis or flushing the ureters via retrograde catheters may be considered. Clearly, prevention of this complication is far better than treatment.

The cancer patient who comes to the ED with renal colic warrants careful evaluation for hyperuricemia. The prognosis depends on the underlying malignancy and degree of renal failure.^{24,31}

■ HYPERCALCEMIA

Hypercalcemia occurs in approximately 20 to 40% of cancer patients and is the most common life-threatening metabolic disorder associated with cancer.³³ It affects multiple organ systems and induces a variety of pathophysiologic events that may be more immediate threats to life than the cancer itself. For the purpose of this discussion, we discuss nonparathyroid hormone-mediated hypercalcemia, which is associated with malignancy.³⁴

Pathophysiology

Two mechanisms have been proposed to explain the development of hypercalcemia associated with malignancy. The first mechanism involves patients with metastatic bone involvement. This hypercalcemia is most likely associated with the release of calcium and phosphate caused by associated increased osteoclastic activity within the bone. The second mechanism involves those patients with no bone disease. A variety of tumor-produced hormone-like substances have been associated with the development of hypercalcemia, including parathyroid hormone, prostaglandins, and peptides, all of which affect bone turnover.

Hypercalcemia is a common feature of many malignancies but most often complicates cancer of the breast, lung, head, and neck, as well as multiple myeloma and leukemia. Bony metastases are not a prerequisite for hypercalcemia and when present do not necessarily cause hypercalcemia. In patients who are hypercalcemic from squamous cell lung cancer, only one in six has bone metastases. In small-cell lung carcinoma, hypercalcemia is almost never seen, despite the presence of bone marrow metastases in 20 to 50% of cases. A complex interaction of various substances (parathyroid hormone, prostaglandins, peptides, steroids, osteoclastic factors) appears to result in both increased bone synthesis and degradation. The exception is multiple myeloma, in which bone destruction is accompanied by minimal bone synthesis. Other entities that cause hypercalcemia are listed in [Box 121-6](#).^{35,36}

Rarer still are factitious hypercalcemia, idiopathic hypercalcemia of infancy (with elfin facies), familial hypocalciuric hypercalcemia, and hypercalcemia from pheochromocytoma or periostitis

Clinical Features

The development of symptoms of hypercalcemia is nonspecific. There is little correlation between serum calcium levels and the presence and severity of symptoms. Acute hypercalcemia results in marked CNS effects ranging from personality changes (depression, paranoia, lethargy, somnolence) to coma. With chronic hypercalcemia, symptoms include a history of anorexia, nausea, vomiting, constipation, polyuria, polydipsia,

BOX 121-6 NON-NEOPLASTIC CAUSES OF HYPERCALCEMIA

Hyperparathyroidism
 Hyperthyroidism
 Renal insufficiency (diuretic phase of acute renal failure, after transplantation, secondary hyperparathyroidism)
 Drugs (thiazide diuretics, lithium, and calcium carbonate)
 Hypervitaminosis (A and D)
 Acute adrenal insufficiency
 Immobilization (Paget's disease, fracture, paraplegia)
 Acromegaly
 Myxedema
 Milk-alkali syndrome
 Sarcoidosis
 Benign monoclonal gammopathy
 Rarer still are factitious hypercalcemia, idiopathic hypercalcemia of infancy (with elfin facies), familial hypocalciuric hypercalcemia, and hypercalcemia from pheochromocytoma or periostitis

BOX 121-7 COMMON SIGNS AND SYMPTOMS OF HYPERCALCEMIA IN MALIGNANCY

General

Itching

Neurologic

Fatigue, muscle weakness, hyporeflexia, lethargy, apathy, disturbances of perception and behavior, stupor, coma

Renal

Polyuria, polydipsia, renal insufficiency

Gastrointestinal

Anorexia, nausea, vomiting, constipation, abdominal pain

Cardiovascular

Hypertension, dysrhythmias, digitalis sensitivity

and memory loss. The signs, symptoms, and complications of hypercalcemia are summarized in [Box 121-7](#).

In patients with carcinoma, any of these symptoms should suggest the diagnosis of hypercalcemia, but the emergency physician should be particularly alert to the possibility of hypercalcemia in any cancer patient with lethargy or a change in mental status. Many may also have electrolyte abnormalities such as hypokalemia and dehydration. Thus evaluation of serum electrolytes should accompany the measurement of serum calcium, phosphorus, albumin, and alkaline phosphate. In general, a serum calcium level above 14 mg/dL constitutes a medical emergency. In chronic hypercalcemia, one may see patients with blood calcium levels as high as 15 mg/dL with only mild symptoms. With an acute onset, one can see patients comatose at a level of only 12 to 13 mg/dL.^{24,31-34}

Many benign conditions can result in hypercalcemia. The most common are hyperparathyroidism and Paget's disease of bone. Clinical features include a long history of hypercalcemia symptoms, particularly renal stones. Chronic changes on bone films, such as subperiosteal reaction and cysts or a "ground-glass" appearance of the skull, suggest hyperparathyroidism. Diagnosis of Paget's disease rests in biopsy results. Vitamin D excess, milk-alkali syndrome, and adrenal insufficiency are other common causes in the differential diagnosis of hypercalcemia.^{24,31}

The acute onset of severe hypercalcemia or chronic exposure of the renal tubules to elevated calcium levels may reduce

the glomerular filtration rate and renal blood flow, resulting in acute renal failure.²⁴

Management

The therapeutic modalities used in the treatment of hypercalcemia are numerous, but they should always be used in conjunction with therapy of the underlying malignant disease. The exception to this is breast cancer, when hormone therapy should be stopped until hypercalcemia is regulated.

The treatment depends on the clinical status of the patient and on the calcium level in the blood, but the general principles of treatment include treating the cancer when possible, encouraging ambulation, correcting dehydration, increasing urinary calcium excretion; inhibiting osteoclastic activity (calcium removal from bone) and reducing calcium intake.

If serum calcium levels are below 14 mg/dL, oral rehydration and ambulation may suffice. Normal saline solution can be administered if the oral intake is not sufficient. If the serum phosphate level is not elevated, oral phosphates may be used cautiously. Phosphosoda (5 mL by mouth, two or three times daily) is usually tolerated with mild to no diarrhea. IV phosphates are able to effectively lower the serum calcium level through precipitation of inorganic calcium phosphate salts in bone. This modality of treatment is usually not recommended, however, and if needed, it should only be done in consultation with a nephrologist or oncologist in view of their serious complications, which include widespread visceral calcifications, shock, and renal failure. This agent is usually reserved for hypercalcemia unresponsive to other agents.

Mithramycin (given as 25 µg/kg IM once every 4 to 5 days) is not generally part of the initial emergency management of hypercalcemia and has been supplanted in most cases by the bisphosphonates.

Prednisone (60–80 mg) or other corticosteroids may be effective within a few days to a week. This drug is more useful for long-term treatment than for acute control. Corticosteroids are particularly valuable in breast carcinoma, myeloma, and lymphoma. They should not be initiated without oncologic consultation because they are chemotherapeutic agents for these malignancies.

If the serum calcium level is greater than 14 mg/dL or significant symptoms are present, a more vigorous management should be undertaken. Continuous cardiac monitoring in the ED is necessary and central venous or pulmonary artery pressure monitoring may be required.

Saline rehydration and diuresis stimulates renal tubular excretion of calcium and is the most important initial component of the emergency management of hypercalcemia. Dehydration should be corrected within 1 to 2 hours with normal saline solution. When urine flow is adequate, furosemide (40–60 mg IV) may be given to increase excretion of calcium. Although the calciuric effect of furosemide is modest, it is also useful in preventing fluid overload in patients predisposed to cardiac failure. Careful attention to fluid input and output to ensure that the patient remains euvolemic is necessary.

Calcitonin is a naturally occurring hormone that inhibits bone resorption and increased excretion of calcium. Calcitonin may be effective in doses of 4 to 8 IU/kg IM/SC. This treatment, although relatively safe when renal function is normal, is not generally part of the initial emergency management of hypercalcemia.

Fifty percent of hypercalcemic cancer patients also have hypokalemia. Serum potassium levels should be monitored every 4 hours and potassium chloride (20–40 mEq, IV or PO) supplemented as necessary to prevent severe hypokalemia.^{24,31,33,35}

In the past 5 years following approval by the Food and Drug Administration, bisphosphonates have become the treatment of choice for management of cancer-induced hypercalcemia supplanting all other pharmacologic approaches except corticosteroids. Bisphosphonates act by binding to hydroxyapatite in bone and thereby inhibiting the dissolution of crystals. These agents prevent osteoclast attachment to bone matrix and interfere with osteoclast recruitment without inhibiting bone formation and mineralization.

Several agents are now available, including clodronate, pamidronate, and ibandronate, with other more potent bisphosphonates in development. Pamidronate (90 mg, given as an infusion over 4–24 hr) effectively and safely achieves normocalcemia within a few days (mean 4 days) in over 90 to 95% of patients.^{32–35}

■ NEOPLASTIC CARDIAC TAMPONADE

Although cardiac tamponade resulting from neoplasm is uncommon, it can occur abruptly and result in death if not treated quickly. In most cases neoplastic cardiac tamponade is observed in patients with a previous diagnosis of cancer, typically at late stages of the disease. It is rarely seen as the initial manifestation of an extracardiac malignancy.

The decompensated state of cardiac function comes from a marked rise in intrapericardial pressure caused by accumulation of fluid within the pericardial sac resulting from malignancy or from pericardial thickening with scar formation, which results in a thick constrictive neoplastic encasement. This condition, if not recognized and decompressed promptly, can lead to circulatory compromise and death. Signs and symptoms are partially affected by the rapidity of development. In the era prior to diagnostic ultrasound this medical/oncologic emergency was often unrecognized. In one early series prior to the advent of ultrasound, the diagnosis was missed by the first physician in 11 of 17 patients and a number of times was missed by more than a single examiner.³⁷

In most instances, pericardial effusion is accompanied by signs and symptoms that presage the development of the clinical picture of tamponade, including dyspnea, apprehension, anxiety, and chest pain. In rare instances, tamponade may be the first manifestation of the malignancy, solid tumor, or leukemia. Any patient in the ED with a history of cancer, shortness of breath, and hypotension should be suspected of having pericardial tamponade. The diagnoses of pulmonary embolism, congestive heart failure, and anxiety can be mistakenly made in this setting.

Etiology

The most common cause of neoplastic pericardial tamponade is malignant pericardial effusion, often associated with postirradiation pericarditis, fibrosis, and effusion. Only rarely does a tumor or radiation fibrosis cause a neoplastic constrictive pericarditis with resultant tamponade. In most reported cases, cardiac tamponade represents a clinical progression of neoplastic or postirradiation pericarditis.

Neoplastic pericarditis can result from any number of benign, malignant, primary, or secondary tumors of the pericardium or mediastinum.^{38–40} The most common benign tumors of the pericardium or mediastinum are fibromas, angiomas, and teratomas. Pericardial mesothelioma can have a clinical course characterized by rapid accumulation of massive quantities of bloody pericardial fluid, eventually leading to tamponade. Secondary involvement of the pericardium may result from either direct invasion from structures or metastases from a distant primary tumor. These metastases are usually multiple rather

than solitary lesions. The tumors most commonly associated with pericardial involvement include those of the lung and breast, leukemia, Hodgkin's and non-Hodgkin's lymphomas, melanomas, GI primary tumors, and sarcomas.^{40,41} Clinically recognizable symptoms or signs of pericardial disease are difficult to appreciate before death. Less than 30% of patients with autopsy-proven malignant pericardial disease were diagnosed antemortem.^{40,41}

Radiation pericarditis has been a well-known complication of radiotherapy since the introduction of modern megavoltage techniques. The cardiac effects of radiotherapy may manifest themselves immediately with acute pericarditis or be delayed for months to years, although the majority develop effusion within the first year. The acute forms are inflammatory or effusive, usually self-limited, and subside without residual constriction; the chronic effusive and constrictive types may lead to tamponade and death.⁴²

Neoplastic constrictive pericarditis, although rare, may be caused by the invasion of the pericardium by metastatic lesions or indirectly from the complication of radiation therapy with resultant fibrous thickening of the pericardium. Each of these entities can progress to cardiac tamponade because of thickening by tumor or radiation fibrosis, resulting in a decrease in the distensibility of the pericardium, thus reaching the critical point of cardiopulmonary decompensation earlier, despite smaller volumes of slowly accumulating effusion.

The symptoms and signs of neoplastic and radiation pericarditis mimic pericarditis from other causes, and because of the usual insidious onset of the effusion of fibrous pericardial thickening, the condition might be attributed to the underlying malignancy and not considered until the full-blown picture of cardiac tamponade develops.

Pathophysiology

The severity of cardiac tamponade and eventual cardiopulmonary decompensation depend on the rate of pericardial fluid accumulation, the fluid volume, and the underlying cardiac function. Clinically the progressive elevation of intracardial pressure interferes with ventricular expansion and results in a decrease in the cardiac volume. Intracardial chamber pressures rise rapidly with subsequent transmission of this pressure peripherally in pulmonary and vena caval beds. In an effort to maintain cardiac output, various compensatory mechanisms come into play (tachycardia, peripheral vasoconstriction, decrease in renal flow with resultant increase in blood volume by sodium and water retention), all to maintain arterial pressure and venous return. When these compensatory mechanisms fail to maintain cardiac output, ventricular end-diastolic pressure increases and subsequent circulatory collapse is impending. The signs and symptoms parallel these pathophysiologic changes. The most common symptoms include extreme anxiety and apprehension, a precordial oppressive feeling, or actual retrosternal chest pain with dyspnea of varying degrees. True orthopnea and paroxysmal nocturnal dyspnea are uncommon, but when they occur the patient assumes a variety of positions to get relief from the chest pain and the dyspnea. Other prominent symptoms include cough, hoarseness, hiccups, and occasional GI manifestations such as dysphagia, nausea, vomiting, and epigastric or right upper quadrant abdominal pain that is probably the result of visceral congestion.^{12,40-43}

Clinical Features

Patients with severe tamponade are acutely ill and may appear ashen, pale, or markedly diaphoretic with an impaired consciousness ranging from mildly confused to unresponsive.

Table 121-1

Physical Evaluation of Neoplastic Cardiac Tamponade

Beck Triad or Acute Compression Triad

Described in 1935, this complex of physical findings refers to increased jugular venous pressure, hypotension, and diminished heart sounds.

These findings result from a rapid accumulation of pericardial fluid. However, this classic triad is usually observed in patients with acute cardiac tamponade.

Pulsus Paradoxus or Paradoxical Pulse

This is an exaggeration (>12 mm Hg, or 9%) of the normal inspiratory decrease in systemic blood pressure.

To measure the pulsus paradoxus, patients are often placed in a semirecumbent position; respirations should be normal. The blood pressure cuff is inflated to at least 20 mm Hg above the systolic pressure and slowly deflated until the first Korotkoff sounds are heard only during expiration. At this pressure reading, if the cuff is not further deflated and a pulsus paradoxus is present, the first Korotkoff sound is not audible during inspiration. As the cuff is further deflated, the point at which the first Korotkoff sound is audible during both inspiration and expiration is recorded. If the difference between the first and second measurement is greater than 12 mm Hg, an abnormal pulsus paradoxus is present.

The paradox is that while listening to the heart sounds during inspiration, the pulse weakens or may not be palpated with certain heartbeats, while S_1 is heard with all heartbeats.

A pulsus paradoxus can be observed in patients with other conditions, such as constrictive pericarditis, severe obstructive pulmonary disease, restrictive cardiomyopathy, pulmonary embolism, rapid and labored breathing, and right ventricular infarction with shock.

A pulsus paradoxus may be absent in patients with markedly elevated left ventricular diastolic pressures, atrial septal defect, pulmonary hypertension, and aortic regurgitation.

Kussmaul's Sign

This was described by Adolph Kussmaul as a paradoxical increase in venous distention and pressure during inspiration.

This sign is usually observed in patients with constrictive pericarditis but occasionally is observed in patients with effusive-constrictive pericarditis and cardiac tamponade.

Rapid, shallow, and occasionally labored breathing may be present along with peripheral cyanosis and distended jugular veins. Seizures have been reported. Striking facial plethora and a full neck secondary to edema (Stoke's collar) can also be seen in SVCS. Pulses are soft and easily compressible. The systolic blood pressure is usually low, with a decreased pulse pressure, although normal systolic, diastolic, and pulse pressures have been reported with moderate degrees of tamponade. Kussmaul's signs (muffled heart sounds, an enlarged cardiomeastinal silhouette, tachycardia, and, most notably, pulsus paradoxus) are extremely useful findings in the physical evaluation of tamponade (Table 121-1).⁴⁴ Ascites, hepatomegaly, peripheral edema, and mottling are other findings that reflect the elevation in venous pressure and decrease in cardiac output.^{12,40,41,45}

Ancillary Evaluation

Low-voltage and the nonspecific findings of pericardial effusion, sinus tachycardia, ST elevation, and nonspecific ST-T wave changes may occur. Electrical alternans with 1:1 total atrial-ventricular complexes has been considered almost pathognomonic of cardiac tamponade. Electrical alternans is the alternation of electrocardiographic QRS complexes, usually

in a 2:1 ratio. This is due to movement of the heart in the pericardial space. Electrical alternans is also observed in patients with myocardial ischemia, acute pulmonary embolism, and tachyarrhythmias.⁴⁴

Approximately two thirds of the reported cases of pulsus alternans occur in patients with tamponade caused by massive pericardial effusion in neoplastic pericarditis. The alternation customarily disappears soon after removal of a small volume of fluid, but it can also disappear spontaneously or be observed in attendance with a fluid increase.⁴¹

Radiographic signs of tamponade suggestive of pericardial effusion include an enlarged cardiac silhouette with clear lung fields and normal vascular pattern, although a normal chest radiograph does not exclude tamponade. The typical “water-bottle” appearance of the heart on a plain radiograph is often present.

Echocardiography is the simplest and most sensitive of diagnostic tests and can be done at the bedside for confirmation of pericardial effusion. Thoracic CT has also become an important diagnostic tool in diagnosing pericardial effusions.^{41,46}

Cardiac tamponade should be considered in any cancer patient with dyspnea. Highly suggestive symptoms include clouded sensorium, thready pulse, pulsus paradoxus exceeding 50% of the pulse pressure, low systolic pressure, engorged neck veins with a rising peripheral venous pressure above 130 mm H₂O, a falling pulse pressure below 20 mm Hg, and electrical alternans. This is an uncommon yet pathognomonic sinusoidal variation in QRS size secondary to the pendular effect of the heart swinging in the fluid medium of the pericardial sac.⁴⁵ In this setting, sudden death may occur and pericardiocentesis should be performed as soon as possible.

Management

In the ED, the only lifesaving treatment for tamponade that is effective is immediate removal of the pericardial effusion via pericardiocentesis. The procedure carries some risk, including induction of cardiac dysrhythmias and hemorrhage from an injured coronary vessel. Aspiration of as little as 50 to 100 mL of fluid has been shown to temporarily alleviate the pathologic process.^{12,40,41}

Emergency subxiphoid percutaneous drainage is a lifesaving bedside procedure. The subxiphoid approach is extrapleural; hence, it is the safest for blind pericardiocentesis. A 16- or 18-gauge needle is inserted at an angle of 30 to 45 degrees to the skin, near the left xiphocostal angle, aiming toward the left shoulder. When performed emergently, this procedure is associated with a reported mortality rate of approximately 4% and a complication rate of 17%.⁴⁴

Echocardiographically guided pericardiocentesis can also be performed in the ED, but the cardiac catheterization laboratory is a more controlled setting and is preferable: this is usually performed from the left intercostal space. First, mark the site of entry based on the area of maximal fluid accumulation closest to the transducer. Then, measure the distance from the skin to the pericardial space. The angle of the transducer should be the trajectory of the needle during the procedure. Avoid the inferior rib margin while advancing the needle to prevent neurovascular injury. Leave a 16-gauge catheter in place for continuous drainage.⁴⁴

Removal of the maximal amount of fluid is advisable, along with inserting of an indwelling catheter, during the first pericardiocentesis since fluid may reaccumulate during the first 24 hours. Once the pericardial fluid has been obtained, it must be sent for biochemical and cytologic analysis. Neoplastic cardiac tamponade accounts for at least 50% of all reported cases of pericardial fluid collection. Other types of supportive therapy may be needed during the evaluation process while

preparing for pericardiocentesis, such as IV hydration with normal saline and oxygen therapy.

Once the patient has been stabilized, additional therapeutic interventions should be planned and initiated by the appropriate admitting services because reaccumulation of effusion in neoplastic tamponade is not easily managed on a short-term basis. Pericardial windows, radiotherapy, intrapericardial chemotherapy, and pericardiectomy may be justified.^{12,40,41}

The prognosis after neoplastic cardiac tamponade depends on the underlying type and extent of cancer. The presence of total electrical alternans is an adverse prognostic sign, even when the alternans disappears with pericardiocentesis. Despite a poor prognosis for patients with cancers such as melanoma or non-small-cell lung cancer, some patients with treatment-responsive lymphomas have survived long-term after neoplastic cardiac tamponade.

■ NEUROLOGIC EMERGENCIES

Of all patients with cancer, 15 to 20% have neurologic complications.⁴⁷ Neurologic symptoms are occasionally the presenting complaint in patients with systemic cancer, but more often symptoms develop in patients known to have cancer. Neurologic emergencies in cancer patients include cerebral herniation, seizures, epidural spinal cord compression, CNS infections, and reversible toxic or metabolic encephalopathies. Treatment is needed urgently after the patient arrives at the ED to prevent permanent neurologic dysfunction or death.

■ CEREBRAL HERNIATION

Pathophysiology

Cerebral herniation occurs when the ICP increases locally within the skull from an expanding mass lesion. The increase produces a shift of brain substance in the direction of least resistance caudally through the tentorial opening and the foramen magnum. Causes of cerebral herniation in cancer patients commonly include primary or metastatic brain tumors and intracerebral hemorrhage. Less common causes include subdural hematoma, brain abscess, acute hydrocephalus, and radiation-induced brain necrosis.⁴⁶ Primary brain tumors account for approximately one half of intracranial tumors. Metastatic brain tumors are seen most commonly in lung, breast, colon, kidney, and testicular cancer and in patients with choriocarcinoma and malignant melanoma.^{47,48}

Clinical Features

Three distinct herniation syndromes have been described: uncal, central, and tonsillar herniation. In *uncal* herniation a lateral mass displaces the temporal lobe, which compresses the upper brainstem. A rapid loss of consciousness is seen in conjunction with unilateral pupillary dilatation and ipsilateral hemiparesis. *Central* herniation usually results from slowly expanding, multifocal lesions that cause a downward and lateral shift of the diencephalon and upper pons. A slowly decreasing level of consciousness, small reactive pupils, and Cheyne-Stokes respirations, without focal signs, are seen clinically. Central herniation is sometimes mistaken for toxic or metabolic encephalopathy because of the lack of focal signs. A history of headache or focal neurologic complaints or any lateralizing findings indicates the need for prompt CT of the head to rule out a herniating mass lesion before lumbar puncture. *Tonsillar* herniation is produced by a large posterior fossa mass that pushes the cerebellar tonsils through the foramen magnum, compressing the medulla and resulting in a rapidly decreasing level of consciousness, occipital headache, vomit-

ing, hiccups, hypertension, meningismus, and abrupt changes in the respiratory pattern.⁴⁷⁻⁵⁰

Management

When the clinical diagnosis of cerebral herniation is made, emergency management is necessary before the cause can be established. Intubation with hyperventilation to a carbon dioxide partial pressure (Pco₂) of 25 to 30 mm Hg temporarily lowers the ICP by producing cerebral vasoconstriction. This should be avoided if possible but may be necessary for brief periods in response to reversible, acute neurologic deterioration. Excessive or prolonged hyperventilation may cause paradoxical vasodilation and should be avoided. Mannitol (1 g/kg IV) should be given and may be repeated in 4 to 6 hours. Dexamethasone (12–24 mg IV) has not been shown to improve outcome or reduce ICP acutely in severe head injury,^{51,52} but is often administered in patients with raised ICP or impending herniation caused by CNS malignancy because of the effect of corticosteroids on reducing cerebral edema associated with the neoplastic process. CT of the brain should be obtained as soon as emergency stabilization is accomplished. Epidural or subdural hematoma and hydrocephalus usually require surgery, whereas abscess and metastases are usually managed with antibiotics and antineoplastics or radiation, or both, respectively. When stabilization and an initial diagnosis have been made, neurologic or neurosurgical consultation and prompt admission to an intensive care unit are mandatory.^{47,49}

SEIZURES

Seizures are common in patients with cancer. Their immediate management is necessary to prevent physical injury, increased ICP, and risk of aspiration. Seizures increase the brain's metabolic requirements and lead to increased cerebral blood flow. This may precipitate increased ICP in susceptible patients. Seizures may be due to brain metastases, toxic or metabolic disturbances (usually hyponatremia or uremia), vascular problems (especially intracerebral hemorrhage or subdural hematomas), and infections. Diagnostic laboratory studies should include a CBC, electrolytes, glucose level, blood urea nitrogen (BUN), measurement of calcium and magnesium levels, liver function tests, coagulation studies, and appropriate cultures. CT of the head should be done and followed by a lumbar puncture, when indicated.^{47,49}

The therapy for seizures depends on the specific cause and the patient's clinical status. For example, a single hypoglycemic or hypoxic seizure usually requires only correction of the underlying metabolic defect. Patients with a single seizure whose workup reveals a chronic problem (e.g., a cerebral metastasis) require anticonvulsants and therapy specific for the malignancy. A loading dose of phenytoin (15–18 mg/kg IV) may be given followed by oral maintenance. Prolonged single seizures or repetitive seizures require more vigorous treatment, including diazepam (5–10 mg IV) or lorazepam (1–2 mg IV) followed by IV phenytoin. Active airway and ventilatory management is essential. A bedside fingerstick glucose level should be obtained promptly. Thiamine and naloxone are not routinely indicated. In addition, when repetitive seizures have occurred, management of the underlying cause should be initiated rapidly and the patient admitted to an intensive care unit.^{47,49}

EPIDURAL SPINAL CORD COMPRESSION

Principles of Disease

Epidural spinal cord compression from metastatic cancer is common, serious, and potentially treatable. It is most often

caused by lymphoma or lung, breast, or prostate carcinoma. With the exception of lymphoma, which extends through the intervertebral foramina from paravertebral lymph nodes, these tumors metastasize to the vertebral body and extend into the spinal canal to compress the spinal cord. Less common causes of spinal cord compression in patients with cancer include melanoma, myeloma, renal cell carcinoma, vertebral subluxation, spinal epidural hematomas, and intramedullary metastasis. Acute myelopathy in patients with cancer may also be caused by radiation, paraneoplastic necrotizing myelitis, a ruptured intervertebral disk, and meningeal carcinomatosis with spinal cord involvement. Most cases (68%) of epidural cord compression occur in the thoracic spine, 15% occur in the cervical spine, and 19% in the lumbosacral spine.⁵³

Clinical Features

Back pain, either local or radicular, is the initial symptom in 95% of patients with epidural metastasis. It may be acute in onset or develop insidiously over weeks to months and usually predates other symptoms. The pain may increase during physical examination with spinal percussion, neck flexion, Valsalva maneuver, or straight leg raising and is usually located at the level of the tumor.^{50,54,55} Other symptoms are usually present at the time of diagnosis and may include weakness (75% of patients) and autonomic or sensory symptoms (50% of patients). Fifty percent of patients are not ambulatory at the time of diagnosis. The neurologic examination usually reveals symmetrical weakness with either flaccidity and hyporeflexia (if the diagnosis is made very early) or spasticity and hyperreflexia (if the diagnosis is made later).

Diagnostic Strategies

Plain films show evidence of tumor in the vertebral body in 70 to 90% of patients with vertebral metastases.^{47,51} Immediate myelography or MRI is indicated if the plain films are abnormal, regardless of whether the neurologic examination is abnormal or is consistent with spinal cord compression or the plain film findings. In cases with questionable findings on plain films of the spine, tomograms, coned-down views, or CT may reveal bone metastases not otherwise appreciated. Myelography can demonstrate a complete or near-complete obstruction of contrast dye flow at the level of vertebral body involvement. MRI has emerged as the procedure of choice for intramedullary metastases and has also replaced myelography, which is associated with significant morbidity related to lumbar puncture and dye insertion at multiple levels (including cisternal puncture), to demonstrate the length of the compression or skip lesions along the spinal cord.^{50,55}

Management

Because minimal weakness at the time of presentation may progress to profound, irreversible weakness over several hours, treatment should be started rapidly. In the ED, a loading dose of dexamethasone (10–100 mg IV), followed by 4 to 24 mg every 6 hours for 3 days to reduce cord edema, is initiated at the time of diagnosis. Immediate oncology and radiation oncology consultations should be obtained. Although corticosteroids are routinely administered to patients with suggested spinal cord compression, high-dose corticosteroids, such as dexamethasone (100 mg), have been associated with complications and their use is controversial.⁵⁵ Radiation treatment is the usual therapy and can be initiated after steroid treatment. The prognosis depends on the radiosensitivity of the tumor, the location of the compression, the pretreatment performance status, and the rate of decompensation. Surgery is indicated

only if the diagnosis is in doubt, if a tissue diagnosis is required, if the spine is unstable, or when maximal doses of radiation have already been given to the involved area.^{47,48,56}

Intramedullary metastases are similar in presentation and treatment to epidural cord compression but are associated with a very poor prognosis. Epidural hematomas have been described in patients with thrombocytopenia or a coagulopathy as a complication of lumbar puncture. A rapidly progressive paraparesis and back pain are seen. MRI or myelography can establish the diagnosis; the treatment is surgical decompression. Platelet transfusions may limit progression in the ED.^{47,56}

■ CENTRAL NERVOUS SYSTEM INFECTIONS

Principles of Disease

Patients with cancer are susceptible to a variety of CNS infections. These patients may have impaired immune responses secondary to their underlying disease or treatment with steroids, chemotherapy, splenectomy, or irradiation. Most CNS infections occur in patients with leukemia, lymphoma, or head and neck cancer. Patients with head and neck cancer are susceptible (in addition to the reasons discussed) because of fistula formation and tumor invasion, which allows organisms access to the CNS. Important CNS infections include meningitis, brain abscess, and encephalitis. These often have similar presentations, making their differentiation in the ED difficult.

Clinical Features

Meningitis is characterized by fever, headache, and altered mental status. Meningismus is often absent. The diagnosis of meningitis in patients with cancer is often delayed because the manifestations of the disease are attributed to other processes: fever to systemic infection, headache to cerebral metastases, and altered mental status to a toxic or metabolic encephalopathy.

Diagnostic Strategies

All cancer patients with fever and an altered mental status require a lumbar puncture, which should be preceded by head CT if cerebral metastases are suggested.^{49,52} In addition, thrombocytopenia and coagulopathy should be considered and either ruled out or treated appropriately with platelet transfusions or fresh frozen plasma, respectively, before a lumbar puncture is done. Platelet transfusion is usually reserved for patients with platelet count less than 10,000/ μ L. The fluid obtained should be sent for a cell count and differential cell count, Gram's stain, India ink stain, protein and glucose levels, bacterial and fungal cultures, cryptococcal antigen level, and cytologic examination. The absence of WBCs in the cerebrospinal fluid does not rule out meningitis, especially in neutropenic patients. The likely organisms responsible for meningitis vary with the underlying disease and the peripheral WBC count.

Differential Considerations

Brain abscess is usually seen in patients with leukemia or head and neck tumors and accounts for 30% of CNS infections in cancer patients.⁴⁹ Patients have symptoms of elevated ICP (headache, vomiting, and papilledema), lateralizing findings, and a source of infection.^{48,52} Fever is usually present. Head CT characteristically demonstrates an ill-defined mass early in

the course of an abscess, with the classic well-defined mass with a low-density center and a contrast-enhancing ring seen later. Edema and mass effect are common. A lumbar puncture is not helpful in making the diagnosis and may precipitate cerebral herniation. Organisms that cause abscess include gram-negative rods, *Aspergillus* and *Phycomycetes* species, and *Toxoplasma gondii*. Emergency management includes high-dosage antibiotics. If herniation develops, immediate steps to reduce the ICP, followed by emergency surgery, are indicated.

Encephalitis is rare in patients with cancer and is most often caused by herpes zoster or *T. gondii*. The presenting complaints are usually headache, fever, and altered mental status. The CT scan is commonly normal but may show diffuse edema, whereas the lumbar puncture may show pleocytosis with an elevated protein level but no demonstrable organism. It is difficult to distinguish encephalitis from meningitis in the ED, but the overall clinical picture in both diseases mandates hospital admission for further evaluation.

Management

Until further evaluation is able to distinguish between meningitis and encephalitis, empirical broad-spectrum antibiotic coverage with a third-generation cephalosporin (ceftriaxone or ceftazidime) and vancomycin should be initiated for all patients. Ampicillin may be added when there is suggestion of *Listeria*. Ceftazidime with or without an aminoglycoside is generally selected when the likelihood of infection with *Pseudomonas* is high. Neutropenic patients (polymorphonuclear WBC count < 1000/ mm^3) with either leukemia or lymphoma usually have a gram-negative infection (often with *P. aeruginosa*). Patients with lymphoma and a normal WBC count are commonly infected with *Listeria monocytogenes*, *Streptococcus pneumoniae*, or *Cryptococcus neoformans*. Infections with *Haemophilus influenzae* and *Neisseria meningitidis* are uncommon. Patients with head and neck tumors may develop staphylococcal infection.⁴⁷⁻⁵⁰

Encephalopathy

Toxic and metabolic encephalopathy should be actively considered when patients with cancer have an acute or subacute altered mental status in the absence of fever or headache. Toxic and metabolic causes should be routinely excluded even when infection or a metastatic complication is suggested. Signs of encephalopathy include confusion, aberrant behavior, and a decreased level of consciousness. These may develop acutely or insidiously over days to weeks. Patients with cancer are particularly susceptible to toxic and metabolic encephalopathy because their disease can have multiple organ system involvement, can cause electrolyte and nutritional abnormalities, and the drugs used to treat the disease (especially chemotherapeutic agents and narcotics) can cause encephalopathy even when used in therapeutic doses.^{47,48} In the ED, encephalopathic patients should first be evaluated carefully for a possible infection or mass lesion. The metabolic workup should include electrolytes; BUN, creatinine, glucose, and calcium levels; arterial blood gases; and liver function tests. Toxicology screens should be considered in possible ingestions and in patients who are unable to give a history. Naloxone and 50% dextrose should be given while the workup is proceeding. Specific treatment is indicated for any abnormalities found during the workup. Hospital admission is usually required unless the cause is easily and rapidly reversible and is unlikely to recur.

KEY CONCEPTS

- Hypercalcemia due to malignancy is unrelated to bone metastases in 20% of patients and is associated with a poor prognosis independent of therapeutic response. Hydration and use of bisphosphonates (e.g., pamidronate) have become the mainstays of initial treatment.
- Spinal cord compression arises as back pain in more than 95% of patients. If ambulatory at the time of diagnosis, 80% of patients maintain the ability to ambulate. MRI has become the diagnostic modality of choice, and high-dose dexamethasone is given to all patients, followed by radiation therapy in most cases.
- Superior vena cava obstruction is rarely life-threatening and requires tissue diagnosis. Although caused by malignancy in 70 to 80% of cases, thrombosis secondary to indwelling central lines is increasing as a cause. Stenting of the SVC has become the approach to SVC obstruction unresponsive to chemotherapy or radiation therapy, or both.
- Fever and neutropenia in the cancer patient are a true medical emergency requiring rapid diagnosis, cultures, and treatment with broad-spectrum, bactericidal, synergistic antimicrobials. An aminoglycoside plus an extended-spectrum penicillin and third-generation cephalosporin with or without vancomycin remain the standard combinations in patients without penicillin allergy.
- Neoplastic pericardial effusion can arise insidiously with symptoms such as apprehension, anxiety, dyspnea, and weakness. Bedside ultrasonography has become a rapid, safe imaging modality for establishing the diagnosis prior to the development of clinically apparent tamponade in a critically ill patient.
- Acute TLS, previously limited to hematologic malignancies, is now being described in patients receiving chemotherapy for solid tumors. It can arise with dyspnea, mental status changes, cardiac dysrhythmia, or seizures. Treatment includes urinary alkalinization and emergency hemodialysis in cases complicated by acute renal failure.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.

PERSPECTIVE

Nausea and vomiting may constitute the primary presentation of many gastrointestinal (GI) disorders (e.g., bowel obstruction, gastroenteritis) or the secondary presentation of numerous systemic conditions (1) caused by severe pain, especially visceral pain; (2) caused by or related to severe systemic illness, such as myocardial infarction, sepsis, or shock; or (3) related to definitive conditions by specific mechanisms, such as pregnancy (hormones), increased intracranial pressure (central mechanism), toxins (homeostatic response), motion sickness (neuroendocrine), and chemotherapy (chemoreceptor trigger zone [CTZ]). Additionally, vomiting may cause serious sequelae, such as aspiration pneumonia, Mallory-Weiss syndrome, esophageal rupture, volume depletion, and metabolic derangement. Classification by duration and frequency of the vomiting (acute, recurrent, chronic, or cyclic) may assist in determination of the underlying cause.¹

Epidemiology

The most common causes of nausea and vomiting are acute gastroenteritis, febrile systemic illnesses, and drug effects. Acute viral gastroenteritis is the most common GI disease in the United States. In adult medicine, nausea and vomiting are caused most often by medications. Emesis associated with pregnancy is common, especially in the first trimester, but hyperemesis gravidarum is not. Although the scope of the differential diagnosis in the pediatric population is broad, acute vomiting is commonly seen with infectious disorders affecting the gastrointestinal tract as well as with infections in other areas of the body.²

Pathophysiology

The act of vomiting can be divided into three distinct phases: nausea, retching, and actual vomiting^{3,4} (Fig. 20-1). Nausea may occur without retching or vomiting, and retching may occur without vomiting. *Nausea* is defined as a vague and extremely unpleasant feeling that often precedes vomiting. The exact neural pathways mediating nausea are not clear, but they are likely to be the same pathways that mediate vomiting. Mild activation of the pathways may result in nausea, whereas more intense stimulation results in vomiting. During nausea, there is an increase in tone in the musculature in the duodenum and jejunum, with a concomitant decrease in gastric tone; this leads to reflux of intestinal contents into the stomach. There is often associated hypersalivation, repetitive swallowing, and tachycardia.

Retching is characterized as rhythmic, synchronous contractions of the diaphragm, abdominal muscles, and intercostals that occur against a closed glottis. There is a resultant increase in abdominal pressure with a concurrent decrease in intrathoracic pressure. This pressure gradient causes gastric contents to move up into the esophagus. The mouth is usually closed.

Vomiting is the forceful expulsion of gastric contents through the mouth. There is contraction of the external oblique and abdominal rectus muscles, and the hiatal portion of the diaphragm relaxes; this increases the pressure in the abdominal and the thoracic compartments. There is contraction of the pyloric portion of the stomach. Simultaneously, there is relaxation of the gastric fundus, cardia, and upper esophageal sphincter as the vomitus is brought up and out the mouth. The glottis closes to prevent aspiration.

The complex act of vomiting is not completely understood but is thought to be coordinated by a *vomiting center* located in the lateral reticular formation of the medulla (Fig. 20-2). The efferent pathways from the vomiting center are mainly through the vagus, phrenic, and spinal nerves. These pathways are responsible for the integrated response of the diaphragm, intercostals, abdominal muscles, stomach, and esophagus. The vomiting center is activated by afferent stimuli from a variety of sources. These include vagal and sympathetic impulses directly from the GI tract. Direct irritation of the stomach lining causes vomiting in this way. Other GI sources of afferent impulses include the pharynx, small bowel, colon, biliary system, and peritoneum. Receptors also are found outside the GI tract in the vestibular system, heart, and genitalia.

The other major source of impulses to the vomiting center is the CTZ. The CTZ is located in the area postrema, the floor of the fourth ventricle. Part of this area is located outside of the blood-brain barrier, enabling it to respond to endogenous and exogenous substances that activate vomiting. It is activated by hormones, peptides, medications, or toxins in the circulation, including opiates, digitalis, chemotherapy agents, salicylate, syrup of ipecac, and dopamine neurotransmitters.

The discovery of various neurotransmitters and their receptor sites within the medulla has improved the understanding and development of therapeutic agents. The CTZ area is rich in dopamine D₂ receptors, which are antagonized by drugs such as prochlorperazine, metoclopramide, and droperidol. The serotonin receptor has been found widely in the area postrema and the GI tract. It may act directly and through the release of dopamine. The serotonin receptor antagonists ondansetron and granisetron have been shown to be effective in preventing chemotherapy-induced nausea and vomiting. Concentrations of cholinergic and histamine receptors are found in the lateral vestibular nucleus and are important in

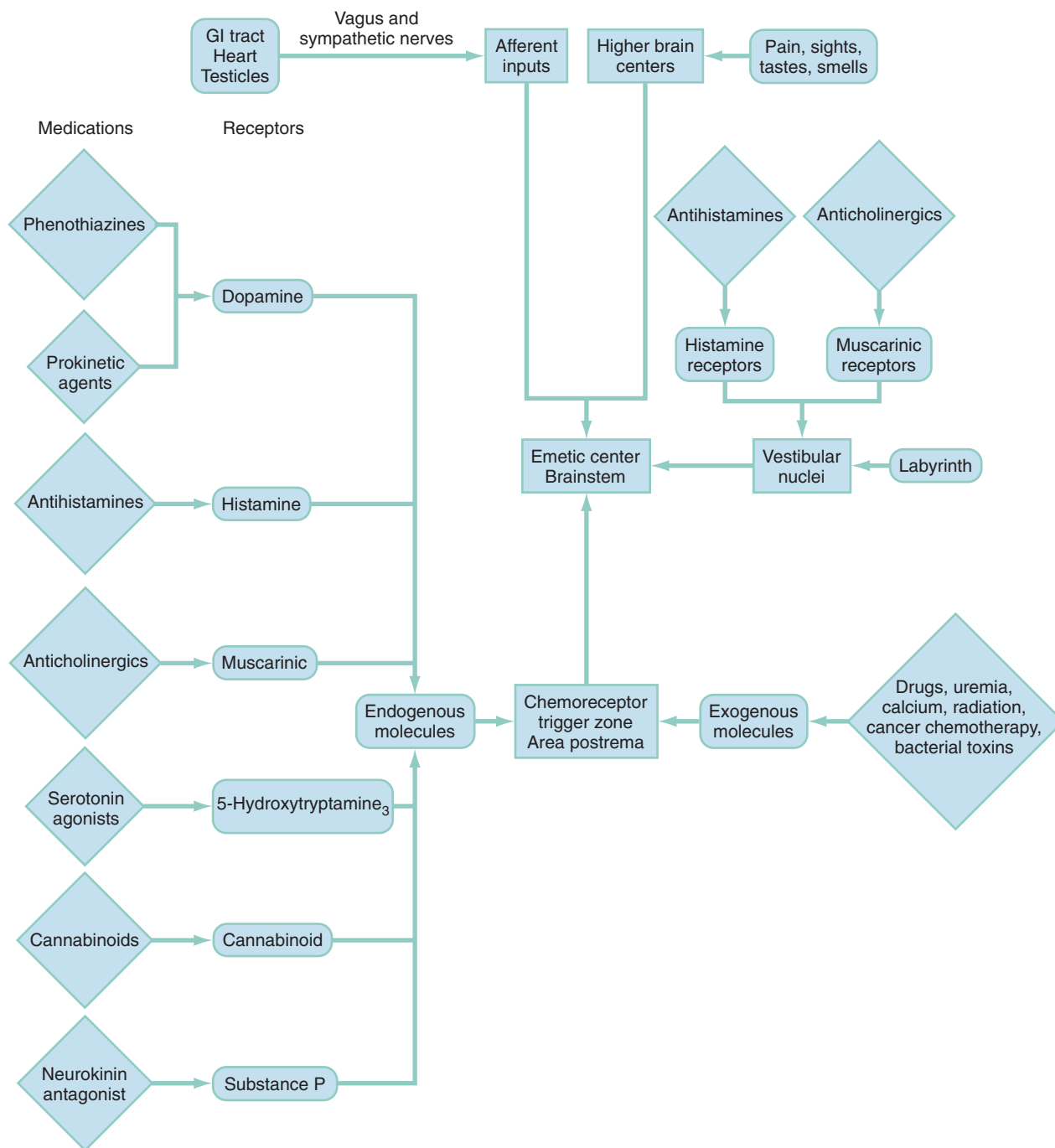


Figure 20-1. Pathophysiology of nausea and vomiting.

motion sickness. Meclizine, diphenhydramine, and scopolamine act by antagonizing these receptors. Cannabinoid receptors have been found to inhibit the emetic reflex.

Rumination is regurgitation of ingested food that subsequently is reswallowed or ejected. Rumination syndrome is found in infants, children, and mentally challenged adults, but rarely in adults with normal intelligence.

■ DIAGNOSTIC APPROACH

Differential Considerations

The differential diagnosis for nausea and vomiting is particularly broad in scope; almost any organ system can be involved (Table 20-1). Vomiting also can result in complications, which

must be considered in addition to the causes. The sequelae of vomiting may include the following various metabolic and traumatic lesions.

Hypovolemia is caused by loss of water and sodium chloride in the vomitus. The contraction of the extracellular fluid space leads to activation of the renin-angiotensin-aldosterone system.

Metabolic alkalosis is produced by loss of hydrogen ions in the vomitus. Many factors serve to maintain the alkalosis, including volume contractions, hypokalemia, chloride depletion, shift of extracellular hydrogen ions into cells, and increased aldosterone.

Hypokalemia is produced primarily by loss of potassium in the urine. The metabolic alkalosis leads to large amounts of sodium bicarbonate being delivered to the distal tubule. Secondary hyperaldosteronism from volume depletion causes

reabsorption of sodium and excretion of large amounts of potassium in the urine.

Mallory-Weiss tears typically result from a forceful bout of retching and vomiting. The lesion itself is a 1- to 4-cm tear through the mucosa and submucosa; 75% of cases occur in the stomach, with the remainder near the gastroesophageal junction. Bleeding usually is mild and self-limited; however, 3% of deaths from upper GI bleeds are due to Mallory-Weiss tears.

Boerhaave's syndrome refers to a perforation of all layers of the esophagus occurring as a result of forceful retching or

vomiting. The overlying pleura is torn so that there is free passage of esophageal contents into the mediastinum and thorax; 80% of cases involve the posterolateral aspect of the distal esophagus. Boerhaave's syndrome constitutes a surgical emergency. The mortality rate is 50% if surgical repair is not performed within 24 hours.

Aspiration of gastric contents is a concern in patients who have altered mental status or pulmonary findings after an episode of vomiting. Patients with pulmonary findings after vomiting need further evaluation for aspiration.

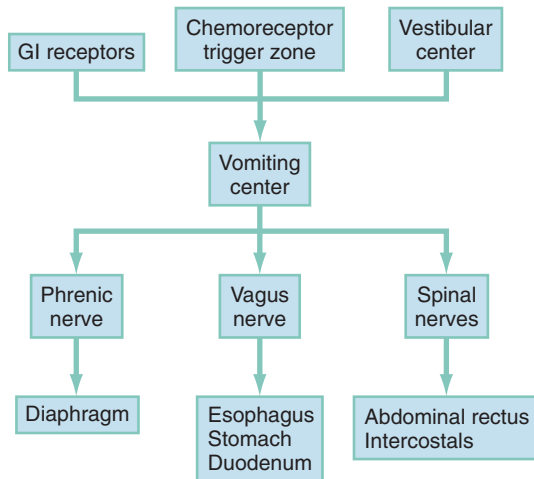


Figure 20-2. Vomiting process. GI, gastrointestinal.

Rapid Assessment and Stabilization

The initial assessment is directed toward the patient's hemodynamic status and identifying the critical causes or sequelae of vomiting (see [Table 20-1](#)). Data gathered include duration of vomiting, whether blood is in the vomitus, symptoms of volume depletion, and associated symptoms pointing to serious underlying disease. Physical findings include level of consciousness, abdominal examination, rapid neurologic screen for focality, and serial vital signs. Initial stabilization may include establishing intravenous access and fluid resuscitation in patients with signs of volume depletion, cardiac monitoring, and therapeutic measures directed toward specific underlying diseases (e.g., blood pressure control in severe hypertension).

Pivotal Findings

A thorough history and physical examination usually yield the underlying cause of nausea and vomiting.

Table 20-1 Differential Diagnosis of Nausea and Vomiting

ETIOLOGIC CATEGORY	CRITICAL DIAGNOSES	EMERGENT DIAGNOSES	NONEMERGENT DIAGNOSES
Gastrointestinal (GI)	Boerhaave's syndrome Ischemic bowel GI bleeding	Gastric outlet obstruction Pancreatitis Cholecystitis/cholangitis Bowel obstruction/ileus Ruptured viscus Appendicitis Peritonitis Spontaneous bacterial peritonitis	Gastritis Gastroparesis Peptic ulcer disease Inflammatory bowel disease Biliary colic Hepatitis Gastroenteritis
Neurologic	Intracerebral bleed Meningitis	Migraine CNS tumor Raised ICP	
Endocrine	DKA	Adrenal insufficiency Uremia	Thyroid
Pregnancy		Hyperemesis gravidarum	Nausea and vomiting of pregnancy
Drug toxicity		Acetaminophen Digoxin Aspirin Theophylline	
Therapeutic drug use			Aspirin Antibiotics Erythromycin Ibuprofen Chemotherapy
Drugs of abuse			Narcotics Narcotic withdrawal Alcohol
Genitourinary		Gonadal torsion	Urinary tract infection Poisoning Nephrolithiasis
Miscellaneous	Myocardial infarction Sepsis	Carbon monoxide Electrolyte disorders Organophosphate poisoning	Motion sickness Labyrinthitis

CNS, central nervous system; DKA, diabetic ketoacidosis; ICP, intracranial pressure.

Duration of the vomiting can lead to a diagnosis. *Acute* vomiting is vomiting occurring for less than 1 week and is associated with obstructive, ischemic, toxic, metabolic, infectious, neurologic, and postoperative causes. *Chronic* vomiting occurs with partial obstructions, motility disorders, and neurologic conditions or may be pregnancy-related or functional in origin.

Timing of the vomiting may be important. An acute onset of nausea and vomiting suggests gastroenteritis, pancreatitis, cholecystitis, or a drug-related side effect. Symptoms occurring primarily in the morning suggest pregnancy, although this pattern also may be seen with uremia, alcohol ingestion, or increased intracranial pressure. Delayed vomiting more than 1 hour after eating suggests gastric outlet obstruction or gastroparesis. Vomiting of material eaten more than 12 hours previously is pathognomonic for outlet obstruction. Nausea and vomiting for more than 1 month are considered chronic. Discrete episodes of intractable vomiting with intervening asymptomatic periods are considered cyclic.

Content of the vomitus may provide clues. The presence of bile indicates a patent connection between the duodenum and the stomach and essentially rules out a gastric outlet obstruction. Regurgitation of undigested food can suggest achalasia, esophageal stricture, or Zenker's diverticulum. Feculent material usually suggests a distal bowel obstruction but also may be seen with gastrocolic fistula or bacterial overgrowth of stomach contents in long-standing outlet obstruction.

Associated symptoms and signs may be helpful. Hypersalivation, defecation, tachycardia, bradycardia, atrial fibrillation, and termination of ventricular tachyarrhythmias are associated phenomena with nausea and vomiting. Chronic headaches with nausea and vomiting should raise the index of suspicion for an intracranial lesion. Also, vomiting without preceding nausea is typical of central nervous system pathology. The *social history* should include inquiries about alcohol or other substance abuse. The *past medical history* will reveal the pres-

ence of any GI disease or previous surgeries. Nutritional history is valuable in the consideration of failure to thrive in infancy. Finally, a thorough *medication list*, including over-the-counter drugs, should be included.

Physical Examination

The important physical examination findings are outlined in Table 20-2. During evaluation, findings of jaundice, lymphadenopathy, vertigo, fever, and goiter can help determine the etiology of the disease. Oral examination may reveal loss of dental enamel commonly seen with bulimia. Abdominal examination may reveal ascites, distention, hernias, abdominal tenderness and masses, organomegaly, or hyper- or hypoactive bowel sounds, with appropriate laboratory testing for occult blood in the stool. Determination of orthostatic vital signs may be valuable in patients with signs of dehydration, lightheadedness, generalized weakness, or toxic appearance. It also is important to evaluate neurologic status to rule out a central cause of a patient's symptoms, which includes cranial nerves, fundoscopic examination, and gait observation. Provocative testing for vertigo such as with the Nylan-Bárány test may elicit nausea and vomiting. Attentive physicians may elicit evidence of depression or anxiety that may lead to a psychiatric diagnosis.

In children, the examination should search for other diagnostic clues. A bulging fontanelle (meningitis), projective vomiting (pyloric stenosis), unusual odors (metabolic), visible bowel loops (obstruction), enlarged parotid, and loss of dental enamel (bulimia) point to specific etiologic disorders. Most of these disorders are age-dependent.

Ancillary Studies

Because of the broad differential diagnosis for nausea and vomiting, there is no standard panel of laboratory tests. Appropriate testing is determined by the specifics of the history and

Table 20-2 Physical Examination of the Patient with Nausea and Vomiting

ORGAN SYSTEM	FINDING	SUGGESTED DIAGNOSES
General	Poor skin turgor Dry mucous membranes	Dehydration
Vital signs	Fever Tachycardia/orthostatic changes	Gastroenteritis, cholecystitis, appendicitis, hepatitis Bowel perforation Dehydration
HEENT	Nystagmus Papilledema	Labyrinthitis Vertebrobasilar insufficiency Cerebellar infarct or bleed CPA tumor Increased ICP from CNS tumor or bleeding
Neck	Goiter	Thyroid disease
Lungs	Rales	Pneumonia
Heart	Arrhythmia Murmur	Acute myocardial infarction
Abdomen	Abdominal distention Peristaltic waves High-pitched bowel sounds Decreased bowel sounds Hernias or surgical scars Peritoneal signs	Bowel obstruction, gastroparesis Gastric outlet obstruction Bowel obstruction Ileus Possible bowel obstruction Appendicitis, cholecystitis Perforated viscus
Neurologic	Abnormal mental status Cerebellar findings Cranial nerve findings	CNS pathology

CNS, central nervous system; CPA, cerebellopontine angle; HEENT, head, eyes, ears, nose, throat; ICP, intracranial pressure.

physical examination. The following general guidelines regarding specific tests are useful.

Complete blood count: Most patients do not require a complete blood count. Elevated hemoglobin may suggest dehydration, but other tests are better for this purpose. An elevated white blood count is entirely nonspecific and of no discriminatory value.

Serum electrolytes: Measurement of serum electrolytes is not indicated in most cases of vomiting. Severe, protracted vomiting can cause a hypochloremic, hypokalemic metabolic alkalosis. Patients with this history or with clinical evidence of dehydration should undergo electrolyte testing. In general, serum electrolyte testing is indicated only in patients with symptoms lasting longer than 3 days or signs of significant dehydration who require intravenous fluid to replenish vascular volume.

Blood urea nitrogen and creatinine: Classically a blood urea nitrogen-to-creatinine ratio greater than 20:1 implies significant dehydration.

Serum lipase: Lipase determination is indicated in cases of suspected pancreatitis.

Urine tests: A urine pregnancy test should be performed in all women of childbearing age. Nitrites, leukocyte esterase, white blood cells, and bacteria indicate a urinary tract infection. Ketones may support a diagnosis of diabetic ketoacidosis or prolonged starvation state. Hematuria indicates a possible renal calculus.

Cultures: Blood cultures may be indicated in the fever patient with nausea and vomiting. Urine cultures may be necessary to determine an underlying cause, and stool cultures looking for enteric pathogens, parasites, or leukocytes may be valuable.

Liver function and ammonia tests: Liver function tests are indicated in cases of suspected hepatitis or biliary disease. Ammonia testing is useful if liver failure is suspected.

Serum drug levels: Serum drug levels may be important in patients on theophylline, digoxin, or salicylates, especially in elderly patients who are taking medication without supervision.

Abdominal imaging: Flat and upright plain radiographs are indicated only in cases of suspected bowel obstruction or ileus. Computed tomography (CT) scan of the abdomen has supplanted plain radiography for the evaluation of many patients with suspected obstruction because of the improved ability to discern the cause of the problem in addition to the presence of obstruction. An abdominal ultrasound study is indicated in cases of suspected choledocholithiasis or cholecystitis in adults, and suspected pyloric stenosis and intussusception in children. Imaging studies such as cranial CT scan or magnetic resonance imaging (MRI) may be needed for evaluation for possible central nervous system (CNS) trauma, tumor, or infectious causes.

Electrocardiogram: An electrocardiogram (ECG) is indicated in cases of suspected coronary artery ischemia.

Thyroid function tests: Although not usually available during the patient's stay in the emergency department (ED), thyroid function tests may indicate a thyroid cause for the vomiting.

■ DIFFERENTIAL DIAGNOSIS

Clinical and diagnostic findings are helpful in differentiating among the common and catastrophic causes of nausea and vomiting (Table 20-3). The differential diagnosis in adults is extensive; etiologic categories include medication-induced, infectious and toxic causes, disorders of the gastrointestinal tract, CNS causes, pregnancy-related, endocrine and metabolic disorders, radiation-induced, postoperative, unknown (as in cyclic vomiting), psychogenic, and other causes such as

acute myocardial infarction and acute graft-versus-host disease.

■ PEDIATRIC CONSIDERATIONS

The evaluation and management of pediatric patients with nausea and vomiting depend on age and likely causative disorders⁵ (Table 20-4). Mild degrees of reflux and associated regurgitation are common in the first few months of life, but vomiting in infancy can be associated with life-threatening illness. In the first week of life, obstructive lesions of the alimentary tract, inborn errors of metabolism, and serious infectious processes are associated with vomiting. After the first week of life, pyloric stenosis needs to be considered. The diagnosis of “feeding problems” should be considered a diagnosis of exclusion. After the first month of life, infections, metabolic diseases, cow's milk intolerance, failure to thrive, and subdural hematoma from abuse should be prime considerations. Thereafter, various disorders are associated with vomiting, including recurrent cyclic vomiting, acute surgical emergencies, food poisoning, toxic ingestion, Henoch-Schönlein purpura, pneumonia, and diabetic ketoacidosis. Anorexia nervosa and bulimia should be considered in teenagers with recurrent vomiting.⁵

■ MANAGEMENT

If an underlying cause for the nausea and vomiting is discovered, treatment of this disorder would take precedence. Decreased oral intake is a major cause of dehydration and malnutrition. If the patient is able to take oral liquids, sports drinks are preferred while avoiding citrus and highly sweetened drinks.⁶ Patients who are dehydrated and in whom intake of oral fluids is not possible or is contraindicated should be given intravenous fluids. Hypokalemia is rarely of clinical significance but may be found with profound vomiting secondary to contraction metabolic alkalosis. Treatment of the underlying condition with administration of intravenous fluids is indicated. Placement of a nasogastric tube is an option in cases such as persistent vomiting, gastroparesis, pancreatitis, and bowel obstruction.

Pharmacologic management of patients with nausea and vomiting is outlined in Figure 20-2. To allow the physician to make an appropriate choice for each patient, the pharmacologic therapies available may be classified into histamine antagonists, muscarinic antagonists, dopamine antagonists, and serotonin antagonists.

The phenothiazines are widely used as general-purpose antiemetics. These agents have multiple complex mechanisms of action. The antiemetic effect is apparently through blockade of the dopamine D₂ receptor in the CTZ. Prochlorperazine (Compazine), droperidol (Inapsine), haloperidol (Haldol), and promethazine (Phenergan) are commonly used medications in this class. Mild to moderate side effects are fairly common and include dystonic reactions and feelings of restlessness. These side effects may be treated with diphenhydramine (Benadryl) or benztropine (Cogentin). Although prochlorperazine was found to be more effective in reducing vomiting than promethazine, use of prochlorperazine has been reported to be associated with a 16% incidence of akathisia and a 4% incidence of dystonia, so patients should be advised about this potential and its mitigation with diphenhydramine or benztropine. Neuroleptic malignant syndrome, blood dyscrasias, and cholestatic jaundice have been documented rarely with use of phenothiazines.

The serotonin receptor antagonists, such as ondansetron, granisetron, and tropisetron, are a class of agents that have

Table 20-3 Disorders Commonly Associated with Vomiting

DISORDER	HISTORY	PREVALENCE	PHYSICAL EXAMINATION	USEFUL TESTS	COMMENTS
Nausea and vomiting of pregnancy (NVP)	Vomiting occurs predominantly in the morning. Associated breast tenderness. NVP typically starts in weeks 4–7, peaks in weeks 10–16, and disappears by week 20. Vomiting that begins after week 12 or continues past week 20 should prompt a search for another cause.	Very common Affects 75% of all pregnancies	Benign abdomen	Urine pregnancy test Serum electrolytes, urine ketones to exclude hyperemesis gravidarum	Consider NVP in all females of childbearing age. Prognosis for mother and infant is excellent. NVP is associated with a decreased risk of miscarriage, fetal growth retardation, and fetal mortality.
Hyperemesis gravidarum	Severe, protracted form of NVP; No universally accepted definition of the disease. Generally accepted hallmarks include 5% weight loss, ketonuria, and disturbance. Hyperemesis is associated with multiple gestation, molar pregnancy, and nulliparity.	Uncommon Affects <1% of pregnancies	Signs of dehydration Benign abdomen	β -hCG Urinalysis for ketones Serum electrolytes Ultrasound exam to exclude molar pregnancy or multiple gestation	Most studies have found no adverse outcomes for the fetus. A few studies, however, have shown a correlation with fetal growth retardation.
Gastroenteritis	Fever, diarrhea, and crampy abdominal pain. Vomiting and pain occur early, usually followed by diarrhea within 24 hr.	Very common	Benign abdomen	Usually not necessary	Early gastroenteritis, when only vomiting and periumbilical pain are present, may be confused with early appendicitis. Diarrhea is usually in the diagnosis of gastroenteritis.
Gastritis	Epigastric pain, belching, bloating, fullness, heartburn, and food intolerance. Use of NSAIDs or ETOH common.	Very common	Mild epigastric tenderness may be present.	Lipase and pregnancy test may be necessary to exclude other diagnoses.	Removal of inciting agent along with antacid therapy will resolve symptoms in most patients.
Peptic ulcer disease (PUD)	Epigastric pain present in 90% of cases. Classically, duodenal ulcer pain is relieved by food while gastric ulcer pain is made worse. Presence of severe pain should raise suspicion of perforation.	Very common	Mild epigastric tenderness	Hemoglobin if bleeding is suspected Heme-positive stool Upright abdominal film if perforation is suspected	Three major causes of PUD are NSAIDs, <i>H. pylori</i> infection, and hypersecretory states.

Biliary disease	Abdominal pain may be midepigastric or right upper quadrant (RUQ). Onset frequently after a fatty meal. May have history of similar episodes in the past.	Very common	RUQ tenderness present in most cases. If instructed to breathe deeply during palpation in the RUQ, the patient experiences heightened tenderness and inspiratory arrest (Murphy's sign).	WBC Lipase Serum bilirubin Alkaline phosphatase RUQ ultrasound exam	Normal temperature, WBC, and spontaneous resolution of symptoms suggest biliary colic. Fever, Murphy's sign, elevated WBC, and suggestive ultrasound indicate cholecystitis.
Myocardial infarction	Patients typically have substernal chest pain that may radiate to left arm or jaw. Often associated with dyspnea, diaphoresis, or dizziness.	Common	Patients often are anxious and in distress from pain. No diagnostic examination findings.	ECG (new Q waves, ST segment changes, or T wave inversions) CPK-MB/troponin	Not all patients present with chest pain. A subset of patients, particularly diabetics and the elderly, may present with only nausea, vomiting, and epigastric discomfort.
Diabetic ketoacidosis (DKA)	Polydipsia and polyuria occur early. Without treatment, altered mental status and coma may develop. In long-standing diabetics, DKA may be triggered by infection, trauma, MI, or surgery.	Common	"Fruity" breath odor results from serum acetone. Tachypnea occurs with attempts to "blow off" carbon dioxide to compensate for metabolic acidosis. Signs of dehydration may be present. Severe cases often manifest with altered mental status or coma.	Serum glucose, urine ketones, ABGs	DKA may be the first manifestation of diabetes in some patients. These patients often do not recognize the importance of polydipsia and polyuria. They often present complaining only of nausea, vomiting, and epigastric pain.
Pancreatitis	Patients present with epigastric pain, which often radiates to the back. Most cases are caused by gallstones or alcoholism. Other causes include hypercalcemia, hyperlipidemia, drugs (sulfas and thiazides), ERCP.	Common	Epigastric tenderness is present. Associated paralytic ileus may cause abdominal distention and decreased bowel sounds. Frank shock may be present in severe cases.	Lipase WBC, serum glucose, LDH, AST Hematoctrit, BUN, calcium, ABGs	Criteria correlating with higher mortality: <i>At admission</i> —Age >55 yr, WBC >16,000/mm ³ , glucose >200 dL, base deficit >4, LDH >350 IU/L, AST >250 F Units <i>Within 48 hours</i> —Hct drop of 10%, BUN >2 mg/dL, Po ₂ < 60 mm Hg, calcium <8 mg, fluid sequestration >4 L
Appendicitis	Abdominal pain classically begins in periumbilical region and later moves to right lower quadrant. Anorexia is common.	Common	Localized tenderness over right lower quadrant. Low-grade fever may be present.	WBC Abdominal CT	Early appendicitis can be a difficult diagnosis to make. It is still frequently missed on the first physician encounter.

Continued

Table 20-3 Disorders Commonly Associated with Vomiting—cont'd

DISORDER	HISTORY	PREVALENCE	PHYSICAL EXAMINATION	USEFUL TESTS	COMMENTS
Bowel obstruction	Classically, abdominal pain consists of intermittent cramps occurring at regular intervals. The frequency of the cramps varies with the level of the obstruction; the higher the level, the more frequent the cramps. The location of the pain also varies with the level of the obstruction; high obstruction causes epigastric pain, mid-level obstruction causes periumbilical pain, colonic obstruction causes hypogastric pain.	Common	Abdominal distention, mild diffuse tenderness, and high-pitched “tinkling” bowel sounds may be present. Thorough search for hernias should be performed.	Supine and upright plain abdominal films Abdominal CT	Adhesions, hernias, and tumors account for 90% of bowel obstructions. Other causes include intussusception, volvulus, foreign bodies, gallstone ileus, inflammatory bowel disease, stricture, cystic fibrosis, and hematoma.
Carbon monoxide (CO) poisoning	Headache is usually present. CO poisoning often occurs during winter months when furnaces are turned on. Family members may have similar symptoms if they also have been exposed.	Uncommon	No reliable signs of early CO poisoning	CO level	Because CO is a tasteless, odorless gas, patients may not realize they have been exposed. It is important to keep a high index of suspicion during the winter months.
Boerhaave's syndrome	Patients may have neck, chest, or epigastric pain. Forceful, protracted vomiting usually causes the tear. Most cases follow a bout of heavy eating and drinking. Other reported causes include childbirth, defecation, seizures, and heavy lifting.	Uncommon	Tachypnea, tachycardia, and hypotension may be present. Escaped air from the esophagus may produce subcutaneous emphysema. Air in the mediastinum produces a “crunching” sound as the heart beats (Hamman's sign).	CXR may show pleural effusion, widened mediastinum, or pneumothorax, or pneumomediastinum. Esophagogram using water-soluble contrast is definitive.	The classic presentation includes forceful vomiting, severe chest pain, subcutaneous emphysema, and multiple CXR findings. There is a growing body of evidence that most cases do not have this “classic” picture. In more subtle presentations, the diagnosis can be difficult to make.

ABGs, arterial blood gases; AST, aspartate aminotransferase; β -hCG, β -human chorionic gonadotropin; BUN, blood urea nitrogen; CK, creatine kinase; CT, computed tomography; CXR, chest radiography; DKA, diabetic ketoacidosis; ECG, electrocardiogram; ERCP, endoscopic retrograde cholangiopancreatography; ETOH, ethyl alcohol; LDH, lactate dehydrogenase; MI, myocardial infarction; NSAID, nonsteroidal anti-inflammatory drug; PUD, peptic ulcer disease; WBC, white blood cell.

Table 20-4 Etiology of Nausea and Vomiting in Pediatric Age Groups

ETIOLOGIC CATEGORY	NEWBORN	INFANT	CHILD	ADOLESCENT
Infectious	Sepsis, meningitis, UTI, thrush	Pneumonia, otitis media, thrush	Gastroenteritis	Gastroenteritis, URI
Anatomic	Atresia and webs, malrotation, stenosis, meconium ileus, Hirschsprung's disease	Pyloric stenosis, intussusception, Hirschsprung's disease	Bezoars, chronic granulomatous disease	PUD, superior mesenteric syndrome
Gastrointestinal	Reflux, overfeeding, gastric outlet obstruction, volvulus	Reflux, gastritis, milk intolerance	Appendicitis, pancreatic, hepatitis, other food intolerance	Achalasia, hepatitis
Neurologic	Subdural hematoma, hydrocephalus	Subdural hematoma	Neoplasia, migraine, Reye's syndrome, motion sickness, hypertension	Neoplasia, migraine, motion sickness, hypertension
Metabolic	Organic or amino acidemias, urea cycle defects, galactosemia, hypercalcemia, phenylketonuria, kernicterus	Hereditary fructose intolerance, disorders of fatty acid metabolism, uremia, adrenal hyperplasia, kernicterus	Diabetes, vitamin A excess	Diabetes, pregnancy, acute intermittent porphyria
Other	Idiopathic, cardiac failure	Rumination, cardiac failure	Cyclic vomiting syndrome, toxins, food poisoning, Munchausen syndrome by proxy	Psychogenic, anorexia

PUD, peptic ulcer disease; URI, upper respiratory infection; UTI, urinary tract infection.

Adapted from Li HK, Sunku BK: Vomiting and nausea. In Wyllie R, Hyams JS (eds): Pediatric Gastrointestinal and Liver Disease: Pathophysiology, Diagnosis, Management. Philadelphia, Saunders, 2005, pp 127–149.

Table 20-5 Commonly Used Medications for the Treatment of Nausea and Vomiting

MEDICATION	DOSE	COMMENTS
Promethazine (Phenergan)	<i>Adult:</i> 12.5–25 mg IV, IM, PO, or by rectum <i>Pediatric:</i> 0.25–1 mg/kg/dose q4–6 h prn IV, IM, PO, or by rectum; max 25 mg/dose	May be repeated every 4–6 hr, until cessation of vomiting. Dry mouth, dizziness, blurred vision. Boxed warning for use under 2 yrs old
Prochlorperazine (Compazine)	<i>Adult:</i> 5–10 mg IM, or PO; 2.5–10 mg IV; 25 mg by rectum <i>Pediatric:</i> 0.4 mg/kg/24 hr tid-qid PO or by rectum; 0.1–0.15 mg/kg/dose tid-qid IM; max 40 mg/24 hr	May be repeated every 4 hr by IV or IM or every 12 hr by rectum, until cessation of vomiting. Lethargy, hypotension, extrapyramidal effects
Metoclopramide (Reglan)	<i>Adult:</i> 10 mg IM or IV, may repeat q6 h <i>Pediatric:</i> 1–2 mg/kg/dose q2–6 h IV q2–3 hr	Dystonic reactions, tardive dyskinesia, neuroleptic malignant syndrome
Ondansetron (Zofran)	<i>Adult:</i> 4 mg IV single dose <i>Pediatric:</i> up to 40 kg: 0.1 mg/kg; >40 kg: 4 mg/dose IV single dose	Headache, dizziness, and musculoskeletal pain

IM, intramuscular; IV, intravenous.

generated much interest because of their beneficial effect in chemotherapy-induced emesis. Their principal site of action is the area postrema, although they also affect receptors in the GI tract. Several studies in small series of patients have looked at their effect in overdose of theophylline and acetaminophen. With both of these agents, overdose causes vomiting, and oral intake is required as part of therapy (multiple-dose charcoal and *N*-acetylcysteine). It is well documented that the vomiting often prevents effective oral therapy in patients with overdose of these agents. These studies showed that ondansetron stopped the vomiting and allowed oral therapy to proceed. The dose was 8 mg given intravenously over 20 minutes. The side effects of the serotonin receptor antagonists are mild and include headache and constipation.^{7,8}

The prokinetic agents are useful in patients with gastroparesis, gastroesophageal reflux disease, and other putative dysmotility syndromes. Metoclopramide (Reglan) has the most applicability in the ED. It has dopamine antagonist activity at the CTZ and exerts anticholinergic and antiserotonin effects. The primary effect is increased gastric emptying; the exact mechanism for this is not understood. Metoclopramide has

multiple antiemetic actions and may be used as a general-purpose agent. Other prokinetic agents, such as cisapride (Propulsid), do not cross the blood-brain barrier. They are not useful as general-purpose antiemetics. Prokinetic agents are used in patients with isolated gastric motility disorders. The most common side effects of metoclopramide are restlessness, drowsiness, and diarrhea. These effects are usually mild and transient.

Antihistamines are useful in nausea and vomiting associated with motion sickness and vertigo. Agents such as dimenhydrinate (Gravol, Dramamine) and meclizine (Antivert) directly inhibit vestibular stimulation and vestibular-cerebellar pathways. Their anticholinergic effect also may contribute to their effectiveness in vertigo and motion sickness. Antihistamines have some role as general antiemetics but are better used in the prevention of motion sickness; for nausea and vomiting, they are less effective than the phenothiazines. The most common side effects of antihistamines are drowsiness, blurred vision, dry mouth, and hypotension. The newer, less-sedating antihistamines are thought to be less effective as antiemetics.

The anticholinergic agent scopolamine in a transdermal patch (Transderm Scō p) or hyoscine (Buscopan) in an oral form may be used for prophylaxis and treatment of motion sickness. These agents also have mild efficacy in preventing cytotoxic chemotherapy-related nausea and vomiting but are not useful in the emergency department.

Benzodiazepine medications have been used for nausea and vomiting, with variable results. Limited studies have evaluated the efficacy of benzodiazepines in the treatment of hyperemesis gravidarum, in prophylaxis for emetogenic chemotherapy, and preoperatively for minor gynecologic surgery. Although this aspect was not directly measured in these studies, the studies inferred that part of the response may be related to the anxiolytic component. No studies have addressed the use of benzodiazepines to treat nonspecific nausea and vomiting in an ED population.

Many of the new medications for nausea and vomiting are first tested as agents for prevention and treatment related to chemotherapy and postoperative nausea and vomiting (PONV). A new oral neurokinin-1 antagonist, aprepitant (Emend), has been found to be an effective adjunctive agent for use in patients receiving cancer chemotherapy.⁹ Aprepitant blocks the effects of substance P in the brain. Currently, it is not indicated for use in patients with established nausea and vomiting.

The medication choice is directed at the underlying cause of the nausea and vomiting, if known, such as motion sickness, PONV, or nausea and vomiting related to cancer chemotherapy. For all other patients, the choice of antiemetic agent has not been well studied in emergency medicine. One study found droperidol to be more effective than prochlorperazine or metoclopramide as compared with placebo for moderate to severe nausea of any cause.¹⁰ The same limitation is true of preferred agents used in the field. One study found that ondansetron was moderately effective in the treatment of nausea and vomiting in this setting.¹¹

As with adults, the underlying cause of nausea and vomiting is first addressed in determining treatment choices.⁶ Most of the same agents used in adults are recommended for children in a weight-based dosing regimen. Ondansetron and metoclopramide have value for antiemetic treatment to reduce nausea and vomiting in pediatric patients. These agents are particularly effective in improving gastroenteritis patients' ability to maintain oral hydration.¹²

Special Situations

Medications such as antihistamines are frequently used to reduce the incidence of nausea and vomiting when opioid analgesics are administered in the ED for pain control. Studies have demonstrated that the incidence of nausea and vomiting related to opioid administration in the ED is low and that these medications have little efficacy in reducing nausea and vomiting.^{13,14}

Many agents have been advocated for the treatment of nausea and vomiting in pregnancy (NVP). The treatments include *nonpharmacologic*—avoiding triggers, dietary changes, acupuncture, acupressure, ginger, and behavioral therapy—and *pharmacologic*—pyridoxine, antihistamines, metoclopramide, ondansetron, or prochlorperazine. Hyperemesis gravidarum is treated essentially as for NVP. For mild symptoms, pyridoxine (vitamin B₆), acupressure, ginger, and administration of antiemetics including antihistamines, metoclopramide, ondansetron, prochlorperazine, and phenothiazines may be used. Pyridoxine, acupressure, and ginger are thought to be of benefit but are not commonly used in the ED. For severe symptoms, hospitalization, fluids, corticosteroids, and electrolyte replacement may be needed. No specific medication has been shown to be superior in the treatment of hyperemesis gravidarum.¹⁵

Treatment of PONV is well known. Approximately one third of the patients undergoing surgery may experience nausea and vomiting unless they receive appropriate prophylactic treatment, and the incidence is surgical procedure-dependent. Droperidol, metoclopramide, ondansetron, and dexamethasone have been used to reduce PONV.¹⁶ However, the need for antiemetic therapy during procedural sedation in the ED is not well studied. Many of the same drugs associated with PONV used by anesthesiologists in the operating room are used in the ED for procedural sedation. Nitrous oxide and propofol have been associated with a higher incidence of nausea and vomiting. Although the optimal medication for PONV has yet to be determined, ondansetron is considered a first-line agent in some studies; this recommendation could be extended to postprocedural sedation-related nausea and vomiting in the ED.

Chemotherapy-related nausea and vomiting may be seen in the ED. The chemotherapy-induced nausea and vomiting may be acute (up to 24 hours) or delayed (after 24 hours).¹⁷ The incidence of nausea and vomiting is correlated with the emetic potential of the chemotherapeutic agents, the patient's risk factors and other comorbid disorders, and antiemetic treatment. Patients commonly are given the serotonin antagonists dexamethasone and aprepitant for both immediate and delayed prophylaxis. Cannabinoids also have been used to control chemotherapy-induced nausea and vomiting. Choice of agents for treatment in the ED has not been studied, but for this indication the serotonin antagonists and aprepitants are used.

■ DISPOSITION

Hospital admission is appropriate when the patient has a significant underlying disease, has an unclear diagnosis and responds poorly to fluid and antiemetic therapy, continues to experience uncontrolled emesis refractory to medication, or is at the extremes of age with poor response to treatment. A category subject to broad interpretation is patients in whom the diagnosis is unclear and prospects for timely follow-up are poor (e.g., the patient has no family physician, lacks transportation, is indigent, habitually abuses drugs or alcohol, or has a language barrier). Discharge may be considered if no serious underlying illness is present, the response to fluid and antiemetic therapy is good, the patient is able to take clear liquids before discharge, and the prospects for follow-up and observation at home are favorable.

Close follow-up is arranged for most discharged patients, preferably with their primary care physician, in 24 to 48 hours. At discharge, the patient is prescribed medications as needed and is advised to restart oral intake with small feedings of a liquid diet with gradual return to a normal diet. Some experts have recommended the nausea and vomiting diet, which requires the least amount of gastric neuromuscular work. It is a three-step diet: Sports drinks and bouillon are recommended in step 1; soups are recommended in step 2; and foods that require the least amount of gastric "work" are recommended in step 3, such as meals high in protein and low in lipids.¹⁶ Clear instructions are given to return to the emergency department if there is a recurrence, change, or deterioration in symptoms.

Causes for nausea and vomiting frequently remain undiagnosed. Some cases declare themselves or resolve over time; reevaluation and close follow-up are imperative for patients with continuing symptoms. In patients with persistent or recurring symptoms, psychogenic causes or cyclic vomiting syndrome should be considered.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.

■ PERSPECTIVE

Abdominal pain is a common emergency department (ED) complaint but, for many reasons, is often diagnostically challenging. The nature and quality of abdominal pain may be difficult for the patient to convey. Physical examination findings with this complaint are variable and can be misleading. The location and severity of the pain may change over time. Benign-appearing symptoms and presentations may evolve into life-threatening conditions. Conversely, patients presenting with severe symptoms may carry a relatively benign diagnosis. All of these factors make evaluation of patients with acute abdominal pain challenging in the ED setting.

Epidemiology

Abdominal pain is a common presenting complaint, accounting for up to 10% of all ED visits. Some of the most common causes of acute abdominal pain are listed in [Table 21-1](#). Many patients present with pain and other symptoms that are not typical of any specific disease process. A specific diagnosis may not be possible in about one in every four individuals presenting with this chief complaint.¹ In addition, several adult groups deserve special consideration: the elderly (older than 65 years of age), the immunocompromised, and women of reproductive age.

Elderly patients with acute abdominal pain are more likely to have a life-threatening process as the cause of their pain. Conditions such as diverticulitis, ruptured abdominal aneurysm, or mesenteric ischemia may manifest atypically and be rapidly progressive. Decreased diagnostic accuracy, coupled with increased probability of severe disease, results in increased mortality in elderly patients with abdominal pain.²

Increasingly, emergency physicians are seeing patients in immunocompromised states secondary to HIV/AIDS, chemotherapy, and immunosuppressive drugs. For many reasons, these patients also prove challenging. Their clinical presentation can be misleading owing to atypical physical and laboratory findings, such as lack of fever or elevated white count. In regard to infection, the scope of the differential diagnosis also should be broader than usual.³⁻⁷ Presentations in the immunocompromised patient may be highly variable and subtle and are discussed in Chapter 181.

The evaluation of abdominal pain in women involves a differential diagnosis of considerable extent and often requires a more in-depth physical exam and further diagnostic testing. Pelvic organs may be the source of significant pathology in

both the pregnant and the nonpregnant patient. The possibility of ectopic pregnancy in women of reproductive age greatly increases the risk of serious disease with a high potential for misdiagnosis. During pregnancy the uterus becomes an abdominal rather than a pelvic organ and may displace the normal intraperitoneal contents, adding complexity to the evaluation of these patients.⁸ Nonpregnant patients require evaluation for various ovarian and uterine pathology states.

Pathophysiology

Pathology in the gastrointestinal and genitourinary tracts remains the most common source of pain perceived in the abdomen. Also, pain can arise from a multitude of other intra-abdominal and extra-abdominal locations ([Box 21-1](#)). Abdominal pain is derived from one or more of three distinct pain pathways: visceral, somatic, and referred.

Visceral pain results from stimulating autonomic nerves invested in the visceral peritoneum surrounding internal organs. It is often the earliest manifestation of a particular disease process. Distention of hollow organs by fluid or gas and capsular stretching of solid organs from edema, blood, cysts, or abscesses are the most common stimuli. This discomfort is poorly characterized and difficult to localize. If the involved organ is affected by peristalsis, the pain often is described as intermittent, crampy, or colicky. In general, visceral pain is perceived from the abdominal region that correlates with the embryonic somatic segment:

- *Foregut* structures (stomach, duodenum, liver, and pancreas) are associated with *upper abdominal pain*.
- *Midgut* derivatives (small bowel, proximal colon, and appendix) are associated with *periumbilical pain*.
- *Hindgut* structures (distal colon and genitourinary tract) are associated with *lower abdominal pain*.

Visceral pain can be perceived in a location remote from the actual disease process. Localization occurs with the extension of the disease process beyond the viscera. A classic example is that of the early periumbilical pain of appendicitis (midgut). When the parietal peritoneum becomes involved, the pain localizes to the right lower quadrant of the abdomen, the usual location of the appendix.

Somatic pain occurs with irritation of the parietal peritoneum. This is usually caused by infection, chemical irritation, or another inflammatory process. Sensations are conducted by

Table 21-1 Common Causes of Abdominal Pain

CAUSATIVE DISORDER/CONDITION	EPIDEMIOLOGY	ETIOLOGY	PRESENTATION	PHYSICAL EXAMINATION	USEFUL TEST(S)
Gastric, esophageal, or duodenal inflammation	Occurs in all age groups.	Caused by gastric hypersecretion, breakdown of mucoprotective barriers, infection, or exogenous sources.	Epigastric radiating or localized, associated with certain foods. Pain may be burning. In some cases, exacerbation in supine position.	Epigastric tenderness without rebound or guarding. Perforation or bleeding leads to more severe clinical findings.	Uncomplicated cases are treated with antacids or histamine H ₂ blockers before invasive studies are contemplated. Gastroduodenoscopy is valuable in diagnosis and biopsy. Testing for <i>H. pylori</i> with blood or biopsy specimens. If perforation is suspected, an upright chest radiograph is obtained early to rule out free air. CT may be beneficial.
Acute appendicitis	Peak age in adolescence and young adulthood; less common in children and elders. Higher perforation rate in women, children, and elders and in pregnancy. Mortality rate is 0.1% but increases to 2–6% with perforation.	Appendiceal lumen obstruction leads to swelling, ischemia, infection, and perforation.	Epigastric or periumbilical pain migrates to RLQ over 8–12 hr (50–60%). Later presentations associated with higher perforation rates. Pain, low-grade fever (15%), and anorexia (80%) common; vomiting less common (50–70%).	Mean temperature 38°C (100.5°F). Higher temperature associated with perforation. RLQ tenderness (90–95%) with rebound (40–70%) in majority of cases. Rectal tenderness in 30%.	Leukocyte count usually elevated or may show left shift. Urinalysis may show sterile pyuria. CT is sensitive and specific. US may have use in women, pregnancy, and children with RLQ pain.
Biliary tract disease	Peak age 35–60 yr; rare in patients younger than 20. Female-to-male ratio of 3:1. Risk factors include multiparity, obesity, alcohol intake, and use of birth control pills.	Passage of gallstones causes biliary colic. Impaction of a stone in cystic duct or common duct causes cholecystitis or cholangitis.	Crampy RUQ pain radiates to right subscapular area. Prior history of pain is common. May have nausea or postprandial pain. Longer duration of pain favors diagnosis of cholecystitis or cholangitis.	Temperature normal in biliary colic, elevated in cholecystitis and cholangitis. RUQ tenderness, rebound, and jaundice (less common) may be present.	WBC count elevated in cholecystitis and cholangitis. Lipase and liver function tests may help differentiate this from gastritis or ulcer disease. Ultrasound shows wall thickening, pericholecystic fluid, stones, or duct dilatation. Hepatobiliary scintigraphy diagnoses gallbladder function.
Ureteral colic	Average age 30–40 yr, primarily in men. Prior history or family history of stones is common.	Family history, gout, <i>Proteus</i> infection. Renal tubular acidosis and cystinuria lead to stone formation.	Acute onset of flank pain radiating to groin. Nausea, vomiting, and pallor are common. Patient usually writhing in pain.	Vital signs usually normal. Tenderness on CVA percussion with benign abdominal examination.	Urinalysis usually shows hematuria. Noncontrast CT is sensitive and specific. US with fluid bolus useful diagnostically.

Diverticulitis	Incidence increases with advancing age, affects males more often than females. Recurrences are common. Often called “left-sided” appendicitis.	Colonic diverticula may become infected or perforated or cause local colitis. Obstruction, peritonitis, abscesses, fistulas result from infection or swelling.	Change in stool frequency or consistency commonly reported. LLQ pain is common. Associated with fever, nausea/vomiting; rectal bleeding may be seen.	Fever usually of low grade. LLQ pain without rebound is common. Stool may be heme-positive.	Results on most tests usually normal. Plain radiographs may show obstruction or mass effect. CT is often diagnostic.
Acute gastroenteritis	Common diagnosis. Seasonal. Most common misdiagnosis of appendicitis. May be seen in multiple family members. History of travel or immune compromise.	Usually viral. Consider invasive bacterial or parasitic in prolonged cases, in travelers, or immune-compromised patients.	Pain usually poorly localized, intermittent, crampy, and diffuse. Diarrhea is key element in diagnosis; usually large-volume, watery. Nausea and vomiting usually begin before pain.	Abdominal examination usually nonspecific without peritoneal signs. Watery diarrhea or no stool noted on rectal examination. Fever is usually present.	Usually symptomatic care with antiemetics and volume repletion. Heme-positive stools may be a clue to invasive pathogens. Key is not using this as a “default” diagnosis and missing more serious disease.
Constipation and obstipation	More common in females, the elderly, the very young, and patients on narcotics.	Idiopathic or hypokinesia secondary to disease states (low motility) or exogenous sources (diet, medications).	Abdominal pain; change in bowel habits.	Variable, nonspecific without peritoneal signs. Rectal exam may reveal hard stool or impaction.	Radiographs may show large amounts of stool. This is a diagnosis of exclusion.
Nonspecific abdominal pain	More common in persons of young and middle age, women of childbearing age or persons of low socioeconomic status, and patients with psychiatric disorders. Up to 10% of patients older than 50 years of age will have intra-abdominal cancer.	Unknown. Early or undiagnosed presentation of pathologic conditions.	Variable but tends to be chronic or recurrent.	Variable but no peritoneal signs. Rectal exam should be done to evaluate for subtle signs of pathology, including heme-positive stool, fistulas, and fissures.	Variable and often can be done on an outpatient basis.

CT, computed tomography; CVA, costovertebral angle; LLQ, left lower quadrant; LUQ, left upper quadrant; RLQ, right lower quadrant; RUQ, right upper quadrant; US, ultrasonography; WBC, white blood cell.

BOX 21-1

IMPORTANT EXTRA-ABDOMINOPELVIC CAUSES OF ABDOMINAL PAIN

Thoracic

Myocardial infarction/unstable angina
Pneumonia
Pulmonary embolism
Herniated thoracic disk (neuralgia)
Pericarditis/myocarditis

Genitourinary

Testicular torsion

Abdominal Wall

Muscle spasm
Muscle hematoma
Herpes zoster

Infectious

Streptococcal pharyngitis (more often in children)
Rocky Mountain spotted fever
Mononucleosis

Systemic

Diabetic ketoacidosis
Alcoholic ketoacidosis
Uremia
Sickle cell disease
Porphyria
Systemic lupus erythematosus
Vasculitis
Glaucoma
Hyperthyroidism

Toxic

Methanol poisoning
Heavy metal toxicity
Scorpion bite
Snake bite
Black widow spider bite

Adapted from Purcell TB: Nonsurgical and extraperitoneal causes of abdominal pain. *Emerg Med Clin North Am* 7:721, 1989.

the peripheral nerves and are better localized than the visceral pain component. [Figure 21-1](#) illustrates some more typical pain locations corresponding to specific disease entities. Somatic pain is often described as intense and constant. As disease processes evolve to peritoneal irritation with inflammation, better localization of the pain to the area of pathology generally occurs.

Referred pain is defined as pain felt at a distance from its source because peripheral afferent nerve fibers from many internal organs enter the spinal cord through nerve roots that also carry nociceptive fibers from other locations, as illustrated in [Figure 21-2](#). This makes interpretation of the location of noxious stimuli difficult for the brain. Both visceral pain and somatic pain can manifest as referred pain. Two examples of referred pain are the epigastric pain associated with an inferior myocardial infarction and the shoulder pain associated with blood in the peritoneal cavity irritating the diaphragm.

Gynecologic and obstetric presentations are discussed in other chapters. Notably, any abdominal pain in a female may represent referred pain from pelvic structures or an extension of a pelvic process, as in the case of perihepatic inflammation with pelvic inflammatory disease.

■ DIAGNOSTIC APPROACH

The clinical approach should focus on early stabilization, history, physical examination, and any ancillary tests collectively facilitating appropriate management and disposition plans.

Differential Considerations

Classically, potential diagnoses are divided into intra-abdominopelvic (intraperitoneal, retroperitoneal, and pelvic) causes (e.g., appendicitis, cholecystitis, pancreatitis) and extra-abdominopelvic processes (e.g., pneumonia, myocardial infarction, ketoacidosis).

Although significant morbidity and mortality can result from many disorders causing abdominal pain, a few processes warrant careful consideration in the ED. [Table 21-2](#) lists important potentially life-threatening nontraumatic causes of abdominal pain. This group represents the major etiologic disorders likely to be associated with hemodynamic compromise and for which early therapeutic intervention is critical.

Rapid Assessment and Stabilization

As with any complaint, triage is the first critical step in management. Most patients presenting with abdominal pain do not have hemodynamic instability, but up to 7% of these patients may have a life-threatening process. This percentage is higher in elders and immunocompromised patients.¹

Physiologically compromised patients should be brought to a treatment area immediately and resuscitation initiated. Profound shock or protracted emesis can lead to airway compromise necessitating intubation. These patients are often severely volume depleted and require rapid intravenous access and volume resuscitation with an isotonic crystalloid solution, titrated to a physiologic endpoint.

Extreme conditions such as ruptured abdominal aortic aneurysm, massive gastrointestinal hemorrhage, ruptured spleen, and hemorrhagic pancreatitis may require blood or blood product replacement. Bedside ultrasonography can be used to quickly evaluate patients for free intraperitoneal fluid, volume status, and presence of aortic pathology. Ultrasound assessment should be part of the initial physical examination and can be invaluable in guiding treatment and disposition. Because any of the immediately life-threatening entities may necessitate surgical intervention or management, early surgical consultation is indicated.

Pivotal Findings**History**

A careful and focused history is central to unlocking the puzzle of abdominal pain. [Box 21-2](#) lists some historical questions with high yields for serious pathology. Language and cultural differences may influence accurate communication and mutual understanding.

Abrupt onset often is indicative of a more serious cause; however, delayed presentations also may represent a surgical condition. Surgical causes of abdominal pain are more likely to manifest with pain first, followed by nausea and vomiting, rather than with nausea and vomiting followed by pain. Localization and pain migration also are helpful components of the pain history. Diffuse pain generally is nonsurgical, but it may represent the early visceral component of a surgical process. Colicky pain is indicative of hollow viscus distention, and

Figure 21-1. Differential diagnosis of acute abdominal pain. CHF, congestive heart failure; GERD, gastroesophageal reflux disease; LLL, left lower lobe; RLL, right lower lobe.

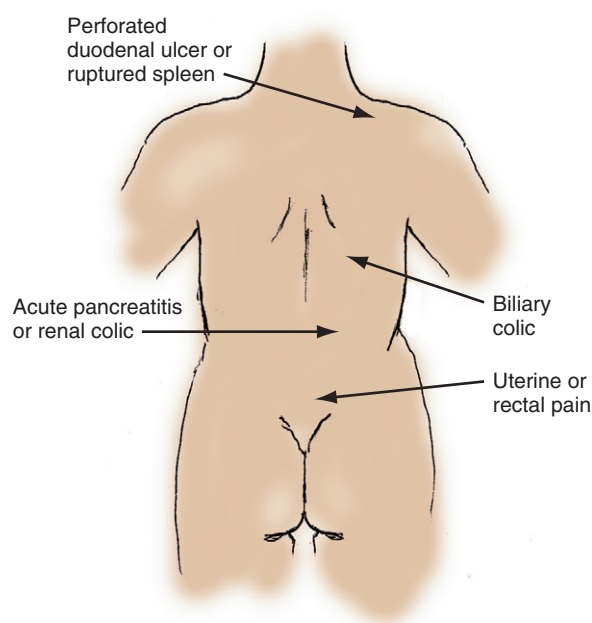
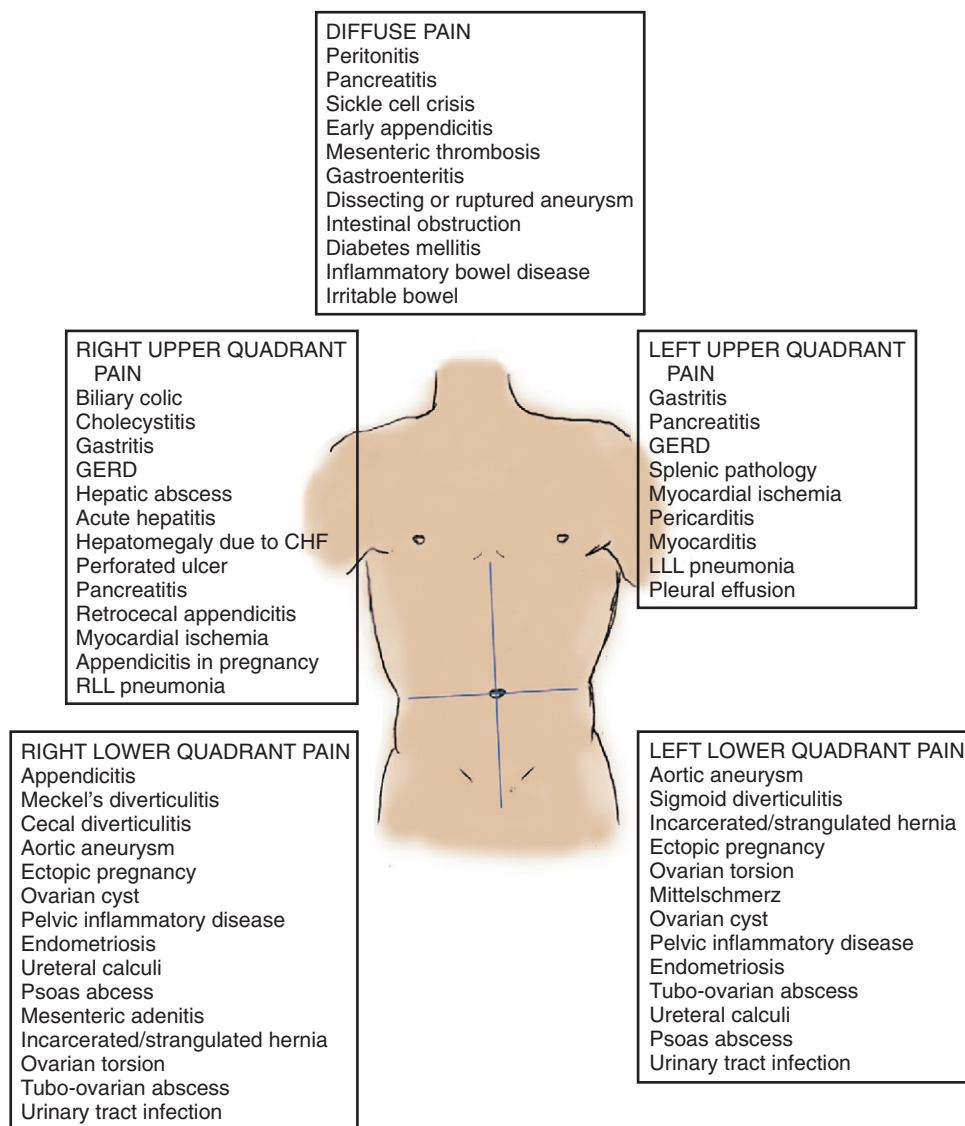


Figure 21-2. Common locations of referred pain from abdominal etiology.

duration and time of colic may give clues to the identity of the culprit organ, as displayed in [Figure 21-3](#).

The severity and descriptive nature of the pain are the most subjective aspects of the pain history, but a few classical descriptions are recognized, such as the following:

- The diffuse, severe, colicky pain of bowel obstruction
- The “pain out of proportion to examination” observed in patients with mesenteric ischemia
- The radiation of pain from the epigastrium straight through to the midback associated with pancreatitis, either related to primary organ inflammation or secondary to a penetrating ulcer
- The radiation of pain to the left shoulder or independent pain in the left shoulder associated with splenic pathology, diaphragmatic irritation, or free intraperitoneal fluid
- The onset of pain associated with syncope seen in perforation of gastric or duodenal ulcer, ruptured aortic aneurysm, or ruptured ectopic pregnancy

Physical Examination

The objective evaluation begins with measurement of the vital signs. Significant tachycardia and hypotension are indicators that hypovolemia or sepsis may be present. Tachypnea

Table 21-2 Potentially Life-threatening Causes of Abdominal Pain

CAUSE	EPIDEMIOLOGY	ETIOLOGY	PRESENTATION	PHYSICAL EXAMINATION	USEFUL TOOL(S)
Ruptured ectopic pregnancy	Occurs in females of childbearing age. No method of contraception prevents ectopic pregnancy. Approximately 1 in every 100 pregnancies.	Risk factors include nonwhite race, older age, history of STD or PID, infertility treatment, intrauterine contraceptive device placed within the past year, tubal sterilization, and previous ectopic pregnancy.	Severe, sharp constant pain localized to the affected side. More diffuse abdominal pain with intraperitoneal hemorrhage. Signs of shock may be present. Midline pain tends not to be ectopic pregnancy.	Shock or evidence of peritonitis may be present. Lateralized abdominal tenderness. Localized adnexal tenderness or cervical motion tenderness increase the likelihood of ectopic pregnancy. Vaginal bleeding does not have to be present.	β -hCG testing necessary in all females of childbearing age (10–55 yr); combined with ultrasonography, preferably transvaginal in early pregnancy, usually is diagnostic. FAST exam is useful in evaluating for free fluid in patients with shock or peritonitis.
Ruptured or leaking abdominal aneurysm	Incidence increases with advancing age. More frequent in men. Risk factors include HTN, DM, smoking, COPD, and CAD.	Exact etiology is undetermined. Contributing factors include atherosclerosis, genetic predisposition, HTN, connective tissue disease, trauma, and infection.	Patient often asymptomatic until rupture. Acute epigastric and back pain often associated with or followed by syncope or signs of shock. Pain may radiate to back, groin, or testes.	Vital signs may be normal (in 70%) to severely hypotensive. Palpation of a pulsatile mass is usually possible in aneurysms 5 cm or greater. The physical examination may be nonspecific. Bruits or inequality of femoral pulses may be evident.	Abdominal plain films abnormal in 80% of cases. Ultrasound can define diameter and length but can be limited by obesity and bowel gas. FAST exam can be helpful in evaluating for leak by looking for free fluid. Spiral CT test of choice in stable patients.
Mesenteric ischemia	Occurs most commonly in elders with CV disease, CHF, cardiac dysrhythmias, DM, sepsis, and dehydration. Responsible for 1 of 1000 hospital admissions. Mortality 70%. Mesenteric venous thrombosis associated with hypercoagulable states, hematologic inflammation, and trauma.	20–30% of lesions are nonocclusive. The causes of ischemia are multifactorial, including transient hypotension in the presence of preexisting atherosclerotic lesion. The arterial occlusive causes (65%) are secondary to emboli (75%) or acute arterial thrombosis (25%).	Severe pain, colicky, that starts in periumbilical region and then becomes diffuse. Often associated with vomiting and diarrhea. Sometimes postprandial. “Mesenteric or abdominal angina.”	Early examination results can be remarkably benign in the presence of severe ischemia. Bowel sounds often still present. Rectal examination important because mild bleeding with positive guaiac stools can be present.	Often a pronounced leukocytosis is present. Elevations of amylase and creatine kinase levels are seen. Metabolic acidosis due to lactic acidemia is often seen with infarction. Plain radiographs of limited benefit. CT, MRI, and angiography are accurate to varying degrees.

Intestinal obstruction	Peaks in infancy and older age. More common with history of previous abdominal surgery.	Adhesions, carcinoma, hernias, abscesses, volvulus, and infarction. Obstruction leads to vomiting, “third spacing” of fluid, or strangulation and necrosis of bowel.	Crampy diffuse abdominal pain associated with vomiting.	Vital signs usually normal unless dehydration or bowel strangulation has occurred. Abdominal distention, hyperactive bowel sounds, and diffuse tenderness. Local peritoneal signs indicate strangulation.	Elevated WBC count suggests strangulation. Electrolytes may be abnormal if associated with vomiting or prolonged symptoms. Abdominal radiographs and CT are useful in diagnosis.
Perforated viscus	Incidence increases with advancing age. History of peptic ulcer disease or diverticular disease common.	More often a duodenal ulcer that erodes through the serosa. Colonic diverticula, large bowel, and gallbladder perforations are rare. Spillage of bowel contents causes peritonitis.	Acute onset of epigastric pain is common. Vomiting in 50%. Fever may develop later. Pain may localize with omental walling off of peritonitis. Shock may be present with bleeding or sepsis.	Fever, usually of low grade, is common; worsens over time. Tachycardia is common. Abdominal examination reveals diffuse guarding and rebound. “Boardlike” abdomen in later stages. Bowel sounds are decreased.	WBC count usually elevated due to peritonitis. Amylase may be elevated; LFT results are variable. Upright radiographic view reveals free air in 70–80% of cases with perforated ulcers.
Acute pancreatitis	Peak age in adulthood; rare in children and elders. Male preponderance. Alcohol abuse and biliary tract disease are risk factors.	Alcohol, gallstones, hyperlipidemia, hypercalcemia, or endoscopic retrograde pancreatography causing pancreatic damage, saponification, and necrosis. ARDS, sepsis, hemorrhage, and renal failure are secondary.	Acute onset of epigastric pain radiating to the back. Nausea and vomiting are common. Pain disproportionate to physical findings. Adequate volume repletion is important in the initial therapy.	Low-grade fever common. Patient may be hypotensive or tachypneic. Some epigastric tenderness usually present. Because pancreas is retroperitoneal organ, guarding or rebound not present unless condition is severe. Flank ecchymosis or periumbilical process is hemorrhagic.	Lipase determination is test of choice. Ultrasound exam may show edema, pseudocyst, or biliary tract disease. CT scan may show abscesses, necrosis, hemorrhage, or pseudocysts. CT is ordered if severe acute pancreatitis is suspected. Rule out gallstones with ultrasound exam.

ARDS, acute respiratory distress syndrome; β -hCG, β human chorionic gonadotropin; CAD, coronary artery disease; CHF, congestive heart failure; COPD, chronic obstructive pulmonary disease; CT, computed tomography; CV, cerebrovascular; DM, diabetes mellitus; FAST, focused assessment with sonography in trauma; HTN, hypertension; LFT, liver function test; MRI, magnetic resonance imaging; PID, pelvic inflammatory disease; STD, sexually transmitted disease; WBC, white blood cell.

BOX 21-2 HIGH-YIELD HISTORICAL QUESTIONS

1. *How old are you?* Advanced age means increased risk.
2. *Which came first—pain or vomiting?* Pain first is worse (i.e., more likely to be caused by surgical disease).
3. *How long have you had the pain?* Pain for less than 48 hours is worse.
4. *Have you ever had abdominal surgery?* Consider obstruction in patients who report previous abdominal surgery.
5. *Is the pain constant or intermittent?* Constant pain is worse.
6. *Have you ever had this before?* A report of no prior episodes is worse.
7. *Do you have a history of cancer, diverticulosis, pancreatitis, kidney failure, gallstones, or inflammatory bowel disease?* All are suggestive of more serious disease.
8. *Do you have human immunodeficiency virus (HIV)?* Consider occult infection or drug-related pancreatitis.
9. *How much alcohol do you drink per day?* Consider pancreatitis, hepatitis, or cirrhosis in patients with history or signs of significant intake.
10. *Are you pregnant?* Test for pregnancy—consider ectopic pregnancy.
11. *Are you taking antibiotics or steroids?* Effects of these drugs may mask infection.
12. *Did the pain start centrally and migrate to the right lower quadrant?* High specificity for appendicitis.
13. *Do you have a history of vascular or heart disease, hypertension, or atrial fibrillation?* Consider mesenteric ischemia and abdominal aneurysm.

From Colucciello SA, Lukens TW, Morgan DL: Abdominal pain: An evidence-based approach. *Emerg Med Pract* 1:2, 1999.

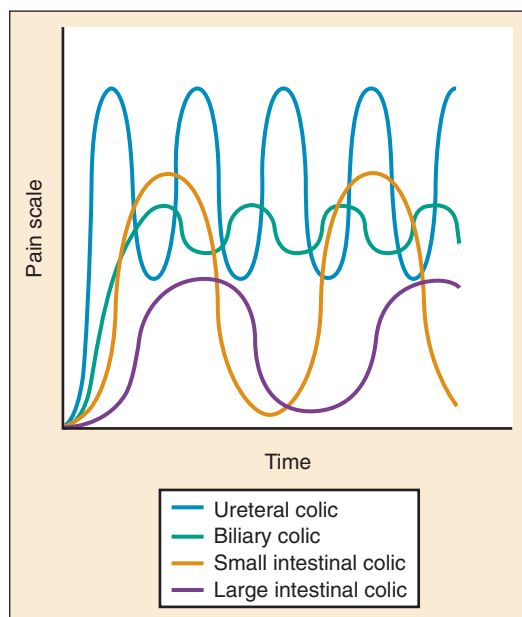


Figure 21-3. The characteristics of colicky abdominal pain.

may be an indication of metabolic acidosis from gangrenous viscera or sepsis, hypoxemia from pneumonia, or simply a catecholamine-induced reaction to pain. Elevated temperature often is associated with intra-abdominal infections. Although important, vital signs may be misleading and should be inter-

preted in the context of the entire presentation. Tachycardia may develop late for various reasons in hypovolemia. Temperature often is normal in elderly patients with laparotomy-proven intraperitoneal infections.⁹ Septic elderly patients also may present with hypothermia.

A thorough abdominal examination is an essential part of evaluation of the patient with abdominal pain. This requires properly positioning the patient supine and exposing the abdomen. The examination should begin with inspection for any signs of trauma, bruising, or skin lesions. The patient should be asked to localize the area of maximal tenderness by pointing with one finger. The abdomen can be mentally divided into four quadrants: right upper, right lower, left upper, and left lower; each area is then examined individually. Tenderness in one quadrant often corresponds with the location of the diseased organ, which will direct the workup (see Fig. 21-1). Some disease processes may manifest with pain that is not exclusively within one specific quadrant, such as the suprapubic pain of a urinary tract infection or the midepigastic pain of a gastric ulcer. Although 80% of patients with suspected appendicitis present with right lower quadrant abdominal tenderness, 20% of patients with proven appendicitis do not.¹⁰

Rectal examination may have limited use in the evaluation of abdominal pain, except that associated with intraluminal gastrointestinal hemorrhage, prostatitis, or perirectal disease. The main utility of the rectal examination is in the detection of heme-positive stool, anal fissures or fistulas, or stool impaction. Rectal examination has not been shown to increase diagnostic accuracy for appendicitis when added to external physical examination of the abdomen.¹¹

The abdominal evaluation should include a pelvic examination in female patients with lower abdominal pain or an otherwise uncertain diagnosis. The pelvic exam should be done early in the evaluation of the female patient with abdominal pain to help differentiate an abdominal from a pelvic source. This information is helpful in choosing an imaging modality. Pelvic ultrasound exam is helpful in evaluating uterine and ovarian pathology, whereas computed tomography (CT) is more beneficial in evaluation of suspected intra-abdominal pathology. Although the pelvic exam may guide the initial choice of imaging modality, overlap in exam findings is common. For example a patient with right lower quadrant tenderness may have both right adnexal tenderness and tenderness over McBurney's point—necessitating exclusion of both appendicitis and ovarian torsion. The diagnosis highest on the differential list should be ruled out first using the corresponding imaging modality.

In the male patient with abdominal pain, the urogenital system should be examined. Diseases such as prostatitis, orchitis, and epididymitis commonly cause abdominal pain in males. Furthermore, inguinal hernias are more common in males, with the possibility of strangulation or incarceration in the inguinal canal making a thorough genitourinary examination mandatory.

In view of the evolving nature of abdominal pain, repetitive examinations may be useful. This is common practice with respect to suspected appendicitis and has improved the diagnostic accuracy in patients whose presentations were atypical.²

Ancillary Testing

Urinalysis and testing for pregnancy are perhaps the most time- and cost-effective adjunctive laboratory tests available. Results often can be obtained quickly, so the former can lead to an early diagnosis and the latter may significantly affect

further evaluation and management approaches. It is necessary to interpret urinalysis results within the context of the patient's clinical picture. Pyuria, with or without bacteriuria, often is present in a variety of conditions besides a simple urinary tract infection. For example, appendicitis may feature sterile pyuria.¹² Similarly, hematuria usually is present with the relatively benign condition of nephrolithiasis but also may indicate an abdominal aortic aneurysm.

Complete blood counts frequently are ordered for patients with abdominal pain, but findings seldom are contributory to a diagnosis. Despite the association of elevated white blood cell (WBC) counts with many infectious and inflammatory processes, the WBC count is neither sufficiently sensitive nor specific to be considered a discriminatory test to help establish or rule out a serious cause for the pain. Even serial WBC counts have failed to differentiate surgical from nonsurgical conditions. The WBC count is therefore not helpful for diagnosis. Serum electrolytes, even in the presence of protracted emesis or diarrhea, are abnormal in less than 1% of patients. These studies are not indicated for most patients in the absence of another indication. Blood urea nitrogen concentrations can be elevated in gastrointestinal hemorrhage and dehydration, but such conditions are better detected and quantified by history and physical examination. Increased serum creatinine usually is indicative of renal dysfunction. Blood glucose, anion gap, and serum ketone determinations are useful in diabetic ketoacidosis, one cause of acute abdominal pain and tachypnea.

Liver enzymes and coagulation studies are helpful only in a small subset of patients with suspected liver disease.¹³ If pancreatitis is suspected, the most useful diagnostic result is serum lipase elevated to at least double the normal value, because it is more specific and more sensitive than serum amylase for this process. Measurement of serum amylase is of no value if a serum lipase level is available.¹⁴ Serum phosphate and serum lactate levels are elevated late in bowel ischemia, and such determinations may be useful if this entity is suspected but cannot be considered either sufficiently sensitive or specific to establish or exclude the diagnosis on their own.

Plain radiography of the abdomen has limited usefulness in the evaluation of acute abdominal pain. Suspected bowel obstruction, foreign body, and perforated viscus are the main indications. CT of the abdomen has become the imaging modality of choice with nonobstetric abdominal pain. It allows visualization of both intraperitoneal and extraperitoneal structures and has a high degree of accuracy, establishing a diagnosis in more than 95% of cases in one study¹⁵ and increasing the confidence in diagnosis in another.¹⁶ Incidental findings are common on CT scans and may lead to a diagnosis. Patients who undergo CT have a change in diagnosis more often than those who do not.¹⁷ The proper execution and interpretation

of CT studies will reduce morbidity, mortality, and medical expenses.^{18,19}

CT has increased diagnostic utility in elderly patients for several reasons. Older people with abdominal pain may have twice the rate of surgery²⁰⁻²² and a six- to eight-fold increase in mortality compared with younger adults.^{20,21} Furthermore, evaluation of abdominal pain in the elderly often is more challenging owing to unreliable findings on physical examination including vital signs, difficulties in history taking, physiologic age-related changes, and comorbid conditions. In the elderly population, CT results change management or disposition decisions in a significant proportion of patients.²³ Table 21-3 lists the most common findings on CT scans in elderly patients with abdominal pain.

Some controversy surrounds the use of oral contrast in abdominal CT in the critically ill ED patient. Technologic advances have improved image acquisition and resolution, and preliminary studies have shown that intravenous contrast alone may now be adequate in the evaluation of certain suspected pathologic processes, such as solid organ or bowel wall disease.²⁴ CT with intravenous contrast alone also has been shown to be sensitive and specific for the confirmation or exclusion of acute appendicitis.²⁵ The exclusion of oral contrast in these patients significantly decreases ED time to disposition and improves patient satisfaction.

Bedside transabdominal and transvaginal ultrasonography have emerged as extremely useful adjuncts, decreasing time to diagnosis of life-threatening abdominopelvic conditions. Useful indications include the following:

- Identification of an intrauterine pregnancy, effectively lowering the chances of an ectopic pregnancy to less than 1 in 20,000 (In women using fertility aids, however, identification of intrauterine pregnancy does not exclude ectopic pregnancy, in keeping with an increased incidence of heterotopic pregnancy.)
- Measurement of the cross-sectional diameter of the abdominal aorta to determine whether an abdominal aortic aneurysm exists
- Detection of free intraperitoneal fluid indicating hemorrhage, pus, or extrusion of gut contents
- Use as a diagnostic aid for detection of the following non-life-threatening conditions:
 - Gallstones or a dilated common bile duct, which may be a clue to the presence of choledocholithiasis
 - Pericholecystic fluid or gallbladder wall thickening, which may be indicative of cholecystitis
 - Free intraperitoneal fluid indicating ascites
 - Hydronephrosis indicating possible obstructive uropathy
 - Inferior vena cava distention or collapse as an indicator of volume status

Table 21-3

Most Common Diagnostic Computed Tomography (CT) Findings in Older Patients Presenting to the Emergency Department with Acute Abdominal Pain

FINDING	PERCENT OF ABDOMINAL CT SCANS
Small bowel obstruction or ileus	18%
Diverticulitis	18%
Urolithiasis	10%
Cholelithiasis	10%
Abdominal mass/neoplasm	8%
Pylonephritis	7%
Pancreatitis	6%

From Hustey FM, Meldon SW, et al: The use of abdominal computed tomography in older ED patients with acute abdominal pain. *Am J Emerg Med* 23:259-265, 2005.

The results of sonographic examinations are operator-dependent, and misdiagnosis can occur because of failure to detect or identify pathology, incorrect identification of normal anatomy as pathologic, or overinterpretation of correctly identified findings (e.g., the mere presence of gallstones does not indicate that cholelithiasis is the cause of the pain). The emergency physician must be properly trained in image acquisition and interpretation, and ultrasound evaluation in the radiology department should be sought if there is ambiguity or uncertainty in findings.

■ DIFFERENTIAL DIAGNOSIS

The differential considerations with abdominal pain include a significant number of potentially life- or organ-threatening entities, particularly in the setting of a hemodynamically unstable or toxic-appearing patient. Severely ill patients require timely resuscitation and expeditious evaluation for potentially life-threatening conditions. A focused history and exam should be performed, and the patient should be placed in a monitored acute care area well equipped for airway control, quick intravenous access, and fluid administration. Only then should appropriate diagnostics be initiated (bedside focused assessment with sonography in trauma [FAST] and aorta ultrasound assessment and radiographic, electrocardiographic, and laboratory studies). This approach is particularly important in dealing with elderly or potentially pregnant patients (see Tables 21-1 and 21-2).

Women of reproductive age who present with abdominal pain should undergo pregnancy testing early, and a known pregnancy or a positive result on urine or serum pregnancy testing associated with abdominal pain in the ED should be considered to represent an ectopic pregnancy until proved otherwise. If evidence of blood loss is present, early obstetric consultation and diagnostic ultrasonography should be promptly sought. Bedside transabdominal sonography may identify free intraperitoneal fluid during the evaluation of shock, which may be sufficient evidence to justify operative intervention in the context of a positive pregnancy test and appropriate history and physical findings.

Despite the limitations already described, the approach to the differential diagnosis of abdominal pain generally is based on the location of maximum tenderness. Figure 21-1 shows locations of subjective pain and maximal tenderness on palpation related to various underlying causes. In women of child-bearing age, a positive result on pregnancy testing may indicate ectopic pregnancy, but the entire spectrum of intra-abdominal conditions remains in the differential diagnosis, as for the non-pregnant patient. When the very broad differential list is compartmentalized by both history and physical examination, ancillary testing should proceed to either confirm or support the clinical suspicion.

Despite the significant variety of tests available, close to one half of the patients presenting to the ED with acute abdominal pain will have no conclusive diagnosis. It is incumbent on the clinician to reconsider the extra-abdominal causes of abdominal pain (see Box 21-1), with special consideration in elderly and immunocompromised patients, before arriving at the diagnosis of “nonspecific abdominal pain.”

■ EMPIRICAL MANAGEMENT

The main therapeutic goals in managing acute abdominal pain are physiologic stabilization, mitigation of symptoms (e.g., emesis control, pain relief), and expeditious diagnosis, with consultation, if required.

There is no evidence to support withholding analgesics from patients with acute abdominal pain to preserve the accuracy of subsequent abdominal exams; in fact, the preponderance of evidence supports the opposite. Pain relief may facilitate the diagnosis in patients ultimately requiring surgery.²⁶⁻²⁸ In the acute setting, analgesia usually is accomplished with intravenously titrated opioids. Meperidine (Demerol) has an unfavorable side effect profile and should be avoided. Intravenous ketorolac, the only parenteral nonsteroidal anti-inflammatory drug available in North America, is useful for both ureteral and biliary colic,^{29,30} as well as some gynecologic conditions, but is not indicated for general treatment of undifferentiated abdominal pain. Among patients with gastrointestinal hemorrhage and potential surgical candidates, ketorolac has been shown to increase bleeding times in healthy volunteers.³¹

Aside from analgesics, a variety of other medications may be helpful to patients with abdominal pain. The burning pain caused by gastric acid may be relieved by antacids.³² Intestinal cramping may be diminished with oral anticholinergics such as the combination agent atropine-scopolamine-hyoscyamine-phenobarbital (Donnatal), although evidence for this is scant and highly variable.

Antiemetics such as promethazine, prochlorperazine, ondansetron, granisetron, or inapsine can be useful for nausea and vomiting. Gastric emptying by nasogastric tube with suction is appropriate for suspected small bowel obstruction and intractable pain or vomiting.

If intra-abdominal infection is suspected, broad-spectrum antibiotic therapy should be initiated promptly. Abdominal infections are often polymicrobial and coverage for enteric gram-negative, gram-positive, and anaerobic bacteria must be included. In the choice of antibiotic or combination, the following should be considered:

- Unless local antibiotic resistance surveillance indicates otherwise, second-generation cephalosporins (e.g., cefamandole, cefotetan, cefoxitin) or quinolone (ciprofloxacin, levofloxacin) may be combined with metronidazole for the initial dose of antibiotics in the ED. Other noncephalosporin, β -lactam agents with β -lactamase antagonists (e.g., ampicillin-sulbactam, piperacillin-tazobactam, ticarcillin-clavulanate) are alternatives.
- Many enteric gram-negative bacilli mutate rapidly to produce β -lactamases that are poorly antagonized by specific drug combinations containing clavulanate, sulbactam, or tazobactam. A carbapenem (e.g., imipenem, meropenem) or ceftazidime is an alternative for patients who may have recently received other antibiotics.^{10,33}

Whether to provide coverage for *Enterococcus* species is still a subject of debate, and the decision to treat for these bacteria specifically can be made after consultation. Immunocompromised patients may require antifungal agents.

■ DISPOSITION

Because up to 40% of patients presenting with acute abdominal pain receive the diagnosis of nonspecific abdominal pain, the disposition can be as difficult as the diagnosis in these patients. Categories for disposition may include surgical versus nonsurgical consultation and management, admission for observation, and discharge to home with follow-up evaluation.³⁴ The decision to admit a patient to an observation unit or a hospital bed must factor in the following:

- Information gained from the history, physical examination, and test results
- The likelihood of any suspected disease
- Any potential ramifications of progression of a known disease, or of incorrect diagnosis or management
- The likelihood of appropriate (or any) and timely follow-up after hospital discharge

Clinically stable patients may be discharged from the ED with appropriate follow-up care, possibly to include repeated physical exam or additional diagnostic imaging if indicated.

In the case of nonspecific abdominal pain that is considered potentially worrisome, it is prudent to have the patient reevaluated after 8 to 12 hours. This can be done through a return visit to the ED, an appointment with a primary care physician, or an observation unit protocol.

Before discharge of a patient with an undiagnosed cause of nonspecific abdominal pain, several conditions should be met: The abdominal examination findings should be benign overall, with normal vital signs. Pain and nausea should be controlled,

and the patient should be able to eat and drink. If a patient is to be discharged home without a specific diagnosis, clear instructions to the patient must include the following information:

- What the patient has to do for relief of symptoms or to maximize chances of resolution of the condition (e.g., avoiding exacerbating food or activities, taking medications as prescribed)
- Under what circumstances, with whom, and in what time frame to seek follow-up evaluation, if all goes as desired on the basis of what is known when the patient is in the ED
- Under what conditions the patient should seek more urgent care because of unexpected changes in his or her condition (such as with natural progression of the process before improvement, incorrect diagnosis made in the ED, or untoward reactions to medications)

The references for this chapter can be found online by accessing the accompanying Expert Consult website.

Philip L. Henneman

PERSPECTIVE

Epidemiology

Gastrointestinal (GI) bleeding is a relatively common problem encountered in emergency medicine that often requires early consultation and hospital admission. The overall mortality rate for GI bleeding is approximately 10% and has not changed significantly since the 1960s. Diagnostic modalities have improved much more than therapeutic techniques. GI bleeding is often easy to identify when there is clear evidence of vomiting blood or passing blood in the stool, but the clinical presentation may be subtle, with signs and symptoms of hypovolemia, such as dizziness, weakness, or syncope.

The approach to GI bleeding depends on whether the hemorrhage is located in the proximal or the distal segment of the GI tract (i.e., upper or lower GI bleeding). These segments are anatomically defined by the ligament of Treitz in the fourth section of the duodenum. In the United States, upper GI bleeding (UGIB) affects 50 to 150 people per 100,000 population each year and results in more than 300,000 admissions and about 30,000 deaths per year at an estimated annual cost of almost \$1 billion. Lower GI bleeding (LGIB) affects a smaller portion of patients and results in proportionally fewer hospital admissions than UGIB.¹

GI bleeding can occur in persons of any age but most commonly affects people in their 40s through 70s (mean age, 59 years). Most deaths caused by GI bleeding occur in patients older than 60 years. UGIB is more common in men than in women (in a 2:1 ratio), whereas LGIB is more common in women. Significant UGIB requiring admission is more common in adults, whereas LGIB requiring admission is more common in children.²

DIAGNOSTIC APPROACH

Differential Considerations

Peptic ulcer disease, gastric erosions, and varices account for approximately three fourths of adult patients with UGIB (Box 22-1). Diverticulosis and angiodysplasia account for approximately 80% of adults with LGIB. In children, esophagitis, gastritis, and peptic ulcer disease are the most common causes of UGIB, and infectious colitis and inflammatory bowel disease are the most common causes of LGIB (Box 22-2). In children younger than 2 years of age, massive LGIB is most often a result of Meckel's diverticulum or intussusception. At all ages, anorectal abnormalities are the most common cause of minor

LGIB. Despite improved diagnostic techniques, no source of bleeding is identified in approximately 10% of patients with GI bleeding. In patients with abdominal aortic grafts who present to the emergency department (ED) with GI bleeding, the possibility of aortoenteric fistula should be considered. Prompt surgical consultation in the ED should be obtained if this is suspected, because bleeding can be massive and fatal.

Rapid Assessment and Stabilization

Most patients with GI bleeding are easy to diagnose because they present to the ED complaining of vomiting blood or passing black or bloody stool. The diagnosis is confirmed quickly by examination of the stool for the presence of blood.

Patients with suspected GI bleeding who are hemodynamically unstable should undergo rapid evaluation and resuscitation. They should be undressed quickly to permit placement of cardiac and oxygen saturation monitors, and supplemental oxygen should be given as needed. At least two large-bore peripheral intravenous lines should be placed (minimum 18-gauge); blood should be drawn for hemoglobin or hematocrit, platelet count, prothrombin time (PT), and type and screen or type and crossmatch studies; and crystalloid resuscitation should be initiated. Intravenous crystalloid fluid should be given as a 2-L bolus in adults or 20 mL/kg in children until the patient's vital signs have stabilized or the patient has received 40 mL/kg of crystalloid in an adult or 60 mL/kg as a child. Patients who remain unstable after 40 to 60 mL/kg of crystalloid should be given type O, type-specific, or cross-matched blood, depending on availability. Persistently unstable patients should receive immediate consultation with a gastroenterologist for UGIB and with a surgeon for LGIB.³

Pivotal Findings

History, physical examination, testing a stool sample for blood, and measuring hemoglobin or hematocrit are the keys to diagnosing GI bleeding in most patients.

History

Patients typically complain of vomiting red blood or coffee grounds–like material, or passing black or bloody stool. *Hematemesis* (vomiting blood) occurs with bleeding of the esophagus, stomach, or proximal small bowel. Approximately

BOX 22-1

ETIOLOGY OF SIGNIFICANT GASTROINTESTINAL (GI) BLEEDING IN ADULTS***Upper**

Peptic ulcer disease
Gastric erosions
Varices
Mallory-Weiss tear
Esophagitis
Duodenitis

Lower

Diverticulosis
Angiodysplasia
Upper GI bleeding
Cancer/polyps
Rectal disease
Inflammatory bowel disease

*Potential causes listed in decreasing frequency.

BOX 22-2

ETIOLOGY OF GASTROINTESTINAL BLEEDING IN CHILDREN***Upper**

Esophagitis
Gastritis
Ulcer
Esophageal varices
Mallory-Weiss tear

Lower

Anal fissure
Infectious colitis
Inflammatory bowel
Polyps
Intussusception

*Potential causes listed in decreasing frequency.

50% of patients with UGIB present with this complaint. Hematemesis may be bright red or darker (i.e., coffee grounds–like) as a result of the conversion of hemoglobin to hematin or other pigments by hydrochloric acid in the stomach. The color of vomited or aspirated blood from the stomach does not differentiate between arterial and venous bleeding.

Melena, or black tarry stool, will result from the presence of approximately 150 to 200 mL of blood in the GI tract for a prolonged period. Melena is seen in approximately 70% of patients with UGIB and in one third of patients with LGIB. Black stool that is not tarlike may result from presence of 60 mL of blood from the upper GI tract. Blood from the duodenum or jejunum must remain in the GI tract for approximately 8 hours before turning black. Occasionally, black stool may follow bleeding into the lower portion of the small bowel and ascending colon. Stool may remain black and tarry for several days, even though bleeding has stopped. Black stool also may be seen after ingestion of bismuth (e.g., Pepto-Bismol), which can confuse the situation because such preparations often are taken for UGI distress. In contrast with melena, stool rendered black by bismuth is not positive on Hemoccult testing.

Hematochezia, or bloody stool (bright red or maroon), most often signifies LGIB but may be due to a brisk UGIB with rapid transit time through the bowel in 10 to 15% of patients. Because UGIB is much more common than LGIB, a more proximal source of significant bleeding must be excluded before assuming the bleeding is from the lower GI tract. Approximately two thirds of patients with LGIB present with red blood from bleeding per rectum. Small amounts of red blood (e.g., 5 mL) from rectal bleeding, such as bleeding due to hemorrhoids, may cause the water in the toilet bowl to appear bright red. Bright red stools also can be seen after ingestion of a large quantity of beets; in this case, Hemoccult testing would be negative and the patient also will report pink-colored water in the toilet bowl.

In taking the history, specific questions should address the duration and quantity of bleeding, associated symptoms, previous history of bleeding, current medications, alcohol, non-steroidal anti-inflammatory drug use and long-term aspirin

ingestion, allergies, associated medical illnesses, previous surgery, treatment by nonhospital personnel, and the response to that treatment.^{4,5} Patients with GI bleeding may report symptoms of hypovolemia, such as dizziness, weakness, or loss of consciousness, most often after standing up. Other nonspecific complaints include dyspnea, confusion, and abdominal pain. Rarely an elderly patient may present with ischemic chest pain precipitated by significant anemia due to a GI bleed. One in five patients with GI bleeding may have only nonspecific complaints.

The history is of limited help in predicting the site or quantity of bleeding. Patients with a previously documented GI lesion bleed from the same site in only 60% of cases. Gross estimates of blood loss based on the volume and color of the vomitus or stool (e.g., brown or black, pink or red) or the number of episodes of hemorrhage are notoriously inaccurate.

Physical Examination

Vital Signs Vital signs and postural changes in heart rate and blood pressure have been used to assess the amount of blood loss in patients with GI bleeding but are insensitive and non-specific, with the exception of significant, sustained heart rate increase and hypotension. All patients with a history suggesting GI bleeding who are hypotensive, are tachycardic, or experience sustained posture-induced changes in heart rate of greater than 20 beats per minute should be assumed to have a significant hemorrhage. Normal vital signs do not exclude a significant hemorrhage, and postural changes in heart rate and blood pressure may occur in individuals who are not bleeding (e.g., elderly patients, many normal individuals, individuals on certain medications such as beta-blockers, individuals with hypovolemia from other causes).

General Examination The physical examination is valuable in establishing a specific diagnosis and assessing the severity of blood loss and the physiologic response to that loss. Careful attention is given to the patient's general appearance, vital signs, mental status (including restlessness), skin signs (e.g., color, warmth, and moisture to assess for shock, or presence of lesions such as telangiectasia, bruises, or petechiae to assess for vascular diseases or hypocoagulable states), pulmonary and cardiac findings, abdominal examination, and rectal and stool examination. Frequent reassessment is important because a patient's status may change quickly.

Rectal Examination Rectal and stool examinations are often key to making or confirming the diagnosis of GI bleeding. The finding of red, black, or melanic stool early in the assessment is helpful in prompting early recognition and management of patients with GI bleeding. The absence of black or bloody stool, however, does not exclude the diagnosis of GI bleeding. Regardless of the apparent character and color of the stool, occult blood testing is indicated.

Ancillary Testing

Tests for Occult Blood The presence of hemoglobin in occult amounts in stool is confirmed by tests such as guaiac assays (e.g., Hemoccult, HemaPrompt). Stool tests for occult blood may have positive results 14 days after a single, major episode of UGIB. False-positive results have been associated with the ingestion of certain fruits (e.g., cantaloupe, grapefruit, figs), uncooked vegetables (e.g., radish, cauliflower, broccoli) and red meat, methylene blue, chlorophyll, iodide, cupric sulfate, and bromide preparations. False-negative results are uncommon but can be caused by bile or ingestion of magnesium-containing antacids or ascorbic acid. Tests to evaluate gastric contents for occult blood (e.g., Gastrocult) can be unreliable

and should not be used for this purpose. In newborns, maternal blood that is swallowed may cause bloody stools; the Apt test may show that it is maternal in origin.

Clinical Laboratory Tests Blood should be drawn for evaluation of baseline hematocrit or hemoglobin, coagulation studies (PT and platelet count), and type and crossmatch studies (or type and screen studies if the patient is stable). The initial hematocrit may be misleading in patients with preexisting anemia or polycythemia. Changes in the hematocrit may lag significantly behind actual blood loss. Infusion of normal saline speeds equilibration of the hematocrit; however, rapid infusion of crystalloid in nonbleeding patients also may cause a decrease in hematocrit by hemodilution. The optimal hematocrit with respect to oxygen-carrying capacity and viscosity in critically ill patients has been reported to be 33%. In general, patients with a hemoglobin concentration of 8 g/dL or less (hematocrit <25%) from acute blood loss usually require blood therapy. After transfusion and in the absence of ongoing blood loss, the hematocrit can be expected to increase approximately 3% for each unit of blood administered (hemoglobin level increases by 1 mg/dL).

The PT should be used to determine whether a patient has a preexisting coagulopathy. An elevated PT may indicate vitamin K deficiency, liver dysfunction, warfarin therapy, or consumptive coagulopathy. Patients receiving therapeutic anticoagulants or patients with an elevated PT and evidence of active bleeding should receive sufficient fresh frozen plasma to correct the PT. Serial platelet counts are used to determine the need for platelet transfusions (i.e., less than 50,000/mm³).

Blood Bank Blood should be sent for “type and hold” or type and crossmatch studies early in the patient’s care. Immediate transfusion needs in unstable patients can be met with O-positive packed red blood cells (O-negative packed red blood cells in women of childbearing age whose Rh status is unknown). Type-specific blood is usually available within 10 to 15 minutes. Group O blood and type-specific blood are safe for patients and cause few transfusion reactions. Fully cross-matched blood may take 60 minutes to prepare. Stable patients can be managed more cost-effectively by ordering “type and hold” without crossmatching for units of blood.

Other Laboratory Tests Electrolytes usually are normal in patients with GI bleeding. However, determination of electrolytes, blood urea nitrogen, and creatinine may be useful in a small percentage of patients with GI bleeding when indicated. For example, in patients with repeated vomiting, hypokalemia, hyponatremia, and metabolic alkalosis may develop, which usually correct with adequate hydration and the resolution of vomiting. Patients with shock often have metabolic acidosis from lactate accumulation. The blood urea nitrogen is elevated in many patients with UGIB as a result of the absorption of blood from the GI tract and hypovolemia causing prerenal azotemia. After 24 hours, hypovolemia probably is the sole determinant of azotemia unless there has been recurrent bleeding or there is baseline renal insufficiency.

Electrocardiogram An electrocardiogram (ECG) should be obtained in all patients with a GI bleed who are older than 50 years of age or have preexisting ischemic cardiac disease, significant anemia, or chest pain, shortness of breath, or persistent hypotension. Asymptomatic myocardial ischemia (ST segment depression greater than 1 mm) or injury (ST segment elevation greater than 1 mm) may develop in the setting of GI bleeding. Patients with GI bleeding and clinical or ECG evidence of myocardial ischemia should receive packed red blood cells as soon as possible, as well as appropriate treatment for myocardial ischemia.

Imaging GI hemorrhage is not an indication for plain abdominal radiography. An upright chest radiograph should be

obtained in patients with UGIB suspected of aspiration or with signs and symptoms of bowel perforation (shock with significant abdominal or peritoneal tenderness). Subdiaphragmatic air consistent with bowel perforation is a rare finding with UGIB, but it is an indication for immediate surgical consultation and operative repair.

■ DIFFERENTIAL DIAGNOSIS

Not all patients complaining of vomiting blood or passing blood in the stool have GI bleeding. Swallowing blood during epistaxis or from the oral cavity may cause hematemesis or melena. Red vomitus may be due to food products (e.g., Jell-O, tomato sauce, wine), and black stool may be due to iron therapy or bismuth (e.g., Pepto-Bismol). Hypovolemia (and its symptoms) may be due to vomiting and diarrhea without bleeding. Poor oral intake with or without fever also may result in hypovolemia. Usually the patient’s hemoglobin or hematocrit is normal or elevated until hemodilution can occur. There are many causes of anemia other than GI bleeding. In the absence of suggestive symptoms or blood in the stool, GI bleeding is less likely to be the cause of observed anemia.⁵

■ MANAGEMENT

Quick identification, aggressive resuscitation, risk stratification, and prompt consultation are the keys to appropriate emergency management. When the diagnosis of GI bleeding is made, emergency management of patients can proceed (Fig. 22-1).

Reassurance

Patients who present to the ED with symptoms and signs of GI bleeding are often frightened by their symptoms. They may be concerned about the possibility of painful procedures and of the real or perceived risk of death. These patients and their families should be treated in a supportive and reassuring manner. They should be provided with accurate information about their problem, and all aspects of the care they are receiving should be explained in a way that they understand.

Nasogastric Tube and Gastric Lavage

After initial resuscitation of the patient, it is important to identify whether the hemorrhage is proximal or distal to the ligament of Treitz (i.e., UGIB or LGIB). If the patient’s vomitus demonstrates blood, then the diagnosis of UGIB is confirmed. If a patient reports bloody or “coffee grounds” emesis or if melanic stool is present, an upper GI bleed is more likely. Placement of a nasogastric (NG) tube formerly was widely undertaken in the belief that it had both diagnostic and therapeutic benefit. Although in some cases, an NG tube may help to establish or confirm the diagnosis of UGIB, it is not useful for risk stratification. Aspiration of bloody contents from the NG tube diagnoses UGIB (or bleeding from nasal or oral passageways), but it does not determine if the bleeding is ongoing or has already stopped. Earlier assertions that gastric lavage “until clear” demonstrated that the bleeding had stopped have been refuted by findings at endoscopy. There is a 10% incidence of failure to aspirate blood through the NG tube in established UGIB. False-negative results are possible if the bleeding is intermittent or has already stopped and the stomach is cleared, or if the bleeding is in the duodenum, and edema or spasm of the pylorus has prevented reflux of

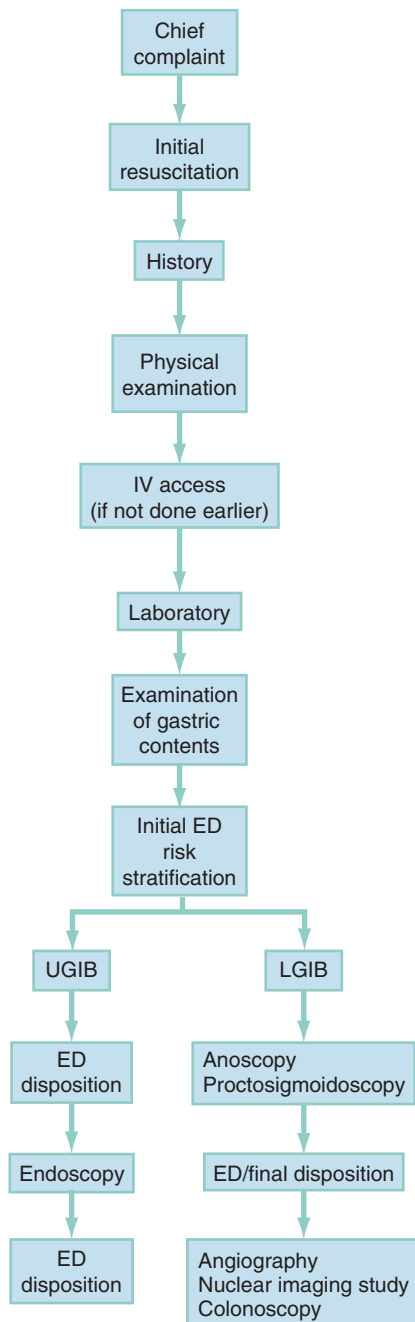


Figure 22-1. Emergency management of patients with gastrointestinal bleeding. ED, emergency department; IV, intravenous; LGIB, lower gastrointestinal bleeding; UGIB, upper gastrointestinal bleeding.

blood into the stomach. The presence of bile in an otherwise clear aspirate excludes the possibility of active bleeding above the ligament of Treitz, but should not be used to exclude UGIB in a patient with documented melena.

False-positive results may occur from nasal bleeding. Gastric contents should not be tested for occult blood; visual inspection of the vomitus or aspirate is insufficient to diagnose subtle bleeding, and testing is unreliable. In patients who have hematochezia, an upper GI origin for the bleeding often is associated with signs and symptoms of shock, because rapid transit time of large quantities of blood is producing the hematochezia. Because up to 10 to 15% of patients with hematochezia have UGIB, it has been recommended that an NG tube

be inserted in most cases of LGIB, but there is no evidence to support an improved outcome related to this practice.^{2,6} Typically, a clinical decision is made regarding the likelihood of an upper versus lower GI origin for the bleeding, and endoscopy is then performed based on that determination. If the first endoscopy approach fails to identify the bleeding site, often endoscopy of the other end of the GI tract is necessary.

Gastric tubes are safe in most patients, but pharyngeal and esophageal perforation, cardiac arrest, ethmoid sinus fracture with brain trauma, and bronchial intubation have been reported. No evidence exists that gastric tube placement aggravates hemorrhage from varices or Mallory-Weiss tears.

Gastric lavage may be helpful to prepare a patient for endoscopy. Before gastric lavage, patients with evidence of a possible perforated viscus (e.g., severe pain, peritoneal signs) should undergo radiologic assessment looking for free air. Lavage should not be performed in the presence of pneumoperitoneum. Gastric lavage does not reduce blood loss in patients with UGIB, and use of iced lavage fluid is not recommended. Gastric lavage in preparation for endoscopy is best performed with a large-bore Ewald tube, passed orally while the patient is in the left lateral decubitus position with the bed in Trendelenburg position. Additional holes may be cut in the distal portion of the Ewald tube to improve aspiration of blood and clots. Clots that cannot be aspirated continue to cause pink return, giving the false impression of continued bleeding. The irrigant need not be sterile; regular tap water may be used. The irrigant should be delivered and removed by gravity in volumes of 200 to 300 mL until the return is clear. Little irrigant is absorbed by the patient. Gastric rupture has been reported as a rare complication of gastric lavage.^{7,8}

Anoscopy/Proctosigmoidoscopy

Patients with mild rectal bleeding who do not have obviously bleeding hemorrhoids should undergo anoscopy or proctosigmoidoscopy. If bleeding internal hemorrhoids are discovered, and the patient does not have portal hypertension, the patient may be discharged with appropriate treatment and follow-up evaluation for hemorrhoids. If hemorrhoids are not detected, it is important to determine if the stool above the rectum contains blood. The absence of blood above the rectum in a patient who is actively bleeding indicates that the source of bleeding is in the rectum. The presence of blood above the anoscope or sigmoidoscope does not invariably indicate a proximal source of bleeding, because retrograde passage of blood into the more proximal colon commonly occurs. Such patients need further evaluation.

Endoscopy

Endoscopy is the most accurate diagnostic tool available for the evaluation of UGIB. It identifies a lesion in 78% to 95% of patients with UGIB if it is performed within 12 to 24 hours of the hemorrhage. Accurate identification of the bleeding site allows for risk stratification with respect to predicting rebleeding and mortality. Endoscopy-based triage significantly reduces hospitalization rates and costs of treating upper GI bleeding.^{8,9} Significant advances in endoscopic hemostasis also make it of therapeutic value in select patients (e.g., for banding or sclerosing of varices). Colonoscopy is an effective tool for diagnosis and selected treatment of LGIB.^{10,11}

Angiography and Tagged Red Blood Cell Scan

Angiography can detect the location of UGIB in two thirds of patients studied. Since the advent of endoscopy, however, the use of angiography has decreased significantly, and today angiography is used in only 1% of patients with UGIB. Angiography is used more commonly in patients with LGIB and usually in consultation with a general surgeon. Although angiography rarely diagnoses the cause of bleeding, it does identify the site of bleeding in approximately 40% of patients who have LGIB and in 65% of patients who eventually require surgical intervention. Angiography ideally is performed during active bleeding; this may be apparent from persistently unstable vital signs or continued transfusion requirements to establish or maintain an optimal hemoglobin or hematocrit level. Arterial embolization can be used in selected cases of LGIB.¹² In some patients with more indolent or elusive bleeding, a nuclear isotope–tagged red blood cell scan, usually performed from the inpatient unit, may identify the bleeding site.

Gastric Acid Secretion Inhibition

All patients with peptic ulcer disease documented by endoscopy should receive therapy with a proton-pump inhibitor (e.g., omeprazole).^{13–15} There is no documented benefit to initiating this therapy or administering H₂ antihistamines in the ED for patients with UGIB, however.

Octreotide (Somatostatin Analogues)

Patients with documented esophageal varices and acute upper GI bleeding should receive an intravenous infusion of octreotide at 25–50 µg/hour for a minimum of 24 hours while being observed in the intensive care unit (ICU). Octreotide is a useful addition to endoscopic sclerotherapy and decreases rebleeding occurrences.^{16,17} (See Chapter 87.) Octreotide may also reduce the incidence of lower GI rebleeding secondary to angiodysplasia.¹⁸

Vasopressin

Intravenous vasopressin has been used in the treatment of UGIB, most commonly in patients with variceal hemorrhage. Controlled studies have not shown a positive effect of vasopressin on overall mortality, however. These results, combined with a relatively high rate of serious complications (9% major and 3% fatal), suggest that use of vasopressin should be limited. A trial of vasopressin may be warranted in an exsanguinating patient with suspected variceal bleeding, especially if endoscopy is not immediately available. The recommended dose of vasopressin is 20 units given intravenously over 20 minutes and then 0.2 to 0.4 unit per minute. Consultation with a gastroenterologist is advisable.

Sengstaken-Blakemore Tube

Placement of a Sengstaken-Blakemore tube stops hemorrhage in approximately 80% of patients bleeding from esophageal varices. The Linton tube may be superior to the Sengstaken-Blakemore tube in patients with bleeding gastric varices; however, either of these tubes is rarely used. In general, these tubes should not be used without endoscopic documentation of the source of bleeding because complications are common and significant (14% major, 3% fatal). A trial of balloon tamponade should be considered, however, in an exsanguinating

patient with probable variceal bleeding in whom endoscopy is not immediately available and vasopressin has not slowed the hemorrhage. Consultation with a surgeon or gastroenterologist is advisable.

Surgery

Surgery is indicated for all hemodynamically unstable patients with active bleeding who do not respond to appropriate intravascular volume replacement, correction of any coagulopathy, and endoscopic intervention (if available). The mortality rate for patients undergoing emergency procedures for GI bleeding is approximately 23%. Generally, surgery is indicated whenever the risk of ineffective medical therapy and continued hemorrhage outweighs that of surgical morbidity and mortality.¹⁹ Emergency surgical consultation should be considered when blood replacement exceeds 5 units within the first 4 to 6 hours or when 2 units of blood is needed every 4 hours (after replacement of initial losses) to maintain normal cardiac output.

DISPOSITION

Risk Stratification

Risk stratification involves combining historical, clinical, and laboratory data to determine the risk of death and rebleeding in patients presenting to an ED with GI bleeding. Patients can be sorted into four risk categories: very low, low, moderate, and high risk. Some patients present to the ED with a vague complaint of vomiting blood or passing blood from the rectum in whom detailed history and examination allows a diagnosis of hemorrhoid, or anal fissure, or there may be little or no objective evidence of significant GI bleeding. These patients can be categorized as very low risk and can be sent home without further diagnostic tests^{2,7,20} (Box 22-3).

Before discharge, patients should be educated about the signs and symptoms of significant GI bleeding and when to return to the ED or when to call their primary care physician. They should be given specific education about the possible or actual cause of the bleeding and specific treatment for that disorder. They should be educated about the side effects of any medications. Patients should undergo specific follow-up evaluation within 24 to 36 hours. They should be instructed to avoid aspirin, nonsteroidal anti-inflammatory drugs, and alcohol.^{2,21,22}

Patients with low-risk, moderate-risk, and high-risk criteria are more complicated and require further assessment. Historically, nearly all patients with significant GI bleeding were admitted to the hospital. As health care has changed, a greater emphasis has been placed on outpatient management of select

BOX 22-3

VERY-LOW-RISK CRITERIA FOR PATIENTS COMPLAINING OF GASTROINTESTINAL BLEEDING WHO CAN BE DISCHARGED HOME

- No comorbid diseases
- Normal vital signs
- Normal or trace positive result on stool guaiac testing
- Negative findings on gastric aspiration, if done
- Normal or near-normal hemoglobin/hematocrit
- Good support systems
- Proper understanding of signs and symptoms of significant bleeding
- Immediate access to emergent care if needed
- Follow-up arranged within 24 hr

Table 22-1 Initial Emergency Department Risk Stratification for Patients with Gastrointestinal Bleeding

LOW RISK	MODERATE RISK	HIGH RISK
Age < 60 yr	Age > 60 yr	
Initial SBP ≥ 100 mm Hg	Initial SBP < 100 mm Hg	Persistent SBP < 100 mm Hg
Normal vital signs for 1 hr	Mild ongoing tachycardia for 1 hr	Persistent moderate/severe tachycardia
No transfusion requirement	Transfusions required, ≤4 units	Transfusion required, >4 units
No active major comorbid diseases	Stable major comorbid diseases	Unstable major comorbid diseases
No liver disease	Mild liver disease—PT normal or near-normal	Decompensated liver disease—e.g., coagulopathy, ascites, encephalopathy
No moderate-risk or high-risk clinical features	No high-risk clinical features	

PT, prothrombin time; SBP, systolic blood pressure.

Data from Terdiman JP, Lindenauer GF: Acute gastrointestinal bleeding. In Wachter RM, Goldman L, Hollander H (eds): Hospital Medicine. Philadelphia, Lippincott Williams & Wilkins, 2005, pp. 767–779.

Table 22-2 Management by Risk Category for Patients with Upper Gastrointestinal Bleeding after Endoscopy

RISK STRATIFICATION	Recommended Management		
	LOW RISK	MODERATE RISK	HIGH RISK
Low risk	Immediate discharge*	23-hr observation (floor) [†]	ICU monitoring for 24 hr [‡] (48- to 72-hr hospitalization)
Moderate risk	48-hr inpatient stay [†]	48- to 72-hr inpatient stay (floor) [†]	ICU monitoring for 24 hr (48- to 72-hr hospitalization)
High risk	ICU monitoring for 48 hr (48- to 72-hr hospitalization)	ICU monitoring for 24 to 48 hr (72-hr hospitalization)	ICU monitoring for 72 hr (≥72-hr hospitalization)

*Patients with low-risk clinical and endoscopic findings can be discharged home with appropriate treatment based on diagnosis, scheduled follow-up evaluation within 24 hours, and proper patient education to ensure immediate return if signs of rebleeding appear.

[†]Patients may be discharged after 24 to 48 hours of in-hospital observation if there is no evidence of rebleeding, vital signs are normal, there is no need for further transfusion, and the hemoglobin or hematocrit has remained stable. They should be provided with appropriate treatment based on diagnosis, scheduled follow-up evaluation within 24 hours, and proper patient education to ensure immediate return if signs of rebleeding appear.

[‡]Patients with high-risk clinical or endoscopic findings should be hospitalized and monitored closely for evidence of rebleeding.

Data from Terdiman JP, Lindenauer GF: Acute gastrointestinal bleeding. In Wachter RM, Goldman L, Hollander H (eds): Hospital Medicine. Philadelphia, Lippincott Williams & Wilkins, 2005, pp. 767–779.

low-risk patients with GI bleeding. Studies have shown that combining clinical and endoscopic criteria provides an accurate estimation of the risk of rebleeding and mortality in patients with UGIB. These combined criteria have been used to identify patients with UGIB at low risk, who can be discharged home, and patients at moderate or high risk, who need to be admitted to an appropriate care site in the hospital. Risk stratification for patients with LGIB is less well studied, so nearly all patients with significant LGIB are admitted. Risk stratification can be used for patients with LGIB, however, to decide an appropriate inpatient care site.

Table 22-1 presents an initial risk stratification tool for patients with upper and lower GI bleeding. Combining clinical and endoscopic findings allows for final risk stratification, as shown in Table 22-2, to decide disposition, inpatient care site, and treatment.^{1,2,6,20,23-25}

Patients with clinical evidence of GI bleeding should undergo endoscopy as soon as it is available for final risk stratification, inpatient triage, and determination of appropriate treatment (see Table 22-2). If endoscopy is not immediately available, patients with low clinical risk may be admitted to an ED observation unit or short-stay hospital bed until endoscopy can be performed. Patients with moderate clinical risk criteria may be admitted to an inpatient floor, intermediate care unit, or ICU, as indicated by specific patient management needs and depending on the capabilities of the institution. Patients with high clinical risk should be admitted to a closely monitored step-down unit or an ICU. The timing of endoscopy depends on availability, the acuity of the patient, the need for

emergent therapy, the need to determine final care site, and the need to minimize length of stay.^{3,26}

Patients with LGIB that is not clearly due to hemorrhoids, fissure, or proctitis should be admitted to an inpatient bed. Patients with low risk may be admitted to an inpatient floor bed and prepared for a nuclear medicine imaging study (e.g., red blood cell-labeled study) or colonoscopy. Patients with high-risk criteria should be admitted to a step-down unit or ICU and considered for angiography to identify the site of LGIB. Patients with moderate-risk criteria require individualized determination of the most appropriate inpatient care site (floor, intermediate care bed, or ICU) and the most useful diagnostic studies (nuclear imaging or angiography).

Consultation with a surgeon should be obtained if it appears that more than 5 units of blood is required to achieve hemodynamic stability or if there is reasonable suspicion that operative intervention may be needed. This is especially true of patients older than 65 years of age. In general, the older the patient, the more aggressive the surgical management ought to be. Patients with a history of varices, persistent postural changes in heart rate, or significant bleeding of bright red blood per rectum are more likely to require surgery than are patients without these findings. Emergent vascular surgical consultation is needed for patients who have abdominal aortic grafts who present to the ED with GI bleeding, because of the possibility of an aortoenteric fistula.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.

PERSPECTIVE

Diarrhea is a common presenting complaint in the emergency department (ED) and can account for up to 5% of all visits. Most cases of diarrhea are self-limited and require only supportive care. Conversely, patients with more serious infection and associated comorbidity may present with life-threatening dehydration and shock. There may be an associated sepsis and septic shock component. Numerous but relatively rare, noninfectious causes of diarrhea should also be considered.

Incidence

Worldwide, diarrhea remains a major health problem, accounting for approximately 4% of all deaths each year, which is estimated by the World Health Organization (WHO) to be 2.2 million victims (World Health Report, 2000).¹ A large proportion of these deaths occur in small children in developing countries. Rotavirus causes 25% to 65% of childhood-associated diarrheal illnesses (3.5 million cases per year in the United States), whereas adults experience 74 million episodes of diarrhea annually. In the United States, 90% of diarrheal illnesses are caused by noroviruses (caliciviruses), of which more than 100 different strains are recognized.² Patients at the extremes of age, those with significant comorbidity, those who are immunologically compromised, and those with iatrogenic illness are most vulnerable to significant morbidity and mortality. An estimated 60% of patients infected with human immunodeficiency virus (HIV) experience significant diarrhea during the course of their illness.³

Most adults experience diarrhea many times during their lifetime. Diarrhea illnesses are the primary cause of many hospitalizations and hours of lost work.

Definition and Categorization

The term *diarrhea* is derived from the Greek words *dia* (“through”) and *rhein* (“to flow”). The two main categories of diarrhea-associated illness are infectious and noninfectious. Infectious causes represent about 85% of cases, whereas noninfectious causes account for only 15% of the total. Infectious diarrhea may be divided into viral, bacterial, and parasitic causes (Box 23-1), with estimates of their relative contributions being 70% for viral, 24% for bacterial, and 6% for parasitic infections.⁴

Definitions for diarrhea have been proposed to standardize nomenclature, help the clinician determine a probable etiology, and direct empirical therapy if indicated⁵:

- *Acute* diarrhea is defined as lasting for 14 days or less.
- *Persistent* diarrhea lasts for longer than 14 days.
- *Chronic* diarrhea lasts 30 days or longer.

Acute diarrhea presentations usually will be infectious. A majority of these cases are self-limited and caused by viral and bacterial pathogens. Persistent diarrhea suggests an enteric pathogen other than viral, such as bacterial or protozoan. Chronic diarrhea usually is associated with noninfectious causes and requires further testing to determine the etiology.

Normally, the small and large bowel absorb 99% of gastrointestinal tract secretions produced and liquids ingested each day. Any pathologic state that reduces water absorption by 1% can cause diarrhea.⁶ Diarrhea results from one or more of four different pathologic processes that are characteristic of the primary cause and that contribute to the decreased absorption of the gut.

Secretory diarrhea is caused by pathogens that produce cytotoxins that increase cellular permeability and cause the oversecretion of water and electrolytes. Most cases of diarrhea encountered in the ED are secretory. Noninfectious causes of secretory diarrhea include medications, toxic substances, endocrine disorders, and neoplasias (Box 23-2).

Inflammatory diarrhea, also described as invasive or severe diarrhea, or dysentery, results from cellular damage to the intestinal mucosa, leading to the hypersecretion of water, electrolytes, blood, mucus, and plasma proteins. This diarrhea most commonly is caused by invasive bacterial and parasitic pathogens that produce dysenteric illnesses (see Box 23-1). Some noninfectious causes of inflammatory diarrhea include chemotherapy, radiation therapy, hypersensitivity reactions, autoimmune disorders, ischemic colitis, and inflammatory bowel disease. With inflammatory diarrhea, fecal leukocytes and erythrocytes typically are present, as are systemic symptoms, and the diarrhea continues despite fasting.

Osmotic diarrhea occurs with the ingestion or malabsorption of osmotically active solutes. These solutes cause the osmotic movement of water into the intestinal lumen, which then overwhelms the gut's ability to reabsorb it. Examples include the effects of osmotic laxatives and carbohydrate malabsorption. Steatorrhea results from osmotic effects of lipids not absorbed in malabsorption and maldigestion syndromes.

Abnormal motility generally is seen in patients with chronic diarrhea but is always a component of acute diarrhea. Hypermotility decreases contact time between luminal contents and the absorbing mucosa, limiting water and electrolyte absorption.

BOX 23-1 ETIOLOGIC AGENTS OF INFECTIOUS DIARRHEA**Viral (60% of cases)**

Astrovirus
 Calicivirus
 Coronavirus
 Cytomegalovirus*
 Enteric adenovirus
 Hepatitis A through G
 Herpes simplex virus
 HIV enteropathy
 Norwalk-like agents
 Norwalk virus
 Pararotavirus
 Picornavirus
 Rotavirus
 Small round viruses

Bacterial (20% of cases)**Invasive***

Aeromonas spp.
Campylobacter spp.
Clostridium difficile
 Enteroinvasive *E. coli*
Mycobacterium spp.
Plesiomonas shigelloides
Salmonella spp.
Shigella spp.
Vibrio fluvialis
Vibrio parahaemolyticus
Vibrio vulnificus
Yersinia enterocolitica
Yersinia pseudotuberculosis

Toxigenic

FOOD POISONING WITH PREFORMED TOXINS

Bacillus cereus
Clostridium botulinum
Staphylococcus aureus

TOXIN FORMATION AFTER COLONIZATION

Aeromonas hydrophila
Clostridium perfringens

Enterohemorrhagic *E. coli** O157:H7
 Enterotoxigenic *E. coli*
Klebsiella pneumoniae
Shigella spp.
Vibrio cholerae

Other bacteria**Parasitic (5% of cases)****Protozoa**

*Balantidium coli**
Blastocystis hominis
Cryptosporidium
Cyclospora
Dientamoeba fragilis
*Entamoeba histolytica**
Entamoeba polecki
Enteromonas hominis
Giardia lamblia
Iso spor a belli
 Microsporidia
Sarcocystis hominis

Helminths

Angiostrongylus costaricensis
 Anisakiasis
Ascaris lumbricoides
Diphyllobothrium latum
Enterobius vermicularis
 Hookworms
Schistosoma spp.
Strongyloides stercoralis
Taenia spp.
Trichinella spiralis
Trichuris trichiura

*Associated with fever, abdominal pain, and fecal red blood cells or white blood cells. % indicates the estimated contribution to total cases.

CLINICAL APPROACH**Emergency Assessment and Stabilization**

An immediate assessment should be made of the patient's stability, including maintenance of the airway, adequacy of oxygenation and ventilation, and circulation, with particular attention to volume status. Tachycardia, orthostatic hypotension, poor skin turgor and color, diaphoresis, and mental status changes all are characteristic of hypovolemia and hypoperfusion. Associated septic shock may contribute to the hypotension and general organ hypoperfusion, and diarrhea may be a manifestation of toxic shock syndrome. A diarrhea-associated acid-base disorder should be suspected in patients with Kussmaul respirations, a significant anion gap on basic metabolic panel reflecting a lactic acidosis from significant volume loss, or a non-anion gap metabolic acidosis associated with massive bicarbonate loss. After stabilization, a secondary survey may elucidate the potential cause of the diarrhea and direct further evaluation and treatment.

Secondary Survey

The physical examination should assess the patient's overall health, toxicity, fever, volume status, signs of a surgical abdomen, and determine the presence of blood in the stool. Young healthy adults may maintain a normal blood pressure and heart rate even with significant dehydration. In patients who are taking antiarrhythmic or beta-blocker medications or have conduction disease or fixed-pace rhythms, heart rate may not be a reliable indicator of volume status. Signs of volume depletion and impending shock include dry mucosa, poor skin turgor, decreased urine output, and mental status changes. Children will present with sunken eyes, depression of the fontanel, decrease in urine output (number of wet diapers), and decrease in alertness and activity.⁷

Particular attention should be given to the abdominal examination. Focal abdominal pain with peritoneal findings may be due to an acute surgical abdomen with symptoms mimicking those of severe gastroenteritis. A rectal examination should be performed to detect fecal impaction, melena, or hematochezia.

BOX 23-2 CAUSES OF NONINFECTIOUS DIARRHEA**Toxins****Drugs**

ACE inhibitors
 Alprazolam (Xanax)
 Antacids (Mg)
 Antibiotics
 Antidepressants
 Antiepileptic drugs
 Antihypertensives
 Antiparkinson drugs
 Beta-blockers
 Caffeine
 Cardiac antiarrhythmics
 Chemotherapy agents
 Cholesterol-lowering drugs
 Cholinergic agents
 Cholinesterase inhibitors
 Colchicine
 Digitalis
 Diuretics
 Fluorouracil
 Fluoxetine (Prozac)
 Histamine H₂-receptor antagonists
 Hydralazine
 Lactulose
 Laxatives/cathartics
 Levodopa
 Lithium
 NSAIDs
 Neomycin
 Podophyllin
 Procainamide
 Prostaglandins
 Quinidine
 Ricinoleic acid
 Theophylline
 Thyroid hormone
 Valproic acid

Dietetic Foods

Mannitol
 Sorbitol
 Xylitol

Fish-Associated Toxins

Amnesic shellfish poisoning
 Ciguatera
 Echinoderms

Neurotoxic shellfish poisoning
 Paralytic shellfish poisoning
 Scombroid
 Tetroton

Plant-Associated Toxins

Herbal preparations
 Horse chestnut
 Mushrooms—*Amanita* spp.
 Nicotine
 Other plant toxins
 Pesticides—organophosphates
 Pokeweed
 Rhubarb
 Miscellaneous
 Allergic reactions
 Carbon monoxide poisoning
 Ethanol
 Heavy metals
 Monosodium glutamate (MSG)
 Opiate withdrawal

Gastrointestinal Pathology

Appendicitis
 Autonomic dysfunction
 Bile acid malabsorption
 Blind loop
 Bowel obstruction
 Celiac disease
 Cirrhosis
 Defects in amino acid transport
 Diverticular disease
 Familial dysautonomia
 Fecal impaction
 Fecal incontinence
 GI bleed
 GI cancer
 Hirschsprung's disease
 Inflammatory bowel disease (ulcerative colitis, Crohn's disease)
 Intussusception
 Irritable bowel syndrome
 Ischemic bowel
 Lactose/fructose intolerance
 Malabsorption syndromes
 Malrotation
 Postsurgical
 Postvagotomy

Radiation therapy
 Short gut syndrome
 Small bowel resection
 Strictures
 Toxic megacolon
 Tropical sprue
 Volvulus
 Whipple's disease

Endocrine-Related

Carcinoid syndrome (serotonin)
 Hormonal hypersecretion
 Hyperthyroidism (thyroid hormone)
 Medullary carcinoma of the thyroid (calcitonin)
 Pancreatic cholera (VIP)
 Somatostatinoma (somatostatin)
 Systemic mastocytosis (histamine)
 Zollinger-Ellison syndrome (gastrin)

Endocrine Pathology

Adrenal insufficiency
 Diabetes enteropathy
 Hypoparathyroidism
 Pancreatic insufficiency

Systemic Illness/Other

Alcoholism
 Amyloidosis
 Connective tissue disease
 Cystic fibrosis
 Ectopic pregnancy
 Hemolytic-uremic syndrome
 Henoch-Schönlein purpura
 Lymphoma
 Otitis media—infants
 Pelvic inflammatory disease
 Pneumonia/sepsis
 Pyelonephritis
 Scleroderma/SLE
 Severe malnutrition
 Stevens-Johnson syndrome
 Toxic shock syndrome
 Wilson's disease
 Miscellaneous
 Factitious diarrhea
 Runner's diarrhea

ACE, angiotensin-converting enzyme; GI, gastrointestinal; NSAIDs, nonsteroidal anti-inflammatory drugs; SLE, systemic lupus erythematosus; VIP, vasoactive intestinal polypeptide.

Gross blood may be consistent with invasive, infectious diarrhea but may be the harbinger of many other pathologic states that manifest with gastrointestinal bleeding. Histamine-induced skin changes may be indicative of an intestinal parasitic infection. The patient should be assessed for specific toxidromes, such as cholinergic or sympathomimetic states that may be clues to a noninfectious cause.

Characterization of the Diarrheal Syndrome

Acute Infectious Diarrhea Most viral and many bacterial agents cause a self-limited, secretory diarrhea that lasts less than 14 days and causes only mild dehydration and minimal systemic symp-

oms. These infections do not require extensive testing and are treated symptomatically. In the United States, monitoring of pathogens causing this type of acute gastroenteritis demonstrates that 90% of the infections are caused by norovirus species.⁸ All other potential causes of diarrhea are highly improbable unless certain historical and clinical findings are present. Bacterial and protozoan agents less commonly cause diarrhea syndromes indistinguishable from norovirus infection with a nontoxic, self-limited course. A Bayesian approach to diagnosing and treating acute diarrhea has been proposed.⁸ The clinical evaluation should screen for all factors (Table 23-1) that may change the probability of “not norovirus” from 10% to 50% or greater.⁸ With one or more of these findings

Table 23-1 Factors Increasing Probability of Nonbenign Diarrhea

FACTOR	SPECIFIC PATHOGEN(S)/OTHER CONSIDERATIONS
Presentation to a health care facility	Degree of illness overall greater in patients presenting for evaluation; increased probability of “not norovirus” etiology to 50%
Travel history	Especially foreign travel and to endemic areas of dysenteric disease
Recent hospitalization	<i>C. difficile</i> from antibiotic exposure
Day care attendance	Rotavirus, <i>Shigella</i> , <i>Giardia</i>
Nursing home residence	<i>C. difficile</i> , medication side effects, tube feedings, ischemic colitis, fecal impaction, and overflow diarrhea
Wilderness exposure	<i>Giardia</i> or <i>Cryptosporidium</i>
Antibiotic therapy	<i>C. difficile</i> , antibiotic side effects
Raw shellfish, farm animals and fair livestock, pet reptiles or amphibians, petting zoos	<i>Salmonella</i> spp., <i>E. coli</i> O157:H7 and non-O157 Shiga toxin-producing <i>E. coli</i> , <i>Vibrio</i> spp.
Epidemic of multiple patients with a short time of onset	Norovirus; less commonly, <i>Campylobacter jejuni</i> , <i>Salmonella</i> spp., <i>Cryptosporidium</i>
Acute vomiting and diarrhea after suspected contaminated food	<i>Bacillus cereus</i> , <i>Clostridium botulinum</i> , <i>Staphylococcus aureus</i>
Epidemic of severe gastroenteritis traced to eggs, poultry, meat, or dairy products	<i>Campylobacter jejuni</i> , <i>Salmonella</i> spp.
Homosexual lifestyle (males)	<i>Giardia lamblia</i> , <i>Entamoeba histolytica</i>
Abdominal pain	Severe bacterial infections: <i>Salmonella</i> , <i>Campylobacter</i> , <i>Shigella</i> , EPEC, <i>Yersinia</i> or <i>Vibrio</i> spp.
Nausea, vomiting	Also consider surgical abdomen, GI bleeding
Bloody stool	Inflammatory bowel disease
Fever	
Rectal pain	
Tenesmus	
Diarrhea >7–14 days' duration	Protozoa and microsporidia, <i>Clostridium difficile</i> , <i>Campylobacter</i> , Shiga toxin-producing <i>E. coli</i>
Hemolytic uremic syndrome	<i>E. coli</i> O157:H7 or other species
Stool WBC count	Not reliable for diagnosis of bacterial etiology
Colonic ulcerations	Inflammatory bowel disease
Proctitis	Bacterial etiology highly probable
Pseudomembranes	Toxic megacolon, <i>Clostridium difficile</i>
Chronic disease (e.g., cirrhosis, DM)	Complicated course expected with any form of diarrheal illness
Organ transplantation	Abnormally severe illness from rotavirus and adenovirus Increased frequency of cytomegalovirus Severe illness from dysenteric diarrhea Spore-forming protozoa and microsporidia
HIV infection, other immunodeficiency disorders	Severe illness from common bacteria/spore-forming protozoa and microsporidia Increased frequency of cytomegalovirus and <i>Mycobacterium avium</i> complex

DM, diabetes mellitus; EPEC, enteropathogenic *E. coli*; WBC, white blood cell.

present, empirical antibiotic or other specific therapy may be indicated, as well as clinical testing to determine the exact etiologic disorder.

Chronic Infectious Diarrhea Persistent diarrhea is defined as that lasting for more than 14 days, and chronic diarrhea, more than 30 days.⁵ Infectious agents of persistent and chronic diarrhea include bacteria, parasites, and rarely viruses.⁹ Common bacterial pathogens include *Aeromonas*, *Plesiomonas*, *Campylobacter*, *Clostridium difficile*, *Salmonella*, and *Mycobacterium tuberculosis*. Parasites causing chronic diarrhea are colonic forms such as *Amoeba*, *Trichuris*, *Yersinia*, and *Schistosoma* species or small intestinal pathogens such as *Giardia*, *Cryptosporidium*, *Cyclospora*, *Isospora*, and *Strongyloides*.⁹ In developing countries, chronic diarrhea is more likely to have a bacterial cause. In developed countries, chronic diarrhea is caused by noninfectious disorders such as irritable bowel syndrome, malabsorption syndromes, laxative abuse, and inflammatory bowel disease.⁶ Categorization of the stool type as watery, inflammatory, or fatty may assist in proper classification of the chronic

diarrhea syndrome as infectious and noninfectious. Testing for HIV or immune deficiency is important because these patients commonly present with chronic diarrhea. Evaluation should include testing for cryptosporidia, microsporidia, mycobacteria (i.e., *Mycobacterium avium* complex), herpes simplex virus (HSV), *Isospora*, *Cyclospora*, and cytomegalovirus. In addition, parasitic and helminthic infections should be ruled out.

Noninfectious Diarrhea Noninfectious causes of diarrhea (see Box 23-2) are responsible for approximately 15% of all cases of diarrheal illness. The distinction between infectious and noninfectious causes may not be clinically apparent. A complete evaluation should consider possible surgical pathology of the abdomen, including gastrointestinal bleeding, ischemic bowel, acute appendicitis, intussusception, ectopic pregnancy, and partial bowel obstruction.⁷ The differential diagnosis also includes possible toxic exposures or ingestions, such as heavy metal poisoning, or ingestion of plant-borne or fish-borne toxins. Endocrine pathology, such as adrenal insufficiency, hyperthyroidism, diabetic enteropathy, and

hormone-secreting tumors, and other systemic illnesses should be considered, and special attention should be directed at underlying medical conditions, medication use, and past surgical history.

Ancillary Testing

Most cases of acute diarrhea are self-limited, and laboratory and diagnostic tests should be kept to a minimum unless required for epidemiologic studies. Testing is indicated in patients who have a high probability of a “non-norovirus” clinical picture and have worrisome historical data, signs, and symptoms associated with an increased probability of those causes. Ancillary testing should never compromise empirical treatment when indicated (as discussed later on). Fever with a toxic appearance and volume depletion, blood- or mucus-containing stools, frequent voluminous stools, and other risk factors (Table 23-1) should prompt a diligent search for a specific causative disorder in order to guide appropriate therapy.^{8,10} A white blood cell count is rarely helpful and not sensitive or specific enough to aid in diagnostic decision-making, although hemoglobin determination is useful to screen for anemia from blood loss, and abnormalities in platelet and coagulation parameters may contribute to identification of a cause for gastrointestinal bleeding. A comprehensive chemistry panel including renal function tests can be important when significant volume loss is suspected, or when significant diarrhea has been present for 48 to 72 hours. Liver function studies, thyroid tests, serum lipase assay, and a pregnancy test may be helpful in selected cases.

Hemocult and fecal cell count: The presence of fecal leukocytes is not specific or sensitive enough to use as the sole criterion to decide which patients with presumed bacterial gastroenteritis should be treated empirically with antibiotics. With inflammatory diarrhea of various causes, red and white blood cells are seen on stool examination. Included are bacterial, parasitic, and noninfectious causes, such as chemotherapy, radiation therapy, hypersensitivity reactions, autoimmune disorders, and inflammatory bowel disease. The presence of fecal leukocytes does not delineate which patients would benefit from empirical antimicrobial therapy. The presence of blood does not always correlate with the presence of fecal leukocytes, so reliance on positive stool guaiac test result alone as a rationale for antibiotic therapy is not recommended. The presence of blood without fecal leukocytes may indicate amebiasis, malignancies, heavy metal poisoning, fissures, hemorrhoids, bowel ischemia, or primary gastrointestinal bleeding.

Assays for calprotectin and lactoferrin, produced by leukocytes, are sensitive and specific and may be more useful than microscopic examination of the stool, but these tests are rarely, if ever, of use in the ED.⁶

Clostridium difficile toxin assay: This test is indicated if the patient reports recent antibiotic use. *C. difficile*-associated diarrhea most commonly occurs during or shortly after the antibiotic course. In 25% to 40% of cases, however, onset of the diarrhea may be delayed as long as 12 weeks after antibiotic therapy. The most commonly implicated antibiotics are cephalosporins, penicillins, and clindamycin. Although *C. difficile* accounts for only 10% to 20% of antibiotic-associated diarrhea, an assay for *C. difficile* toxin gives a positive result in nearly all cases of antibiotic-associated pseudomembranous colitis.¹¹ Approximately 3% of adult patients and 65% of newborns may be colonized with *C. difficile*.

E. coli O157:H7 toxin assay: This test is considered in endemic areas and in patients with suspected hemolytic-uremic syndrome.¹²

Stool culture for bacteria: Stool cultures may be warranted in patients who are febrile, toxic-appearing, immunocompromised, at the extremes of age, experiencing a prolonged course, or not responding to conventional treatment. Studies have shown a 2% positive rate, thus proving that routine cultures are of limited value.¹²

Stool examination for ova and parasites: The assessment of stool for ova and parasites is not routinely recommended. This study is used in patients with chronic diarrhea (*E. histolytica*, *Cryptosporidium*); patients with a history of travel to developing countries, particularly to Nepal or areas of Russia (*Cryptosporidium*, *Giardia*, *Cyclospora*)¹³; patients with exposure to infants in day care centers (*Cryptosporidium*, *Giardia*); and patients with HIV infection (*E. histolytica*, *Giardia*).¹⁴

Giardia antigen assay and serologic testing for amebiasis may be considered in patients exposed to poor sanitation, HIV-infected patients, patients with a history of travel to developing countries, patients with a history of backpacking, and patients with day care exposures.

Urinalysis: A urinalysis and a urine pregnancy test should be obtained only when urinary tract infection is a possibility, a gastrointestinal origin for the symptoms is not clear, or pregnancy is suspected.

Radiographic studies: Plain radiographs and contrast computed tomography (CT) may be indicated for patients thought to have a surgical abdomen and to identify pathologic abnormalities, such as tumor, obstruction, free air, fistulas, blind loops, and those associated with Crohn's disease.

Gastrointestinal referral: Referral may be indicated in the evaluation of chronic diarrhea and for workup beyond the scope of the ED (e.g., endoscopy, further stool studies, biopsy).

■ EMPIRICAL MANAGEMENT

Oral rehydration is the treatment of choice for mild to moderate fluid losses (Fig. 23-1). Oral rehydration can be accomplished using sports beverages, commercial rehydration solutions, or a balanced clear liquid diet in the home (e.g.,

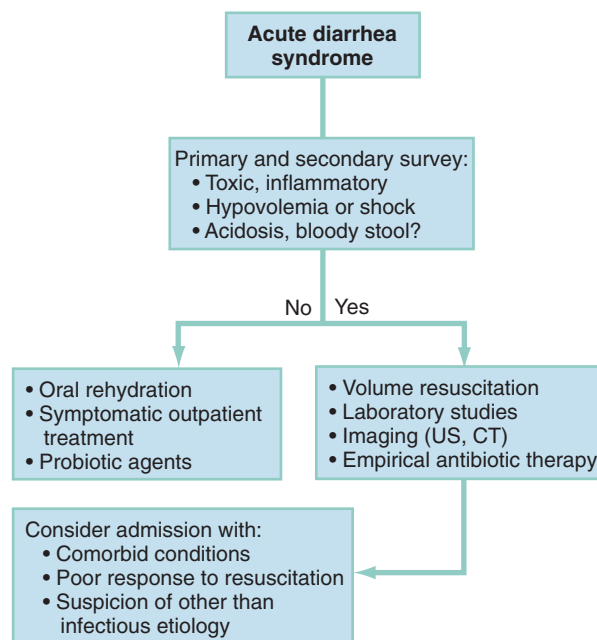


Figure 23-1. Approach to the patient with acute diarrhea. CT, computed tomography; US, ultrasonography.

consisting of water, salt-containing liquids such as canned soups, and potassium from oranges or bananas). The WHO has defined an oral rehydration solution (WHO-ORS) that can be made by dissolving the following in 1 liter of clean water:

3.5 g of sodium chloride
2.9 g of trisodium citrate or 2.5 g of sodium bicarbonate
1.5 g of potassium chloride
20 g of glucose or 40 g of sucrose

Replacement of micronutrients, particularly copper and zinc, has been recommended, especially in developing countries.¹⁵ The concept of bowel rest has been abandoned because it may worsen diarrhea and lead to more severe dehydration. The choice of oral rehydration fluids depends on the extent of dehydration and the underlying health of the patient. In otherwise healthy patients with mild to moderate dehydration, fluids such as sports drinks, diluted fruit juices, and soft drinks supplemented with soups, broths, or crackers may be sufficient to replace the fluid and sodium losses associated with acute diarrhea. Such frequently used “clear liquids” may contain excess sugars and insufficient sodium content, however, leading to an osmotic diarrhea. Beverages containing caffeine should be avoided because caffeine increases cyclic adenosine monophosphate levels and may cause a secretory diarrhea. Milk and other products containing lactose also should be avoided because viral and bacterial pathogens, responsible for many cases of diarrhea, may cause a transient lactase deficiency, leading to malabsorption and osmotic diarrhea. Food intake is encouraged, but foods high in simple sugars should be avoided because the osmotic effect is counterproductive. Foods with a high fat content may delay gastric emptying and should be avoided. The *BRAT* (bananas, rice, apples, and toast) diet has long been recommended, particularly with pediatric patients. Although no controlled studies have examined the efficacy of the *BRAT* diet, it remains a commonly recommended strategy. The pectin in the peel of apples is constipating (pectin, found in fruit peel, is the “pectate” in Kaopectate), and bananas provide potassium. If this diet is used for extended periods, adequate provision of protein and energy needs of the patient becomes a concern.

In patients with evidence of more severe dehydration, intravenous fluid resuscitation with normal saline or lactated Ringer's solution is the preferred treatment. Pediatric patients should receive a bolus of 20 mL/kg of normal saline, which may be repeated as indicated. Specific treatment for diarrhea should be directed toward the suspected cause. In patients with suspected surgical pathology, further diagnostic testing and surgical consultation may be required. With toxic exposures, treatment consists of early decontamination, supportive care, and, if appropriate, administration of specific antidotes. Other noninfectious causes of diarrhea are treated as indicated.

Because the specific pathogen causing infectious diarrhea is rarely identified in the ED, and the results of cultures are usually unavailable, any antimicrobial treatment must be empirical and guided by knowledge of the common causes of infectious diarrhea (see [Box 23-1](#)). Viral and noninvasive bacterial gastroenteritis tend to be self-limiting and require only supportive therapy. Empirical antibiotic treatment is directed against invasive bacterial and parasitic organisms that cause the greatest harm. Antibiotic treatment is initiated in patients with a suspected invasive process and severe diarrhea, sys-

temic symptoms, fever, or abdominal pain and in patients who appear toxic. The current recommendation for empirical treatment of a systemically ill-appearing adult is ciprofloxacin, 500 mg orally twice a day, or levofloxacin, 500 mg orally every 24 hours for 3 to 5 days.¹⁶ Fluoroquinolones are efficacious against most organisms that cause dysenteric illnesses and have been shown to be more effective than trimethoprim-sulfamethoxazole.^{13,17} Fluoroquinolones should not be administered to pregnant patients or children younger than 18 years of age. The antibiotic treatment of severe gastroenteritis in children has been associated with the development of hemolytic-uremic syndrome and thrombotic thrombocytopenic purpura if the bacterial cause is enterohemorrhagic *E. coli* O157:H7, although *Salmonella*, *Shigella*, and *Campylobacter* also have been implicated. If possible, treatment for pediatric patients should be based on culture results with supportive care initially.

If amebic dysentery is of concern in high-risk patients (see [Table 23-1](#)), treatment with metronidazole after stool analysis for ova and parasites is recommended. In patients with a history of recent antibiotic use suspected of having *C. difficile* colitis, a *C. difficile* toxin assay followed by vancomycin or metronidazole is appropriate.¹⁸

The use of antimotility agents in the treatment of acute enteritis has been controversial, with the literature divided over this issue. Patients with simple, acute viral gastroenteritis benefit from antimotility agents and often obtain significant relief of symptoms, with less fluid loss and without significant complications. Loperamide is the safest and most effective medication. Relief of symptoms is achieved much more rapidly than with bismuth subsalicylate (Pepto-Bismol) in patients with inflammatory diarrhea or antibiotic-associated colitis. In pediatric age groups, the use of opioids, loperamide, or diphenoxylate with atropine rarely has been associated with the precipitation of toxic megacolon and hemolytic-uremic syndrome. Because the beneficial effects of these medications are modest, they should be avoided or used with extreme caution in these high-risk patients.

Probiotics have been used as an alternative to traditional antibiotic therapy for diarrhea. *Lactobacillus* and other bacteria have proved to be effective in restoring the normal gastrointestinal flora that is disrupted during diarrhea illness. This approach has been most effective with traveler's diarrhea and nonspecific diarrhea in children.

■ DISPOSITION

Most patients with uncomplicated, acute diarrhea can be discharged home after assessment and symptomatic relief. Hospitalization rarely is required for diarrhea secondary to viral and many forms of bacterial gastroenteritis, which tend to be self-limiting. Often the exact etiologic agent of diarrhea is not identified in the ED. An understanding of common causes and their treatment and recognition of patients at risk for a more severe clinical course are essential to make the appropriate disposition. In patients with severe dehydration, hemodynamic instability, or a toxic appearance and in high-risk groups, hospital admission is warranted for continuous monitoring, further treatment, and definitive management when initial evaluation and stabilization are complete.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.

■ PERSPECTIVE

Constipation is a symptom, not a disease. Patients and doctors often define constipation differently. Patients often use the term *constipation* to describe a broad set of complaints including straining, hard or infrequent stools, feeling of incomplete evacuation, and abdominal discomfort. Constipation may be acute (new for the patient) or chronic. Chronic constipation is defined as the presence of symptoms for at least 3 months. The Rome III criteria constitute a consensus definition of functional chronic constipation often used in research (Table 24-1).¹ Attempting to identify the cause of this symptom will often result in the best chance of effective treatment and will help determine disposition. A definitive diagnosis often is not possible in the emergency department, and appropriate follow-up evaluation should be arranged in those cases. When constipation becomes severe with constant pain, some clinicians use the term *obstipation*. Obstipation represents the progression of the symptom of constipation toward bowel obstruction.

In the emergency department (ED), the complaint of constipation should be of concern when it represents a significant change from a patient's own normal pattern that is creating discomfort for the patient. This change may manifest as a decrease in frequency of defecation, sudden and persistent change in the character or amount of stools (especially decrease in stool caliber), blood in the stool, or problems expelling the stool.²

Epidemiology

The prevalence of constipation in North America is approximately 15%. Constipation is more common in women than in men, in nonwhites than in whites, and in the elderly. A consistent trend of increasing prevalence of constipation is observed with age, with significant increases after the age of 70 years. The high prevalence among elderly patients is multifactorial and related to a diet low in fiber, sedentary habits, multiple medications, and various disease processes that impair neurologic and motor control. Constipation also is common with patients who are institutionalized, debilitated, or neurologically impaired.³

Pathophysiology

Normally the gastrointestinal tract is presented with 9 to 10 L/day of secretions and ingested fluids. The small intestine

usually absorbs all of this except for approximately 500 mL. The colon mixes the ileal effluent, ferments and salvages the unabsorbed carbohydrate residues, and desiccates the contents to form stool. The process of stool transport and evacuation is complex and is regulated by neurotransmitters, intrinsic colonic reflexes, and a multitude of learned and reflex mechanisms that are not fully understood. Constipation may result from structural, metabolic, mechanical, neurologic, or behavioral disorders that affect the colon or anorectum either directly or indirectly.⁴⁻⁶

■ DIAGNOSTIC APPROACH

Differential Considerations

The causes of constipation are numerous. Causes of constipation can be divided into *primary* (no apparent external cause) and *secondary* causes (summarized in Table 24-2). These two groupings have some overlap. In the ED, patients commonly present with acute constipation due to side effects of medications or avoidance of defecation secondary to presence of painful perianal lesions such as fissures, hemorrhoids, or perirectal abscesses.⁶

Pivotal Findings

History

A thorough, detailed history usually identifies the most likely cause of the patient's constipation. Defining what the patient means by "constipation" is a good starting point. Essential information includes the presence or absence of alarming signs or symptoms. These include fevers, anorexia, nausea, vomiting, new onset or worsening of constipation, blood in the stool, weight loss, and a family history of inflammatory bowel disease or colon cancer.

Additional elements of the history are directed toward elucidating a possible cause. Questions about the character of the stools may reveal a decrease in caliber of the stool, suggesting possible mass lesion, or diarrhea alternating with constipation, which may indicate irritable bowel syndrome. Frequency of stools and what the patient considers "normal" should be assessed.

The review of systems may need to include questions regarding associated symptoms if no obvious cause is elicited in the cursory history. Questions directed at associated neuro-

Table 24-1 Rome III Criteria for Functional Constipation

- (1) At least 2 of the following for a minimum of 3 months, with symptom onset at least 6 months before diagnosis:
 - (a) Straining during $\geq 25\%$ of bowel movements
 - (b) Lumpy or hard stools for $\geq 25\%$ of bowel movements
 - (c) Sensation of incomplete evacuation for $\geq 25\%$ of bowel movements
 - (d) Manual maneuvers to facilitate $\geq 25\%$ of bowel movements (e.g., digital evacuation, support of the pelvic floor)
 - (e) < 3 bowel movements per week
- (2) Loose stools rarely present without use of laxatives
- (3) Insufficient criteria for irritable bowel syndrome

logic symptoms, activity level, and status of comorbid diseases may provide clues to contributing factors.⁷

A medication history is essential and should include any recent changes in dosing of any prescription medications, herbal agents, and over-the-counter (OTC) medications. Many patients experience constipation as a side effect of medication. Drugs of abuse also may cause changes in bowel patterns. Opiates are the most common cause of constipation among medications and drugs of abuse.

Physical Examination

The physical examination should initially focus on two major aspects: the abdominal and rectal portions of the physical examination. The abdominal examination usually yields normal findings but may reveal tenderness, a mass, distention, or possibly evidence of obstruction. Bowel sounds should be auscultated.

The anorectal examination and an evaluation of the stool are the most important parts of the physical assessment. Anorectal inspection may reveal fissures, skin excoriations, hemorrhoids, or rectal prolapse. The digital rectal examination should include careful palpation for masses, and the presence or absence of pain should be noted. Other possible findings include strictures, high sphincter tone, and the presence of blood. Having the patient bear down may be helpful in assessing sphincter function and may reveal milder forms of prolapse. The quantity and the characteristics of the stool should be recorded. Testing the stool for occult blood may yield additional information, although straining at stool can produce local anal lesions and bleeding. If results of occult blood testing are positive, diverticular disease, carcinoma, and simply trauma from repeated attempts at straining all are possibilities. Patients with acute constipation who present to the ED most commonly have large amounts of hard stool in the rectum. Results of rectal examination have not been shown, however, to correlate with complaints of constipation or with evidence of colonic loading on abdominal radiographs. The rectal examination alone should not be used to confirm or exclude the presence of constipation.⁸

Ancillary Testing

A majority of patients who present to the ED with a chief complaint of constipation do not need any testing. Plain radiographs may provide information about extent of stool retention but also may suggest emergent diagnoses such as megacolon or volvulus. Although constipation can cause cramping and abdominal pain, plain radiographs documenting an increased stool load in the constipated patient cannot be used to rule out more serious underlying etiologic disorders, especially if the

Table 24-2 Causes of Constipation

Primary Causes

Functional Disorders

Idiopathic
Irritable bowel syndrome
Pelvic dyssynergia (anismus)
Slow-transit constipation

Neuropathic

Congenital anal sphincter myopathy
Hirschsprung's disease
Spinal cord injury

Obstructive

Anal stenosis
Crohn's disease
Colon cancer
Stricture
Rectal prolapse

Gynecologic

Large rectocele
Pelvic relaxation

Secondary Causes

Lifestyle/General Condition

Dehydration
Inadequate dietary fiber
Sedentary
Voluntary suppression of defecation

Medications

Antacids
Anticholinergics
Anticonvulsants
Antidepressants
Antihistamines
Antiparkinsonian drugs
Antipsychotics
Calcium channel blockers
Calcium supplements
Diuretics
Iron supplements
Laxatives (chronic abuse)
Nonsteroidal anti-inflammatory drugs
Opiates

Metabolic/Endocrine

Diabetes mellitus
Hypercalcemia
Hypokalemia
Hypothyroidism
Hypomagnesemia
Porphyria
Uremia

Myopathic

Scleroderma
Amyloidosis
Neurologic
Cerebrovascular accident
Autonomic neuropathy
Multiple sclerosis
Paraneoplastic neuropathy
Parkinson's disease
Amyotrophic lateral sclerosis

Psychological

Anxiety
Depression
Eating disorders
Situational stress
Sexual abuse

Adapted from Swegle JM, Logemann C: Management of common opioid-induced adverse effects. *Am Fam Physician* 74:1347, 2006.

patient has a significant amount of abdominal pain or tenderness on examination.

Clinical laboratory studies are not routinely indicated in the workup for constipation. When blood is found in the stool, a hemoglobin level may reveal an accompanying anemia, which may suggest an occult carcinoma. The white blood cell count is nonspecific and not helpful.

Patients with acute constipation for which the cause is not readily apparent should receive symptomatic treatment, with referral for outpatient evaluation and reassessment as needed. The patient who presents to the ED with chronic constipation and no alarming signs or symptoms should receive empirical treatment without any ancillary testing. Outpatient tests may eventually include blood tests to investigate metabolic or endocrine causes and possibly specialized tests such as colonic transit studies, defecography, and anorectal manometry with balloon expulsion. Consensus recommendations state that the

routine use of colonoscopy to exclude organic disorders in patients with chronic constipation symptoms is not indicated, although it is still recommended for colon cancer screening in all patients older than 50 years of age.^{9,10}

■ DIAGNOSTIC ALGORITHM

The approach to the patient with constipation starts with assessing whether or not this symptom is accompanied by the additional symptom of abdominal pain. If such pain is present, the workup should be geared toward this symptom, which may ultimately reveal the cause of the constipation. Constipation may itself cause abdominal pain; however, this should be a diagnosis of exclusion once other, more serious potential etiologic disorders are ruled out.

Figure 24-1 presents a diagnostic algorithm. If the physical examination reveals a structural or mechanical cause, such as

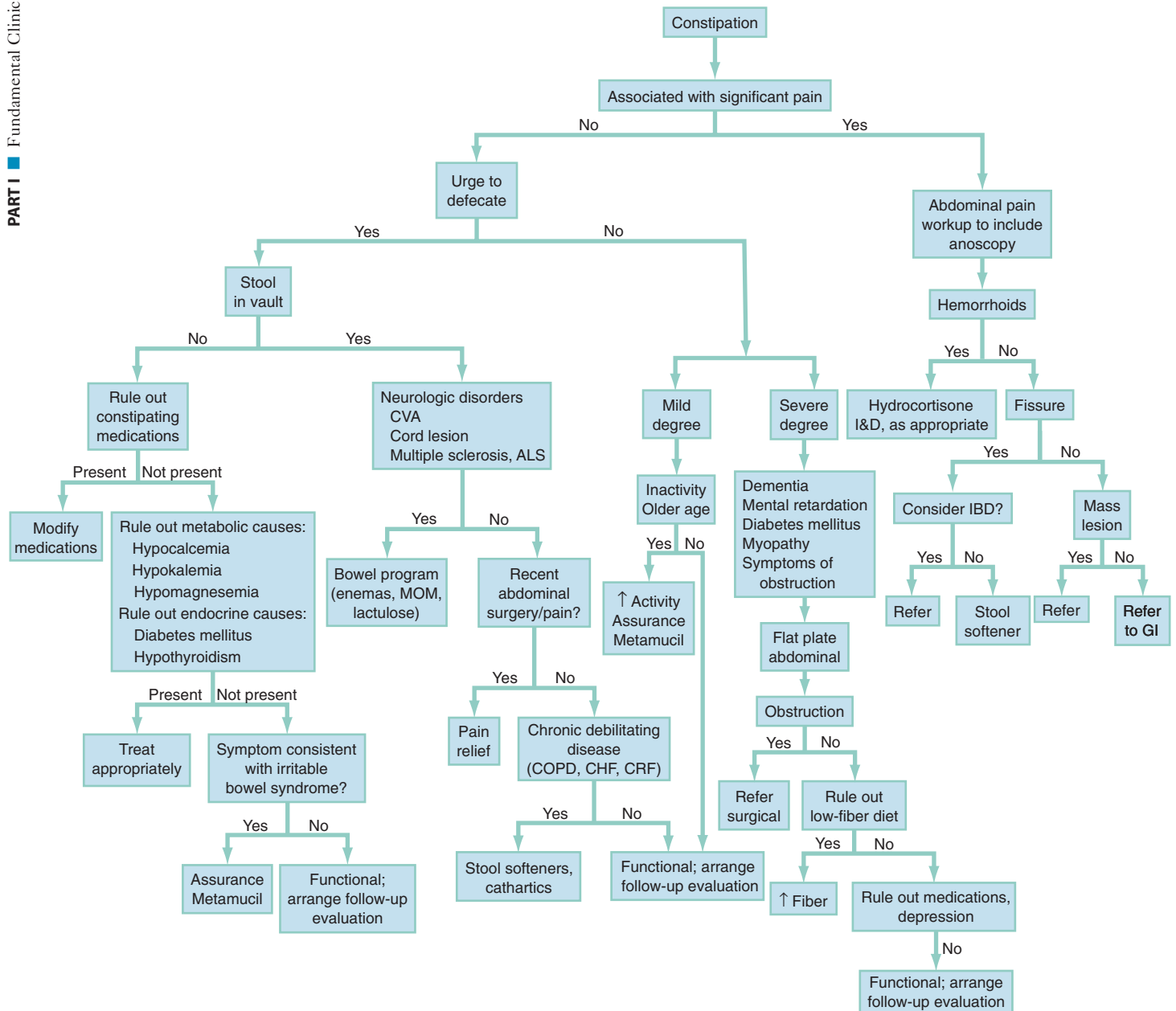


Figure 24-1. Algorithmic approach to the diagnosis of constipation. ALS, amyotrophic lateral sclerosis; CHF, congestive heart failure; COPD, chronic obstructive pulmonary disease; CRF, chronic renal failure; IBD, inflammatory bowel disease; I&D, incision and drainage; MOM, milk of magnesia.

pain from hemorrhoids, fissures, or mass lesion, the appropriate treatment or referral is indicated; the constipation will resolve once the cause is addressed. If no obvious cause is found on examination, then determination of the presence or absence of stool in the rectal vault may be helpful. History will be very helpful in differentiating between causes such as medication side effect and possible neurologic disease.

Constipation is rarely associated with morbidity or mortality. Most bad outcomes are due to missed diagnosis of bowel obstruction or perforation. These conditions are diagnosed with physical examination, plain radiographs, and computed tomography (CT) scan if needed. Surgical consultation is needed for suspected perforation and obstruction.

■ EMPIRICAL MANAGEMENT

Treatment of acute constipation is directed toward eradicating the underlying cause and providing symptom relief. Prevention of further episodes of constipation may include recommending increased fluid intake, increased dietary fiber, and, if necessary, additional sources of bulk in the form of synthetic bulk agents. These interventions will not usually help the acutely constipated patient in the short term.¹⁰ Laxatives often will be required¹¹ (see Table 24-3). Specific therapy also may include actions such as withholding a causal medication, management of an anal fissure, or draining of a perirectal abscess. Stool softeners have not been shown to be any more effective than placebo at relieving acute constipation, although they may be somewhat helpful in patients with anal fissures or hemorrhoids, which can make defecation painful.^{4,12}

Specific agents for symptomatic treatment of constipation are listed in Table 24-4. Most of these agents are designated category B or C as determined by the U.S. Food and Drug Administration (FDA). A consensus panel recently concluded that polyethylene glycol (category C) was the optimal laxative for pregnant women because it is effective and minimally absorbed, has few side effects, and is low risk.¹³ Patients who are on chronic, medically necessary medications that cause constipation (e.g., opioids in patients with chronic pain or cancer) should be on so-called bowel regimens. These regimens usually include preventive measures such as high levels of dietary fiber (e.g., in prunes or figs) as well as stimulant laxatives. Patients on chronic opioids with acute constipation may also respond to naloxone although precipitating withdrawal symptoms are possible.¹⁴ The new specific peripheral opioid receptor agents on the horizon may prove to be very useful in treating the constipation associated with chronic opioid use without reversing central actions.¹⁵ Elderly patients who are prescribed opiates from the ED for home use should be warned about constipation and given instructions to prevent and treat it.

Enemas are sometimes necessary if laxatives have failed to provide relief or if the patient has a large volume of stool in the lower colon or rectum that cannot be expelled. Warm tap-water enemas probably are the safest. For immediate relief, manual disimpaction may be necessary in some patients, especially in elderly persons with large amounts of stool present in the rectal vault. In the rare case, disimpaction may need to be performed with procedural sedation.

There are alternatives to the traditional laxatives and enemas for patients who suffer from chronic constipation. Patients with recalcitrant constipation may benefit from interventions such as acupuncture, biofeedback, and bowel training.^{16,17}

Table 24-3

General Approach to Treatment of Constipation

For specific agents, dosages, and precautions, see Table 24-4.

- I. Core program for all patients
 - A. Adequate intake of fluid and fiber is one key to preventing constipation. Fiber is available primarily from grains and bran cereals. Flatulence, bloating, and cramps are common side effects encountered when bran fiber is introduced.
 - B. Another source of bulk is from synthetic bulk agents (e.g., psyllium). Bulk agents require an adequate amount of fluid intake; otherwise, they may worsen constipation.
 - C. Avoid irritant laxatives as part of a core program because long-term use may decrease bowel motility. Encourage the patient to exercise and respond promptly to the urge to defecate.
- II. Individualized program—specific indications and general comments
 - A. *Stimulant laxatives*: Many believe that long-term use of these agents leads to dependency and habituation, but this is not substantiated. When used appropriately, these medications are not harmful and are very effective. Senna is probably the first-line choice among this class of laxatives.
 - B. *Osmotic laxatives*: These agents are most commonly used for colonic preparation before bowel procedures. This class of drugs includes magnesium-containing laxatives, polyethylene glycol (PEG), and nonabsorbable sugars such as lactulose and sorbitol. These agents are safe and well tolerated. PEG has been shown to be slightly more effective than lactulose and causes less bloating and flatus.
 - C. *Lubricants*: Oral mineral oil lubricants are particularly helpful in patients who have acute painful perianal lesions. The softening and coating of the stool can make passage much easier and less painful, preventing constipation. It is also helpful in elderly patients who have chronically hard stools and usually is well tolerated. Mineral oil is contraindicated in patients with swallowing problems or in those who are particularly debilitated, to prevent aspiration leading to lipid pneumonia.
 - D. *Stool softeners*: Stool softeners are wetting agents believed to enhance the moisture content of fecal material. Evidence exists that stool softeners are no more effective than placebo and certainly not any better than other agents available.
 - E. *Suppositories and enemas*: These agents are especially helpful in patients who tend to have trouble expelling soft stool from the rectum. Glycerin suppositories may have a soothing effect and be helpful in patients with constipation caused by local, painful perianal lesions. Tap-water enemas or soapsuds enemas are helpful when disimpaction is necessary.

■ DISPOSITION

Constipation is appropriately treated at home, and only the most severe cases require disimpaction or enema treatment in the ED. With complications or presence of a serious disorder as a cause for the constipation, such as fecal impaction beyond that able to be resolved by digital disimpaction, megacolon, volvulus, or bowel obstruction, the patient should be admitted to the hospital for further evaluation and treatment.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.

Table 24-4 Preparations Used in the Symptomatic Treatment of Constipation

MEDICATION	MAXIMAL RECOMMENDED DOSE	ONSET OF ACTION	COMMENTS
Bulk laxatives			
Psyllium (Metamucil)	Titrate up to 20 g	12–72 hr	Increases colonic residue, stimulates peristalsis. Natural fiber that undergoes bacterial degradation, which may contribute to bloating and flatus. Should be taken with plenty of water to avoid intestinal obstruction.
Methylcellulose (Citrucel)	Titrate up to 20 g		Semisynthetic cellulose fiber that is relatively resistant to colonic bacterial degradation.
Polycarbophil (Fibercon)	Titrate up to 20 g		Synthetic fiber of polymer of acrylic acid, resistant to bacterial degradation.
Osmotic laxatives			Draws water into the intestines along osmotic gradient.
<i>Magnesium/sodium salts</i>			
Magnesium hydroxide (Milk of Magnesia)	15–30 mL once or twice daily	0.5–3 hr	A small percentage of magnesium is absorbed—caution in renal insufficiency and in children.
Magnesium citrate	150–300 mL as needed		
Sodium phosphate (Fleet phospho-soda)	20–45 mL with 12 oz of water as needed		Hyperphosphatemia may result if patient has renal insufficiency. Commonly used before colonoscopy.
<i>Poorly absorbed sugars</i>			
Lactulose	15–30 mL once or twice a day	24–72 hr	Synthetic disaccharide not absorbed by the small intestine. Gas and bloating common.
Sorbitol	15–30 mL once or twice a day		Poorly absorbed by small intestine.
Polyethylene glycol and electrolytes (GoLYTELY, Miralax)	17–36 g once or twice a day	1–24 hr	Organic polymers that are poorly absorbed and not metabolized by bacteria, thus may cause less bloating and cramping. Can be mixed with noncarbonated beverages.
Stimulant laxatives			Stimulate intestinal motility or secretion.
Senna (Senokot, Ex-lax)	8–34 mg daily	6–12 hr	Stimulates secretion and motility of small intestine and colon.
Bisacodyl (Dulcolax, Correctol)	5–10 mg daily		
Castor oil	15–30 mL daily		Causes cramping and severe diarrhea.
Stool softeners			
Docusate sodium (Colace)	100 mg twice a day; some use higher doses	6–8 hr	In many studies, no better than placebo.
Mineral oil (Fleet mineral oil)	5–15 mL orally at night		Provides lubrication for the passage of stool. Long-term use is not recommended. Lipid pneumonia can occur in patients predisposed to aspiration.
Enterokinetic agents			
Lubiprostone (Amitiza)	24 µg once or twice per day	1 hr	A chloride channel activator. Approved for treatment of chronic idiopathic constipation in adults. Headache, nausea, possible diarrhea.

CHAPTER 25 Jaundice

Matthew A. Wheatley and Katherine L. Heilpern

■ PERSPECTIVE

Epidemiology and Pathophysiology

Jaundice affects patients of all ages, from neonates to the elderly. It is not a common chief complaint; however, the jaundiced patient may present with a related symptom such as abdominal pain, pruritus, or substance ingestion. Jaundice is the manifestation of elevated serum bilirubin, so an understanding of the metabolism of bilirubin is crucial for the emergency evaluation and management of jaundice.

Bilirubin Metabolism

Bilirubin is generated from heme products, primarily senescent red blood cells. A small portion is derived from myoglobin and maturing erythroid cells. Within the reticuloendothelial system, heme is oxidized to biliverdin, which is then converted to bilirubin. Bilirubin forms a tight but reversible bond with albumin in circulation. It is passively taken into the hepatocytes, where it undergoes glucuronidation. This conjugated fraction is secreted into the biliary system and emptied into the gut. Colonic bacteria metabolize the major portion of the bilirubin to urobilinogen and stercobilin. Stercobilin is excreted in the stool and urobilinogen is reabsorbed and excreted in the urine. The remaining conjugated bilirubin is deconjugated and reenters the portal circulation to be taken up again by the liver (enterohepatic circulation).

In the laboratory, conjugated bilirubin is the fraction that reacts directly with reagents whereas unconjugated bilirubin requires the addition of an accelerator compound. They are reported as direct and indirect fractions, respectively.

Pathophysiology

Clinical jaundice usually is not evident until the serum bilirubin concentration rises above 2.5 mg/dL. It is observed in tissues with high albumin concentrations, for example, the skin and eyes. It is absent in albumin-poor fluids, such as tears or saliva. The physiology of bile metabolism may be altered on four principal levels: overproduction of heme products, failure of the hepatocyte to take up the bilirubin for processing, failure of the hepatocyte to conjugate or excrete bilirubin, or an obstruction of biliary excretion into the intestine. Unconjugated bilirubin that is not bound to albumin can cross the blood-brain barrier, causing adverse neurologic effects ranging from subtle developmental abnormalities to encephalopathy and death. The risk of neurotoxicity is increased by conditions

that favor the unbound fraction of unconjugated bilirubin, including hemolysis, hypoalbuminemia, acidemia, and drugs that bind competitively to albumin. Conjugated bilirubins are not neurotoxic, although they may indicate serious disease.

■ DIAGNOSTIC APPROACH

Differential Considerations

The three major diagnostic categories to consider as causes of jaundice are biliary obstructive disorders, liver injury or dysfunction, and hematologic disorders. [Figure 25-1](#) outlines a laboratory-based approach to differentiating among these three categories.

Pivotal Findings

The pivotal findings related to history, physical examination, and ancillary testing are listed in [Figure 25-2](#).

History

Patients may be asymptomatic at presentation or have nonspecific symptoms, such as pruritus, malaise, or nausea. There are a few symptom complexes that, if present, can help narrow the differential diagnosis. Jaundice with abdominal pain suggests significant hepatic inflammation. New-onset painless jaundice is the classic presentation for a neoplasm involving the head of the pancreas. Patients may complain of ill-fitting clothing because of weight loss or increasing abdominal girth related to ascites. The patient or caregiver may note personality changes or confusion, suggestive of hepatic encephalopathy. Unexplained liver failure and jaundice may be the downstream sequelae of an intentional overdose of acetaminophen taken 48 to 72 hours (or more) earlier.

Physical Examination

A thorough examination should be performed in patients presenting with jaundice, because the physical findings can help narrow the differential diagnosis. Pertinent examination findings are summarized in [Figure 25-2](#). Jaundice is first apparent sublingually, on the hard palate, and in the conjunctiva. From there, it spreads caudally. Studies in both adults and neonates suggest that the “level” of cephalocaudal progression of jaundice cannot accurately estimate the serum bilirubin concentration.^{1,2}

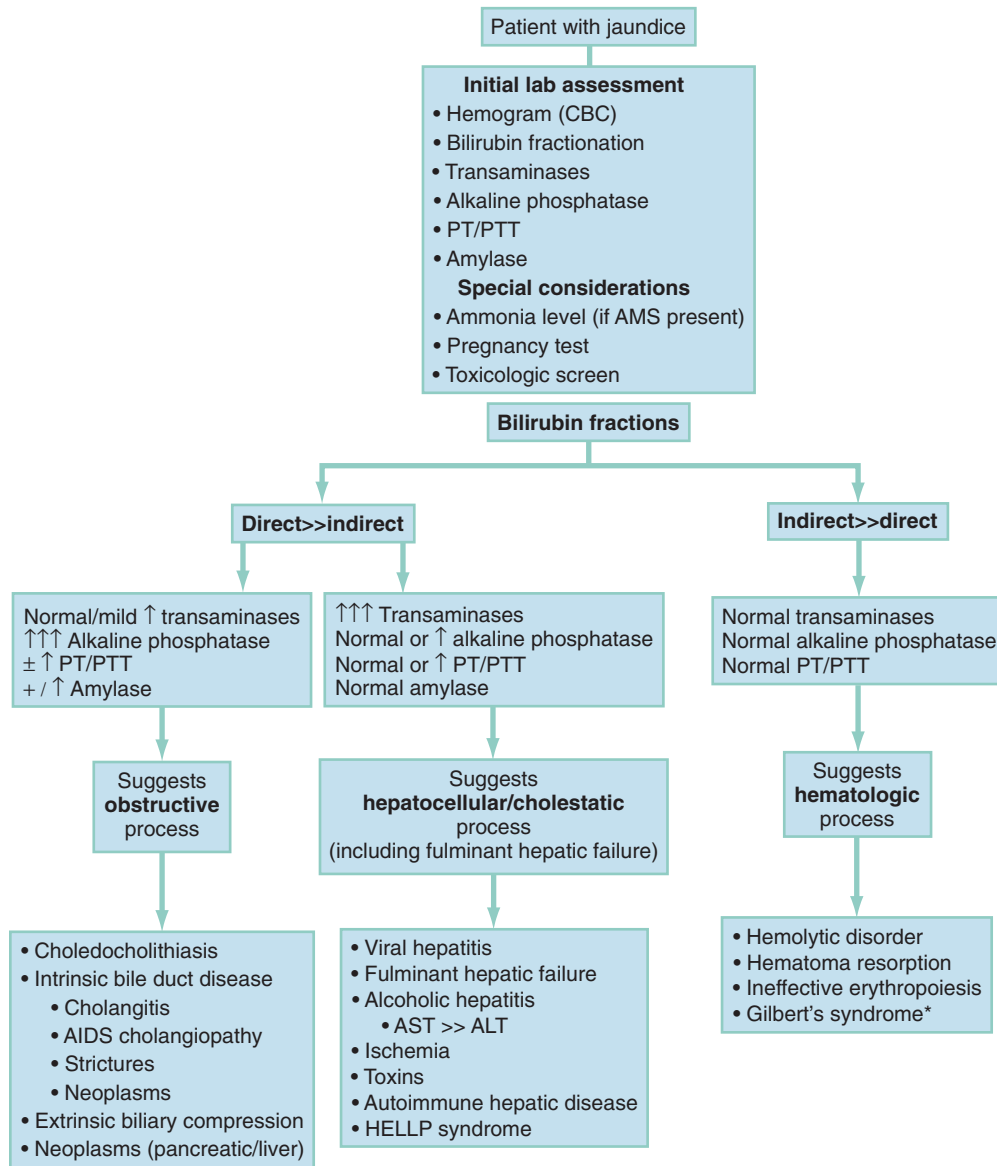


Figure 25-1. Laboratory approach to differential diagnosis of jaundice. AIDS, acquired immunodeficiency syndrome; ALT, alanine aminotransferase; AMS, altered mental status; AST, aspartate aminotransferase; CBC, complete blood count; HELLP, hemolysis, elevated liver enzymes, and low platelets; PT, prothrombin time; PTT, partial thromboplastin time.

*A benign hereditary condition characterized by hyperbilirubinemia and jaundice due to inadequate hepatic conjugation of bilirubin.

Fever with right upper quadrant tenderness suggests cholangitis.³ In this clinical scenario, the liver should not be engorged. A large tender liver may represent an exacerbation of acute or chronic hepatitis or malignant infiltration. A palpable gallbladder, a rare finding, suggests chronic cholestasis or malignancy. The presence of splenomegaly suggests hemolysis, malignancy, or portal hypertension. Ascites may be associated with acute or chronic liver disease. Ascites associated with abdominal tenderness raises suspicion for spontaneous bacterial peritonitis. Rapid onset of hepatomegaly and ascites may indicate portal vein thrombosis (Budd-Chiari syndrome).⁴ Jaundice associated with a large pulsatile abdominal mass may indicate a rapidly enlarging or ruptured abdominal aortic aneurysm. The patient's mental status should be assessed for evidence of hepatic encephalopathy.

Physical examination findings associated with chronic liver disease and cirrhosis include spider angiomas, gynecomastia, testicular atrophy, and caput medusae. Excoriations from scratching in attempts to relieve pruritus suggest chronic liver

disease. Asterix, a sign of hepatic encephalopathy, usually is found only in patients with chronic liver disease. Table 25-1 summarizes the clinical stages of hepatic encephalopathy.

Ancillary Testing

Figure 25-1 lists the laboratory tests that should be considered in the evaluation of the patient with jaundice. Alkaline phosphatase (AP) also can be elevated in diseases affecting bone or the placenta in the first trimester. In the setting of isolated elevated AP, increased serum gamma-glutamyl transpeptidase (GGT) or 5'-nucleotidase points to a hepatic source. A reticulocyte count and evaluation of the peripheral blood smear may identify hemolysis. In the setting of toxic ingestion or unexplained hepatocellular injury, serum acetaminophen concentration level is indicated. Rapid stool guaiac testing should be performed to assess for the presence of gastrointestinal bleeding. Patients with altered mental status should have a rapid bedside glucose assessment in addition to determination of

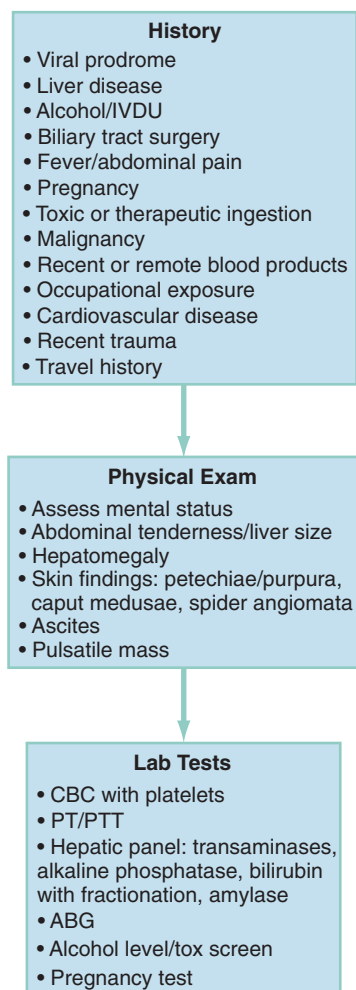


Figure 25-2. Pivotal points in the assessment of the jaundiced patient. ABG, arterial blood gas (analysis); Alk phos, alkaline phosphatase; CBC, complete blood count; IVDU, intravenous drug use; PT, prothrombin time; PTT, partial thromboplastin time.

serum ammonia concentration. Although elevated serum ammonia may aid in diagnosis, the degree of hyperammonemia has not been shown to correlate with the degree of encephalopathy.⁵ In the presence of abdominal tenderness and ascites, ascitic fluid should be tested for cell count, Gram staining, culture, and protein.⁶ Two sets of blood cultures should be obtained in patients with fever and jaundice. If the patient appears ill or there is evidence of gastrointestinal bleeding, type and screen or type and crossmatch studies should be performed.

Imaging

The best radiologic study for the emergent evaluation of obstructive biliary disease remains somewhat controversial. Both ultrasonography (US) and computed tomography (CT) are available in the emergency department (ED), and each has its advantages. The choice of imaging procedure depends on the pretest probability of biliary obstruction and on the index of suspicion of malignancy. In cases in which the probability of malignant obstruction is high, CT is the preferred imaging methodology. It is more sensitive than US in locating the site of the obstruction. Additionally, it is 70% accurate in staging disease and determining resectable versus unresectable disease.⁷ Patients with a high likelihood of benign obstruction

Table 25-1 Clinical Stages of Hepatic Encephalopathy

CLINICAL STAGE	INTELLECTUAL FUNCTION	NEUROMUSCULAR FUNCTION
Subclinical	Normal examination findings, but work or driving may be impaired	Subtle changes in psychometric testing
Stage 1	Impaired attention, irritability, depression, or personality changes	Tremor, incoordination, apraxia
Stage 2	Drowsiness, behavioral changes, poor memory, disturbed sleep	Asterixis, slowed or slurred speech, ataxia
Stage 3	Confusion, disorientation, somnolence, amnesia	Hypoactive reflexes, nystagmus, clonus, muscular rigidity
Stage 4	Stupor and coma	Dilated pupils and decerebrate posturing, oculocephalic reflex

From Fitz G: Hepatic encephalopathy, hepatopulmonary syndromes, hepatorenal syndrome and other complications of liver disease. In Feldman M, Friedman L (eds.): *Gastroenterology and Liver Disease*, 8th ed., Philadelphia, WB Saunders, 2006, p 1966.

are best screened with US. It is safe, rapid, and less expensive and less invasive than CT. Some common duct stones may be missed with US, but it is as sensitive as CT in determining the presence of obstruction. US with Doppler flow can detect obstruction of the hepatic, portal, and splenic veins. Sonographic features that suggest acute cholecystitis include presence of pericholecystic fluid and gallbladder wall thickening.⁸ If gallstones are present on ultrasound images, a sonographic Murphy sign has a positive predictive value of 90% for acute cholecystitis. In patients with low or intermediate clinical likelihood of mechanical obstruction, US is the preferred modality to evaluate whether or not biliary obstruction is present. CT is preferred if the entire abdomen needs to be evaluated.

DIFFERENTIAL DIAGNOSIS

Using a systems approach, jaundice can be classified into critical, emergent, and nonemergent categories (Table 25-2). Patients are considered to be critically ill if they present with jaundice and any of the following: altered level of consciousness, hypotension, fever with abdominal pain, or active bleeding. Any patient with a *new* triad of jaundice, encephalopathy, and coagulopathy is considered to have fulminant hepatic failure.⁹ In general, these patients have no previous history of liver disease and experience sudden onset of illness or toxic exposure that leads to hepatic necrosis. The time course from insult to fulminant hepatic failure ranges from 1 to 8 weeks. Patients with fulminant hepatic failure require aggressive stabilization, consideration for toxic exposures, and admission to an intensive care unit or possible transfer to a center with liver transplantation capabilities.

Table 25-2 Causes of Jaundice Grouped by Level of Urgency

ETIOLOGIC CATEGORY	CRITICAL	EMERGENT	NONEMERGENT
Hepatic	Fulminant hepatic failure Toxin Virus Alcohol Ischemic insult Reye's syndrome	Hepatitis of any etiology with confusion, bleeding, or coagulopathy Wilson's disease* Primary biliary cirrhosis Autoimmune hepatitis Liver transplant rejection Infiltrative liver disease Drug-induced (isoniazid, phenytoin, acetaminophen, ritonavir, halothane, sulfonamides) Toxin ingestion or exposure	Hepatitis with normal mental status, normal vital signs, and no active bleeding
Biliary	Cholangitis	Bile duct obstruction (stone, inflammation, stricture, neoplasm)	
Systemic	Sepsis Heatstroke	Sarcoidosis Amyloidosis Graft-versus-host disease	Post-traumatic hematoma resorption Total parenteral nutrition
Cardiovascular	Obstructing AAA Budd-Chiari syndrome Severe congestive heart failure	Right-sided congestive heart failure Veno-occlusive disease	
Hematologic-oncologic	Transfusion reaction	Hemolytic anemia Massive malignant infiltration Inborn error of metabolism Pancreatic head tumor Metastatic disease	Gilbert's syndrome Physiologic neonatal jaundice
Reproductive	Preclampsia/HELLP syndrome Acute fatty liver of pregnancy	Hyperemesis gravidarum	Cholestasis of pregnancy

*In Wilson's disease, hereditary deficiency of ceruloplasmin causes copper to accumulate in the liver, leading to fulminant hepatic failure.
 AAA, abdominal aortic aneurysm; HELLP, hemolysis, elevated liver enzymes, low platelets.

■ EMPIRICAL MANAGEMENT AND DISPOSITION

Specific therapies depend on the likely clinical entity causing the jaundice (Fig. 25-3). The patient with depressed mental status should have bedside glucose testing. If mental status remains significantly depressed, endotracheal intubation for maintaining airway patency or protection may be required.

Intravenous access should be obtained immediately, and crystalloid infusion may be indicated in the hypotensive patient. A quick assessment of volume status is required because hepatic congestion with jaundice can occur in the setting of congestive heart failure. Owing to the risk of coagulopathy, compressible sites should be used for central venous access. Significant bleeding from any source requires aggressive management. Crystalloid infusion is initiated and continued until blood products become available. Coagulopathy should be corrected with fresh frozen plasma and blood volume replenished with packed red blood cells.

If ascites is present, diagnostic paracentesis should be considered to rule out spontaneous bacterial peritonitis (SBP). This disease can have a subtle presentation and may be missed without a diagnostic paracentesis. The presence of more than 250 polymorphonuclear cells per cm³ of ascitic fluid is diagnostic for SBP. The empiric antibiotic of choice is a third-generation cephalosporin.⁶

Patients with jaundice and elevated transaminase levels out of proportion to the elevation of alkaline phosphatase have a hepatocellular injury pattern. Liver failure with hepatic encephalopathy, if present, can be treated with lactulose, either 60 mg orally or 300 mg by retention enema. Patients with fulminant hepatic failure should be admitted to the intensive care unit or possibly transferred to a liver transplantation center.

Even in the absence of acute liver failure, patients with encephalopathy or unstable vital signs should be hospitalized. On the basis of laboratory data alone, patients with new-onset jaundice should be hospitalized if transaminases are greater than 1000 IU/L, the bilirubin exceeds 10 mg/dL, or there is evidence of coagulopathy. Any of these laboratory abnormalities suggests significant hepatic dysfunction. Patients with hepatitis or cholestatic jaundice may be managed as outpatients if they have a normal mental status and stable vital signs, are able to tolerate oral fluids, have no evidence of acute bleeding, and have no complicating infectious process. Intravenous fluids and antiemetics may be required in the ED. Medications with potential hepatotoxicity, particularly acetaminophen, should be avoided.

If the laboratory evaluation and diagnostic imaging point to an obstructive picture, ascending cholangitis must be ruled out. If it is suspected, blood cultures should be obtained, followed by prompt administration of broad-spectrum antibiotics with coverage for gram-negative aerobes and anaerobes. Patients with this disorder usually require emergent decompression by means of endoscopic retrograde cholangiopancreatography (ERCP) or cholecystostomy, which dramatically improves survival.¹⁰ Some stable patients can undergo a trial of antibiotics and have drainage performed subacutely.¹¹ However, conservative treatment is more likely to fail in patients older than 75 years or chronic smokers.¹²

Patients with extrahepatic obstructive jaundice without cholangitis should be admitted for drainage. ERCP is therapeutic for benign obstructions such as gallstones or strictures. Patients with obstructive jaundice due to malignancy also benefit from biliary decompression, whether operative, endoscopic, or palliative. Once jaundice develops, malignancy is associated with more advanced disease and increased morbidity.

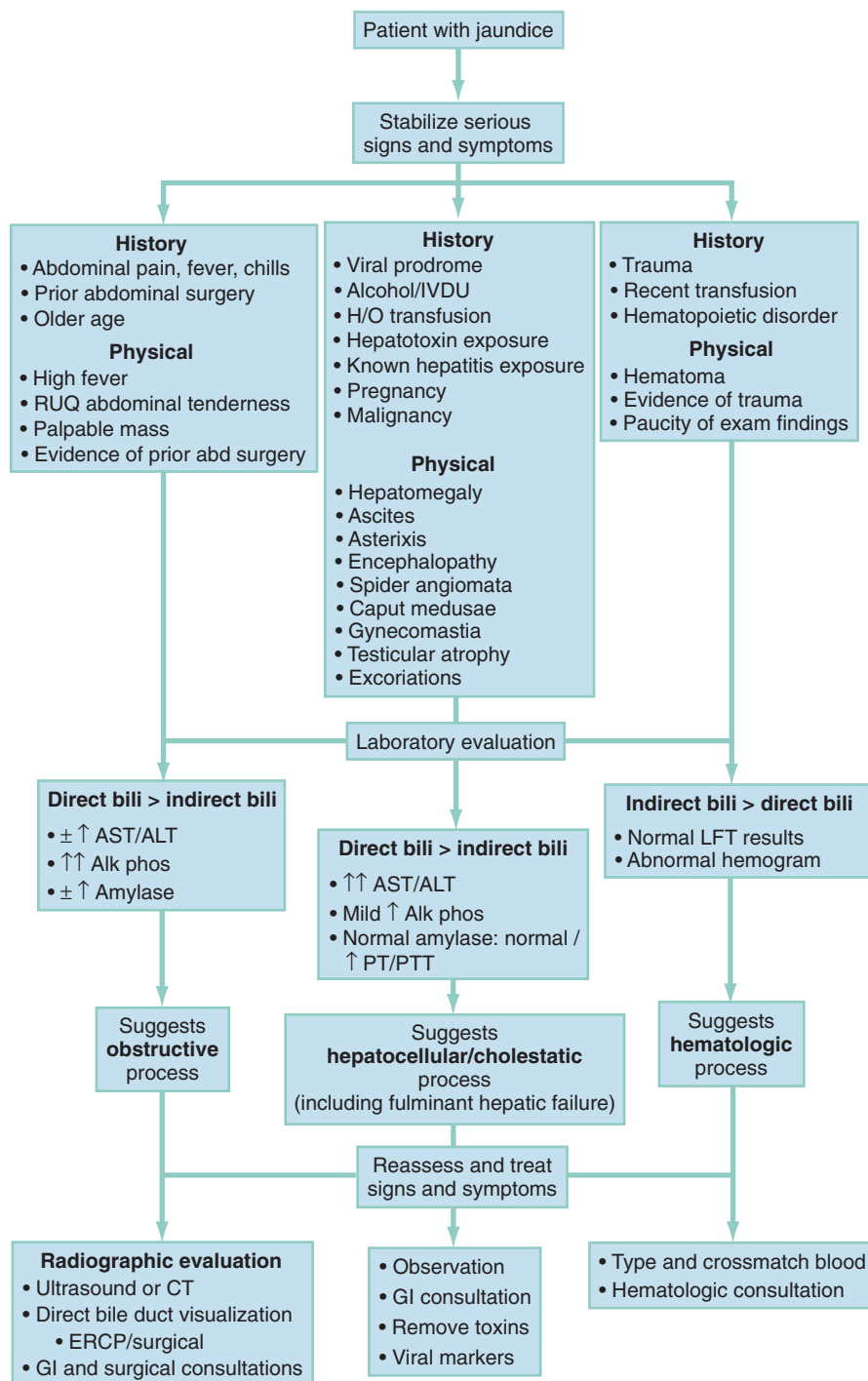


Figure 25-3. Management of the patient with jaundice. Alk phos, alkaline phosphatase; ALT, alanine aminotransferase; AST, aspartate aminotransferase; bili, bilirubin; CT, computed tomography; ERCP, endoscopic retrograde cholangiopancreatography; GI, gastrointestinal; H/O, history of; IVDU, intravenous drug use; LFT, liver function test; PT, prothrombin time; PTT, partial thromboplastin time; RUQ, right upper quadrant.

ity and mortality.^{13,14} Biliary drainage has been correlated with improvements in cardiac function¹⁵ and, not insignificantly, food intake.¹⁶ In patients with obstructive jaundice secondary to malignancy, preoperative drainage is not beneficial in those undergoing surgery.¹⁷ Palliative biliary drainage is recommended for patients who are not surgical candidates. Endoscopic drainage with biliary stenting has been found to result in fewer complications, although the rate of recurrent obstruction is higher¹⁸ than with percutaneous drainage.

In general, patients with uncomplicated cholecystitis should receive intravenous fluids in the ED, parenteral analgesics, and antiemetics as needed and should be hospitalized. For uncomplicated cholecystitis, antibiotic therapy usually is not indicated. Patients with temperature greater than 38.8°C

(102°F), a toxic appearance, or frank sepsis should receive broad-spectrum antibiotic therapy with coverage for enteric pathogens, streptococcal species, and anaerobes. These patients should undergo emergent imaging and consultation with a surgeon or gastroenterologist.⁸

Cholelithiasis, presence of a stone in the common bile duct, may not be as easily visualized by sonography but is suggested by significant obstructive signs and symptoms and dilation of the common bile duct beyond 6 mm. Affected patients require hospitalization for possible ERCP and cholecystectomy.¹⁹⁻²¹

In immune-mediated hemolytic anemia, appropriate cross-matching may be difficult and fatal if not done properly. The decision to transfuse should be based on the achievable level

of oxygenation and the feasibility of instituting alternative treatments. An urgent hematology consultation is recommended. In the case of drug-induced hemolytic anemia, the mainstay of treatment is removal of the offending agent. For patients with glucose-6-phosphate deficiency, blood transfusions are rarely indicated, and the focus of management should be on maintaining urine output to prevent renal failure. Patients with hemoglobinopathies rarely require transfusion therapy unless they present with severe anemia without evidence of reticulocytosis. Fluids, oxygen, and analgesics can be given for an acute crisis.

■ SPECIAL POPULATIONS

One specific presentation that warrants discussion is the pregnant patient who presents with jaundice. Normal physiologic changes in pregnancy have little effect on the liver, so jaundice always indicates serious pathology. Jaundice can occur in pregnancy as a result of any of the conditions discussed earlier, as well as conditions specific to pregnancy, such as hyperemesis gravidarum, acute fatty liver of pregnancy, and intrahepatic cholestasis of pregnancy.

Hyperemesis gravidarum usually manifests in the first trimester and, in severe cases, can be associated with elevated serum bilirubin. The exact mechanism for jaundice is unknown but is likely to be related to malnutrition and impaired excretion of bilirubin. ED treatment is unchanged in these cases: hydration and antiemetics. Patients with hyperemesis and jaundice should be admitted for intravenous hydration.

Intrahepatic cholestasis of pregnancy is an idiopathic cause of jaundice that occurs early in the third trimester. It manifests with pruritus mainly on the trunk, extremities, palms, and soles, followed by jaundice after 1 to 4 weeks. Other features of obstructive jaundice such as acholic stools and dark urine may be present. Laboratory analysis reveals a cholestatic picture. Affected patients are at increased risk for preterm delivery and intrauterine fetal demise and should therefore be managed in conjunction with the obstetric team or transferred to a center capable of caring for premature neonates. Specific treatments include cholestyramine for pruritus and vitamin K.

Acute fatty liver of pregnancy (AFLP) occurs in the third trimester and is characterized by accumulation of microvesicular fat within hepatocytes. It is rare, occurring in 1 in 13,000 deliveries. There is a slight predilection toward primiparous and multiple gestation pregnancies. Clinical manifestations include nausea, vomiting, right upper quadrant or epigastric pain, malaise, anorexia, and jaundice progressing to fulminant hepatic failure and encephalopathy. Treatment consists of prompt delivery. Jaundice and liver dysfunction may progress after delivery but generally resolve. AFLP generally does not recur in subsequent pregnancies. Liver transplantation has been successful for this condition.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.

Ari Lipsky

■ PERSPECTIVE

Women of childbearing age who present with low abdominal pain often have pathologic conditions related to the female reproductive system or bladder, although additional causes must also be considered. Potential etiologic disorders range from the very benign to the immediately life-threatening. Pregnancy presents its series of considerations, and pregnancy status should be determined in all patients.

Epidemiology

Acute pain due to pelvic pathology is common, although the presenting complaint is often abdominal pain or lower abdominal pain; a complaint of low back pain may also signal pelvic pathology. A flare of chronic pelvic pain may manifest as an acute process.

In a survey of reproductive-age adult women, 39% reported that they experience nonmenstrual pelvic pain at least sometimes.¹ Among women who present to an emergency department (ED) and receive a gynecologic diagnosis, 24% of those diagnoses are for pelvic inflammatory disease (PID), 23% for lower genital tract infections (e.g., cervicitis, candidiasis, Bartholin's abscess), 12% for menstrual disorders, 12% for non-inflammatory ovarian and tubal pathology (including cysts and torsion), and 4.3% for ectopic pregnancy.² In the general population, annually 5.8 of every 1000 women present to an ED and receive a diagnosis of PID, and 1.1 of every 1000 women are diagnosed with an ectopic pregnancy.²

Younger patients and those with multiple sexual partners are more likely to have PID, and a previous episode increases the likelihood of a subsequent episode.³ The risk of ectopic pregnancy is higher in women who have had PID, pelvic surgery, or an intrauterine device. Heterotopic pregnancy is of special concern in women undergoing fertility treatment.⁴ Common nongynecologic diseases, such as appendicitis, diverticulitis, urinary tract infection, and urolithiasis, remain important considerations in the woman with acute pelvic pain. [Box 26-1](#) lists conditions accounting for pelvic pain in most women.^{5,6}

Some causes of pelvic pain may lead to serious sequelae. PID carries the short-term risk of tubo-ovarian abscess, and the long-term risks of impaired fertility, chronic pelvic pain, and increased predisposition to ectopic pregnancy.³ Rupture of an ectopic pregnancy or a hemorrhagic ovarian cyst may be acutely life-threatening. Unrecognized abuse may have serious or lethal consequences as well.

Pathophysiology

The female pelvis contains the vagina, uterus, fallopian tubes and ovaries, ureters and urinary bladder, and sigmoid colon and rectum, as well as components of the musculoskeletal system. Although pelvic pain often originates from the reproductive organs, it may arise from any structures that lie adjacent to or course through the pelvis. Visceral pain afferents supplying the pelvic organs have common innervation with the appendix, ureters, and colon. Their significant overlap makes accurate localization difficult for both patient and clinician. Pain may be initiated by inflammation, distention, or ischemia of an organ, or by spillage of blood, pus, or other material into the pelvis. Parietal pain develops when the afferent nerves in the parietal peritoneum adjacent to an affected organ are stimulated.

■ DIAGNOSTIC APPROACH

Differential Considerations

The differential diagnosis of pelvic pain is broad in scope (see [Box 26-1](#)). Most causes of pelvic pain fit into three categories, however: (1) those that originate in the reproductive tract, (2) those that originate in the urinary tract, and (3) those that originate in the intestinal tract. Within the reproductive tract, a subset of causes of pelvic pain is only found in pregnancy; the pregnancy test is therefore a key branch point in the diagnostic process. Potential pregnancy-related disorders can be divided into complications of early pregnancy and complications that occur further along in pregnancy. Although the specific cause of pelvic pain is not always determined at the initial ED visit, an organized approach usually leads to the confirmation or exclusion of disorders most likely to result in significant morbidity.

Pivotal Findings

It is rare that any particular finding on history or physical examination (summarized in [Table 26-1](#)) is reliable enough to conclusively make or exclude a particular diagnosis, so ancillary testing (beyond a simple pregnancy test) is commonly required in the evaluation of patients with acute pelvic pain.

The bimanual examination may at times provide important and convincing information. Unfortunately, however, findings on pelvic examination are somewhat subjective and unreliable,^{7,8} and the test may be more helpful to localize the process to one side or the other, or to help focus the workup of the

BOX 26-1 POTENTIAL CAUSES OF PELVIC PAIN IN WOMEN**Reproductive Tract**

Ovarian torsion
 Ovarian cyst
 Salpingitis/tubo-ovarian abscess
 Septic pelvic thrombophlebitis
 Endometritis
 Endometriosis
 Uterine perforation
 Uterine fibroids
 Dysmenorrhea

Pregnancy-Related**First Trimester**

Ectopic pregnancy
 Threatened abortion
 Nonviable pregnancy
 Ovarian hyperstimulation syndrome

Second and Third Trimesters

Placenta previa
 Placental abruption
 Round ligament pain

Intestinal Tract

Appendicitis
 Diverticulitis
 Ischemic bowel
 Perforated viscus
 Bowel obstruction
 Incarcerated/strangulated hernia
 Inflammatory bowel disease
 Gastroenteritis

Urinary Tract

Pyelonephritis
 Cystitis
 Ureteral stone

pathologic process to the reproductive organs. For instance, tenderness on examination that seems to arise from the right ovary may be appropriately used to guide the subsequent workup, perhaps the ordering of a pelvic ultrasound study. The lack of certainty of the findings on the bimanual examination, however, do not allow the examiner to completely exclude appendicitis, especially if the pelvic ultrasound study fails to identify a clear explanation for the pain.

A sequential approach, as outlined next, allows the clinician to progressively limit the diagnostic possibilities until a sound provisional diagnosis is reached.

Symptoms

The location of *pain* and the *radiation* pattern often are helpful in focusing the differential diagnosis toward a specific cause or group of causes. Lateral pelvic pain usually is related to a process in the tube or ovary. In right-sided pain, appendicitis is considered, and in left-sided pain (especially in patients older than 40 years of age), the differential diagnosis includes diverticulitis and colitis. Urolithiasis may also manifest as lateral pelvic pain, especially when the stone is impacted at the ureterovesicular junction. Central pelvic pain usually is due to processes involving the uterus or bladder, or involving both adnexae. Pain radiating to the rectum may be secondary to pooling of fluid or blood in the cul-de-sac. Diffuse pain may occur with a bilateral process, such as PID, or with diffuse peritonitis secondary to infection or intra-abdominal hemorrhage.

Information regarding the *onset* and *duration* of pain may also be useful. Patients with uncomplicated appendicitis (without perforation or abscess) typically present within 48 hours of symptom onset. Sudden-onset pain suggests acute intrapelvic hemorrhage, cystic rupture, or ovarian torsion. Gradual-onset pain is more consistent with inflammation (such as in PID) or obstruction. Chronic or recurrent pain is consistent with endometriosis, recurrent ovarian cysts, or a persistent ovarian mass. The quality of pain may differentiate the crampy, intermittent pattern of muscular contractions along a hollow viscus (arising from, e.g., uterine, ureteral, or bowel pathology) from the steady, progressive pain associated with inflammatory or neoplastic causes, but this finding is highly variable. Pain associated with PID often manifests at the end of menses. Ovarian cyst pain may fluctuate through several menstrual cycles, finally manifesting as rupture, which often occurs in the middle of the menstrual cycle.

A complaint of fever and chills is more common with an infectious process. Nausea and vomiting occur more frequently when the process originates within the gastrointestinal tract but also may accompany ovarian torsion, ureteral colic, other causes of severe pain, and pregnancy. Dysuria and frequency occur in many local vulvar and vaginal processes, such as herpesvirus infection, candidiasis, and other types of vulvovaginitis, but urgency typically signals an irritated bladder or urethra, focusing attention on the urinary tract.

Information about the patient's last menstrual period, pattern of menses, and sexual activity pattern is useful, although such data cannot be used to rule out pregnancy. Accordingly, a pregnancy test is always indicated except in women who have had a hysterectomy or are clearly postmenopausal. In a pregnant patient, the obstetric history may provide some helpful diagnostic clues. Recurrent spontaneous abortion or previous ectopic pregnancy increases the likelihood of these conditions, respectively. Patients who are actively undergoing infertility treatment are at increased risk for ectopic pregnancy, heterotopic pregnancy, ovarian torsion, and ovarian hyperstimulation syndrome. Round ligament pain usually is noted in the second trimester. Postpartum patients are at increased risk for endometritis.

The presence, quantity, and duration of associated vaginal bleeding should be ascertained. (See also Chapters 27 and 176.) In a nonpregnant patient, bleeding may be associated with PID, trauma, dysfunctional uterine bleeding, or cervical or uterine cancer. In a pregnant patient, bleeding may be associated with a subchorionic hemorrhage in an otherwise viable pregnancy or with an ectopic pregnancy or a nonviable intrauterine pregnancy (which may continue to cause bleeding after expulsion of the uterine contents, especially if any products of conception are retained), or later in pregnancy with placenta previa or abruption. In some cases, the amount of bleeding may be substantial enough to necessitate blood transfusion and surgical intervention.

As part of the past medical history, any recent procedures should be ascertained. All women are interviewed in private to permit disclosure of sensitive information, such as a known pregnancy or recent abortion. The onset of pelvic pain shortly after uterine instrumentation increases the possibility of uterine perforation or infection. Sexual history is important, with an emphasis on recent sexual contact and previous history of sexually transmitted diseases.

Signs

The physical examination is directed toward the abdomen and pelvis. Pelvic examination is performed in virtually all patients, including pregnant patients at less than 20 weeks of gestation.

Table 26-1 Differentiation of Common or Potentially Catastrophic Causes of Pelvic Pain

CAUSATIVE DISORDER/CONDITION	PAIN HISTORY	ASSOCIATED SYMPTOMS	SUPPORTING HISTORY	PREVALENCE IN ED	PHYSICAL EXAMINATION	USEFUL TESTS	ATYPICAL OR ADDITIONAL ASPECTS
Ectopic pregnancy (critical if ruptured)	Classically severe, sharp, lateral pelvic pain, but severity, location, and quality highly variable	Vaginal bleeding	Missed period; history of previous ectopic pregnancy; infertility, tubal ligation, PID, or IUD use	Common	Classically unilateral adnexal tenderness, adnexal mass, and CMT	Pelvic US, quantitative β hCG, T&C progesterone?, laparoscopy	Cannot reliably exclude diagnosis based on history and physical; severe pain, hypotension, or peritonitis suggests rupture.
Ruptured corpus luteum cyst (emergent-critical with significant hemorrhage; otherwise, urgent)	Abrupt moderate to severe lateral pain	Light-headedness if bleeding is severe; rectal pain arises from fluid in cul-de-sac.		Uncommon	Hypotension and tachycardia if blood loss is significant; possible peritonitis	Pelvic US, CBC, T&C	Physical examination findings often do not correlate with volume of blood in pelvis at US.
Ovarian torsion (emergent)	Acute onset of moderate to severe lateral pain	Nausea and vomiting	History of ovarian mass	Uncommon	Adnexal mass and tenderness, possible peritonitis	US with Doppler flow studies, laparoscopy	Torsion can be intermittent.
Appendicitis (emergent)	Duration often <48 hr, generalized followed by localized RLQ	Low-grade fever, nausea, anorexia	Migration of pain to RLQ from center, abdominal pain before vomiting	Common	RLQ tenderness, possible peritonitis	US or CT in unclear cases	Early in course, tenderness may be minimal or poorly localized.
PID/TOA (TOA: emergent; PID: urgent-emergent)	Without TOA, pain usually bilateral. May present acutely within 48 hr, or subacutely with up to 3 wk of pain.	Fever, vaginal discharge	Vaginal discharge, history of PID, unprotected intercourse/multiple partners	PID: common TOA: uncommon	Pus from cervical os, (+) CMT, adnexal tenderness. Peritonitis suggests severe PID or TOA.	CBC, ESR, CRP, pelvic US, laparoscopy, cervical cultures, cervical smear for WBCs	History and physical may be inaccurate for diagnosis, particularly in patients presenting subacutely.
UTI (urgent)	Pain with urination usually is not severe unless patient has flank pain from associated pyelonephritis.	Urinary urgency and frequency; fever and vomiting if patient has associated pyelonephritis	Recent urologic procedure, prior history of UTI	Common	Suprapubic tenderness, flank tenderness, and fever with pyelonephritis	Urinalysis, urine culture	WBC can be present in urine with PID and appendicitis.
Ureteral colic (urgent)	Acute onset, presents within hours. Pain is lateral, usually moderate to severe. Often radiates into the groin.	Nausea and vomiting	Prior history of stones	Common	Patient often appears uncomfortable, but physical examination can be otherwise unremarkable	Urinalysis; hematuria present in ~80% of cases; abdominal CT	If stone is at junction of ureter and bladder, can have localized pain that can mimic appendicitis or other acute pelvic pathology
Nonruptured ovarian cyst/tumor	Lateral ache, gradual onset	Often minimal	Prior history of similar pain	Common	Lateral pelvic tenderness, with or without a mass	Pelvic US, CBC	
Endometriosis	Unilateral or bilateral pelvic pain, often recurrent	Dysmenorrhea, dyspareunia	Prior history of same type of pain in association with menstrual cycle	Common	Unilateral or bilateral adnexal tenderness, occasionally pelvic mass present, peritoneal findings uncommon	Pelvic US, laparoscopy	Symptoms can mimic other types of pelvic pathology; laparoscopy often is needed for confirmation.

CBC, complete blood count; CMT, cervical motion tenderness; CRP, C-reactive protein; CT, computed tomography; ED, emergency department; ESR, erythrocyte sedimentation rate; β hCG, β human chorionic gonadotropin; IUD, intrauterine device; PID, pelvic inflammatory disease; RLQ, right lower quadrant; T&C, type and crossmatch; TOA, tubo-ovarian abscess; US, ultrasonography; UTI, urinary tract infection.

Pregnant patients beyond 20 weeks of gestation with complaints of vaginal bleeding undergo transabdominal pelvic ultrasound study for placental localization before the pelvic examination (see Chapter 27). Timely obstetric consultation should be obtained for patients beyond 20 weeks of gestation.

Abnormal vaginal discharge may be seen in a variety of conditions, including vaginitis, cervicitis, endometritis, and PID more generally, as well as retained foreign body. Cervical motion tenderness most commonly indicates reproductive tract inflammation, but irritation of adjacent structures (e.g., cystitis, appendicitis) also may give rise to this finding. Although an open os is most consistent with an incomplete or inevitable abortion, it does not definitively exclude an ectopic pregnancy. A large uterus in a nonpregnant patient may indicate fibroids. Fundal tenderness often is difficult to distinguish from cystitis but could suggest endometritis or necrotic fibroids. Adnexal masses and tenderness suggest cystic disease, as well as ectopic pregnancy, tubo-ovarian abscess, and torsion, especially if these findings are unilateral.

The constellation of bilateral lower abdominal tenderness, bilateral adnexal tenderness, and cervical motion tenderness is classically associated with PID, particularly when onset of the pain occurs during or just after menstruation, although the diagnosis may (and often should) be made without the presence of all three signs.

Laboratory Tests

A pregnancy test is required in almost all patients. A positive test may indicate intra- or extrauterine pregnancy or, rarely,

molar pregnancy or cancer. Urine dipstick testing of a clean-catch specimen can be used to identify pyuria, typically seen in the setting of urinary tract infection, or hematuria, which is consistent with urolithiasis and also hemorrhagic cystitis. The absence of hematuria does not rule out a ureteral stone, although it lowers the likelihood. Urinalysis should be performed in all pregnant patients, even if their symptomatology does not include urinary tract complaints.

Patients who may be hemorrhaging either internally or externally should have blood drawn for a hemoglobin and hematocrit, as well as for typing and crossmatching.

Patients with a positive pregnancy test should undergo formal ultrasound assessment or bedside ED ultrasound examination to evaluate for ectopic pregnancy.^{9,10} Identification of an intrauterine pregnancy by ultrasound imaging excludes ectopic pregnancy with a high degree of certainty. Heterotopic pregnancy is exceedingly rare in patients who are not undergoing assisted reproduction. Conversely, a patient with a positive pregnancy test result in whom a definite intrauterine pregnancy cannot be seen is presumed to have an ectopic pregnancy until proved otherwise. Furthermore, presence of free intra-abdominal fluid on ultrasound images is consistent with hemorrhage from either an ectopic pregnancy or a ruptured ovarian cyst and must be addressed expediently.

■ DIAGNOSTIC ALGORITHM

The algorithm in Figure 26-1 is designed to help focus further testing and progress to a rational provisional diagnosis. It is not unusual, however, for common diseases to present in uncommon ways or for more than one disease to be present, and tests

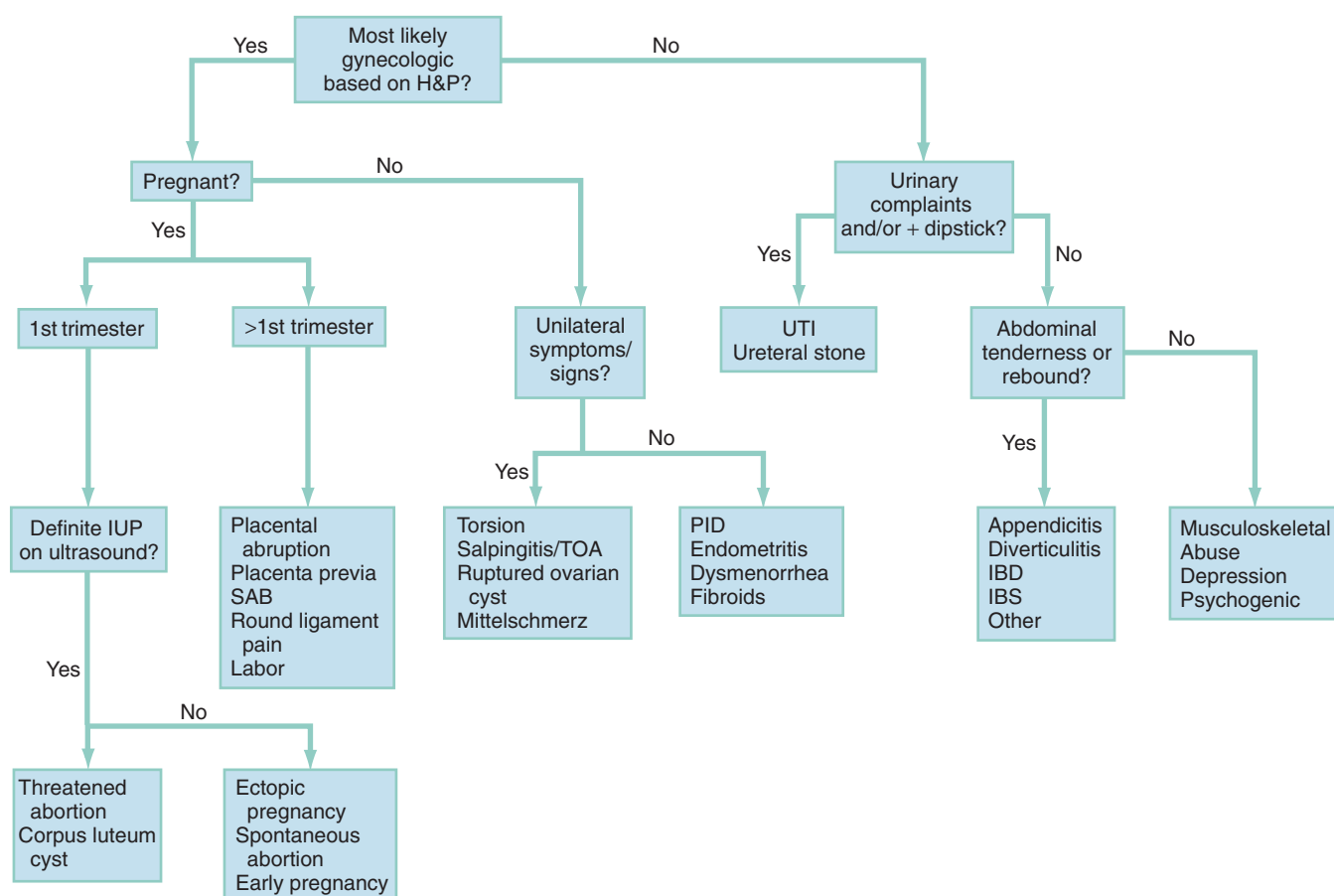


Figure 26-1. Diagnostic algorithm for acute pelvic pain. H&P, history and physical; IBD, inflammatory bowel disease; IBS, irritable bowel syndrome; IUP, intrauterine pregnancy; PID, pelvic inflammatory disease; SAB, spontaneous abortion; TOA, tubo-ovarian abscess; UTI, urinary tract infection.

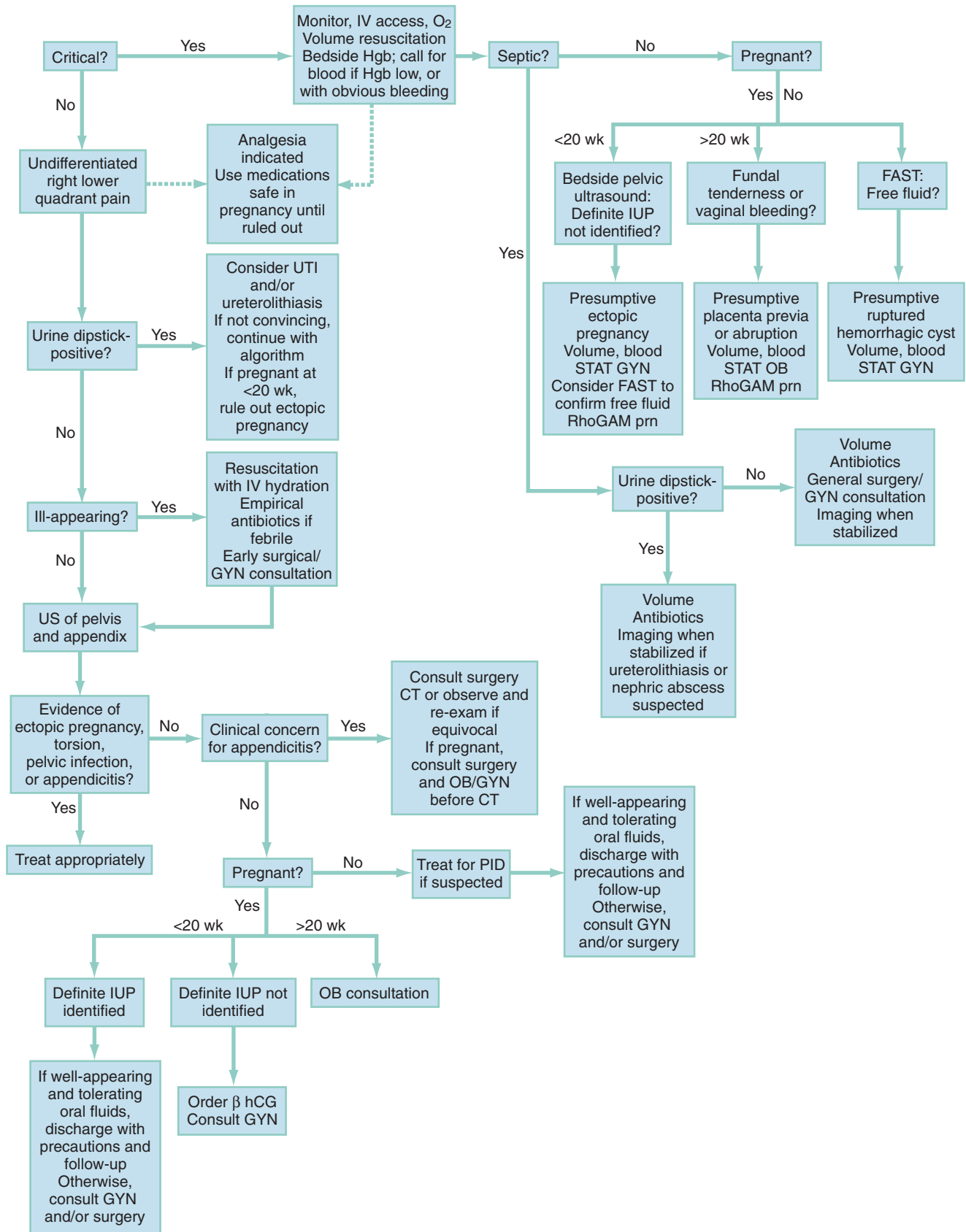


Figure 26-2. Management algorithm for acute pelvic pain: critical patients and right lower quadrant pain presentations. CT, computed tomography; FAST, focused assessment with sonography for trauma; GYN, gynecology; β hCG, β human chorionic gonadotropin; Hgb, hemoglobin; IUP, intrauterine pregnancy; IV, intravenous; OB, obstetrics; PID, pelvic inflammatory disease; US, ultrasound; UTI, urinary tract infection.

must be interpreted carefully in the context of the individual patient's presentation. As examples, patients with a positive result on urine dipstick testing may have appendicitis, and pregnant patients may suffer from ovarian torsion. With certain diseases, such as endometriosis, definitive testing is not available in the ED, and the patient's history may become the most important discriminator.

After an initial history and physical examination, the pregnancy test determines the subsequent priorities. If the patient is in early pregnancy, an ectopic pregnancy is the most emergent diagnosis to consider. Bedside or formal ultrasound assessment may rapidly confirm an intrauterine pregnancy, in which case a threatened abortion is most likely, although unilateral pain may prompt further evaluation for torsion. An empty uterus on ultrasound imaging (or any ultrasound study that cannot confirm a definite intrauterine pregnancy) is consistent with both an ectopic pregnancy and a spontaneous abortion; a very normal pregnancy is also possible. Later in pregnancy, formal ultrasound study often is indicated, and many women whose pregnancies are past 20 weeks' gestation will require observation with monitoring.

Nonpregnant patients with pain that seems to be gynecologic in nature must be assessed for hemorrhage from a ruptured ovarian cyst; for ovarian torsion; and for infection, including cervicitis, endometritis, salpingitis, and tubo-ovarian abscess. Although the history and physical examination often are sufficient to diagnose infection, formal ultrasound assessment usually is required if torsion or tubo-ovarian abscess is suspected. Ultrasound findings also may support a diagnosis of PID if evidence of salpingitis is noted, or of a ruptured cyst if a characteristic ovarian appearance is combined with presence of a small amount of free fluid. Although not as reliable as CT scanning, the ultrasound study also may be used to examine the appendix.

Because in practice it is difficult to differentiate some gynecologic origins of pain from classic intra-abdominal causes (such as right ovarian pathology from appendicitis), the workup often will require an ultrasound study or a CT scan, or both. If the cause appears to be most likely gynecologic, then an ultrasound exam of both the ovary and the appendix is more reasonable, followed by a CT scan if the ultrasound findings are negative and the presentation is possibly consistent with (say) appendicitis. Patients whose pain does not seem to be from the reproductive tract usually are found to have urinary infections or stones, abdominal sources of pain (see Chapter 21), or musculoskeletal pathology, or may be suffering from abuse or depression.

If the available data either do not make sense or conflict with the clinical gestalt, execution of the following three

steps should be considered: (1) Ensure that emergent, life-threatening diagnoses have been addressed (e.g., is a reliable, negative pregnancy test recorded, so that ectopic pregnancy is ruled out?). (2) Move back up the algorithm and reassess whether the presentation may be atypical (e.g., is the examiner confident that appendicitis is not a consideration?). (3) If it seems reasonable that emergent causes are unlikely and sufficient consideration was given to less likely etiologic disorders without uncovering an apparent cause, review the possibility of depression or abuse before disposition. Follow-up planning for all patients is recommended.

■ EMPIRICAL MANAGEMENT

An algorithm for management of patients with acute pelvic pain is presented in [Figure 26-2](#). Patients who are in extremis are most likely to be hemorrhaging, although on occasion their critical condition arises from septic shock. Presentations related to vaginal bleeding are discussed in Chapter 27. Ectopic pregnancy, placental abruption, and hemorrhagic ovarian cyst also may cause life-threatening hemorrhage with no or minimal vaginal bleeding. Patients with these disorders need rapid treatment with fluid and blood products and may require surgical intervention before stabilization can be achieved. A bedside ultrasound assessment by an appropriately trained operator may help the clinician reach the presumptive diagnosis expediently. The obstetric-gynecologic service should be consulted promptly. Septic shock may be a consequence of abdominal or pelvic processes and may require both general surgical and gynecologic consultations, as well as admission to an intensive care setting.

In both critical and noncritical patients, early administration of analgesia is advisable, both for patient comfort and to improve the yield of examinations. Intravenous opioids, such as morphine, are rapid and effective, titratable, and safe in pregnancy. Patients who do not appear ill and for whom a sound provisional diagnosis is reached may be discharged with close follow-up and appropriate precautions. However, pregnant patients who are at more than 20 weeks of gestation should be referred to the obstetrics service for observation. Abdominal trauma in pregnancy, especially in patients who present later in pregnancy, arouses additional concerns not addressed in this chapter.

The author would like to thank Robert Dart, MD, the previous author for this chapter.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.

Vaginal Bleeding

Hilarie Cranmer and Mark Foran

PERSPECTIVE

Vaginal bleeding is one of the most frequent chief complaints of women presenting for emergency care. Normal vaginal bleeding occurs cyclically in women who have achieved menarche, mean age 12.5 years, until menopause, mean age 51 years, in North America. The normal cycle, defined as the first day of bleeding of one cycle to the first day of bleeding of the next cycle, lasts 28 days, plus or minus 7 days, and average volume of blood loss is 60 mL. Vaginal bleeding is defined temporally as midcycle (ovulatory), premenstrual, menstrual, and postmenstrual. Abnormal vaginal bleeding is classified on the basis of the duration, amount, and frequency of bleeding (Table 27-1). It occurs in women of all ages, and it can result from a number of causes, including anatomic abnormalities, complications of pregnancy, malignancies, infections, systemic diseases, and endocrinologic imbalances. Typically, premenarchal or postmenopausal vaginal bleeding is rarely life-threatening, but bleeding as a complication of pregnancy has a significantly increased risk of morbidity and mortality for the mother and fetus.^{1,2}

Epidemiology

Approximately 5% of women aged 30 to 45 years will see a physician for vaginal bleeding. Nonpregnancy causes are classified as ovulatory, anovulatory, and nonuterine. Menorrhagia secondary to anovulation is seen in 10 to 15% of all gynecologic patients. It is common in perimenarchal and perimenopausal women, as well as in patients with endocrine disorders, polycystic ovary syndrome, exogenous hormone use, and liver or renal disease. Nonuterine bleeding must also be considered.³ Approximately 20% of all pregnant patients have vaginal bleeding before the 20th week of gestation; more than 50% of these women spontaneously abort. Vaginal bleeding is reported in 50 to 80% of ectopic pregnancies. Ectopic pregnancy is the most common cause of maternal death in the first trimester of pregnancy, accounting for 9% of pregnancy-related maternal deaths in the United States, and the second leading cause for maternal mortality overall, after postpartum hemorrhage. Teenagers and women of color have the highest risk of death related to ectopic pregnancy. Vaginal bleeding after the 20th week of gestation occurs in approximately 4% of pregnancies; approximately 30% of cases are due to placental abruption (abruptio placentae), and 20% are due to placenta previa. Postpartum hemorrhage accounts for nearly 30% of pregnancy-related maternal deaths. The most common cause of postpartum

hemorrhage in the first 24 hours is uterine atony. After 24 hours, retained products of conception are frequently the etiology.⁴

Pathophysiology

Pregnant Patients

The differential diagnosis of vaginal bleeding in early pregnancy (before the 20th week of gestation) includes ectopic pregnancy; threatened, inevitable, missed, or incomplete abortion; implantation bleeding; cervicitis; cervical conditions such as polyp or ectropion; bleeding from the urinary or gastrointestinal tract; and cervical carcinoma. Risk factors for ectopic pregnancy should increase clinical suspicion but are often absent. These include tubal abnormalities due to past infection or surgical scarring and assisted reproductive techniques. Disruption of the blood supply to the ectopic gestational sac can cause hemorrhage into the fallopian tube, or the size of the developing sac fetus can lead to rupture through the tubal wall.⁵

Spontaneous abortion is the most common complication of pregnancy and is defined as the passing of a pregnancy prior to completion of the 20th gestational week. It implies delivery of all or any part of the products of conception, with or without a fetus weighing less than 500 g. Threatened abortion is bleeding of intrauterine origin occurring before the 20th completed week, with or without uterine contractions, without dilatation of the cervix, and without expulsion of the products of conception. Complete abortion is the expulsion of all of the products of conception before the 20th completed week of gestation, whereas incomplete abortion is the expulsion of some, but not all, of the products of conception. Inevitable abortion refers to bleeding of intrauterine origin before the 20th completed week, with dilatation of the cervix without expulsion of the products of conception. In missed abortion, the embryo or fetus dies, but the products of conception are retained in utero. In septic abortion, infection of the uterus and sometimes surrounding structures occurs.⁶

Placental abruption can occur spontaneously or secondary to abdominal trauma with transmission of forces to the uterus. An increased incidence is seen in association with cocaine use, hypertension, preeclampsia, HELLP (hemolysis, elevated liver enzymes, and low platelets) syndrome, smoking, increased maternal age, and abnormal implantation of the placenta (e.g., placenta previa, accreta, increta, or percreta). Placenta previa

occurs when the implanted placenta overlays the cervical os. Bleeding is due to partial separation of the placenta from the uterine wall. Uterine atony occurs when myometrial dysfunction prevents the uterine corpus from contracting, allowing continued bleeding at the placental site. Atony is more likely to occur with conditions that overdistend the uterus, such as polyhydramnios, multiparity, prolonged labor, induced labor, high pitocin usage during labor, precipitous labor, magnesium therapy, or intrauterine infection (chorioamnionitis).⁷

Nonpregnant Patients

The pathophysiology of nonpregnant vaginal bleeding varies with age group. Children may present with foreign bodies, genital trauma, or severe vulvovaginitis causing mucosal breakdown and hemorrhage. Sexual abuse must always be considered. In adolescent girls and women, anovulatory uterine bleeding occurs when estrogen stimulates endometrium proliferation without the stabilizing effect of progesterone, causing

spontaneous sloughing of the endometrium. Submucosal leiomyomas cause hemorrhage by disrupting the endometrial vascular supply and the ability of the uterus to contract to stop bleeding. Cervical and endometrial polyps have vascular pedicles and are prone to bleed.

■ DIAGNOSTIC APPROACH

Differential Considerations

The differential diagnosis can be categorized by age of presentation and frequency of cause (Table 27-2). Primary coagulation disorders account for almost 20% of acute menorrhagia in adolescents. Von Willebrand's disease is the most common; however, myeloproliferative disorders and immune thrombocytopenia are also possibilities.⁸ After immediate resuscitation and stabilization of unstable patients, pregnancy status is determined. Patients presenting with hemodynamic instability require intravenous access, fluid resuscitation, stabilization with blood components, and consultation with obstetrics/gynecology (or, less often, surgery). Concurrently, steps must be taken to prevent further vaginal bleeding. In hemodynamically unstable patients, surgical intervention is often necessary to control bleeding effectively. Ectopic pregnancy should be considered in all women of childbearing age who present with abdominal or pelvic complaints or with unexplained signs or symptoms of hypovolemia.

Nonuterine causes of vaginal bleeding must be included in the differential diagnosis, systematically addressed during the history taking and physical examination, and pursued with relevant investigations and consultations, if indicated. Potential sources of nonuterine bleeding include the cervix, vagina, lower urinary tract, and lower gastrointestinal tract. Cervical causes include carcinoma, polyps, condylomata, eversion of squamocolumnar junction associated with oral contraceptive use or pregnancy, trauma, and some infections. Vaginal sources of bleeding include carcinoma, sarcoma, adenosis, lacerations, infections, and retained foreign bodies. Lower urinary tract lesions, such as urethral faruncles and infected urethral diverticula, may also mimic vaginal bleeding.

Pivotal Findings (Symptoms, Signs, and Laboratory)

Symptoms

The volume, duration, and timing of bleeding should be ascertained. The average tampon or pad absorbs 20 to 30 mL of

Table 27-1 Definitions of Vaginal Bleeding

Polymenorrhea	Abnormally shortened cycle, with bleeding occurring every 21 days or sooner
Oligomenorrhea	A cycle duration of 35 days or longer
Menorrhagia	Cycle occurs at regular intervals but lasts for more than 7 days and involves the loss of more than 80 mL of blood
Hypomenorrhea	Cycle occurs at regular intervals but has a decrease in monthly blood loss
Intermenstrual bleeding	Bleeding that occurs between regular periods
Metrorrhagia	Bleeding that is frequent and irregular
Menometrorrhagia	When metrorrhagia becomes prolonged
Dysfunctional uterine bleeding	Abnormal vaginal bleeding due to anovulation
Postcoital bleeding	Bleeding after sexual intercourse, suggesting cervical pathology
Postmenopausal bleeding	Any bleeding that occurs more than 6 months after the cessation of menstruation

Table 27-2 Causes of Vaginal Bleeding by Age in Descending Order of Frequency

	PREPUBERTAL	ADOLESCENT	REPRODUCTIVE	PERIMENOPAUSAL	POSTMENOPAUSAL
Most common	Vaginitis	Anovulation	Pregnancy	Anovulation	Endometrial lesions, including cancer (30%)
	Anovulation	Pregnancy	Anovulation	Uterine leiomyomas	Exogenous hormone use (30%)
	Genital trauma or foreign bodies	Exogenous hormone use	Exogenous hormone use	Cervical and endometrial polyps	Atrophic vaginitis (30%)
		Coagulopathy (von Willebrand's disease)	Uterine leiomyomas	Thyroid dysfunction	Other tumor: vulvar, vaginal, cervical (10%)
Least common			Cervical and endometrial polyps		
			Thyroid dysfunction		

vaginal effluent, although the number of pads or tampons used is unreliable because personal habits vary greatly among women. Amenorrhea may not indicate pregnancy, and bleeding during approximately the time of the last expected period does not exclude pregnancy. Bleeding during or after intercourse may indicate a cervical lesion and is more common in pregnancy because of increased blood flow to the cervix. Abdominal pain may indicate critical, emergent, or noncritical causes, depending on the severity of pain, bleeding, and hemodynamic state. During active labor, a history of previous cesarean section, cocaine abuse, or high doses of oxytocin or prostaglandins should raise the suspicion of uterine rupture. A history of trauma should be considered in an adolescent with bleeding, and sexual assault should be considered in an adult in whom abuse is present. In the pregnant patient, there is significant increased risk of maternal and fetal morbidity and mortality after blunt trauma, such as motor vehicle accident, interpersonal violence, or falls. Associated symptoms of nausea, breast tenderness, urinary frequency, and fatigue may indicate that the patient is pregnant. In the absence of pregnancy, vaginal discharge, pelvic pain, and fever may suggest pelvic inflammatory disease. Pelvic inflammatory disease is very rare during pregnancy.

Signs

A thorough evaluation includes recording and interpreting vital signs, abdominal and pelvic examinations, and, in the pregnant patient of sufficient gestational age, fetal heart tones and fundal height. Vaginal bleeding associated with hemodynamic shock alerts the clinician to ruptured ectopic pregnancy. Fetal heart tones that are diminished to less than 100 or that are absent in a gravid female may indicate fetal distress. Pelvic examination may reveal the source of bleeding; however, after the 20th week of gestation, ultrasound should precede pelvic examination to avoid disruption of a possible placenta previa. Bedside transabdominal ultrasound imaging may reveal free intraperitoneal fluid in an unstable patient, which should lead to immediate gynecologic or surgical evaluation.

Uterine size, measured from the symphysis pubis to the fundus, is the quickest means of roughly estimating gestational age. This distance in centimeters equals the gestational age in weeks (e.g., 24 cm = 24 weeks), which allows some early indication of fetal viability if delivery is necessary. Usually, 24 or 25 weeks is used as the cutoff point for fetal viability. As a rough guide, the fetus is potentially viable when the dome of the uterus extends beyond the umbilicus. Fetal heart tones can be detected by auscultation at 20 weeks of gestation or by Doppler probe at 10 to 14 weeks. If either the uterus is less than 24 cm in size or fetal heart tones are absent, the pregnancy is probably too early to be viable, and treatment is directed solely at the mother.

Ancillary Testing

In hemodynamically compromised patients, blood is obtained for hematocrit, platelet count, prothrombin time, partial thromboplastin time, ABO and Rh typing, and cross-matching of blood. Ultrasound is the imaging modality of choice for simultaneous assessment of the mother and the fetus. In the pregnant trauma patient, it is useful in the detection of major abdominal injury (sensitivity 80%, specificity 100%) and for establishing fetal well-being or demise, gestational age, and placental location.⁹ Computed tomography and magnetic resonance imaging are rarely indicated in the evaluation of vaginal bleeding, except in the case of pregnant trauma patients to

diagnose potentially life-threatening injuries in those patients not proceeding directly to surgical intervention.

Qualitative pregnancy tests in clinical use are typically reported as positive when the β -hCG concentration is 20 mIU/mL or higher in urine and 10 mIU/mL or higher in serum. At this level of detection, the false-negative rate for detection of pregnancy will not be more than 1% for urine and 0.5% or less for serum. In clinical use, the performance of urine qualitative testing has been found to be 95 to 100% sensitive and specific compared with serum testing. When a bedside urine test is negative and ectopic pregnancy is still being considered, a quantitative serum test should be performed. The sensitivity of quantitative serum testing for the diagnosis of pregnancy is virtually 100% when an assay capable of detecting 5 mIU/mL or more of β -hCG is used.¹⁰ The discriminatory level of serum β -hCG for ectopic pregnancy is 1500 to 2000 mIU/mL.¹¹ Below this level, with no evidence of an intrauterine pregnancy (IUP) on transvaginal ultrasound, ectopic pregnancy as well as normal IUP are still possible. Above this level, ectopic pregnancy is diagnosed by the absence of an IUP on transvaginal ultrasound. In stable patients with minimal symptoms who are below the discriminatory level, serial quantitative β -hCG levels every 48 hours may distinguish ectopic pregnancy from IUP and spontaneous abortion in pregnancies less than 5 to 7 weeks of gestation. A system for close follow-up with gynecology is essential to an outpatient strategy for such patients. Additional testing such as progesterone level may help to distinguish normal versus abnormal pregnancy. A progesterone level of less than or equal to 5 ng/mL indicates a nonviable pregnancy, ectopic pregnancy, or IUP and excludes normal pregnancy with 100% sensitivity (Figs. 27-1 and 27-2).¹²

EMPIRICAL MANAGEMENT

All patients who present in shock with a surgical abdomen or evidence of intra-abdominal free fluid should be resuscitated and promptly evaluated with immediate consideration of operative intervention in consultation with obstetrics/gynecology and surgery.

Pregnant Patients

If ectopic pregnancy is suspected and the serum or urine β -hCG is positive, and the patient is hemodynamically unstable, immediate surgical consultation is indicated. If bleeding presents with shock after the 20th week of pregnancy, stabilization is performed while obtaining a transabdominal ultrasound to evaluate the placenta (location in placenta previa and separation and hemorrhage in placenta abruptio). In the presence of vaginal bleeding in these patients, bimanual or speculum vaginal examination or transvaginal ultrasound should not be undertaken until placenta previa is excluded. High-grade third-trimester bleeding should prompt immediate obstetric consultation, even before diagnostic studies elucidate the possible cause. Vaginal delivery is the preferred management of third-trimester vaginal bleeding in the absence of placenta previa, but cesarean section is indicated if (1) fetal distress is present and vaginal delivery is not imminent, (2) there is severe abruptio with a viable fetus, (3) life-threatening hemorrhage exists, or (4) the patient has failed a trial of labor.

Uterine rupture may present with excessive vaginal bleeding, uterine pain, and a change in abdominal contour. A soft horizontal lump often appears below a hard fundus, representing expanding hematoma and a retracting uterus, respectively. Emergent surgical delivery is indicated.

Urgent cesarean section is performed if excessive vaginal bleeding accompanies the rupture of membranes and the fetus

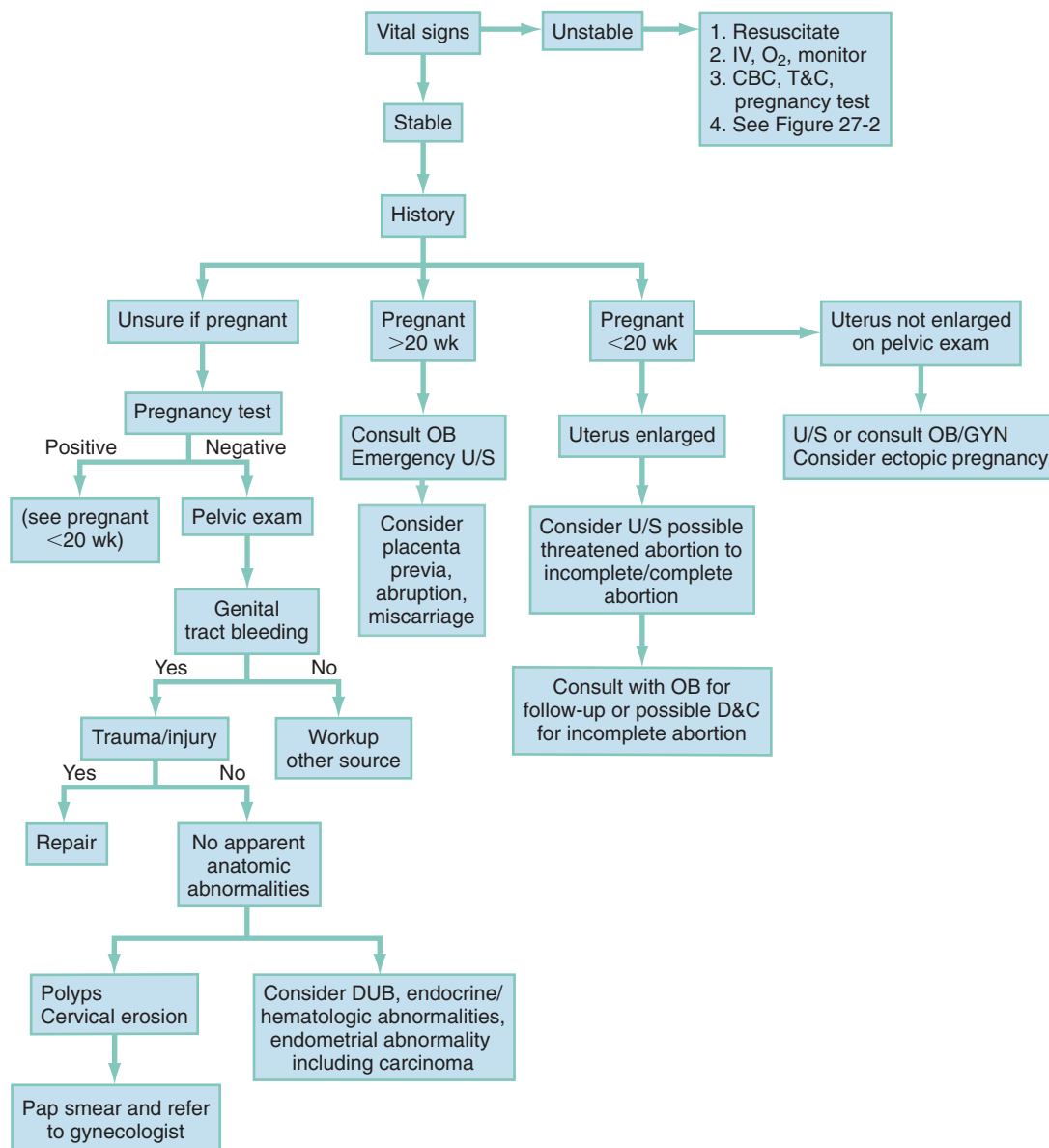


Figure 27-1. Diagnostic approach to patient with vaginal bleeding.

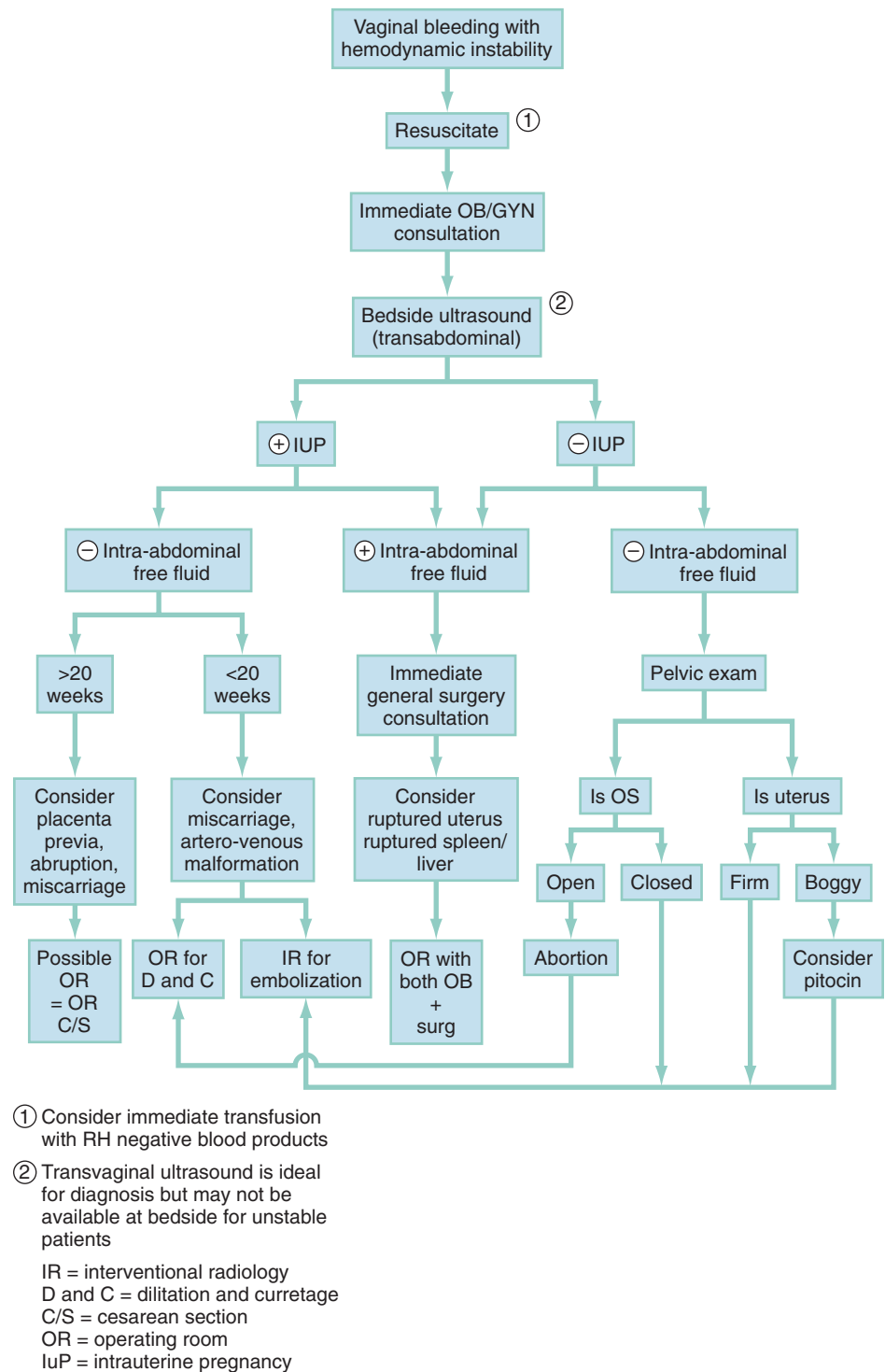
shows signs of distress. Painless vaginal bleeding with rupture of membranes classically suggests vasa previa; it indicates fetal bleeding and requires emergent cesarean section. If after delivery of the fetus the placenta adheres abnormally and has difficulty separating, placenta accreta is likely present and may require urgent hysterectomy to prevent life-threatening hemorrhage. If available, interventional radiology for thromboembolization may be considered. Firm bimanual compression of the uterus or insertion and inflation of a Foley catheter with a 30-mL balloon may limit hemorrhage until surgery is arranged. Uterine atony often responds to vigorous uterine massage and intravenous oxytocin.¹³

Evidence for the administration of anti-D immunoglobulin (Rhogam) for the prevention of Rh seroconversion in pregnant women is limited. Nevertheless, it is recommended to administer anti-D immunoglobulin to Rh-negative women in all cases of documented first-trimester loss of established pregnancy, including threatened abortion, incomplete abortion, and ectopic pregnancy. One may consider administration of anti-D immunoglobulin in cases of minor trauma in Rh-negative pregnant women.¹⁴

Nonpregnant Patients

In nonpregnant patients, heavy vaginal bleeding may be under ovulatory control or related to anovulatory dysfunctional uterine bleeding. Nonsteroidal anti-inflammatory drugs are the mainstay of treatment for both conditions, although the exact mechanism of action is not clearly understood.¹⁵ In nonpregnant hemodynamically unstable patients, consider administering IV conjugated estrogen (Premarin) 25 mg and repeat doses if necessary until bleeding stops, usually within 1 to 5 hours. If bleeding continues after IV estrogen, insert a pediatric Foley catheter into the cervical os and inflate to tamponade the bleeding. Distend the balloon with saline until the bleeding stops. A larger balloon may be needed and this can be left in place for 12 to 24 hours.¹⁶ Hemodynamically stable patients can be referred for outpatient ultrasound and/or endometrial biopsy. All patients with abnormal uterine bleeding should receive close follow-up from a primary care physician or gynecologist. Outpatient treatment with oral contraceptives can arrest bleeding. Patients older than 35 years or with risk factors for endometrial cancer should have an endometrial

Figure 27-2. Diagnostic approach to unstable patient with vaginal bleeding.



biopsy within one week of starting hormonal manipulation. A baseline hemoglobin/hematocrit is recommended. Finally, other medical causes, such as hypothyroidism, hemostasis disorders, or anticoagulant therapy, must be considered and appropriate outpatient consultation obtained.

DISPOSITION

In a patient with postpartum uterine atony or coagulopathy, medical management is often sufficient. Obstetrics consultation is rarely indicated. In a preadolescent patient, abuse must be ruled out before the patient is discharged to her current

environment. In a nonpregnant stable patient, malignancy always should be suspected, and additional inpatient or timely outpatient gynecologic workup is indicated. Laboratory studies such as thyroid function and prolactin levels may be helpful to the consultant or in the initial outpatient workup of dysfunctional uterine bleeding, but they are not required in the emergency department setting.¹⁷

The references for this chapter can be found online by accessing the accompanying Expert Consult website.

PERSPECTIVE

Back pain is a common symptom causing patients to seek care in the emergency department (ED). It accounts for 2.3% of all physician visits,¹ and 84% of the adult population have experienced low back pain in their lifetime.² Forty-nine percent have suffered low back pain in the last 6 months² and 26% in the last 3 months.¹ Total costs of low back pain in the United States exceed \$100 billion per year.³ Although mechanical or nonspecific low back is the most common cause, the differential diagnosis includes several life-threatening and disabling conditions (see Box 28-2). Developing a systematic approach that screens all the potential causes of back pain is the key to accurate clinical decision-making.

Epidemiology

Ninety-seven percent of patients presenting to a physician for evaluation of acute back pain, defined as lasting less than 6 weeks, are finally diagnosed with mechanical or nonspecific low back pain.⁴ Most will recover but many will have a recurrence within a year.⁵ For those with chronic back pain, defined as lasting longer than 3 months, persistence or a recurrence within 12 months is very common.⁵ About 1% of all patients with back pain have true sciatica.⁵ Back pain is the second most common cause of lost time in the workplace and an enormous source of health care expenditures and lost productivity.^{6,7}

Before considering these common mechanical causes, several emergent diagnoses must be excluded, including aortic dissection, abdominal aortic aneurysm, cauda equina syndrome, epidural abscess, osteomyelitis, and tumor. The presence of “red flags” (Box 28-1) should prompt a more thorough examination of these possibilities. Visceral causes constitute about 2% of the diagnoses in patients presenting with back pain.⁴ Aortic dissection is a rare but catastrophic event, with mortality rates exceeding 90% if it is not diagnosed. Cauda equina syndrome (bilateral leg pain and weakness, urinary retention with overflow incontinence, fecal incontinence or decreased rectal tone, and “saddle anesthesia”) is a rare but disabling complication usually due to a large central herniated disk, and less often to tumor or infection. Epidural abscess and vertebral osteomyelitis comprise 0.01% of the diagnoses in patients complaining primarily of back pain.⁴ Spinal carcinoma is uncommon (0.7%) in the general population presenting

with back pain.⁴ Of cancer patients, 80% who present with back pain have spinal metastases. Metastasis to bone is seen commonly in breast, lung, prostate, kidney, and thyroid carcinomas. Inflammatory arthritis is the diagnosis for 0.3% of patients presenting with back pain.⁴

Pathophysiology

The pathophysiology of back pain is diverse. Sources of pain include vascular, visceral, infectious, mechanical, and rheumatologic causes. Pain may originate in the spinal column, cord or root, or musculature or may be referred from thoracic or abdominal organs.

The gelatinous nucleus pulposus is surrounded by the tough anulus fibrosus. The anulus thins posteriorly, creating the opportunity for the nucleus pulposus to herniate. This varies from bulging, to protrusion, to extrusion, to sequestration. Ninety-five percent of herniations occur at the L4-5 and L5-S1 disk spaces, causing radicular pain in the L5 and S1 dermatomes.⁴ Sciatica radiates below the knees, causing focal motor and sensory loss. It worsens with bending, sitting, coughing, sneezing, and straining. Involvement of the L5 nerve root presents with decreased sensation in the first web space, weakness with extension of the great toe, and normal reflexes. An S1 radiculopathy is characterized by diminished sensation of the lateral small toe, impaired plantar flexion, and a decreased or absent ankle jerk. Disk bulging (52–81%) and annular tears with focal disk protrusion (32–67%) are commonly found in asymptomatic patients, whereas disk extrusion (0–18%) is not.⁸⁻¹¹ Serial magnetic resonance imaging (MRI) studies show that two thirds of herniated disks regress or resolve over 6 months.⁴ The fact that so many patients with herniated disk improve over time, and the high incidence of findings in asymptomatic patients argues against early MRI or computed tomography (CT). This natural resolution of symptoms in disk disease is in contrast to spinal stenosis, which tends to remain the same or worsen over time.⁴

The spinal cord ends at L1 in the adult where it gives rise to the cauda equina. Compressive lesions above the cauda equina cause upper motor neurologic signs. Compression of the cauda equina leads to lower motor neurologic findings. The ligamentum flavum can thicken with age and along with degenerative changes contributes to spinal stenosis.

DIAGNOSTIC APPROACH

Differential Considerations

The emergency physician must first rule out life-threatening and disabling causes of back pain, including thoracic aortic dissection, ruptured abdominal aortic aneurysm, epidural

*I gratefully acknowledge the work of Kevin G. Rodgers and James B. Jones on this chapter in the last edition and who figure so prominently in this new edition.

BOX 28-1**COMMON HISTORICAL AND PHYSICAL EXAMINATION
“RED FLAGS”****Historical Information**

Recent significant trauma
 Recent mild trauma in patients older than 50 years
 History of prolonged steroid use
 History of osteoporosis
 Patients older than 70 years
 Syncope
 Acute onset of back, flank, or testicular pain
 Diaphoresis or nausea associated with pain
 History of cancer
 Low back pain worse at rest or night pain
 Unexplained weight loss
 Recent bacterial infection
 Unexplained fever > 38°C (>100°F)
 Intravenous drug use
 Immunocompromised status

Physical Examination

Abnormal vital signs—hypotension, tachycardia, fever
 Unequal blood pressure readings in the upper extremities
 Pulse deficit or circulatory compromise of the lower extremities
 Pulsatile abdominal mass
 Loss of rectal sphincter tone, urinary retention, or focal lower extremity weakness
 Focal back pain with fever

abscess or compressive mass, spinal column injury with cord or root compression, and cauda equina syndrome. An accurate history and physical examination guides the investigation of possible more serious underlying pathologic process (Boxes 28-1 and 28-2).^{4,12} Laboratory and imaging are needed in some cases, but it is usually possible to rule out significant pathology without recourse to extensive testing.

Rapid Assessment and Stabilization

If the initial history and physical examination identify any suggestion of serious disease, rapid stabilization measures should ensue consistent with the cause of concern (Fig. 28-1). Management of aortic dissection, ruptured abdominal aortic aneurysm, and spinal cord and column injuries are covered in other chapters. If epidural abscess or cauda equina syndrome is suggested, emergent MRI and neurosurgical consultation then should be obtained based on the results of the scan. For epidural abscess, blood cultures are obtained followed by intravenous (IV) administration of antibiotics against *Staphylococcus aureus*. For cauda equina syndrome, an urgent neurosurgical consultation is required. Although the evidence supporting steroid use is conflicting, dexamethasone is commonly used with the hope of decreasing compression from inflammation or to shrink tumor mass. For all patients with significant pain, including patients with “benign” causes for back pain, effective analgesia should be provided early in the evaluation.

Pivotal Findings**History**

History of Present Illness. The history helps to localize pain to the most likely structure and mechanism. The following questions are useful in differentiating between mechanical and nonmechanical causes and will help guide appropriate management.

BOX 28-2**DIFFERENTIAL CONSIDERATIONS IN ACUTE LOW
BACK PAIN****Emergent**

Aortic dissection
 Cauda equina syndrome
 Epidural abscess or hematoma
 Meningitis
 Ruptured/expanding aortic aneurysm
 Spinal fracture or subluxation with cord or root impingement

Urgent

Back pain with neurologic deficits
 Disk herniation causing neurologic compromise
 Malignancy
 Sciatica with motor nerve root compression
 Spinal fractures without cord impingement
 Spinal stenosis
 Transverse myelitis
 Vertebral osteomyelitis

Common or Stable

Acute ligamentous injury
 Acute muscle strain
 Ankylosing spondylitis
 Degenerative joint disease
 Intervertebral disk disease without impingement
 Pathologic fracture without impingement
 Seropositive arthritis
 Spondylolisthesis

Referred or Visceral

Cholecystitis
 Esophageal disease
 Nephrolithiasis
 Ovarian torsion, mass, or tumor
 Pancreatitis
 Peptic ulcer disease
 Pleural effusion
 Pneumonia
 Pulmonary embolism
 Pyelonephritis
 Retroperitoneal hemorrhage or mass

Where is the pain? The patient is asked to point with one finger to the one spot where it hurts the most. Does the pain radiate to the legs and, if so, specifically where in the legs? Does the pain conform to a specific dermatomal area? Radicular pain, particularly extending below the knee in a dermatomal distribution, implies nerve root involvement. Pain mainly in the paraspinal musculature without dermatomal radiculopathy implies nonspecific low back pain. Any associated chest or abdominal pain may indicate a possible visceral cause. Flank location implies a renal origin, and a higher location can be from the chest or pleura.

When did the pain start? The patient should describe in detail what he or she was doing when the pain started. Has there been a recent change in type or intensity of physical activity? Is there any past history of back pain, and what therapeutic modalities were used to treat it? If there is a history of back pain, is there any difference between present and past pain? Acute onset associated with a specific task suggests a mechanical cause. Sudden-onset, severe back pain suggests aortic dissection. Slow onset or onset unrelated to activity suggests a nonmechanical cause (e.g., tumor). Non-

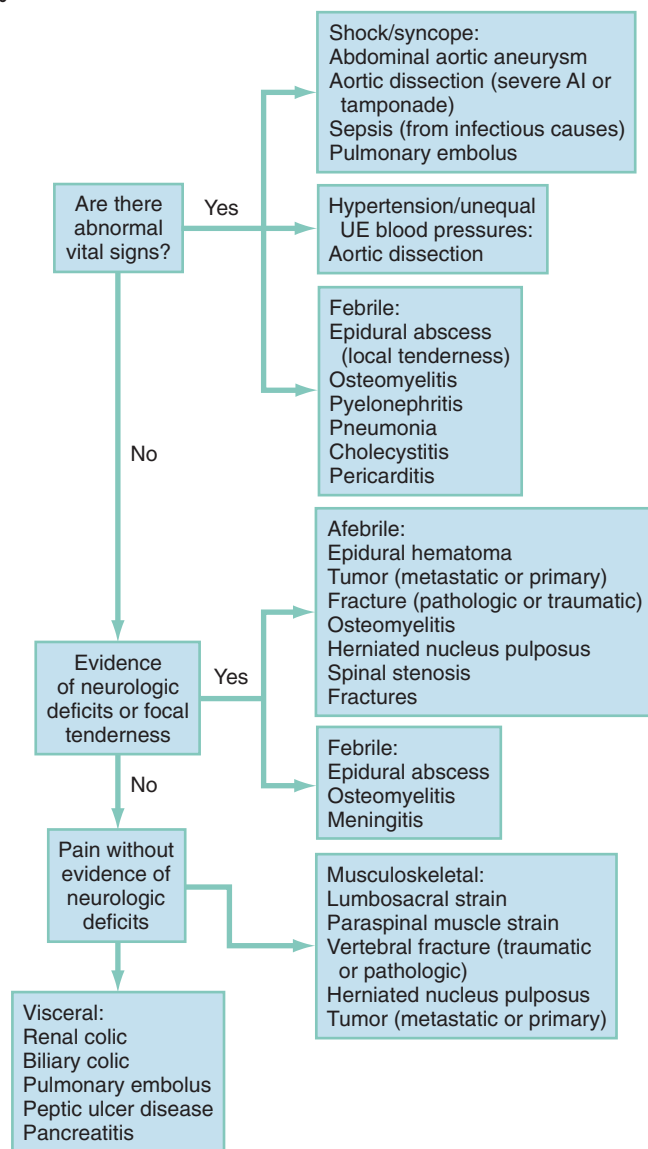


Figure 28-1. Rapid assessment of acute low back pain. AI, aortic insufficiency; UE, upper extremity.

mechanical pain may improve then recur, but the trend is progressive worsening.

Are there any aggravating or alleviating factors? Cough or Valsalva maneuver that aggravates the pain in general favors a mechanical cause and may point specifically to a herniated disk. Patients with back pain associated with tumors and infectious causes often present with nighttime pain and persistent pain unrelieved by rest and analgesics. Spinal stenosis presents with diffuse back pain, numbness, and tingling in one or both legs (pseudoclaudication). Symptoms are aggravated by ambulation (especially “downhill”) and relieved with spinal flexion, which increases spinal canal diameter, temporarily relieving the stenosis. Direct trauma may suggest contusion, strain or fracture, while deceleration may suggest aortic dissection.

Is there motor or sensory loss, bowel or bladder dysfunction? Back pain associated with progressive or severe neurologic symptoms, motor loss, or urinary retention or bowel incontinence requires MRI or CT, which may indicate the need for emergent decompression.

Is there other pertinent history? Other pertinent history should include work history, past and present (a history of repeated

loading would suggest mechanical cause); fever (suggesting infectious cause); medications (anticoagulants associated with epidural hematomas, steroids associated with infection and compression fractures); hematuria (suggest nephrolithiasis or pyelonephritis); and pending litigation or worker’s compensation status (possible secondary gains).

Past Medical History. In addition to any history of back disorders, a thorough inquiry about any systemic disease is important. Ask if there is a history of (1) cancer (metastatic disease), (2) inflammatory disease, (3) IV drug abuse (diskitis), (4) arthropathies, (5) endocrinopathies (hyperparathyroidism), (6) bleeding disorders, (7) osteoporosis, or (8) sickle cell disease. Previous atherosclerotic or vascular disease suggests aortic disease; previous kidney stones or alcohol-related disease may suggest related disease. Knowledge of medications or other modalities used to treat present and past symptoms informs direct treatment decisions. Knowledge of current medications used by the patient gives clues about the presence of other systemic disease. The family history also is assessed. Diseases such as spondyloarthropathies (e.g., ankylosing spondylitis) have a familial component.

Physical Examination

Vital Signs. Vital signs are important because alterations may suggest a life-threatening process (e.g., hypotension and tachycardia with ruptured abdominal aortic aneurysm, hypertension with aortic dissection, fever with abscess, osteomyelitis, or diskitis).

Lower Back Inspection.

1. Observe the patient’s gait and movement in the examining room. Does the patient move cautiously, protecting himself or herself, or freely and appear to be in little pain?
2. Examine the patient while standing, searching for scoliosis (may be structural or secondary to muscle spasm), increase or decrease of lumbar lordosis or thoracic kyphosis (may predispose to mechanical pain), or pelvic obliquity (may indicate muscle spasm, leg-length discrepancy, or uncompensated scoliosis).
3. Assess the range of motion for the low back. Patients with significant mechanical pain usually flex without reversing the normal lumbar lordosis, and extension may aggravate facet causes or nerve root impingement.
4. Perform the palpation in an orderly fashion with the fingertips to localize the area of greatest tenderness (e.g., specific spinous process, paravertebral musculature).

Other Examinations, Including Neurologic Examination.

1. The neurologic assessment evaluates the asymmetry of reflexes (clinically, reflexes diminish with age, and uncovering asymmetry is key), dermatomal sensory loss, and focal muscle weakness (suggests nerve root impingement). If possible, motor testing of the legs is best done with the patient standing. Heel-walking and toe-walking indicate normal plantar and dorsiflexion strength, and a partial knee bend while bearing weight on one leg, then the other, indicate normal hip, buttock, and thigh muscle strength. A patient with a long history of back pain should be asked about previous motor, sensory, or reflex abnormality. The presence of clonus, hyper-reflexia, or upgoing toes (Babinski’s sign) indicate an upper motor neuron lesion.
2. A rectal examination can assess sphincter tone and anal wink. Testing for perianal sensation is necessary if there is any history of bowel or bladder dysfunction.

3. A head-to-toe examination looking for signs of systemic disease should include cardiac and pulmonary auscultation; abdominal examination for tenderness, aneurysm, or masses; and palpation of peripheral pulses.
4. The hips are examined for a musculoskeletal or inflammatory focus other than the back.

Straight Leg Raise. The straight leg raise is the classic test for sciatic nerve root irritation. It is sensitive but not specific for disk disease.⁴ This test is often negative in patients with spinal stenosis. With the knee extended, the leg is elevated until pain is elicited. A positive result is pain radiating down the leg below the knee in a dermatomal distribution when the leg is elevated to less than 90° (not back, buttocks, or thigh pain). Pain referred to an affected leg (“crossover pain”) with straight leg raise of the unaffected leg is insensitive but highly specific for nerve root irritation. In a patient who may be malingering, the straight leg raise can be done with the patient sitting with the knees flexed at the side of the bed and then passively straightening the legs. If there is true nerve root irritation, results should be similar in the sitting and the supine positions.

Ancillary Testing

Laboratory Tests. For mechanical causes of back pain, laboratory studies are of little use. For nonmechanical causes, erythrocyte sedimentation rate and complete blood count may be useful if inflammatory disease is suggested, but are rarely of use in the ED. Urinalysis is helpful in possible cases of renal disease

with referred back pain (nephrolithiasis, pyelonephritis, urinary tract infection).

Imaging. Although patient satisfaction is reportedly improved when imaging is performed,¹³ plain radiographs are not useful in uncomplicated mechanical low back pain of less than 6 weeks duration.¹² If the patient has a history of trauma with bony tenderness or focal signs of trauma, neurologic deficit, cancer, unexplained weight loss, pain that persists at rest or at night, advanced age, osteoporosis, prolonged glucocorticoid use, or fever, plain radiographs may be helpful.¹³ Plain radiographs should not be obtained, however, if advanced imaging (e.g., CT, MRI) is planned. Most patients do not require radiographic evaluation while in the ED.¹⁴

Emergency MRI, CT, or myelogram (in order of preference) is indicated if an acute, significant neurologic deficit such as motor loss or cauda equina syndrome is present. For patients with acute back and radicular pain but no motor weakness, and for those with chronic low back pain without neurologic deficit, obtaining MRI and CT does not improve outcome.¹⁵⁻¹⁷ Eighty-four percent of patients with sciatica will recover without surgery.¹⁸ For patients in whom infection or tumor is suggested, MRI (or bone scan followed by MRI) is the diagnostic test of choice.¹⁹ The degree of neurologic impairment and patient stability dictates whether these tests are obtained on an emergent or urgent basis.

DIFFERENTIAL DIAGNOSIS

After stabilization and assessment, the clinical findings aid in narrowing the differential diagnosis (Table 28-1). An algorithm

Table 28-1 Classic Findings in Selected Serious Causes of Acute Back Pain

	DIAGNOSES	HISTORY	IMPORTANT PHYSICAL EXAMINATION FINDINGS	ANCILLARY TESTING	COMMENTS	
Critical	Vascular	Aortic dissection	Often sudden-onset, “tearing” severe pain. Associated nausea, vomiting, acute anxiety are common. Syncope can occur	Associated diaphoresis, unstable vital signs. Hypertension is common. Unequal upper extremity blood pressure. New-onset aortic insufficiency murmur. Central and peripheral neurologic deficits secondary to ischemia	Choice of CT, MRI, aortogram depends on patient stability and availability of equipment	More common as a chest pain cause, but low back pain may be only complaint
		Abdominal aortic aneurysm (ruptured/expanding)	Pain may radiate to back, flank, or testicle. Patient may present with syncope	Pulsatile abdominal mass (especially if right of midline), abdominal bruits. Diminished lower extremity pulses or hypoperfusion or both	Bedside US. If “stable,” abdominal CT with contrast. Plain films may show a calcified enlarged aortic contour	Can also mimic renal colic, GI bleeding, diverticulitis, and myocardial infarction. 30% of signs are misdiagnosed
Infectious	Spinal epidural abscess	At-risk population with diabetes, chronic renal failure, intravenous drug use, alcoholism, cancer, or recent spinal surgery or trauma. Sepsis-linked history is common	Fever, reproducible radicular pain, other signs of sepsis. Localized body tenderness along spine Focal neurologic deficits are late findings (<50% patients). Rare cauda equina–like syndrome	CBC, blood cultures useful but nonspecific. MRI modality of choice. CT or myelography can be used. Search for source of infection. <i>Staphylococcus aureus</i> common cause (70%)	Presents as mass-occupying lesion compressing spinal cord; may be hematoma, malignancy, disk. Often begins as focal pyogenic infection in disk. Biopsy may be necessary	

Table 28-1 Classic Findings in Selected Serious Causes of Acute Back Pain—cont'd

	DIAGNOSES	HISTORY	IMPORTANT PHYSICAL EXAMINATION FINDINGS	ANCILLARY TESTING	COMMENTS
Mechanical	Cauda equina syndrome	Usually a history of back pain. Symptoms may develop over hours	Urinary retention and fecal incontinence. Saddle anesthesia, bilateral leg pain. Lower extremity weakness with hypo-reflexia	CT with or without contrast, MRI useful	Can result in severe dysfunction. An emergent condition caused by compression of lumbosacral nerve roots
	Spinal fracture with cord impingement	Acute onset, localized pain. Usually trauma history. Older population with osteoporosis also at risk	Bone tenderness, radicular, or cord compression findings	Plain films initially, then CT or MRI	Symptoms/signs depend on level
	Epidural hematoma	Usually patient with coagulation disorder, hereditary or acquired (e.g., anticoagulants). May occur after epidural anesthesia	Radicular findings (neurologic defects). Neurologic pattern similar to abscess	MRI, CT, or myelography	Can also occur in AV malformations
Emergent					
Infectious	Vertebral osteomyelitis	At-risk group similar to that for epidural abscess. Onset may be insidious. Back pain, tenderness, and stiffness may precede neurologic findings by significant time period	Fever and other constitutional symptoms. Localized body tenderness of two adjacent vertebrae	CBC, blood cultures generally low yield. Plain films diagnostic 80–95%, but MRI more accurate and detailed	Biopsy may be necessary for diagnosis. <i>S. aureus</i> most common
Immune	Transverse myelitis	Back pain and neurologic deficits. Almost 50% of patients worsen maximally in 24 hr	Partial/total loss sensory, motor, autonomic, and sphincter function below the level of the lesion. Leg weakness more common; arm involvement is rare. Bladder (bowel control) involved in most patients	Goal is to rule out mass lesion compressing the cord. Thought to be autoimmune origin. MRI is imaging modality of choice. Contrast CT and CT myelogram may be obtained	May be associated with multiple sclerosis, SLE, sarcoidosis. Also associated with Lyme disease. Epstein-Barr virus, and other viral (herpes, enterovirus) or bacterial (tuberculosis syphilis) infections
Mechanical	Back pain with neurologic deficits Intervertebral disk herniation Spinal stenosis Spinal fractures without cord impingement Malignancy Sciatica with potential of nerve root compression	Most patients recall atraumatic mechanisms (lifting, twisting). Common complaints are stiffness, tenderness, decreased range of motion	Positive straight leg raise test. Muscular weakness. Potential for sensory deficits. Absent or diminished deep tendon reflexes	Selective use of plain films. CT or MRI performed for complete assessment when “red flag” present	Search for “red flags” (see Box 28-1) to rule out serious underlying disease

AV, arteriovenous; CBC, complete blood count; CT, computed tomography; GI, gastrointestinal; MRI, magnetic resonance imaging; SLE, systemic lupus erythematosus.

(see Fig. 28-1) that takes into account important differential considerations, such as abnormal vital signs, the presence of fever, and an abnormal neurologic examination, is a useful tool. After collecting this information the emergency physician should be able to answer two additional key questions. *Does the patient need emergent, urgent, or more routine treatment?* *Does the patient need surgical or medical treatment?* Young children for whom there is no clear explanation of their back pain warrant earlier and more extensive evaluation for infection and tumor.

■ EMPIRICAL MANAGEMENT

The initial empirical management of acute back pain depends on the presenting vital signs and the patient's overall appearance. Figure 28-2 details the specific management considerations for treating unstable patients.²⁰⁻²⁶ For a stable patient,

early effective pain management can be of significant value. The choice of analgesic is dictated by the patient's and physician's perception of the degree of pain. Physicians notoriously underestimate and undertreat pain, especially acute and chronic low back pain. If the pain is severe, IV opioids are the preferred analgesic and should be given in a titrated fashion. Frequent reassessment of the patient for an adequate response is required. After adequate response to the initial IV opioid, an oral agent can be administered in preparation for discharge. For patients with less acute symptoms, an oral opioid or a nonsteroidal anti-inflammatory drug (NSAID) is appropriate. NSAIDs are effective for short-term relief of acute low back pain but are not superior to acetaminophen.²⁷ Their safety profiles must be considered in patients with acid peptic disease, renal insufficiency, diabetes, and liver pathology. NSAIDs are routinely used for chronic back pain, but evidence for their effectiveness is lacking.²⁸ Muscle relaxants such as

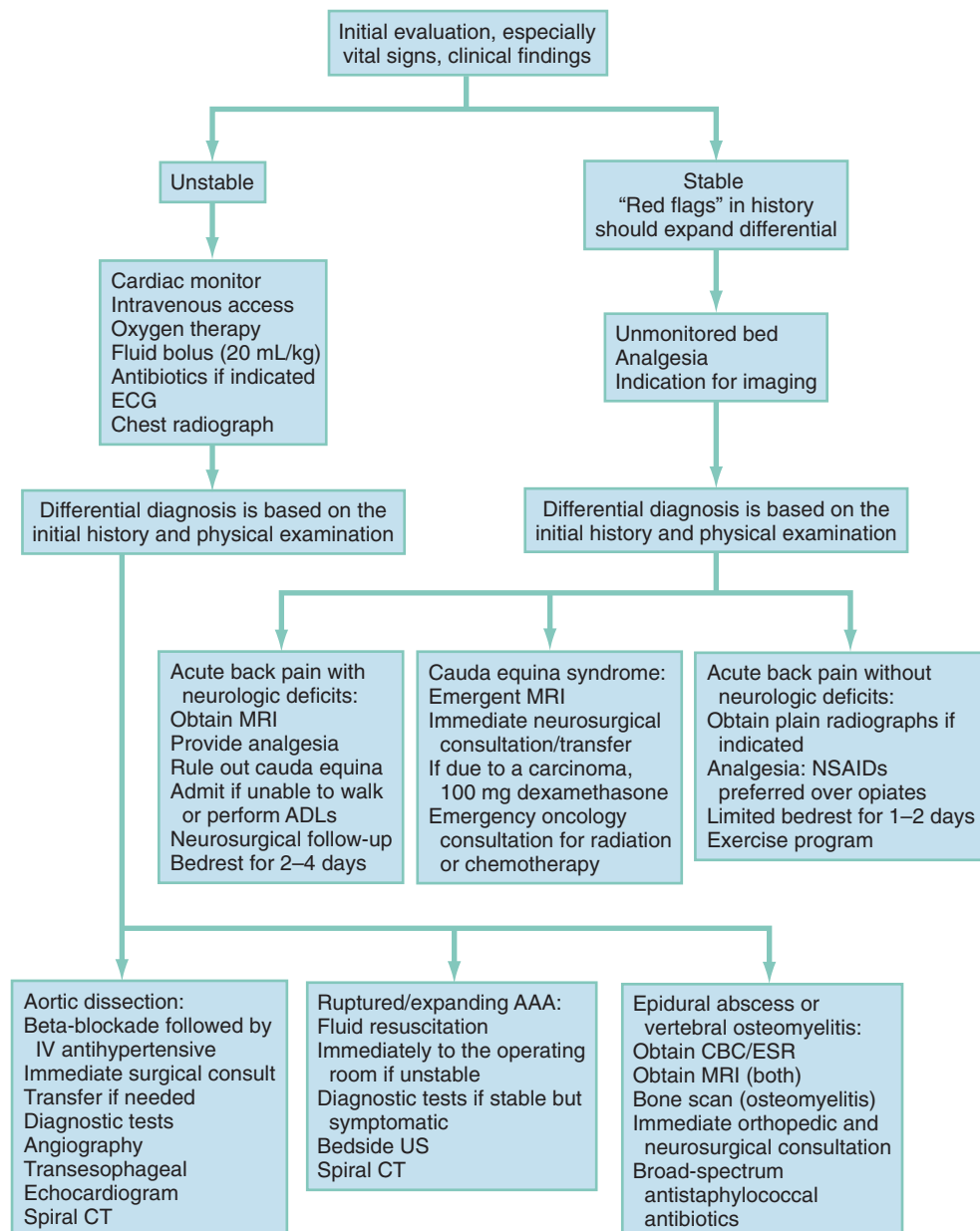


Figure 28-2. Management of acute low back pain. AAA, abdominal aortic aneurysm; ADLs, activities of daily living; CBC, complete blood count; CT, computed tomography; ECG, electrocardiogram; ESR, erythrocyte sedimentation rate; IV, intravenous; MRI, magnetic resonance imaging; NSAIDs, nonsteroidal anti-inflammatory drugs; US, ultrasound.

benzodiazepines and cyclobenzaprine (Flexeril) may be effective adjuncts for acute low back pain, but their significant adverse side effects require they be used with caution.^{29,30}

For some patients, chronic recurrent mechanical back pain is a long-term issue. Such patients need support, and often a multidisciplinary approach to help manage their chronic pain or recurrent flare-ups. Management and follow-up require referral to primary care or a pain clinic. Tools that may be tried by their primary care or pain clinic may include lumbar supports,^{31,32} traction,³³ acupuncture,³⁴ spinal manipulative therapy,³⁵ physical therapy or chiropractic therapy,³⁶ back education,³⁷ massage,³⁸ exercise therapy,³⁹ transcutaneous electrical nerve stimulation (TENS),⁴⁰ heat therapy,⁴¹ epidural injection of methylprednisolone,^{42–44} and tricyclic or tetracyclic antidepressant therapy.⁴⁵ ED treatment is directed to relief of their current exacerbation.

■ DISPOSITION

The disposition of patients with back pain depends on their diagnosis. Patients with one of the life-threatening or disabling causes require admission and further emergent treatment. Patients with acute cord compression from a fracture, disk protrusion, abscess, or hematoma require urgent neurosurgical evaluation.

Patients who are unable to walk or require IV analgesics for adequate pain control should be considered for admission to hospital or the ED observation unit. If pain can be controlled

using oral analgesics, patients can be discharged with appropriate follow-up. Reassure the patient with acute mechanical back pain that the vast majority of patients eventually experience spontaneous relief of pain. Prescribe NSAIDs, supplemented with oral opioids for moderate to severe pain, and refer the patient for primary care. Oral opioids may be effective for short-term relief, but long-term efficacy is less clear and aberrant medication taking occurs in up to 24% of cases of chronic back pain.^{46,47} Repeat visits to the ED for chronic back pain can be a source of frustration for the physician and the patient. Patients frequently receive prescriptions for pain relief and referral for primary care but for many reasons do not complete this referral. For such a chronic, recurring, painful condition, repeat visits to the doctor are to be expected. Short-term oral opioids plus NSAIDs or acetaminophen may be what the patient needs. Rather than a consistent relationship with a primary care provider, repeat visits to the ED raise the possibility of drug seeking. For those patients, it may become necessary for the ED physician to prescribe simple analgesics such as NSAIDs instead of narcotics. Encourage patients to maintain activities as their pain allows, avoiding heavy lifting or twisting during the acute phase. Advise patients to avoid strict or prolonged bedrest as it is no more effective and may be worse than maintaining reasonable activities.^{48,49}

The references for this chapter can be found online by accessing the accompanying Expert Consult website.

CHAPTER 29 Cyanosis

Madonna Fernández-Frackelton and Jennifer Bocock

■ PERSPECTIVE

Epidemiology

Cyanosis is a blue or purple appearance of the skin or mucous membranes. This clinical finding is secondary to inadequately oxygenated blood perfusing peripheral tissues or the presence of abnormal hemoglobin forms unable to bind oxygen or to supply adequate oxygen to end organs and tissues. Cyanosis is a relatively rare presenting chief complaint in the emergency department (ED) and is most commonly noted in patients with a hypoperfused state or known cardiopulmonary disease, including congenital heart disease.¹ Although carbon monoxide poisoning and cyanide toxicity result in difficulty with hemoglobin oxygenation or tissue hypoxia, these entities typically do not present with the clinical finding of cyanosis and are discussed in other chapters.

Pathophysiology

Cyanosis is evident on physical examination when the absolute amount of desaturated (unoxxygenated) hemoglobin in the circulating capillary blood ($>4\text{--}5\text{ g/dL}$ in whole blood) is elevated. It is not a percent of desaturated total hemoglobin mass or a decreased amount of oxyhemoglobin. For this reason patients with a relatively low hemoglobin exhibit cyanosis at a much lower partial pressure of oxygen (Pao_2) and arterial oxygen saturation (Sao_2) than those with normal hemoglobin levels. Cyanosis is an insensitive indicator of tissue oxygenation.² Its presence suggests hypoxia, but its absence does not exclude it.

Abnormal hemoglobin forms contribute significantly to cyanotic disease. Under normal conditions, red blood cells (RBCs) contain hemoglobin with iron in the reduced ferrous state (Fe^{2+}). The iron molecule may be oxidized to the ferric state (Fe^{3+}) to produce methemoglobin. This reaction impairs the ability of hemoglobin to transport oxygen to and carbon dioxide from the tissues. The oxygen dissociation curve is shifted to the left, resulting in tissue hypoxia and lactic acid production (Fig. 29-1). Methemoglobin normally accounts for less than 1% of total hemoglobin.³ Cyanosis results when greater than 10 to 15% of the total hemoglobin is methemoglobin ($\geq 1.5\text{ g/dL}$) that has a dark purple-brown color, even when exposed to room air. Methemoglobin is reduced to ferrous hemoglobin primarily by nicotinamide adenine dinucleotide (NADH) cytochrome- b_5 reductase, an enzyme system present within RBCs. A secondary NADPH-dependant system uses glutathi-

one production and glucose-6-phosphate dehydrogenase (G6PD) to reduce methemoglobin to hemoglobin. This secondary pathway normally plays a minor role, but is accelerated by methylene blue.³

Primary methemoglobinemia is a congenital error of enzyme metabolism, with either diminished levels of NADH reductase or an abnormally functioning enzyme. Patients may present with cyanosis in a stable compensated state. Acquired methemoglobinemia occurs when methemoglobin production (hemoglobin oxidation) is accelerated beyond the capacity of NADH reductase activity. This usually occurs as a drug reaction. (See Box 29-1 for common causes.) Newborns are at risk for methemoglobinemia because their NADH reductase activity is relatively low.³

■ DIAGNOSTIC APPROACH

Differential considerations for patients presenting with cyanosis are listed in Box 29-2.

Pivotal Findings

Symptoms

The onset, duration, and time of day of symptoms, and any previous episodes should be noted. Precipitating factors may include exposure to cold air or water, high altitude, or exercise in patients with a history of cardiopulmonary disease. Additional history should include known congenital heart disease or cardiopulmonary disease, hypercoagulable states, and any family history of cyanotic disease or hematologic illness. A history of home or occupational exposures to fumes or chemicals should be obtained, including aniline, azo dyes (pyridium), phenacetin, and nitrates.⁴ A drug history should be reviewed, including use of prescription and over-the-counter medications, health food supplements, and herbal or alternative preparations.⁵ The potential of pseudocyanosis resulting from exposure to dyes, heavy metals, or topically absorbed pigments should be explored.²

In infants, congenital heart disease is suggested by difficulty feeding, sweating, lethargy, poor weight gain, or respiratory distress. Episodic cyanotic events, or “Tet spells,” may be seen in children with tetralogy of Fallot (ventricular septal defect, overriding aorta, pulmonary stenosis or atresia, and right ventricular hypertrophy with outlet obstruction). These patients present with cyanosis, tachypnea, and anxiety due to

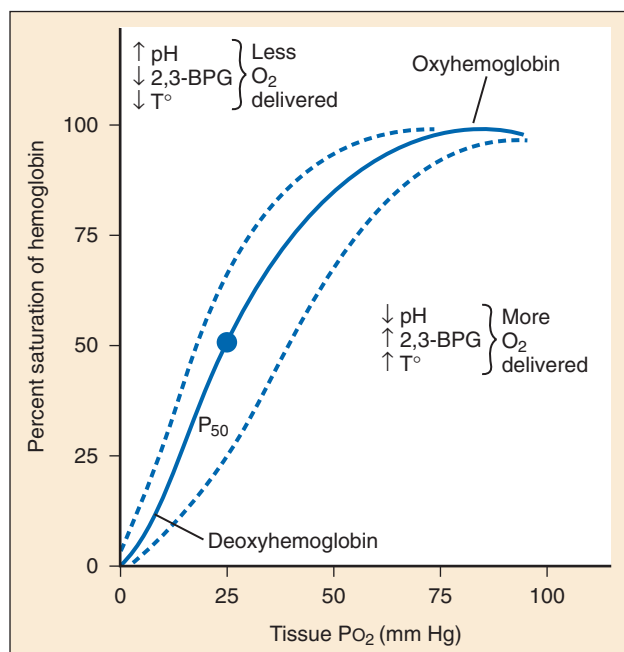


Figure 29-1. Hemoglobin-oxygen dissociation curve. Deoxyhemoglobin does not bind oxygen efficiently. Methemoglobin has a high affinity for oxygen molecules and does not readily release oxygen to the peripheral tissues. This shifts the normal oxygen dissociation curve to the left, resulting in hypoxia and lactic acid production. Typically, when acid is produced in the tissues, the dissociation curve shifts back to the right, facilitating oxygen release; however, the high affinity of methemoglobin prevents this normal process. (Redrawn from Benz EJ Jr: Hemoglobinopathies. In Harrison's online.) 2,3-BPG, 2,3-bisphosphoglycerate; PO₂, partial pressure of oxygen; P₅₀, oxygen half saturation pressure of hemoglobin; T°, temperature.

decreased pulmonary blood flow with shunting of unoxygenated blood into the peripheral circulation.^{6,7}

Signs

There is significant interobserver variability in detecting cyanosis on physical examination. Room lighting and temperature may affect examination of the skin and mucous membranes. A patient's natural skin tone, thickness, and pigmentation also may alter findings.

Central cyanosis is often secondary to the shunting of venous unsaturated hemoglobin into the arterial circulation or the presence of abnormal hemoglobin. A bluish discoloration of the skin and mucous membranes is best seen on perioral skin, oral mucosa, or conjunctivae.

Peripheral cyanosis is secondary to vasoconstriction and slow flow of normally oxygenated hemoglobin in arterial blood, allowing for greater oxygen extraction by the tissues. Peripheral cyanosis affects capillary beds and typically is seen in the extremities and nail beds. Differential cyanosis may occur in either the upper or lower (or the right or the left) half of the body, with the remainder appearing well oxygenated. This form of cyanosis usually is seen in cases of cyanotic heart disease with multiple anomalies.

Vital signs should be obtained on all patients. Temperature is typically normal. Blood pressure and heart rate may be high, normal, or low depending on the underlying cause. Upper airway obstruction and other signs of respiratory insufficiency should be sought. Intermittent apnea in infants suggests central nervous system immaturity or a central lesion. Infants with cyanosis, increased respiratory depth, periodic apnea epi-

BOX 29-1 COMMON CAUSES OF METHEMOGLOBINEMIA

Hereditary

Hemoglobin M

NADH methemoglobin reductase deficiency (homozygote and heterozygote)

Acquired

Medications

Amyl nitrite

Antineoplastics (cyclophosphamide, ifosfamide, flutamide)

Celecoxib

Dapsone

Local anesthetics (benzocaine, lidocaine, prilocaine)

Nitroglycerin

Nitroprusside

Phenacetin

Phenazopyridine (pyridium)

Quinones (chloroquine, primaquine)

Sulfonamides (sulfanilamide, sulfathiazide, sulfapyridine, sulfamethoxazole)

Chemical Agents

Aniline dye derivatives (shoe dyes, marking inks)

Butyl nitrite

Chlorobenzene

Fires (heat-induced denaturation)

Food adulterated with nitrites

Food high in nitrates

Isobutyl nitrite

Naphthalene (mothballs)

Nitrophenol

Nitrous gases (seen in arc welders)

Paraquat

Silver nitrate

Trinitrotoluene

Well water (nitrates)

Pediatric

Reduced NADH methemoglobin reductase activity in infants (4 mo)

Seen in association with low birth weight, prematurity, dehydration, acidosis, diarrhea, and hyperchloremia

NADH, reduced nicotinamide adenine dinucleotide.

Modified from Goldfrank LR: Toxicologic Emergencies, 6th ed. Stamford, Conn, Appleton and Lange, 1998.

sodes, or diaphoresis with feeding may have congenital heart disease.⁶ Tachypnea (>60 breaths/min) in a newborn may indicate a pulmonary disorder, congenital heart disease, infection, a metabolic disorder, or central nervous system pathology.⁸

General appearance and mental status should be evaluated. The head, eyes, ears, nose, and throat examination may reveal central cyanosis. Funduscopic examination may detect dilated tortuous veins and papilledema in patients with cyanotic congenital heart disease.⁹ Jugular venous distention may be seen on the neck examination in patients with pulmonary edema.

The chest examination may reveal crackles, wheezing, or inadequate ventilation. Heart sounds should be assessed for tachycardia, abnormal rhythm, or gallops, and the presence and quality of murmurs, especially in newborns. Central pulse strength should be noted. The abdomen should be examined for the presence of hepatosplenomegaly, a pulsatile mass, or abdominal bruit.

Extremity examination includes evaluation of nail beds for peripheral cyanosis, strength and symmetry of distal pulses,

BOX 29-2 DIFFERENTIAL DIAGNOSIS OF CYANOSIS

- I. Peripheral cyanosis
 - A. Low cardiac output states
 - 1. Shock
 - 2. Left ventricular failure
 - 3. Hypovolemia
 - B. Environmental exposure (cold)
 - 1. Air or water
 - C. Arterial occlusion
 - 1. Thrombosis
 - 2. Embolism
 - 3. Vasospasm (Raynaud's phenomenon)
 - 4. Peripheral vascular disease
 - D. Venous obstruction
 - E. Redistribution of blood flow from extremities
- II. Central cyanosis
 - A. Decreased arterial oxygen saturation
 - 1. High altitude (>8000 ft)
 - 2. Impaired pulmonary function
 - a. Hypoventilation
 - b. Impaired oxygen diffusion
 - c. Ventilation-perfusion mismatching
 - (1) Pulmonary embolism
 - (2) Acute respiratory distress syndrome
 - (3) Pulmonary hypertension
 - d. Respiratory compromise
 - (1) Upper airway obstruction
 - (2) Pneumonia
 - (3) Diaphragmatic hernia
 - (4) Tension pneumothorax
 - (5) Polycythemia
 - B. Anatomic shunts
 - 1. Pulmonary arteriovenous fistulae and intrapulmonary shunts
 - 2. Cerebral, hepatic, peripheral arteriovenous fistulae
 - 3. Cyanotic congenital heart disease
 - a. Endocardial cushion defects
 - b. Ventricular septal defects
 - c. Coarctation of aorta
 - d. Tetralogy of Fallot
 - e. Total anomalous pulmonary venous drainage
 - f. Hypoplastic left ventricle
 - g. Pulmonary vein stenosis
 - h. Tricuspid atresia and anomalies
 - i. Premature closure of foramen ovale
 - j. Dextrocardia
 - k. Pulmonary stenosis of atrial septal defect
 - l. Patent ductus arteriosus with reversed shunt
 - C. Abnormal hemoglobin
 - 1. Methemoglobinemia
 - a. Hereditary
 - b. Acquired
 - 2. Sulfhemoglobinemia
 - 3. Mutant hemoglobin with low oxygen affinity (e.g., hemoglobin Kansas)



Figure 29-2. Symmetrical cyanosis. Equal cyanosis and clubbing of hands and feet resulting from transposition of great vessels and a ventricular septal defect without patent ductus arteriosus.

monary disease, cystic fibrosis), and some gastrointestinal disorders (cirrhosis, Crohn's disease, and regional enteritis). Thrombotic events should also be considered with findings of skin and nail bed hemorrhages or end-organ damage (eye, kidney).

A neurologic examination should be performed focusing on mental status, symmetry of motor and sensory function, and any gross deficit.

Laboratory and Ancillary Testing

The complete blood count should be checked to assess for polycythemia or anemia.¹⁰ Peripheral smear assesses RBC morphology and fragments, as well as white blood cell differential count.

Interpretation of pulse oximetry is problematic in the setting of cyanosis (see Chapter 3). Assessment of distal perfusion usually determines if poor circulation is a cause of low pulse oximetry. Pulse oximetry measures light absorbance of tissue at 660 nm (red reduced hemoglobin) and 940 nm (infrared oxyhemoglobin). The ratio of these two readings is the basis of the pulse oximetry calculation. Methemoglobin absorbs well at both wavelengths, resulting in a saturation approximation of 85%, regardless of the actual P_{aO_2} and S_{aO_2} .¹¹

Arterial blood gas testing assesses S_{aO_2} , often sampled when the patient is breathing room air (see Fig. 29-1). Co-oximetry measurements should be specifically ordered if carbon monoxide exposure or methemoglobinemia is suspected. Sulfhemoglobin is reported as methemoglobin on co-oximetry, so if sulfhemoglobinemia is possible, measured oxygen saturation should be specifically requested.¹²

Imaging

A chest radiograph should be ordered to evaluate lung fields for consolidation, infiltrates, and increased vasculature or pulmonary edema. The cardiac silhouette and mediastinum may suggest congenital heart disease. In patients thought to have pulmonary embolism, imaging may include lower extremity venous Doppler ultrasound (if deep venous thrombosis

and capillary refill. Evidence of chronic vascular disease, such as hair loss and temperature difference, should be noted. Clubbing of the nails may occur due to increased soft tissue and expansion of the capillary beds (Fig. 29-2). Clubbing may be idiopathic or hereditary, but is usually the result of chronic hypoxemic states, such as cyanotic heart disease, infective endocarditis, pulmonary disease (chronic obstructive pul-

symptoms are present), ventilation-perfusion scanning or computerized tomography pulmonary angiogram.

Electrocardiogram and Echocardiogram

An electrocardiogram should be performed on all patients with cyanosis to assess for arrhythmias and acute ischemic changes. Right-axis deviation or right ventricular hypertrophy may be seen with significant cardiopulmonary disease (e.g., cor pulmonale, acute pulmonary hypertension). An echocardiogram may be helpful in detecting septal defects in infants or valvular disease in infants and adults.

DIFFERENTIAL ALGORITHMS

Figures 29-3 and 29-4 outline the differential diagnosis and treatment for peripheral and central cyanosis, respectively. After the initial assessment is completed, and the distribution of cyanosis is noted, the clinician should begin 100% oxygen therapy and follow steps to determine the cause of cyanosis. Clinical improvement with oxygen suggests diffusion impairment. Patients who do not respond to oxygen are more likely to have ventilation-perfusion ratio abnormalities, such as shunting from a consolidated pulmonary lobule, or congenital heart disease with right-to-left shunting. Cardiac size and silhouette on chest radiograph may provide

a clue to the presence of congenital cardiac disease. If heart size is normal, impaired pulmonary function, pulmonary embolus, or other noncardiac causes should be considered. If no improvement occurs with 100% oxygen therapy, the patient's respiratory status should be reassessed, and tension pneumothorax or upper airway obstruction considered. Pulmonary embolus should be considered and a ventilation-perfusion scan or spiral computed tomography pulmonary angiogram performed. If a patient exhibits no respiratory distress and remains resistant to oxygen therapy, cardiac shunting or abnormal hemoglobin forms should be considered and treated accordingly.

Critical Diagnoses

Acute cardiovascular and respiratory compromise must be considered in a patient presenting with cyanosis and symptoms or signs of shock. The differential diagnosis for these critical presentations includes acute congestive heart failure, acute coronary syndromes, hypovolemic or cardiogenic shock, acute respiratory insufficiency or failure, massive pulmonary embolism, an exacerbation or decompensation in a patient with known congenital heart disease, or the first presentation of pediatric congenital heart disease. These patients require emergent treatment, critical therapeutic intervention, and admission to the intensive care unit.

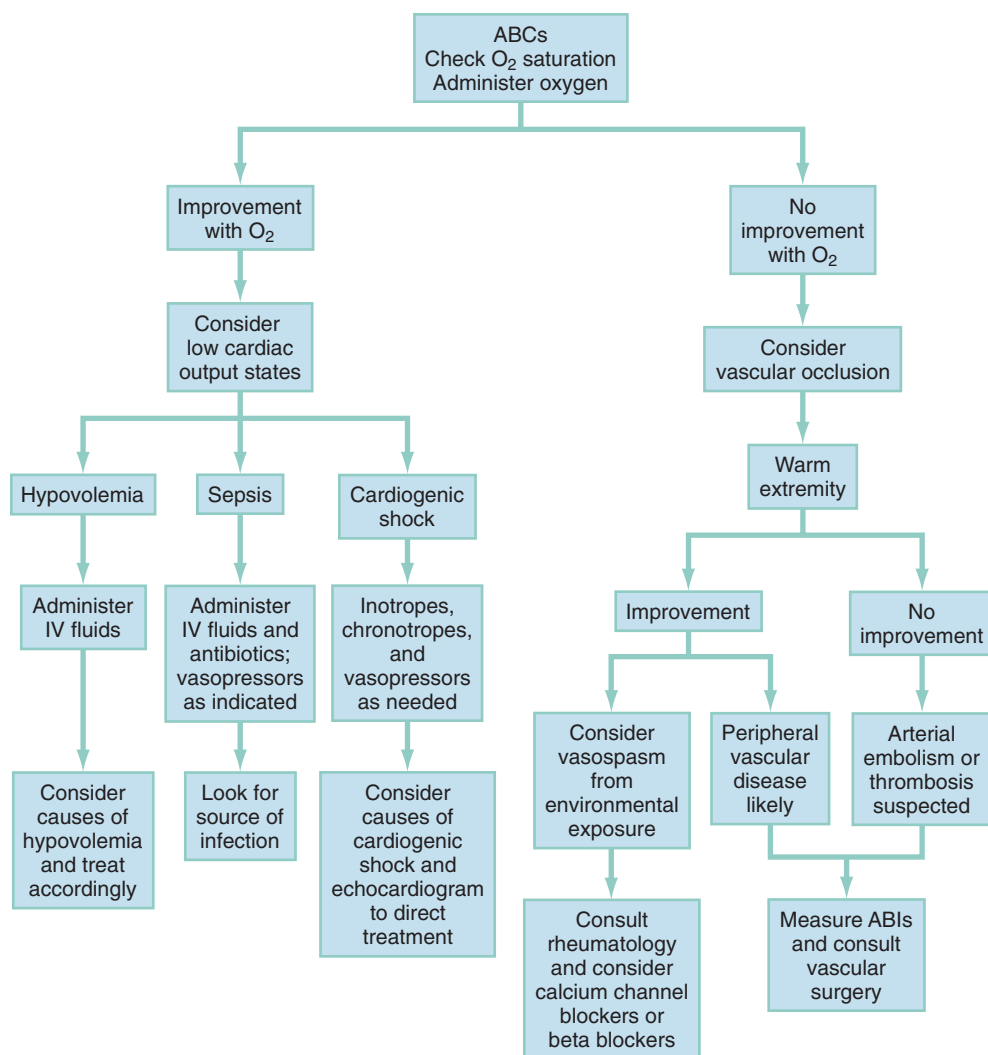


Figure 29-3. An algorithmic approach to peripheral cyanosis. ABCs, airway, breathing, circulation; ABI, ankle brachial index; IV, intravenous.

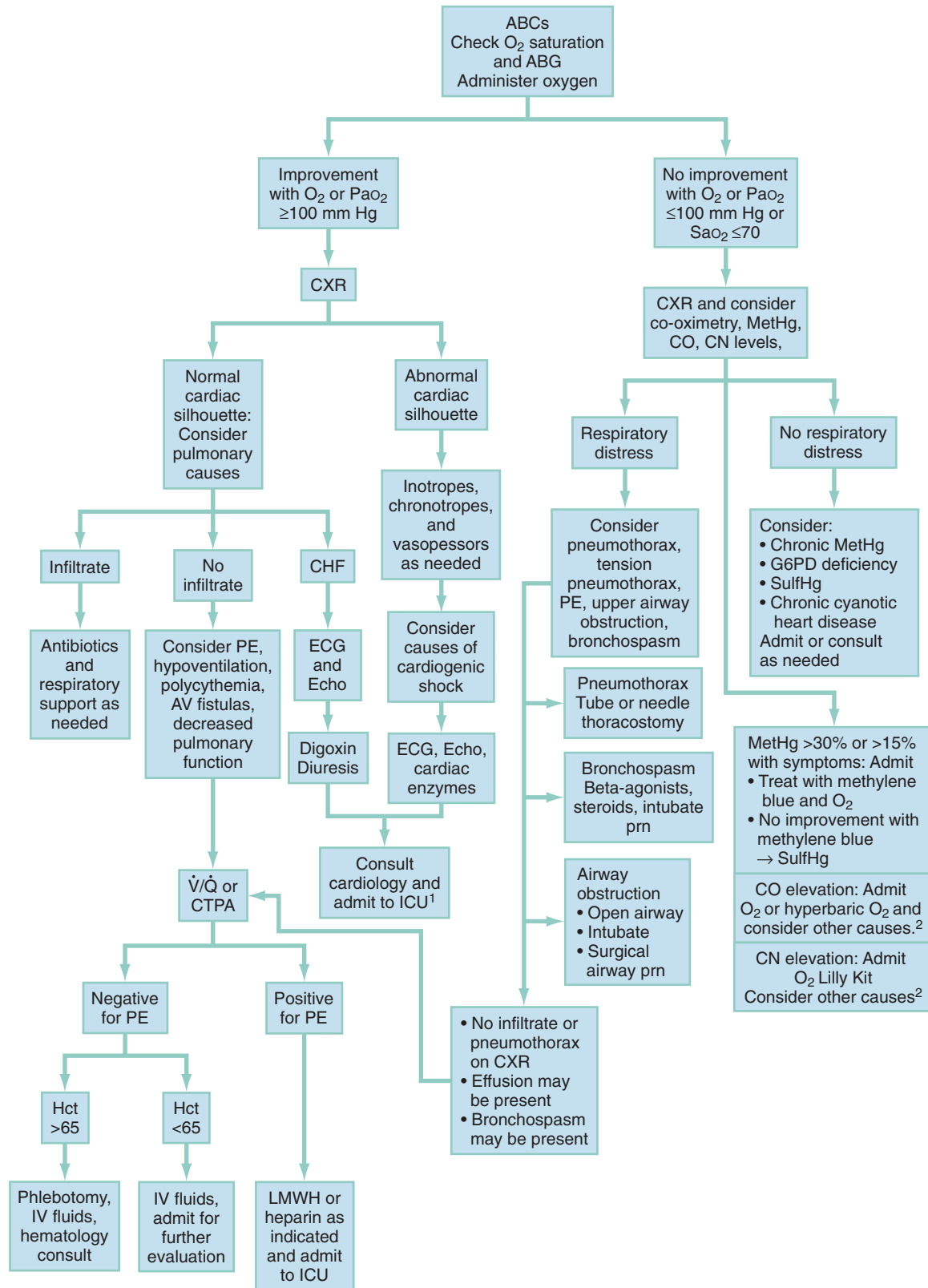


Figure 29-4. An algorithmic approach to central cyanosis. ABCs, airway, breathing, circulation; ABG, arterial blood gas; AV, arteriovenous; CHF, congestive heart failure; CN, cyanide; CO, carbon monoxide; CTPA, computed tomography pulmonary angiography; CXR, chest radiograph; ECG, electrocardiogram; Echo, echocardiogram; G6PD, glucose-6-phosphate dehydrogenase; Hct, hematocrit; ICU, intensive care unit; IV, intravenous; LMWH, low-molecular-weight heparin; MetHgb, methemoglobin; PaO₂, partial pressure of arterial oxygen; PE, pulmonary embolus; prn, as needed; RA, room air; Sao₂, arterial oxygen saturation; SulfHg, sulfhemoglobin; V/Q, ventilation-perfusion scan.

¹ Patients with chronic cyanotic heart disease may not require ICU care or even admission. Disposition should be discussed with patient's cardiologist.

² Cyanide and carbon monoxide toxicity do not present with cyanosis. If either of these is present, consider coexisting diagnosis.

Emergent Diagnoses

Methemoglobinemia is an infrequent cause of cyanosis, but should be considered in patients presenting without a history or physical findings suggestive of cardiovascular or pulmonary disease.

Sulfhemoglobinemia is a rare cause of cyanosis, most commonly occurring after exposure to hydrogen sulfide from organic sources, medications that are sulfonamide derivatives, or gastrointestinal sources (bacterial overgrowth). Strong consideration should be given to sulfhemoglobin toxicity in patients with cyanotic findings and methemoglobin on co-oximetry, but who do not improve with methylene blue treatment.

Polycythemia is defined as an elevated RBC mass due to one of three causes. Polycythemia vera is a disorder of bone marrow stem cells with increased RBC mass, cyanosis, and splenomegaly. Patients may present with hyperviscosity syndrome. Secondary polycythemia occurs with either an appropriate or inappropriate increase of erythropoietin, a physiologic response to chronic hypoxemia ($\leq 92\%$ oxygen saturation), cyanotic congenital heart disease, cigarette smoking, or high altitude exposures. Relative polycythemia is an increased RBC mass, often due to dehydration or reduced plasma volumes.

Finally, vascular disease, such as Raynaud's phenomenon, may present with a cyanotic appearance. Raynaud's phenomenon occurs in 15% of the population and has a female predominance. Patients have an abnormal response to excessive cold or emotional stress and report vasoconstriction, profound cold sensitivity, and recurrent events of sharply demarcated pallor or cyanosis of the digits. Most commonly, the cutaneous arterial capillary beds of the fingers and toes are affected, but tongue, ear, and other distal areas are sometimes also affected.¹³

EMPIRICAL MANAGEMENT

Administration of high-flow oxygen is the first therapy for patients with cyanosis. Any clinical improvement, or lack thereof, should be noted. At this point, consideration of abnormal hemoglobin and toxin-induced cyanosis is crucial because the administration of appropriate antidotes and systemic therapies may decrease morbidity and improve outcome.

Intravenous fluid resuscitation should be initiated in patients with hypovolemia. Treatment of congestive heart failure, arrhythmia, or poor cardiac output should occur as clinical conditions indicate. Cardiology consultation is recommended in patients thought to have congenital or ischemic heart disease. Although several specific treatments are discussed here, the cause of the cyanosis may be elusive, and hospitalization is required to determine it.

Specific Strategies

Methemoglobinemia and Sulfhemoglobinemia

If cutaneous exposure with an inciting agent occurred (i.e., aniline dyes), complete decontamination with soap and water is recommended. The staff should use appropriate protective

equipment. Urgent treatment with oxygen and methylene blue (1–2 mg/kg IV over 5 minutes)¹⁴ is indicated for patients with symptomatic hypoxia (dysrhythmias, angina, respiratory distress, seizures, or coma) and methemoglobin levels greater than 30%. Sulfhemoglobinemia is suggested when the laboratory reports an elevated methemoglobin level and the patient does not respond to methylene blue. Treatment of sulfhemoglobinemia is supportive in addition to removing the causative agent.

Other Causes of Cyanosis

Acute therapy for patients with symptomatic hyperviscosity syndrome and secondary polycythemia includes phlebotomy and volume expansion with isotonic crystalloid. The goal of therapy is a normal hematocrit (45% in men and 42% in women). Long-term therapy is focused on the underlying cause, and patients may require referral to a hematologist.^{10,15}

Raynaud's phenomena is treated with warming the affected digits and extremities. Systemic vasodilating agents (e.g., calcium channel blockers [nifedipine] or nitrates) may be useful in the acute setting.¹⁶ If there is no improvement of peripheral cyanosis with warming and administration of 100% oxygen, arterial insufficiency or occlusion may be present. In cases of critical limb ischemia, intravenous heparin should be considered in consultation with a vascular surgeon. Embolic sources, such as endocarditis and abdominal aortic aneurysms should be considered. Vascular bypass, intra-arterial thrombolysis, or stenting may be indicated.

Carbon monoxide and cyanide poisoning do not typically present with cyanosis and are covered elsewhere.

PATIENT DISPOSITION

Admission

All patients with a first episode of cyanosis or an uncertain cause require admission. Cardiology consultation and referral is recommended for children with a first episode of congestive heart failure and newly diagnosed or suggested congenital heart disease. Surgical consultation and intervention are indicated for acute arterial occlusion from embolic or thrombotic sources.

Discharge

Patients with peripheral cyanosis from vasospasm, asymptomatic methemoglobinemia less than 15%, and stable patients with primary pulmonary disease may be treated as outpatients, after several hours of monitoring in the ED. Unless the patient has a previous diagnosis of chronic cyanosis, follow-up must be arranged within 24 hours. Instructions should clearly state that if the cyanosis worsens, or if dyspnea, altered mentation, or chest pain occur after discharge, the patient must return immediately to the ED.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.

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■ PERSPECTIVE

Epidemiology

Sore throat is a frequent complaint among patients presenting to the emergency department (ED). The National Health Care Survey in 2001–2002 reported more than 2.4 million ED visits with complaints relating to the throat and acute pharyngitis diagnosed in more than 1.9 million patients.¹ The chief complaint of sore throat is seen in every age group and has no sex predilection. Sore throat and other upper respiratory tract infections are some of the most common diseases for which care is sought and for which antibiotics are prescribed.^{1,2}

Pathophysiology

Sore throat results from irritation or inflammation of any anatomic surface within the oropharynx. The oropharynx is defined by the following borders: (1) posteriorly by the prevertebral fascia, (2) laterally by the buccinator muscle groups, (3) superiorly by the base of the skull, and (4) inferiorly by the vocal cords (Fig. 30-1). Pain may originate within the buccal mucosa, tongue, palatine tonsils, lingual tonsils, adenoids, soft palate, and posterior pharyngeal wall. In addition, pain may result from infection, inflammation, or invasive diseases of the potential spaces within and surrounding the oropharynx—the peritonsillar, retropharyngeal, sublingual, submental, lateral pharyngeal, parotid, buccal, and pretracheal spaces. Sore throat also occurs with inflammatory changes of the epiglottis, aryepiglottic folds, vocal cords, and subglottic region. Infectious diseases of dental structures and cervical nodes and the presence of middle ear fluid may cause sore throat through referred pain. The 9th and 10th cranial nerves provide sensory input from the oropharynx, larynx, middle ear, and external auditory canal.³ Many systemic diseases, including hepatitis, infectious mononucleosis, retroviral disease, and neutropenia, may also have sore throat as part of their symptom complex or initial presentation.

Sore throat commonly results from infections within the oropharynx, and the majority of these illnesses are self-limited. Table 30-1 lists common infectious and noninfectious causes of sore throat. Although the majority of infections are mild and not associated with serious complications, several infections may result in airway compromise, systemic disease, or sepsis.

Viruses cause the majority of cases of sore throat—up to 80% by some reports.^{2,4} Enterovirus infection accounts for the majority of sore throats in all age groups from late spring through

autumn. Adenovirus, rhinovirus, parainfluenza virus, influenza virus, and respiratory syncytial virus predominate during winter months. Epstein-Barr virus (EBV), herpes simplex, and varicella-zoster virus have less seasonal predilection.

Acute pharyngitis due to bacterial infection is much less common than viral infection, and the cause can usually be discerned by a combination of clinical evaluation and rapid strep testing, because group A β -hemolytic streptococcus (GABHS) is the most common bacterial pathogen.^{2,4,5} Aerobes such as GABHS with anaerobes or anaerobes alone cause infection in the deeper planes of the pharynx and neck. GABHS is isolated as the offending pathogen in 10 to 15% of all patients with sore throat. The incidence of GABHS in school-age children with sore throat may reach 15 to 30%, and some studies have reported the incidence as high as 50%.^{2,5-8} GABHS is implicated in as few as 5% of adults with sore throat, but 47 to 73% of adults with pharyngitis are prescribed antibiotics.^{2,9} GABHS is most often isolated from patients between late winter and spring. GABHS infection may cause coinfection with other viral agents, but distinguishing acute infection from carrier state is difficult.

Fungal colonization and systemic infection with *Candida albicans* may occur throughout the oral cavity. Immunocompromised patients may present with severe infections or repeated infections. Recent antibiotic therapy, chemotherapy, and radiation therapy increase the risk for fungal colonization with *Candida* species.

Sore throat may be a manifestation of noninfectious systemic disease, trauma, tumor, or congenital anomaly. Additional systemic complaints or physical findings will often accompany these diseases.

■ DIAGNOSTIC APPROACH

Differential Considerations

The stable patient should receive a directed history and physical examination followed by judicious use of ancillary testing. Table 30-1 lists the possible causes of acute sore throat.

Pivotal Findings

History

Characteristic of Pain. Rapidly progressing symptoms, high fever, or severe pain suggest the possibility of invasive disease.^{10,11} A duration of several days accompanied by fever suggests deeper plane infection or systemic disease. Inflam-

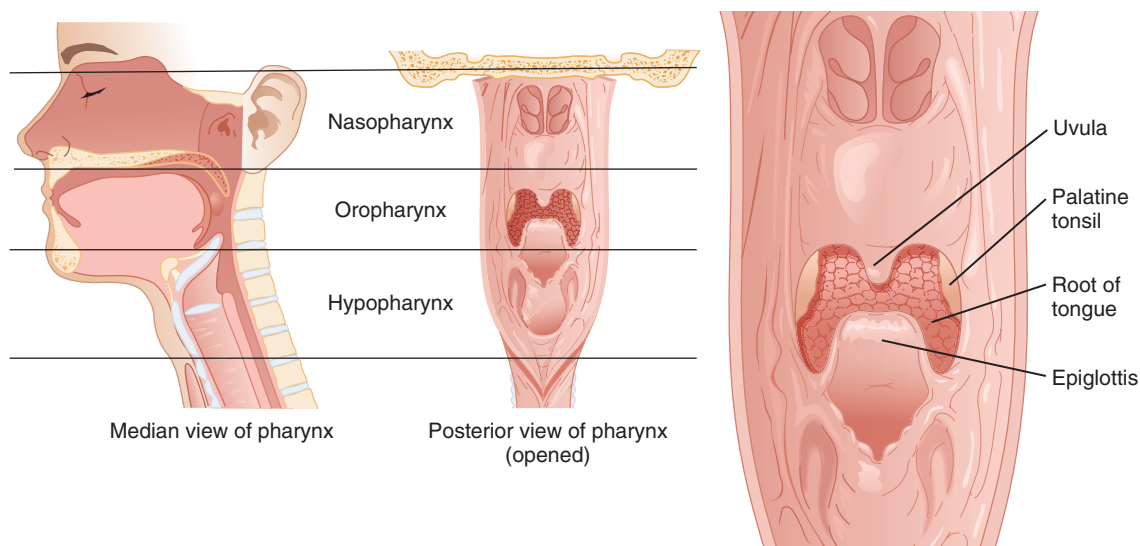


Figure 30-1. Anatomy of the nasopharynx, oropharynx, and hypopharynx.

Table 30-1 Differential Diagnosis for Sore Throat

INFECTIOUS CAUSES				
AEROBES				
VIRAL	COMMON	UNCOMMON	ANAEROBES	OTHER
Rhinovirus	<i>Streptococcus pyogenes</i> (GABHS)	<i>Haemophilus influenzae</i>	<i>Bacteroides</i> sp.	<i>Candida</i> sp.
Adenovirus	GABHS	<i>Haemophilus parainfluenzae</i>		
	<i>Peptostreptococcus</i> sp.	<i>Coccidioides</i> sp.		
Coronavirus	Non-group A streptococcus	<i>Corynebacterium diphtheriae</i>	<i>Peptococcus</i> sp.	
Herpes simplex 1, 2	<i>Neisseria gonorrhoeae</i>	<i>Streptococcus pneumoniae</i>	<i>Clostridium</i> sp.	
Influenza A, B	<i>Neisseria meningitidis</i>	<i>Yersinia enterocolitica</i>	<i>Fusobacterium</i> sp.	
Parainfluenza	<i>Mycoplasma pneumoniae</i>	<i>Treponema pallidum</i>	<i>Prevotella</i> sp.	
Cytomegalovirus	<i>Arcanobacterium hemolyticum</i>	<i>Francisella tularensis</i>		
Epstein-Barr	<i>Chlamydia trachomatis</i>	<i>Legionella pneumophila</i>		
Varicella-zoster	<i>Staphylococcus aureus</i>	<i>Mycobacterium</i> sp.		
Hepatitis virus				
NONINFECTIOUS CAUSES				
SYSTEMIC	TRAUMA, MISCELLANEOUS		TUMOR	
Kawasaki disease	Penetrating injury		Tongue	
	Angioneurotic edema			
Stevens-Johnson syndrome	Retained foreign body		Larynx	
	Anomalous aortic arch			
Cyclic neutropenia	Laryngeal fracture		Thyroid	
	Calcific retropharyngeal tendinitis			
Thyroiditis	Retropharyngeal hematoma		Leukemia	
Connective tissue disease	Caustic exposure			

GABHS, group A beta-hemolytic streptococcus.

mation or infection within Waldeyer's ring is accompanied by pain localized to the oropharynx. Pain that radiates to the back of the neck or between the shoulder blades suggests prevertebral or retropharyngeal pathology (abscess or calcific tendinitis). Sore throat with radiation to the jaw or ear may be seen with dental abscess or deeper tissue plane infection.¹²

Associated Complaints. Odynophagia is almost universal, and many viral infections can cause a raspy dysphonia (laryngitis). The presence of severe pain or significant dysphagia, drooling, voice muffling ("hot potato" voice), or difficulty

breathing, however, may indicate serious infection and potential for airway compromise. In the febrile patient, these symptoms suggest glossal abscess, severe infection of the lingual tonsils or palatine tonsils (peritonsillar cellulitis or abscess), epiglottitis, or Ludwig's angina (submental or sublingual space infection).^{3,12}

Systemic Symptoms. Prolonged fever (more than 5 to 7 days) may be seen in Kawasaki disease. Cough, myalgia, and arthralgia are seen with influenza A and B, parainfluenza, *Neisseria meningitidis*, and *Mycoplasma pneumoniae* infection. Hepatitis,

infectious mononucleosis, cytomegalovirus (CMV), and human herpesvirus 6 are associated with fatigue, malaise, and loss of appetite.^{13,14} Retroviral disease may present with similar symptoms and will often be accompanied by a rash.¹³

Epidemiology. For children, sick contacts within a daycare or school setting may provide important clues to infectious causes of sore throat. Secondary spread of disease is common for persons exposed to *M. pneumoniae*, GABHS, *Haemophilus influenzae*, *N. meningitidis*, and many viruses. Among adults, intra-familial spread is common with viruses, *Mycoplasma* sp., and GABHS. A history of recent orogenital contact may point to gonococcal or herpetic infection.

Trauma. Blunt or penetrating trauma to the oropharynx can result in deep space infections. Recent medical or dental procedures may increase the likelihood of certain infections. Potential exposures to caustics or foreign body ingestions should be sought, especially in young children.

Immunizations/Specific GABHS History. The patient's immunization status is assessed for diphtheria, pertussis, *H. influenzae*, and tetanus vaccines. The presence of previous GABHS infections should be ascertained.

Immune Status. The immune status of the patient should be assessed, including (1) the presence of diabetes, (2) known immune disorders, or (3) recent chemotherapeutic or radiation therapy. Underlying alcoholism or malnutrition may place the patient at risk for more serious infections. Recent antibiotic use may indicate the presence of resistant organisms or atypical pathogens.

Physical Examination

Assessing for airway compromise or potential airway compromise is the critical first step in the approach to the patient with sore throat (Fig. 30-2). Rapid assessment of the patient can be accomplished by observing the patient's posture, color, level of consciousness, and phonation. Observation alone is especially important in the pediatric patient with potential airway compromise because attempts at a more thorough physical examination may result in agitation and progression to complete airway obstruction. The presence of air hunger, stridor, drooling, or toxic appearance may indicate pending airway obstruction.^{3,4} A complete ear, nose, and throat (ENT) and general examination will help narrow the differential diagnosis (Table 30-2).

A reduced functional caliber of the airway may occur acutely, subacutely, or insidiously, depending on the cause of the disease process.^{10,11,15,16} Pending airway loss leads to air-preserving posturing especially in children. Infants unable to sit without support choose the lateral decubitus position with the neck hyperextended when partial obstruction occurs. Children capable of sitting may support their heads with their hands. Airway obstruction in an older child is typically associated with fixed upright posturing. The patient has forced flexion at the waist and maintains the neck flexed and the head extended with an open mouth. Alternatively, patients may assume tripod posturing, in which additional support is gained by hands held on a surface.

Ancillary Testing

Laboratory procedures, other than rapid group A strep testing or throat culture, generally are not necessary to develop a working diagnosis of viral pharyngitis or GABHS pharyngitis (Table 30-3). Use of the Centor criteria, with or without rapid antigen detection test or culture, is a rational but not universally accepted approach.^{8,17-19} Patients with a score of 0 or 1 do not require treatment or additional testing. The goal is to

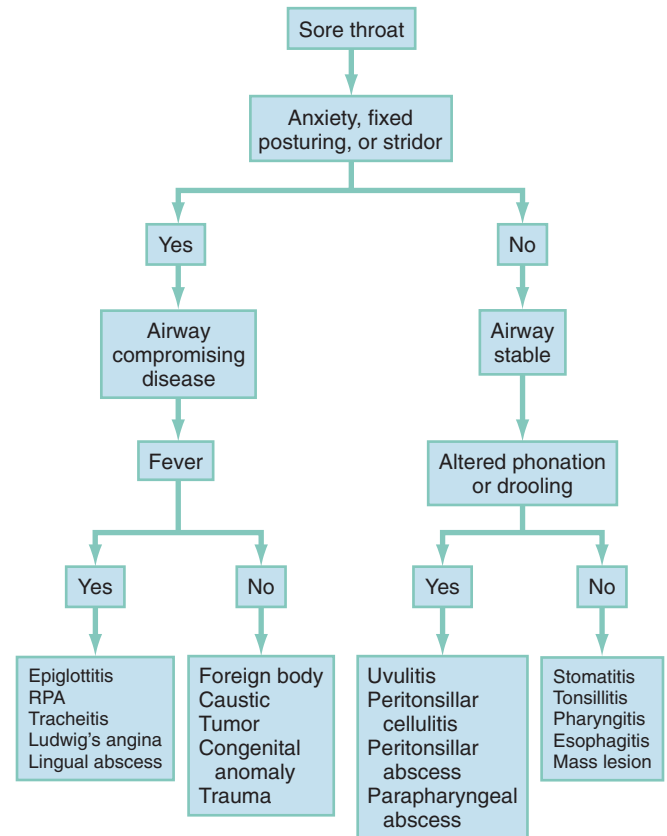


Figure 30-2. Diagnostic algorithm for the patient with a sore throat. RPA, retropharyngeal abscess.

decrease the cost of additional testing and decrease the inappropriate use of antibiotics while still treating those with GABHS to prevent suppurative and nonsuppurative complications.

A complete blood count is rarely helpful but may be used, along with serologic test for EBV, for the patient with a compatible presentation—severe sore throat, fever, and lymphadenopathy. Hematologic findings of leukocytosis, relative and absolute lymphocyte predominance, and the presence of atypical lymphocytes constituting more than 10% of the total leukocyte count suggest EBV. A serologic test such as the heterophile antibody screen (Monospot) may provide evidence of primary EBV infection.¹³ Patients with a negative serologic test but with compatible symptom complex should be retested a week later because heterophile antibodies may not be present in the first week in 10% of patients.^{13,14} In addition, CMV, acute retroviral illness, herpes simplex virus, and human herpes 6 viral infections should be considered.

A lateral portable upright neck radiograph may be used in the pediatric patient to narrow the differential diagnosis of infectious conditions associated with potential airway obstruction. The lateral neck film may demonstrate swelling in the prevertebral soft tissue in a patient with a retropharyngeal abscess (RPA).⁴ Plain radiographic imaging is rarely warranted in the adult patient with an acute sore throat. The adult with severe symptoms should be considered for direct nasopharyngoscopy to search for epiglottitis. Use of the *H. influenzae* b vaccine in children has resulted in a dramatic decrease in the incidence of epiglottitis, but the incidence in adults has not changed.^{10,11,16,20} Ultrasonography may be a useful tool in the diagnosis and treatment of some deep space infections. The advantages of ultrasound are as follows: (1) It can be used at the bedside, (2) it can be used to guide incision and drainage

Table 30-2 Pivotal Findings in Physical Examination

SIGN	FINDING	DIAGNOSES
Appearance	Toxic	Epiglottitis RPA Bacterial tracheitis Kawasaki disease
Posturing	Fixed, upright, leaning forward	Epiglottitis RPA Tracheitis Laryngotracheobronchitis Parapharyngeal abscess
Phonation	Torticollis Absent Muffled	Epiglottitis RPA Peritonsillar cellulitis Peritonsillar abscess
Stridor, drool	Either present	Epiglottitis RPA Tracheitis Peritonsillar abscess
Noninvasive ENT	Conjunctivitis	Kawasaki disease Stevens-Johnson syndrome Adenovirus
	Mucous membrane sore	Stevens-Johnson syndrome Behçet disease Enterovirus Herpes simplex Ludwig's angina Adenovirus
	Submental, sublingual mass Adenopathy	EBV <i>Mycobacterium</i> sp. HIV
	Tender hyoid Tender thyroid	Epiglottitis Thyroiditis Thyroglossal duct cyst infection
Augmented ENT findings	Trismus	Parapharyngeal abscess Peritonsillar abscess
	Tongue coating	Kawasaki disease GABHS
	Palatal petechiae	GABHS
	Pharyngeal hyperemia	Infectious tonsillopharyngitis Caustic Trauma GABHS <i>Corynebacterium diphtheriae</i> <i>Fusobacterium</i> sp. EBV Adenovirus
	Exudative tonsillitis	RPA Uvulitis Peritonsillar abscess Parapharyngeal abscess
	Bulged retropharynx Uvular erythema Displaced uvula	Epiglottitis
Abdomen	Inflamed epiglottis	EBV, hepatitis
Joint examination	Hepatosplenomegaly	Lemierre's syndrome
Rash	Arthritis	GABHS <i>Arcanobacterium</i> sp. EBV Kawasaki disease
	Scarlatiniform	

EBV, Epstein-Barr virus; ENT, ear, nose, throat; GABHS, group A beta-hemolytic streptococcus; HIV, human immunodeficiency virus; RPA, retropharyngeal abscess.

of peritonsillar abscesses (PTAs), and (3) it decreases the exposure to ionizing radiation in computed tomography (CT) scans.⁴ CT scanning defines the extent of infection and is superior to ultrasonography for this purpose. It can also help distinguish cellulitis from abscess.^{4,21} Magnetic resonance imaging provides superior resolution of deep tissue planes and may supplant the use of CT scanning in the future.

Table 30-3

Centers for Disease Control and Prevention: Practice Guidelines for Acute Pharyngitis in Adults

Population: Adults (patients older than 15 years)
Patients with viral symptoms: Do not test or treat
Patients with symptoms of GABHS: Use Centor criteria*
 Centor score = 4: Perform RADT or treat presumptively
 Centor score = 3: Perform RADT or treat presumptively
 Centor score = 2: Perform RADT or do not test or treat
 Centor score = 1 or 0: Do not test or treat
 In all cases in which an RADT is performed, only those with positive results are treated.
Culture after negative RADT: No
Recommended antibiotic: Penicillin (erythromycin if penicillin allergic)

*Centor criteria history of fever; absence of cough; swollen, tender anterior cervical lymph nodes; and tonsillar exudate.
 GABHS, group A beta-hemolytic streptococcus; RADT, rapid antigen detection test.

■ DIFFERENTIAL DIAGNOSIS

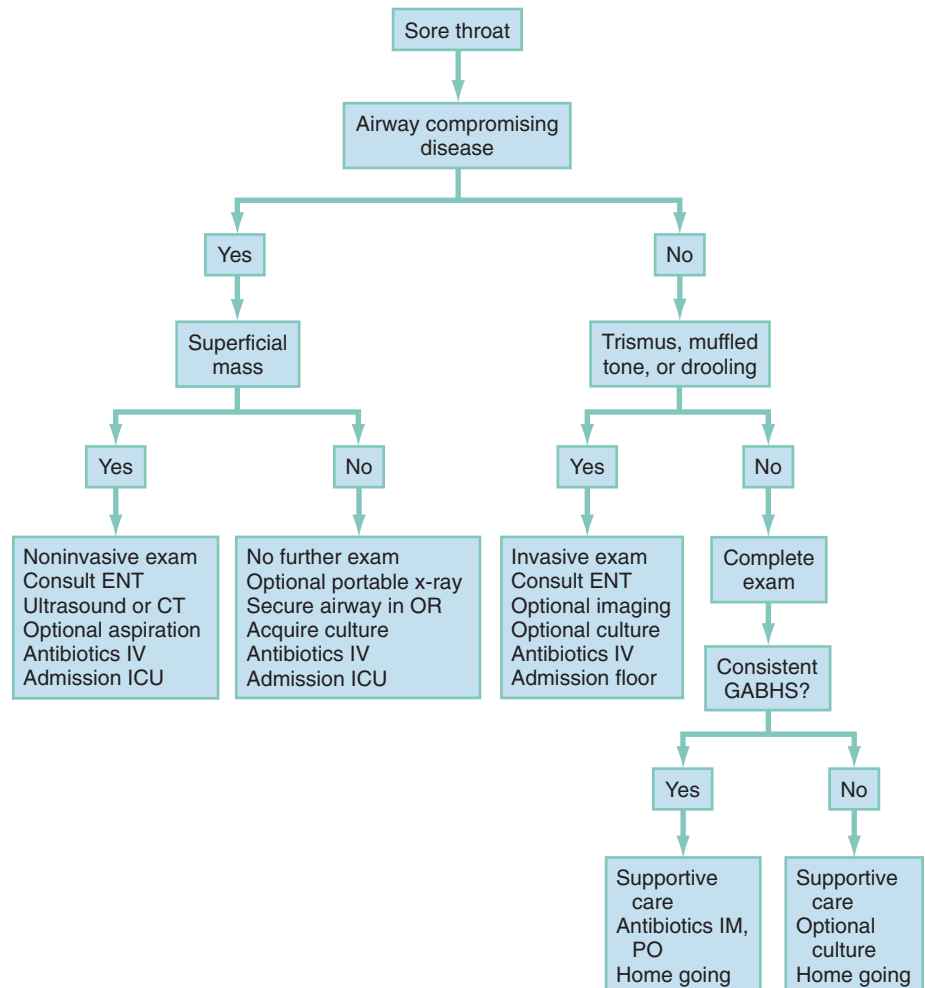
Table 30-1 lists the infectious and noninfectious causes of sore throat.

■ EMPIRICAL MANAGEMENT

The management of the patient presenting with a sore throat begins with a rapid assessment for potential airway compromise (Figs. 30-2 and 30-3). If the patient is in extremis, immediate airway control is obviously necessary. If the airway is patent and ventilation is adequate, diagnostic and therapeutic efforts may simultaneously commence. Infections within the parotid, buccal, parapharyngeal, submental, and sublingual spaces create masses that are readily apparent.^{3,12,21,22} The purulent material rapidly expands the tissues but rarely occludes the airway. A thorough head and neck examination accompanied by fiberoptic nasopharyngoscopy, ultrasonography, or CT scan may be necessary to identify the severity and extent of the process. Needle aspiration of a PTA can be both diagnostic and therapeutic. Intravenous antibiotics are administered to treat mixed infection with aerobic and anaerobic organisms. ENT consultation is often necessary for definitive management of PTA, RPA, and other infectious or mass lesions, and early consultation is often warranted.

The patient who is febrile and appears toxic, is in respiratory distress, has an abnormal voice or prefers not to speak, or is drooling through a persistently open mouth may require emergent airway management before any other diagnostic

Figure 30-3. Management algorithm for the patient with a sore throat. CT, computed tomography, ENT, ear, nose, and throat; GABHS, group A beta-hemolytic streptococcus.



maneuvers are attempted due to impending airway compromise. If time permits, immediate transfer to the operating room may be warranted, with ENT and anesthesiology consultation. This requires that the operating room be ready to receive the patient and the patient be accompanied by a physician or surgeon capable of surgical cricothyrotomy. If the patient cannot be transported, then fiberoptic intubation (nasal or oral) is the preferred route, with light sedation and topical anesthesia (see Chapter 1). Equipment for cricothyrotomy should be readily available because instrumentation can lead to airway obstruction or laryngospasm. After the airway is secured, the infected surface and secretions can be swabbed for culture; tissue aspiration and blood culture specimens can be submitted for culturing.¹⁵ Broad-spectrum parenteral antibiotics are begun for mixed aerobic and anaerobic infection, and the patient should be admitted to the intensive care unit.

If the febrile patient does not have evidence of airway compromise but has vocal changes (e.g., muffled or “hot potato” voice), epiglottitis, peritonsillar cellulitis, or abscess may be present. If examination of the oropharynx does not identify

the offending condition, then fiberoptic examination of the upper airway for epiglottitis is indicated. ENT need not be consulted for peritonsillar cellulitis or uvulitis. ENT may be consulted for peritonsillar abscess, even after needle aspiration, for incision and drainage. Intravenous antibiotics are provided to cover *Streptococcus pyogenes*, non-group A streptococci, and *Staphylococcus aureus*. The patient may require admission for further care if he or she has severe symptoms or is unable to tolerate liquids by mouth.

In the patient with a sore throat who has no evidence of airway compromise, the pain may be a problem within the oropharynx, referred from another location, or part of a systemic illness. Further workup may continue in the ED or on an outpatient basis. The patient with presumed or proven GABHS should be treated with antibiotics. Penicillin remains the drug of choice.^{7-9,23} Details of treatment are provided in Chapter 73.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.

■ PERSPECTIVE

Epidemiology

Hemoptysis is defined as the expectoration of blood from the respiratory tract that originates below the vocal cords. Most cases are mild and consist of blood-tinged sputum or minute amounts of frank blood. The most common cause of small-volume hemoptysis is bronchitis. Rarely, hemoptysis is accompanied by massive blood loss, generally accepted as 100 to 600 mL of blood loss in any 24-hour period. In addition to manifesting as hemoptysis, endobronchial bleeding may impair alveolar oxygen exchange and cause significant morbidity and mortality. Rapid blood loss can also result in hemodynamic instability and shock.

Although hemoptysis is a common complaint in emergency populations, only 1 to 5% of hemoptysis patients have massive or life-threatening hemorrhage, with mortality rates approaching 80%.¹ Large, contemporary series of patients with massive hemoptysis are lacking. Most etiologic data originate from small, often rural studies where tuberculosis (TB) and bronchiectasis are responsible for the vast majority of cases.^{2,3} In developed nations, cancer, cystic fibrosis, arteriovenous malformations, and postprocedural complications play a more prominent role. Pediatric hemoptysis is rare but can be caused by infection, congenital heart disease, cystic fibrosis, or bleeding from a preexisting tracheostomy.⁴

Pathophysiology

Trace hemoptysis typically originates from tracheobronchial capillaries that become disrupted with vigorous coughing or minor bronchial infections. Massive hemoptysis almost exclusively involves one of the two sets of vessels that constitute the lung's dual blood supply. Bronchial arteries, direct branches from the thoracic aorta, are responsible for supplying oxygenated blood to lung parenchyma. Disruption of these vessels from arteritis, trauma, or bronchiectasis or erosion from an adjacent malignancy can result in sudden and profound hemorrhage. Although small in caliber, the bronchial circulation is a high-pressure system and the culprit in nearly 90% of the cases of massive hemoptysis requiring embolization. Pulmonary arteries, although transmitting large volumes of blood, are at much lower pressure and, unless affected at a very central location, are less likely to cause massive hemoptysis.

Nearly all causes of hemoptysis have a common mechanism—vascular disruption within the trachea, bronchi, small-

caliber airways, or lung parenchyma. Modes of vessel injury include acute and chronic inflammation (from bronchitis and arteritis), local infection (especially lung abscesses, TB, and aspergillosis), trauma, invasion from a growing tumor, infarction following a pulmonary embolus and fistula formation (specifically aortobronchial fistulae).

Bronchiectasis, a chronic necrotizing infection resulting in bronchial wall inflammation and dilation, is one of the most common causes of massive hemoptysis. As tissue destruction and remodeling continue, rupture of nearby bronchial vessels results in bleeding. Bronchiectasis can complicate chronic airway obstruction, necrotizing pneumonia, TB, or cystic fibrosis. Broncholithiasis, the formation of calcified endobronchial lesions following a wide array of granulomatous infections, is an uncommon problem with a propensity to erode nearby vessels. Hemorrhage control often requires surgical intervention.⁵⁻⁷

Iatrogenic hemoptysis may complicate 2 to 10% of all endobronchial procedures, especially percutaneous lung biopsies.^{8,9} Additionally, bleeding can be exacerbated by coagulopathy and thrombocytopenia. An uncommon cause includes ectopic endometrial tissue within the lung that can result in monthly catamenial episodes of hemoptysis. Diffuse alveolar hemorrhage can be seen with autoimmune vasculitides such as Wegener's granulomatosis, systemic lupus erythematosus, and Goodpasture's syndrome. Still others include pulmonary hereditary telangiectasias and hydatiform infections.

■ DIAGNOSTIC APPROACH

Differential Considerations

When a patient presents with apparent hemoptysis, two other potential sources of bleeding should be investigated. Nasal, oral, or hypopharyngeal bleeding sometimes inadvertently contaminates the tracheobronchial tree and can mimic true hemoptysis. The clinician should closely inspect the nasopharynx and oral cavity to exclude this possibility. Differentiating hemoptysis from a gastric or proximal duodenal source of bleeding is the principle diagnostic dilemma, since further evaluation and management follow divergent pathways. Usually, this can be done by the patient and physician discriminating coughing from vomiting. In unclear cases, inspection and pH testing may help to distinguish gastrointestinal from tracheobronchial hemorrhage. Unless an active, brisk upper GI hemorrhage is present, the acidification of blood in the stomach results in fragmentation and

darkening of its color. This produces specks of brown or black material often referred to as “coffee-grounds” emesis. Pulmonary blood appears bright red or as slightly darker clots and is alkaline.

Rapid Assessment and Stabilization

Although hemodynamic instability can occur as a result of hemorrhage, the most lethal sequela of massive hemoptysis is hypoxia resulting from the ventilation-perfusion mismatch that occurs as small airways and alveoli are submerged with blood. The clinician should consider the standard indications for emergency airway management in such cases.

As a mitigating maneuver in patients with a known lateralizing source of bleeding, the “lung down” position can be employed in which the patient is positioned so the bleeding lung is more dependent. This position can promote continued protection and ventilation of the unaffected lung and improve oxygenation.^{10,11} Large-bore (8.0) endotracheal tubes should be used to facilitate emergent fiberoptic bronchoscopy. In selected cases of confirmed left-sided bleeding, a single-lumen right-mainstem intubation can be successfully performed by advancing the tube in either the neutral position or by using a 90° rotational technique.¹² Left-mainstem intubations are more difficult and should be attempted with caution.

The use of double-lumen endotracheal tubes for lung isolation should be reserved for dire circumstances and usually requires an experienced anesthetist. The correct positioning of blindly placed double-lumen tubes is difficult and requires confirmation by auscultation and fiberoptic bronchoscopy, both of which have severely impaired accuracy in massive hemoptysis. Complications of double-lumen tubes include unilateral and bilateral pneumothorax, pneumomediastinum, carinal rupture, lobar collapse, and tube malposition.¹³

Pivotal Findings

History

Although patient reports of bleeding severity are historically inaccurate, a rough estimate of the rate, volume, and appearance of expectorated blood should be obtained.

Any history of parenchymal pulmonary disorders should be obtained, including the presence of bronchiectasis, recurrent pneumonia, chronic obstructive pulmonary disease, bronchitis, TB, and fungal infection. Inflammatory disorders that secondarily involve the lungs or pulmonary vasculature include Wegener’s granulomatosis, Goodpasture’s syndrome, and systemic lupus erythematosus. Risk factors for platelet dysfunction, thrombocytopenia, and coagulopathy may be present. Hypercoagulable states can contribute to deep venous thrombi and pulmonary embolism.

The presence of primary or metastatic cancer can cause hemoptysis by erosion into pulmonary and bronchial vessels. Recent percutaneous or transbronchial procedures can cause immediate or delayed postprocedural bleeding, and any recent history of trauma should also be noted. A pertinent travel history to areas endemic with TB or pulmonary paragonimiasis is crucial.

Physical Examination

After a primary survey and stabilization, a targeted examination may suggest the location and etiology of bleeding, but does so in less than 50% of cases.¹⁴ Focal adventitious breath sounds may indicate pneumonia or pulmonary abscess. A new heart murmur, especially in a febrile patient, might reflect

endocarditis causing septic pulmonary emboli. Symptoms and signs of deep venous thrombosis should suggest pulmonary embolism. Ecchymoses and petechiae can indicate coagulopathy and thrombocytopenia, respectively.

Ancillary Testing

Initial laboratory studies include a complete blood count, coagulation tests, and a type and screen or crossmatch. Renal function tests should be obtained if vasculitis is suggested or contrast computed tomography (CT) is planned. Plain chest radiography should be ordered, although its sensitivity is marginal. A prospective study of 184 consecutive patients with varying degrees of hemoptysis reveals that more than 40% of patients with a normal chest radiograph have a positive chest CT.¹⁵

In patients with massive hemoptysis, plain films may localize the site of hemorrhage in as many as 80% of patients.⁶ High-resolution multidetector CT of the chest is the principle diagnostic test for investigating both bronchial and non-bronchial arterial causes of massive hemoptysis. CT is diagnostically comparable, yet less invasive, than conventional angiography, which is now done primarily as a combined diagnostic-therapeutic modality.¹⁶⁻¹⁸ A chest CT should be obtained in the high risk patient (smokers, oncology patients) or in any patient with moderate to severe bleeding even if the initial chest radiography is normal. CT localization of hemorrhage can expedite bronchoscopic evaluation or guide subsequent interventional procedures.

■ DIFFERENTIAL DIAGNOSIS

Potential causes of hemoptysis vary and include systemic illnesses as well as pulmonary parenchymal disease. **Box 31-1** includes the most common causes.

■ MANAGEMENT

Since the advent of high-resolution CT, radiologic evaluation has had an integral role in the evaluation and treatment of patients with hemoptysis. The challenge to the emergency physician is to rapidly assess the need for airway control prior to hemodynamic stabilization. Unless the initial chest radiograph is diagnostic or the patient is hemodynamically unstable, a chest CT should be obtained in most cases. Further management strategy should be developed in conjunction with pulmonary and thoracic surgery consultants, guided by the CT results (**Fig. 31-1**).

Bronchoscopy

Early bronchoscopy facilitates both localization of bleeding and therapeutic intervention. Balloon and topical hemostatic tamponade, thermocoagulation, and injection of vasoactive agents can all control arterial bleeding. Optimal timing for bronchoscopy remains conjectural. Stable patients with mild to moderate bleeding may benefit from early bronchoscopy. In unstable patients or those with brisk hemorrhaging, bronchoscopy sometimes can facilitate airway management, but is less likely to control bleeding.

Chest CT is as diagnostically accurate as bronchoscopy in locating peripheral vessels not accessible by a flexible bronchoscope.¹⁹ Chest CT is used to identify the bleeding site and to determine whether angiography is indicated. There may be little benefit to bronchoscopy prior to interventional angiography if a CT scan has accurately identified a bleeding source.²⁰

BOX 31-1 DIFFERENTIAL DIAGNOSIS: HEMOPTYSIS**Airway Disease**

Bronchitis (acute or chronic)
Bronchiectasis
Neoplasm (primary and metastatic)
Trauma
Foreign body

Parenchymal Disease

Tuberculosis
Pneumonia/lung abscess
Fungal infection
Neoplasm

Vascular Disease

Pulmonary embolism
Arteriovenous malformation
Aortic aneurysm
Pulmonary hypertension
Vasculitis (Wegener's granulomatosis, SLE, Goodpasture's syndrome)

Hematologic Disease

Coagulopathy (cirrhosis or warfarin therapy)
Disseminated intravascular coagulation
Platelet dysfunction
Thrombocytopenia

Cardiac Disease

Congenital heart disease (especially in children)
Valvular heart disease
Endocarditis

Miscellaneous

Cocaine
Post-procedural injury
Tracheal-arterial fistula

SLE, systemic lupus erythematosus.

Interventional Angiography

Bronchial arterial embolization is an effective first-line therapy and is the procedure of choice for patients unable to tolerate surgery, or those in whom bronchoscopy was unsuccessful. Hemostatic rates range from 91 to 98%, but as many as 20 to 50% of patients have early episodes of repeat bleeding. The risk of delayed bleeding may exist for up to 36 months.²¹⁻²⁴ In order to guide therapy, initial localization of bleeding by bronchoscopy or CT is preferred. Rare complications include arterial perforation and dissection.

Surgery

Emergency thoracotomy is reserved for life-threatening hemoptysis or for persistent, rapid bleeding that is uncontrolled by bronchoscopy and percutaneous embolization. Pulmonary arterial hemorrhage from tumor necrosis represents a surgical emergency.²⁵

DISPOSITION

Healthy patients with blood-streaked sputum and normal vital signs do not require imaging beyond plain chest radiography

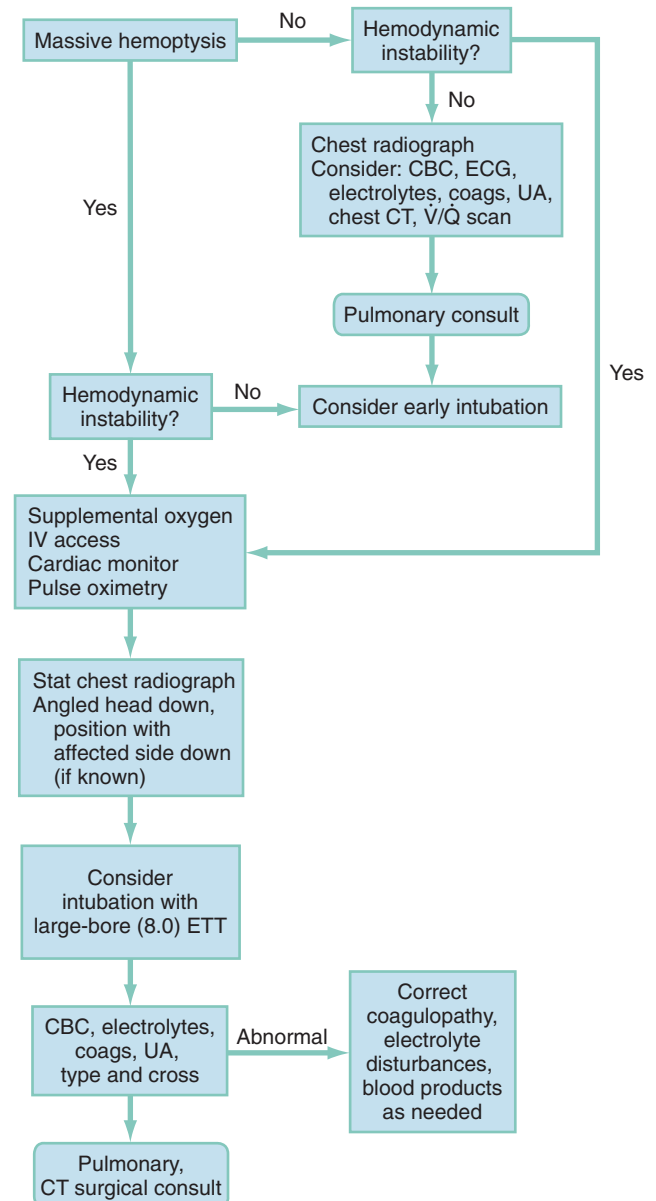


Figure 31-1. Emergency department management of hemoptysis. CBC, complete blood count; coags, coagulation studies; CT, computed tomography; ECG, electrocardiogram; ETT, endotracheal tube; IV, intravenous; UA, urinalysis; V/Q, ventilation-perfusion.

and can be discharged with follow-up. High risk patients with minor hemoptysis and all patients with moderate or large amounts of hemoptysis should undergo plain chest radiography followed by emergent chest CT. Brief hospitalization or admission to an observation unit for bronchoscopy should be considered. All patients with massive hemoptysis require admission to an intensive care unit and expedited multidisciplinary treatment involving the emergency physician, pulmonologist, and thoracic surgeon.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.

Red and Painful Eye

Joshua L. Wright and John M. Wightman

PERSPECTIVE

Epidemiology and Pathophysiology

Most eye complaints are not immediately sight-threatening and can be managed by an emergency physician. Nontraumatic diseases, such as glaucoma and peripheral vascular disease leading to retinal ischemia, are more common with advancing age. Ocular injuries are the leading cause of visual impairment and blindness in the United States.¹ More patients with postoperative complications can be expected to present to the emergency department as more vision correction surgeries are performed.

The external and internal anatomy of the eye is depicted in [Figure 32-1A and B](#). The globe has a complex layer of blood vessels in the conjunctiva, sclera, and retina. Redness reflects vascular dilation and may occur with processes that produce inflammation of the eye or surrounding tissues. Eye pain may originate from the cornea, conjunctiva, iris, or vasculature. Each is sensitive to processes causing irritation or inflammation.

DIAGNOSTIC APPROACH

Rapid and accurate triage is the most critical consideration in the approach to the red and painful eye. The first question should be, “Did anything get in your eye?” If so, the second question should be, “What do you think it is?” This helps separate trauma from nontrauma, but, more importantly, seeks to identify quickly eyes that may have been exposed to a caustic substance. Patients exposed to caustic substances require rapid decontamination to prevent permanent loss of visual acuity.

Differential Considerations

Diagnoses are classically divided into traumatic and nontraumatic. Traumatic pain and redness can be caused by caustic fluids and solid materials, low-velocity contact with a host of materials that can fall or be rubbed into the eye, higher velocity blunt-force impacts to the orbit or globe, or potentially penetrating injuries. Causes of nontraumatic pain and redness require a more detailed history, including contact lens use and questions directed toward determining the likelihood of systemic illnesses.

Pivotal Findings

Measurement of the patient’s best corrected visual acuity (i.e., with glasses on, if available) with each eye individually and with both eyes provides vital information when evaluating eye complaints. Only a few situations preclude early and accurate visual acuity testing. Eyes exposed to caustic materials should be decontaminated as soon as possible. Patients with sudden and complete visual loss in one eye require prompt funduscopic examination to determine the possibility of acute central retinal artery occlusion. This condition is readily apparent as a diffusely pale retina with indistinct or unseen retinal arteries ([Fig. 32-2](#)).

Other pivotal findings, which are more likely to be associated with a serious diagnosis, in patients with a red or painful eye are listed in [Box 32-1](#).

History

Chief complaints of pain can be manifestations of a variety of sensations. When carefully questioned, some patients may differentiate between itching, burning, dull pain, sharp pain, and perception of a foreign body. Itching tends to be more often due to blepharitis, conjunctivitis, or dry eye syndrome. Burning is associated with these conditions and with other mostly extraocular problems such as irritation of a pterygium or pinguecula, episcleritis, or limbic keratoconjunctivitis. Dull pain may be a manifestation of increased intraocular pressure (IOP) or referred from an extraorbital process such as sinusitis, migraine headache, or temporal arteritis. Sharp pain generally results from abnormalities of the anterior eye, such as keratitis, uveitis, and acute angle-closure glaucoma. A foreign body sensation is more typical of corneal irritation or inflammation.

A chief complaint of redness commonly results from palpebral or limbal injection of the conjunctiva. However, free blood can be noted behind the bulbar conjunctiva (i.e., subconjunctival hemorrhage) or in the anterior chamber (i.e., hyphema). Both of these can be spontaneous or post-traumatic. Spontaneous subconjunctival hemorrhages may follow coughing or straining or may be due to systemic hypertension. Often, it occurs without any identifiable precipitating incident and is simply noticed by the patient when looking in the mirror. Spontaneous subconjunctival hemorrhage is painless, and the presence of pain raises concern for a more serious cause of the hemorrhage, such as direct globe injury. Hyphema of

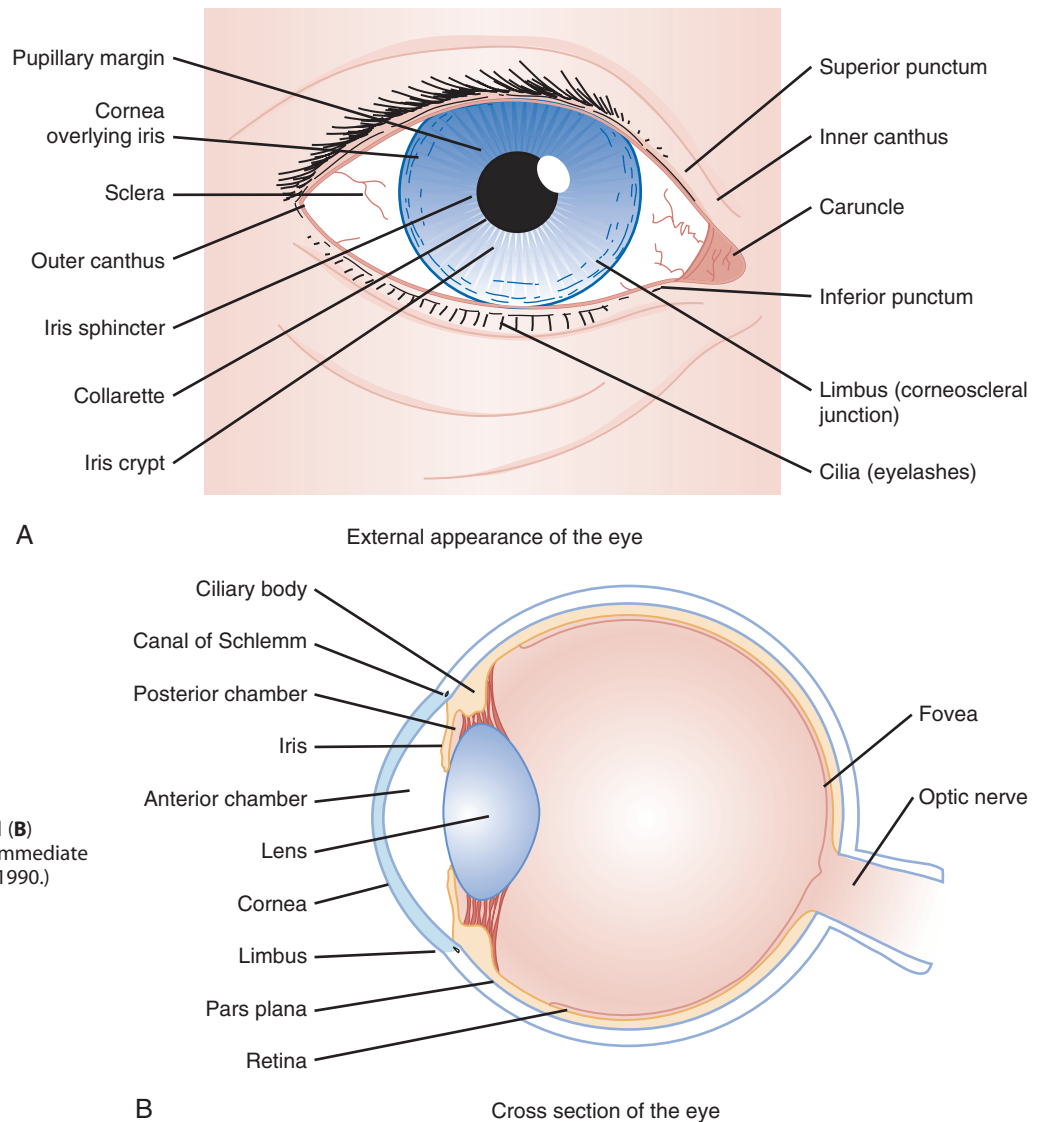


Figure 32-1. External (A) and internal (B) anatomy. (From Ragge NK, Easty DL: Immediate Eye Care. St. Louis, Mosby-Year Book, 1990.)

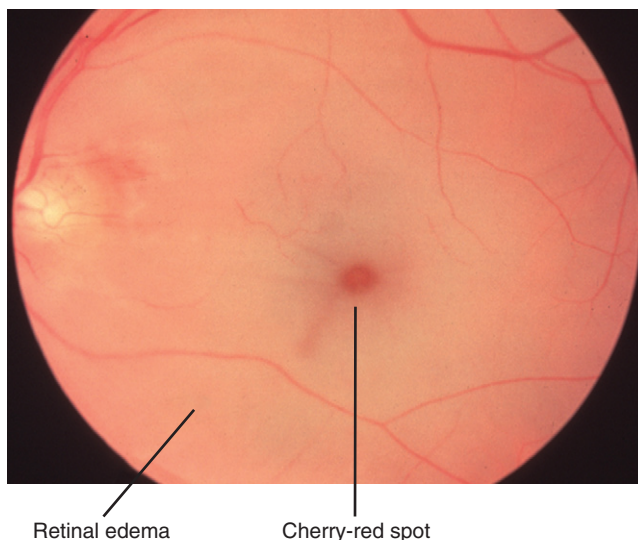


Figure 32-2. Key funduscopic findings in acute central retinal artery occlusion include general pallor of the retina (except for a characteristic cherry-red spot where the perfused choroid shows through the thinner fovea) and attenuation of retinal arteries (possibly with retinal veins preserved as in the photograph). (From Kaiser PK, Friedman NJ, Pineda R, II: The Massachusetts Eye and Ear Infirmary Illustrated Manual of Ophthalmology, 2nd ed. Philadelphia, WB Saunders, 2004, p 297.)

BOX 32-1

PIVOTAL FINDINGS MORE LIKELY ASSOCIATED WITH A SERIOUS DIAGNOSIS IN PATIENTS WITH A RED OR PAINFUL EYE

- Severe ocular pain
- Persistently blurred vision
- Proptosis
- Reduced ocular light reflection
- Corneal epithelial defect or opacity
- Limbal injection (i.e., ciliary flush)
- Pupil unreactive to a direct light stimulus
- Wearer of soft contact lenses
- Neonate
- Immunocompromised host
- Worsening signs after 3 days of pharmacologic treatment

Adapted and reprinted, with permission, from Trobe JD: The Physician's Guide to Eye Care. San Francisco, Foundation of the American Academy of Ophthalmology, 2001.

sufficient size to be noted by the patient or bystander usually arises with pain and blurred vision.

Other subjective findings may be transient and detected only by history. The patient may relate lid swelling, tearing, discharge, crusting, or sensitivity to light. Lid swelling can be caused by inflammatory and noninflammatory processes. Concurrent erythema of the lid favors the former. In the absence of trauma or other external irritant (e.g., contact dermatitis), inflammatory processes include primary lid problems such as hordeolum (i.e., sty) or blepharitis as well as extension from concomitant conjunctivitis or cellulitis in orbital or periorbital structures. When pain is present, tearing is usually secondary. Discharge and crusting are most commonly associated with conjunctivitis, whether allergic, viral, or bacterial. Blepharitis, dacryocystitis, and canaliculitis are other inflammatory processes that may create a discharge and subsequent crusting.

Other eye status review questions include the following:

- Are contact lenses used? If so, what type, how are they cleaned, and how old are the lenses? Has there been a change in the pattern of use (especially increased use)? Were the lenses worn for a particularly long period recently? Are there problems with the lenses drying out? Does insertion of the lenses worsen or relieve the symptoms?
- Are glasses worn? If so, when was the last assessment for adequate refraction?
- Has previous eye surgery or injury occurred?
- What is the patient's usual state of health?
- What medications are being taken? Are there any allergies, including environmental allergies?

Physical Examination

A complete eye examination usually includes eight components, although many patients require only a limited or directed eye examination, depending on the presentation.² The mnemonic VVEPP (pronounced “veep”) plus slit-lamp and funduscopic examinations represent these components (Box 32-2).³ Slit-lamp examination is recommended for any complaint involving trauma and for any medical presentation involving foreign body sensation or alteration of vision.

BOX 32-2 COMPLETE EYE EXAMINATION

Visual acuity (best possible using correction)
 Visual fields (tested by confrontation)
 External examination
 Globe position in orbit
 Conjugate gaze
 Periorbital soft tissues, bones, and sensation
 Extraocular muscle movement
 Pupillary evaluation (absolute and relative)
 Pressure determination (tonometry)
 Slit-lamp examination
 Lids and lashes
 Conjunctiva and sclera
 Cornea (with fluorescein in some cases)
 Anterior chamber
 Iris
 Lens
 Funduscopic examination

Adapted from Wightman JM, Hurley LD: Emergency department management of eye injuries. *Crit Decis Emerg Med* 12:1, 1998.

Funduscopic examination is usually pursued if there is visual loss, visual alteration, or suggestion of serious pathology in the history and initial physical examination. A thorough physical examination can be conducted in the following order.

Visual Acuity

The initial determination of a patient's visual acuity provides a baseline from which deterioration or improvement may be followed. It is also predictive of functional outcome after ocular trauma. Visual acuity is quantitatively assessed by use of a Snellen chart test at a distance of 20 feet (6 m) or a Rosenbaum chart at a distance of 14 inches. Young patients who cannot yet read letters and numbers should be tested with an Allen chart that depicts easily recognizable shapes. Each eye is tested separately with the opposite eye carefully covered. Patients who present without their prescribed corrective lenses may be evaluated by having them view the chart through a pinhole eye cover, which negates most refractive errors in vision.

If the patient cannot distinguish letters or shapes on a chart, visual acuity must be determined qualitatively. Any printed material suffices. The result may be recorded as, for example, “patient able to read newsprint at 3 feet.” If this is not possible, visual acuity is recorded as:

- Unable/able to count fingers (CF)
- Unable/able to perceive hand motion (HM)
- Unable/able to perceive light (LP)

Visual Field Testing

Confrontation is the most common method of testing visual fields in the emergency department.⁴ Detection of a scotoma usually represents a retinal problem. However, glaucoma may cause scotomata that can be crescent-shaped, involve just the binasal visual fields, or affect all peripheral vision. Hemis- or quadrantanopia is more commonly a problem of the neural pathways to the brain.

External Examination

Gross abnormalities are assessed by a visual inspection of both eyes simultaneously. Findings may be more apparent if compared with the opposite side. Fractures of facial bones are associated with ocular injuries, some of which require immediate intervention by an ophthalmologist.⁵

Globe position is part of the external examination. Subtle exophthalmos and enophthalmos are rare, and are best detected by looking inferiorly, tangentially across the forehead, from over the patient's scalp.⁶ Exophthalmos may have traumatic or nontraumatic causes, but is due to increased pressure or a space-occupying lesion within the orbit, which may manifest as pain. Medical causes include cellulitis or intraorbital or lacrimal tumors. Hyperthyroidism may cause enlargement of extraocular muscles. The most important cause of exophthalmos in the emergency department is retrobulbar hematoma, a condition characterized by hemorrhage within the bony orbit, behind the globe. Orbital compartment syndrome pushes the globe forward, stretching the optic nerve and retinal artery and increasing IOP. The resulting microvascular ischemia is sight-threatening if sufficiently severe and persistent. Orbital emphysema and inflammation caused by a retained foreign body behind the eye are other causes of exophthalmos. The discovery of exophthalmos should prompt ocular tonometry measurements to determine the urgency of intervention. Trauma, particularly penetrating globe injury with extrusion

of vitreous, can cause the globe to recede into the orbit, but the most common cause of enophthalmos is actually pseudoenophthalmos when the contralateral globe is proptotic.

Inspection also involves examination of the upper and lower palpebral sulci for foreign bodies or other abnormalities. The lower sulcus is easily viewed after manual retraction of the lower lid toward the cheek and having the patient gaze upward. The upper sulcus is inspected by pulling its lashes directly forward and looking under the lid with white light. The lid can then be everted by pressing a cotton-tipped applicator in the external lid crease and folding the lid margin over the applicator.

Extraocular Muscle Function

Limitation of ocular movement in one eye may be detected by having the patient follow the examiner's finger or a bright light through the cardinal movements of gaze. The eyes may move in a disconjugate fashion, or the patient may admit to diplopia if asked. Diplopia on extreme gaze in one direction may indicate entrapment of one of the extraocular muscles within a fracture site, but more often is caused simply by edema or hemorrhage related to the injury and is functional rather than actual entrapment. In the absence of trauma, diplopia is rarely associated with redness or pain.

Pupillary Evaluation

The pupils are inspected for abnormalities of shape, size, and reactivity. These examinations are conducted with light specifically directed into the pupil and by means of the swinging flashlight test.

Previous surgery (e.g., iridotomy for cataract extraction) and synechiae from prior iritis or other inflammatory condition are the most common causes of irregularly shaped pupils. Asymmetrically sized pupils may represent normal or pathologic conditions. Physiologic anisocoria is a slight difference in pupil size that occurs in up to 10% of the population. Topical or systemic medications, drugs, and toxins may cause abnormal pupillary constriction or dilation.

Pathologic reasons for failure of one pupil to constrict with a direct light stimulus include globe injury, abnormalities of afferent or efferent nerves, and paralysis of the ciliaris or sphincter pupillae muscles in the iris. Potentially serious problems, which also cause pain and redness, include uveitis and acute angle-closure glaucoma.

The swinging flashlight test is used to determine whether a relative afferent pupillary defect (RAPD) exists.⁴ The patient fixes the gaze on a distant object and the examination room is darkened. The size of the pupils in lowered light is noted, and unless there is physiologic anisocoria, the pupils should be equal in size. The direct and consensual light responses of the eyes are compared as a light source, angled into the pupil from in front of the cheeks, is swung back and forth between the two. When the light source shines into an eye with an RAPD, the pupil dilates because the consensual response from withdrawal of light from the opposite eye with normal afferent activity is stronger than the direct constrictive response to light in the affected eye with inhibited afferent activity. It is termed "relative" because the response is compared with that of the opposite side as the light source is alternated between eyes. An RAPD may be partial or complete and due to inhibition of light transmission to the retina because of vitreous hemorrhage, loss of some or all of the retinal surface for light contact because of ischemia or detachment, or the presence of lesions affecting the prechiasmal optic nerve (e.g., optic neuritis).

Pressure Determination

Ocular tonometry is usually the last examination performed in the emergency department. Common methods of determining the IOP in the emergency department include use of electronic, manual (e.g., Schiøtz), or applanation tonometers. IOPs in the 10- to 20-mm Hg range are considered normal. Causes of intraocular hypertension include glaucoma in its many forms, suprachoroidal hemorrhage, and space-occupying retrobulbar pathology. Patients presenting with IOPs exceeding 20 mm Hg should have ophthalmologic consultation. Rapid treatment is usually not necessary until the pressure exceeds 30 mm Hg.

Slit-Lamp Examination

The slit lamp permits a magnified, binocular view of the conjunctivae and anterior globe for diagnostic purposes and to facilitate delicate procedures. It allows depth perception in otherwise clear structures, such as the cornea, aqueous humor, and lens. The slit-lamp examination can include the following:

- Lids and lashes may be inspected for blepharitis and pointing of a lid abscess (i.e., hordeolum). The inner canthus and lacrimal punctum may be better viewed for evidence of dacryocystitis.
- Punctures, lacerations, and inflammatory patterns of the conjunctiva or sclera may be discovered with magnification.
- Corneal abrasions, ulcers, foreign bodies, and other abnormalities may be seen. The depth of these lesions may be accurately assessed with an angled beam. Edema, which appears as a white haze or cloudiness within clear structures, can be differentiated within the epithelium or deeper stroma.
- The anterior chamber may be examined for cells (e.g., red and white blood cells) and "flare." Cells are seen as small floating objects caught in the beam of a highly angulated slit-lamp light, as dust floating in the movie theater glows from the reflected light of the projector beam. Flare is a diffuse haziness, related to cells and proteins suspended in the aqueous humor, and is often visible only when illuminated directly (Fig. 32-3). It usually represents deep

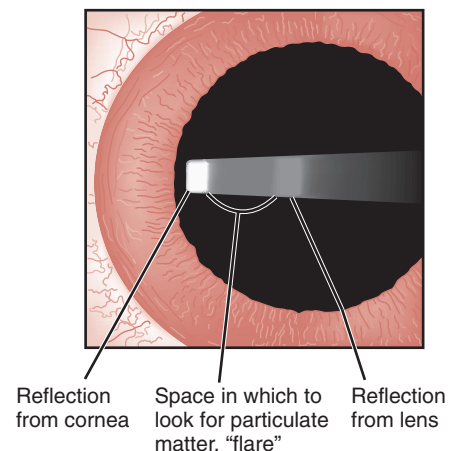


Figure 32-3. Technique of slit-lamp examination with a short, narrow light beam projected from an extreme temporal angle across the contrasting black pupil to better find cells or "flare" indicative of acute anterior uveitis. (From Ragge NK, Easty DL: Immediate Eye Care. St. Louis, Mosby-Year Book, 1990.)

inflammation of the eye and is often seen in iritis. Collections of layered blood or pus in the dependent portions of the anterior chamber are called hyphema or hypopyon, respectively, and are graded by the percentage of the vertical diameter of the visible iris when the head is upright. Foreign bodies that have penetrated the cornea may be found floating in the anterior chamber.

- The trabeculated pattern of the iris can be seen in detail. Spiraling muscle fibers may be seen in acute angle-closure glaucoma. If the beam is shown almost coaxially with the examiner's line of sight such that the red reflex is elicited, tears in the iris may be seen by light returning through the iris itself instead of just through the pupil.
- The lens should be examined for position, general clarity, and the presence of opacities or foreign bodies. The type and position of any lens implants can also be better assessed during a slit-lamp examination.

Direct Funduscopy Examination

Emergency physicians most commonly perform a nondilated funduscopy examination because there are several eye conditions in which dilation may be harmful (e.g., glaucoma). Iridodialysis, lens dislocation, and conditions requiring early intervention are usually identifiable along the visual axis.

Inability to obtain a red reflex or visualize the fundus of the eye can be due to:

- Opacification of the cornea, most commonly by edema secondary to injury or infection
- Hyphema or hypopyon within the anterior chamber
- Extremely miotic pupil
- Cataract of the lens
- Blood in the vitreous or posterior eye wall
- Retinal detachment

In the absence of trauma, few posterior findings are associated with chief complaints of external redness. Findings associated with visual loss include pallor of the retina indicating ischemia, "cupping" of the optic disk indicating glaucoma, indistinctness of disk margins indicating papilledema or optic neuritis or neuropathy, air or plaque emboli in retinal arteries, and a host of other signs indicating more chronic ocular or systemic pathology not normally amenable to management in the emergency department.

Bedside Testing

Fluorescein solution and the cobalt blue lamp are the best means for identifying damage to the corneal epithelium, including that which cannot be seen with conventional slit-lamp examination. Fluorescein highlights defects, making them easy to identify, because the fluorescing liquid is thicker in defects than it is across the normally smooth corneal surface. Use of fluorescein may reveal corneal abrasions and ulcers as well as damage from keratitis related to chemicals, ultraviolet light, or infections (e.g., herpes).

Relief of discomfort after instillation of a topical anesthetic can be used as a diagnostic test for an external source of pain. In general, abolition of pain by local anesthetic drops indicates pain of corneal origin. Modest but incomplete relief suggests a conjunctival process. Intraocular pain is not diminished by local anesthetic solution.⁷ When ocular penetration is suggested, Seidel's test can be used. This test involves placing a fluorescein strip directly over an area of possible corneal disruption. The high localized concentration of fluorescein may facilitate identification of the corneal defect with a slit lamp by allowing visualization of leaking aqueous fluid diluting the fluorescein. This test does not work on the conjunctiva overlying

the sclera, and a negative test result does not rule out a full-thickness corneal injury.

Ancillary Testing

An erythrocyte sedimentation rate may be used to evaluate for temporal arteritis, which may arise with eye pain and decreased visual acuity.

Infections are usually evident by examination, and laboratory tests such as a complete blood count are not necessary. Microbiologic cultures are rarely ordered in the emergency department.

Plain radiography is used to identify facial fractures associated with facial or ocular trauma or indirectly by detecting an air-fluid level in the orbit or fluid in the paranasal sinuses. Computed tomography (CT), using 1.5-mm axial and coronal cuts, provides superior imaging, but is not necessary in many cases.

CT also reliably localizes metal and many nonradiopaque foreign bodies in the globe and orbit. It can also detect small amounts of intraocular air following penetrating trauma. Magnetic resonance imaging (MRI) clearly delineates the orbital and retro-orbital structures, but cannot be employed with metallic (magnetic) foreign bodies, which can migrate to cause additional damage.⁸ It is less often used in emergency eye assessment, for which, in general, CT is the initial imaging modality of choice.⁹ Ultrasonography is more sensitive for detecting intraocular foreign bodies, but CT is better at delineating the damage caused by them, so they are complementary tests.¹⁰

■ DIFFERENTIAL DIAGNOSIS

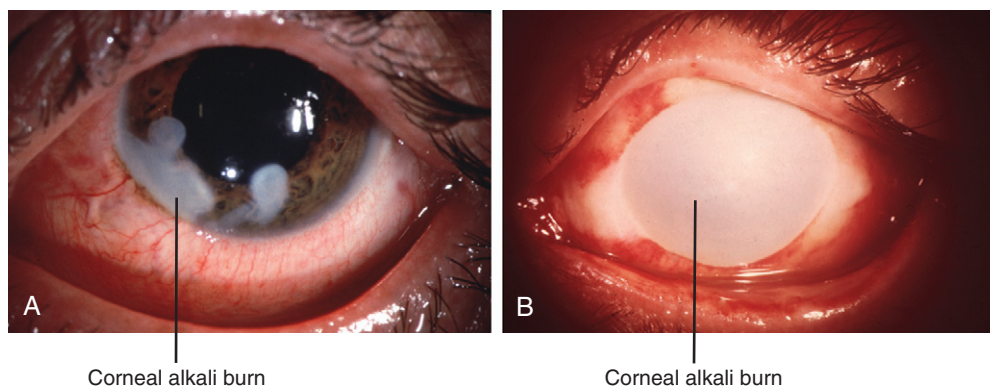
Clinical findings most indicative of serious eye disorder are listed in [Box 32-1](#).

Critical Diagnoses

Caustic injury to the eye can rapidly lead to a destructive keratoconjunctivitis ([Fig. 32-4A and B](#)) if the agent is not removed immediately. The diagnosis is made on history alone, before any other examination is performed. Early and copious irrigation is indicated. Many patients have already undergone extensive irrigation at the job site, but when the exposure has occurred in the home, irrigation prior to arrival in the emergency department is uncommon. Alkaline caustic agents cause a liquefactive necrosis of the cornea by progressively reacting with the corneal layers, and destruction is severe and relentless. Continuous irrigation is the only effective method to terminate the reaction and should be continued for at least 30 minutes. Acid injury is much less severe and requires less irrigation than alkaline exposures, but irrigation should continue until the pH of the tears is neutral or the patient is essentially asymptomatic.

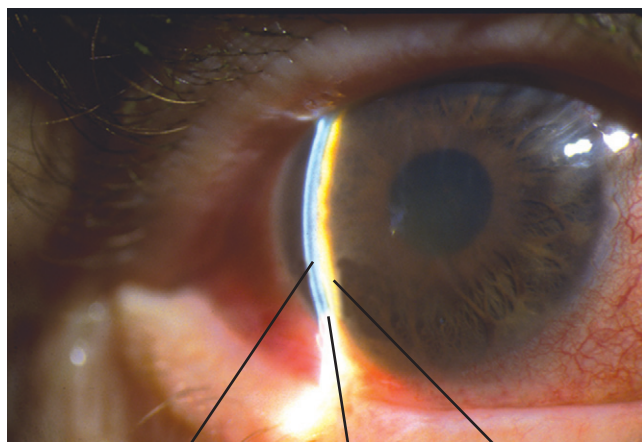
Acute angle-closure glaucoma is a relatively rare but important critical diagnosis to make in the emergency department. Patients present with pain, the onset of which is often sudden in low-light conditions requiring pupillary dilation through contraction and thickening of the iris peripherally. The iris becomes immobile and often irregular, and the pupil is commonly fixed at 5 to 6 mm in diameter. Inability of the pupil to constrict may result in photophobia, and accommodation may be affected. These reactions and the increased IOP can lead to frontal headache, nausea, and vomiting. As inflammation progresses, limbal injection of the conjunctiva is almost universally seen. [Figure 32-5](#) demonstrates many of these findings. Immediate medical intervention in the emergency

Figure 32-4. A, Alkali burn demonstrating corneal burns and conjunctival injection on the day of the accident. B, Complete corneal tissue destruction 7 days after alkali burn. (From Kaiser PK, Friedman NJ, Pineda R, II: *The Massachusetts Eye and Ear Infirmary Illustrated Manual of Ophthalmology*, 2nd ed. Philadelphia, WB Saunders, 2004.)



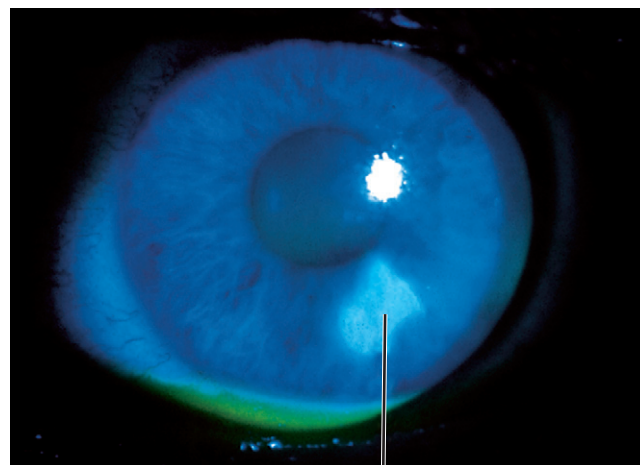
Corneal alkali burn

Corneal alkali burn



Posterior cornea Slit-beam Iris surface

Figure 32-5. Primary angle-closure glaucoma with very shallow anterior chamber and iridocorneal touch (no space between slit-beam view of cornea and iris). (From Kaiser PK, Friedman NJ, Pineda R, II: *The Massachusetts Eye and Ear Infirmary Illustrated Manual of Ophthalmology*, 2nd ed. Philadelphia, WB Saunders, 2004.)



Corneal abrasion

Figure 32-6. Corneal abrasion demonstrating fluorescein pooling of a small inferior epithelial defect. (From Kaiser PK, Friedman NJ, Pineda R, II: *The Massachusetts Eye and Ear Infirmary Illustrated Manual of Ophthalmology*, 2nd ed. Philadelphia, WB Saunders, 2004.)

department and urgent ophthalmologic consultation are warranted.

Retrobulbar hematoma (blood) is usually caused by orbital trauma, but it can also occur spontaneously in patients with coagulopathy. Retrobulbar abscess (pus) or emphysema (air) can also occur.^{11,12} Elevated IOP in any of these conditions constitutes orbital compartment syndrome and a surgical emergency. Emergency intervention is to decompress the orbit by performing lateral canthotomy and cantholysis.^{13,14}

Emergent Diagnoses

Most emergent diagnoses involve some kind of inflammation secondary to trauma, infection, or systemic disease. These include keratitis, anterior uveitis, scleritis, and endophthalmitis. Any of these may be complications of surgical procedures, and an appropriate ophthalmologic history must be obtained.

Keratitis, or inflammation of the cornea, is most commonly viral in origin but can also be caused by exposure to intense ultraviolet light (e.g., snow blindness, arc welder's blindness), various chemicals, or ischemia related to contact lens use. Patients present with an intense foreign-body sensation, ciliary spasm causes photophobia that is often severe, and the affected eyes are often clenched shut. Topical anesthesia provides immediate (but temporary) relief of pain, thus reinforcing the

corneal origin of the process and facilitating examination and definitive diagnosis.⁶ Corneal abrasions are very common and may be identified by white light or fluorescein-facilitated blue light using a slit lamp or any other magnification (Fig. 32-6). Following thorough irrigation, thermal and chemical burns must receive a careful slit-lamp examination for potential full-thickness injury. If this is not found, the corneal injury may be treated similarly to an abrasion.

In immunocompetent hosts, corneal ulcerations are most commonly due to overuse of contact lenses. They are seen as a denuding of epithelium with surrounding edema, the increased interstitial water of which is seen as whitish clouding of the normally clear tissue (Fig. 32-7). Almost all ulcerations require same-day evaluation by an ophthalmologist. Infections of the cornea with herpes simplex virus can rapidly lead to opacification and significant visual loss. It is most commonly recognized by a characteristic dendritic pattern of fluorescein pooling under blue light (Fig. 32-8). Anterior uveitis, which includes iritis and iridocyclitis, often occurs secondary to a traumatic injury or infectious process or can be associated with serious systemic immune diseases, such as adult and juvenile rheumatoid arthritis, sarcoidosis, and ankylosing spondylitis.

Scleritis is rare and may be difficult to differentiate from episcleritis, which is somewhat more common and a more

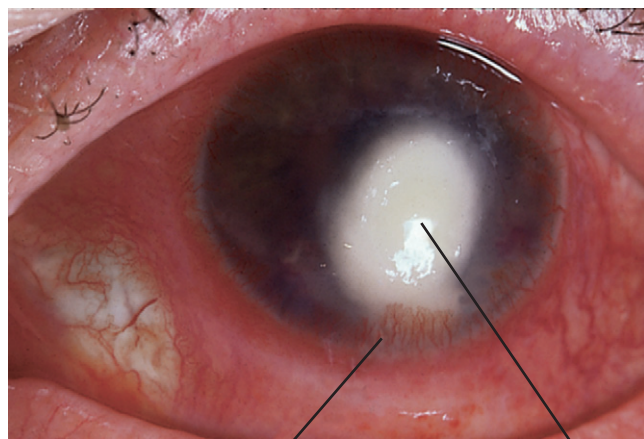


Figure 32-7. Bacterial keratitis demonstrating large, central *Streptococcus pneumoniae* corneal ulcer. Note the dense, white corneal infiltrate and the extreme conjunctival injection. (From Kaiser PK, Friedman NJ, Pineda R, II: The Massachusetts Eye and Ear Infirmary Illustrated Manual of Ophthalmology, 2nd ed. Philadelphia, WB Saunders, 2004.)

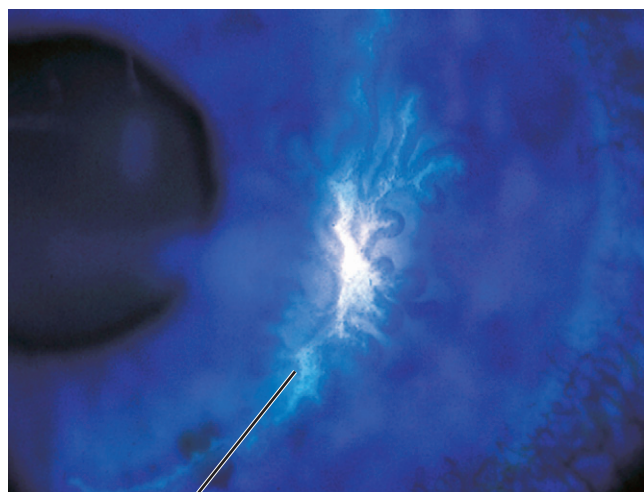


Figure 32-8. Patient demonstrating fluorescein pooling of herpes simplex virus dendrite. (From Kaiser PK, Friedman NJ, Pineda R, II: The Massachusetts Eye and Ear Infirmary Illustrated Manual of Ophthalmology, 2nd ed. Philadelphia, WB Saunders, 2004.)

benign inflammation. The former is commonly idiopathic but may be associated with a systemic inflammatory process, such as a connective tissue disease, gout, or infection (e.g., Lyme disease, syphilis, tuberculosis). Eye redness in episcleritis results from dilation of the episcleral blood vessels just underneath the conjunctiva, usually in a small sector of the visible portion of the globe. If the location of the involved layer is in doubt, a topical anesthetic allows the examiner to move the conjunctiva and its contained vessels with a cotton-tipped applicator, thus differentiating between vessels of scleral or conjunctival origin. The pain of scleritis is typically slower in onset but is often described as a severe “boring” pain that radiates to the ipsilateral forehead, cheek, or jaw. Engorgement of scleral vessels is usually more prominent and more diffuse than that of the episcleral vessels in episcleritis. A bluish hue may be seen as the underlying pigmented epithelium shows through the edematous, and hence more

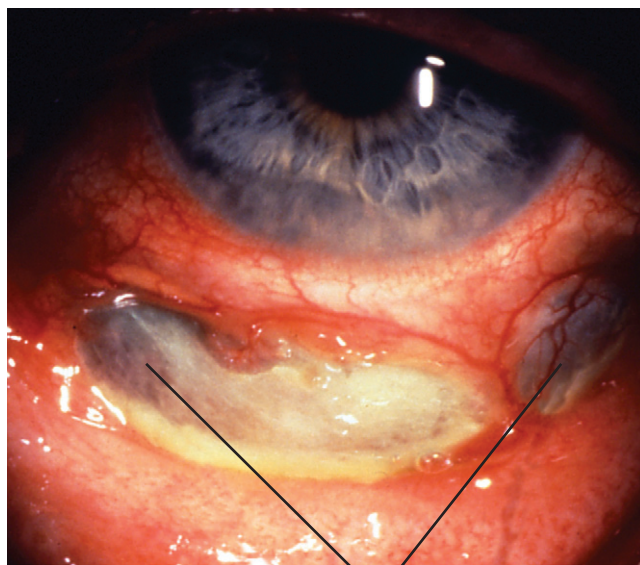


Figure 32-9. Diffuse scleritis with slight bluish region in addition to injection of scleral, episcleral, and conjunctival vessels. (From Kaiser PK, Friedman NJ, Pineda R, II: The Massachusetts Eye and Ear Infirmary Illustrated Manual of Ophthalmology, 2nd ed. Philadelphia, WB Saunders, 2004.)

translucent, sclera (Fig. 32-9). Scleritis may be associated with anterior uveitis, cataract, and secondary glaucoma.

Endophthalmitis usually results from an infection of structures inside the globe. It is most common following penetrating trauma but may begin after hematogenous seeding from a remote or systemic infection, particularly in immunocompromised hosts. Unless it is detected early, and is responsive to aggressive antimicrobial therapy, endophthalmitis is a devastating process that frequently requires enucleation.

Urgent Diagnoses

Penetrating ocular trauma is evaluated by history (e.g., working with high-speed grinding equipment), examination (extrusion of aqueous humor or other globe content; direct visualization of a foreign body in the anterior chamber, vitreous, or retina), or identification of the offending object by biplanar plain radiography, thin-cut CT, or ultrasonography. MRI should not be used if there is any possibility that the foreign object may be metallic. Indirect indicators of globe penetration are hyphema, an irregularly shaped pupil from traction on or injury to the iris' attachments, or lack of a red reflex. If penetrating ocular injury is confirmed or if the possibility persists after evaluation, an ophthalmologic consultation is indicated.⁴

Spontaneous or traumatic hyphema is often managed conservatively. Blood in the anterior chamber is usually the result of direct ocular trauma and may be associated with traumatic mydriasis or an obvious tear of the iris. If penetration and rupture can be reasonably excluded, the hyphema should be graded and IOP determined. Intraocular hypertension (or hypotension in the case of occult globe rupture) following trauma must also be evaluated by an ophthalmologist urgently. Inability to view posterior structures through the anterior blood may necessitate radiologic or ultrasonographic imaging.

Diagnostic Algorithm

A recommended algorithmic approach to the patient with an acute red eye is provided in Figure 32-10.

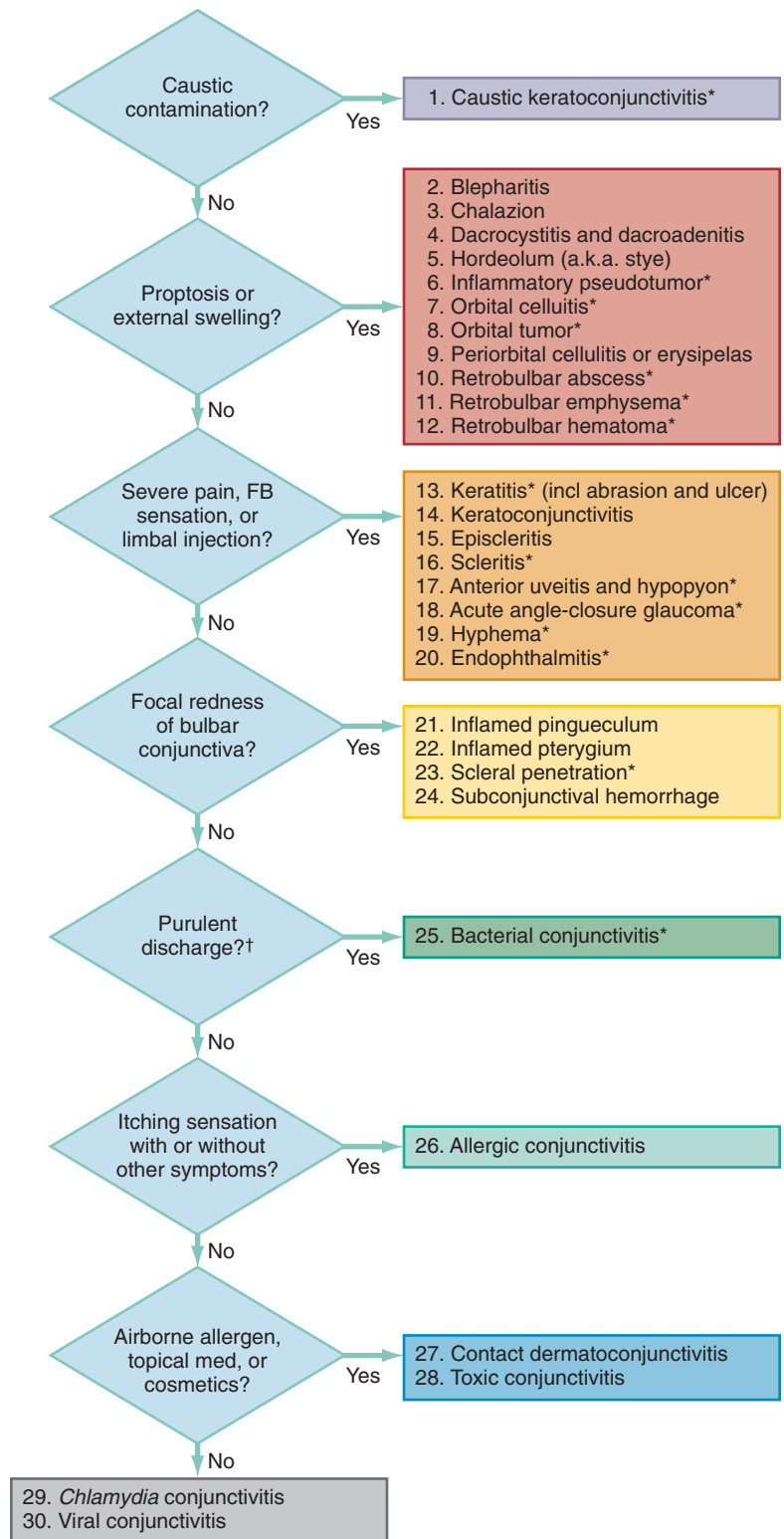


Figure 32-10. Diagnostic algorithm for red eyes. *Indicates potentially serious diagnoses if not identified on initial emergency department evaluation. †Purulent implies true pus, as opposed to the mucoid discharge more commonly associated with nonbacterial causes of conjunctivitis. a.k.a., also known as; FB, foreign body; incl, including. (Modified from Trobe JB: The Physician's Guide to Eye Care. San Francisco, Foundation of the American Academy of Ophthalmology, 2001.)

EMPIRICAL MANAGEMENT

Irrigation

Any clean water is appropriate for irrigation, and prompt initiation takes precedence over procurement of a particular irrigating solution. The most important principles are *rapid and copious* dilution and removal of the offending material. An eyewash station or faucet with tap water may be employed.

Normal saline may be instilled through the end of macrodrip intravenous administration tubing. If there is no gross eye injury, a Morgan lens may be attached to this tubing, but emergency department staff do not have to help the patient hold the eye open. Quickly administering two drops of topical anesthetic and allowing 30 seconds or so for the anesthetic to become effective greatly facilitates patients' tolerance of the prolonged irrigation required. It is recommended that the first 500 to 1000 mL of irrigation fluid be

administered while examining the eye; then the Morgan lens may be placed.

Pain Relief

Pain often interferes with obtaining an adequate assessment. A topical anesthetic, such as proparacaine 0.5%, may facilitate cooperation in patients with possible injury or inflammation of the anterior eye by reducing pain and blepharospasm long enough to obtain a targeted history and focused examination. Topical anesthetic agents should not be given to patients to use at home. Parenteral or oral analgesics can be used for severe deep pain not amenable to topical relief in the emergency department, or for outpatient management of discomfort after discharge.

Mydriatic and Cycloplegic Agents

Dilation of the pupil is not usually necessary in the emergency department for funduscopic examination, but may relieve pain associated with ciliary spasm in anterior uveitis. Mydriatic agents (e.g., phenylephrine, tropicamide) merely prevent constriction of the pupil by paralyzing the sphincter pupillae muscle of the iris. Cycloplegic agents (e.g., cyclopentolate, homatropine) paralyze the ciliaris muscle, with an accompanying mydriatic effect. The agent chosen should be guided by the desired length of time of mydriasis for the particular condition being treated (Table 32-1). Mydriatic agents are contraindicated in patients with narrow-angle glaucoma.

Antimicrobial Agents

Most conjunctivitis is viral in origin, but it is often difficult to distinguish bacterial from viral types of conjunctivitis based solely on clinical grounds.^{15,16} Although no definitive empirical evidence dictates the use of antibiotic solutions or ointments for surface infections, the use of broad-spectrum topical antibiotics in cases of proven bacterial conjunctivitis is associated with benefit showing significantly higher clinical remission rates.¹⁷ Antimicrobial prophylaxis should be used for penetrating wounds to prevent bacterial keratitis or endophthalmitis.

Antibiotics are typically used to treat identified or suggested bacterial infections, even if the exact bacterial agent has not been determined. The most common causes of bacterial conjunctivitis are nontypable *Haemophilus influenzae*, *Streptococcus pneumoniae*, and *Staphylococcus aureus*.^{18,19} Trachoma, a chronic keratoconjunctivitis caused by *Chlamydia trachomatis*, is the

most common infectious cause of blindness.²⁰ Bacterial keratitis is usually seen in contact lens wearers, particularly those who wear them overnight.²¹ In descending order of frequency of cultured organisms, microbial keratitis is caused by *Pseudomonas aeruginosa*, streptococcal or staphylococcal species, filamentous fungi, nonpseudomonal gram-negative rods, *Acanthamoeba*, other bacteria, and yeast.²²

The most common organisms cultured from deeper eye structures, particularly following open-globe injuries, are *Bacillus cereus*, *Propionibacterium acnes*, and various species of *Bacillus*, *Streptococcus*, and *Staphylococcus*.^{23,24} While awaiting emergent ophthalmologic consultation for possible vitrectomy, empirical parenteral antibiotic combinations include cefazolin plus gentamicin or vancomycin plus cefotaxime, ceftazidime, or ceftriaxone. Possible cases of mycotic endophthalmitis have historically been treated with amphotericin B,²³ though voriconazole has been shown to have good intraocular penetration, broad-spectrum activity, and relatively low systemic toxicity.²⁵

Open wounds also require tetanus prophylaxis, if the patient's immunization status is not up to date. There is no current evidence supporting the practice of administering tetanus immunization to patients with superficial corneal abrasions.

Other Protective Interventions

Significantly increased IOP must be reversed as rapidly as possible, often before the specific cause is known. After placing the patient in at least a 30° head-up position, two drops of timolol 0.5%, a topical beta-adrenergic blocking medication, should be administered as a first-line agent to decrease the production of aqueous humor. This may be followed by two drops of dorzolamide 2%, a topical carbonic anhydrase inhibitor, to reduce aqueous humor production further. If not available, 500 mg of acetazolamide may be given orally or intravenously. If the patient has sickle cell disease or trait, oral methazolamide 50 mg must be used instead. Patients with suggested intraocular hypertension who also have nausea or vomiting should receive a parenteral antiemetic so that they do not gag or vomit, which may further increase IOP.

Specific Management

Management of the specific entities listed in the diagnostic algorithm presented in Figure 32-10 is presented in Table 32-2. Specific management of ophthalmologic conditions is also discussed in Chapter 69.

SPECIAL CONSIDERATIONS

Pediatrics

A red eye in a neonate or infant is always abnormal. It is usually caused by corneal abrasion or infection. Corneal abrasions can also be a cause of inconsolable crying in an infant. Fluorescein examination helps to identify abrasions and herpes keratitis, acquired from the birth canal. *Chlamydia* infections may also be acquired during vaginal deliveries but may not arise for weeks. These infections should be treated with oral azithromycin as well as parenteral ceftriaxone to cover *Neisseria gonorrhoeae*. Conjunctivitis associated with respiratory symptoms or infiltrates on a chest radiograph in an infant younger than 3 months should be treated with an oral macrolide. Oral antibiotics are also indicated for conjunctivitis associated with otitis media. *Mycoplasma* is a common infectious agent in these cases, and a macrolide is indicated.¹⁴

Table 32-1

Duration of Action for Common Mydriatic and Cycloplegic Medications

NAME	CONCENTRATION (%)	COMMON DURATION	MAXIMUM DURATION
Ephedrine*	5.0	0.5–1 hr	3 hr
Phenylephrine*	2.5	0.5–1 hr	3 hr
Tropicamide	0.5	3–4 hr	6 hr
Cyclopentolate	0.5	12–18 hr	24 hr
Homatropine	1.0	1–2 days	3 days
Scopolamine	0.5	2–5 days	7 days
Atropine	0.5	5–10 days	14 days

*Mydriatic action only, no cycloplegic effect. Combination products such as Cyclomydril, which is cyclopentolate 0.2% and phenylephrine 10%, are also available.

Table 32-2 Management Algorithm for Red Eyes Extended from Diagnostic Algorithm in Figure 32-10

DIAGNOSIS FROM FIGURE 32-10	MANAGEMENT	CONSULTATION	DISPOSITION
1. Caustic keratoconjunctivitis	Immediate and copious irrigation with tap water or sterile normal saline until tear-film pH = 7. <i>Solids:</i> lift particles out with dry swab before irrigation <i>Acids:</i> minimum of 2 L and 20 min <i>Alkalis:</i> minimum of 4 L and 40 min None except artificial tears for dry eye.	Ophthalmologist must come to ED if there is any abnormal visual acuity or objective finding on examination after sufficient irrigation, with exception of expected injection of conjunctiva secondary to treatment.	May discharge only if tear film pH = 7 and no findings on examination except conjunctival injection and ophthalmologist can reevaluate next day.
2. Blepharitis Inflammation of eyelid margins often a/w crusts on awakening, FB sensation, and tearing.	None.	Outpatient referral only for treatment failure after 2 wk.	Discharge with instructions to apply warm compresses to eyelids for 15 min qid and scrub lid margins and lashes with mild shampoo on washcloth bid.
3. Chalazion Inflammation of meibomian gland causing subcutaneous nodule within the eyelid.	None.	Outpatient referral only for treatment failure after 2 wk.	Discharge with instructions to apply warm compresses to eyelids for 15 min and gently massage nodule qid.
4. Dacryocystitis and dacryoadenitis Eye tearing and inflammation of lower eyelid inferior to lacrimal punctum finding redness and tenderness over nasal aspect of lower lid and adjacent periorbital skin.	First t/o periorbital cellulitis (#9) and orbital cellulitis (#7). Inspect for obstruction of punctum by SLE, may express pus by pressing on sac, PO Rx for nasal and skin flora if not admitting. <i>External:</i> Warm compresses often all that is needed, may Rx anti- <i>Staph</i> ointment bid. <i>Internal:</i> PO Rx for β -lactamase <i>Staph</i> .	Ophthalmologist may admit if systemically ill, case is moderate or severe, or no social support for patient. Ask about culturing before Rx if admitting, then Rx same as for periorbital cellulitis (#9). Outpatient referral only for treatment failure after 2 wk.	May discharge mild cases with PO analgesics and antibiotics (e.g., amoxicillin/clavulanate), and instructions to apply warm compresses to eyelids for 15 min and gently massage inner canthal area qid. Discharge with instructions to apply warm compresses to eyelids for 15 min and gently massage abscess qid.
5. Hordeolum (a.k.a. srye) Abscess in eyelash follicle or modified sebaceous gland at lid margin: <i>external</i> or <i>internal</i> based on side of lid margin that abscess is pointing.	Measure IOP. Evaluate for infection, diabetes mellitus, and vasculitis with CBC, BMP, UA, and ESR. Obtain axial CT of brain and axial and coronal CT of orbits and sinuses.	IOP > 20 mm Hg may be surgical emergency. Rx to decrease IOP in ED.	May discharge if no systemic problems, no findings of particular concern on CT, and IOP > 20 mm Hg. Start high-dose PO steroids after discussion with ophthalmologist and ensure reevaluation in 2–3 days.
6. Inflammatory pseudotumor* Nonspecific idiopathic retrobulbar inflammation with eyelid swelling, palpebral injection of conjunctiva, chemosis, proptosis, blurred vision, painful or limited ocular mobility, binocular diplopia, edema of optic disk, or venous engorgement of retina.	Measure IOP. Start IV Rx with second-generation cephalosporin (e.g., cefuroxime, cefoxitin, or cefotetan) or with ampicillin/sulbactam to cover sinus and skin flora. Alternative Rx is ticarcillin/clavulanate, piperacillin/tazobactam, vancomycin, or clindamycin + third-generation cephalosporin (e.g., ceftriaxone) or ceftriaxone.	IOP > 20 mm Hg may be surgical emergency. Rx to decrease IOP in ED. Obtain blood cultures and start antibiotics. Axial and coronal CT of orbits and sinuses to r/o FB, retrobulbar abscess, orbital gas, subperiosteal abscess, osteomyelitis, and changes in cavernous sinus. Consider LP.	Admit all cases of orbital cellulitis.
7. Orbital cellulitis* Eyelid swelling, redness and warmth of skin overlying orbit, tenderness of skin overlying bone palpebral injection of conjunctiva, and chemosis. Differentiated from periorbital cellulitis by presence of any finding of fever, ill appearance, blurred vision, proptosis, painful or limited ocular mobility, binocular diplopia, edema of optic disk, or venous engorgement of retina.	Measure IOP. Evaluate for extraocular signs of malignancy. Obtain axial CT of brain and axial and coronal CT of orbits and sinuses.	IOP > 20 mm Hg may be surgical emergency. Rx to decrease IOP in ED. Ophthalmologist may want MRI, MRA, or orbital US.	Based on findings and discussion with consultant.
8. Orbital tumor* Blurred vision, proptosis or other displacement of globe, painful or limited ocular mobility, or binocular diplopia (but can be asymptomatic).			

Continued

Table 32-2 Management Algorithm for Red Eyes Extended from Diagnostic Algorithm in Figure 32-10—cont'd

DIAGNOSIS FROM FIGURE 32-10	MANAGEMENT	CONSULTATION	DISPOSITION
9. Periorbital cellulitis or erysipelas swelling, redness and warmth of skin overlying orbit, tenderness of skin overlying bone, palpebral injection of conjunctiva, and chemosis. Differentiated from orbital cellulitis by <i>absence</i> of any other finding listed in #7.	Eyelid overlying orbit, tenderness of skin overlying bone, palpebral injection of conjunctiva, and chemosis.	Ophthalmologist may admit if systemically ill, case is moderate or severe, or no social support for patient.	May discharge mild cases with PO antibiotics. Ophthalmologist must reevaluate next day to ensure no orbital extension.
10. Retrobulbar abscess* Findings of orbital cellulitis (#7) but a/w increased IOP.	Findings of orbital cellulitis (#7) but a/w increased IOP.	IOP > 20 mm Hg may be surgical emergency, Rx to decrease IOP in ED. Obtain axial CT of brain and axial and coronal CT of orbits and sinuses.	Admit all cases of retrobulbar pathology causing increased IOP. Others might be candidates for discharge depending on cause of problem.
11. Retrobulbar emphysema* Findings of pseudotumor (#6) but a/w increased IOP.	Findings of pseudotumor (#6) but a/w increased IOP.	<i>Abscess:</i> Antibiotics as in orbital cellulitis (#7). <i>Emphysema:</i> Prophylax with antibiotics to cover sinus flora. <i>Hematoma:</i> Correct any coagulopathy or thrombocytopenia.	
12. Retrobulbar hematoma* Findings of pseudotumor (#6) but occurs due to trauma, coagulopathy, or thrombocytopenia and a/w diffuse subconjunctival hemorrhage anteriorly and extending posteriorly as well as increased IOP.	Findings of pseudotumor (#6) but occurs due to trauma, coagulopathy, or thrombocytopenia and a/w diffuse subconjunctival hemorrhage anteriorly and extending posteriorly as well as increased IOP.		
13. Keratitis (abrasion or UV injury) Pain, FB sensation, blepharospasm, tearing, photophobia, epithelial disruption on inspection under white light or fluorescein pooling under blue light. SPK appears as stippling of corneal surface [often lower 2/3 of cornea if due to light exposure].	Pain, FB sensation, blepharospasm, tearing, photophobia, epithelial disruption on inspection under white light or fluorescein pooling under blue light. SPK appears as stippling of corneal surface [often lower 2/3 of cornea if due to light exposure].	Ophthalmologist must come to ED if there is any concern for globe penetration. Otherwise consult for follow-up examination in 1–2 days. One-time administration of cycloplegic agent may limit photophobia until follow-up examination.	May discharge cases not infected or ulcerated on topical antibiotic prophylaxis using polymyxin B combinations with bacitracin (ointment) or trimethoprim (solution). Gentamicin and sulfacetamide are less desirable single-agent alternatives. PO NSAIDs or narcotics for analgesia. Patching not necessary.
Keratitis (ulceration)* Symptoms and signs as above. Ulceration from complications of contact wear or neglected corneal abrasion has “scooped out” epithelium with surrounding edema appearing as white “cloudiness” in clear tissue.	Symptoms and signs as above. Ulceration from complications of contact wear or neglected corneal abrasion has “scooped out” epithelium with surrounding edema appearing as white “cloudiness” in clear tissue.	Discuss any potential need to debride or culture before starting antibiotic.	Based on findings and discussion with consultant. Typical ciprofloxacin dosing is 1 gt. q 15 min for 1 hr, then 1 gt. q hr for 8 hr, then 1 gt. q 4 hr until seen by consultant next day. PO NSAIDs or narcotics for analgesia. No patch.
Keratitis (herpetic infection)* Symptoms and signs as above. Look for other signs of herpes, varicella, zoster (or CMV infection in immunocompromised patient). Look for “dendritic” defects of cornea with fluorescein under blue light.	Symptoms and signs as above. Look for other signs of herpes, varicella, zoster (or CMV infection in immunocompromised patient). Look for “dendritic” defects of cornea with fluorescein under blue light.	Discuss with ophthalmologist any potential need to debride or culture before starting antiviral.	Based on findings and discussion with consultant. Typical trifluridine dosing is 1 gt. q 2 hr for 7 days, then taper over 2 more wk. Typical vidarabine or acyclovir dosing is five times a day for 7 days, then taper over 2 more wk. PO NSAIDs or narcotics for analgesia. No patch.

14. Keratoconjunctivitis Conjunctivitis with subepithelial infiltrates in cornea causing pain and decreased vision, possibly with halos reported.	Treat for conjunctivitis by likely etiologic category (#25–30).	Discuss findings and use of prednisolone acetate 1% (frequency determined by ophthalmologist).	May discharge patient with medications recommended by ophthalmologist and ensure reevaluation in 2–3 days.
15. Episcleritis Rapid onset of localized pain, injection of episcleral vessels, and localized tenderness.	Relieve irritation with artificial tears and decrease inflammation with ketorolac gtt.	Outpatient referral only for treatment failure after 2 wk.	May discharge patient with PO NSAIDs alone or in combination with topical ketorolac gtt.
16. Scleritis * Progressively increasing eye pain with radiation to ipsilateral face and decreasing vision, photophobia, tearing, and possible pain with eye motion.	Decrease inflammation with PO NSAIDs.	Discuss findings and use of topical or PO steroids.	May discharge patient with medications recommended by ophthalmologist and ensure reevaluation in 2–3 days.
17. Anterior uveitis and hypopyon * Eye pain, photophobia, tearing, limbal injection of conjunctiva, and cells or flare in anterior chamber. Hypopyon is layering of white cells (pus) in anterior chamber.	First t/o glaucoma with IOP measurement. Rx in ED if IOP > 20 mm Hg. Otherwise OK to dilate pupil with 2 gtt. of cyclopentolate 1%.	Discuss findings and use of prednisolone acetate 1% (frequency determined by ophthalmologist but range is q 1–6 hr).	May discharge patient with medications recommended by ophthalmologist and ensure reevaluation in 2–3 days. Patients with hypopyon are generally admitted.
18. Acute angle-closure glaucoma Sudden-onset eye pain and blurred vision that may be a/w frontal headache, nausea, and vomiting. Anterior eye may manifest shallow or closed angle between iris and cornea, pupil fixed in mid-dilation, or limbal injection of conjunctiva.	Decrease production of aqueous humor. Timolol 0.5% 1 gt., then repeat in 30 min. Apraclonidine 1% 1 gt. once. Dorzolamide 2% 2 gtt. <i>or if sickle cell disease or trait</i> then methazolamide 50 mg PO. Decrease inflammation. Prednisolone 1% 1 gt. every 15 min four times.	Discuss any IOP > 20 mm Hg with ophthalmologist.	Based on findings and discussion with consultant, which primarily depends on speed of onset and response to treatment.
Rx in ED if IOP > 30 mm Hg.	Constrict pupil. Pilocarpine 4% 1 gt., then repeat in 15 min Consider establishing osmotic gradient Mannitol 2 g/kg IV.		
19. Hypohphema * Pain, decreased visual acuity, gross or microscopic blood in anterior chamber, may be a/w dilated and fixed pupil following blunt trauma. Graded by amount of blood Percentage of vertical diameter of anterior chamber when blood layers with patient in upright position. Microhyphema shows no layering and only suspended red blood cells.	First t/o globe rupture. May require ultrasound if cannot visualize posterior structures. Measure IOP unless possibility of ruptured globe. IOP > 30 mm Hg may require acute treatment as in glaucoma (#18). If IOP > 20 mm Hg and no iridodialysis, may use cycloplegic to prevent iris motion.	Discuss findings and use of ϵ -aminocaproic acid and steroids, other medical therapy, best disposition, and follow-up examination by ophthalmologist within 2 days. Some patients may be admitted for observation, bed-rest, head elevation, and frequent medication administration.	Most patients can be discharged with careful instructions to return for any increased pain or change in vision. Patients should decrease physical activity and sleep with an eye shield in place. Eyes should be left open while awake, so any change in vision can be immediately recognized. PO NSAIDs or narcotics for analgesia.

Continued

Table 32-2 Management Algorithm for Red Eyes Extended from Diagnostic Algorithm in Figure 32-10—cont'd

DIAGNOSIS FROM FIGURE 32-10	MANAGEMENT	CONSULTATION	DISPOSITION
20. Endophthalmitis * Progressively increasing eye pain and decreasing vision, diminished red reflex, cells and flare (and possibly hypopyon) in anterior chamber, chemosis, and eyelid edema.	Empirical parenteral antibiotic administration with cefazolin + gentamicin or vancomycin + cefotaxime, ceftazidime, or ceftriaxone to cover <i>Bacillus</i> , <i>enterococcus</i> , and <i>Staphylococcus</i> spp.	Ophthalmologist must admit for parenteral and possibly intraocular antibiotics.	Admit all cases of endophthalmitis.
21. Inflamed pingueculum Inflammation of soft yellow patches in temporal and nasal edges of limbal margin.	Decrease inflammation with naphazoline or ketorolac gtt.	Outpatient referral only for treatment failure after 2 wk.	Discharge to follow-up with ophthalmologist for possible steroid therapy or surgical removal. Same as #21
22. Inflamed intervegium Inflammation of firmer white nodules extending from limbal conjunctiva onto cornea.	Same as #21	Same as #21	Same as #21
23. Scleral penetration * Localized redness at site of entry, teardrop pupil, blood in anterior chamber or loss of red reflex.	Protect eye from further pressure, provide pain relief, and prevent vomiting. Tetanus prophylaxis.	Ophthalmologist must come to ED if there is any concern for globe penetration.	Admit for IV antibiotics and possible procedural intervention.
24. Subconjunctival hemorrhage Red blood beneath clear conjunctival membrane.	Exclude coagulopathy or thrombocytopenia, if indicated by history.	None required if no complications.	Reassure patient that red should resolve over 2–3 wk.
25. Bacterial conjunctivitis * Hyperpurulent discharge not typical of common “pink eye” and more commonly unilateral in adults. Inflammation of eyelid margins a/w lid edema, chemosis, and possibly subconjunctival hemorrhage, but usually no follicular “cobblestoning.”	Topical polymyxin B trimethoprim in infants and children, because more <i>Staph.</i> spp. Topical sulfacetamide or gentamicin clinically effective in 90% of uncomplicated adult cases. Use topical fluoroquinolone if <i>Pseudomonas</i> possible.	Culture drainage and ophthalmology consult in all neonates and those at risk for vision loss or systemic sepsis. <i>Neisseria gonorrhoeae</i> can be rapidly sight-threatening.	Discharge uncomplicated cases with 10 days of topical antibiotics in both eyes, regardless of laterality of apparent infection. Use ointments in infants and gtt. in others.
26. Allergic conjunctivitis Often bilateral palpebral injection of conjunctiva and follicular cobblestoning of inner surface of lids that may be seasonal and a/w other allergic symptoms such as rhinitis.	Decrease irritation with naphazoline gtt.	Outpatient referral only for treatment failure after 2 wk.	Identify antigen if possible. Consider treating other allergic symptoms with PO antihistamines.
27. Contact dermatitis/conjunctivitis Localized lid and conjunctival redness and edema.	Irrigation with tap water or sterile normal saline. Decrease irritation with naphazoline gtt.	Outpatient referral only for severe cases or treatment failure after 2 wk.	Identify offending agent and avoid subsequent exposure. Discharge uncomplicated cases on continued naphazoline. Same as #27
28. Toxic conjunctivitis Diffuse conjunctival injection, chemosis, and lid edema.	Same as #27	Same as #27	Same as #27
29. Chlamydia conjunctivitis Often bilateral palpebral injection of conjunctiva in neonate or other individual at risk for sexually transmitted disease.	Rx PO azithromycin for <i>Chlamydia</i> . Consider parenteral ceftriaxone for concurrent <i>Neisseria gonorrhoeae</i> .	Culture drainage and consult ophthalmology in all neonates and those at risk for vision loss or systemic sepsis.	Discharge uncomplicated cases on 5 days of PO azithromycin.
30. Viral conjunctivitis Often bilateral palpebral injection of conjunctiva and follicular cobblestoning of inner surface of lids. Inflammation of eyelid margins often a/w crusts on awakening, FB sensation, and tearing.	Decrease irritation with artificial tears, naphazoline, or ketorolac gtt.	Culture drainage and consult ophthalmology in all neonates and those at risk for vision loss or systemic sepsis.	Ask about pregnant mothers, infants, and immunocompromised individuals in close contact. Discharge uncomplicated cases with instructions on respiratory and direct-contact contagion for 2 wk.

*Potentially serious diagnoses if not identified on initial emergency department evaluation. Antibiotic choices should be based on current practice.

a.k.a., also known as; a/w, associated with; bid, twice daily; BMP, basic metabolic profile (includes electrolytes, glucose, and renal function tests); CBC, complete blood count; CMV, cytomegalovirus; CT, computed tomography; ED, emergency department; ESR, erythrocyte sedimentation rate; FB, foreign body; gt., drop; gtt, drops; IOP, intraocular pressure; LP, lumbar puncture; MRA, magnetic resonance angiography; MRI, magnetic resonance imaging; NSAIDs, nonsteroidal anti-inflammatory drugs; PO, oral; q, every; qid, 4 times a day; r/o, rule out; Rx, prescribe; SLE, slit-lamp examination; SPK, superficial punctuate keratitis; spp, species; *Staph.*, *Staphylococcus*; *Strep.*, *Streptococcus*; UA, urinalysis; US, ultrasonography; UV, ultraviolet.

Trauma

Blunt trauma is a common cause of a red and painful eye. Large hyphemas and those with clots are likely to require hospitalization for bedrest with 30° of head elevation. Systemic analgesia and, if required, antiemetics are indicated. Medications affecting platelet function should be avoided. Treatment may be indicated when the IOP exceeds 30 mm Hg, as it is in acute angle-closure glaucoma. If the iris is not injured, a long-acting cycloplegic agent (e.g., topical homatropine) may be recommended to prevent repetitive motion of the iris. Some reliable adult patients may be discharged with daily follow-up by a specialist. Strong analgesia and patching are not indicated, so that the patient may immediately identify increases in pain or decreases in visual acuity.

Corneal abrasions are common problems in the emergency department. When the emergency physician is convinced that the cornea has not received a full-thickness laceration or penetration by a foreign body, management is relatively simple. Foreign bodies (on or in the epithelium only) should be removed when possible. These may frequently adhere to a saline-moistened cotton-tipped applicator. Ones that do not may sometimes be lifted off with a blunt-tipped tool (“spud”) under the binocular magnification of a slit lamp. The common use of hypodermic needle removal may damage surrounding cornea and is not recommended. Whether or not the object can be successfully removed, management is the same as for corneal abrasions. Rust staining of the corneal epithelium does not require removal in the emergency department, but patients are referred to a specialist for examination within 3 days. Prophylactic topical antibiotics are indicated for all epithelial defects of the cornea. Patching is not necessary and may be harmful. Systemic analgesia appropriate to the patient’s level of pain should be provided. Larger lesions may require a prophylactic mydriatic or cycloplegic agent anticipating a secondary iritis. Topical anesthetics should not be given to the patient for home use.⁴

DISPOSITION

Most emergency department patients with eye complaints are candidates for discharge and, if indicated, follow-up in the

emergency department or with an ophthalmologist in 1 to 2 days. Others may require referral only if there is lack of resolution or treatment fails. A few patients require admission for procedural intervention, parenteral antibiotic regimens, management of intractable pain, or further diagnostic evaluation. General consultation and disposition considerations for the most important entities are outlined in Table 32-2.

KEY CONCEPTS

- Prompt and prolonged irrigation is advised for patients who experience caustic injury to the eye.
- Headache and nausea may be prominent symptoms in patients with acute angle-closure glaucoma.
- Keratitis, inflammation of the cornea, is most commonly caused by a viral infection, but may also be caused by recent ultraviolet light exposure, chemical injury, or hypoxic injury from contact lens use.
- A localized corneal defect with edematous, inflammatory changes may signal corneal ulceration.
- A corneal dendritic pattern may signal a herpetic infection, which can progress to corneal opacification and visual loss.
- Pain, consensual photophobia, perilimbal conjunctival infection, and a miotic pupil that is caused by ciliary spasm could signal iritis, which is inflammation of the iris and ciliary body, or uveitis, inflammation of the iris, ciliary body, and also choroids. The cause may be trauma or underlying autoimmune disease. The presence of cells and flare in the anterior chamber can help signal these conditions.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.

PART II

Trauma

CHAPTER 33 Multiple Trauma

Eric Gross and Marc Martel

■ INTRODUCTION

The care of the injured patient remains one of the mainstays of emergency medicine practice. Emergency physicians play a vital role in the stabilization and diagnostic phases of trauma care. Management of these patients involves complex, time-dependent decision-making, leadership capability, and technical skill. Proper resuscitation can lead to functional outcomes, even in severely injured patients.¹

■ EPIDEMIOLOGY

In 2004, there were 167,184 injury-related deaths, of which 73% were motor vehicle–related, firearms-related, or caused by poisonings or falls. Unintentional accidents were the leading cause of death in people ages 1 to 44 years. Motor vehicle collisions (MVCs) made up the largest percentage of those accidents, accounting for 26% of all injury-related deaths.² The number of motor vehicle deaths has remained relatively stable for the past decade. The number of people injured in motor vehicle crashes, however, has declined 22% during the same time period, to 2.7 million in 2005.³ Homicide is one of the top five leading causes of death in people ages 1 to 44 years, with firearm injuries accounting for 17.7% of all injury deaths in 2004.²

The economic cost of traumatic injuries is staggering. It is estimated that the total cost of injuries that occurred in 2000 is \$406 billion; this includes medical costs and lost productivity. Motor vehicle and fall injuries account for 22% (\$89 billion) and 20% (\$81 billion) of this total, respectively.⁴

Many of these injuries are avoidable. Proper use of lap/shoulder belts can reduce the risk of fatal injury by 45%.⁵ Yet, in 2005, it is estimated that 55% of MVC occupant fatalities were unrestrained; this percentage increases to 65% for the 21- to 24-year-old age group.⁵ Child safety seats reduce the risk of death in passenger cars by 71% for infants and by 54% for toddlers ages 1 to 4 years.⁶ Educational and law enforcement initiatives addressing seat belts, proper child restraint, drinking and driving, gun safety, and fall prevention can assist in raising public awareness. The National Highway Traffic Safety Administration's (NHTSA) "Click-it or Ticket" campaign increased belt use in 41 of 50 states and the District of Columbia during a 2-month time period.⁷ NHTSA has a similar program aimed at drunk driving called "Over the limit. Under arrest." The effect of firearm laws on decreasing firearm violence

is less concrete (even though a decrease has been seen).⁸ Further study is needed to determine the impact of legislation, public education, and prevention programs on firearm violence.

■ TRAUMA SYSTEMS

The first document to set criteria for categorizing hospitals as trauma centers was promulgated by the American College of Surgeons (ACS) Committee on Trauma in 1976.⁹ As other groups recognized the importance of structured trauma care, legislation and funding to promote the development of trauma systems grew. In the early 1990s, the Health Resources and Services Administration developed the Model Trauma Care System Plan, a well-designed framework for progress measurement in trauma systems. Unfortunately, this program lost funding in 2006. As of 1998, 38 states and the District of Columbia had at least one critical element in place for a formal trauma system.¹⁰ More up-to-date data are not readily available because the program was the main source for this information.

The benefit of regionalized trauma systems has been shown in multiple studies. A meta-analysis of 14 studies demonstrated an overall 15% decline in mortality due to the presence of a trauma system.¹¹ However, this apparent decline may be confounded by other factors. A nationwide study suggests that mortality reduction could not be solely attributed to the presence of a trauma system because its impact was small and statistically not significant. Rather, the presence of a primary seat belt law and mean per capita income were associated with a reduction in occupant mortality rates, whereas rural population and speed limits faster than 65 mph were associated with an increase in mortality rates.¹² As new trauma systems mature, more research will be needed to guide implementation of new system strategies to further reduce morbidity and mortality from traumatic injury.

One goal of the out-of-hospital trauma system is to transport the patient to the closest appropriate facility in a timely manner. Problems with over- and undertriage occur. Most efforts are aimed at reducing undertriage (transport of severely injured patients to lower level trauma centers), which may result in preventable morbidity and mortality from delay in definitive care. Overtriage (transport of minimally injured patients to higher level trauma centers) has no deleterious effects on patient care; however, it may contribute to unneces-

sary resource utilization and potential overcrowding in tertiary care, level 1 trauma centers. The ACS published a field triage decision scheme to assist in appropriate transport decisions (Fig. 33-1).¹⁰

Limitations to effective use of regionalized trauma systems remain. Hospital crowding, ambulance diversion, lack of specialist on-call availability, and reimbursement issues all contribute. In addition, from 1995 to 2005, the number of hospital emergency departments declined from 4176 to 3795.¹³ Trends such as this increase the burden of emergency care on those remaining hospitals, potentially compromising care. Future funding and legislative decisions at the state and national levels will have a significant impact on the future of quality trauma care in the United States.

■ PRINCIPLES OF DISEASE

The emergency physician faces significant clinical uncertainty when a multiple trauma patient presents to the emergency department. Much of the diagnostic dilemma and subsequent evaluation can be directed based on knowledge of the mechanism of injury. Although mechanisms of injury alone are not good predictors of major trauma,¹⁴ common patterns of injuries can be anticipated and specifically assessed in emergency department patients. Table 33-1 outlines several blunt trauma mechanisms of injury with potential associated clinical findings.

Basic anatomic principles are useful in the assessment of patients with penetrating trauma. In contrast to penetrating trauma from knife wounds, in which injuries can be expected along the track of the weapon, gunshot wounds depend on several factors. The amount of tissue damage is related to the kinetic energy of the bullet imparted to the patient. The bullet weight (caliber) and velocity (determined by the weapon) play a role in anticipating injuries. Gunshot wounds result in trauma to the surrounding tissue by direct laceration, crush injury, shock waves and cavitation—the displacement of tissue forward and radially. Because of these dynamic forces, the emergency physician should anticipate more widespread injuries from high-velocity weapons, such as rifles, than from low-velocity weapons, such as handguns. Similar to knife wounds, handguns generally cause injury based on direct laceration and crush generated by the missile along its track. Shotgun wounds from close range are characterized by massive tissue injury.

Injury patterns can differ significantly between adults and children subjected to similar mechanisms of trauma. The major anatomic distinctions relate to the smaller size and surface area, larger head-to-body ratio, and less protected abdominal cavity of the child. As a result, children are more vulnerable to multisystem injury in blunt trauma, more frequently sustain significant head and intra-abdominal injuries, and are more at risk for hypothermia.¹⁵⁻¹⁸

Trauma is the seventh leading cause of death in patients older than the age of 65 years.¹⁹ Elder patients commonly sustain extremity, craniofacial, and closed head injuries. The majority of these occur as the result of a fall or an MCV. Elder trauma patients typically have normal, age-related changes in organ system function related to decreased cardiopulmonary functional reserve, decreased renal function, decreased bone density, and cerebral atrophy. These changes can increase susceptibility to sheer forces and other aspects of trauma.²⁰

Comorbidities and preexisting medication use further complicate the management of elder trauma patients. Lower extremity weakness, gait disturbances, decreased visual acuity, and the use of psychotropics, antihypertensives, and sedatives have been associated with falls in elders, resulting in major injury.²¹ The use of these medications, particularly antihyper-

tensives, should not be considered causative in trauma patients with hypotension until acute hemorrhage is assessed and managed. In addition, anticoagulants, antiplatelet drugs, and aspirin are commonly prescribed, and their effects should be suspected and reversed if possible in elder trauma patients.

■ MANAGEMENT

Out-of-Hospital

Management of the trauma patient frequently begins prior to arrival in the emergency department by first responders. The goals of out-of-hospital care include intervening in immediately life-threatening injuries, preventing additional injury, and rapid transport to trauma centers for definitive care. Although accepted as tenets of out-of-hospital care, controversy exists regarding each of these goals.

The majority of life-threatening injuries that require intervention by out-of-hospital providers are related to airway, breathing, and circulation (the ABCs). Preventing aspiration of gastric contents and providing adequate tissue oxygenation are the primary goals of endotracheal intubation. Although controversy exists regarding the use of out-of-hospital rapid sequence induction,²²⁻²⁷ securing an unprotected airway is essential in this phase of trauma management. Tension pneumothorax is the fundamental threat to adequate ventilation and requires immediate needle thoracostomy. Systemic hypotension with impaired end-organ perfusion mandates treatment in the trauma patient, despite the debate surrounding controlled hypotension versus aggressive fluid resuscitation.

Preventing additional injury requires an awareness of not only clinically evident abnormalities but also potentially more serious injuries. Coordinated extrication and transport with rigid cervical immobilization, complete spinal precautions, intensive hemodynamic monitoring, and stabilization of fractures to prevent neurovascular compromise are examples of assuming the most serious injuries exist in multiple trauma patients.

In the United States, rapid transport to the nearest appropriate facility is one of the fundamental concepts in trauma management. Much of the controversy regarding various out-of-hospital approaches to the ABCs is rooted in attempts to limit transport times and avoid further infringement on the “golden hour” of trauma care. In contrast, physician-operated emergency medical service (EMS) systems more aggressively manage airway and ventilatory issues and are more likely to commit out-of-hospital time resources to establishing hemodynamic stability prior to transport.^{28,29} Rural EMS systems in the United States, where transport times may be prolonged because of the distance to a receiving facility, may benefit from more advanced interventions such as rapid sequence induction/intubation and more aggressive fluid resuscitative measures.

Emergency Department

General Principles

Care of the multiple trauma patient is complex and involves the coordination of multiple providers, including EMS personnel, emergency physicians, nurses, technicians, trauma surgeons, and subspecialists. A systematic and comprehensive approach to these patients is necessary, incorporating providers from each discipline. Advanced Trauma Life Support (ATLS) guidelines delineate the use of defined trauma response teams, with providers performing assessments, diagnostics, and interventions simultaneously. With this approach,

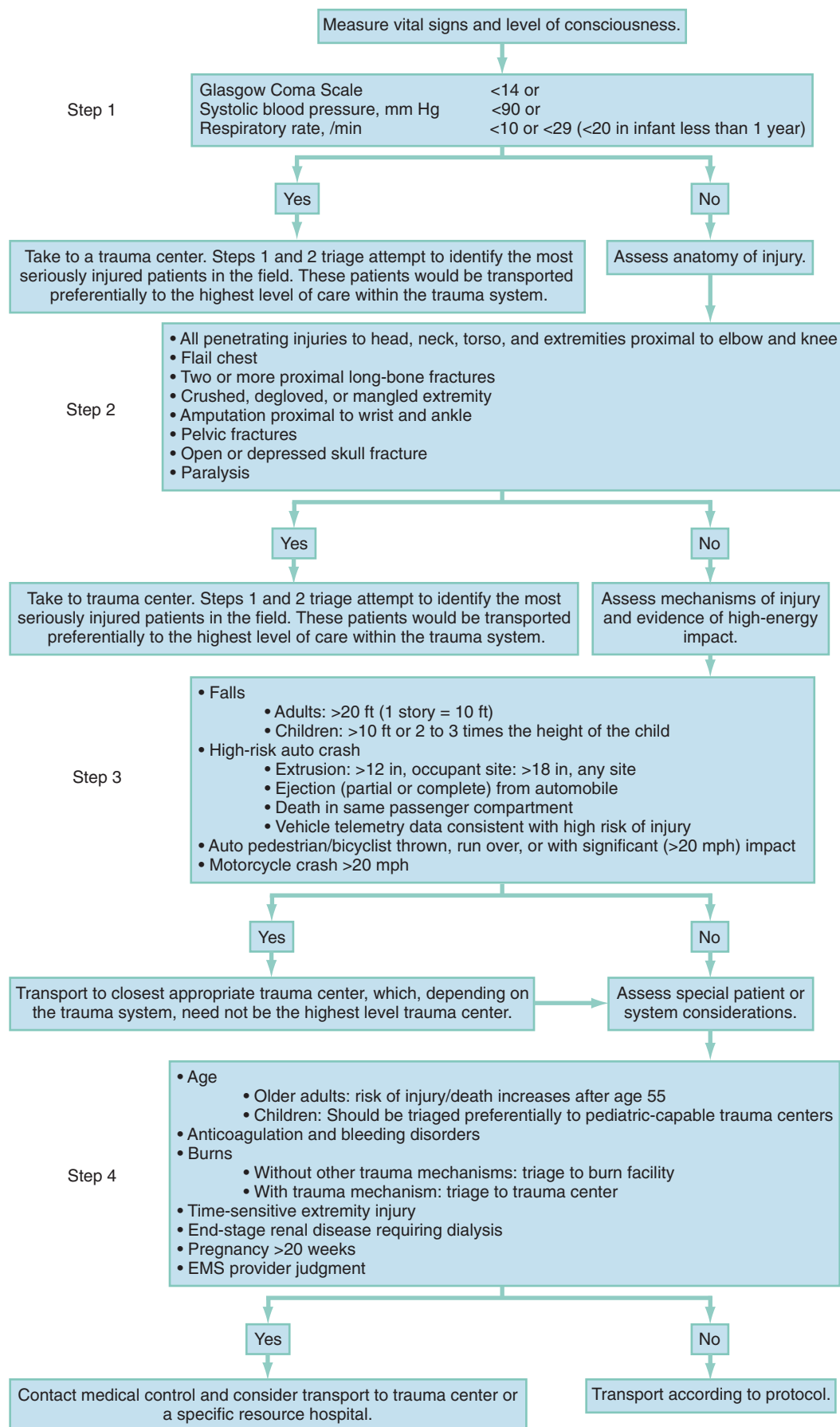


Figure 33-1. Triage decision scheme. Redrawn from American College of Surgeons, Committee on Trauma: Resources for the Optimal Care of the Injured Patient. Chicago, American College of Surgeons, 2006.

Table 33-1 Blunt Trauma Mechanisms and Associated Injuries

MECHANISM OF INJURY	ADDITIONAL CONSIDERATIONS	POTENTIAL ASSOCIATED INJURIES
Motor vehicle collisions		
Head-on collision		Facial injuries Lower extremity injuries Aortic injuries
Rear-end collision		Hyperextension injuries of cervical spine Cervical spine fractures Central cord syndrome
Lateral (T-bone) collision		Thoracic injuries Abdominal injuries—spleen, liver Pelvic injuries Clavicle, humerus, rib fractures
Rollover	Greater chance of ejection Significant mechanism of injury	Crush injuries Compression fractures of spine
Ejected from vehicle	Likely unrestrained Significant mortality	Spinal injuries
Windshield damage	Likely unrestrained	Closed head injuries, coup and countercoup injuries Facial fractures Skull fractures Cervical spine fractures Thoracic injuries
Steering wheel damage	Likely unrestrained	Sternal and rib fractures, flail chest Cardiac contusion Aortic injuries Hemo/pneumothoraces
Dashboard involvement/damage		Pelvic and acetabular injuries Dislocated hip
Restraint/seat belt use		
Proper three-point restraint	Decreased morbidity	Sternal and rib fractures, pulmonary contusions
Lap belt only		Chance fractures, abdominal injuries, head and facial injuries/fractures
Shoulder belt only		Cervical spine injuries/fractures, “submarine” out of restraint devices (possible ejection)
Airbag deployment	Front-end collisions Less severe head/upper torso injuries Not effective for lateral impacts More severe injuries in children (improper front seat placement)	Upper extremity soft tissue injuries/fractures Lower extremity injuries/fractures
Pedestrian versus automobile		
Low speed (braking automobile)		Tibia and fibula fractures, knee injuries
High speed		Waddle’s triad—tibia/fibula or femur fractures, truncal injuries, craniofacial injuries “Thrown” pedestrians at risk for multisystem injuries
Bicycle		
Automobile related		Closed head injuries “Handlebar” injuries Spleen/liver lacerations Additional intra-abdominal injuries Consider penetrating injuries
Nonautomobile related		Extremity injuries “Handlebar” injuries
Falls		
Vertical impact	LD ₅₀ 36–60 ft	Calcaneal and lower extremity fractures Pelvic fractures Closed head injuries Cervical spine fractures Renal and renal vascular injuries
Horizontal impact		Craniofacial fractures Hand and wrist fractures Abdominal and thoracic visceral injuries Aortic injuries

Table 33-2

American College of Surgeons Requirements for the Presence of a Surgeon in Major Resuscitations

A surgeon should be present in the emergency department on trauma patient arrival or within 15 minutes if any of the following major criteria are found:

- Confirmed hypotension (systolic blood pressure < 90 mm Hg)
- Respiratory compromise requiring intubation
- Penetrating gunshot wound to the neck, chest, abdomen, or pelvis
- Glasgow Coma Scale score of <8 attributed to trauma
- Discretion of emergency physician

the physician team leader coordinates the management of the patient, considering the presence of life- or limb-threatening injuries in a sequential manner.

For level 1 trauma centers, the ACS mandates the presence of a surgeon or an appropriate representative (e.g., a fourth- or fifth-year surgery resident) to be present in the hospital 24 hours a day. The attending surgeon is expected to be present in the emergency department no later than 15 minutes after the emergency department arrival of trauma patients (Table 33-2).¹⁰ As the specialty of emergency medicine has evolved and the number of residency-trained and board-certified emergency physicians has increased, the need for a surgeon for all trauma patients has been increasingly debated.³⁰⁻³⁴ Optimal patient care is best provided in a collaborative, patient-centered manner.

The priorities in the treatment of trauma patients are similar to those for any other life-threatening condition. Securing the airway, maintaining ventilation, controlling hemorrhage, and treating shock are first priorities because of their crucial importance for survival. The emergency physician should give consideration to the worst possible injury and act accordingly until the diagnosis is confirmed or excluded. The consequences of an overzealous evaluation are more acceptable than those of a missed injury. Based on ATLS, the phases of emergency department care are divided into the primary survey with interventions, initial diagnostics and imaging, the secondary survey, and disposition.³⁵

Primary Survey

Airway and Breathing. Proper assessment and management of airway, oxygenation, and ventilation in the trauma patient is of utmost importance but can be challenging. In a review of 44,404 trauma patient admissions and 2594 deaths, airway management was responsible for 16% of preventable errors likely contributing to trauma mortality.³⁶ The goals of airway management are threefold: airway protection, adequate oxygenation, and adequate ventilation.

Airway protection is necessary in a variety of trauma patients. Airway obstruction mandates immediate intervention. Obstruction from debris, blood, or vomitus may be easily removed with suction. Neck or facial trauma may be more problematic. Swelling, distorted anatomy, and hematoma formation may all contribute to impending obstruction. Early airway control is safest because these conditions may rapidly worsen. Inability to adequately protect the airway, such as in patients with depressed levels of consciousness, is another indication for intervention. Airway control is recommended in patients with significant head injury (GCS ≤ 8).³⁷

As a general rule, all trauma patients should be placed on supplemental oxygen. Adequate oxygenation has a direct

effect on outcome of many trauma patients. In head-injured patients, hypoxia in both out-of-hospital and hospital phases of resuscitation has been associated with poorer outcomes.³⁸⁻⁴¹ Hypoxia may also worsen outcome in spinal cord injury.⁴² Maintenance of Pao₂ greater than 60 mm Hg has been recommended.⁴³ Inadequate ventilation, which may lead to respiratory acidosis, can be noted by the rate and quality of respirations. Signs of inadequate oxygenation may be more subtle and include agitation and restlessness. Assessment for injury that may compromise oxygenation, ventilation, or both requires careful inspection and auscultation of the chest. Signs of such compromising injury include increased work of breathing, tachypnea, penetrating wounds, flail segments, tracheal deviation, and distended neck veins. In determining the need for more aggressive airway management, these data are put into the context of the patient's overall presentation. Certain ventilatory problems, such as pneumothorax or hemothorax, may require tube thoracostomy in addition to intubation. Early intervention is preferable in the tenuous patient.

Once the decision to intubate the patient has been made, many considerations must be taken into account. If the patient's condition allows, a brief neurologic examination prior to paralytics can be helpful in determining the extent of injury. Also, cervical spine injury precautions should be considered for patients with blunt trauma and gunshot wounds to the neck. Rapid sequence induction and orotracheal intubation with in-line cervical stabilization provides a safe method. There have been no reported cases of spinal cord injury from orotracheal intubation if proper stabilization has been applied.⁴⁴ There are many approaches to airway control, and many alternative devices, such as the flexible fiberoptic scope, intubating laryngeal mask airway, and video-assisted laryngoscope, are now available to assist in intubation. The choice will be based on clinical scenario and physician comfort. A review of the literature did not reveal one superior modality for intubation of the patient with suspected spinal cord injury.⁴⁵ Nasotracheal intubation is generally undesirable in trauma patients due to the potential for abrupt rises in intracranial pressure, a higher complication rate than that for orotracheal intubation,⁴⁶ and relative contraindications of severe midface trauma or severe basilar skull fracture.

Surgical airways are indicated in cases of failure or contraindication to orotracheal or nasotracheal intubation. Cricothyrotomy is the preferred method. A variety of devices for percutaneous cricothyrotomy are available that show good success rates and are easy to use.⁴⁷⁻⁴⁹ If there is any question of the ability to identify the cricothyroid membrane, the traditional surgical approach with a vertical incision should be used.

Circulation. Assessment of hemodynamics and circulatory status are of critical importance after the airway has been evaluated and controlled and adequate ventilation has been ensured. The assessment of circulation is multidimensional. Clinical indicators of adequate perfusion include mental status, skin color and temperature, heart rate, blood pressure, and capillary refill. A normal finding of any single sign does not rule out shock. Mental status changes associated with hypoperfusion can include anxiety, agitation, or sedation. Cool, pale skin or extremities with delayed capillary refill suggest inadequate perfusion and shock. A normal heart rate and/or blood pressure can be present despite significant hemorrhage. Conversely, tachycardia may be seen without evidence of significant volume loss.

Control of external hemorrhage is crucial. Traditionally, direct pressure to external bleeding sites has been advocated and the use of tourniquets has been discouraged. The use of direct pressure on bleeding remains first-line therapy; recent

data have suggested the more liberal use of tourniquets for massive extremity bleeding that is not easily controlled.⁵⁰⁻⁵³ Similarly, recent studies of newer hemostatic agents have shown potential application both in combat and out-of-hospital settings.⁵⁴⁻⁵⁷

Intravenous access is required early in the assessment of circulation. Two large-bore (14- or 16-gauge) intravenous catheters are recommended. Routine intravenous access may be difficult or unobtainable in certain cases. Intraosseous vascular access can be obtained rapidly in both pediatric⁵⁸ and adult⁵⁹ patients and allows the safe infusion of large amounts of fluid or blood products. Compact, battery-operated intraosseous drills have recently been introduced. Ultrasound-guided peripheral venous access should be considered in patients when blind peripheral attempts are unsuccessful.⁶⁰⁻⁶² Central venous access may also be indicated in the appropriate clinical scenario or based on physician discretion. The use of ultrasound has been shown to increase successful vein cannulation and decrease complications in the placement of central venous lines.⁶³⁻⁶⁷ Central venous pressure measurements may be used to direct resuscitative efforts but should not delay definitive care.

The choice of fluids for resuscitation includes crystalloid, colloid, and blood products. Fluid replacement is generally based on a 3:1 ratio of fluids to blood loss. There are few clinically significant differences between lactated Ringer's and normal saline. The debate regarding the choice of fluid for resuscitation is ongoing. No indisputable advantages of colloids have been demonstrated. Therefore, the less expensive and more readily available crystalloids are the routine mainstay as first line of therapy. No clear benefit to the use of hypertonic saline has been established.⁶⁸⁻⁷¹ Current ATLS guidelines standardize the ratio of replacement fluids to loss and recommend 2 L of crystalloid be infused in all patients in shock, followed by blood products. O-positive blood should be used except in women of childbearing age. Type-specific blood should be used when available, but emergent transfusion should not be delayed.

The concept of "permissive hypotension" is based on the concern that resuscitation to normal blood pressures may increase bleeding from a site that is contained and not actively hemorrhaging.⁷² Clinically, restoration of normal blood pressure is delayed until active bleeding foci are ruled out. Although data exist to support this strategy,^{72,73} a Cochrane review of the six available clinical trials meeting inclusion criteria did not support (or disprove) the use of early or larger volume intravenous fluids in uncontrolled bleeding.⁷⁴ Permissive hypotension is contraindicated in the management of traumatic brain injury because of the risk of hypoperfusion.^{75,76}

The extended focused abdominal sonography in trauma (eFAST) examination should be performed on all patients as an adjunct to the assessment of circulation. The presence or absence of free intra-abdominal, -thoracic, or -pelvic hemorrhage or pneumothorax diagnosed by ultrasound will direct the management of trauma patients. Pericardial effusions or tamponade can be readily identified. In addition, ultrasound evaluation of the inferior vena cava may be useful in the overall assessment of fluid status in resuscitation.⁷⁷⁻⁸¹

Disability. A rapid assessment of the patient's neurologic status is necessary early in the emergency department course. The Glasgow Coma Scale score is commonly employed (see Chapter 38).

Exposure. The final phase of the primary survey is completely undressing the patient in order to assess for inconspicuous injuries. Special attention to the axilla, perineum, and skin folds is needed. Preventing hypothermia is essential in this

phase. Blankets, warming lights, and warm fluids may be used as indicated.

Secondary Survey

The goals of the secondary survey are to obtain pertinent historical data about the patient and injury as well as evaluate and treat injuries not found on the primary survey. An AMPLE (allergies, medications, past medical history, last meal, environments and events) history should be obtained. Frequent reassessment of the ABCs throughout the emergency department phase of management is necessary. If deterioration occurs, a complete reevaluation of the primary survey should be initiated. Features of the secondary survey and management are listed in Table 33-3.

Table 33-3 Secondary Survey

REGION/SYSTEM	ASSESSMENT/EXAMINATION
General	Level of consciousness GCS score Specific complaints
Head	Pupils (size, shape, reactivity, visual fields) Contusions Lacerations Evidence of skull fracture (hemotympanum, Battle's sign, raccoon eyes, palpable defects)
Face	Contusions Lacerations Midface instability Malocclusion
Neck (maintain cervical immobilization)	Penetrating injury/lacerations Tracheal deviation Jugular venous distention Subcutaneous emphysema Hematoma Midline cervical tenderness
Chest	Respiratory effort/excursion Contusions Lacerations Focal tenderness/crepitus Subcutaneous emphysema Heart tones (muffled) Breath sounds (symmetrical)
Abdomen/flank	Contusions Penetrating injury/lacerations Tenderness Peritoneal signs
Pelvis/genitourinary	Contusions Lacerations Stability/symphyseal tenderness Blood (urethral meatus, vaginal bleeding, hematuria) Rectal examination
Neurologic/spinal cord	Midline bony spinal tenderness Mental status Paresthesias Sensory level Motor function, including sphincter tone
Extremities	Contusions Lacerations Deformity Focal tenderness Pulses Capillary refill Evaluation of compartments

Pitfalls

The goal of trauma management in the emergency department is to provide life-saving interventions and evaluate for injuries. The tenets of ATLS provide a structured approach to ensure a thorough assessment is performed. Because multiple trauma patients present with a variety of injuries from varying mechanisms, the initial focus is directed at defining the most serious pathology. The emergency physician must be aware of potential pitfalls. Studies from large trauma centers have shown that preventable deaths frequently result from human error and are related to the inability to intubate or secure the airway; delayed control of thoracic, abdominal, and pelvic hemorrhage; and several inpatient factors.^{36,82} Head-injured, unconscious, and intubated patients frequently have injuries that are not identified early in their hospital course.⁸³⁻⁸⁵ More aggressive diagnostics may be indicated in this subset of patients when history and patient-specific complaints are not obtainable. When cervical radiography is indicated, it is essential that the imaging obtained is complete and adequate. Limited radiographic series and inadequate cervical spine imaging have been shown to result in missed or delayed diagnosis of injuries.⁸⁶

Radiographic Evaluation

The approach to radiographic evaluation of the trauma patient has changed in recent years. The mantra of C-spine, chest, and pelvis radiographs for all trauma patients has been challenged, and new recommendations continue to emerge. However, in the critically injured patient, imaging of the chest and pelvis and an eFAST exam should be obtained early in the evaluation. These studies provide essential information on possible sources of hemorrhage in either the chest or abdomen or from a pelvic fracture.

Imaging of the cervical spine can usually be delayed; however, in a patient with neurologic findings and persistent moderate hypotension (e.g., 70 mm Hg), neurogenic shock should be considered. A positive finding on C-spine imaging (e.g., fracture, soft tissue swelling, and subluxation) may confirm this if other causes of hypotension have been ruled out. A single cross-table lateral cervical radiograph is not adequate to fully assess the cervical spine; in trauma patients, the sensitivity is too low to rule out fracture.⁸⁷⁻⁹⁰

Cervical spine radiographs may not be necessary in all trauma patients. The NEXUS criteria allows clinical clearance of the cervical spine in patients without posterior midline tenderness, focal neurologic deficit, altered mental status, intoxication, or distracting injury.⁹¹ The Canadian C-spine rule uses a different set of criteria and, although more complex, has higher specificity and therefore allows the elimination of a higher percentage of unnecessary x-rays.⁹² In a patient with a concerning mechanism of injury or physical examination, computed tomography (CT) scanning is more efficient and effective than plain radiography⁸⁷ and should be considered the primary imaging modality. Plain C-spine radiographs are often inadequate to identify fractures and often miss secondary injury.⁹³ In the patient with a low-risk mechanism of injury and no neurologic findings on examination, plain radiography alone may be sufficient to rule out cervical fracture.⁹⁴ If this approach is used, three views of the cervical spine (lateral, anteroposterior, and odontoid) should be obtained.⁹⁵

Imaging studies of the thoracolumbar spine and extremities can also be delayed until higher priority assessments and interventions are complete. Once the patient has been stabilized,

the clinical examination can direct whether additional radiographic examinations are necessary.

Imaging of the thorax early in the evaluation of the trauma patient can give important information about potentially life-threatening injuries. However, the dogmatic “chest x-ray (CXR) for all trauma patients” approach to radiographic evaluation of the chest has undergone some recent challenges. Plain radiography of the chest is considered an acceptable first screening modality. However, in the patient with a significant blunt mechanism of injury, CT scanning may be considered the imaging modality of choice. The sensitivity of CXR alone, *even in patients with a normal physical exam*, may be too low for use as the primary screening tool to rule out injury to the mediastinum or a blunt aortic dissection,^{96,97} and the results of those radiographs likely do not affect decision-making.⁹⁸ In patients with penetrating thoracic injury, primary CT scan, rather than repeat CXRs, can speed disposition.⁹⁹ Cost and radiation considerations must, of course, be taken into account. Other preliminary evidence suggests that some patients may not require chest imaging.¹⁰⁰ Ultrasound is gaining favor as the initial screening tool for pneumothorax and hemothorax because it shows better sensitivity and is more rapid than CXR.^{101,102} One possible approach for initial screening of the blunt trauma patient with a significant mechanism of injury could be carried out by thoracic ultrasound to identify life-threatening pneumo- or hemothorax that must be immediately managed, followed by chest CT scan to further elucidate other injuries.

Pelvis radiographs are useful in the severely injured trauma patient. Pelvic fractures can account for significant hemorrhage, and early recognition of fracture and closure of the pelvic space can mitigate hypotension in these patients. Although not validated in large multicenter studies, evidence suggests that pelvic radiographs may be omitted in patients without altered level of consciousness, complaint of pelvic pain, pelvic tenderness on examination, distracting injury, or clinical intoxication.^{103,104} Stable patients undergoing CT scan of the abdomen and pelvis can be further evaluated by bone windows of the CT scan.

As mentioned previously, the eFAST exam should take place early in the evaluation of the trauma patient, ideally as part of the primary survey. A positive scan in hypotensive patients can identify, with good sensitivity, those in need of emergent laparotomy.⁵² In addition, valuable information regarding the presence or absence of pericardial effusion can be obtained. False-negative scans can occur with hollow viscous injuries or solid organ injuries without free fluid.¹⁰⁵ A normal scan does not necessarily eliminate the need for further abdominal imaging in significantly injured patients. Further discussion and suggested algorithms are found in Chapter 42.

Laboratory Evaluation

Laboratory evaluation of the trauma patient can provide an objective measure of the adequacy of resuscitation. It also provides much needed information for proper transfusion products and the onset of coagulopathy. Finally, it provides baseline information and values for follow-up studies (e.g., tracking hemoglobin values in a patient with a nonoperative splenic injury).

Lactate and base deficit have both been used to measure adequacy of resuscitation in the trauma patient.^{106,107} New non-invasive devices, such as muscle tissue oxygenation and sublingual capnometry, have shown promise in assessing severity of shock and predicting multiorgan dysfunction and mortality.¹⁰⁸⁻¹¹⁰ Using one of these objective markers can assist in ensuring proper resuscitation of these patients.

All patients with potentially serious injuries should have blood type and screening done. Cross-matched blood should be immediately ordered for those with apparent serious hemorrhage. As transfusion requirements increase, attention should be paid to the development of coagulopathy. INR and fibrinogen should be followed in these patients. Routine electrolytes, blood urea nitrogen, creatinine, complete blood count, and pregnancy testing (when appropriate) should be obtained as well.

■ DISPOSITION

The emergency department management of the multiple trauma patient is but one critical phase of the spectrum of care. The decision to admit the patient or transfer to a tertiary care facility should be coordinated based on available resources, consultation with the trauma surgeon, and consideration of institutional and regional guidelines.

Ultimate disposition is dictated by a number of factors, including the patient's condition, the nature of the injury, and the availability of surgeons, subspecialists, and anesthesiologists. Possible dispositions include transfer to the operating room, admission to the surgical service, limited observation in the emergency department, or transfer to another hospital. The level of care and monitoring established in the emergency department should be maintained throughout transfer. All equipment and medications needed for resuscitation and maintenance of vital functions should be available during the transfer, as should qualified personnel to oversee the patient's care.

In cases of interhospital transfers, all arrangements should be carefully coordinated by physicians at the two institutions. Stabilizing measures are begun before the patient's transfer, but decompensation in transit should be anticipated. Qualified personnel and necessary resuscitative equipment must accompany the patient. The compelling reason for transferring a patient with life-threatening trauma is the lack of resources or personnel to care for the patient's particular injuries. Transfer should not be delayed for nonessential diagnostic procedures. All documentation and results of ancillary testing should accompany the patient in transfer.

In certain circumstances, the multiple trauma patient may not need admission or interhospital transfer. The decision to discharge these patients must be evaluated carefully because many traumatic injuries may present in a delayed manner. When discharge is considered, thorough emergency department evaluation is necessary with resources in place to ensure an optimal outcome: surgical consultation where appropriate, attending radiologist support for radiographic image interpretation, and timely, scheduled follow-up as an outpatient.

■ SPECIAL CONSIDERATIONS

The role of emergency department thoracotomy (EDT) has become more selective in order to limit futile resuscitation efforts and minimize risk to providers. Patients with penetrating trauma who have cardiac arrest while in transport or the emergency department are most likely to benefit from EDT. In contrast, cardiac arrest patients with blunt trauma, prolonged cardiopulmonary resuscitation (CPR), or delayed transport times generally have dismal outcomes not altered by EDT.¹¹¹⁻¹¹³ Most institutions have protocols in place outlining criteria for which EDT would be performed. The National Association of EMS Physicians and the ACS Committee on Trauma have published guidelines for withholding or terminating resuscitation efforts in out-of-hospital traumatic cardiac arrest patients. As a result, these guidelines often limit the

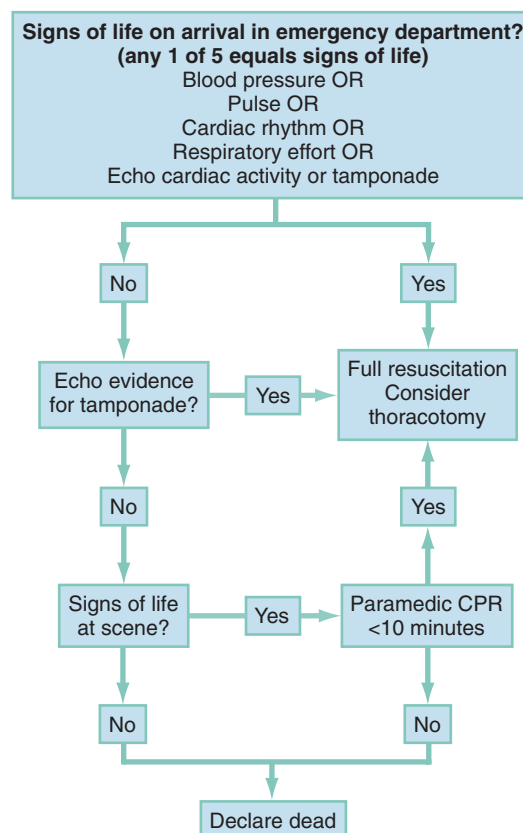


Figure 33-2. Penetrating trauma emergency department thoracotomy algorithm.

transport of patients who would not likely benefit from EDT. Patients who may not be transported include any blunt trauma patient without vital signs at the scene, apneic or pulseless penetrating trauma victims without other signs of life, patients receiving more than 15 minutes of CPR, or patients with transport times of more than 15 minutes after arrest.¹¹⁴ Suggested algorithms for the application of EDT are outlined in [Figures 33-2 and 33-3](#).

When performed, the goal of EDT is to manage rapidly correctable traumatic injuries and allow for transfer to definitive operative intervention. The use of ultrasound should be employed to assist in determining the presence or absence of pericardial effusions and tamponade. When the chest is open, a number of therapeutic measures can be undertaken, depending on the injuries present. After identifying the phrenic nerve, tamponade should be relieved by pericardotomy. Cardiac injuries are sutured or hemorrhage is controlled with digital pressure or placement of a Foley catheter balloon. Compressing or cross-clamping the pulmonary hilum can control major pulmonary bleeding, but damage to the bronchus is likely to occur and may require repair if the patient survives. The descending aorta is compressed to maximize coronary and cerebral perfusion. The aorta should remain clamped until hemorrhage has been controlled and volume replaced. Open cardiac massage can also be performed.

Acknowledgments

We acknowledge the previous authors of this chapter, Drs. Susan L. Gin-Shaw and Robert C. Jordan.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.

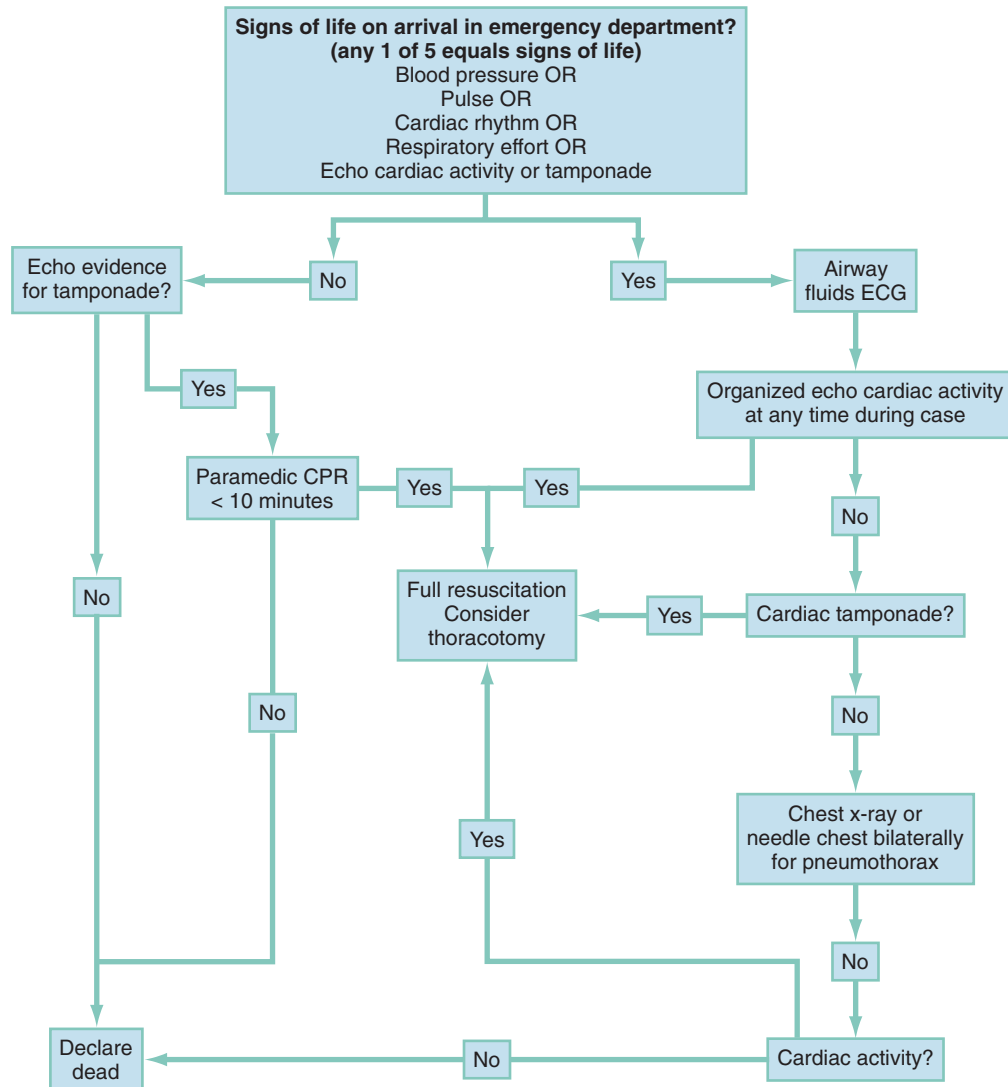


Figure 33-3. Blunt trauma emergency department thoracotomy algorithm.

Trauma in Pregnancy

Kriti Bhatia and Hilarie Cranmer*

PERSPECTIVE

Trauma occurs in 6 to 7% of all pregnancies. It is the leading cause of maternal death due to nonobstetric causes, accounting for close to 50% of fatalities in pregnant women.¹ The most common causes of injury in pregnancy, in order of frequency, that result in emergency department (ED) visits are motor vehicle crashes (MVCs), interpersonal violence, and falls.²⁻⁶ Patients with penetrating injuries present more frequently to EDs in inner city medical centers.⁷ Of note, 8% of women, aged 15 to 40, admitted to a trauma center do not yet know they are pregnant.⁸ Commonly used thresholds of fetal viability are an estimated gestational age of 24 to 26 weeks or an estimated fetal weight of 500 g. Only viable fetuses are monitored, because no obstetric intervention will alter the outcome with a previable fetus.⁹ Counseling on proper seatbelt and alcohol use and screening for interpersonal violence may help to reduce the morbidity and mortality rates for pregnant patients. Although the essential principles of trauma management remain unchanged in the pregnant patient, a number of special points need to be considered. Pregnancy causes alterations in physiology and anatomy that affect multiple organ systems. Although there are two lives involved, maternal life takes priority.

PRINCIPLES OF DISEASE—CHANGES OF PREGNANCY

Physiology

Cardiovascular

The normal cardiovascular changes of pregnancy can alter the presentation of shock and vascular events (Table 34-1).

Some Alterations Mimic Shock. Blood pressure declines in the first trimester, levels out in the second trimester, and then returns to nonpregnant levels during the third trimester. The decline in systole is small, 2 to 4 mm Hg, whereas diastole falls 5 to 15 mm Hg. Heart rate increases in pregnancy but does not rise by more than 10 to 15 beats per minute above baseline (mean of approximately 90 beats/min).¹⁰

A major contributor to maternal hypotension is the supine hypotensive syndrome. After 20 weeks' gestation, the uterus has risen to the level of the inferior vena cava, resulting in compression when the mother is supine. Such caval obstruction

diminishes cardiac preload, which can decrease cardiac output 28%, resulting in reduced systolic blood pressure by 30 mm Hg.¹⁰ In late pregnancy, it is common for the inferior vena cava to become completely occluded when the pregnant patient is supine. Attempts at resuscitation will be improved if compression is relieved. To determine whether observed hypotension is related to positioning, the pregnant woman's pelvis can be tilted so that the uterus is displaced from the inferior vena cava, which is to the patient's left, unless otherwise prevented due to other injuries (e.g., hip fracture). The uterus can also be manually pushed to the left by using two hands and pushing it toward the patient's head. One study found that tilting limited to only about 15 degrees may only partially resolve vena caval obstruction;¹¹ thus, maintaining a position between 15 and 30 degrees is optimal. Elevating the patient's legs, where blood may pool due to increased capacitance, will improve venous return.

Similarly, central venous pressure (CVP) measurements can be lowered in the last two trimesters by inferior vena caval compression. Normal CVP during pregnancy is approximately 12 mm Hg.

Alterations That May Mask Hypovolemic Shock. Blood volume gradually increases during pregnancy, starting at 6 to 8 weeks' gestation, to as much as 45% above normal, peaking at 32 to 34 weeks' gestation. Blood volumes become increasingly larger for multigravidas, twins, triplets, and quadruplets.¹² With this increased circulatory reserve, clinical signs of maternal hypotension from acute traumatic bleeding may be delayed.

Some Alterations Can Exacerbate Traumatic Bleeding. By the beginning of the second trimester and throughout the remaining pregnancy, cardiac output is increased 40%, to 6 L/min.¹⁰ Blood flow to the uterus increases from 60 mL/min before pregnancy to 600 mL/min at term.¹³ This hyperdynamic state is needed to maintain adequate oxygen delivery to the fetus. Because the mother's total circulating blood volume flows through the uterus every 8 to 11 minutes at term, this organ can be a major source of blood loss when injured. By the third trimester there is also marked venous congestion in the pelvis and lower extremities, increasing the potential for hemorrhage from both bony and soft tissue pelvic injuries.

Compression of the lower abdominal venous system by the gravid uterus increases peripheral venous pressure and volume in the legs, creating the potential for brisk blood loss from leg wounds.

This alteration can play an important role in procedures that may be necessary for maternal resuscitation, such as central venous catheter placement.

*The contributors would like to sincerely thank John D. G. Neufeld, MD, for his previous work on this chapter.

Table 34-1 Hemodynamic Changes of Pregnancy (Mean Values)

PARAMETER	NONPREGNANT	TRIMESTER 1	TRIMESTER 2	TRIMESTER 3
Heart rate (beats/min)	70	78	82	85
Systolic blood pressure (mm Hg)	115	112	112	114
Diastolic blood pressure (mm Hg)	70	60	63	70
Cardiac output (L/min)	4.5	4.5	6	6
Central venous pressure (mm Hg)	9.0	7.5	4.0	3.8
Blood volume (mL)	4000	4200	5000	5600
Hematocrit without iron (%)	40	36	33	34
Hematocrit with iron (%)	40	36	34	36
White blood cell (cell/mm ³)	7200	9100	9700	9800

Data from de Swiet M: The cardiovascular system. In Hytten F, Chamberlain G (eds): Clinical Physiology in Obstetrics. Oxford, UK, Blackwell Scientific Publications, 1980, pp 3–42; Colditz RB, Josey WE: Central venous pressure in supine position during normal pregnancy. Comparative determinations during first, second and third trimesters. *Obstet Gynecol* 36:769, 1970; Letsky E: The haematological system. In Hytten RF, Chamberlain G (eds): Clinical Physiology in Obstetrics. Oxford, UK, Blackwell Scientific Publications, 1980, pp 43–78; and Cruikshank DP: Anatomic and physiologic alterations of pregnancy that modify the response to trauma. In Buchsbaum HJ (ed): Trauma in Pregnancy. Philadelphia, WB Saunders, 1979, pp 21–39.

Pulmonary

The term pregnant woman has a significantly reduced oxygen reserve. This effect comes from a 20% reduction in functional residual capacity caused by diaphragm elevation and a 15% increase in oxygen consumption related to the growing fetus, uterus, and placenta.¹⁴ Archer and Marx observed that mean arterial oxygen tension dropped by 29% in term pregnant women during 60 seconds of apnea but just 11% in nonpregnant women. Labor accelerates this decline by a further 7%.¹⁵ Additionally, minute ventilation increases, leading to hypocapnea. Therefore, an arterial partial pressure of carbon dioxide (Paco₂) of 35 to 40 mm Hg may indicate inadequate ventilation and impending respiratory decompensation in the pregnant patient. At signs of respiratory compromise or hypoxia, endotracheal intubation should be considered, as maternal hypoxia rapidly leads to fetal hypoxia, distress, and possibly demise. There are no contraindications to rapid sequence intubation during pregnancy. Bag-valve-mask ventilation is more difficult in the pregnant patient.

Gastrointestinal

Gastroesophageal sphincter response is reduced in pregnancy and gastrointestinal motility is decreased, both of which increase the possibility of aspiration during reduced levels of consciousness and intubation.¹⁶ The stomach's increased acid production in pregnancy makes aspiration more ominous than usual.¹⁷ Therefore early gastric decompression should be considered under appropriate circumstances.

Anatomic Changes in Pregnancy

The uterus remains an intrapelvic organ until approximately the 12th week of gestation. It reaches the umbilicus by 20 weeks and the costal margins by 34 to 36 weeks. It grows from a 7-cm, 70-g organ to a 36-cm, 1000-g structure at term. As a result of this growing mass, the normal anatomic location and function of multiple structures are altered.

The diaphragm progressively rises an extra 4 cm in pregnancy with compensatory flaring of the ribs.¹⁸ Pneumothorax may be exacerbated and tension pneumothorax can develop more quickly in pregnancy because of this diaphragm elevation, combined with pulmonary hyperventilation. For thoracostomies done in the third trimester, the chest tube should be placed one or two interspaces higher than the usual fifth interspace site to allow for diaphragm elevation.

Abdominal viscera are pushed upward by the enlarging uterus, resulting in altered pain location patterns. The gravid uterus itself tends to protect abdominal organs from trauma but substantially increases the likelihood of bowel injury with penetrating trauma to the upper abdomen. Conversely, the upward displacement of the bowel makes it less susceptible to blunt trauma. The stretching of the abdominal wall modifies the normal response to peritoneal irritation. Therefore, expected muscle guarding and rebound can be progressively blunted as pregnancy approaches term, despite significant intra-abdominal bleeding and organ injury, another factor that may lead to underestimation of the extent and gravity of maternal trauma.

Total weight for the gravid uterus and its contents typically reaches 4500 g.¹³ In the first trimester, the bony pelvis shields the uterus. After the third month, the uterus rises out of the pelvis and becomes vulnerable to direct injury. The bladder is also displaced into the abdominal cavity beyond 12 weeks' gestation, thereby becoming more vulnerable to injury. Like the uterus, the bladder becomes hyperemic, and injury may lead to a marked increase in blood loss compared with similar injury in a nonpregnant patient.

Imaging studies may show ureteral dilation that can be physiologic secondary to smooth muscle relaxation or caused by compression from the gravid uterus. Thus hydronephrosis is not necessarily pathologic. The ligaments of the symphysis pubis and sacroiliac joints are loosened during pregnancy. As a result, a baseline diastasis of the pubic symphysis may exist that can be mistaken for pelvic disruption on a radiograph.

Changes in Laboratory Values with Pregnancy

The physiologic anemia of pregnancy, resulting from a 48 to 58% increase in plasma volume and only an 18% increase in red blood cells, causes hematocrits of 32 to 34% by the 32nd to 34th week. Despite the lower hematocrit, there is actually an overall increase in oxygen-carrying capacity because of an increased total red blood cell mass.

Placental progesterone directly stimulates the medullary respiratory center, producing a Paco₂ of 30 mm Hg from the second trimester until term. The subsequent compensatory lowering of serum bicarbonate to 21 mEq/L slightly reduces blood-buffering capacity for stress situations.¹⁴ A Paco₂ of 40 mm Hg in the latter half of pregnancy reflects inadequate

ventilation and potential respiratory acidosis that could precipitate fetal distress.

Electrocardiographic changes include a left-axis shift averaging 15 degrees, caused by diaphragm elevation. Consequently, flattened T waves or Q waves in leads III and augmented voltage unipolar in left foot lead may be seen.

■ CLINICAL FEATURES OF TRAUMA IN PREGNANCY

Blunt and Penetrating Trauma

The findings of the physical examination in the pregnant woman with blunt trauma are not reliable in predicting adverse obstetric outcomes. However, risk factors that are significantly predictive of contractions or preterm labor include gestational age greater than 35 weeks, assaults, and pedestrian collisions. In gravid patients, penetrating trauma of the abdomen has an increased likelihood of causing injuries of the bowel, liver, or spleen.

Fetal mortality rates range between 4 and 40% after maternal trauma, with most likely causes of fetal death occurring from placental abruption, maternal shock, and maternal death, in order of decreasing incidence. Risk factors significantly predictive of fetal death included ejections, motorcycle and pedestrian collisions, maternal death, maternal tachycardia, abnormal fetal heart rate, lack of restraints, and an Injury Severity Score greater than 9.⁹

Unbelted or improperly restrained pregnant women are twice as likely to experience excessive maternal bleeding, and fetal death is three times more likely to occur.^{19,20} For low- to moderate-severity crashes (constituting 95% of all MVCs), proper restraint use, with or without air bag deployment, generally leads to acceptable fetal outcomes. For high-severity crashes, even proper restraint does not improve fetal outcome.²¹

Pregnant crash-test-dummy trials show that improper placement of the lap belt over the pregnant abdomen causes a three- to fourfold increase in force transmission through the uterus. The lowest force transmission readings through the uterus occur when a three-point seat belt is used properly. For correct position, the lap belt should be placed under the gravid abdomen, snugly over the thighs, with the shoulder harness off to the side of the uterus, between the breasts and over the midline of the clavicle.²² Women who receive information on seat belt use during pregnancy from a health care worker are statistically more likely to use seat belts and to use them properly than uninformed controls.²³

Interpersonal Violence

Although it has been previously documented that intimate partner violence against women affects one in four U.S. women, and numerous health consequences have been associated with being a victim of such violence, a recent study by Silverman and colleagues conclusively demonstrates that physical abuse from husbands or boyfriends compromises a woman's health during pregnancy, as well as her likelihood of carrying a child to term and the health of her newborn.²⁴ Women experiencing abuse in the year prior to or during a recent pregnancy were 40 to 60% more likely than nonabused women to report high blood pressure, vaginal bleeding, severe nausea, kidney or urinary tract infections, and hospitalization during this pregnancy. Abused women were 37% more likely to deliver preterm, and children of abused women were 17% more likely to be born underweight. These conditions pose grave health risks to newborns, and children born to abused

mothers were over 30% more likely than other children to require intensive care at birth.²⁴⁻²⁷ Physicians detect only 4 to 10% of cases, which supports the need for routine screening for interpersonal violence in pregnant patients.²⁷

Falls

Falls become more prevalent after the 20th week of pregnancy.² Protuberance of the abdomen, loosening of pelvic ligaments, strain on the lower back, and fatigability contribute to this problem. In a given pregnancy, about 2% of pregnant women sustain repeated direct blows to the abdomen because of falling more than once. Although repeated falls often trigger premature contractions, they seldom result in immediate labor and delivery.²⁸

Penetrating Trauma

The gravid uterus alters injury patterns to the mother. There is an increased probability of harm (approaching 100%) to the bowel, liver, or spleen if the entrance of the penetrating object is in the upper abdomen. When the entry site is anterior and below the uterine fundus, visceral injuries are less likely. Although the enlarging uterus can act as a shield against intra-abdominal injuries in the mother, it makes the fetus more susceptible to injury. Awwad and colleagues observed a 67% fetal death rate from penetrating trauma to the uterus but a lower fetal death rate (38%) for maternal injuries above the uterus.²⁹

Fetal Injury

Pregnancy does not alter rates of maternal mortality caused by trauma. However, trauma is associated with a high risk for fetal loss. When the mother suffers a severe level of injury, poor fetal outcome is predicted by maternal hypotension and acidosis (hypoxia, lowered pH, lowered bicarbonate) and a fetal heart rate of less than 110 beats per minute.^{6,7,30-34} When the mother suffers life-threatening injuries, there is a 40% chance of fetal demise, compared with a less than 2% chance in cases of non-life-threatening maternal injuries. Maternal age and gestational age may also be important factors in determining fetal outcome.³⁵

For women with less severe trauma, fetal outcome is not predicted by maternal vital signs, abdominal tenderness, blood tests, or ultrasonography (US) results. Only cardiotocographic monitoring for a minimum of 4 hours is useful in predicting fetal outcome.³

Fatal in utero fetal injuries from blunt trauma usually involve intracranial hemorrhage and skull fractures. Such head injury is often secondary to fractured maternal pelvic bones striking the fetal skull due to vertex lie.^{36,37} Pelvic and acetabular fractures during pregnancy are associated with a high maternal (9%) and a higher fetal (38%) mortality rate.³⁸ With penetrating trauma, gunshot wounds to the uterus are associated with a high incidence of fetal injury (59–89%) and fetal mortality (41–71%).³⁹ Stab wounds to the uterus can produce 93% morbidity and 50% mortality rates, respectively, to the fetus.⁴⁰

Placental Injury

In blunt trauma, 50 to 70% of all fetal losses result from placental abruption.^{41,42} It is the leading cause of fetal death after blunt trauma.

Placental separation results when the inelastic placenta shears away from the elastic uterus during sudden deformation of the uterus. Because deceleration forces can be as damaging

to the placenta as direct uterine trauma, abruption can occur with little or no external sign of injury to the abdominal wall.⁴³ Because all gas exchange between the mother and fetus occurs across the placenta, abruption inhibits the flow of oxygen to the fetus and causes in utero CO₂ accumulation. Such hypoxia and acidosis can lead to fetal distress.¹³ Sustained uterine contractions induced by intrauterine hemorrhage also inhibit uterine blood flow, further contributing to fetal hypoxia.⁴⁴

The diagnosis of abruption is a clinical one, and ultrasonography and the Kleihauer-Betke test are of limited value.⁴⁵ Classical clinical findings of abruption may include vaginal bleeding, abdominal cramps, uterine tenderness, maternal hypovolemia (up to 2 L of blood can accumulate in the gravid uterus), or a change in the fetal heart rate. However, in some trauma studies, as many as 63% of cases showed no evidence of vaginal bleeding.⁴⁶

The most sensitive indicator of placental abruption is fetal distress. Hence, prompt fetal monitoring is a very important assessment technique in trauma during pregnancy. There is also a close linkage of abruption to uterine activity. One study reported that if 12 or more contractions occurred in any hour of a 4-hour cardiotocographic monitoring period, the risk of abruption was 14%; abruption did not occur in this study if contractions occurred less than once every 10 minutes.³ Ultrasound (US) is less than 50% accurate as a first-line test in detecting placental abruption.^{3,47} If the abruption bleeds externally, not enough blood collects to be seen sonographically. Even with significant intrauterine blood accumulation, accurate US diagnosis may be difficult because of placental position (i.e., posterior) and confounding uterine or placental structural conditions.⁴⁸

Placental abruption is associated with an overall 8.9-fold increased risk of stillbirth (>20 weeks) and a 3.9-fold increased risk of preterm delivery (before 37 weeks). The extent of placental separation affects stillbirth rates. At 50% separation there is a fourfold increase of stillbirth and a more profound 31.5-fold increased risk of stillbirth at 75% separation. The risk of preterm delivery is substantially increased with even mild abruptions; a 25% separation carries a 5.5-fold increased risk of preterm.⁴⁹

When mother and fetus are stable, expectant management can be tried for partial placental abruptions of less than 25%. This usually applies to fetuses of less than 32 weeks' gestation in whom the likelihood of morbidity and mortality associated with prematurity makes delivery management risky. Expectant care in stable patients may allow further fetal maturation and improved outcome. Metzger and associates recommended intervention if the fetus is older than 32 weeks' gestation because the risk of further placental separation outweighs the benefit of further fetal maturation.⁵⁰ If expectant management is pursued, close maternal and fetal monitoring is needed to ensure the well-being of both patients. The ability to perform an immediate cesarean section is necessary because there may be little time between the appearance of fetal distress from further placental separation and the occurrence of fetal death.⁵¹

Women with placental abruption are more likely to have coagulopathies than those without abruption.⁵¹ The injured placenta can release thromboplastin into the maternal circulation, resulting in disseminated intravascular coagulation, whereas the damaged uterus can disperse plasminogen activator and trigger fibrinolysis.⁵² The precipitation of disseminated intravascular coagulation is directly related to the degree of placental separation. Severe clotting disorders rarely occur unless separation of the placenta is significant enough to result in fetal demise.⁵³

Uterine Injury

The most common obstetric problem caused by maternal trauma is uterine contractions.^{3,4,54} Myometrial and decidual cells, irritated by contusion or placental separation, release prostaglandins that stimulate uterine contractions. Progression to labor depends on the extent of uterine damage, the amount of prostaglandins released, and the gestational age of the pregnancy. The routine use of tocolytics for premature labor has come under question because 90% of contractions stop spontaneously.³ Contractions that are not self-limited are often induced by some pathologic condition, such as underlying placental abruption, which is a contraindication to tocolytic therapy. Others consider this contraindication relative and have used tocolysis successfully with careful evaluation and intensive monitoring to continue the pregnancy and enhance fetal maturity.⁵⁵ The option to use tocolytics ends when cervical dilation reaches 4 cm.

Uterine rupture is a rare event. It is most often caused by severe vehicular crashes in which pelvic fractures strike directly against the uterus. There have been a few reports of uterine rupture from stab wounds and gunshot injuries.⁵⁶ Maternal shock, abdominal pain, easily palpable fetal anatomy caused by extrusion into the abdomen, and fetal demise are typical findings on examination. Diagnosing uterine rupture can be difficult. A fractured liver or spleen can produce similar signs and symptoms of peritoneal irritation, hemoperitoneum, and unstable vital signs. Optimal treatment, between suturing the tear or performing a hysterectomy, depends on the extent of uterus and uterine vessel tears and the importance of future childbearing.

■ DIAGNOSTIC STRATEGIES

Radiography

Plain Radiographs

Adverse effects are unlikely at less than 5 to 10 radiation-absorbed doses (rad). Less than 1% of trauma patients are exposed to more than 3 rad. Sensitivity to radiation is greater during intrauterine development than at any other time of life, especially in the first trimester (i.e., when the embryo undergoes organogenesis in weeks 2–9). However, the risk to the fetus of a 1-rad (1000 mrad) exposure, approximately 0.003%, is thousands of times smaller than the spontaneous risks of malformations, abortions, or genetic disease.⁵⁷ Studies show that intrauterine exposure to 10 rad causes no significant increase in congenital malformations, intrauterine growth retardation, or miscarriage but is associated with a small increase in the number of childhood cancers.^{58–60} Pathologic conditions more readily appear with intrauterine radiation doses of 15 rad. At 15 rad there is approximately a 6% chance that the fetus could experience severe mental retardation, a less than 3% chance of developing childhood cancer, and a 15% chance of the having a small head, although this does not necessarily affect normal cerebral function.⁶¹

Providing information on radiation exposure from diagnostic radiographs is difficult. The individual amount of fetal dosage may vary by a factor of 50 or more, depending on the equipment used, technique, number of radiographs done in a complete study, maternal size, and fetal-uterine size. In general, coned x-ray beams aimed more than 10 cm away from the fetus are not harmful.⁶²

Diagnostic radiographic studies should be performed with regard for fetal protection, but necessary diagnostic studies should not be withheld out of concern for fetal radiation exposure. When appropriate, fetal irradiation should be minimized

Table 34-2

Estimated Radiation Dose to the Unshielded Ovaries/Pelvic Uterus

IMAGING STUDY	UTERINE RADIATION DOSE (MRAD)*
Plain-film Radiography	
Cervical spine	Undetectable
Thoracic spine	<1
Chest (PA)	<1
Chest (AP)	<5
Extremities (femur)	<50
Hip	10–210
Lumbar spine	31–400
Pelvis	140–2200
KUB	200–503
Intravenous pyelogram	503–880
Urethrocytogram	1500
Computed Tomography	
Head	<50
Thorax	10–590
Abdomen	2800–4600
Pelvis	1940–5000
Angiography	
Cerebral	<100
Cardiac catheterization	<500
Aortography	<100

*mrad, millirad; dose increases as the fetus grows to occupy more of the abdomen.

AP, anteroposterior; KUB, kidney, ureter, and bladder; PA, posteroanterior.

Data from Berlin L. Radiation exposure and the pregnant patient. *AJR Am J Roentgenol* 167:1377, 1996; North DL. Radiation doses in pregnant women. *J Am Coll Surg* 194:100, 2002; Damilakis J, Perisinakis K, Voloudaki A, Gourtsoyiannis N. Estimation of fetal radiation dose from computed tomography scanning in late pregnancy: Depth-dose data from routine examinations [published correction appears in *Invest Radiol* 2000;35:706]. *Invest Radiol* 35:527, 2000.

by limiting the scope of the examination and using technical means such as shielding and collimation.⁵⁸ Table 34-2 provides estimated radiation doses from various types of examinations.⁶³⁻⁶⁵ For comparison, the amount of naturally occurring radiation that the fetus receives during 9 months of gestation is approximately 50 to 100 mrad.⁶¹ If ionizing radiation is used, one must adhere to the principle of using a dose that is as low as reasonably achievable after a discussion of risks versus benefits with the patient.

Ultrasonography

US is the best modality for simultaneous assessment of both the mother and the fetus. It has a sensitivity of 88%, a specificity of 99%, and an accuracy of 97% for detecting intra-abdominal injuries in blunt trauma in all patients. In the pregnant patient, it is most useful in detecting major abdominal injury (sensitivity 80%, specificity 100%) and establishing fetal well-being or demise, gestational age, and placental location.^{66,67}

Sonography is a safe and effective screening examination that can obviate more hazardous tests such as computed tomography (CT), cystography, and diagnostic peritoneal lavage (DPL) in most pregnant patients with trauma who require an objective evaluation of the abdomen. Limitations in accuracy include operator experience, patient obesity, the presence of subcutaneous air, and a history of multiple abdominal operations. If an ultrasound is equivocal and the patient is hemodynamically unstable, DPL performed in an open and suprauterine fashion is indicated.

Computed Tomography and Magnetic Resonance Imaging Scans

CT and, increasingly, magnetic resonance imaging (MRI) studies are used in evaluating abdominal trauma in pregnancy. If US is indeterminate and the patient's condition is stable, CT and MRI have the potential to identify specific organ damage.⁶⁸ They are particularly useful in assessing penetrating wounds of the flank and back. CT can miss diaphragm and bowel injuries.⁶⁹ Both of these studies generally lack portability, and trauma patients may have to be taken from the closely monitored environment of the ED to a distant room.

Radiation from CT is a concern in the pregnant trauma patient. However, with shielding, fetal exposure from head and chest CT scans can be kept below an acceptable 1-rad limit. CT of the abdomen above the uterus can be done with less than 3 rad of exposure to the fetus.⁶¹ Pelvic CT, centered over the fetus, produces a more prohibitive 3- to 9-rad dose.⁷⁰ Fortunately, spiral CT can reduce radiation dosage by a further 14 to 30%.⁷¹ Radiation exposure ultimately depends on the patient, scanner, and technique used in performing the study (see Table 34-2). MRI scanners use no radiation and cause no fetal disease or disability.⁷²

SPECIAL PROCEDURES

Diagnostic Peritoneal Lavage

In unstable trauma patients with equivocal or negative findings on US, DPL can be done quickly and safely in any trimester by an open technique above the uterus. Blunt trauma studies indicate that the gravid uterus does not compartmentalize intraperitoneal hemorrhage and does not reduce the accuracy of DPL for selecting patients who need operative intervention for intra-abdominal bleeding.⁷³ DPL is limited in detecting bowel perforations and does not assess retroperitoneal and intrauterine pathology.

MANAGEMENT

As with any trauma, advance notification and preparation can be helpful. Depending on mechanism, maternal condition, and gestational age, the emergency physician should consider early notification or consultation with an obstetrician, neonatologist, or pediatrician (or all three). A fetal monitor, portable US, and neonatal resuscitation equipment should be available in the ED.⁷⁴

Maternal Resuscitation

Primary Survey

The primary survey focuses on the mother. However, because two patients are present, it is reasonable also to gather preliminary information about the fetus in the primary survey (Fig. 34-1).

Airway and Breathing. Oxygen therapy should be instituted early. Because of reduced oxygen reserve and increased oxygen consumption, the traumatized pregnant woman can quickly become hypoxic. The fetus is very vulnerable to any reduction in oxygen delivery. Animal studies show that severe hypoxia causes a 30% reduction in uterine blood flow.⁷⁵ Therefore, supplemental oxygen should be continued throughout maternal resuscitation and evaluation.⁷⁴

A secure airway is critical to an optimum outcome. Not only does it enable proper oxygenation but it negates the higher risk of aspiration in pregnancy. Rapid sequence intubation is

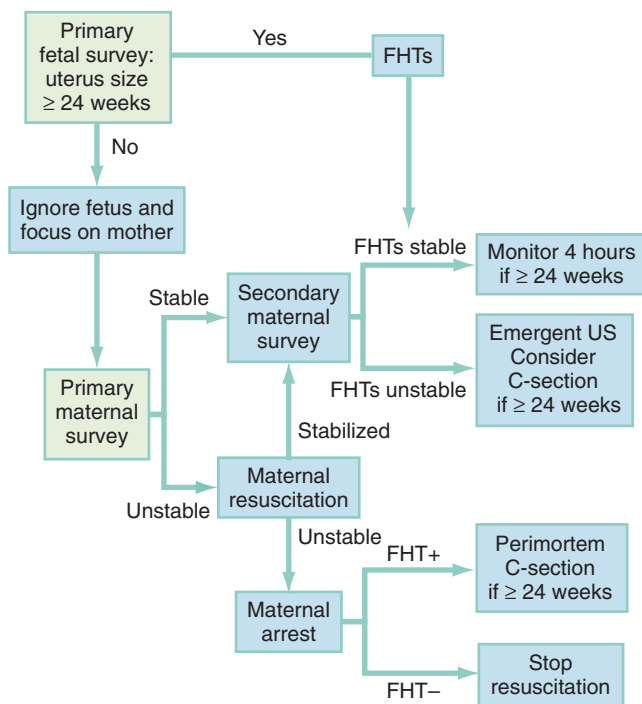


Figure 34-1. Decision-making algorithm in emergency obstetric care. C-section, cesarean section; FHTs, fetal heart tones; US, ultrasonography.

recommended when intubation is performed. Mechanical respirators need to be adjusted for increased tidal volumes and respiratory alkalosis consistent with the physiologic P_{aCO_2} of 30 mm Hg in the last stage of pregnancy.

Circulation. Any time significant maternal injury is suggested by the mechanism of injury or clinical findings, early IV access for volume resuscitation is indicated. Maternal blood pressure and heart rate are not consistently reliable predictors of fetal and maternal well-being.⁷⁴ Because of an expanded circulating volume, the mother can be bleeding but not show early signs of hypotension. The uterus is not a critical organ, and its blood flow is markedly reduced when the maternal circulation must be maintained. As a result, after an acute blood loss, uterine blood flow can be decreased 10 to 20% while maternal blood pressure remains normal.⁷⁶ Consequently, the mother who presents with borderline stability probably already has a jeopardized fetus. When traditional signs of shock appear, fetal compromise can be far advanced. Vasopressors should be avoided because they produce fetal distress by further decreasing uterine blood flow.

Beyond 20 weeks' gestation, the patient should be tilted to approximately 30 degrees to the left when on a backboard or should have the right hip elevated, unless otherwise precluded by right-sided injuries. This reduces the compression on the inferior vena cava caused by the gravid uterus. Tilting to the right is less effective in removing the uterus from the inferior vena cava; so consider manually displacing the uterus upward and leftward as discussed earlier.

For severe injuries, a CVP line is helpful in assessing cardiac preload. CVP pressures decline as pregnancy progresses because of inferior vena caval compression by the gravid uterus. Therefore, correction to nonpregnant normal pressures may be unnecessary. Instead, it is more valuable to focus on trends of how the CVP responds to fluid challenges. A Foley catheter for measuring urine output provides further information on circulatory volume status.

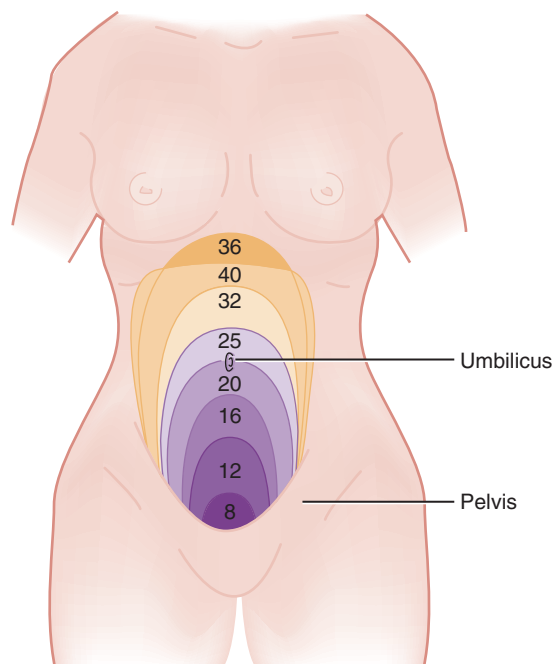


Figure 34-2. Uterine size at different weeks of gestation. (From Kravis TC, Warner CG [eds]: *Emergency Medicine: A Comprehensive Review*. Rockville, Md, Aspen Publishers, 1979.)

Table 34-3 Fetal Viability

WEEKS' GESTATION	6-MONTH SURVIVAL (%)	SURVIVAL WITH NO SEVERE ABNORMALITIES (%)
22	0	0
23	15	2
24	56	21
25	79	69

Data from Morris JA Jr et al: Infant survival after cesarean section for trauma. *Ann Surg* 223:481, 1996.

With trauma in pregnancy, the primary survey can be modified to assess uterine size and the presence of fetal heart tones. Uterine size, measured from the symphysis pubis to the fundus, is the quickest means of estimating gestational age. This distance in centimeters equals the gestational age in weeks (e.g., 24 cm = 24 weeks), which allows some early indication of fetal viability if delivery is necessary (Fig. 34-2). Usually, 24 to 26 weeks is used as the cutoff point for fetal viability (Table 34-3). As a rough guide, the fetus is potentially viable when the dome of the uterus extends beyond the umbilicus. Fetal heart tones can be detected by auscultation at 20 weeks' gestation or by Doppler probe at 10 to 14 weeks. If either the uterus is less than 24 cm in size or fetal heart tones are absent, the pregnancy is probably too early to be viable, and treatment is directed solely at the mother.

Secondary Survey

The secondary survey involves a detailed examination of the patient but is also modified to gather additional information about the maternal abdomen and the fetus. Physical examination of the abdomen, frequently unreliable in the nonpregnant patient, is more inaccurate with changing organ position, abdominal wall stretching in advancing pregnancy, and uterine

contraction pains. Still, information can be gathered about uterine tenderness, contraction frequency, and vaginal bleeding.

Pelvic examination begins with sterile speculum examination to allow direct visualization to enable detection of possible trauma in the genital tract, the degree of cervical dilation, and the source of any observed vaginal fluid. Vaginal bleeding suggests placental abruption, and a watery discharge suggests rupture of the membranes. If a vaginal fluid sample placed on a slide dries and crystallizes in a ferning pattern, it is amniotic fluid and not urine. Cervical cultures for group B streptococci, *Neisseria gonorrhoeae*, and *Chlamydia* should be obtained if there is evidence of amniotic fluid leak. Bimanual examination should be limited to assessing for pelvic bone injury or progression of advanced labor. This examination is preferably performed by an obstetrician. If the mechanism of injury is significant enough and the fetus is judged to be viable, it is strongly preferred that an obstetrician be involved in the treatment.

Fetal Evaluation

Fetal evaluation in the secondary survey focuses on the fetal heart rate and detection of fetal movement. When the presence of fetal heart tones has been confirmed, intermittent monitoring of fetal heart rate is sufficient for the previable fetus. If the fetus is viable (i.e., 24 weeks or more), continuous external monitoring should be initiated quickly and maintained throughout all diagnostic and therapeutic procedures. Such monitoring can also benefit the mother because fetal hemodynamics are more sensitive to decreases in maternal blood flow and oxygenation than are most measures of the mother. Fetal distress can be a sign of occult maternal distress. Signs of fetal distress include an abnormal baseline rate, decreased variability of heart rate, and fetal decelerations after contractions.

The normal fetal heart rate ranges between 120 and 160 beats per minute. Rates outside or trending toward these limits are ominous. Heart rate variability has two components. Beat-to-beat variability measures autonomic nervous function, whereas long-term variability indicates fetal activity. Heart

rate variability increases with gestational age. The loss of beat-to-beat and long-term variability warns of fetal central nervous system depression and reduced fetal movement caused by fetal distress⁷⁴ (Fig. 34-3).

Late decelerations are an indication of fetal hypoxia. These decelerations are relatively small in amplitude and occur after the peak or conclusion of a uterine contraction. By comparison, early decelerations are larger, occur with the contraction, and recover to baseline immediately after the contraction. Early decelerations may be vagally mediated when uterine contractions squeeze the fetal head, stretch the neck, or compress the umbilical cord. Variable decelerations are large, occur at any time, and are possibly caused by umbilical cord compression⁷⁴ (Fig. 34-4).

Laboratory

Besides routine trauma blood work, emergency laboratory tests should include a blood type with Rh status. In apparently stable pregnant patients, a low serum bicarbonate level may indicate occult maternal shock.⁷⁷ Interpretation of bicarbonate results must consider that the normal bicarbonate level is 21 mEq/L in the later stages of pregnancy due to respiratory alkalosis. Arterial blood gases can allow detection of maternal hypoxia and acidosis, whereas pulse oximetry can be used to monitor oxygen saturation. Coagulation studies should be obtained for patients with multisystem trauma or when the diagnosis of placental abruption is considered. The main difference between the nonpregnant trauma patient and the pregnant trauma patient's laboratory workup is the need to determine Rh status and quantitative β -hCG, if indicated based on mechanism; besides this, the physician should order any and all laboratory tests that would be ordered for any trauma patient.

Kleihauer-Betke Test and Fetomaternal Hemorrhage

Fetomaternal hemorrhage (FMH), the transplacental bleeding of fetal blood into the normally separate maternal circulation, is a unique complication of pregnancy. The reported incidence

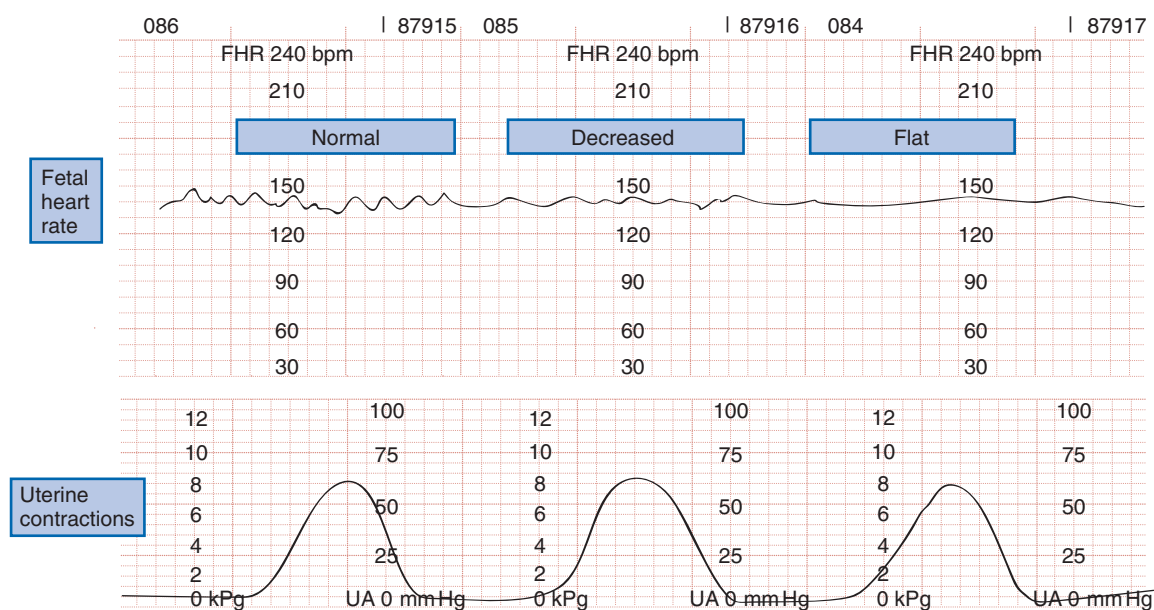


Figure 34-3. Types of fetal heart rate variability. bpm, beats per minute; FHR, fetal heart rate; UA, uterine activity.

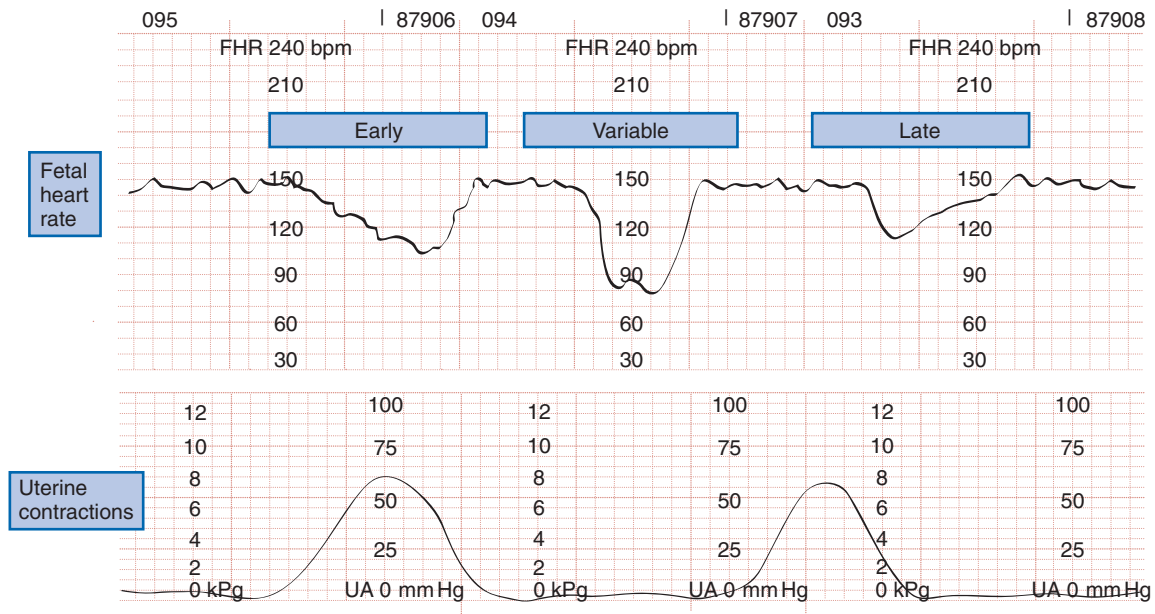


Figure 34-4. Types of fetal heart rate decelerations. bpm, beats per minute; FHR, fetal heart rate; UA, uterine activity.

of FMH after trauma is 8 to 30% (with a range of 2.5–115 mL of blood) compared with 2 to 8% (range of 0.1–8 mL) for control studies. MVCs, anterior placental location, and uterine tenderness are associated with an increased risk of FMH, but gestational age is not.^{3,5,78} Massive fetomaternal transplacental hemorrhage causes alloimmunization in Rh incompatibility but also endangers the fetus by severe fetal anemia and resulting fetal distress and possible exsanguination. ABO incompatibility causes less severe disease.

In theory, it is possible that trauma can result in FMH as early as the fourth week of gestation, when the fetal and placental circulations first form. In practice, FMH is usually of more concern after 12 weeks' gestation, when the uterus rises above the pelvis and becomes susceptible to direct trauma.³

The Kleihauer-Betke test identifies fetal cells in a maternal blood sample. Most laboratories screen for FMH of 5 mL or more. Unfortunately, the amount of FMH sufficient to sensitize most Rh-negative women is well below this 5-mL sensitivity level. Therefore, all Rh-negative mothers who have a history of abdominal trauma should receive one prophylactic dose of Rhesus immune globulin (RhIG). In the first trimester, one 50- μ g dose is used because total fetal blood volume is only 4.2 mL by 12 weeks' gestation and a 50- μ g dose covers 5 mL of bleeding. During the second and third trimesters, a 300- μ g dose of RhIG is given, which protects against 30 mL of FMH. Beyond 16 weeks' gestation, the total fetal blood volume reaches 30 mL, so it is quite possible that massive FMH may exceed the efficacy of one 300- μ g dose of RhIG. Therefore, it is unlikely that a Kleihauer-Betke test is useful in the treatment of severely injured pregnant trauma patients.

Trauma patients at risk for massive FMH present with major injuries or abnormal obstetric findings, such as uterine tenderness, contractions, or vaginal bleeding. Less than 1% of all pregnant trauma patients and only 3.1% of major trauma cases exceed the coverage of one 300- μ g RhIG dose.⁵

Because RhIG can effectively prevent Rh isoimmunization when administered within 72 hours of antigenic exposure, the results of the Kleihauer-Betke test are not immediately needed in the ED.

Mother Stable, Fetus Stable

Minor trauma does not necessarily exempt the fetus from significant injury. It is estimated that 1 to 3% of all minor trauma results in fetal loss, typically from placental abruption.⁷⁹ Therefore, once the traumatized mother is stabilized, the focus of care is directed toward the fetus. For the viable fetus (greater than 24 weeks' gestation), monitoring is the next step. Monitoring must be continuous and should be maintained throughout all diagnostic and therapeutic actions. Because direct impact is not necessary for fetoplacental pathology to occur, the mother with no obvious abdominal injury still needs monitoring.

The recommended 4 hours of cardiotocographic observation of the viable fetus should be extended to 24 hours if, at any time during the first 4 hours, there are more than three uterine contractions per hour, uterine tenderness persists, results on a fetal monitor strip are worrisome, vaginal bleeding occurs, the membranes rupture, or if any serious maternal injury is present. In Pearlman's study, all cases of placental abruption after maternal trauma were detected within the first 4 hours of monitoring.³ These mothers typically had more than 12 uterine contractions per hour. Although 70% of patients required admission beyond the 4-hour observation period, all patients who were discharged at the end of the 4-hour or 24-hour monitoring periods had subsequent live births.

On release from the hospital, the mother should be instructed to record fetal movements during the next week. If fewer than four movements per monitored hour are noted, the patient should see her obstetrician immediately and a nonstress test should be performed. The occurrence of preterm labor, membrane rupture, vaginal bleeding, or uterine pain also necessitates prompt reevaluation. Serial US and fetal heart rate tests should be performed on viable fetuses a few days after all trauma episodes and periodically throughout the remaining portion of the pregnancy to monitor fetal well-being.

Mother Stable, Fetus Unstable

Fetal death rates following maternal trauma are three to nine times higher than maternal death rates.⁶ If a viable fetus

remains in distress despite optimization of maternal physiology, cesarean section should be performed.

Although fetal viability is first reached at 24 weeks, the ultimate determinant of the age of fetal viability is the level of neonatal care provided by the intensive care nursery unit in each hospital or accessible regional facility. Note that determining gestational age for fetuses less than 29 weeks is difficult. Even with the best US criteria, unless the time of conception is known exactly, the assignment of gestational age is subject to 1 to 2 weeks of uncertainty.⁸⁰ Emergency decisions on fetal viability are, therefore, made on the basis of the best gestational age information available.

Morris and colleagues found that the presence of fetal heart tones is an important survival marker for fetuses about to undergo emergency cesarean section.⁸¹ No infant survives if there is no fetal heart tone before emergency cesarean section commences. If fetal heart tones are present and the gestational age is 26 weeks or more, then infant survival rate is 75%. Sixty percent of fetal deaths result from underuse of cardiotocographic monitoring and delayed recognition of fetal distress.

Besides fetal distress, other reasons for a cesarean section include uterine rupture, placental rupture with significant vaginal bleeding, fetal malpresentation during premature labor, and situations in which the uterus mechanically limits maternal repair. Fetal demise without any of the aforementioned conditions is not an indication for cesarean section because most will pass spontaneously within 1 week.

Mother Unstable, Fetus Unstable

If the mother's condition is critical, primary repair of her wounds is the best course. This may apply even when the fetus is in distress because a critically ill mother may not be able to withstand an additional operative procedure such as cesarean section, which prolongs laparotomy time and increases blood loss by at least 1000 mL. The best initial action on behalf of the fetus is early restoration of normal maternal physiology. If it is felt that the unstable mother can tolerate an emergency cesarean section, it can be attempted for the distressed, viable fetus.

As with nonpregnant patients, operative intervention for blunt trauma and above-the-uterus stab wounds is dictated by clinical findings and diagnostic testing results. Above-the-uterus intraperitoneal gunshot wounds should be explored.

There is little evidence to support a definitive management strategy for penetrating trauma to the gravid uterus. In situations of a hemodynamically stable mother, expectant management has been recommended. However, no prospective study has verified this. Damage to the uterus alone can be quite devastating because of its increased circulation. Meizner and Potashnik reported a shrapnel injury to only the uterus in a case in which an initially normal examination quickly changed to a hypotensive emergency.⁸² At celiotomy, 1000 mL of blood was found in the abdomen from a perforated corner injury to the uterus. Without exploration it is impossible to know the occurrence, size, or depth of uterine penetration, and there are no guidelines indicating whether a uterine wound can be left unsutured without incurring an increased risk of infection or delayed uterine rupture. Laparotomy or laparoscopy seems to be the safest means of managing penetrating uterine wounds because missed maternal injuries can quickly compromise the fragile fetus.

Perimortem Cesarean Section

Restoration of maternal and thus fetal circulation is the optimal goal. However, extended and exclusive attention to the mother

in cardiopulmonary arrest may prevent recovery of a potentially viable fetus. During maternal resuscitation, adequate oxygenation, fluid loading, and a 30-degree left tilting position should be tried to determine whether maternal circulation can be improved. If there is no response to advanced cardiac life support, a decision for perimortem cesarean section must be made. If there are no fetal heart tones, a cesarean section is not warranted.⁸³

Perimortem cesarean section in the ED should be performed if uterine size exceeds the umbilicus and fetal heart tones are present. Time since maternal circulation ceased is the critical factor in fetal outcome. Published reports from more than 20 years ago support, but fall far from proving, that perimortem cesarean delivery should be initiated within 4 minutes if no pulse to obtain cardiac return by 5 minutes. Beyond 20 minutes, there is virtually never survival or favorable neurologic outcome for either mother or fetus.⁸⁴

In the event of maternal cardiopulmonary arrest, perimortem cesarean section is indicated. The most experienced physician, preferably an obstetrician, should perform the procedure. However, this task ultimately may fall to the emergency physician or trauma surgeon, given the time constraints of the situation. A pediatric consultation should be obtained emergently. While continuing CPR, a "classic midline vertical incision is made, using a large scalpel, extending from the epigastrium to the symphysis pubis and carried through all layers to the peritoneal cavity. A vertical incision is then made in the anterior uterus from the fundus to the bladder reflection. Assistants and other surgical instruments (e.g., clamps, retractors) are helpful but not required. If, when the uterus is entered, an anterior placenta is encountered, it should be incised in order to reach the fetus. The cord should be promptly clamped and cut following delivery of the child."⁸⁴ Maternal revival after delivery of the fetus is reported in a few perimortem circumstances, presumably because vena caval compression is relieved.⁸⁵

■ DISPOSITION

Any pregnant woman at 24 or more weeks of gestation who has suffered blunt body trauma should undergo at least 4 hours of fetal monitoring even if she looks well. Other admission and operative criteria are similar for pregnant and nonpregnant trauma patients. The emergency physician must always consider the stability of the mother and the viability of the growing fetus when making management and disposition decisions.

■ MISCELLANEOUS

Tetanus toxoid and immune globulin have no detrimental effect on the fetus. Proper immunization of pregnant women decreases the incidence of neonatal tetanus because the tetanus antibody crosses the placenta.

Electrical flow that bypasses the fetus has little effect on the pregnancy. Maternal elective and emergent cardioversion has been performed safely for cardiac dysrhythmias in all three stages of pregnancy. Energies up to 300 watt-seconds have been used without affecting the fetus or inducing premature labor. Cullhead reported no disruption of a monitored fetal heart rhythm during maternal cardioversion with 80 and 200 watt-seconds.⁸⁶ Although the amount of energy reaching the fetal heart is thought to be small, the fetal heart should be monitored during cardioversion.

KEY CONCEPTS

- Management of life- and limb-threatening injury in the mother comes first.
- Even in the noninjured pregnant patient, the fetus is at increased risk of morbidity and mortality.
- The fetus is viable at 24 weeks' gestation. A fetus is estimated to be viable if the fundus is at or above the umbilicus.
- When assessing the injured pregnant patient, it is important to keep in mind the alterations in anatomy and physiology that occur during pregnancy.
- Stable pregnancies with a viable fetus should be monitored after trauma continuously for a minimum of 4 hours.
- Keeping the mother tilted 30 degrees to the left or in the left lateral decubitus position may alleviate hypotension and improve perfusion for the mother and fetus.
- Perimortem cesarean section should be performed only for a viable fetus with signs of life.
- Minimize the use of ionizing radiation to the pregnant patient, including CT and plain radiography, but imaging should not be withheld if it may provide significant diagnostic information.
- Nonionizing radiation, including ultrasound and MRI, is preferred for pregnant patients.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.

CHAPTER 35 Pediatric Trauma

Randolph J. Cordle and Richard M. Cantor

PERSPECTIVE

Half of all deaths in children 1 to 14 years old are the result of trauma, and more than 15,000 traumatic deaths per year occur within this age group. More than half of these mortalities are directly related to motor vehicle collisions (MVCs). Injury accounts for approximately 30% of infant deaths as well.^{1,2} In the United States, estimates of mortality for children hospitalized after injury are uniformly low; however, most fatalities occur in the field before arrival at a health care facility, which contributes to an underestimation of the magnitude of overall mortality figures.

The most common single organ system injury associated with death in injured children is head trauma.³ Rates of 80% have been reported in patients with combined thoracoabdominal injuries.^{1,4} Because multiple injuries are common in children, the emergency physician must evaluate all organ systems in any injured child, regardless of the actual mechanism of injury.

Within the subset of MVCs, death rates increase steeply in adolescents (>13 years old). MVC mortality statistics show that the youngest occupant in the vehicle is the most vulnerable to injury. Among school-age children (5–9 years old), pedestrian injuries and bicycle crashes predominate. Falls from heights account for 25 to 30% of injury; submersion injuries account for 10 to 15%, and burns account for 5 to 10%.^{4,5} Throughout the United States, the number of children who are victims of violent acts has increased. Some children's hospitals report that 25 to 35% of all pediatric trauma deaths are related to child abuse.⁶

PRINCIPLES OF DISEASE

There are major anatomic and physiologic differences between pediatric and adult patients that play a significant role in the evaluation and management of a pediatric trauma patient (Box 35-1 and Table 35-1). Compared with adults, any given force is more widely distributed through the body of a child, making multiple injuries significantly more likely to occur in children. The proportionately large surface area of infants and children relative to weight predisposes them to greater amounts of heat loss as a result of evaporation. During resuscitation, even mild to moderate hypothermia has direct negative effects on cardiac function, inotropy, left-ventricular contractility, catecholamine responsiveness, platelet function, renal/hepatic drug clearance, and metabolic acidemia. Maintenance requirements for free water, trace metals, and minerals are therefore magnified compared to those for adults. Oxygen extraction and consumption, as well as glucose utilization, is much higher per kilogram

in infants and small children than in adults. These factors contribute to a significantly higher energy and caloric requirement for an injured child compared with an injured adult. Finally, a child's physiologic response to injury is different from an adult's response, depending on the age and maturation of the child and the severity of the injury. In contrast to adults, children have a great capacity to maintain blood pressure despite significant acute blood losses comprising 25 to 30% of total blood volume. However, changes in heart rate, blood pressure, and extremity perfusion may precede cardiorespiratory failure and should not be overlooked. Small children respond to decreased cardiac output primarily through an increase in heart rate, not contractility. Similarly, children have decreased pulmonary reserve and respond differently to stress than do adults. Children are primarily diaphragmatic breathers dependent on the excursion of the diaphragm to increase the volume of the thoracic cage, thereby increasing the volume of air exchanged per breath. Unlike adults, children's elastic barrel-shaped chest wall prevents them from using their extrathoracic muscles to lift the ribs in order to generate an increase in intrathoracic volume and, hence, increased tidal volume. When infants create greater negative pressures in their pleural spaces, their more elastic chest wall caves inward, leading to chest wall retractions.

CLINICAL FEATURES

Initial Assessment Priorities and Primary Survey

The highest priority in the approach to the injured child is ruling out the presence of life-threatening or limb-threatening injury. Treatment of these injuries must occur before proceeding with the rest of the physical examination. This initial assessment (the primary survey) and necessary initial resuscitation efforts must occur simultaneously. In general, the assessment and resuscitation should be addressed within the first 5 to 10 minutes of evaluation. Any infant or child with a potentially serious or unstable injury requires continual reassessment. Vital signs should be repeated every 5 minutes during the primary survey and every 15 minutes thereafter until the patient is considered stable. The primary survey for pediatric trauma patients can be remembered by A, B, C, D, E, and F.

A—Airway and Cervical Spine Stabilization

Table 35-1 describes anatomic considerations that have implications in the management of the pediatric airway. The physi-

Table 35-1 Anatomic Differences in the Pediatric Airway—Implications in Pediatric Trauma Management

DIFFERENCES	IMPLICATIONS
Increased vagal response to laryngoscopy	Bradycardia during intubation; in infants and small children may be abated through the use of glycopyrrolate or atropine
Relatively larger tongue	Most common cause of airway obstruction in children May necessitate better head positioning or use of airway adjunct (oropharyngeal or nasopharyngeal airway)
Larger mass of adenoidal tissues may make nasotracheal intubation more difficult	Nasopharyngeal airways may also be more difficult to pass in infants
Epiglottis floppy and more U shaped	Necessitates use of a straight blade in young children
Larynx more cephalad and anterior	More difficult to visualize the cords; may need to get lower than the patient and look up at 45-degree angle or greater while intubating
Cricoid ring the narrowest portion of the airway	Allows for use of uncuffed tubes in children up to size 6 mm or approximately 8 years old Can use cuffed tube with or without balloon blown up; cuffed tube size typically (age/4) + 3
Narrow tracheal diameter and distance between the rings, making tracheostomy more difficult	Needle cricothyrotomy for the difficult airway versus a surgical cricothyrotomy for the same reason
Shorter tracheal length (4–5 cm in newborns and 7–8 cm in 18-month-olds)	Leads to intubation of right mainstem or dislodgment of the endotracheal tube Ensure tube position is checked before taping with head in neutral position or it can be driven into the right mainstem when the head is flexed or withdrawn when the head is extended to get to neutral
Large airways more narrow	Leads to greater airway resistance (R proportional to $1/\text{radius}^4$)

BOX 35-1**ANATOMIC DIFFERENCES IN ADULTS AND CHILDREN: IMPLICATIONS FOR PEDIATRIC TRAUMA MANAGEMENT**

The child's body size allows for a greater distribution of traumatic injuries, so multiple trauma is common. The child's greater relative body surface area causes greater heat loss.

The child's internal organs are more susceptible to injury based on more anterior placement of liver and spleen and less protective musculature and subcutaneous tissue mass.

The child's kidney is less well protected and more mobile, making it susceptible to deceleration injury.

Fifteen percent of pediatric patients presenting with hematuria after trauma have underlying congenital abnormalities.

Growth plates are not yet closed in pediatric patients, leading to Salter-type fractures with possible limb-length resultant abnormalities.

The child's head-to-body ratio is greater, the brain is less myelinated, and cranial bones are thinner, resulting in more serious head injury.

cian assesses for possible airway obstruction from injury, teeth, blood, constriction, or vomitus. The physician must know normal dental anatomy and development in order to better recognize the possibility of missing primary or secondary teeth. Efforts to perform cricoid pressure, or ligatures such as ties on gowns, can easily occlude the infant/child's airway with as little as 0.2 pounds of force. Excessive cricoid pressure is a preventable but common reason for difficulty passing the endotracheal tube once it has passed through the cords.⁷ Gurgling or stridor may indicate upper airway obstruction. While stabilizing the neck, the airway is opened with a jaw-thrust maneuver. Efforts should be directed toward clearing the oro-

Table 35-2 Airway: Assessment and Treatment

ASSESSMENT PRIORITIES	INTERVENTIONS
Airway patency	Jaw thrust, suction, airway adjuncts Stabilize/remove loose teeth or other foreign bodies
Level of consciousness	Cervical spine immobilization
Maxillofacial injury	Apply 100% O ₂ by mask Monitor patient closely for emesis
Stridor or cyanosis	Intubate for Glasgow Coma Scale ≤ 8 or absent gag reflex in a patient with a clinically concerning head injury or $\text{Po}_2 < 50$ mm Hg or $\text{Pco}_2 > 50$ mm Hg Needle cricothyrotomy if intubation impossible and cannot oxygenate and ventilate by bag-valve-mask until successful airway control by alternative method or provider

pharynx of debris. The clinician must consider the possibility of cervical cord injuries in all seriously traumatized children. Evaluation of the cervical spine in children is discussed later: A gentle, developmentally appropriate approach must be used if reliable information is to be gained. Any complaint of past or current neurologic deficit, neck pain, or significant trauma to the head, chest, abdomen, or other spinal level injury should raise concern for a cervical spine injury. Repeatedly asking a toddler, "Does this hurt?" will typically lead to a response of, "No, No, No, Yes" as the child tries to accommodate to what he or she believes the examiner must want for an answer. It is often more useful to watch facial expression and for other cues of discomfort. Table 35-2 describes priorities in the assessment of the pediatric airway.

B—Breathing and Ventilation

The physician assesses for adequacy of chest rise; in a young child, this occurs in the lower chest and upper abdomen. Both the chest and the abdomen should move concordantly. Discordant motion is referred to as *paradoxical breathing* and is a sign of impending respiratory failure. The respiratory rate is also assessed. Rates that are too fast or too slow can indicate impending respiratory failure. Treatment is assisted ventilation. If ventilation is necessary, a bag-valve-mask device is recommended initially. Only the volume necessary to cause the chest to rise should be provided because excessive volume or rate of ventilation can increase the likelihood of gastric distention (increasing the risk of vomiting and aspiration) and impair ventilation further. Cricoid pressure may help decrease the amount of air entering the esophagus during positive-pressure ventilation. A nasogastric/orogastric (NG/OG) tube should be placed during the first few minutes of bag-valve-mask ventilation and should be considered in all seriously injured children. Gastric distention due to bag-valve-mask and air swallowing often leads to respiratory embarrassment and potential hypotension due to decreased venous return. In conscious children, placement of an NG or OG should be preceded by local anesthesia, using agents such as atomized or nebulized lidocaine plus lidocaine jelly. The overall amount of lidocaine given should be closely monitored to prevent accidental overdose.

Indications for endotracheal intubation of a pediatric trauma patient include (1) any inability to ventilate by bag-valve-mask methods or the need for prolonged control of the airway, (2) Glasgow Coma Scale (GCS) score of less than 9 to secure the airway and provide controlled hyperventilation as indicated, (3) respiratory failure from hypoxemia (e.g., flail chest and pulmonary contusions) or hypoventilation (e.g., injury to airway structures or spinal cord injury), and (4) the presence of decompensated shock resistant to initial fluid administration.

Compared with adults, intubation of pediatric patients involves special considerations (see [Table 35-1](#)).

In children younger than age 8 years, the cricoid ring is the narrowest portion of the airway. For this reason, the cricoid ring often forms a physiologic cuff on uncuffed endotracheal tubes. In general, uncuffed tubes are used in children younger than age 8 years; cuffed tubes may be indicated when more airway protection than that from an uncuffed tube is necessary or when high-pressure or more precise ventilatory management is necessary. In general, the orotracheal approach to intubation is recommended. Problems associated with nasotracheal intubation in children include impairment of tube passage by the acute angle of the posterior pharynx, the potential for causing or worsening bleeding within the oral cavity, sinusitis, and increasing intracranial pressure (ICP) with insertion.

There are many limiting factors that compromise ventilatory function in an injured child, including depressed sensorium, occlusion of the airway, painful restriction of lung expansion, and direct pulmonary injury. Determination of adequate ventilation is possible only in the face of airway patency and adequate air exchange. As noted previously, the diaphragm plays a special role in the maintenance of proper ventilatory status in children. It is easily fatigued in a young child and often is displaced by any process that promotes distention of the stomach. As such, it is advisable to consider early placement of an NG/OG tube to facilitate decompression of the stomach.

To assess “ventilation,” pulse oximetry is useful; however, pulse oximetry measures adequacy of oxygenation only. The

measurement of exhaled CO₂ is useful to confirm endotracheal tube position. Historically, a colorimetric semiquantitative device was used to detect the presence of exhaled CO₂ in patients with perfusion. The use of continuous end-tidal CO₂ capnography provides far more information and continues to be underutilized. In addition to serving as an initial qualitative device to confirm successful intubation of the trachea, it may also provide an early warning of unintended extubation, tube kinking or partial occlusion, or ventilator malfunction. It also characterizes the response to therapeutic maneuvers instantaneously, provides a quantitative tool to manage the ventilatory aspects of respiration, and may provide prognostic information when used in patients with cardiac arrest. The lack of appropriate CO₂ production when the tube is in proper position often indicates poor perfusion. The use of end-tidal CO₂ capnography allows better ventilatory management during head injury resuscitation, and its values can be confirmed with a single venous or arterial blood gas. This can assist greatly with continuing ventilatory management without the need for recurrent blood draws and the inherent delays and discomfort of acquiring arterial blood gases. [Table 35-3](#) describes priorities in the assessment of breathing in pediatric trauma patients.

Table 35-3 Breathing: Assessment and Treatment

ASSESSMENT PRIORITIES	INTERVENTIONS
Respiratory rate	100% O ₂ by nonrebreather mask or intubate if in respiratory failure; fast rates may indicate shock (fluid resuscitation) or pain (parenteral analgesics).
Chest wall movements	For significant pneumothorax or hemothorax: Place chest tube. Small pneumothorax in a patient spontaneously breathing may only require close monitoring and/or oxygen washout. Transfer to operating room if initial drainage >20 cc/kg or subsequent output >2 mL/kg/hr.
Percussion	Open pneumothorax: Seal with three-sided occlusive dressing (Vaseline gauze) followed by tube thoracostomy and then seal remaining side of occlusive dressing.
Paradoxical breathing	Contusion/flail chest: Intubate if tachypneic or Po ₂ < 50 mm Hg or Pco ₂ > 50 mm Hg.
Tracheal deviation	Tension pneumothorax: Needle decompression at second intercostal space, midclavicular line, followed by placement of chest tube unless chest tube incision can be immediately placed, negating the necessity of needle decompression.
Flail segments	O ₂ by nonrebreather mask or intubate if in respiratory failure.
Open wounds	Compress bleeding sites and cover as indicated. Consider use of hemostatic dressing.

Table 35-4 Circulation: Assessment and Treatment

ASSESSMENT PRIORITIES	INTERVENTIONS
Capillary refill	Oximeter and cardiac monitor, O ₂ and fluid resuscitation 20 mL/kg. Consider intubation and ventilation to decrease workup breathing.
Heart rate	Monitor vital signs every 5 min.
Peripheral pulses	Two large-bore intravenous sites (above and below diaphragm when indicated).
Sensorium	Bolus with 20 mL/kg lactated Ringer's or normal saline solution (warm all intravenous fluids).
Pulse pressure increase	Repeat fluid bolus two times if necessary.
Skin condition/perfusion	Packed red blood cells, 10–20 mL/kg for decompensated shock secondary to blood loss.

C—Circulation and Hemorrhage Control

Shock is not defined by any specific blood pressure but is instead a state in which the body is unable to maintain adequate tissue perfusion. Maintenance of systolic blood pressure does not ensure that the patient is not in shock. The pediatric vasculature has the ability to constrict and increase systemic vascular resistance in an attempt to maintain perfusion. Signs of poor perfusion, such as cool distal extremities, decreases in peripheral versus central pulse quality, and delayed capillary refill time, are signs of pediatric shock, even when blood pressure is maintained at normal levels. Palpable pulses are detectable at a systolic blood pressure greater than 80 mm Hg in children over approximately 10 years of age; however, it may be felt at lower pressures in infants and children younger than 10 years of age. Pulses can frequently be felt at lower systolic blood pressures in infants. Normal capillary refill time is less than 2 seconds; however, many variables affect this clinical finding. Alteration in a child's response to the environment or interaction with caregivers may indicate respiratory failure or shock. External hemorrhage should be sought and controlled with direct pressure. The assessment of circulation in pediatric trauma patients is described in [Table 35-4](#).

**D—Disability Assessment
(Thorough Neurologic Examination)**

The assessment of disability in pediatric trauma patients is described in [Table 35-5](#). To assess patient disability, a rapid neurologic evaluation is needed. Many methods are available to the clinician, specifically the AVPU system ([Box 35-2](#)) and the modified GCS ([Table 35-6](#)).

E—Exposure and Thorough Examination

The final component of the primary survey involves fully undressing the patient to assess for hidden injury. Maintenance of normothermia is paramount in the undressed toddler and infant because metabolic needs are greatly increased by hypothermia. As soon as possible, in addition to increased ambient temperature, additional warming methods such as warmed humidified oxygen, warmed fluids and/or blood, head wraps, and convective warmers or radiant heat sources should

Table 35-5 Disability: Assessment and Treatment

ASSESSMENT PRIORITIES	INTERVENTIONS
Level of consciousness	Maintain blood pressure and oxygenation and ventilation.
AVPU scale or GCS	If head injury with GCS <9: RSI and intubate; head computed tomography, neurosurgical consult. If normotensive, consider mannitol 0.25–0.5 g/kg. Maintain CO ₂ at approximately 35 mm Hg. Maintain cerebral perfusion pressure of at least 50 mm Hg in children and 70 mm Hg in adults.
Pupil size and reactivity	Hyperventilate: PCO ₂ to 30–35 mm Hg with signs of herniation. Consider alternative causes of pupillary dilatation, such as traumatic mydriasis or drug effect from atropine.
Extremity movement and tone	Stabilize spinal column. If blunt cord trauma, consider methylprednisolone sodium succinate (Solu-Medrol) 30 mg/kg IV bolus, then 5.4 mg/kg/hr for 23 hr IV.
Posturing	Hyperventilate: PCO ₂ to 30–35 mm Hg.
Reflexes	Assess for signs of respiratory failure/bulbocavernosus reflex or anal wink for spinal injury “completeness.”

AVPU, alert, verbal, painful, unresponsive; GCS, Glasgow Coma Scale; RSI, rapid sequence intubation.

BOX 35-2 AVPU SYSTEM

A Alert
V Responds to verbal stimuli
P Responds to painful stimuli
U Unresponsive

be used. Preventing and treating hypothermia is not a matter of comfort for traumatized infants and children but, instead, one of survival ([Table 35-7](#)).

F—Family

In the management of children, the family could be added to the primary survey. Rapidly informing the family of what has happened and the evaluation that is proceeding helps lessen the stress of the caregivers. Allowing family members to be present during resuscitations is acceptable and often preferred by families. Some caregivers choose not to be present, but that choice should be given to them. If a caregiver is present, it is advisable to assign a staff member to be with him or her during the trauma resuscitation to explain the process.

Child life specialists and clergy are valuable members of the resuscitation team. They can not only serve the patient directly through their provision of comfort and developmentally appropriate explanations of medical activities but also serve as a single caring person for the child to focus on throughout their evaluation. They can also be instrumental in assisting the family to better understand what they may and may not do during and soon after the resuscitation. Families often wonder, but are afraid to ask, if they may touch the child, who can help with siblings, which siblings should be allowed to visit, what is the next step, and a myriad of other concerns. These special-

Table 35-6 Glasgow Coma Scale Modified for Pediatric Patients*

EYE OPENING RESPONSE			
SCORE	>1 YR		<1 YR
4	Spontaneous		Spontaneous
3	To verbal command		To shout
2	To pain		To pain
1	None		None
MOTOR RESPONSE			
SCORE	>1 YR		<1 YR
6	Obeys commands		Spontaneous
5	Localizes pain		Localizes pain
4	Withdraws to pain		Withdraws to pain
3	Abnormal flexion to pain (decorticate)		Abnormal flexion to pain (decorticate)
2	Abnormal extension to pain (decerebrate)		Abnormal extension to pain (decerebrate)
1	None		None
VERBAL RESPONSE			
SCORE	>5 YR	2–5 YR	0–2 YR
5	Oriented and converses	Appropriate words and phrases	Babbles, coos appropriately
4	Confused conversation	Inappropriate words	Cries but is consolable
3	Inappropriate words	Persistent crying or screaming to pain	Persistent crying or screaming to pain
2	Incomprehensible sounds	Grunts or moans to pain	Grunts or moans to pain
1	None	None	None

*Total score key: severe, <9; moderate, 9–13; mild, 14–15.

Table 35-7 Exposure: Assessment and Treatment

ASSESSMENT PRIORITIES	INTERVENTIONS
Undress	Trauma examination, including rectal examination when indicated.
Look under collar and splints	
Log roll and examine back	Remove from board when not contraindicated.
Radiology	Consider cervical spine, chest, and pelvis radiographs.
Laboratory	Complete blood cell count, type and crossmatch, amylase, urinalysis, urine pregnancy test.
Interventions	Place urinary catheter and nasogastric or orogastric tube as indicated.
Immunization	Appropriate tetanus vaccine if indicated. Consider tetanus immune globulin in appropriate cases.
Pelvic fracture	Consider binding pelvis to decrease pelvic volume and improve hemostasis.

ists can assist in all these areas. They are also expert in “playing”—the art of distraction and visual imagery. They may also play a role as the quintessential patient advocate, ensuring that the health care providers focus on the patient and not only on the individual medical issue at hand. Child life specialists should be viewed as part of the resuscitation team.

Secondary Survey

After completion of the primary survey and requisite procedures, the secondary survey is performed. The secondary survey is an organized complete assessment to detect additional injury not found on the primary survey. A more complete and detailed history is obtained at this time. Features of the history that need to be obtained can be remembered by the mnemonic *AMPLE* (Box 35-3). Ongoing assessment of the

patient occurs after the secondary survey, and key points are summarized in Box 35-4.

MANAGEMENT AND DIAGNOSTIC STRATEGIES

General Management Principles

All pediatric patients who have sustained major trauma should be placed on a cardiac monitor; receive supplemental oxygen; and have constant reassessment of vital signs, oximetry, and, when possible, end-tidal CO₂. Vascular access is best obtained by accessing the upper extremity for the establishment of two large-bore intravenous lines. In the absence of available upper extremity peripheral sites, lower extremity sites could be used. Many clinicians favor the femoral vein as a safe site for insertion of a central line by use of a guidewire technique. A guide

BOX 35-3 AMPLE HISTORY

- A** Allergies
- M** Medications
- P** Past medical history
- L** Last meal
- E** Environments and events

BOX 35-4 TASKS TO BE COMPLETED AFTER THE SECONDARY SURVEY

- Complete head-to-toe examination
- Appropriate tetanus immunization
- Antibiotics as indicated
- Continued monitoring of vital signs
- Ensure urine output of 1 mL/kg/hr

BOX 35-5 FEMORAL LINE SIZING ESTIMATES

3 F	<3 kg
4 F	3–10 kg
5 F	10–20 kg
6 F	>20 kg

to suggested sizing of femoral catheters is shown in Box 35-5.

If cutdowns are necessary, the antecubital or saphenous sites are preferred; however, intraosseous access is typically a quicker and more reliable procedure to obtain access to the vascular space. Essentially all labs with the exception of the peripheral WBC and peripheral smear can be obtained from an intraosseous needle (including blood type and cross). Although most commonly started in the proximal medial tibia just below the growth plate, intraosseous access often can be obtained more easily in the flattened area of the anterior distal femur. The intraosseous route serves as an appropriate venous access site; however, the delivery rate of large amounts of crystalloid solutions is limited, based on maximal flow rate of approximately 25 mL/min.⁸ More than one intraosseous needle may need to be placed (in separate bones), and a separate peripheral or a central line may be more easily obtained once fluids have been given via the intraosseous route. Intraosseous placement in a fractured extremity is contraindicated. Umbilical vein cannulation can be obtained in infants up to approximately 2 weeks of age. Three- and 5-F single and 5-F double-lumen catheters are available for use in the umbilical vein. If a specialized catheter is not available, a feeding tube or even a flexible intravenous catheter can be used for the infusion of crystalloid or blood. If vasopressors or highly osmotic agents are to be used, a more formal umbilical venous line placed above the liver should be considered to avoid hepatic injury.

Most hypovolemic pediatric trauma patients respond to 20 mL/kg boluses of isotonic crystalloid solutions. If 40 mL/kg has not reversed systemic signs of hypoperfusion, infusion of packed red blood cells at 10 mL/kg should be considered. In patients who present in decompensated shock or cardiopulmonary failure and occult bleeding is a potential cause for the shock, crystalloid and blood products may be administered simultaneously.

The use of blood products during trauma resuscitation is controversial. Although it is desirable to reduce risks of infectious disease and conserve precious resources, coagulopathy is

to be expected after trauma and massive transfusion. When massive transfusion (>1 blood volume = approximately 80 mL/kg) is expected, most current guidelines appear to underutilize additional blood products. This leads to continued coagulopathy and likely contributes to unnecessary death and morbidity. Some experts now recommend, based predominantly on adult studies, that if massive transfusion is expected, blood and fresh frozen plasma (FFP) be given in a near 1:1 ratio, although other experts believe a ratio closer to 1:2.5 may suffice and decrease the risk of multiorgan failure. In general, FFP should be dosed at 15 to 25 mL/kg. Platelet transfusion dosing can be very confusing. Practically all platelet units currently used are apheresis packs from a single donor. Each apheresis unit roughly equates to six of the older concentrate units. The usual dose in trauma is 10 mL/kg; however, the response may be quite variable (i.e., it can vary by more than a factor of two) due partly to the heterogeneity of the concentration of platelets in an apheresis pack. A general goal in trauma patients is to raise the platelet count above $50 \times 10^9/L$. The platelet count should be rechecked at 1 and 24 hours after transfusion, more often if the patient has ongoing difficulties with hemostasis or need for recurrent transfusion of red blood cells. The primary goal of giving cryoprecipitate is to increase the fibrinogen to levels of 1 to 1.5 g/dL, especially after central nervous system trauma. Although dependent on the fibrinogen concentration in the individual cryoprecipitate bags, the dose is typically 0.1 to 0.2 bags/kg. Each bag of cryoprecipitate contains approximately 150 mg of fibrinogen and 80 units of factor VIII.

In contrast to adults, cardiogenic shock is a rare event in childhood injury.^{3,9} However, any degree of chest trauma associated with the presence of shock must alert the clinician to the possibility of concomitant myocardial contusion or rupture. Myocardial rupture should be apparent during the focused abdominal sonography in trauma (FAST) exam. The classic presentation of neurogenic shock due to loss of sympathetic tone and contractility involves hypotension with a relative bradycardia. Vasodilatation leading to a suboptimal systemic vascular resistance is the root cause of this hypotension. These patients are often hypothermic. Neurogenic shock typically occurs after injury to the sympathetic outflow tracts between T1 and L2. Generally, dopamine is the first-line agent for treatment of neurogenic shock. The mechanism and clinical findings of neurogenic shock are distinct from those of spinal shock. Spinal shock often presents with decreased systemic vascular resistance, a relative hypovolemia, and tachycardia. It is often treated with fluids and, when necessary, pressors with primarily alpha vasoconstrictive effects, such as phenylephrine.

Specifics of the head examination include pupillary size and reactivity, funduscopic examination, and palpation of the skull. Assessment of the cervical spine must be done carefully, with the patient in full cervical spine immobilization. As soon as feasible, the patient should be removed from the backboard with cervical spine immobilization maintained. Backboards are not only uncomfortable but also often rapidly cause necrosis at pressure points. There are no common indications to justify leaving children on backboards after their initial evaluation.

Assessment of the chest and internal structures involves inspection for wounds and flail segments, palpation for tenderness and crepitus, and auscultation for asymmetry, poorly transmitted breath sounds, or cardiac impulses. When airbags have deployed, occult trauma (e.g., ocular injury) should be expected and specifically ruled out. Similarly, a seat belt sign across the abdomen is a significant harbinger of serious traumatic injury.

Examination of the pediatric abdomen is most reliable when performed on a cooperative patient. It is an insensitive screen-

BOX 35-6 INDICATIONS FOR LAPAROTOMY

Hemodynamic instability despite aggressive resuscitation and appropriate emergency department procedures (e.g., a decompression hemothorax or tension pneumothorax)
 Transfusion of $\geq 50\%$ of total blood volume because of massive intraperitoneal bleeding
 Radiographic evidence of pneumoperitoneum, intraperitoneal bladder rupture, grade V renovascular injury
 Gunshot wound to the abdomen
 Evisceration of intraperitoneal or stomach contents
 Signs of peritonitis
 Evidence of fecal or bowel contamination on diagnostic peritoneal lavage

ing test for the presence of an injury, however. The diagnostic test of choice to assess intra-abdominal injury in stable trauma patients is rapid abdominal computed tomography (CT).¹⁰ The role of diagnostic peritoneal lavage (DPL) and bedside ultrasonography is more limited. The finding of intraperitoneal hemorrhage alone is not necessarily an indication for surgery in a stable pediatric patient. The ultrasound FAST exam is a useful screen, however, because when positive, it will clarify the need for abdominal CT, close observation, and possible repeat ultrasound exams. In hemodynamically unstable children, FAST or DPL may point to the abdomen as the primary area in need of hemorrhage control. Because of the desire for splenic salvage to maintain immunocompetency, an injured spleen is often left in place as long as the patient can be resuscitated adequately with crystalloid and blood products. Indications for surgery are listed in **Box 35-6**. Whatever the surgical preference within a health care facility, it is important to establish a protocol for approaching these challenging patients. Patients who remain hypotensive after adequate crystalloid infusion, have active arterial bleeding on CT scan, or have consistent decreases in their hemoglobin level may be candidates for early operative intervention.

A rectal examination provides information concerning sphincter tone, prostatic position, and the presence of blood in the stool. Unfortunately, the rectal exam lacks sensitivity, so its findings, when negative, are often misleading. Studies have demonstrated that the rectal exam has a sensitivity of only approximately 33% for any injury, and it misses the majority of urethral injuries, rectal wall injuries, pelvic injuries, bowel injuries, and spinal injuries. In a subset analysis of children younger than age 18 years, it performed even more poorly. The rectal exam is not required in all cases and should only be performed when its result has a reasonable chance of meaningfully changing the patient's treatment.¹¹ Although urethral injury is rare in children, all trauma patients should be assessed for a perineal or lower abdominal hematoma and blood from the urethral meatus. A retrograde urethrogram should be completed to assess for urethral trauma prior to the insertion of a urinary catheter in cases with suggestive physical findings.

Examination of the extremities is directed toward the evaluation of any deformities, penetrations, and interruptions of perfusion. Most fracture sites may be stabilized with splinting until surgical intervention can be carried out. Careful neurologic examination and documentation should occur in all cases of significant extremity injury, with repeat assessments as indicated. Early orthopedic consultation is advisable.

The pediatric patient is at great risk for the development of hypothermia. This risk is based on the large amount of surface

area relative to body weight as well as increased transcutaneous heat conductance, especially in neonates. As a major contributor to the "triad of death" (acidosis, coagulopathy, and hypothermia), the importance of maintaining normothermia in trauma patients cannot be overemphasized.¹² Core temperatures should be monitored in these vulnerable patients, typically with temperature-sensing urinary catheters. Supplemental external warming techniques should be employed when necessary to maintain normothermia.

Pain Control

Pain control is an essential part of any trauma patient's management. Pain control may include not only medications but also techniques designed to change the perception and attention of the patient away from noxious stimuli and toward more pleasant experiences. The mainstay of pain control is narcotic analgesics, which should be used appropriately. Fentanyl has an advantage over morphine due to its hemodynamic profile and should be used readily but appropriately. It does not cause the release of histamine with secondary hypotension. Initial orders should generally be for both a loading dose and a PRN dose so that the nurse can continue to adequately control and assess the patient's pain after the physician has left the bedside. In addition to narcotics for pain control, immobilization of fractures and extremities with significant soft tissue injury also helps control pain. Visual imagery and distraction techniques, as well as a consistent and calm approach to the patient, should be utilized. Child life specialists (when available), patient representatives, chaplaincy, and most parents can assist in this endeavor. When asked if they want pain medicine, many children will say "No," even when they are in pain, because they are afraid of getting a shot or they interpret "pain medicine" as medicine that will cause pain. Basing pain medication on pain scales and common sense seems to be most appropriate.

In head-injured patients, fentanyl has the additional advantage of a short duration of action. If the patient has a mental status change, fentanyl clears quickly, making it possible to differentiate worsening brain injury from side effects of the medication. This is generally a better option than reversing the pain medicine and pain control with a narcotic antagonist. It is not humane to withhold pain medication completely in a traumatized patient whose mental status is of concern; it is better to titrate with smaller doses of short-lived medications. If immediate concern arises, the narcotic can be reversed with very small doses of naloxone.

Diagnostic Evaluation

Laboratory Studies

Blood sampling for a pediatric trauma patient is no different than that for an adult trauma patient; however, use of smaller blood collection tubes and microtechnique by laboratory staff may be necessary in infants and small children. All older pediatric trauma patients should be assessed for the possible use of drugs or alcohol as contributing factors to the traumatic event. In patients with hypovolemic shock, the hemoglobin alone is unreliable because equilibration will not have occurred at the time of presentation to the emergency department.¹³

A bedside glucose should be obtained on all patients with significant trauma. Repeat testing 30 to 60 minutes after significant pediatric trauma may be indicated as well. Children's glucose utilization and metabolic rate per kilogram is much greater than that of an adult, and they have far less substrate reserve in the form of glycogen stores. Any child with a change in mental status after trauma should have a bedside glucose

checked immediately. Any child requiring dextrose due to hypoglycemia will likely need an ongoing dextrose supply to prevent recurrence of hypoglycemia. In patients who can eat, this may be a meal with starches, fats, and protein. In others, it may require intravenous dextrose.

Radiology

The most important “traditional” radiographs to obtain on moderately to severely injured children are of the chest and pelvis to assess for sites of blood loss or potential causes of shock. In stable, alert children without distracting injuries, the pelvic film may be eliminated if no suggestion of sacral or pelvic fracture is found on thorough clinical exam. The following seven criteria are required to rule out any relevant pelvic fracture: patient age older than 3 years, no impairment of consciousness, no other major distracting injury, no complaint of pelvic pain, no signs of fracture on inspection, no pain on iliac or pubic symphysis compression, and no pain on hip rotation or flexion.¹⁴⁻¹⁷ In patients with remarkable sacral tenderness and negative plain films, a CT scan should be strongly considered. Sacral fractures can be difficult to discern reliably on plain films. The radiographs of the cervical spine may be delayed until after further diagnostic studies are obtained, depending on the clinical presentation of the patient.

Other radiographs are obtained based on the physical examination. For patients sustaining minor trauma, no radiographs may be needed. Children younger than 2 years with injuries consistent with child abuse should undergo a skeletal survey, including skull, chest, abdomen, and long bone radiographs. Generally, these should be completed on a nonemergent basis while in the hospital. In most cases, they can be scheduled in the inpatient radiology department with the pediatric radiologist or the most experienced radiologist on staff available to interpret the films.

Although not considered part of the primary survey, consideration of cervical spine CT instead of plain radiographs in those with mental status changes and significant trauma, those receiving a head CT scan for a head injury, and those at high clinical pretest likelihood for cervical fracture may be appropriate. Although the negative predictive value of plain radiographs in low-risk populations seems high, the sensitivity of plain films to detect fractures is far less impressive. The pretest likelihood of fracture must be considered when making decisions regarding the removal of cervical immobilization in children with apparently normal radiographs. Patients with continued neck pain despite negative radiographs and/or CT may require magnetic resonance imaging (MRI) evaluation, delayed flexion-extension views, or, in rare cases, evaluation by neurosurgery under fluoroscopy. The use of immediate flexion-extension views is rarely indicated or helpful.

Electrocardiogram after Electrical Injury

The majority of pediatric electrocution cases are due to household current (≤ 240 V). Asymptomatic patients who did not have ventricular tachydysrhythmias in the field or water contact at the time of their electrocution with household current and do not have dysrhythmias in the emergency department are at very low risk for significant arrhythmias. Current literature does not indicate a need for an electrocardiogram (ECG) or monitoring in these children.¹⁸ Those patients with normal and nonspecific ECG changes, when one is performed, remain at low risk. Nonspecific changes generally resolve within 24 hours. ECGs are indicated, as is cardiac monitoring for at least 4 hours, in patients who experience high-voltage electrocution.

SPECIFIC DISORDERS/INJURIES

Head Injury

Perspective

Head trauma is the leading cause of death among injured children and is responsible for 80% of all trauma deaths.³ Each year, 29,000 children younger than age 19 years experience permanent disability from traumatic brain injury. Falls account for 37% of pediatric head injuries,¹⁹ MVCs account for 18%,²⁰ pedestrian injuries account for 17%, and falls from bicycles account for 10%. On an age-related basis, infants and toddlers are more prone to falls from their own height, school-age children are involved in sports injuries and MVCs, and all ages are subject to the sequelae of abuse.

Principles of Disease

An important anatomic difference of a pediatric patient compared with an adult is that the cranial vault of a child is larger and heavier in proportion to the total body mass. This anatomic characteristic predisposes children, specifically toddlers and infants, to high degrees of torque that are generated by any forces along the cervical spine axis. Sutures within the pediatric skull are both protective and detrimental to the outcome of head injury in these patients. Although the cranium may be more pliable relative to traumatic insult, forces are generated internally that predispose the pediatric patient to parenchymal injury in the absence of skull fractures. The pediatric brain is less myelinated, predisposing it to shearing forces and further injury.^{3,21}

Clinical Features

The clinician must obtain as many details regarding the traumatic event as possible. The height of the fall or injury is particularly important with regard to the development of associated injury. Most children fall from their own height. It is important to consider the quality of the surface at the point of impact, specifically the presence or absence of carpeting within the home or location of injury. Impact with an object increases the localized force even after a short fall and may lead to increased risk for fracture and intraparenchymal injury. Children involved in MVCs are best evaluated by the degree of restraint that was present during the time of the accident. An infant in a properly installed car seat is likely to have a better outcome than an unrestrained infant. Unrestrained children involved in high-speed crashes are prone to serious injury.

In most cases, it is important to establish whether there was alteration of consciousness at the time of the injury event. With playground trauma, the history may be vague, and the interpretation of any change in consciousness of the child may be regarded as an actual loss of consciousness. The behavior of the child after the event should include questions related to the presence or absence of irritability, lethargy, personality change, abnormal gait, or other alterations in behavior. Any worsening of these symptoms since the injury should also be reported.

The prognostic significance of vomiting after pediatric head trauma is unclear. There is no adequate study defining an acceptable time frame in which vomiting after head injury is benign in nature. The development of seizures after head trauma, in contrast to vomiting, has been well studied.²¹ A brief seizure that occurs immediately after the insult (with rapid return of normal level of consciousness) is commonly called an *impact seizure*. This seizure usually is not associated with intracranial parenchymal injury. A CT is not necessary if the only

concern is the impact seizure; the decision to scan should take into account the mechanism of injury and current neurological status of the child. An impact seizure does not mandate the institution of anticonvulsant therapy. Seizures that occur later (>20 minutes after the insult) portend the greater possibility of traumatic brain injury and the development of seizures at a later date. CT is indicated for children with late seizures after head injury. Patients who experience seizures later in the course of the posttraumatic event are best evaluated by the neurosurgical service. As in all instances of trauma, a careful history related to the possibility of substance abuse must be obtained.

The physical examination of a head-injured child must include strict attention to the ABCs of emergency care. Although internal injuries are important in the outcome of these patients, the maintenance of oxygenation and perfusion is paramount in eliminating further insult. Because the pediatric brain is sensitive to decreases in glucose, oxygen, and perfusion, their maintenance optimizes the chances of good recovery. Strict attention must be paid to the maintenance of euvolemia because cerebral perfusion pressure (CPP) is adequate only in the face of a normal mean arterial pressure (MAP). Conceptually, CPP is equal to MAP minus intracranial pressure (ICP): $CPP = MAP - ICP$. As the blood pressure is reduced, so is CPP. Localized cerebral perfusion pressure at the site of injury and in the areas surrounding it may vary greatly from that approximated by this formula. Pediatric patients with any form of head injury should be evaluated and protected from cervical spine injury.

Several methods are available for evaluating head-injured patients, including AVPU and the GCS. A commonly used modification of the GCS for children is shown in Table 35-6. Although widely utilized, none of the pediatric modifications of the GCS have been well verified.²² However, studies on the reliability of the GCS in predicting the outcome in children with traumatic brain injury provide optimism compared with those on head-injured adults. In a study involving 80 children with traumatic brain injuries admitted to an intensive care unit (ICU), initial GCS scores were compared with eventual outcome.²³ ICU length of stay and time to cognition relative to GCS scores indicated that scores greater than or equal to 6 were associated with favorable outcomes and neurologic status. Although the number of patients in this study was small, the important message is that no matter how the patient presents neurologically, all efforts should be initiated to ensure survival and maintain stable neurologic status in the emergency department.

Examining a brain-injured child involves mental status testing, cranial nerve testing, motor testing, sensory testing, and short-term memory testing, with additional cognitive function testing under stress when indicated. The evaluation of cranial nerve function is essentially no different from that of an adult. The most important aspect of motor and cranial nerve evaluation involves ruling out the presence of increased ICP. Common symptoms and signs of increased ICP in infants and children should be sought (Boxes 35-7 and 35-8).

Minor injury to the scalp of infants and children involves the development of three common injury complexes.²⁴⁻²⁷ In order to better understand these injury complexes, the layers of the “SCALP” (skin, connective tissue, aponeurosis, loose areolar tissue, and periosteum) must be considered. *Caput succedaneum* refers to injury with hematoma in the connective tissue layer. This is freely mobile and crosses suture lines. A *subgaleal hematoma* refers to a hematoma that is subgaleal within the loose areolar tissue above the periosteum. Lastly, *cephalohematoma* refers to a collection of blood under the peri-

BOX 35-7

COMMON SYMPTOMS AND SIGNS OF INCREASED INTRACRANIAL PRESSURE IN INFANTS

- Full fontanel
- Split sutures
- Altered state of consciousness
- Paradoxical irritability
- Persistent emesis
- “Setting sun” sign (bilateral downward gaze of the eyes with apparent inability to elevate the eyes superiorly in a normal manner leading to an area of sclera being seen between the iris and the upper palpebra when the child attempts to look upward). This finding can be a normal finding when intermittent in infants younger than 7 months of age, especially with the withdrawal of a light stimulus.

BOX 35-8

COMMON SYMPTOMS AND SIGNS OF INCREASED INTRACRANIAL PRESSURE IN CHILDREN

- Headache
- Stiff neck
- Photophobia
- Altered state of consciousness
- Persistent emesis
- Cranial nerve involvement
- Papilledema
- Hypertension, bradycardia, and hypoventilation
- Decorticate or decerebrate posturing

osteum. Since the periosteum is tightly adhered to the various suture lines, the cephalohematoma does not cross them. Bleeding from scalp wounds is often profuse and can lead to hemodynamic embarrassment in infants and small children if not quickly controlled. Although children may develop shock from a scalp injury, it is prudent to look elsewhere while controlling this bleeding.

Skull fractures in children occur in many different configurations.^{19,21,28} Linear fractures, the most common type of skull fracture, rarely require therapy and often are associated with good outcomes. Factors favoring a poor outcome include the presence of the fracture overlying a vascular channel, depression, a diastatic fracture, or a fracture that extends over the area of the middle meningeal artery. Diastatic fractures, or defects extending through suture lines, are different from linear fractures in that leptomeningeal cysts (growing fractures) may develop at these sites. Fractures of the basilar portions of the occipital, temporal, sphenoid, or ethmoid bones commonly occur in children. The presence of cerebrospinal fluid rhinorrhea and otorrhea has been associated with these injuries. Signs of basilar skull fractures in children are similar to signs in adults and include posterior auricular ecchymosis (Battle’s sign) and raccoon eyes (the presence of periorbital subcutaneous hematoma).²⁹

Strictly speaking, *concussion* is defined as a brain insult with transient impairment of consciousness. Amnesia is often involved. Patients who sustain concussive insults have anorexia, vomiting, or pallor soon after the insult. This transitional period is followed by rapid recovery to baseline. A CT scan is usually not indicated; if one is obtained, it is most often normal. In contrast, contusions are often the result of coup and contrecoup forces at work. Contusions may not be associated with

any loss of consciousness at the time of insult. Patients often present with associated symptoms, such as altered level of consciousness, severe headache, vomiting, or focal deficits on neurologic assessment. These injuries are clearly demonstrable on CT.

Traditional teaching regarding the development of epidural hematomas involves the typical triad of head injury followed by a lucid interval, followed by rapid deterioration as intracranial hemorrhage worsens. In contrast to epidural hematomas in adults, pediatric epidural hematomas may be the result of venous bleeding, which predisposes them to a delay in the development of symptoms. Guardians should always be informed of delayed signs and symptoms following head trauma that should prompt immediate reassessment. In any event, epidural hematomas are associated with a high incidence of overlying skull fractures (60–80% of cases). Patients with small fracture-related epidurals localized only to the site of the inner table fracture should be monitored closely in the hospital but often do not require surgical intervention.

Special attention should be directed toward infants and toddlers to rule out the presence of subdural hematomas. This clinical scenario is most often secondary to rupture of bridging veins and rarely is associated with the presence of overlying fractures (<30%). Subdural hematomas most commonly occur in patients younger than age 2 years, with 93% of cases involving children younger than age 1 year. Chronic subdural hematomas are most often encountered in patients who have been subjected to what has been termed the “shaken baby syndrome.” This clinical complex involves forcible shaking of the child with accelerating and decelerating forces impacting the cranial vault.³⁰ This syndrome is most often due to child abuse; 22% of abused children have central nervous system injuries. Patients present with nonspecific findings, such as vomiting, failure to thrive, change in level of consciousness, or seizures. Retinal hemorrhages are present in the majority of cases, and all patients should have careful funduscopic examinations to rule out the presence of these nearly pathognomonic findings. Definitive exams should be performed by an ophthalmologist after pupil dilation to characterize the specific type of retinal hemorrhage. Those that occur in multiple retinal layers, are diffuse, and extend to the periphery are more likely secondary to abuse. Similarly, in less severe evident trauma, subdural hematomas at multiple sites, over areas other than the convexities, in the posterior fossa, or in the posterior interhemispheric fissure should strongly suggest the possibility of nonaccidental trauma.^{31,32} Left to their own development, the worst cases may manifest with signs of increased ICP. Retinal hemorrhages are not observed in children with mild to moderate trauma from other causes and are not associated with a prior history of cardiopulmonary resuscitation; the presence of retinal hemorrhages suggests child abuse. Coagulation studies, platelet count, platelet function assays, and, when indicated, metabolic tests for glutaric aciduria should be performed in these cases as well.

Diagnostic Strategies and Management

As a basic rule, serial examinations are the most reliable indicators of clinical deterioration.^{21,28} The presence of focality is a reliable indicator of a localized insult, whereas the absence of focality may be misleading. The signs of increased ICP usually develop late in the course of the process in infants. As in an adult, papilledema may require days to develop. The classic Cushing’s response (bradycardia and hypertension) is also unreliable in children. If ICP elevation is suspected, emergency intervention must be initiated immediately (Table 35-8).

The Monroe-Kelly doctrine describes the contents of the skull to be made up of essentially three compartments: brain, cerebrospinal fluid, and blood. The volume of the skull is fixed. Although not a perfect model, this doctrine suggests the effects that changes in each compartment may have on the others. For example, in the presence of an intracerebral hemorrhage of significant volume, either cerebral spinal fluid or brain must leave the cranial vault. Similarly, if the brain swells, cerebral spinal fluid, blood, or both must leave the cranial vault. When this balance is disrupted, and the autoregulatory system’s capacity to adapt is exceeded, the ICP rapidly increases. ICP can quickly reach a level that is not conducive to localized brain survival or continued blood flow to the brain. If left untreated, herniation may occur: An ICP over 20 to 25 mm Hg should be treated, but the absolute value less than this that should trigger treatment is unclear. From the standpoint of global cerebral perfusion, CPP is equated with MAP – ICP. However, this model does not allow the accurate prediction of CPP at the specific site of injury or within the ischemic penumbra. Measurement of oxygen extraction (using modifications of the Fick principle) and outcome studies have played a role in the following recommendations. In general, it is best to keep the cerebral perfusion pressure greater than 50 to 65 mm Hg in children and greater than 70 mm Hg in adults. There appears to be an age continuum with regard to necessary cerebral perfusion pressure. Hackbarth and coworkers demonstrated that the single greatest prognostic sign of outcome from traumatic brain injury in children was the ability to maintain a cerebral perfusion pressure greater than 50 mm Hg.³³ Many have adopted this as the minimum acceptable cerebral perfusion pressure.

The use of anticonvulsants after moderate to severe head injury in children is controversial. Early prophylaxis does not decrease the incidence of late seizures and is not recommended for this purpose. Clearly, the effects on temperature, intracranial oxygenation, and cerebral perfusion during an early seizure after trauma are discordant with the management principles of acute brain injury. In addition, early seizures often disrupt the evaluation and management of the patient’s head and other trauma. However, the evidence for phenytoin effectiveness in preventing early seizures after trauma is weak at best. Also, Young and coauthors demonstrated that in moderate to severe head injury the incidence of early seizure was much lower than expected and that phenytoin did not substantially lower this risk.³⁴ Others have suggested that topiramate or levetiracetam may be more effective with decreased risks of side effects.^{35,36} It may be prudent to treat seizures aggressively if they occur and to consider using sedative medications with anticonvulsant properties such as benzodiazepines while reserving the use of prophylactic phenytoin or fosphenytoin for the highest risk patients.

Most clinicians favor early and controlled intubation in pediatric patients with GCS scores that are deteriorating or are less than 9. However, in the out-of-hospital phase of care, or if the physician is not knowledgeable and experienced in pediatric rapid sequence induction, bag-valve-mask ventilation should be strongly considered during short transports and until additional, more experienced support personnel are available. An OG tube should be placed if bag-valve-mask ventilation is utilized to decrease the chance of emesis and to prevent respiratory embarrassment from gastric distension with air. Isolated head injury is uncommon; a careful search for other injuries should be made using meticulous and repetitive examinations as well as indicated laboratory and imaging tests.

Herniation syndromes in children are similar to those in adults. Uncal herniation is suggested early on by the presence of a unilaterally dilated pupil (compression of ipsilateral third

Table 35-8 Emergent Management of Increased Intracranial Pressure

THERAPY	DOSE	MECHANISM OF ACTION
Head elevation (30 degrees)		Lowers intracranial venous pressure
Head in midline		Prevents jugular vein compression
Hyperventilation	Maintenance P_{aCO_2} 38–42 mm Hg If acute increase in ICP then reduce P_{aCO_2} to 30–35 mm Hg	Promptly but temporarily decreases cerebral blood volume and thus intracranial pressure Only recommended for short-term treatment of acute ICP elevation
Mannitol	0.25–0.5 g/kg IV	Both agents effect rapid osmotic diuresis.
Hypertonic saline (HTS)	0.1–1 mL/kg of 3% Titrate to effect	Diuresis may decrease BP and CPP. Mannitol should be given through filter. HTS may require central line. Effect from osmotic and rheologic effects Avoid dehydration
Pentobarbital	5–10 mg/kg over 30 minutes, then 5 mg/kg/hr for 3 hours, then 1 mg/kg/hr Rarely indicated or started in emergency department	Thought to lower cerebral metabolism; also may have some effect on free radical formation. Other barbiturates (phenobarbital) also have been used. May decrease BP and CPP
Decompressive craniotomy		Allows more space for swelling and decreases ICP Potential value in children
Mild hypothermia (35° C)		Thought to decrease cerebral blood flow and metabolic rate Can cause cardiac dysrhythmias Is currently under investigation
Maintain euvolemia	Clinically or invasive monitoring	Maintenance of mean arterial pressure
Pressors if needed to maintain CBF	Depends on agent used	Maintain CBF and CPP by increasing MAP
Neuromuscular blockade	Depends on agent used	Helps maintain lower ICP
Sedation	Depends on agent used	Do not assume they are completely incapable of response to noxious stimuli or situation.
Prevent fever	Acetaminophen 15 mg/kg OG	Fever raises ICP and cardiac work.
Treat seizure aggressively	Depends on agent used	Prophylactic treatment controversial. Treatment of seizure not controversial and must be aggressive to prevent increased ICP, hypoxia, hyperpyrexia, and hypercarbia.

BP, blood pressure; CBF, cerebral blood flow; CPP, cerebral perfusion pressure; ICP, intracranial pressure; MAP, mean arterial pressure.

nerve parasympathetic fibers), contralateral hemiplegia (due to ipsilateral cerebral peduncle compression against the tentorium), and spontaneous hyperventilation. With progression, the ipsilateral eye may be noted to be looking downward and outward secondary to the loss of third nerve motor function but continued fourth and sixth cranial nerve function. Often, bilateral third nerve compression occurs very early, leading to bilateral “blown” pupils. In Kernohan’s phenomena, the temporal lobe compresses the contralateral cerebral peduncle against the tentorium, leading to ipsilateral paresis, making localization of the lesion challenging without neuroimaging. Small, sluggish pupils, decorticate posturing, and Cheyne-Stokes respirations characterize early central diencephalic herniation. If this progresses and extends to the pons or medulla, the patient will present with fixed and dilated pupils, flaccid muscle tone, and slow or apneustic breathing or frank apnea and cardiorespiratory arrest. Management of suspected acute herniation begins with immediate controlled hyperventilation.^{37,38} Clinical endpoints of hyperventilation are improved patient status or constriction of dilated pupils. End-tidal CO_2 capnography is used with arterial or venous blood gas correlation to assess adequacy of hyperventilation with a target partial pressure of carbon dioxide (P_{CO_2}) of 30 to 35 mm Hg. Excessive hyperventilation can result in excessive cerebral vasoconstriction and secondary brain injury; ventilation is started at an age-appropriate rate, and then the rate is increased until pupil-

lary function returns. Subsequent management of herniation includes hyperosmolar agents acutely, followed by other specific interventions in the ICU.³⁷⁻⁴²

Radiology

Skull Radiographs. Most clinicians agree that firm indications for skull radiographs alone include the skeletal survey involved with the evaluation of child abuse, establishment of a functioning ventricular peritoneal shunt, some penetrating wounds of the scalp, or the suspicion of foreign bodies underlying scalp lacerations. In children requiring neuroimaging due to concern for intracranial injury, plain skull radiography lacks sufficient sensitivity to be used as a screening tool and a noncontrast CT scan is the recommended test.

Computed Tomography of the Head. There has been a considerable amount of research on the indications and relative value of CT scanning in pediatric head-injured patients. A large study evaluated 185 children ages 2 to 17 years with loss of consciousness and GCS scores of 15 after mild head injury.⁴³ The children were grouped according to physical examination findings, neurologic status, and whether the head injury was isolated or nonisolated. Patients with obvious skull fractures were excluded. Two variables were highly associated with the presence of intracranial hemorrhage: the presenting neurologic status and the presence of multiple injuries. None of the 49

neurologically normal children with isolated head injury had intracranial hemorrhages. All patients with intracranial hemorrhages were noted to have other traumatic insults on physical examination. The authors concluded that after isolated head injury with any loss of consciousness, children older than 2 years of age who were neurologically normal could be discharged without a CT scan after careful physical examination alone.

Other studies contradict these findings, establishing a clear association with parenchymal injury and loss of consciousness.^{43,44} Currently, recommendations for CT scanning include the presence of neurologic deficits, GCS scores of less than 14, and injury patterns that are the result of major forcible insults. Studies have shown various combinations of characteristics that make significant intracranial injury very unlikely but have provided less guidance in selection of which patients actually need a head CT (high negative predictive value but low positive predictive value). Studies have shown that if these rules are utilized, they must be used exactly as they were performed in the study to be effective. Dunning and coauthors' meta-analysis showed a statistically significant correlation of intracranial hemorrhage with focality (relative risk [RR] = 9.4), skull fracture (RR = 6.1), altered level of consciousness (RR = 2.23), and GCS scores greater than 15 (RR = 5.51).³⁶ Children younger than 1 year of age are a special challenge to the clinician because their neurologic milestones are more difficult to evaluate. Within this age group, any loss of consciousness, protracted vomiting, irritability, poor feeding, or suspicion of abuse should trigger strong consideration for CT scanning. The value of brief loss of consciousness and the determination of the need for CT in the child more than 1 year old are less clear, but loss of consciousness lasting longer than a minute is considered an indication for neuroimaging by many practitioners.

The evaluation of infants with minor closed-head injury was studied in a series of 668 infants younger than 2 years of age who underwent CT scanning, in which a subset of 92 infants younger than 2 months of age was further scrutinized. The presence of a significant scalp hematoma highly correlated with underlying parenchymal brain injury. The authors recommended CT scans for these patients.⁴⁵

Cervical Spine Injury

Perspective

In the United States, more than 1100 children sustain spinal injury annually.⁴⁶ Cervical injury patterns vary with the age of the patient. Fractures below the C3 level account for only 30% of spinal lesions in children younger than 8 years of age, which differs dramatically from the patterns seen in adults. Likewise, spinal cord injury (SCI) without radiographic abnormality (SCIWORA) has been found in 25 to 50% of spinal cord injuries in this same age group.⁴²⁻⁴⁴ SCIWORA may be a misnomer in the era of MRI. Intra- or extraneural findings are usually seen immediately on MRI but may be delayed, necessitating immobilization and a follow-up MRI to prevent late or recurrent injury. Length of immobilization is controversial, but it may be up to 12 weeks.

Principles of Disease

Anatomic features of the cervical spine approach adult patterns between the ages of 8 and 10 years (Box 35-9).² Injury patterns identical to those of adults are often not fully manifested, however, until age 15 years. The pediatric spine has greater elasticity of the supporting ligamentous structures than the

BOX 35-9

ANATOMIC DIFFERENCES IN THE PEDIATRIC CERVICAL SPINE

Cervical spine fulcrum changes from C2–C3 in toddlers to C5–C6 by age 8–12 years.
 Relatively larger head size, resulting in greater flexion and extension injuries.
 Relatively large occiput in children younger than age 2 years leads to flexion of cervical spine if they are laid flat on standard backboard without support under their scapula and pelvis.
 Smaller neck muscle mass with ligamentous injuries more common than fractures.
 Anterior wedge appearance of cervical vertebral bodies is common.
 Increased flexibility of interspinous ligaments.
 Flatter facet joints with a more horizontal orientation.
 Upper 30°→65 degrees and lower 55°→70 degrees as one ages to adulthood.
 Incomplete ossification, making interpretation of bony alignment difficult (spondylolysis).
 Uncinate processes do not calcify until approximately 7 years of age.
 Basilar odontoid spondylolysis fuses at 3–7 years of age.
 Apical odontoid epiphyses radiographically apparent at 7 years of age but may not fuse until approximately 12 years of age.
 Posterior arch of C1 fuses at 4 years of age.
 Anterior C1 arch may not be visible until one year of age and fuses at 7–10 years of age.
 More inferior cervical vertebral segments.
 Neural arches fuse to body by approximately 7 years of age.
 Posterior arches fuse by 3–5 years of age.
 Epiphyses of spinous process tips may mimic fractures.
 Preodontoid space 4–5 mm in those <8 years of age and <3 mm in those 8 years or older.
 Pseudosubluxation of C2 on C3 seen in 40% of children <8–12 years of age.
 Prevertebral space size varies with phase of respiration.

adult spine. The joint capsules of the child have greater elastic properties, and the cartilaginous structures are less calcified than in adults. In the spine, there is a relatively more horizontal orientation of the facet joints and uncinate processes, and the anterior surfaces of the vertebral bodies have a more wedge-shaped appearance. Compared to the adult, the child has relatively underdeveloped neck musculature and a head that is disproportionately large and heavy compared with the body. Both of these differences lead to an “anatomic fulcrum of the spine” in children that is at the level of the C2 and C3 vertebrae versus the lower cervical vertebrae as found in adults.

Clinical Features

Any patient with severe multiple injuries should be considered to have an SCI until proved otherwise. Likewise, significant head, neck, or back trauma and trauma associated with speed, MVCs, and falls from any height (especially those with associated head injury) should raise suspicions for SCI and be evaluated appropriately. The evaluation of a pediatric patient should begin with a primary survey to assess airway patency, ventilatory status, and perfusion. After initial evaluation and stabilization, the cervical region can be examined. Palpation

of the neck for pain and bony deformity should be performed. If the patient has pain or tenderness, closely watching his or her facial expression will generally indicate more than asking, "Do you have pain?" Any continued concern or perceived discomfort should be taken seriously because ligamentous discomfort is sometimes subtle. In academic institutions, it is common for three or four people to repeat each aspect of the exam. It is not uncommon for children who originally tell you that something hurts to tell new examiners that it does not hurt because they quickly learn that when they affirm that they have pain, stimulus will continue to be applied, making the pain worse. One examiner finding tenderness should be enough to consider further evaluation.

Some factors, such as tenderness or pain with palpation, may be underappreciated in a child who is not yet old enough to talk. Similarly, patients with head injury, decreased level of consciousness, or distracting injury or those who are intoxicated may not reliably localize pain in the cervical region, and spinal precautions should be maintained to avoid potential additional injury.

The neurologic examination in a pediatric patient can be difficult, but several factors should be evaluated in a patient with suspected cervical spine injury. Pain in the cervical region should raise suspicion of cervical spine injury. Likewise, paralysis, perceived paresthesias, ptosis, and priapism are neurologic signs highly correlated with SCIs. Complaints of paralysis or paresthesias, even if completely resolved at the time of examination, should be considered an indication of SCI until proven otherwise. Finally, upper extremity position and function can help elucidate the presence and level of an SCI.

Several characteristic SCI syndromes can be diagnosed on initial emergency department evaluation. Spinal cord injuries are generally described as either complete or incomplete depending on the presence or absence of sensory or motor function. Incomplete SCI has a better prognosis for recovery of some motor function after spinal shock resolves. Incomplete lesions have some preservation, even if slight, of sensory or motor function below the area of SCI and at the area of sacral nerve distribution. The determination of complete or incomplete is not a one-time assessment and cannot be reliably made until after spinal shock has resolved. The performance of a rectal exam (or anal wink testing) or bulbocavernosus reflex testing in males can assess for sensation and motor ability in the sacral distribution. Central cord syndrome (seen mostly in extension injuries to the cervical spine) typically consists of arm findings (e.g., decreased tone) greater than leg findings and distal symptoms greater than proximal symptoms (e.g., burning pain in the fingers and hands). Anterior cord syndrome (associated with flexion injuries to the cervical spine) is characterized by complete motor paralysis with loss of pain and temperature sensation; however, position and vibration sensation are preserved in this disorder. Finally, Brown-Séquard syndrome represents hemisection of the spinal cord with ipsilateral loss of motor function and proprioception. There is also contralateral loss of pain and temperature sensation. SCI syndromes are rare in children.

Radiology

Some experts believe that children with neck pain, involvement in an MVC, or any suspicion of cervical injury should receive radiographic evaluation because when used as a screening tool, these factors may be very sensitive in identifying cervical spine injuries in this patient group. Other experts support the use of the NEXUS criteria to determine who needs cervical radiographs. These criteria were derived from a study of 3065 children younger than age 18 years; however,

only 4 of 30 cervical spine fractures were in children younger than age 9 years, and none of the 88 children younger than age 2 years had cervical fractures.^{47,48} No pediatric cases of SCIWORA were found and the data showed that 45.9% of cervical spine injuries in their cohort were between the levels of C5 and C7. This may reflect the fact that 2160 of the pediatric patients were between 8 and 17 years old. The sensitivity for detection of cervical fractures was reported as 100% (95% confidence interval = 87.8–100%); however, less than 1% of children in the study had an injury, making the 100% negative predictive value less meaningful and the sensitivity (at least in young infants) suspect.⁴⁷ The majority of injured patients were older than age 9 years and had characteristics more similar to adults than infants.⁴⁸ Because of the limitations of the NEXUS criteria as they pertain to children, a low threshold for imaging must be maintained in children with mechanisms worrisome for cervical injury. A report of discomfort, any significant distracting injury, or even transient neurologic symptoms should be considered an indication for radiologic evaluation.

Radiographic evaluation routinely should consist of three views: a cross-table lateral view, an anteroposterior view, and an open-mouth odontoid view to help visualize the odontoid process of C1. The sensitivity of the three-view cervical spine series is highly variable. The pretest likelihood of fracture must be taken into account when acting on the result or choosing an imaging modality. Interpretation of plain cervical spine radiographs in children may be especially challenging because of the anatomic changes that occur with growth (see Box 35-9). In addition, pseudosubluxation of C2 on C3 is common in children up to adolescence, occurring in approximately 40% of patients.⁴⁹ The emergency physician distinguishes between pseudosubluxation and true subluxation by the posterior cervical line and the relationship of the spinolaminar line, also called the line of Swischuk, to the anterior cortical margin of the spinous process at C2. A line is drawn from the anterior cortical margin of the spinous process of C1 down through the anterior cortical margin of C3. If this line at C2 crosses the anterior cortical margin of the spinous process at C2 or is anterior to it by less than 2 mm and no fractures are visualized, the patient likely has pseudosubluxation versus true subluxation at that level (Fig. 35-1).

An important criterion for radiographic clearing of the cervical spine is complete visualization of all seven cervical vertebral bodies down to and including the C7-T1 interface. The predental space should not exceed 4 or 5 mm in children younger than 10 years of age, and the prevertebral soft tissue space should not be greater than normal (variable but generally <1/3 to 1/2 vertebral body width). The four cervical radiographic lines should be evaluated, and the atlanto-occipital alignment should be assessed for dislocation in this region. Other imaging modalities that can be used to delineate cervical fractures include thin-section CT and magnetic resonance imaging. If the dens cannot be adequately assessed by the open-mouth odontoid view, then a transforaminal view or CT scan should be utilized. Patients with high clinical suspicion for fracture but negative plain radiographs should be considered candidates for computed tomographic evaluation and radiologic, orthopedic, or neurosurgical consultation. Eubanks and co-workers provide a discussion on clearing the pediatric cervical spine in their review article.⁵⁰

Classically, young children have been considered at greater risk for upper cervical spine injury. Unfortunately, many occipital cervical junction injuries are immediately fatal. However, survival is possible in some cases.⁵¹ Early detection and immobilization is crucial. Occipital cervical junction injuries should be suspected in any child pedestrian versus vehicle

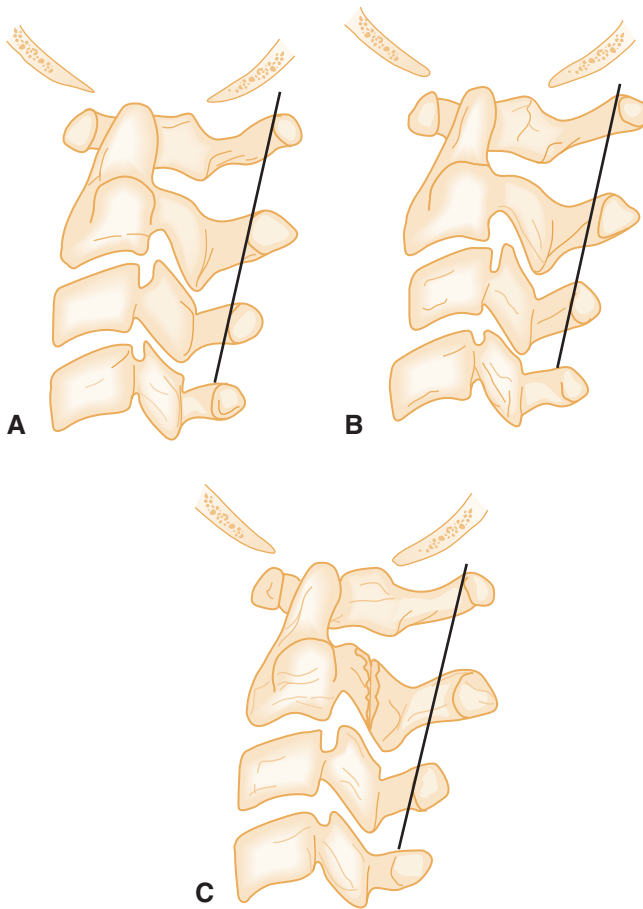


Figure 35-1. Spinalolaminar line. Use only to access anterior displacement of C2 on C3. A line is drawn from the cortex of the spinous process of C1 to the cortex of the spinous process of C3, and the relationship of the spinous process of C2 is noted. **A**, Normal line passing through the cortex of C2. **B**, Normal line passing within 1.5 mm of the cortex of C2. **C**, Abnormal line passing greater than 2 mm anterior to the cortex of C2, suggesting underlying fracture of posterior elements of C2. (From American College of Emergency Physicians, American Academy of Pediatrics: APLS: The Pediatric Emergency Medicine Resource, 4th edition, Dallas, Elk Grove Village, Ill, 2004, the College and the Academy.)

accident, especially if a child presents with a laceration under the chin from a forward fall. In many fatal cases, distraction and displacement is obvious. However, in nonfatal cases it can be subtle. A Power's ratio greater than 1 indicates an atlanto-occipital dislocation until proven otherwise (normal, approximately 0.77). Power's ratio is calculated as the ratio of the distance from the basion to the anterior cortex of the posterior arch of the atlas divided by the distance from the opisthion to the posterior cortex of the anterior arch of the atlas. An additional method to suggest this injury is to draw a vertical line from the posterior border of the odontoid and then measure the distance from this line to the basion. If this distance is greater than 12 mm, then atlanto-occipital separation should be suspected. In addition, a traumatic or even sometimes non-traumatic atlantoaxial rotatory subluxation should be suspected in a child with a fixed rotatory cervical abnormality. Classically, this can be differentiated from a muscular torticollis in non-traumatic cases by the history, the time course, and palpation of the sternocleidomastoid muscle on the side contralateral to the direction in which the chin is pointing. When atlantoaxial rotatory subluxation cannot be confidently ruled out clinically, plain radiographs or CT scan should be utilized. In children with upper cervical spine tenderness, it is prudent to consider

a fracture of the synchondrosis between the odontoid and C2. This can be difficult to diagnose on plain radiograph, but it is often recognized as a subtle anterior tilt to the odontoid on C2. A CT scan with sagittal reconstructions will clarify this entity.⁵²

Management

There are two phases of SCI. Direct injury (initial phase) results in largely irreversible injury to the spinal cord. Indirect injury results from preventable or reversible injury to the spinal cord secondary to ischemia, hypoxemia, and tissue toxicity. Resuscitation of a patient with injury to the cervical spine should focus on prevention or minimization of the indirect causes of injury to the cervical spine. Management of possible spinal cord or column injury should begin in the out-of-hospital phase of emergency care. Most injured children arrive at the emergency department with adequate immobilization. Some more recent evaluations of traditional cervical collars and rigid backboards have shown less than adequate neutral positioning of pediatric patients related to their relatively large cranium in proportion to the rest of their body. Nevertheless, in the absence of modified backboards with cutouts for the occiput of the child, the child should be immobilized with stiff cervical collar, rigid backboard, and external fixation by means of head blocks, cloth tape, or straps to provide adequate precautions. Appropriate padding should be placed under the patient to approximate neutral alignment of the cervical spine and help prevent pressure-related injury. Some emergency medical service agencies' protocols call for small children to be immobilized in their car seats in some circumstances.

Breathing should be assessed to determine the presence of hypoventilation. Patients with SCI may hypoventilate because of diminished diaphragmatic activity or intercostal muscle paralysis. Otherwise normal children held in a supine position have shown reduced ventilatory abilities as measured by the forced vital capacity. Head or chest injury or pulmonary compromise related to contusion, aspiration, or other causes likewise may contribute to ventilatory embarrassment. Supplemental oxygen should be given routinely, and ventilatory assistance by bag-valve-mask ventilation or definitive airway management should be considered in the presence of clinically significant hypoventilation. Finally, circulatory status must be assessed early in the trauma patient and needs to be addressed promptly to prevent end-organ perfusion deficits. Hypotension can result from hypovolemia, neurogenic shock, spinal shock, or a myriad of other less common causes. Spinal shock presents with lower extremity findings of SCI, with flaccid paralysis of skeletal and smooth muscle leading to the appearance of a relative hypovolemia due to diminished systemic vascular resistance. Spinal shock generally resolves in hours to approximately 1 day once some spinal level reflexes return below the site of injury. Neurogenic shock typically occurs after injury to the spinal cord above the level of approximately T6. Patients with neurogenic shock lose their sympathetic tone and present with hypotension in the face of unopposed parasympathetic action such as bradycardia. In each case, fluid administration, parasympathetic receptor blocking agents such as atropine or glycopyrrolate, and vasopressors with chronotropic, vasoactive, and inotropic characteristics (e.g., dopamine) are used. If spinal shock with normal chronotropy and inotropy is found, then fluids and agents with more peripheral vascular vasoconstrictive properties may be preferable, such as phenylephrine or norepinephrine. Spinal shock remains a diagnosis of exclusion, once hemorrhagic shock has been definitively eliminated.

Any patient with definite SCI requires added precautions to ensure appropriate immobilization of the cervical spine. The use of intravenous steroids for blunt injury to the spinal cord continues to be debated in the literature (see Table 35-8). Immediate evaluation by a spinal cord specialist should be sought for all children with SCI. In the absence of such a specialist, the patient should be transported to a center with adequate facilities to care for spinal cord-injured patients.

Even when thoracic or lumbar fractures exist, patients should be expeditiously removed from the backboard to prevent discomfort and morbidity. Sliding boards (smooth movers) can be used to move patients onto scanner tables and back to their trauma beds. Mandatory interhospital transfer rules that require patients to remain on backboards during transport should be discouraged. The initial physician receiving the patient should be able to determine the necessity of the backboard for cervical spine immobilization during transport and, when appropriate, remove the patient from the board before transport.

Cardiothoracic Injury

Perspective

Most serious chest injuries in children (83%) are the result of blunt trauma.^{6,53} Most result from MVCs. Isolated chest injury is a relatively infrequent occurrence considering the typical mechanisms of blunt trauma in the pediatric patient. The presence of significant chest injury increases the potential for multisystem trauma mortality by a factor of 10. Sequelae of blunt injury include rib fractures and pulmonary contusion (50%), pneumothorax (20%), and hemothorax (10%).

The overall mortality of pediatric chest trauma is nearly equivalent for blunt versus penetrating trauma. Children subjected to penetrating trauma, in contrast to the injuries associated with blunt trauma, often die from the primary insult. Penetrating trauma accounts for only 15% of thoracic insults in children.⁵³⁻⁵⁵ Nationwide misuse of firearms has resulted in an increasing incidence of penetrating trauma, often with children as victims. The vast majority of these cases are related to the criminal use of handguns; however, improper storage and poor parental supervision lead to devastating consequences in a relatively small, but nevertheless preventable, number of cases each year. Families of children with emotional difficulties or depression should consider removing guns from their homes due to their lethality when used as an instrument of suicide. Specific clinical patterns should alert the clinician to the potential for concurrent abdominal and thoracic injury. Any patient with penetrating trauma at or below the level of the nipples falls into this category. Apparent isolated thoracic trauma does not exclude abdominal injury. All patients with self-inflicted penetrating trauma should also be assessed for ingested toxins.

Principles of Disease

It is important to understand the physiology of pediatric respiration when considering the potential for early decompensation following chest injury. Infants and young children are preferential diaphragm breathers, and any impairment of diaphragmatic mobility compromises ventilation. The presence of gastric distention elevates the diaphragm and severely diminishes the vital capacity of a child. In addition, the particular types of muscle fibers involved in the diaphragm of infants and young children predispose to the sudden development of apnea when these muscles become fatigued. Unlike adults, whose thoracic wall musculature can pull the ribs up

anteriorly giving a larger circumference to the chest wall, children's chest wall circumference does not change drastically during respiration because their chest is barrel-like throughout the respiratory cycle. This also decreases the ability of children to increase their vital capacity. For these reasons, children will increase ventilation typically by increasing their respiratory rate. Most important, the presence of adequate oxygenation in a pediatric patient does not always ensure sufficiency of ventilation; confirmatory auscultatory and other physical findings are essential. End-tidal CO₂ capnography can be very useful in this regard in both the intubated and the nonintubated trauma patient.

Infants and children are anatomically protected against blunt thoracic cage trauma because of the compliance of the rib cage. Compressibility of the rib cage dissipates the force of impact, which lessens the likelihood of bony injury. These protective mechanisms also may mask fairly complex pediatric thoracic insults. The compliance of the rib cage allows significant injury to occur with little apparent external signs of trauma. Multiple rib fractures are a marker of serious injury in children, with child abuse being the most likely etiology, especially when fractures are posterior and in various stages of healing. In addition, the pediatric mediastinum is mobile, which favors the development of rapid ventilatory and circulatory collapse in the presence of a tension pneumothorax.

Specific Disorders

Pneumothorax. The development of a traumatic pneumothorax is commonly associated with significant pulmonary injury. In contrast to spontaneous pneumothoraces, these insults do not resolve spontaneously and often are associated with the presence of a hemothorax. Signs and symptoms include external evidence of chest trauma, such as abrasion, contusion, or ecchymoses; tachypnea; respiratory distress; hypoxemia; and chest pain. Decreased breath sounds may not be appreciated in children with pneumothoraces because of the wide transmission of breath sounds in the chest and upper abdomen. It is critical to listen to the chest from the axilla in children. This location helps with lateralization to distinguish decreased breath sounds on one side compared to the other and, after intubation, to assess for proper endotracheal tube position.

Management of a hemopneumothorax noted on chest radiograph includes the placement of a large-caliber chest tube placed far enough posteriorly, near the mid-axillary line, to prevent encroaching on more anterior soft tissue that will later become part of the breast. Chest tube size for hemopneumothorax management can be estimated as four times the size of the endotracheal tube that would be used in the patient (the age plus 12–16) or can be found on a length-based resuscitation tape. A chest tube should be considered for any patient with a pneumothorax who will be undergoing mechanical ventilation. In the most conservative of scenarios, such as small (<20%) simple pneumothoraces that are not under tension in a child who will not be mechanically ventilated, the child may be observed carefully for extended periods with 100% oxygen supplementation for nitrogen washout, and reassessment can be done by repeat chest radiographs at selected intervals or a pigtail catheter can be placed percutaneously using a modified Seldinger technique.

Open Pneumothorax. An open pneumothorax exists when the chest wall is injured sufficiently to allow bidirectional flow of air through the wound. The patient is unable to expand the lung because of equalization of pressures between the atmosphere and the chest cavity. Ventilation and oxygenation are severely impaired.

Management of an open pneumothorax is dictated by the size of the defect and the amount of respiratory compromise. Simple, small puncture wounds in a breathing patient may be treated by covering the chest wall defect with occlusive dressing, such as sterile petroleum gauze. A separate incision should be made for the placement of a thoracostomy tube. As in all cases, defects that are too large to seal adequately or patients who are severely impaired with regard to ventilation are candidates for intubation.

Tension Pneumothorax. Pulmonary air leaks that occur in a one-way valve arrangement favor the development of a tension pneumothorax. Increasing amounts of free air within the pleural cavity cause the mediastinal structures to shift toward the opposite side, compromising cardiac output. The final common pathway involves hypoxia, hypotension, and refractive shock. Most patients with tension pneumothoraces present with severe respiratory distress, decreased breath sounds (often bilaterally), and a shift in the point of maximal cardiac impulse. In the worst scenario, the shifted mediastinum forces contralateral tracheal deviation and distention of the neck veins due to decreased venous return to the thorax. In pediatric patients, signs of tension pneumothorax are often subtle. A short neck and increased soft tissue may make detection of tracheal deviation difficult. Pediatric patients with tension pneumothorax may only have subtle signs or present with only tachycardia, shock, and respiratory distress. The emergency physician should consider the diagnosis of tension pneumothorax and, if detected or strongly suspected, should treat the patient immediately with decompression. Without adequate decompression, respiratory embarrassment, hypotension, and circulatory collapse will occur.

In the out-of-hospital setting, treatment includes needle thoracostomy placed in the second intercostal space in the midclavicular line or possibly in the fourth intercostal space just above the rib and anterior to the mid-axillary line. The needle should be placed above the rib margin to avoid injuring the intercostal vessels. In the emergency department, definitive treatment involves the use of a large-caliber thoracostomy tube that favors drainage of the tension pneumothorax and any accompanying hemothorax.

Hemothorax. Significant bleeding may occur when injury is directed toward the intercostal vessels, internal mammary vessels, or lung parenchyma. Without an upright chest film, it is difficult to quantify the degree of bleeding on plain radiographs. A slightly less radiolucent appearance on the affected side of the chest may be the only sign on a supine radiograph, often associated with a pneumothorax. Development of a massive hemothorax is rare in children and is associated most often with severe impact, such as that seen in high-velocity MVCs, falls from extreme heights, or the use of high-powered firearms. These injuries must be evaluated and treated quickly. Clinically, patients present with decreased breath sounds and dullness to percussion on the affected side. A pneumothorax may coexist with a hemothorax. The pediatric patient may present with early or late signs of hypovolemic shock.

Any alteration in cardiovascular sufficiency should be treated with rapid fluid replacement with isotonic crystalloid solutions. The clinician must also prepare for transfusion with the institution of red blood cell replacement as necessary. Patients who present with profound shock may receive either type-specific or O-negative blood; crossmatched blood may be used for more stable patients. The amount of blood that is salvaged from the chest tube should be quantified to help determine the need for red blood cell replacement. Many centers have the capability to salvage blood from hemothoraces and reinfuse using an autotransfuser. As in all cases of trauma, initial measurement of the hemoglobin is often unreliable and typi-

cally underestimates the amount of blood loss due to inadequate time for equilibration.

The treatment of hemothorax includes a tube thoracostomy. The tube needs to be large enough to occupy most of the intercostal space and should be placed laterally and directed posteriorly. In the supine patient with simple pneumothorax, chest tubes are directed superiorly; in hemothorax, they are directed posteromedially. As in all interventions, repeat chest radiographs should be obtained to confirm tube position and document improvement in lung expansion. The emergency physician is often able to stabilize the patient with red blood cell replacement until surgical intervention is achieved.

Indications for thoracotomy include evacuated blood volumes exceeding 10 to 15 mL/kg of blood immediately after the placement of the chest tube, persistent blood loss (e.g., exceeding 2–4 mL/kg/hr over 3 hours), or continued air leak. Emergency department thoracotomy is reserved for patients with penetrating trauma who deteriorate to cardiopulmonary failure despite maximal resuscitation in the out-of-hospital setting or emergency department. Guidelines for emergency pediatric thoracotomy are often institution specific. Cothren and Moore have suggested an algorithm to guide emergency department thoracotomy in multiply injured trauma patients.⁵⁶ Contraindications for emergency department resuscitative thoracotomy after out-of-hospital cardiopulmonary resuscitation (CPR) include (1) blunt trauma with CPR for greater than 5 minutes with asystole and no signs of life on presentation without ultrasound evidence of cardiac tamponade and (2) penetrating trauma with CPR for greater than 15 minutes and asystole with no signs of life on arrival without ultrasound evidence of cardiac tamponade. Patients with penetrating chest trauma and CPR for less than 5 minutes may warrant a left anterior thoracotomy, whereas patients with blunt trauma should have rapid assessment by ultrasound for tamponade. If tamponade is present and CPR has been performed for less than 15 minutes, then a left anterior thoracotomy may be indicated.

Pulmonary Contusion. Penetrating and blunt thoracic trauma may result in the development of a pulmonary contusion. The compliance of the rib cage in children renders them susceptible to the development of pulmonary contusion even in the absence of external signs of chest trauma. Injury to capillary membranes allows the collection of blood within the interstitial spaces, resulting in hypoxia and respiratory distress. If bleeding is severe enough, oxygenation and ventilation are impaired. Initial chest radiographs may not show the classic findings of pulmonary consolidation. In addition, in the early stages of injury, blood gases may be normal.

Treatment of pulmonary contusions includes a careful evaluation for the presence of additional injuries because significant force is necessary to cause the contusions. Most patients may be treated with supplemental oxygen and close monitoring. Most pulmonary contusions resolve without sequelae. Rare cases are associated with the development of acute respiratory distress syndrome.

Traumatic Diaphragmatic Hernia. Children involved in MVCs who are wearing lap belts are predisposed to the development of diaphragmatic herniation.^{55,57-59} Mechanisms of injury involve sudden increases in intra-abdominal pressure. Patients initially present in stable condition, with the degree of respiratory distress directly proportional to the amount of abdominal contents that protrude into the pulmonary space. The presence of bruising from lap belt-only compression should alert the clinician to the possibility of diaphragmatic hernia and other intra-abdominal injuries (small bowel injury) and the possibility of associated thoracolumbar spinal insults such as Chance

fractures. Most commonly, the herniation occurs on the left side because the liver is protective against diaphragmatic rupture on the right.

Initial management for these patients involves placement of an NG tube to decompress the stomach. In cases of severe respiratory distress, intubation is indicated. Bag-valve-mask ventilation is avoided whenever possible. Surgery is required for repair of the injury.

Cardiac and Vascular Injuries. Injuries to the heart and large vessels are uncommon in children.⁵⁹⁻⁶¹ The most common traumatic cardiovascular injury sustained by children is myocardial contusion. Patients often present with chest wall tenderness or may have a complaint of generalized chest pain. Tachycardia is the most common finding. Elevation of myocardial enzymes may be diagnostic. Patients with myocardial contusions should be monitored closely for the development of dysrhythmias and impaired myocardial function; however, in most cases of myocardial contusion, there are no long-term sequelae. The most life-threatening scenario involving the cardiac structures is the development of cardiac tamponade. Penetrating wounds to the chest are not rare but are potentially survivable if myocardial penetration and tamponade are recognized immediately. Extravasated blood fills the pericardial space and impairs cardiac filling during diastole. Tamponade is most often the result of a penetrating wound. Firearm insult often causes sudden death, and blunt trauma rarely results in the development of cardiac tamponade. Clinically, patients present with tachycardia, distant heart sounds, narrow pulse pressure, jugular venous distention, and pulsus paradoxus. In the scenario of profound hypovolemia, venous distention is absent. The final common pathway involves the development of pulseless electrical activity. Ultrasound can characterize this injury in seconds and guide therapy.

In cardiac and vascular injuries, the electrocardiogram may show anything from tachycardia with low voltage (pericardial tamponade) to findings consistent with acute myocardial injury (ST segment elevation). In the subacute scenario, echocardiography often makes the diagnosis. Bedside transthoracic echocardiography defines the degree of pericardial effusion present and the significance of any diastolic dysfunction present. A simple single subxyphoid view provides the emergency physician with an excellent view of the pericardial sac and heart. Pericardiocentesis may be diagnostic and therapeutic. Definitive treatment involves drainage of the fluid from the pericardial sac. In certain situations, the amount of pericardial blood and clot necessitates the performance of a thoracotomy to evacuate the pericardium adequately.

Comotio Cordis. Comotio cordis is a disorder described in pediatric patients that results from sudden impact to the anterior chest wall (e.g., as seen in baseball injuries), which causes cessation of normal cardiac function.⁶¹⁻⁶³ The patient may have an immediate dysrhythmia or ventricular fibrillation that is refractory to resuscitation efforts. Significant morbidity and mortality are associated with this disorder, and although most recover completely, some patients require extended treatment with antiarrhythmic agents, cardiac pacemaker placement, inotropic agents, or intra-aortic balloon pump. In patients with prolonged cardiac instability, cardiogenic shock and death are common, despite maximal therapeutic intervention.^{62,63}

Abdominal Injury

Perspective

Serious abdominal injury accounts for approximately 8% of admissions to pediatric trauma centers.² Abdominal trauma is

the third leading cause of traumatic death in children after head and thoracic injuries. Abdominal trauma is the most common cause of unrecognized fatal injury in children. Pediatric abdominal trauma results from blunt causes in the vast majority of cases. Of patients presenting primarily for other associated injuries, 9% die from abdominal trauma associated with these injuries.

Blunt trauma related to MVCs causes more than 50% of abdominal injuries in children and is the most lethal. “Lap belt” injury, including small bowel injury and Chance fractures, may occur in approximately 5 to 10% of restrained children involved in MVCs.⁶⁴⁻⁶⁶ Another common cause of abdominal injury involves bicycle crashes. Handlebar injuries represent a serious cause of injury and subsequent hospitalization for the pediatric population; patients requiring admission have a mean hospital stay greater than 3 weeks. Often, the effects of bicycle injuries may not be seen on initial presentation, with the mean elapsed time to onset of symptoms being nearly 24 hours after injury. All children with epigastric pain after blunt trauma, especially when concentrated force has been applied in this area, should be considered to have duodenal hematoma until proven otherwise. Pancreatic injury, including transection, should be strongly considered as well.

Sports-related injuries are another common cause of pediatric abdominal trauma. Sports-related injuries are associated most commonly with isolated organ injury as a result of a blow to the abdomen. At particular risk are the spleen, kidney, and intestinal tract in children. Finally, significant abdominal injury occurs in only approximately 5% of child abuse cases, but it is the second most common cause of death in these cases, following deaths resulting from head injury.

Principles of Disease

The anatomy of the child lends special protection from some abdominal injury patterns and predisposes the child to other types of injuries in blunt and penetrating abdominal trauma. Children have proportionally larger solid organs, less subcutaneous fat, and less protective abdominal musculature than adults and relatively more solid-organ injury from blunt and penetrating mechanisms. Children have relatively larger kidneys with fetal lobulations that predispose them to renal injury. Children also have a fairly flexible cartilaginous rib cage that allows for significant excursion of the lower chest wall, permitting compression of the internal organs. The combination of these factors provides the basis for the differences in abdominal injury patterns seen between children and adults.

Clinical Features

Pediatric patients with multiple injuries often present with blunt abdominal injury. In children, history is often limited, traditional signs of decompensation seen in adults are often not as evident, and physical examination can be difficult. Subtle, early abdominal findings may be overlooked, leading to significant morbidity and mortality. The history and examination of young children who have sustained trauma is challenging because it may be difficult to know if the child hurts “all over” or has focal findings. The emergency physician may use distraction with toys, lights, bubbles, or keys to get the child’s mind off the examiner and onto the distraction; in this way, areas of tenderness may be located. Child life specialists can assist in distraction techniques, allowing the physician to better concentrate on subtle signs of injury.

Signs and symptoms of abdominal injury in children include tachypnea from impaired diaphragmatic excursion, abdominal

tenderness, ecchymoses, and signs of shock. Lutz and co-workers demonstrated that among restrained children involved in MVCs, those with abdominal bruising were much more likely to have an intra-abdominal injury than those without bruising.⁶⁷ One in every nine patients with ecchymosis was found to have an intra-abdominal injury, some requiring surgery. Abdominal distention is a common nonspecific finding that is often the result of air swallowing subsequent to a painful event. Children with hepatic and splenic injuries may have trouble localizing their pain. Kehr's sign (left shoulder pain with spleen injury) may be the only indication of an intra-abdominal injury. Any abdominal tenderness on examination should prompt further evaluation of the abdomen. Vomiting is usually a late sign or one associated with duodenal hematoma or traumatic pancreatic injury. Signs of small bowel injury may be delayed and noted clinically only with serial examinations. Pelvic bone stability and a rectal examination searching for signs of urethral injury (rare) in boys or blood in the stool (girls and boys) and spinal cord injury (girls and boys) may need to be performed in selected cases of trauma. Rectal examination is insensitive and nonspecific when used as a general screening test for all patients after serious trauma. Cases with suspected injury should receive further evaluation even when the rectal exam is unremarkable.

Even minor falls can result in significant splenic injury but with only minimal findings on examination. Repeated examination, prolonged observation, and close attention to vital signs are warranted. Any child with a clinically suspicious abdominal examination should be evaluated further with additional radiologic and laboratory studies and/or admission for serial examination.

Diagnostic Strategies and Management

In patients with suspected abdominal injury or with mechanisms of possible injury, management and resuscitation must be rapid. Because of fear and pain, children can compound the difficulties in the management of serious penetrating or blunt abdominal trauma. Children tend to distend the stomach greatly with ingested air, which can decrease the diaphragmatic excursion. This can compromise respiratory efforts, and early decompression via NG or OG tube insertion should be considered. Children with a stable pelvis and who are not at risk of urethral trauma should have a urinary catheter inserted to decompress the bladder, evaluate for the presence of urinary retention, and examine for the presence of blood in the urine. The bladder should be decompressed before any invasive evaluation of the abdomen, such as DPL, to prevent accidental laceration during the procedure. A rule of thumb for urinary catheter size in children is 5 F in newborn, 6 F in preschool, 8 F in elementary school, 10 F in middle school, and 12 to 14 F in high school.

DPL and diagnostic peritoneal aspiration are occasionally still useful in modern trauma practice. In the unstable multi-trauma patient with an equivocal FAST exam, the aspiration of 10 cc of blood, fecal, or vegetable matter from the abdomen typically would indicate intraperitoneal hemorrhage and/or bowel injury and the likely need for laparotomy. The DPL has less use in today's practice but is performed by placing 15 cc/kg of NaCl in the peritoneal space and then removing the fluid by gravity. In blunt trauma, a positive DPL is defined as having greater than 100,000 red blood cells/mL or greater than 500 white blood cells/mL, or gram-negative bacteria or vegetative material (stool) seen on microscopy. DPL does not evaluate for retroperitoneal bleeding. The threshold values must be lowered for penetrating trauma. In general, DPLs are not performed on stable patients because CT scanning can be done

quickly and give the clinician far more information, especially about intraparenchymal injury and retroperitoneal injury. CT scanning is not very sensitive for bowel injury and results in significant radiation exposure. This must be kept in mind in decision making and when interpreting test results.

Spleen Injury. Injuries to the spleen are the most common injuries in pediatric abdominal trauma. Children with injuries from MVCs, sudden deceleration injuries, and contact sports-related injuries may sustain splenic trauma. Typical findings include left upper quadrant abdominal pain radiating to the left shoulder. The abdominal examination may show evidence for peritoneal irritation in the left upper quadrant of the abdomen. Patients may be hemodynamically stable or, after significant splenic rupture or laceration, may be persistently hypotensive or in fulminant cardiovascular collapse. Stable patients may undergo CT for radiologic evaluation. Most often, with minor splenic trauma, bleeding is controlled spontaneously without operative intervention; however, all patients with a splenic injury should be evaluated by a surgeon. In cases with a contained splenic subcapsular hematoma, extracapsular bleeding may occur days later. Patients with splenic injury should be admitted to the hospital for close observation and repeated examinations.

Liver Injury. The liver is the second most commonly injured solid organ in the pediatric patient with abdominal trauma. However, it is the most common cause of lethal hemorrhage, with a mortality of 10 to 20% in severe liver injury. Mechanisms of injury causing splenic injury also may cause liver trauma. Tenderness on palpation of the right upper quadrant of the abdomen and the complaint of abdominal pain in this region or in the right shoulder are signs of possible liver injury. Patients managed conservatively often do well; however, patients who are initially treated conservatively but then go on to require delayed laparotomy often have significant morbidity and mortality. Close observation in the hospital, serial abdominal examinations, and serial hemoglobin are recommended.

Renal Injury. The kidney is less susceptible to trauma from forces applied to the anterior abdomen, but it is often injured in the pediatric patient with multiple injuries.⁶⁸ Because this organ is retroperitoneal, signs and symptoms of kidney injury are often less obvious and more diffuse than signs and symptoms of other abdominal organ injuries. Often, dull back pain, ecchymosis in the costovertebral region, and hematuria are the only clues to renal injury.^{69,70} Renal ultrasound and CT may be used in a stable patient to assess the degree of renal involvement. Other organs, such as the pancreas and gastrointestinal tract, are less frequently injured in pediatric patients.

Penetrating Injury. Penetrating wounds to the abdomen usually require rapid evaluation by a surgeon and, in some cases, operative intervention. The role of DPL in the management of pediatric trauma is controversial. DPL provides the most rapid, objective evaluation of possible intraperitoneal injury. Patients who remain unstable despite fluid resuscitation may be candidates for DPL if they are too unstable for CT and there are multiple potential sites of blood loss. An important role for DPL is in the setting of an underlying small bowel injury. In some patients with small bowel injury, CT findings of free fluid may be ascribed improperly to underlying splenic bleeding. Finally, DPL may be considered in the operating room for patients undergoing emergent craniotomy, when adequate evaluation of the abdomen cannot take place because of the urgency required for intervention for head injury.

Radiology. Because pediatric patients suffer more from injury to the spleen, liver, kidneys, and gastrointestinal tract, CT of the abdomen can provide high sensitivity (except with intestinal injury) and specificity for identification of these

injuries while being relatively noninvasive.¹⁰ Recent studies have clarified that oral contrast does not add to the accuracy of CT; thus, one can avoid delays in evaluation, difficulty with administration, and risk for aspiration.

Another useful procedure in an acutely traumatized pediatric patient is bedside abdominal ultrasound. When used by an experienced clinician, ultrasonography can provide sensitive information about intraperitoneal hemorrhage without invasive measures. Although radiologic evaluation can provide important diagnostic information in a pediatric patient with possible abdominal trauma, any patient with unstable vital signs from an obvious surgically correctable cause should receive immediate operative intervention and not be subjected to delay while obtaining radiographic screening studies. Children with persistent or recurrent hypotension, continued abdominal pain, or persistent abdominal distention should have expedient evaluation by a surgeon.

■ DISPOSITION

A key decision for the emergency physician is whether to admit a pediatric trauma patient or transfer the patient to a tertiary care facility. The decision for admission in questionable cases should be based on consultation with the surgeon and the patient's primary care physician. Infants and children who are moderately to severely injured have improved outcomes in a pediatric (specific) ICU versus an adult ICU or an adult ICU with a few segregated pediatric beds. The primary role of the emergency physician is to evaluate and stabilize the patient before admission to a pediatric ICU or before transfer to a tertiary care facility. Before transport, it is vital that the child is appropriately stabilized and that the emergency physician communicates directly with the accepting physician at the accepting facility. Extensive radiologic testing should generally not be completed in a facility that cannot potentially manage that which is being looked for unless the emergency physician is confident that it will not delay the transfer to more definitive care and has discussed the plan with the receiving physician. All radiographs, documents, and results of laboratory testing should be sent with the patient. Parents should be informed of the exact location to which the child is being taken and given a map from the transferring facility to the receiving facility. Under no circumstances should the child's likely outcome be downplayed to the patient's parents before transfer because this leads to false expectations and the assumption of poor care if the outcome promised is not achieved.

Indications for admission are many, but the main criterion is to admit patients requiring ongoing monitoring for deterioration or complications of their injuries. In addition, children with suspected physical injury from child abuse may be admitted for their protection and for medical treatment. The threshold for admission should be very low in cases in which the health care team does not believe the child will have the social support or oversight necessary to be appropriately observed or to recover in the home environment. The family should be asked if they have transportation, a phone, and access to emergency medical service if needed.

Cessation of Care

Despite advances in trauma care and system improvement, some injured children will die—some in the emergency department. Cases in which death is certain or has already clearly occurred, such as with decapitation and findings of livor

mortis or rigor mortis, do not present treatment dilemmas. Those who do present with signs of life (respirations, blood pressure, pulse, pupil reactivity, or cardiac and electrical activity) should have resuscitative efforts initiated. Cardiac echo can be helpful in some cases to confirm the lack of cardiac activity in patients without a pulse who continue to have electrical activity. Patients who lose their vital signs en route to or in the resuscitation room should receive maximum resuscitative efforts, potentially including emergency thoracotomy.

Organ donation should be considered part of the “Omega survey,” which is the final review after death is declared. Signs of maltreatment, congenital abnormality not previously recognized, possible personal identifying marks in unknown cases, and points for future education and study can be gained from one last mental and visual review, as can the potential for some insight related to the cause of death. Organ donation should be offered under local and state guidelines to all families of deceased children. Often, it is viewed as one way parents can make sense of the death of their child. Knowing that their child's death helped many others to live can help parents in their healing.

Parental presence should be considered in all pediatric resuscitation cases. Assigning someone to be with the parent to explain what is happening is essential. Often, the parents' presence can be useful to the resuscitative effort. Information can be obtained immediately when needed from the parents, and they witness the effort that goes into trauma resuscitation. Very rarely, parental presence can present a true hindrance to medical care, in which case the parent can be asked to leave or be escorted out of the room. Parents who witness what the team does for their children during resuscitation seem to better understand the ability and limitations of medicine. In the final analysis, most parents want to be there, and frankly, at the time of death, their presence is more important than the presence of the medical team.

KEY CONCEPTS

- Trauma is the leading cause of death in children in the United States. It accounts for 64% of all deaths in children, totaling 1.5 million injuries and 250,000 hospitalizations annually.
- Proper management of a pediatric trauma patient involves most of the components of standard adult trauma protocols. By paying strict attention to the anatomic and physiologic differences in children, the clinician is assured of the best patient outcomes.
- The leading cause of traumatic shock in children is hypovolemia; management priorities include appropriate ventilation and oxygenation and fluid resuscitation to maintain perfusion.
- The diagnostic test of choice for the evaluation of intra-abdominal injury in a stable patient is CT of the abdomen.
- Controlled hyperventilation should be performed on children with signs of impending brain herniation, and Pco₂ levels should be maintained at 30 to 35 mm Hg to avoid secondary brain injury from excessive hyperventilation and subsequent cerebral vasoconstriction.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.

■ PERSPECTIVE

Background

At the beginning of the 21st century, nearly 13% of the population in the United States was older than 65 years, and by 2040 one in five people will be within this age range.¹ The financial impact of this aging has been tremendous and will continue to accelerate. The first baby boomer became eligible for Social Security on January 1, 2008, and it is estimated that 365 baby boomers will reach retirement age every hour. More than 35% of total health care dollars spent in the late 1990s was for medical care for patients older than 65 years, and undoubtedly this percentage will continue to rise.¹

With regard to trauma, elders (patients older than 65 years) account for only 10 to 14% of all victims, but they consume 25 to 33% of trauma-related health care dollars. Injury is the fifth leading cause of death in this age group² because of a combination of physiologic changes that alter the older patient's response to trauma and injury mechanisms and patterns that are demographically different from those of younger trauma patients. Even among patients older than 65 years, risk stratification exists. Although the risk of death and significant injury is increased in patients 65 to 80 years of age, traumatized patients older than 80 years are four times more likely to die from their trauma than are younger patients.^{3,4} Emergency practitioners must familiarize themselves with these differences among traumatized elder patients as well as understand the differences in resuscitation and stabilization of these patients.

Epidemiology

In younger patients, assault and motor vehicle crashes (MVCs) account for the vast majority of traumatic injuries. In elders, the most common cause of injury is falls,^{4,5} followed by MVCs, pedestrians struck by cars, and assaults.⁴ In general, elder patients are more likely than younger patients to be injured as a result of activities of daily living.

Falls

Falls are the most common mechanism of injury in elders, accounting for 40% of trauma in patients older than 65 years, and they are the leading cause of injury-related death in this patient population.^{4,5} One third of elder patients suffer a significant fall each year, and serious injuries occur in up to one fourth; this rate is increasing rapidly.^{5,6} Risk factors for falls

include medications (sedatives in particular), cognitive and visual impairment, history of stroke, and arthritis. Most falls occur at home and are same-level falls (i.e., falls from the standing position). Because as many as a one fourth of these falls occur as a result of an underlying medical problem,^{7,8} an appropriate medical evaluation is indicated in addition to the trauma assessment and stabilization. Some of the medical causes of falls include strokes, syncope, near-syncope, medications, elder abuse, and hypovolemia (e.g., related to gastrointestinal bleeding, ruptured abdominal aortic aneurysm, sepsis, or dehydration).

The most common injuries sustained in falls are fractures, occurring in 5 to 10% of fall victims.^{5,6,9} Up to 10% of patients may sustain a major injury,⁹ and head injury is the most concerning of these. Many elder patients are on anticoagulants; this makes them more susceptible to significant head injury as a result of a fall. Some studies have shown the incidence of trauma-related abnormalities on head computed tomography (CT) scans in this group of patients to be as high as 16%, with 1 in 50 requiring neurosurgery.¹⁰ The most common abnormal head CT finding is a cerebral contusion, followed by subdural hematoma; epidural hematomas are rarer in elder patients than in younger patients. The greater the height of the fall, the more likely the patient is to have an abnormal CT scan, but serious head injuries may also be seen in patients who suffer a same-level fall. Overall, the peri-injury mortality rate in elders from falls approaches 12%, and up to 50% die within 1 year of the fall,⁹ often related to either recurrent falls or significant medical complications.

Motor Vehicle Crashes

MVCs are the second most common cause of trauma in elders, accounting for 20 to 59% of trauma in this age group.^{4,11} Compared with younger patients, older drivers are more likely to be killed or hospitalized because of an MVC even when they are using seat belts.¹¹ Cognitive impairment, decreased hearing and vision, and slower reaction time are significant risk factors for MVCs in this age group.

Most MVCs involving elders are daytime crashes occurring close to home. These crashes often occur at an intersection and usually involve two cars.¹¹ An elder is twice as likely as a younger person to be in an MVC when making a left-hand turn, and injuries sustained in these broadside crashes tend to be more severe. A single-vehicle crash should raise suspicion of a medical problem that may have caused the crash and requires a careful history, examination, and workup. MVCs in

elders are less likely to involve alcohol, excessive speeds, or reckless driving than those involving younger patients. The overall fatality rate among elder MVC victims is as high as 21%.^{4,11}

Auto versus Pedestrian Incidents

The third most common cause of injury in elders is auto versus pedestrian incidents.⁴ As with MVCs, poor eyesight and hearing, as well as decreased mobility and longer reaction times, make pedestrian elders more likely than younger patients to be hit by a motor vehicle. This mechanism accounts for 9 to 25% of trauma in elders and carries the highest fatality rate, reported to be from 30 to 55%.⁴

■ PRINCIPLES OF DISEASE

Physiology and Pathophysiology

Physiologic changes are inevitable with aging. These changes affect the mechanisms of trauma, types of injuries sustained, response to injury, approach to resuscitation, and prognosis for the injured elder patient.

Compared with younger patients, elders have a more severe injury response to any given trauma mechanism and have a decreased ability to respond to the trauma. In addition, preexisting medical problems may be exacerbated by the trauma, and morbidity and mortality may result from underlying diseases such as cardiovascular or cerebrovascular disease rather than the trauma.

Elders are less likely to be involved in reckless trauma than their younger counterparts. Diminished hearing and sight increase the risk of falls, pedestrian injuries, and traffic collisions. Difficulty with gait and coordination because of impaired sensation and proprioception, muscle weakness, degenerative joint disease (DJD), neuromuscular disorders, and dementia lead to increased risk of falls and affect reaction times in pedestrians and MVCs. Finally, medications have a significant impact on the traumatized elder patient. Medications that may alter mental status (sedatives and antidepressants) make the elder patient more susceptible to traffic collisions and falls. Cardiac medications affect the response to hypovolemia and shock as well as resuscitation. Diuretics may lead to volume contraction before the trauma occurs. Profound hypokalemia secondary to diuresis can also cause weakness and an impaired ability to ambulate or effectively operate foot pedals. Anticoagulants clearly affect the risk of bleeding. Many elder patients are taking these medications; studies show that the most commonly prescribed medications in the elderly are diuretics, cardiac agents, psychotropic drugs, and anticoagulants.^{12,13}

Cardiovascular System

Cardiovascular reserve decreases with age, and because the aging heart cannot easily increase cardiac output (the maximum achievable heart rate decreases with age), elders tend to respond to hypovolemia with increased systemic vascular resistance. In addition, they are less responsive to the increased circulating catecholamine response to shock, making them at risk for earlier decompensation from hypovolemia. Medications such as antihypertensives may affect their ability to increase systemic vascular resistance, and beta-blockers, calcium channel blockers, and digoxin decrease their ability to develop a tachycardic response to shock. Underlying coronary artery disease increases the risk of myocardial ischemia from hypotension and blood loss. Overall, elders are less able to respond to fluctuations of blood pressure and blood volume

that may accompany a traumatic injury than are younger patients, and these changes may exacerbate underlying cerebrovascular and cardiovascular disease.

Pulmonary System

Aging significantly affects the pulmonary system. Reductions in arterial partial pressure of oxygen (PaO_2), forced expiratory volume in 1 second (FEV_1), and vital capacity occur with aging; the lungs become less compliant; and the muscles of respiration weaken. As a result, elders are less tolerant of the volume resuscitation and spinal immobilization that are often needed in resuscitating a trauma victim. In addition, the chest wall is more brittle because of osteoporosis and more rigid because of DJD, making injuries to the chest wall more likely in an elder patient. Combined with the decreased pulmonary reserve in elders, chest wall injuries may quickly lead to respiratory failure in this population of patients. Elders are therefore more likely to require intubation after trauma and are more difficult to wean from respirators after intubation.

Central Nervous System

With aging, the dura mater adheres to the inside of the skull, making epidural hematomas rarer in elders than in younger patients. With age, the brain often atrophies, making it more mobile within the skull during trauma. This atrophy and the resultant stretching of the bridging veins seen in elders make them more susceptible to subdural hematomas than younger patients, even with seemingly minor mechanisms of injury or minimal external evidence of trauma.

Skeletal System

Osteoporosis, a common condition in elders, is a significant risk factor for skeletal injuries such as compression fractures of the thoracic and lumbar spine and other injuries including hip and wrist fractures. These injuries may occur even with relatively minor trauma. Decreased mobility of the joints is also a problem, particularly in the spinal column. This limited mobility increases the risk of spinal injuries, and the locations of these injuries differ in the elder patient. Spinal stenosis, more common in elders than in younger patients, increases the risk of spinal cord damage even in the absence of spinal column injury.

Skin

Skin trauma is common in the elder patient. Aging skin thins and is susceptible to tears and lacerations even with relatively minor trauma. These injuries may be very difficult to repair and often require débridement of devitalized tissue. Prolonged immobilization on a backboard or in a C-collar can result in decubitus ulcers of the back, buttock, or occiput. In addition, elders may be prone to tetanus due to lapses in their active tetanus immunization.

■ SPECIFIC DISORDERS AND INJURIES

Perspective

Multiple trauma in the elder patient is more lethal than in younger patients; a multiply traumatized patient 70 years of age is three times more likely to die than a patient 20 years of age.⁴ The combination of comorbid illnesses, increased propensity to trauma, and decreased physiologic reserve often

leads to exacerbation of underlying medical problems and a higher risk of multiple-system organ failure and death in the elder patient.¹⁴

Spinal Injuries

Physiologic changes with aging predispose the traumatized elder patient to both spinal column and spinal cord injury. DJD leads to decreased spinal mobility and a more brittle spinal column, and osteoporosis makes the bones more likely to fracture. Spinal stenosis often occurs as a result of the aging process; this increases the risk of cord injury even in the absence of a bone injury. Baseline cognitive impairment or acute brain injury may make evaluation of the spine in an elder trauma patient particularly difficult.

The most common mechanism of spinal injury in an older person is a fall. Because of the relative immobility of the cervical spine related to DJD, the most common level of cervical spine injury in elders is C1 to C3,¹⁵⁻¹⁸ a higher level than in younger patients. The most common fracture of the cervical spine in elders is a type 2 odontoid fracture,¹⁵⁻¹⁷ which necessitates adequate visualization of this area of the cervical spine when imaging is performed, often requiring CT scanning with reconstruction. Even if the patient does not suffer a fracture, spinal cord injuries may result from contusion of the cord. Contusion occurs most frequently in hyperextension injuries leading to central cord syndrome (upper extremity greater than lower extremity weakness and sensory loss). Overall mortality from cervical spinal injuries in elders is approximately 14%, triple that of younger patients.¹⁹ Compression fractures of the thoracic and lumbar spine may occur with falls, even those that are seemingly relatively minor, particularly in elder women with osteoporosis. Although spinal cord injuries are relatively rare with these fractures, disability from pain can be significant, and these patients may need admission for adequate pain control. It may also be necessary to differentiate compression fractures from burst fractures, and CT is often helpful in making this distinction.

Head Injuries

Head injuries are the most common cause of mortality directly related to trauma in elder patients. The most common mechanism of significant head injury in elders is falls. Epidural hematomas are rare because of the adherence of the dura mater to the inside of the skull. Cerebral contusions, however, occur in up to one third of head-injured elder patients, and subdural hematomas become more common with age because of the stretching of the fragile bridging veins as the brain atrophies. This atrophied brain is more mobile within the skull, and head trauma may result in shearing of these veins. These patients may present with a broad range of symptoms, from frank coma to a relatively remote history of head trauma and slightly altered mental status.

Mortality from head injury in elders is double that of younger patients, with mortality from subdural hematoma in elders being up to four times higher than in younger patients.²⁰ When patients have minor head injury, elders may eventually recover full function but often need either in-hospital or home rehabilitation before they can return to their baseline function.²¹

Head CT scanning is the diagnostic test of choice for brain injury, and a contrast study may be necessary if the injury is 7 to 20 days old and an isodense subdural hematoma is suspected. Magnetic resonance imaging (MRI), if available, is an alternative imaging modality in these patients when the injury is subacute and an isodense lesion is suspected.

Chest Injuries

Rigidity of the chest wall related to DJD and osteoporosis makes chest wall injuries more common in elders, even with relatively minor trauma. Because of the frailty of the chest in these patients, lap and shoulder belts in automobiles may actually cause injuries, including multiple rib fractures, flail chest, and sternal fractures. Rib fractures are the most common, and because elders have less pulmonary reserve than younger patients, these fractures may lead to respiratory insufficiency. Elder patients more frequently develop respiratory failure from their trauma and are more likely to require mechanical ventilation. In addition, elders may develop atelectasis, pneumonia, and acute respiratory distress syndrome. With proper care (including pain medication), meticulous attention to pulmonary hygiene, and careful hemodynamic management and monitoring, up to 90% of patients with chest injuries may return to normal life after their injuries.

Abdominal Injuries

Depending on the mechanism of injury, up to 30% of elder trauma patients may suffer a significant intra-abdominal injury, but the abdominal examination may be unreliable in these patients. Because mortality from abdominal injuries in elders is four or five times higher than in younger patients, a diligent search for potential intra-abdominal injuries is crucial. Frequently, this requires some combination of focused abdominal sonography in trauma exam (FAST) and CT scanning of the abdomen, depending on the patient's hemodynamic stability and other system injuries.

Extremity Injuries

Because of the increased bone fragility and predisposition to falls with aging, the musculoskeletal system is the most commonly injured organ system in elder trauma patients. By the age of 75 years, 30 to 70% of patients with osteoporosis sustain a fracture.²² Although rarely life threatening, these injuries can severely limit the daily activities of elder patients to the degree that these patients may need admission for pain control as well as to arrange adequate home support or rehabilitation.

Upper extremity fractures are common. Distal radial fractures are the most common upper extremity fractures in elders, accounting for up to 50% of fractures, followed by proximal humeral fractures (30%) and elbow injuries (radial head fractures and elbow dislocations; 15%).

Pelvic fractures are common in elder trauma patients, accounting for 25% of these injuries.²³ Pubic rami fractures, the most common pelvic fractures in this age group, may be seen with same-level falls. Although these injuries tend to be stable, pain control and gait training may necessitate hospitalization. High-velocity injury mechanisms (MVCs or auto versus pedestrian incidents) and falls from heights may result in unstable pelvic fractures, which are associated with a mortality of up to 80% if the fracture is open.²³

Hip fractures are the most frequent lower extremity fractures and the most common cause of admission in elder trauma patients. These injuries are associated with an early mortality rate of 5% and a risk of death of 13 to 30% during the year after the injury (often related to other factors, such as recurrent falls and underlying medical problems).⁵ Plain radiographs are often diagnostic, but CT or MRI scanning may be necessary to discover or further delineate subtle fractures in the elder patient with hip pain after a fall.

Tibial plateau fractures may occur with a fall or MVC and most commonly involve the lateral tibial plateau. Patellar frac-

tures may result from a fall directly onto the kneecap, and sunrise views of the patella may be the only way to visualize these injuries. Ankle fractures account for 25% of all lower extremity fractures and most commonly involve the lateral malleolus; treatment often consists of a walking cast.

Soft Tissue Injuries

Elder patients are susceptible to skin injuries related to the thinning of the skin that occurs with aging. Treatment of these injuries often proves to be difficult, and débridement of devitalized tissue and careful local care are often necessary. Elder patients frequently are not up to date with their tetanus immunizations and because of this are at risk for developing this infection. Treatment with both active and passive immunization is often indicated in this group.

Burns

Burns are particularly devastating in the elder patient. More than 90% of burns occur at home, and because elder patients often live alone and have decreased reaction times, deeper and more extensive burns may occur in this age group. Flame burns account for 50% of all burns in this group and 20% of burn-related deaths. Some of these injuries are cooking related; scalds account for 19% and flammable liquid burns for 10%. Despite the fact that the incidence of burns is lower in elders than in younger patients, mortality from this injury is high. Until the mid-1980s, Baux's formula (risk of mortality = age in years + percentage body surface area burned)²⁴ provided a gross estimate of risk of death from burns. Although advances in burn care during the past two decades have decreased the mortality rate, elders are still at high risk for mortality from burns, with recent data suggesting a mortality rate of approximately 30%.²⁵ Thinning of the skin and decreased immunocompetence contribute to this higher risk of mortality as well as exacerbation of underlying medical conditions that may be precipitated by the stress of an extensive burn injury and its treatment.

CLINICAL FEATURES

Because elder patients may have significant injuries with subtle findings, a thorough examination supplemented by laboratory testing and radiographic studies is often the most prudent approach to even seemingly minor injuries, depending on the mechanism of injury and the presence of comorbid conditions and particularly certain medications (e.g., anticoagulants).

History

A complete history of the events leading to the injury is needed, and out-of-hospital personnel can be invaluable in providing this information. Falls and MVCs, particularly if they involve a single vehicle, should trigger questioning about possible syncope, hypovolemia, cardiovascular or cerebrovascular events, or a complication of medications. The mechanism of injury should be considered and the different patterns of injury in elder patients evaluated (e.g., higher risk for subdural hematoma, high cervical spine injury, and bone injuries).

Physical Examination

In patients with anything more than the most minor of mechanisms of injury, a thorough head-to-toe examination should be

performed. Clothing should be removed in order to allow a complete physical examination. Vital signs may be normal, even in the presence of significant blood loss. Because many elder patients are on antihypertensives, normal blood pressure may in fact signify the presence of hemorrhage. It is important to keep elder trauma patients warm because they are more likely to develop hypothermia when disrobed for examination, and hypothermia increases the risk of mortality related to trauma.

■ DIAGNOSTIC STRATEGIES

Laboratory

Laboratory evaluation should include serial hemoglobin, hematocrit, or both; prothrombin time and partial thromboplastin time and an international normalized ratio; serum electrolytes; rapid and formal glucose measurements; and medication levels if indicated. An electrocardiogram is also useful to evaluate the patient for a precipitating event as well as to assess any cardiac ischemia that may be caused by the trauma and resultant injuries.

Radiology

Radiographic studies should be ordered as indicated by the history and physical examination. Plain films of the cervical spine are often difficult to interpret because of baseline DJD; therefore, CT scans of the neck, with particular attention to the more likely injured higher cervical spine, may be necessary to rule out spinal injury. This is particularly true if clinical findings warrant and plain films are inadequate or demonstrate suspicious areas. Plain films of the thoracic and lumbosacral spine should be obtained in patients with posttraumatic pain in these areas. A chest radiograph may be of particular importance both to evaluate the patient for traumatic injuries and to search for signs of congestive heart failure precipitated by the trauma or resuscitation efforts. Plain films of the pelvis are indicated in patients with suggestive mechanisms of injury or pain on examination of the pelvis. Extremity films should include all areas of concern, and CT or MRI scanning may be necessary to diagnose subtle hip fractures. Adequate radiographic imaging to rule out significant intra-abdominal injury may require FAST, CT scanning, or both, depending in the clinical presentation of the patient.

■ MANAGEMENT

Prehospital Considerations

Because elder patients may have significant injuries even with minor mechanisms of injury, prehospital management is particularly important. Scene assessment is important because prehospital personnel are often the “eyes and ears” for assessing the mechanism of injury, and this information should be solicited from prehospital personnel by emergency department staff. Rapid transport to the hospital is of prime importance. Because elder trauma patients are more likely to suffer significant injuries after even relatively minor events, transportation to a trauma center should be considered for elders who sustain anything more significant than isolated extremity trauma.

Emergency Department

Emergency department assessment of elder patients requires an organized and rapid evaluation for significant injuries and

frequent reassessment to identify deterioration early. Frequent monitoring of vital signs and maintaining a normal core temperature are important in the management of elder trauma patients.

Airway and Breathing

Supplemental oxygen should be administered to all elder trauma patients. Pulmonary insufficiency may develop quickly, and airway management equipment must be readily available. Airway management may be particularly difficult in elders, and potential problems should be anticipated. Cachectic or edentulous patients may be difficult to ventilate with bag, valve, and mask. Decreased mouth opening and limited neck mobility related to DJD may interfere with orotracheal intubation, and preexisting medical problems such as cerebrovascular accidents or renal failure may alter the choice of neuromuscular blocking agents used to facilitate intubation. Dosing of any agent that may affect cardiovascular stability must be carefully considered, and administering lower doses of these drugs is prudent.

Circulation

Fluid and blood resuscitation is particularly challenging in the elder trauma patient. Underlying congestive heart failure may be exacerbated by aggressive circulatory resuscitation, but hypotension and hypovolemia are poorly tolerated, particularly in patients with cardiovascular or cerebrovascular disease. Elder patients who go to the operating room before hemodynamic stabilization have an extremely high mortality rate. The most prudent approach is controlled boluses of warmed isotonic fluids with frequent assessment of physical examination, vital signs, pulse oximetry, and urine output. Hypotension is often an ominous finding and should be corrected, with attention to the potential effects of large fluid volumes on the respiratory system. Normotension in the usually hypertensive patient may be a subtle indication of hemorrhage. Blood transfusion should be strongly considered when the hematocrit drops below 30, and there should be a diligent search for potential sites of blood loss.

Disability

Underlying hearing deficits and residual neurologic deficits from stroke, such as aphasia, motor deficit, or slurred speech, can make assessment of mental status and evaluation for neurologic injury problematic. Information on previous history of deafness or stroke or other neurologic disease should be obtained quickly from the patient or family or both, and an assessment should be made of whether the patient's current condition represents a new or old finding.

DISPOSITION

Typical criteria for admission related to traumatic injuries apply in elders, but other considerations often lower the threshold for hospitalization. If the patient does not have a support system or home situation amenable to careful observation and recovery from even relatively minor injuries, hospital admission may be required. Patients with significant underlying diseases may need admission for monitoring and reassessment until their injuries begin to heal. Often, elder patients may need admission for pain control, particularly those with compression fractures of the spine or pubic rami fractures who may require frequent doses of narcotics for pain control. The use of narcotics can have additional adverse effects for elders that may put them at risk for additional injury, such as postural hypotension or confusion. Chest injuries may be particularly problematic and susceptible to complications, and elder patients with multiple rib fractures (three or more) or one or more displaced rib fractures should be admitted for aggressive pain management and supportive care. In addition, patients with multiple displaced rib fractures may be at risk of delayed death (24–48 hours) from exsanguinations due to severed intercostals vessels. Elders with minor injuries, particularly extremity injuries, may be discharged with appropriate follow-up care and medications.

KEY CONCEPTS

- Elder patients are more susceptible to injuries than younger patients and have a higher mortality rate for any given injury.
- Mechanisms of injury are different in elders than in younger patients. Elder patients are more likely to sustain their injury from a fall, an MVC, or an auto versus pedestrian incident than from an assault.
- Physiologic changes that occur with aging alter the way in which these patients may manifest significant injuries as well as how they tolerate these injuries.
- Emergency physicians must remember that elder trauma patients may have suffered a medical event that precipitated their trauma, or vice versa, and evaluate patients accordingly.
- Resuscitation of elder trauma patients requires oxygen supplementation, a lower threshold for advanced airway control (endotracheal intubation), and aggressive but judicious fluid and blood resuscitation with frequent reevaluation.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.

CHAPTER 37 Injury Prevention and Control

Stephen W. Hargarten and E. Brooke Lerner

PERSPECTIVE

The science of injury control is based on the concept that trauma is like a disease rather than just the consequence of fate or random occurrences. The principles of disease control are applied to injury as they are successfully applied to infectious diseases. Control of a disease as widespread and multifactorial as injury can occur only through broad interdisciplinary effort, including that of medicine, public health, policy makers, law enforcement, educated citizens, and others (Box 37-1).

Emergency medicine plays a pivotal role in the care of injured patients and in injury control. Approximately one third of all emergency department visits are for the care of injuries, representing 14.4 visits per 100 people.¹ Injury is the leading cause of death for many age groups (Table 37-1). In 2004, 29.6 million people were treated in emergency departments for injuries, of which 93% were not admitted to the hospital.² The medical cost of injury-related medical care in the United States is estimated to be \$117 billion annually.² The emergency care system may be the patient's only interface with the health care system. In addition to providing state-of-the-art acute care, emergency physicians should provide state-of-the-art clinical preventive services for these injured patients,³⁻⁵ as well as work with surgeons, pediatricians, and other specialists to decrease injuries through clinical and policy-relevant research and education.⁶

PRINCIPLES OF THE DISEASE OF INJURY

The major causes of injury include falls, car crashes, gunshots, drownings, and poisonings. Similar to other disease models, injury occurs from the interaction of agent and host through a vector and an environment that is conducive to exposure (Fig. 37-1). *Injury* is a harmful event caused by the acute transfer of energy to a patient that results in tissue and/or organ damage.⁷ The energy may be in any form, such as kinetic (e.g., falls and motor vehicle crashes), thermal (e.g., burns and hypothermia), chemical (e.g., poisoning), electrical (e.g., lightning strike), or the absence of energy (e.g., hanging or drowning).⁸ Energy is the agent that is delivered to the host (patient) by a vector in an environment with variable risk. Cars and guns are examples of vectors of energy transmission that cause the injury. A car on an icy road is an example of the environment and the vector interacting to increase the likelihood of kinetic energy reaching the host and causing injury.

The goal of injury control, similar to other forms of disease control, is to prevent or decrease the transfer of energy to the

host by (1) separating the host from the agent through modification of the environment, (2) equipping the host with protection against the agent, or (3) eliminating or modifying the vector that transmits the energy.⁸⁻¹³

The first step in the control of injuries is the recognition that injury is preventable. Common public perception is that injuries are from accidents or random, unexpected events, similar to the way infectious disease was regarded before the discovery of bacteria. Similar to other diseases, characteristics of the host affect prevention strategies, acute care, and rehabilitation outcomes. These include physical characteristics, such as age, gender, size, and motor skills; and mental/behavioral characteristics, such as intelligence, fatigue, alcohol use and abuse, emotional stability, social norms, and lifestyle. Risks for injury and death vary by age (Table 37-1). To decrease the likelihood of an injury, changes in some of these predisposing factors can be made in the host (e.g., through improvement in driving skills or a commitment to wear a seat belt or not to drink and drive) and should be age specific for maximum effect.

Energy is transmitted to the host through a *vector*, such as motorized and nonmotorized vehicles (e.g., car, bicycle, and skateboard), firearms, piercing instruments (e.g., knife and arrow), explosives, and lit cigarettes. Modifying the vector (by elimination or modification of design) and separating the vector from the host are important methods to reduce injury. For example, understanding the biomechanical forces released during an injury event is crucial to understanding vehicle modification.¹⁴⁻¹⁶ This information can assist physicians in educating patients and families to modify or eliminate vectors they encounter during their daily lives—for example, recommending that families properly store and/or use gun locks with household firearms.¹⁷

When an injury occurs, host-agent interaction and energy transfer take place in an *environment*. This environment can be modified to decrease injury. If the environment does not permit energy transmission, the risk for injury, including intentional injuries to the host (patient), is eliminated.¹⁸ In contrast to altering host risk factors, most environmental modifications require no cooperation or action on the part of the host and are more effective when implemented. Examples are implementing safer road design and lighting to prevent motor vehicle crashes, removing throw rugs to prevent falls, placing fences around pools, and separating bicycle paths and sidewalks from the roadway to protect pedestrians and bicyclists from cars.^{12,19}

William Haddon, the first physician administrator for the National Highway Traffic Safety Administration, first described

BOX 37-1**MULTIDISCIPLINARY TEAM APPROACH TO INJURY CONTROL: PREVENTION, ACUTE CARE, AND REHABILITATION****Prevention**

Epidemiology
Biomechanics
Education
Public policy
Law enforcement
Engineering
Outcomes research
Emergency preparedness

Acute Care

Trauma system
Emergency medical services
Emergency department care
Hospital care
Clinical guidelines
Clinical prevention services
Outcomes research

Rehabilitation

Physical therapy
Occupational therapy
Mental health providers

using this type of approach in a landmark article on injury control in 1970.²⁰ He laid out 10 methods for preventing injury (Table 37-2). Haddon later expanded these ideas to incorporate time and developed the Haddon matrix. The matrix recognizes that preventative interventions should be considered for the host, agent, vector, and environment prior to the event, during the event, and after the event. Any type or cluster of injuries can be prevented or reduced in severity using this matrix when the environment, vector, and population most at risk are identified and modified. An example of using this process to reduce car crash injuries is given in Table 37-3. The key to energy transfer reduction or prevention is understanding that (1) injuries are predictable, (2) they follow predictable patterns (e.g., by age or gender), and (3) reliance solely on human factors for prevention has significant limits. A fragile item sent through the mail arrives at its destination intact only if it is properly packaged to reduce the energy we anticipate being placed on the item during transport. Likewise, injuries to people can be prevented by using the cells of the Haddon matrix to mitigate the energy transfer of predictable events.

The History of Injury Control and Emergency Medicine

The avoidance of personal injury is a goal of modern public health, but until the 1940s and 1950s, unintentional injuries were attributed primarily to human error, and prevention was based on educating people to act safely.¹⁰ Unsafe roads were built, and motorized vehicles and other consumer products were manufactured with safety design flaws.²¹ This is analogous to supplying untreated tap water to homes and relying on educating people to purify their drinking water to prevent cholera.¹¹

In the 1920s, attributing vehicle crashes to poor driver performance led to mandatory licensing of drivers. In the 1930s, when it was realized that vehicle crashes were due not simply to human error but to mechanical factors as well, President

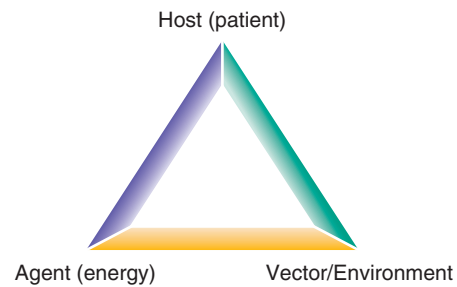
INJURY TRIANGLE

Figure 37-1. The epidemiologic triad can be used as a framework for injury prevention. An injury occurs by the interaction of the host and agent through a vector and an environment that is conducive to injury. Alteration of any of these interactions prevents the injury.¹¹¹

Roosevelt called for automobiles to be made more crashworthy.⁸ In 1942, DeHaven, a former World War I pilot turned physiologist, pondering his own survival in an airplane crash when another occupant had been killed, suggested structural provisions be made to vehicles that would distribute the forces of energy over the human body to attenuate the energy transfer and reduce injuries in crashes. He advocated a focus on defining the physical factors that influence survival rather than the human error that caused the crash.⁸ Physical factors became a focus of interest as the United States embarked on its space program. Air Force researchers showed that people could withstand splashdown with a sled that decelerates from 30 to 0 mph over a stopping distance of 2 feet.²² This demonstration was a significant advance in understanding the biomechanics of sudden deceleration.

The recognition that injuries could be addressed similar to diseases occurred in the 1940s, when Gordon, an epidemiologist, suggested that injuries have epidemic patterns, seasonal variation, long-term trends, and demographic distribution and can be examined with methodologies applied to infectious diseases. Gordon also believed that similar to infectious diseases, injury results from the interaction of the agent, host, and environment.⁸ In the 1960s, Haddon developed a two-dimensional approach to injury analysis by dividing the factors of agent, host, and environment into three phases: preinjury, injury, and postinjury. This phase-factor matrix has become a mainstay of injury control development. Any injury event can be broken down into the component factors, allowing specific interventions to target specific factors (Table 37-4).^{12,23}

In 1985, the publication *Injury in America: A Continuing Public Health Problem* by the National Research Council and the Institute of Medicine called on the public health and health care community to address the injury epidemic.¹³ With the establishment of the National Center for Injury Prevention and Control in the Centers for Disease Control and Prevention (CDC), it was acknowledged that the control of injuries belongs in the disease control community and includes health care providers such as emergency physicians.

Soon after its inception, the National Center for Injury Prevention began sponsoring injury control research centers at academic institutions throughout the country. These centers conduct research in all three core phases of injury control (i.e., prevention, acute care, and rehabilitation), and they serve as training centers for injury control specialists and as information centers for the public.²⁴ In many places, these centers are led by physicians, including emergency physicians. To continue and expand this work, the Society for the Advancement of Violence and Injury Research was created, which is open to

Table 37-1 Ten Leading Causes of Death, United States, 2005, All Races, Both Sexes

RANK	AGE GROUP											ALL AGES
	<1	1-4	5-9	10-14	15-24	25-34	35-44	45-54	55-64	65+		
1	Congenital anomalies 5552	Unintentional injury 1664	Unintentional injury 1072	Unintentional injury 1343	Unintentional injury 15,753	Unintentional injury 13,997	Unintentional injury 16,919	Malignant neoplasms 50,405	Malignant neoplasms 99,240	Heart disease 530,926	Heart disease 652,091	
2	Short gestation 4714	Congenital anomalies 522	Malignant neoplasms 485	Malignant neoplasms 515	Homicide 5466	Suicide 4990	Malignant neoplasms 14,566	Heart disease 38,103	Heart disease 65,208	Malignant neoplasms 388,322	Malignant neoplasms 559,312	
3	SIDS	Malignant neoplasms	Congenital anomalies	Suicide	Suicide	Homicide	Heart disease	Unintentional injury	Chronic low. respiratory disease	Cerebrovascular	Cerebrovascular	
4	Maternal pregnancy comp. 1776	Homicide 377	Homicide 196	Homicide 270	Malignant neoplasms 4212	Malignant neoplasms 4752	Suicide 12,688	Liver disease 18,339	Diabetes mellitus 12,747	Chronic low. respiratory disease 123,881	Chronic low. respiratory disease 143,579	
5	Placenta cord membranes 1110	Heart disease 375	Heart disease 121	Congenital anomalies 200	Heart disease 1717	Heart disease 3601	HIV 4363	Suicide 6991	Unintentional injury 11,301	Alzheimer's disease 112,716	Unintentional injury 130,933	
6	Unintentional injury 1083	Influenza and pneumonia 110	Cerebrovascular 52	Heart disease 146	Congenital anomalies 504	HIV 1318	Homicide 3109	Cerebrovascular 6381	Cerebrovascular 10,853	Influenza and pneumonia 55,453	Diabetes mellitus 75,119	
7	Respiratory distress	Septicemia	Influenza and pneumonia	Chronic low. respiratory disease	Diabetes mellitus	Diabetes mellitus	Liver disease	Diabetes mellitus	Liver disease	Diabetes mellitus	Alzheimer's disease	
8	Bacterial sepsis	Cerebrovascular	Chronic low. respiratory disease	Influenza and pneumonia	Cerebrovascular	Cerebrovascular	Cerebrovascular	HIV	Suicide	Unintentional injury	Influenza and pneumonia	
9	Neonatal hemorrhage	Perinatal period	Benign neoplasms	Septicemia	Complicated pregnancy	Congenital anomalies	Diabetes mellitus	Chronic low. respiratory disease	Nephritis	Nephritis	Nephritis	
10	Necrotizing enterocolitis	Chronic low. respiratory disease	Septicemia	Cerebrovascular	Influenza and pneumonia	Influenza and pneumonia	Influenza and pneumonia	Viral hepatitis	Septicemia	Septicemia	Septicemia	
	546	56	36	43	172	354	934	2314	3912	26,243	34,136	

Data from the National Center for Health Statistics, National Vital Statistics System.

Table 37-2 Haddon's Strategies for Preventing the Transfer of Energy to the Host

TECHNIQUE	CAR CRASH	FALLS
1. Prevent the initial marshaling of energy	Manual task/Breathalyzer ignition interlocks Use of alternative transportation	Remove floor obstacles Prevent unnecessary climbing
2. Reduce the amount of energy marshaled	Speed reduction Vehicle mass restrictions	Climbing height restrictions
3. Prevent the release of energy	Breakaway light poles, roadway obstacle removal	Ambulation aids for elderly Worker safety harnesses
4. Modify the rate of spatial distribution of the release of energy from its source	Autobody crumple zones Safety belts, air bags Water barrel barriers	Land with a "roll" Use of safety nets
5. Separate the energy from the host in space or time	Reduce traffic density Homogeneous traffic flow Increase following distance Sidewalks for pedestrians	Safety zones at edge of raised work areas
6. Separate the energy from the host by barrier	Guardrails, concrete median barriers	Guardrails for scaffolds, raised work areas
7. Modify the surface or structure of impact	Collapsible steering columns, padded pillars and bolsters, safety glass	Padded flooring Helmets and hard hats
8. Strengthen the host receiving the energy	Detect and treat premorbid medical conditions	Prevent/treat osteoporosis and strengthen hip flexion in elderly patients
9. Rapidly detect and evaluate damage and counter its continuation and extension	911 and EMS availability Trauma system planning and implementations, and provision of state-of-the-art emergency care	911 and EMS availability Trauma system planning and implementation, and provision of state-of-the-art emergency care
10. Reparative and rehabilitative measures	Provision of state-of-the-art trauma care, rehabilitation, and aftercare	Provision of state-of-the-art trauma care, rehabilitation, and aftercare

Table 37-3 Typical Haddon Matrix (Constructed for Motor Vehicle Injury)

	HOST (DRIVER)	AGENT/VECTOR (CAR)	ENVIRONMENT
Pre-event (before the crash)	Alcohol use Fatigue Experience and judgment Vision	Brake condition Tire quality Center of gravity Load weight	Visibility of hazards Road curvature and gradient Surface coefficient of friction Shoulder height
Event (during the crash)	Medications Motor skills Cognitive function Age	Speed capacity Visual obstructions Speed at impact Vehicle size	Intersections, access control Weather Signalization Speed limits
Postevent (after the crash)	Age Physical condition Medications Social situation	Load containment Deformation zones Fuel system integrity	911 access EMS response Triage and transfer protocols Nearby level 1 trauma center

Adapted from Baker S, et al: *The Injury Fact Book*, 2nd ed. New York, Oxford University Press, 1992.

any group or individual with an interest in advancing research in violence and injury (<http://www.savirweb.org>).

In 2003, the National Center for Injury Prevention and Control began updating the *CDC Injury Research Agenda* to include acute care research, focusing on research that will improve acute injury care systems.²⁵ Following this, the National Center for Injury Prevention's Division of Injury and Disability Outcomes and Programs changed its name to the Division of Injury Response, whose mission is "to increase the capacity to prevent injuries and their adverse health effects by working with partners to develop, evaluate, and promote evidence-based surveillance, prevention, and care practices."²⁶ This center gives a home for acute injury care and acknowledges its importance for prevention. This division has also become a focus point for response to terrorist-related injuries, an important area of injury control.

Methods of Prevention

The history of injury control began with the first injury. Pain from injury is a powerful stimulus for avoidance behaviors. Early prevention technology is seen in the instruments and garb of the earliest armies, shielding people from the harmful kinetic energy of weapons. Such technology included helmets, shields, and suits of armor. As the delivery of kinetic energy became more sophisticated, prevention technology did not keep pace.^{15,16} Firearms and automobiles represent new plateaus in harnessing kinetic energy as well as creating a new source of morbidity and mortality.^{21,27} The cultural belief was that individuals could avoid vehicular injury by safe driving, and if not, an "accident" occurred. It took more than 50 years to acknowledge that behavior modification alone was insufficient to mitigate high-energy transmission to persons in a haz-

Table 37-4 Ten Leading Causes of Injury Deaths, United States, 2005, All Races, Both Sexes

RANK	<1	AGE GROUP										65+	55-64	45-54	35-44	25-34	15-24	10-14	5-9	1-4	ALL AGES
		1	2	3	4	5	6	7	8	9	10										
1	Unintentional Suffocation	748	Unintentional Suffocation	Unintentional Drowning	Unintentional MV traffic	Unintentional MV traffic	Unintentional MV traffic	Unintentional MV traffic	Unintentional MV traffic	Unintentional MV traffic	Unintentional MV traffic	Unintentional MV traffic	Unintentional MV traffic	Unintentional MV traffic	Unintentional MV traffic	Unintentional MV traffic	Unintentional MV traffic	Unintentional MV traffic	Unintentional MV traffic	Unintentional MV traffic	Unintentional MV traffic
2	Unintentional MV traffic	140	Unintentional MV traffic	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn
3	Homicide	129	Unintentional Fire/burn	Unintentional Drowning	Unintentional Drowning	Unintentional Drowning	Unintentional Drowning	Unintentional Drowning	Unintentional Drowning	Unintentional Drowning	Unintentional Drowning	Unintentional Drowning	Unintentional Drowning	Unintentional Drowning	Unintentional Drowning	Unintentional Drowning	Unintentional Drowning	Unintentional Drowning	Unintentional Drowning	Unintentional Drowning	Unintentional Drowning
4	Homicide	Other, Spec., classifiable	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn
5	Unintentional Drowning	99	Unintentional Drowning	Unintentional Drowning	Unintentional Drowning	Unintentional Drowning	Unintentional Drowning	Unintentional Drowning	Unintentional Drowning	Unintentional Drowning	Unintentional Drowning	Unintentional Drowning	Unintentional Drowning	Unintentional Drowning	Unintentional Drowning	Unintentional Drowning	Unintentional Drowning	Unintentional Drowning	Unintentional Drowning	Unintentional Drowning	Unintentional Drowning
6	Unintentional Fire/burn	64	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn
7	Unintentional Fire/burn	50	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn
8	Unintentional Fire/burn	36	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn
9	Unintentional Fire/burn	30	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn
10	Unintentional Fire/burn	27	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn	Unintentional Fire/burn
		22																			

Data from the National Center for Health Statistics, National Vital Statistics System.

*Unintentional firearm, unintentional struck by or against, and unintentional/unspecified were all tied for 10th place.

ardous environment. Cars were not uniformly equipped with seat belts until the 1960s.²¹ It took another two decades before air bags became standard.^{15,16,28}

Implementation of effective injury control strategies depends on collaborative efforts with physicians, nurses, out-of-hospital care providers, epidemiologists, biomechanical engineers, public policy makers, law enforcement officers, and lawyers.^{19,28,29} A major challenge is the wide diversity of disciplines and interests involved in safety and injury control, many of which are isolated from one another.¹⁰ In the 1990s, important strides were made in community-based injury control programs that rely on coalitions of existing resources with the support of the National Highway Traffic Safety Administration (NHTSA), the CDC, and the State and Territorial Injury Prevention Directors Association. These community coalitions were created to garner existing resources and implement injury countermeasures using “the 3 E’s” of public education, enforcement of laws, and engineering modification of hazardous devices and environmental conditions.¹⁹

■ INJURY CONTROL IN MEDICAL PRACTICE

Traditionally, physicians have focused on treating the patient after the disease has occurred. As the causes of many diseases have become increasingly understood, education about risk assessment and clinically based prevention has been integrated into medical practice, particularly in areas such as infectious (immunizations) and cardiovascular (smoking cessation) disease. Emergency physicians are incorporating risk assessment, counseling, and referral of patients in high-risk groups for injury, such as with domestic violence patients.^{3,5} The Joint Commission for Hospital Accreditation now has emergency department requirements for addressing abused patients.³⁰

Emergency care providers are pivotal in the recording and accumulation of data about the injury event, which is useful for surveillance and epidemiologic analysis. Injury control techniques can be incorporated easily into emergency medicine practice as well.^{5,31-34} A rational approach to improve injury care in a community (Box 37-2) requires that emergency physicians, surgeons, pediatricians, and physiatrists assume specific roles and activities in promoting injury control. This role for

emergency physicians in injury control has been advocated by the American College of Emergency Physicians.³⁵ Documentation of injury information in the medical record, assessing risk factors in individual patients, counseling and referral, provision of systematized acute trauma care, and public health advocacy are all important.³⁶⁻³⁸

Injury Epidemiology and Documentation

Gathering accurate data is essential to understand the characteristics of a disease—its endemic populations, cyclical variations, geographic characteristics, and effectiveness of interventions. Consistent and comprehensive data must be gathered across the population of injured patients to be used for research,^{39,40} hypothesis generation, ongoing monitoring of disease patterns and characteristics,^{34,41} and understanding outcomes of prevention and policy interventions. The goal of injury data collection is to discover who is being injured (host), what is injuring them (vector), and the circumstances surrounding the injury (environment).^{42,43}

Until recently, good data on the disease of injury have been lacking, and knowledge gaps still exist.⁴⁴ Before 1980, the only large civilian databases on injury available for study were mortality data collected by coroners and medical examiners. The Fatal Analysis Reporting System, a comprehensive data set on all car crash deaths in the United States, was established by the NHTSA in 1975⁴⁵ for examining the epidemiology of car crashes.³⁹

Because death results in only 1 in 1000 people who receive medical care for an injury, conclusions based solely on mortality data are limited.^{13,46} The advent of trauma registries in the 1980s increased the sample from patients who die to patients admitted to trauma centers. Because these data are skewed toward the most severe injuries, the conclusions based on these data may have limitations when generalized to the larger population of injured people.⁴⁷

Approximately 93% of injured patients seeking medical care for injury are treated and discharged from the emergency department, many of whom experience significant morbidity resulting in long-term disability and significant cost to society.^{13,34,41,46} In recognition of the importance of emergency department data, the CDC developed Data Elements for Emergency Department Systems to define the minimum data set essential for physicians and information systems developers.^{48,49} The most crucial data element for the understanding of injury is the *E code*, a system for identifying the cause of injury in a patient’s medical record, according to a classification published in the *International Classification of Diseases* (ICD-9-CM).¹⁹ The “E” stands for external cause of injury, such as car crash, fall, or bicycle crash. The cause of injury cannot be extracted from diagnosis codes in medical records. These are “N” codes—the nature of the injury, such as skull fracture, laceration, or contusion. Because injury control depends on identifying the vector that is causing injuries and not just the resultant injuries, the only way to accomplish this systematically is by documenting the appropriate E code for each patient visit. Some states have mandated documentation of E codes for all emergency department discharges.^{34,41-43}

The greatest barrier to the collection of E codes in the emergency department is inadequate physician documentation to assign accurate E codes retrospectively from the medical record.^{46,50,51} The first step in data gathering should be to completely and legibly document the cause of injury in patient medical records. Because injury problems in a community differ greatly from region to region, community-specific injury control efforts can be generated, implemented, and evaluated.^{19,40}

BOX 37-2 INJURY CONTROL IN EMERGENCY MEDICINE PRACTICE

Clinical Preventive Services

- Document injury information in the medical record
- Ensure that medical records of injury cases contain E codes
- Assess behavioral and comorbid risk factors for future injury
- Provide risk screening, counseling, and referral
- Assess biomechanical risk factors in individual patients
- Use biomechanical risk factors for directed evaluation of injured patients
- Provide systematized acute trauma care

Population Health, Research, and Policy

- Participate in and advocate for inclusive trauma systems
- Direct and advocate for rapid, competent emergency medical services response
- Lead efforts in policy development, implementation, and evaluation
- Lead efforts in educating high-risk groups
- Lead efforts to address and modify the environment to reduce risk of injury
- Collaborate in multidisciplinary research to reduce injury risk and to improve care

E-coded hospital records can provide the *who*, *what*, and *when* of injury. The question of *where* can be either place of occurrence or place of residence, both of which are important and have implications for planned countermeasures. Hospital records are helpful in determining place of residence of injured patients, which is useful for community education in high-risk neighborhoods. Location-of-injury data are available only from other sources, such as emergency medical services (EMS), police, or other records. These data are more likely to be useful for environmental modification through engineering enhancements, police enforcement, or hazard removal. Linkage of these records to patient visits, either manually for specific research studies or electronically for surveillance, is the next step in gaining a comprehensive understanding of the epidemiology of injury.^{52,53} With the increasing availability of desktop computer-based geographic information system programs, this powerful tool can be used to study injury locations with minimal training and resources. The maps generated by these programs can be used to identify areas with injury clusters so that prevention efforts can be focused on areas with the greatest need.^{54,55}

Statewide injury data linkages exist in some states and are available for surveillance information⁵⁶ and research for unintentional and intentional injuries.^{32,40,57} For example, the Crash Outcome Data Evaluation System uses probabilistic linkage to create a database of crash, hospital, EMS, and emergency department information for motor vehicle crashes.⁵⁸

Interest and opportunity to apply injury control principles are growing for medical injuries, also known as medical errors. Medical injuries account for an estimated 50,000 to 98,000 deaths each year in the United States, with hundreds of thousands of nonfatal events occurring in emergency departments, intensive care units, and operating rooms.⁴⁴ Emergency physicians can play an important role in using injury control principles and science to reduce medical injuries.⁵⁹⁻⁶² Application of injury control principles for the identification of injury patterns and for the development and evaluation of injury prevention strategies has great potential.^{61,62}

Risk Factor Assessment

Biomechanical Risk Factors

Biomechanical factors responsible for the injury are challenging to understand, occurring in car crashes or gunshots in less than a 10th of a second. Emergency physicians are not trained in engineering principles and have had limited exposure to this “pathophysiology” during training. Ascertaining the forces released on the patient during a blunt (car crash or fall) or penetrating (gunshot or stabbing) injury leads to a directed approach to injury management.⁶³⁻⁶⁶ Extensive research has been done using crash dummies, mathematical models, and computer models to understand the mechanical forces applied in injury and human impact tolerance.^{15,16} Although used extensively by the engineering community for design of products, such knowledge is also valuable to the emergency physician in guiding evaluation and treatment based on energy transfer, tissue tolerance, and risk of occult injury.⁷ Furthermore, as this information becomes more readily available at the time of crash response through automatic crash notification (e.g., General Motor’s On-Star system), it will become more important to understand and correctly interpret what it means in terms of potential injuries.⁶⁷

Injury occurs when energy is delivered to the host in levels that exceed tissue and organ tolerance. This energy can be expressed in G-forces. The G-force that results from a motor vehicle crash, for instance, can be expressed using the following formula:

$$G = \Delta V^2 / (\text{stopping distance} \times k)$$

where ΔV is the change in velocity, *stopping distance* is the distance over which the change in velocity occurs, and k is a constant. G-force is inversely related to stopping distance. To minimize energy transfer to the body during a car crash, one must maximize the stopping distance during the event. The formula shows that doubling the stopping distance reduces the G-force by half, but doubling the speed quadruples the force. Less G-force is applied as velocity is reduced over increasing distance, as the vehicle slows during pre-impact braking or deforms during a crash. The same principle underlies engineering features, such as interior padding, collapsible steering columns, water barrel barriers at bridge abutments, and flexible guardrails. All are designed to increase stopping distance, a major principle of automobile and highway safety engineering.^{15,16,68}

The addition of the air bag to vehicles in the mid-1980s was a significant improvement in safety engineering because it resulted in increased occupant stopping distance during a crash. The NHTSA estimates that as of 2005, approximately 20,000 lives have been saved by air bags.⁶⁹ Initially, these benefits were confined to frontal crashes, but the advent of side curtain air bags and their wide implementation has greatly contributed to the number of lives saved.⁷⁰

As with many new safety countermeasures, there were unintended consequences. First-generation air bags deployed with tremendous force to protect unbelted occupants. These early air bags deployed aggressively at speeds of 140 to 200 mph over 50 msec.⁷¹ Such forces can be lethal to children in the front passenger seat, especially when unrestrained by safety belts, or seated in rear-facing infant seats.^{72,73} A new generation of advanced, less aggressive air bags has been used in the vehicle fleet since the late 1990s, which is expected to reduce injuries associated with air bag deployment. For an emergency physician to estimate risk when assessing a patient from a motor vehicle crash, it is essential to understand the differences in risk posed by seating position, restraint type and use, and vehicle type. This information also must be understood to counsel patients properly on safety belt and child restraint use.^{39,74}

Understanding mechanisms of injury leads to more effective patient counseling to protect patients and their families against injury. Children less than 55 inches tall should only ride in the rear seats of vehicles. Infant seats should never be positioned in the front seat within range of the air bag. Infants 12 months old or younger and weighing 20 pounds or less should always ride in a rear-facing infant seat, and children older than 12 months and weighing more than 20 pounds should ride forward facing in a toddler seat. A booster seat should be used for children weighing 40 to 80 pounds, allowing for better seat belt positioning and discouraging the child from sitting out of position to see out the windows. If circumstances dictate that a smaller child must ride in the front passenger seat, that seat should be positioned as far to the rear as possible and a seat belt should always be worn.⁷⁴ Federal rules allow for air bags to be disabled if there are circumstances necessitating that small children ride in the front seat and for certain medical conditions.⁷⁵ Furthermore, increasingly more vehicles are equipped with sensors that turn off the airbag when certain weight thresholds are not met. Physicians caring for short-stature individuals should counsel these patients about the risk of air bag injuries and recommend they sit with at least 10 inches between the sternum and the steering wheel equipped with an air bag. This distance should be measured objectively because people tend not to estimate this distance correctly.⁷⁶

Other safety features have been associated with specific injuries. Automatic “passive” shoulder belts that require manual fastening of the lap portion may result in “submarining” of the torso toward the floorboard when the lap portion is not fastened, while the shoulder belt squeezes the lower rib cage. Such a mechanism explains the association of these devices with liver, spleen, and lung injuries⁷⁷ and has led to the discontinuation of these devices. However, since cars can be driven over a number of years, it takes many years to completely remove these dangers from the streets. Since automotive safety improves with each new model year, it is important to learn about these safety features and how they might change an injured patient’s presentation.

Knowing the contact surface in falls affects diagnostic and therapeutic interventions because soft surfaces increase stopping distance compared with concrete or packed earth.^{15,16} Understanding the biomechanical risks of other injuries, such as tissue forces from the ballistics of bullet wounds, can guide treatment decisions.^{14,78}

Behavioral and Comorbid Risk Factors

Recognition of patients at high risk of injury affords opportunity for intervention. Counseling a patient about specific ways to avoid injury in the “teachable moment” after injury is more likely to have an effect than diffuse public education.⁷⁹ Family or friends can be recruited to enforce the message to patients or to assist patients in modifying their behavior or environment. Other patient encounters may be used as an opportunity to counsel high-risk patients, such as children at developmental stages that put them at risk for auto versus pedestrian injuries, climbing injuries, or poisoning. It is particularly important to explore the circumstances surrounding injuries to children. A brief review of the injury incident would help physicians and parents identify risks for future injury and opportunities for intervention.⁸⁰ Children who come to the emergency department for an injury are likely to be injured again, commonly during falls and motor vehicle crashes.⁸¹ Preschool-age children admitted to the hospital for injury were twice as likely as community controls to have been treated previously in the emergency department for injury and more likely to have been in the emergency department more than once.⁸²

Risk factors for intentional injury are complex and involve behavioral, social, and environmental factors, but risk factors for all types of injury include male, low income, illicit drug involvement, previous arrest, and young age.⁸²⁻⁸⁴ In studies using psychosocial inventories, recidivists are more likely to have a low sense of autonomy, to have low levels of spirituality, and to have been a victim of crime in the past.⁸² As a practical matter in the emergency department, the most obvious risk factor for future violent injury is prior violent injury. A history of prior significant injury is a strong predictor of injury recidivism, with 10 times the risk of patients with no prior trauma.^{84,85} Emergency departments should have protocols in place for the detection and referral of patients likely to be victims of injury in the future, including victims of domestic violence,³ and for children younger than 18 years injured intentionally, regardless of the age of the perpetrator. In many states, there are mandatory reporting laws for gunshot wounds, stabbing, and other violent acts.^{86,87} Interventions provided to injured patients in the health care setting can reduce injury recidivism.^{83,88}

In the case of motor vehicle injury, the three behavioral risk factors most likely to result in future injury are speeding, seat belt nonuse, and driving after drinking alcohol. Giving patients the necessary data for them to make an accurate self-assessment about their risks is the essence of patient behav-

ioral intervention.⁸⁹ These messages should be part of every injury patient’s encounter, when feasible. Particularly important in this context is the screening and referral for alcohol use disorders (AUDs). Alcohol-related crash injury is a national epidemic in the United States, claiming more than 17,000 lives annually and injuring an estimated 870,000 people.⁹⁰ Reductions in alcohol-related deaths due to more stringent laws to curb impaired driving, more vigilant public education, and a societal shift toward the condemnation of driving while impaired have not had significant effects on people with AUD. Of patients seen in the emergency department after a motor vehicle crash, 17 to 20% meet criteria for AUD.^{91,92} Patients with AUD have higher rates of illness and motor vehicle crash injury than the rest of the population, and patients with AUD are more likely to drive after drinking.⁸⁷

Emergency physicians have a unique role to play in the identification of high-risk patients. In particular, patients with AUD should be detected and referred for formal evaluation and treatment. A structured approach to detect and treat the disease must be brief and effective if it is to be used in a busy emergency department. Screening techniques validated in the emergency department and methods of brief intervention have been thoroughly described.⁸⁷

Successfully treating AUD leads to reduction in alcohol consumption and, consequently, fewer impaired driving episodes, thus leading to a reduction in alcohol-related crash injuries. Evidence suggests that being treated for injury in the emergency department may be an important motivational opportunity to reduce drinking and presents a “teachable moment.”^{87,92-94} The American College of Surgeons’ Committee on Trauma recommends that all trauma centers screen for alcohol and provide interventions as a “part of routine trauma care.”⁹⁵

Motor vehicle crashes are the leading cause of death for children older than 24 months in the United States (Table 37-4; see also Table 37-1). The risk of death in a motor vehicle crash can be reduced by half with the use of age-appropriate child restraints. Emergency physicians should understand the various restraint types and recommendations for their use based on age, weight, and height. Every pediatric visit to the emergency department involves transportation to and from the emergency department and is an opportunity to counsel parents on the safe transport of their children.

Acute Care

The acute care component of injury control involves trauma system planning, medical direction of out-of-hospital care, and providing systematized resuscitative care after the injury, whether it occurs close to or far from a trauma center.⁹⁶ A crucial part of injury assessment is identification of local resources for management of the injured patient. Algorithms and agreements to transfer the patient to definitive care should be established to avoid secondary injury from delays in transfer or inappropriate care.⁹⁷ Likewise, an environment with ready availability of trauma physician specialists should have clear protocols in place for use of those resources.⁹⁸ Cost-effective mobilization of injury care resources dictates that in-hospital triage criteria be developed for the care of the injured patient to avoid unneeded overuse of trauma surgery teams.^{38,99,100}

Trauma systems can be created that recognize and complement the exigencies of budgetary, geographic, and political constraints that are specific to states or regions. Such flexibility is often impossible when a trauma system is based only on the locations of hospitals that seek trauma center verification or designation.⁹⁹

An *inclusive* trauma care system is one that comprises all acute care and rehabilitation facilities that treat injured patients

and deals with the issues of community access, EMS dispatch and response, triage, transport and transfer protocols, training, communications, availability of definitive care and rehabilitation, and a data collection system. In an inclusive trauma care system, every injured patient (not just patients who live near trauma centers) is cared for by a part of the system. Every hospital has a role in an inclusive trauma system according to the services it is capable of offering, whether it is the expeditious transfer of patients, the treatment of patients without neurotrauma, or the definitive care provided at a trauma center. The system should be designed to monitor patient outcomes and system performance.¹⁰¹ The finding that level 1 trauma centers improve severe trauma patient outcomes by 25% is further evidence of the importance of an inclusive trauma care system.¹⁰²

Out-of-hospital emergency care is an integral part of injury control.³⁷ EMS response, triage, and treatment are the first critical steps in injury control after an injury event has occurred. Triage protocols must be well established to avoid unnecessary delays in definitive care.⁹⁷ EMS providers have a unique vantage point to help the trauma physician assess a patient's risk factors for immediate injuries and the risk of injury recurrence. EMS providers can observe the environment for information about mechanism of injury. Accurately reporting vehicle damage and other environmental circumstances associated with the injury event elucidates important biomechanical risk factors.¹⁰³ EMS providers have also become more involved in primary injury prevention through injury risk identification, documentation of injury data, and safety education programs.³⁷

Emergency Medicine Leadership: Advocacy of Public Policy

Passing and enforcing laws are more effective than education in effecting individual behavior change for increasing safety actions such as seat belt and helmet usage.^{8,28} Emergency physicians and other trauma physicians are well positioned to provide lawmakers with factual information coupled with the perspective of firsthand experience of the effects of injury. Effective prevention interventions and policies with documented cost savings are more likely to occur when sound, scientific studies are made available to policymakers.¹⁰⁴ Most public health regulations and traffic safety laws are under the jurisdiction of state legislatures and city and county governments. These policymakers are generally much more accessible to physicians and in need of local expertise than are policymakers at the federal level. Emergency physicians need

to accept an important advocacy role for reducing injuries and incorporate injury control as a professional activity.^{6,105,106}

Community education aimed at people not yet injured may be effective when provided by an emergency physician. Emergency physicians are in a leadership role to deliver the message to school systems, the local housing authority, law enforcement, community service organizations, and policymakers.^{32,33} Trauma physicians can be effective spokespersons for injury prevention through the news media, especially after a newsworthy injury event, and can reframe the event from one of personal blame and behavior failure to a broader biosocial issue that requires environmental and policy interventions.¹⁰⁷

Public policy also determines where resources are used in a community. Environmental modifications and elimination of hazards are effective but often expensive. In contrast to education and law enforcement, environmental modifications are *passive countermeasures* that do not require any action by people. Such modifications might be lengthening a “walk” signal at a busy intersection to reduce auto–pedestrian injuries, especially in the elderly¹⁰⁸; increasing lighting in areas where personal assaults occur¹⁸; or changing a playground surface from hard-packed earth to wood mulch.¹⁰⁹ The need for such modifications may be known only if the physician is alert to the circumstances by asking “How did this happen?” and documenting the location and circumstances of injuries seen in daily practice.

KEY CONCEPTS

- Injury is the second most costly disease to society and the most serious disease of young people.¹¹⁰
- Through interdisciplinary research, a better understanding of the epidemiology and biomechanics of injury will lead to new control strategies. These strategies would complement advances in acute care and trauma systems, which improve care to the patient after the injury occurs.
- Emergency physicians can incorporate injury control techniques into daily practice through clinical prevention services.
- Increasingly, emergency physicians are leaders in addressing and preventing injuries and complex biosocial problems.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.

CHAPTER 38 Head Injury

Michelle H. Biros and William G. Heegaard

■ PERSPECTIVE

Epidemiology

The devastating consequences of head injury have been recorded since ancient times. Early neurosurgical records were primarily observational, with very few suggestions for treatment (Fig. 38-1). Despite centuries of investigation and the development of new and better intensive care, we still have not discovered effective therapies that can be applied after the injury to reverse most pathologic aspects of traumatic brain injury (TBI).

Each year, an estimated 1.5 million people sustain head injury in the United States. Of these, 1.1 million undergo emergency evaluation, including approximately 500,000 children younger than age 14 years.¹ Overall, 80% sustain minor head trauma (Glasgow Coma Scale [GCS] score = 14–15), 10% have moderate head injuries (GCS = 9–13), and 10% have severe head injuries (GCS = ≤8). Almost 20% of all head-injured patients are hospitalized, and approximately 52,000 patients die each year from TBI.

The leading causes of head injury in the civilian population are falls (28%) and motor vehicle collisions. Traumatic brain injury due to blasts has been called the signature injury of the war in Iraq and Afghanistan: TBI of any severity is estimated to affect as many as 10 to 20% of war-time service members.² Head injury is the leading cause of traumatic death in patients younger than 25 years and accounts for nearly one third of all trauma deaths.³ Head injury from child abuse is common and estimated to represent up to two thirds of cases in the 0- to 4-year-old age group.⁴ The Centers for Disease Control and Prevention estimates that there are at least 5.3 million Americans currently suffering from some degree of disability due to TBI. As veterans return to the United States, the numbers of patients suffering from the consequences of TBI are projected to increase markedly.

These facts confirm that TBI is a major public health problem. The emergency physician sees patients with head injuries of different clinical severity caused by a variety of mechanisms. External physical signs of head trauma only confirm that injury has occurred; they are not always present in the patient who has sustained serious underlying TBI. The ultimate survival and neurologic outcome of the head trauma patient depend on the extent of TBI occurring at the time of injury, alone or in combination with secondary systemic insults such as hypotension and hypoxia, which worsen the resulting neurochemical and neuroanatomic pathophysiology. Research aimed at reducing or preventing the neurological consequences

of head trauma is ongoing, but currently the clinical outcome following TBI depends on the circumstances of injury and early clinical management aimed at reducing the occurrence of secondary brain insults. No effective intervention has been found to reverse the pathologic events initiated by the traumatic event.

■ PRINCIPLES OF DISEASE

Anatomy and Physiology

Scalp and Cranium

The scalp consists of five tissue layers. The dermis is the outermost layer and is among the thickest layers of skin on the body. The underlying subcutaneous tissue contains the hair follicles and the rich blood supply of the scalp. The large blood vessels of the scalp do not fully constrict if they are lacerated and can be the source of significant blood loss. The middle scalp layer is the galea, which is made of tough fascial tissue. It contains the occipitofrontalis and temporoparietalis muscles, which move the scalp backward and forward, elevate the eyebrows, and wrinkle the forehead. Under the galea is a loose areolar tissue layer. Because the areolar attachments to the rest of the scalp are loose, scalp avulsions frequently occur through this layer. This is also the site for development of subgaleal hematomas, which can become quite large because blood easily dissects through the loose areolar tissue. The deepest layer of the scalp, the pericranium, is firmly adhered to the skull.

The skull comprises the frontal, ethmoid, sphenoid, and occipital bones and two parietal and two temporal bones. The unique layered architecture of the bones of the skull enhances its strength. Each bone consists of solid inner and outer layers, separated by a layer of cancellous bone tissue (the diploe). In adults, the bones of the skull average between 2 and 6 mm in thickness; the bones in the temporal region are usually the thinnest of the skull.⁵ The cranial bones form a smooth outer surface of the skull, but within the cranial vault are many bone protrusions and ridges. Contrecoup injuries and contusions far from the site of head impact often occur as the accelerating brain strikes against these uneven bone surfaces.

The inner aspect of the skull is lined with the periosteal dura, which is a thick connective tissue layer that adheres closely to the bone surface. The inner meningeal layer of the dura is the outermost covering of the brain. This dural membrane reflects back on itself to make folds within the cranial



Figure 38-1. Title page of a 16th-century neurosurgical textbook. (From Fringer S: *Origins of Science*. New York, Oxford University Press, 2001, p 422.)

space. These folds serve to protect and compartmentalize different components of the brain. The midline falx cerebri separates the two cerebral hemispheres from each other. The tentorium cerebelli partitions the cerebellum and brainstem from the cerebral hemispheres. The U-shaped free margin of this dural fold is important in the pathology of the transtentorial herniation syndromes that can complicate severe head injury. Within the margins of the dural reflections, the two dural layers separate to form large dural venous sinuses. Injury to the dural sinuses is associated with significant morbidity and mortality because of the potential for uncontrolled hemorrhage and the difficulty in repairing these structures.

The cranial vault is rigid and nonexpandable, with an average volume in adults of approximately 1900 mL.⁶ Cranial contents exit or enter the skull through many foramina. The largest, the foramen magnum, is the site of exit of the brainstem and spinal cord from the cranium.

Brain and Cerebrospinal Fluid

The brain is a semisolid structure, which weighs approximately 1400 g (3 pounds) and occupies approximately 80% of the cranial vault.⁶ It is covered by three distinct membranes: the meningeal dura, the arachnoid layer, and the pia. The location of traumatic hematomas relative to these membranes defines the pathologic condition and determines the consequences of the injury.

The major divisions of the brain are the cerebrum, cerebellum, and brainstem. Each lobe of the cerebrum is the source of highly specific neurobehavior, and specific injury to each lobe can disrupt normal behavior patterns. The brain is suspended in the cerebrospinal fluid (CSF), which provides some buffering for the brain during trauma. CSF is produced by the choroid plexus, located primarily in the lateral ventricles of the brain. CSF passes from the ventricular system into the sub-

arachnoid space that surrounds the brain and spinal cord. CSF provides a fluid pathway for delivery of substances to brain cells, elimination of the products of brain metabolism, and transport of peptide hormones and hormone-stimulating proteins from their site of production within the central nervous system (CNS) to their peripheral sites of action.

The normal pressure exerted by the CSF is 65 to 195 mm H₂O or 5 to 15 mm Hg. Blood within the ventricles can obstruct the flow of CSF, causing a traumatic hydrocephalus. Brain injury and its complications can also alter the pH of CSF. Because the pH of CSF influences pulmonary drive and cerebral blood flow (CBF), any alteration can produce detrimental neurophysiologic consequences.⁶

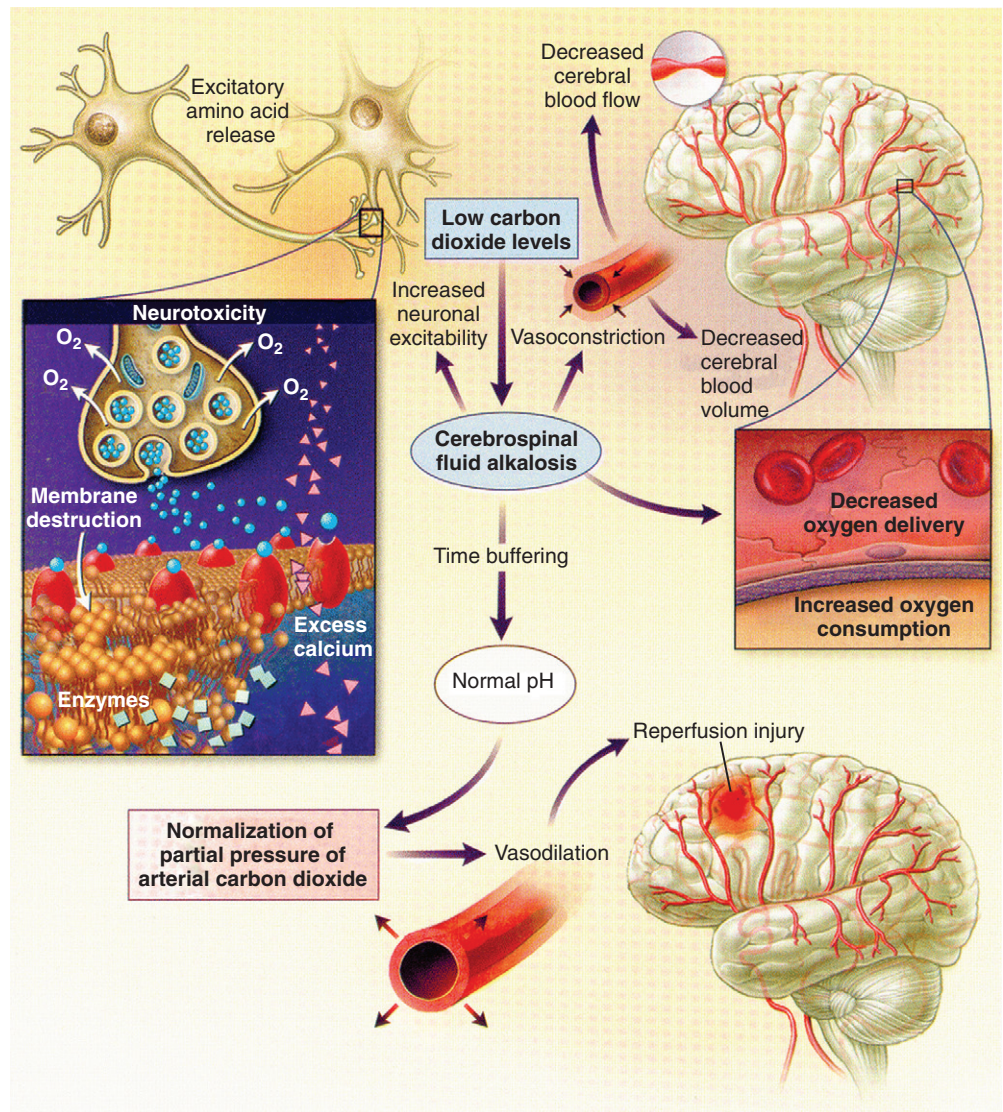
Cerebral Hemodynamics

Blood-Brain Barrier. The blood-brain barrier (BBB) maintains the microenvironment of the brain tissue. Extracellular ion and neurotransmitter concentrations are regulated by movement across this barrier. When the BBB is intact, the ability of neuroactive drugs to penetrate into the brain tissue usually depends on their lipid solubility. Post-traumatic cerebral edema and possibly the biomechanics of the injury itself can cause a prolonged disruption of the BBB for up to several hours after trauma.^{7,8} Prolonged disruption of the BBB contributes to the development of post-traumatic vasogenic cerebral edema.

The brain has an extremely high metabolic rate, using approximately 20% of the entire oxygen volume consumed by the body. To provide for its high metabolic demands, the brain requires approximately 15% of the total cardiac output. Optimal regional CBF is maintained by the ability of cerebral vessels to alter their diameter in response to changing physiologic conditions.⁸ Hypertension, alkalosis, and hypocarbia promote cerebral vasoconstriction; hypotension, acidosis, and hypercarbia cause cerebral vasodilation. In the normal brain, CBF is maintained at constant levels with a mean arterial pressure (MAP) of 60 to 150 mm Hg. This is referred to as *autoregulation*. Outside this range, the CBF varies linearly with MAP.

Cerebral vasoactivity is very sensitive to changes in systemic carbon dioxide and oxygen partial pressures (Pco₂ and Po₂, respectively). The response to changes in Pco₂ is nearly linear between Pco₂ values of 20 and 60 mm Hg.⁹ In this range, lowering Pco₂ by as little as 1 mm Hg decreases the diameter of cerebral vessels by 2 or 3%, which corresponds to an overall change in CBF of 1.1 mL per 100 g of tissue per minute. The physiologic response of blood vessels to Pco₂ is the rationale for the acute use of brief hyperventilation to control increased intracranial pressure (ICP) after head injury. As Pco₂ decreases with hyperventilation, cerebral vasoconstriction occurs. As a result, the volume of blood per unit area of brain tissue decreases. This decrease (even if small) may buffer the effects of increasing edema or an expanding hematoma within the rigid cranial vault. The vasoconstriction produced by extreme changes in Pco₂ (20 mm Hg or less) can be so pronounced that some areas of brain experience ischemia; subsequently, tissue hypoxia can occur.^{8,10,11} Therefore, hyperventilation must be controlled and monitored, with a goal of maintaining the Pco₂ between 30 and 35 mm Hg,^{10,11} and reserved for patients who are showing signs of acute herniation. Over 12 to 24 hours, injured vessels may lose their responsiveness to hyperventilation-induced hypocarbia and become vasodilated. Blood may then be shunted to the injured area, resulting in increased brain swelling and mass effect. Prolonged (i.e., beyond the acute resuscitation) or prophylactic hyperventilation is therefore not recommended as a treatment for increased ICP, and hyperventilation is not used for the routine management of head-injured patients with no signs of increased ICP.⁹⁻¹¹ The neurologic effects of hypocapnia are illustrated in Figure 38-2.

Figure 38-2. Neurologic effects of hypocapnia. Systemic hypocapnia results in cerebrospinal fluid alkalosis, which decreases cerebral blood flow, cerebral oxygen delivery, and, to a lesser extent, cerebral blood volume. The reduction in intracranial pressure may be lifesaving in patients in whom the pressure is severely elevated. However, hypocapnia-induced brain ischemia may occur because of vasoconstriction (impairing cerebral perfusion), reduced oxygen release from hemoglobin, and increased neuronal excitability, with the possible release of excitotoxins such as glutamate. Over time, cerebrospinal fluid pH and, hence, cerebral blood flow gradually return to normal. Subsequent normalization of the partial pressure of arterial carbon dioxide can then result in cerebral hyperemia, causing reperfusion injury to previously ischemic brain regions. (From Laffey JG, Kavanagh BP: Hypocapnia. *N Engl J Med* 347:43, 2002.)



The cerebral vessels also respond to changes in P_{O_2} . As P_{O_2} declines, cerebral vessels dilate to ensure adequate oxygen delivery to brain tissue. When brain injury has occurred, increased CBF in the presence of a disrupted BBB can promote the formation of vasogenic edema. Avoiding or reversing hypoxia is therefore an essential goal in the acute management of the head-injured patient.¹² The responses of the cerebral vasculature to changing physiologic conditions protect the brain by increasing the delivery of oxygen to tissue, enhancing the removal of metabolic end products and allowing nearly instantaneous adjustments of regional blood flow to meet the changing metabolic demands.

Cerebral Perfusion Pressure. CBF also depends on cerebral perfusion pressure (CPP), which is the pressure gradient across the brain. The determinants of CPP are MAP and the resistance to CBF produced by mean systemic venous pressure and ICP. Because ICP is higher than mean systemic venous pressure, ICP effects predominate. Therefore, CPP is estimated as MAP minus ICP. CBF remains constant when CPP is 50 to 160 mm Hg. If CPP falls below 40 mm Hg, the autoregulation of CBF is lost, CBF declines, and the resultant tissue ischemia critically affects cerebral metabolism.^{8,10} It is essential to avoid or correct hypotension in the patient with multiple trauma who is also head injured so that the CPP can be maintained. Management must also be directed at reducing or preventing

increased ICP to ensure adequate CPP to sustain cerebral metabolic needs.

Biomechanics of Head Trauma

Direct Injury. Direct impact head injury occurs when the head is struck by an object or its motion is arrested by another object. The resulting damage depends on the consistency, mass, surface area, and velocity of the object striking the head. Direct injury can also be caused by compression of the head. External signs of trauma are frequently noted at the site of application of the impact or compression force. The skull initially bends inward at the point of contact. If the force is sufficient, a skull fracture can occur. The cranium absorbs some of the applied energy, and some energy is transmitted to the brain by shock waves that travel distant to the site of impact or compression. These shock waves distort and disrupt intracranial contents and temporarily alter regional ICP as they propagate. In general, the more rapidly a force is applied, the greater the damage it causes. The extent of direct injury depends on the viscoelastic properties of the underlying region of brain tissue, the duration of the force applied, the magnitude of the force reaching the brain tissue, and the surface area of the brain that is affected by the application of the force. In cases of penetrating trauma, the mass, shape, direction, and speed of the penetrating object also affect the extent of direct injury.

Direct injury from compression of the head requires significant force because the architecture of the skull provides substantial resistance to deformation. In the clinical setting, compression injury is less common than other types of direct impact. With sufficient and prolonged application of compression force, the ability of the skull to absorb the force is overcome, and multiple linear skull fractures occur. Resulting fractures can be depressed if a high-energy rapid compression force is applied to a small area of the skull. Isolated direct impact injury is rare; direct impact usually sets the head in motion, resulting in simultaneous direct and indirect injury.

Indirect Injury. In indirect brain injury, the cranial contents are set into motion by forces other than the direct contact of the skull with another object. A common example is acceleration–deceleration injury, such as the shaken impact syndrome.¹³ No direct mechanical impact is sustained, but the cranial contents are set into vigorous motion. The brain moves within the skull, and bridging subdural vessels are strained. Subdural hematomas may result. Differential acceleration of the cranial contents occurs, depending on the physical characteristics of the brain region. As one brain region slides past another, shear and strain injuries are produced. This movement results in diffuse injuries, such as diffuse axonal injury or concussion. Additional injury occurs as the movement of the intracranial contents is abruptly arrested and the brain strikes the skull or a dural structure. Contrecoup contusions are an example of the injury produced in this manner. In penetrating injury, the traversal of the object produces pressure waves that can strike structures distal to the path of the missile.

Brain Cellular Damage and Death

Primary and Secondary Brain Injuries

The acute clinical picture of the patient with TBI is dynamic and represents the sum of primary and secondary injury. Primary brain injury is mechanical irreversible damage that occurs at the time of head trauma and includes brain lacerations, hemorrhages, contusions, and tissue avulsions. On the microscopic level, primary injury causes permanent mechanical cellular disruption and microvascular injury.¹⁴ No specific intervention exists to repair or reverse primary brain injury; the only way to decrease brain injury is through public health interventions aimed at reducing the occurrence of head trauma.

The circumstances and extent of the primary injury are not the only contributors to the final neurologic outcome after head injury. The traumatic event also produces injury at the functional and anatomic cellular level, which begins soon after the impact and continues for several hours and even days after injury. Secondary brain injury results from intracellular and extracellular derangements that are probably initiated at the time of trauma by a massive depolarization of brain cells and subsequent ionic shifts.¹⁴ Animal studies have revealed a complicated series of neurochemical, neuroanatomic, and neurophysiologic reactions after head injury (Fig. 38-3). The cell has some compensatory mechanisms to protect itself from widespread damage, such as endogenous free radical scavengers and antioxidants. With significant trauma, however, these systems are quickly overwhelmed, and the functional and structural integrity of the cell is threatened. Human studies document similar changes. Studies suggest that abnormal genetic responses may play a role in response to injury, such as prompting apoptotic cell death.¹⁵ The relative importance and contribution of each adverse reaction to the final functional status of the damaged cell are uncertain, as are the rate and duration of each detrimental event. All currently used

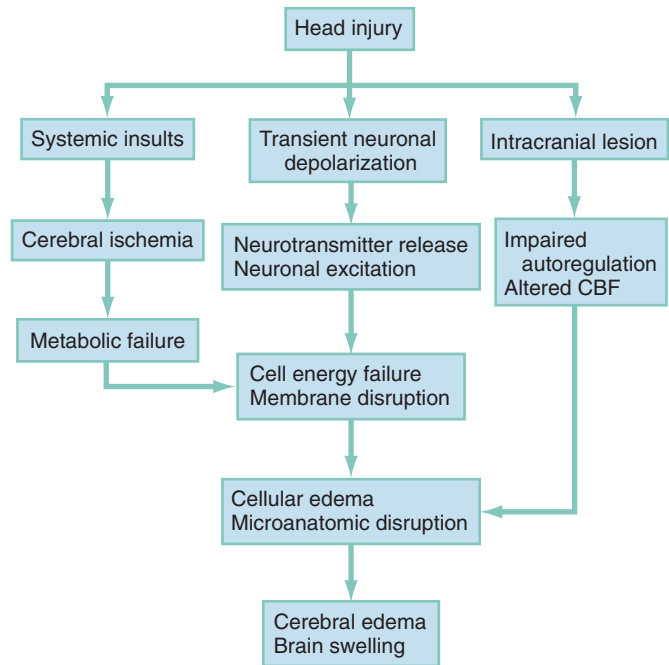


Figure 38-3. Contributing events in the pathophysiology of secondary brain injury. CBF, cerebral blood flow.

acute therapies for TBI are directed at reversing or preventing secondary injury. Experimental evidence for many investigational agents aimed at specific steps in the destructive processes suggests that some aspects of secondary brain injury may be reversed or modified. Multiple ongoing head injury trials have been performed with numerous investigational therapeutic interventions; to date, none have proved useful in the clinical setting.^{15,16}

Secondary Systemic Insults

The final neurologic outcome after head trauma is influenced by the extent and degree of secondary brain injury. In turn, the amount of secondary brain injury depends on certain pre-morbid and comorbid conditions, such as the age of the patient and trauma-related systemic events.^{17,18} A primary goal in the emergency care of the head-injured patient is prevention or reduction of systemic conditions that are known to worsen outcome after TBI.

Common secondary systemic insults in trauma patients include hypotension, hypoxia, and anemia and hyperpyrexia. **Hypotension**, defined as a systolic blood pressure less than 90 mm Hg, has been found to have negative impact on severe head injury outcome.¹⁹ Systemic hypotension reduces cerebral perfusion, thereby potentiating ischemia and infarction. The presence of hypotension nearly doubles the mortality from head injury and worsens the outcome of the patients who survive.^{17,19}

Hyperpyrexia (core body temperature >38.5 °C) is also correlated with worsened outcomes after TBI, and both its magnitude and its duration seem to contribute. The exact mechanism by which it causes damage is yet to be determined but likely involves stimulation of metabolism in injured areas of the brain, thus recruiting blood flow with a resultant increase in ICP.¹⁵

Hypoxia, defined as a P_{O_2} less than 60 mm Hg, probably occurs often in the head-injured patient. Causes include (1) transient or prolonged apnea caused by brainstem compression

or injury after the traumatic event; (2) partial airway obstruction caused by blood, vomitus, or other debris in the airway of the traumatized patient; (3) injury to the chest wall that interferes with normal respiratory excursion; (4) pulmonary injury that reduces effective oxygenation; and (5) ineffective airway management, such as the inability to bag-valve-mask or intubate the patient in an effective or timely manner, respectively. The exact incidence of hypoxia in the head-injured patient is difficult to estimate because it is often unnoticed or undocumented in the out-of-hospital setting. When its occurrence is documented, the overall mortality from severe head injury may double or quadruple.^{19,20} Increased recognition of the potentially devastating consequences of hypoxia has led to more vigilance in the out-of-hospital and emergency setting.

Anemia caused by blood loss can be detrimental to the head-injured patient by reducing the oxygen-carrying capacity of the blood, thus reducing the amount of necessary substrate delivered to the injured brain tissue. When anemia (hematocrit <30%) occurs in patients with severe head injury, the mortality rate increases.¹⁵ Other potential reversible causes of systemic insult in head injury include hypercarbia, hyperthermia, coagulopathy, and seizures.

Pathophysiology

Increased Intracranial Pressure

ICP represents a balance of the pressures exerted by the contents of the cranial cavity. This relationship is explained by the Monro-Kellie doctrine.^{21,22} Because the craniospinal intradural space is almost nonexpandable, the sum of the volume of brain, CSF, and blood within the cranium must remain constant. If the volume of any of these components increases, the volume of another must decrease to maintain a constant ICP. Increased ICP is defined as CSF pressure greater than 15 mm Hg (or 195 mm H₂O) and is a frequent consequence of severe head injury. Initially, as ICP increases as a result of a traumatic mass lesion or edema formation, the CSF is displaced from the cranial vault to the spinal canal, offsetting the increased blood or brain volume. When this compensatory mechanism is overwhelmed, the elastic properties of the brain substance allow tissue compression to provide buffering for the increasing pressure. Depending on the location and the rate of expansion of the traumatic mass lesion and the rate of cerebral edema formation, the intracranial compensatory mechanisms can accommodate an increased volume of 50 to 100 mL. Beyond that, even small additional changes in intracranial volume relationships, such as those caused by vasodilation, CSF obstruction, or small areas of focal edema, cause a dramatic increase in ICP. If ICP increases to the point where CPP is compromised, vasoparalysis occurs and autoregulation is lost. The CBF then depends directly on the systemic MAP. With the loss of autoregulation, massive cerebral vasodilation occurs. Systemic pressure is transmitted to the capillaries, and the outpouring of fluids into the extravascular space can contribute to vasogenic edema and thus further increase ICP. If ICP rises to the level of the systemic arterial pressure, CBF ceases and brain death occurs.

Methods to reduce elevated ICP include hyperventilation, use of osmotic and diuretic agents, and CSF drainage. Uncontrollable increased ICP is defined as an ICP of 20 mm Hg or higher refractory to treatment. If ICP is not controlled, herniation syndromes can occur, resulting in brainstem compression and subsequent cardiorespiratory arrest. In the United States, the use of ICP monitoring and control has become standard in cases of moderate and severe TBI despite the lack of prospec-

tive controlled research showing clear efficacy as an individual patient treatment modality.¹⁵

Brain Swelling and Cerebral Edema

Two primary types of brain swelling occur after head injury. *Congestive brain swelling* results from an increased intracranial blood volume. Hyperemia occurs early after trauma and can persist for the first few days after injury.²³ It is especially common in children. The increased blood volume is most likely caused by vasodilation, which occurs as a compensatory mechanism to maintain optimal CBF in the presence of increased metabolic needs of the damaged brain tissue.

Cerebral edema is an increase in brain volume caused by an absolute increase in cerebral tissue water content. Diffuse cerebral edema may develop soon after head injury; however, its presence and extent do not always correlate with the severity of head injury. On computed tomography (CT) scans, diffuse edema is manifest as bilateral compression of the ventricles, loss of definition of the cortical sulci, or effacement of the basal cisterns (Fig. 38-4). Focal edema adjacent to traumatic mass lesions demonstrates decreased density on CT scans compared with normal tissue. CT can also detect a *mass effect*, caused by edema surrounding a traumatic lesion.

Both vasogenic and cytotoxic cerebral edema occur in the setting of trauma; the incidence and onset of each relative to the other depend on the nature of the injury. *Vasogenic edema* arises from transvascular leakage caused by mechanical failure of the tight endothelial junctions of the BBB.^{23,24} Vasogenic edema accumulates preferentially in white matter and can become widespread. It is frequently associated with focal contusions or hematomas. Vasogenic edema eventually resolves as edema fluid is reabsorbed into the vascular space or the ventricular system.



Figure 38-4. Non-contrast-enhanced computed tomography scan showing diffuse cerebral edema. Loss of gray-white differentiation in brain parenchyma is present. Bilateral compression of the ventricles has occurred with loss of cortical sulci.

Cytotoxic edema is an intracellular process that results from membrane pump failure. It is common after head injury and is frequently associated with post-traumatic ischemia and tissue hypoxia. Normal membrane pump activity depends on adequate CBF to ensure adequate substrate and oxygen delivery to brain tissue. If the CBF is reduced to 40% or less of baseline, cytotoxic edema begins to develop. If CBF drops to 25% of baseline, membrane pumps fail and cells begin to die. Congestive brain swelling can contribute to cytotoxic edema if it becomes severe enough to increase ICP and reduce CPP so that cerebral circulation cannot be maintained. Recent work suggests that cytotoxic cerebral edema is the predominant form of edema in patients who have experienced TBI.²⁵

Altered Levels of Consciousness

Consciousness is the state of awareness of the self and of the environment, and it requires intact functioning of the cerebral cortices and the reticular activating system (RAS) of the brainstem. An altered level of consciousness is the hallmark of brain insult from any cause and results from an interruption of the RAS or a global event that affects the cortices of both hemispheres.

A patient who has sustained TBI typically has an altered level of consciousness. Head trauma patients may be hypoxic from injury to respiratory centers or from concomitant pulmonary injury. Hypotension from other associated injuries can compromise CBF and affect consciousness. Global suppression may result from an intoxicant consumed before the injury, hypoglycemia, a post-traumatic seizure, or a postictal period following a seizure from any cause. With increasing ICP from

brain swelling or an expanding mass lesion, brainstem compression and subsequent RAS compression can occur.

Patients with altered levels of consciousness require careful monitoring and observation. Reversible conditions that can alter mental status, such as hypoxia, hypotension, or hypoglycemia, should be corrected as they are identified.

Cushing's Reflex

Progressive hypertension associated with bradycardia and diminished respiratory effort is a specific response to acute, potentially lethal increases in ICP. This response is called the *Cushing reflex*, or *Cushing's phenomenon*, and its occurrence indicates that the ICP has reached life-threatening levels. The Cushing reflex can occur whenever ICP is increased, regardless of the cause. The full triad of hypertension, bradycardia, and respiratory irregularity is seen in only one third of cases of life-threatening increased ICP.²⁴

Cerebral Herniation

Cerebral herniation occurs when increasing cranial volume and ICP overwhelm the natural compensatory capacities of the CNS (Fig. 38-5). Increased ICP may be the result of post-traumatic brain swelling, edema formation, traumatic mass lesion expansion, or any combination of the three. When increasing ICP cannot be controlled, the intracranial contents shift and herniate through the cranial foramen. Herniation can occur within minutes or up to days after TBI. When the signs of herniation syndrome are present, however, mortality approaches 100% without rapid implementation of temporizing emergency measures and definitive neurosurgical therapy.

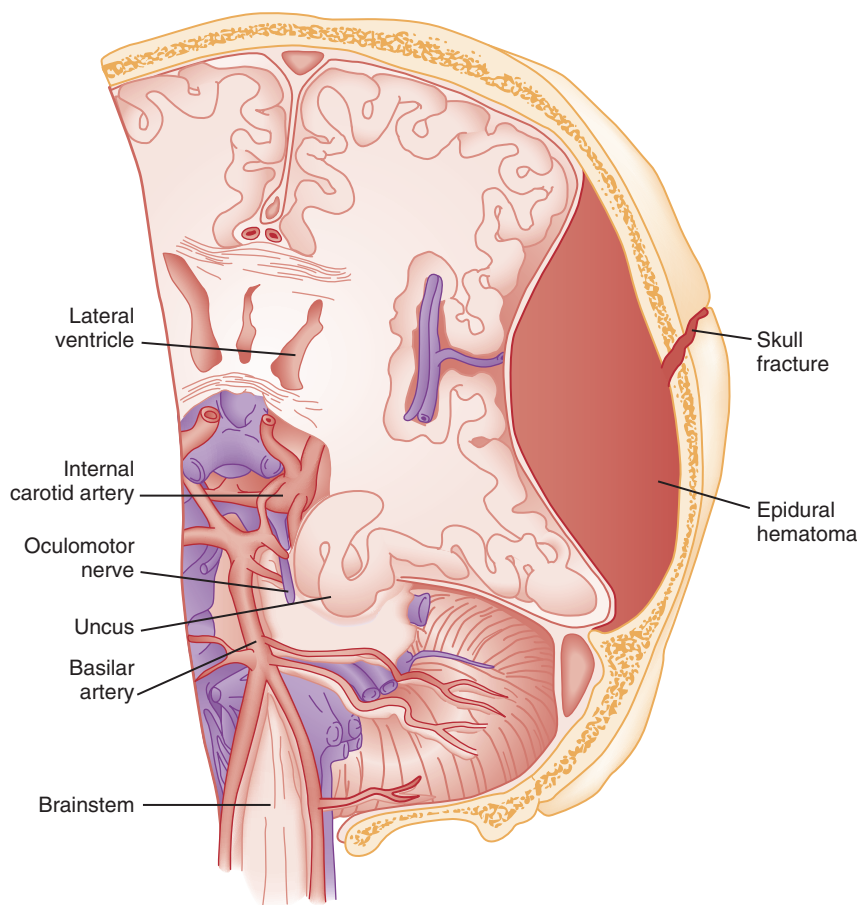


Figure 38-5. Anterior view of transtentorial herniation caused by large epidural hematoma. Skull fracture overlies hematoma. (From Rockswold GL: Head injury. In Tintinalli JE, et al [eds]: Emergency Medicine. New York, McGraw-Hill, 1992, p 915.)

Uncal Herniation. The most common clinically significant traumatic herniation syndrome is uncal herniation, a form of transtentorial herniation. Uncal herniation is often associated with traumatic extra-axial hematomas in the lateral middle fossa or the temporal lobe. The classic signs and symptoms are caused by compression of the ipsilateral uncus of the temporal lobe on the U-shaped edge of the tentorium cerebelli as the brain is forced through the tentorial hiatus. As compression of the uncus begins, the third cranial nerve is compressed; anisocoria, ptosis, impaired extraocular movements, and a sluggish pupillary light reflex develop on the side ipsilateral to the expanding mass lesion. This phase may last for minutes to hours, depending on how rapidly the expanding lesion is changing. As the herniation progresses, compression of the ipsilateral oculomotor nerve eventually causes ipsilateral pupillary dilation and nonreactivity.

Initially in the uncal herniation process, the motor examination can be normal, but contralateral Babinski's responses develop early.²⁴ Babinski's sign is dorsiflexion of the great toe and fanning of the other toes. Contralateral hemiparesis develops as the ipsilateral peduncle is compressed against the tentorium. With continued progression of the herniation, bilateral decerebrate posturing eventually occurs; decorticate posturing is not always seen with the uncal herniation syndrome. In up to 25% of patients, the contralateral cerebral peduncle is forced against the opposite edge of the tentorial hiatus. Hemiparesis is then detected ipsilateral to the dilated pupil and the mass lesion. This is termed *Kernohan's notch syndrome* and causes false-localizing motor findings.

As uncal herniation progresses, direct brainstem compression causes additional alterations in the level of consciousness, respiratory pattern, and cardiovascular system. Mental status changes may initially be quite subtle, such as agitation, restlessness, or confusion, but soon lethargy occurs with progression to frank coma. The patient's respiratory pattern may initially be normal, followed by sustained hyperventilation. With continued brainstem compression, an ataxic respiratory pattern develops. The patient's hemodynamic status may change, with rapid fluctuations in blood pressure and cardiac conduction. Herniation that is uncontrolled progresses rapidly to brainstem failure, cardiovascular collapse, and death.

Central Transtentorial Herniation. The central transtentorial herniation syndrome is demonstrated by rostrocaudal neurologic deterioration caused by an expanding lesion at the vertex or the frontal or occipital pole of the brain. It is less common than uncal transtentorial herniation. Clinical deterioration occurs as bilateral central pressure is exerted on the brain from above. The initial clinical manifestation may be a subtle change in mental status or decreased level of consciousness, bilateral motor weakness, and pinpoint pupils (<2 mm). Light reflexes are still present but are often difficult to detect. Muscle tone is increased bilaterally, and bilateral Babinski's signs may be present. As central herniation progresses, both pupils become midpoint and lose light responsiveness. Respiratory patterns are affected and sustained hyperventilation may occur. Motor tone increases. Decorticate posturing is elicited by noxious stimuli. This progresses to bilateral decorticate and then spontaneous decerebrate posturing. Respiratory patterns initially include yawns and sighs and progress to sustained tachypnea, followed by shallow slow and irregular breaths immediately before respiratory arrest.

Cerebellotonsillar Herniation. Cerebellotonsillar herniation occurs when the cerebellar tonsils herniate downward through the foramen magnum. This is usually caused by a cerebellar mass or a large central vertex mass causing the rapid displacement of the entire brainstem.²⁴ Clinically, patients demonstrate sudden respiratory and cardiovascular collapse as the medulla

is impinged. Pinpoint pupils are noted. Flaccid quadriplegia is the most common motor presentation because of bilateral compression of the corticospinal tracts. Mortality from cerebellar herniation approaches 70%.²⁴

Upward Transtentorial Herniation. Upward transtentorial herniation occasionally occurs as a result of an expanding posterior fossa lesion. Level of consciousness declines rapidly. These patients may have pinpoint pupils from compression of the pons. Downward conjugate gaze is accompanied by the absence of vertical eye movements.²⁴

CLINICAL FEATURES AND DIAGNOSTIC STRATEGIES

History

Details regarding the mechanism of injury should be solicited from witnesses or the victim to determine whether the head-injured patient is at high risk for intracranial injury. The patient's condition before trauma may give clues to important, otherwise unsuspected, comorbid factors such as preexisting coagulopathy (i.e., hemophilia). Past medical history, medications (particularly anticoagulants), recent drug or alcohol use, and complaints immediately preceding the traumatic event should be determined.

The patient's current level of consciousness, as well as that immediately before and after the injury and at the arrival of first responders, should be determined. Witnessed post-traumatic seizures or apnea should be reported. If the patient is now awake but was unconscious at some point, it should be determined if the patient has returned to baseline mental status.

Acute Neurologic Examination

General

The goals of the acute neurologic assessment of head-injured patients include detection of life-threatening injuries and identification of neurologic changes in the immediate post-trauma period. An awake, stable patient can undergo a relatively complete neurologic examination. In other patients, an efficient neurologic examination in the emergency setting includes evaluation of mental status, GCS score, pupillary size and responsiveness, and motor strength and symmetry. An accurate neurologic assessment in the immediate posttrauma period serves as a basis for comparison in subsequent examinations. If a formal GCS measure is not possible or is difficult because of comorbid confounders, the patient's mental status should be described in as much detail as possible. Declining mental status after head trauma suggests increasing ICP from an expanding mass lesion or worsening cerebral edema, which may rapidly become life threatening.

Glasgow Coma Scale

The GCS is an objective method of following the patient's neurologic status (Table 38-1). The GCS assesses a patient's best eye, verbal, and motor responsiveness. It was developed for the clinical evaluation of head trauma patients at 6 hours after trauma, and all initial validation studies investigated its application at this time. It was designed for assessment of patients with isolated head trauma who were hemodynamically stable and adequately oxygenated.²⁶ The GCS is only one aspect of the neurologic examination (i.e., the motor score reflects best limb movement, and it cannot detect subtle changes in mental status). However, because of its interrater

Table 38-1 Glasgow Coma Scale

RESPONSE	SCORE	SIGNIFICANCE
EYE OPENING		
Spontaneously	4	Reticular activating system is intact; patient may not be aware
To verbal command	3	Opens eyes when told to do so
To pain	2	Opens eyes in response to pain
None	1	Does not open eyes to any stimuli
VERBAL STIMULI		
Oriented, converses	5	Relatively intact CNS, aware of self and environment
Disoriented, converses	4	Well articulated, organized, but disoriented
Inappropriate words	3	Random exclamatory words
Incomprehensible	2	Moaning, no recognizable words
No response	1	No response or intubated
MOTOR RESPONSE		
Obeys verbal commands	6	Readily moves limbs when told to
Localizes to painful stimuli	5	Moves limb in an effort to remove painful stimuli
Flexion withdrawal	4	Pulls away from pain in flexion
Abnormal flexion	3	Decorticate rigidity
Extension	2	Decerebrate rigidity
No response	1	Hypotonia, flaccid; suggests loss of medullary function or concomitant spinal cord injury

CNS, central nervous system.

reliability, reliance on objective clinical data, and ease of application, the GCS has become a standard acute measure of neurologic function in patients with altered mental status from any cause, including head trauma.

The acute application (<6 hours) of the GCS in head-injured patients has limitations. Hypoxia, hypotension, and intoxication can falsely lower the initial GCS.²⁶ Intubation lowers the patient's GCS by automatically assigning a score of 1 for verbal response, regardless of the actual contribution of head injury to the clinical examination. Periorbital edema from direct eye trauma may make assessment of spontaneous eye opening difficult. Extremity fractures or occult spinal cord injuries may interfere with the motor examination. Children and non-English-speaking patients are difficult to assess with the GCS. The GCS may miss subtle mental status changes and does not assess brainstem reflexes or pupillary reflexes. Decisions regarding continued resuscitation of severely head-injured patients should not be based on the initial GCS because of these limitations. Patients must be fully resuscitated, with evacuation of all surgical lesions, must remain hemodynamically stable, and must not be intoxicated before the GCS can be used to predict their prognosis.²⁶

Pupillary Examination

An evaluation of the patient's pupil size and responsiveness must be done early in the initial assessment of the head-

injured patient. Pupillary asymmetry, the loss of the light reflex, or a dilated pupil suggests herniation syndrome. Traumatic mydriasis, resulting from direct injury to the eye and periorbital structures, may confuse the assessment of the pupillary responsiveness.

Motor Examination: Posturing

The patient's acute motor examination assesses for strength and symmetry. Paralysis obscures involuntary reflexes; attempts should be made to assess the motor exam before paralytic agents are given. Hemiparesis contralateral to a fixed and dilated pupil suggests herniation syndrome. A false-localizing motor examination can be caused by contralateral cerebral parenchymal injury occurring simultaneously with the expanding mass lesion or by Kernohan's notch syndrome (compression of the contralateral cerebral peduncle). False-localizing signs for the motor examination can also be caused by occult extremity trauma, spinal cord, or nerve root injury that makes the examination painful or difficult. If the patient is not cooperative or is comatose, motor movement should be elicited by application of noxious stimuli. Any movement should be recorded. Voluntary purposeful movement must be distinguished from abnormal motor posturing. *Decorticate posturing* is abnormal flexion of the upper extremity and extension of the lower extremity. The arm, wrist, and elbow slowly flex, and the arm is adducted. The leg extends and internally rotates, with plantar flexion of the foot. Decorticate posturing implies injury above the midbrain. *Decerebrate posturing* is the result of a more caudal injury and therefore is associated with a worse prognosis.¹⁹ The arms extend abnormally and become adducted. The wrist and fingers are flexed, and the entire arm is internally rotated at the shoulder. The neck undergoes abnormal extension, and the teeth may become clenched. The leg is internally rotated and extended, and the feet and toes are plantar flexed.

Brainstem Function

In the acute setting, brainstem activity is assessed by the patient's respiratory pattern, pupillary size, and eye movements. The *oculocephalic response* (doll's eyes maneuver) tests the integrity of the pontine gaze centers. This response cannot be tested until cervical spine fractures have been ruled out. The *oculovestibular response* (cold water caloric) also assesses the brainstem. Comatose patients no longer demonstrate nystagmus when cold water is placed in the ear canal; the only response is tonic deviation of the eyes toward the instilled cold water.²⁴ This response is dampened by cerumen or blood in the patient's ear canal, and the tympanic membrane must be intact to perform this test.

In the severely head-injured patient, the cranial nerve (CN) examination is often limited to the pupillary responses (CN III), gag reflex (CNs IX and X), and corneal reflex (CNs V and VII). Facial symmetry (CN VII) can sometimes be assessed if the patient grimaces with noxious stimuli. In patients who are awake and can cooperate, a formal CN examination should be performed.

Deep Tendon Reflexes and Pathologic Reflexes

Tendon reflexes should be tested for symmetry. An extensor plantar reflex (Babinski's sign) is nonspecific and can be caused by injury anywhere along the corticospinal tract. Rectal sphincter tone and anal reflexes should be determined to assess for spinal cord integrity.

BOX 38-1**CLINICAL CHARACTERISTICS OF BASILAR SKULL FRACTURES**

Blood in ear canal
 Hemotympanum
 Rhinorrhea
 Otorrhea
 Battle's sign (retroauricular hematoma)
 Raccoon sign (periorbital ecchymosis)
 Cranial nerve deficits
 Facial paralysis
 Decreased auditory acuity
 Dizziness
 Tinnitus
 Nystagmus

Other Examination Findings

The head and neck should be carefully examined for external signs of trauma that may have also produced underlying TBI. A scalp laceration, contusion, abrasion, or avulsion may overlie a depressed skull fracture. Basilar skull fractures are usually diagnosed by the clinical examination (Box 38-1). Although not always related to severe brain injury, their presence implies that a significant impact force was sustained during head trauma. Carotid artery dissections caused by a hyperflexion-extension neck injury can occasionally be detected by auscultation of a carotid bruit.²⁴ In these patients, a careful neurologic examination should assess for subtle asymmetry between the carotid arteries. The percentage of concurrent cervical spine injury in patients with severe head trauma may be as high as 10.2%.²⁷ Often, other spinal regions are also injured.

MANAGEMENT**Severe Traumatic Brain Injury**

The neurosurgical literature defines severe head injury as TBI manifested by a postresuscitation GCS of 8 or less within 48 hours. In the emergency setting, however, this definition is not practical because the outcome for the patient beyond the initial resuscitation is not known. Most emergency medicine research defines severe head injury by a GCS score of 8 or less at the acute presentation after injury. The presence of any intracranial contusion, hematoma, or laceration is also considered severe injury (Fig. 38-5).

Approximately 10% of all head-injured patients who reach the emergency department alive have severe head trauma.¹ The clinical prognostic indicators in the acute setting are initial motor activity, pupillary responsiveness, the patient's age and premorbid condition, and the occurrence of secondary systemic insult during the acute period.^{25,26} Up to 25% of these patients have lesions requiring neurosurgical evacuation.¹⁵ The prognosis cannot be reliably predicted by the initial GCS or initial CT scan.

The overall mortality in severe head trauma approaches 60%.^{1,24} Mortality for children is lower. For nonsurvivors of head injury who reach the hospital alive, the average time to death is 2 days after trauma. Adult survivors of severe head trauma are usually severely disabled; currently, only 7% have moderate disability or a good outcome. Children older than 2 years who survive a severe closed-head injury have a better outcome than adults.

Out-of-Hospital Care

The goals of the out-of-hospital management of the head-injured patient are necessary airway interventions to prevent hypoxia and establishing intravenous (IV) access to treat trauma-related hypotension. An accurate neurologic assessment provides a means to determine the subsequent effectiveness of treatment and should focus on the GCS, pupillary responsiveness and size, level of consciousness, and motor strength and symmetry.

Head trauma can produce profound effects on the cardiovascular system if compression of the brainstem and medulla occurs. Any cardiac dysrhythmia can occur and produce cardiac instability.²⁸ All head-injured patients should be placed on a cardiac monitor during transport from the accident scene.

The secondary survey of the head-injured patient should include a search for external signs of head trauma. Scalp lacerations may bleed a large volume into a bulky dressing. A less bulky dressing should be used with firm constant manual pressure applied to avoid excessive blood loss. Many severely head-injured patients are initially combative or agitated. Transporting an agitated patient who is fighting against physical restraints may exacerbate physical injury, cause an increase in ICP, and interfere with appropriate stabilization and management. It may be necessary to use out-of-hospital sedation or neuromuscular blockade for control. The use of sedatives or neuromuscular blockade may influence the initial emergency department evaluation of the neurotrauma patient. Therefore, the risks and benefits of this acute intervention must be carefully considered and decisions made on a case-by-case basis. Out-of-hospital protocols allowing the use of sedative agents for selected agitated head-injured patients should be established. Currently used agents include lorazepam (Ativan), diazepam (Valium), midazolam (Versed), and certain butyrophenones (i.e., haloperidol, droperidol, and tripartolol).

Severe head injury is the most common reason for helicopter transfers in trauma care. Although the decision to transport by helicopter should be made on a case-by-case basis, considerations for helicopter use from an accident scene include a long extrication time, ground transport of longer than 30 minutes to an appropriate emergency department and trauma care facility, two or more severely injured patients at a scene, and assistance in performing expedient lifesaving procedures, especially airway management.

Controversy exists regarding the benefits of out-of-hospital intubations in patients with severe and moderate head injuries. It is unclear if field intubations truly improve neurologic outcome or survival. Unsuccessful attempts at field intubations may add to out-of-hospital time and increase the risk for aspiration or hypoxia.

In 1997, Winchell and Hoyt²⁹ showed that patients who had sustained severe head injuries and who were intubated in the out-of-hospital setting had an improved survival compared with those who were not intubated. Since that time, others have challenged this finding. In the San Diego paramedic rapid sequence intubation (RSI) trial, Davis and colleagues³⁰ found an increase in mortality and morbidity in patients who sustained severe head injuries and underwent out-of-hospital RSI compared to matched historical controls. Potential explanations included frequent hypoxic episodes with associated bradycardia, unintentional hyperventilation, and prolonged scene times for those undergoing out-of-hospital RSI. Wang and colleagues²⁰ found a fourfold increase in mortality among patients who sustained severe TBI and received ground ambulance intubation compared with emergency department intubation. Flight clinician out-of-hospital intubation was associated

with decreased mortality and improved neurologic outcome, likely due to the higher airway management training requirements of air medical programs. These studies are somewhat limited by lack of generalizability³⁰⁻³³ (i.e., most emergency medical service systems do not have RSI capabilities) or by the use of nonvalidated outcome measures of neurologic function.²⁰ Out-of-hospital intubation carries the following risks: frequent hypoxic episodes during intubation attempts with or without concurrent bradycardia; unintentional hyperventilation of intubated patients; prolonged scene times because of the time demands associated with the intubation process; and persistent hypotension, likely from other injuries that go unattended while the airway management occurs. However, hypoxia must be avoided in head-injured patients, and out-of-hospital airway protocols must balance the risks of emergency intubation in an uncontrolled setting with the need to secure an airway at risk.

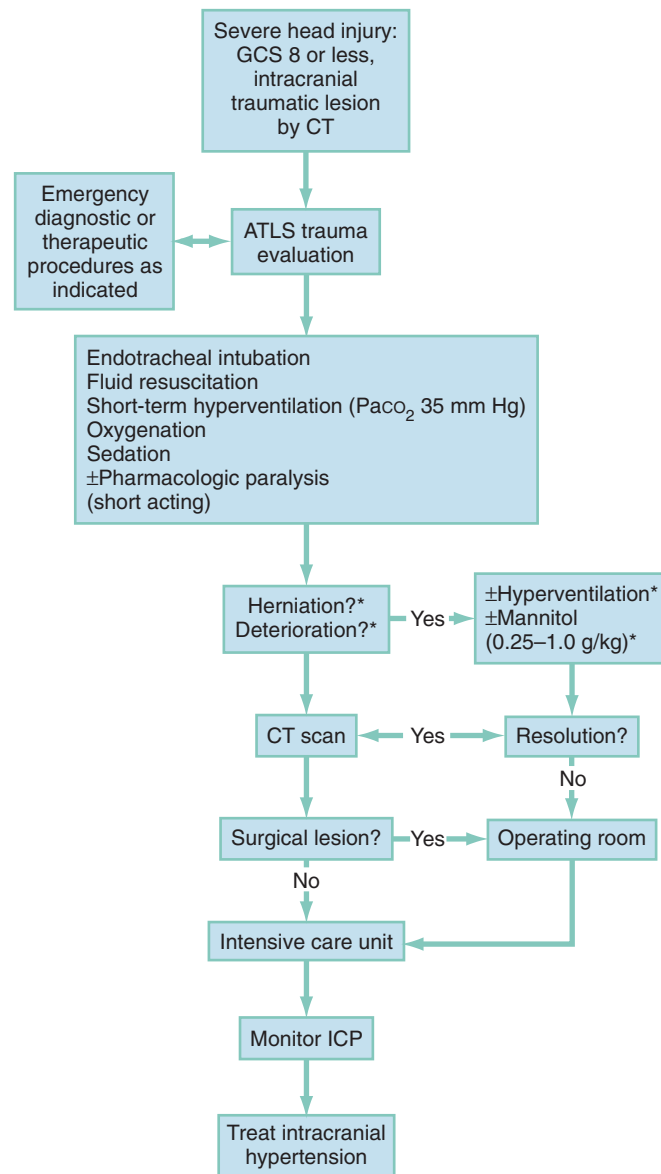
The key to a successful out-of-hospital RSI program involves well-trained clinicians with specific RSI protocols, involved medical control, frequent continuing education, and consistent quality assurance/improvement. Fakhry and colleagues³⁴ reported that their helicopter clinicians had a 96.6% RSI success rate with few complications and no esophageal intubations.

Emergency Department (Fig. 38-6)

Airway. Rapid sequence intubation is an effective and the preferred method for securing the airway in combative or agitated patients. If possible, a brief neurologic examination should be performed before the patient is given any sedative or neuromuscular blocking agents. In general, the agents used for RSI in the head-injured patient are the same as those used for other patients, although attention must be given to the increased ICP that can potentially occur with any physical stimulation of the respiratory tract. Lidocaine (1.5–2 mg/kg IV push) may attenuate the cough reflex, hypertensive response, and increased ICP associated with intubation, although this is vigorously debated.^{35,36} If succinylcholine is used, premedication with a subparalytic dose of a nondepolarizing agent can be considered if time permits because fasciculations produced by succinylcholine may increase ICP. The degree of ICP elevation and its clinical significance are unclear, however, and must be balanced against the need for rapidly establishing an airway. Etomidate (0.3 mg/kg IV), a short-acting sedative-hypnotic agent, has beneficial effects on ICP by reducing CBF and metabolism.³⁷ In addition, etomidate has minimal adverse effects on blood pressure and cardiac output and fewer respiratory depressant effects than other agents.

Hypotension. Hypotension is rarely caused by head injury except as a terminal event. If hypotension is detected at any time in the emergent management of a head-injured patient, a cause other than the head injury should be sought. Some important exceptions occur. Profound blood loss from scalp lacerations can cause hypovolemic hypotension. In small children, hemorrhage into an epidural or subgaleal hematoma can produce profound hypovolemic shock. In the presence of a concomitant high spinal cord injury, neurogenic hypotension may occur. This is rare and usually the cord injury is apparent on physical examination. In less obvious cases, neurogenic hypotension can be differentiated from hypovolemic hypotension by its nonresponsiveness to fluid administration and by the presence of inappropriate bradycardia in the face of hypotension in neurogenic shock.

Systemic hypotension cannot be tolerated in the head-injured patient without profound worsening of neurologic outcome; fluids or blood transfusion should therefore be deliv-



*Only in the presence of signs of herniation or progressive neurologic deterioration not attributable to extracranial factors.

Figure 38-6. Initial resuscitation of patient with severe head injury: treatment options. ATLS, advanced trauma life support; CT, computed tomography; GCS, Glasgow Coma Scale; ICP, intracranial pressure; Paco₂, arterial carbon dioxide partial pressure. (From the Brain Trauma Foundation, American Association of Neurological Surgeons, Joint Section on Neurotrauma and Critical Care: Introduction. Guidelines for the management of severe traumatic brain injury. *J Neurotrauma* 17:465, 2000.)

ered to maintain a systolic blood pressure of at least 90 mm Hg.¹⁹ The delivery of large amounts of fluid to severely head-injured patients who are hypotensive from other injuries does not produce clinically significant increases in ICP; fluids should never be withheld in the head trauma patient with hypovolemic hypotension for fear of increasing cerebral edema and ICP. Hypotension may interfere with the accurate neurologic assessment of the brain-injured patient. Often, when blood pressure is restored, an improved neurologic status is observed.

Traditionally, normal saline or lactated Ringer's solution has been used for resuscitation of trauma patients with hypovolemic hypotension. Although it is not yet included in practice

guidelines on the management of head-injured patients, some researchers suggest that fluid resuscitation with hypertonic saline rather than normal saline may improve neurologic outcome after TBI.¹⁹

As many as 60% of patients with severe head injury are victims of multiple trauma.³⁸ The dramatic presentation of the head injury should not distract the clinician from a thorough search for other life threats.

The emergency department neurologic assessment should be compared with the initial out-of-hospital examination, focusing on evidence of neurologic deterioration or signs of increasing ICP. If the patient is deteriorating or has signs of increased ICP, active intervention must be initiated in the emergency department.

Hyperventilation. Acute hyperventilation is a life-saving intervention that can prevent or delay herniation in the patient with severe TBI. The goal is to reduce the P_{CO_2} to the range of 30 to 35 mm Hg. Hyperventilation will reduce ICP by causing cerebral vasoconstriction; the onset of effect is within 30 seconds²⁴ and probably peaks within 8 minutes after the P_{CO_2} drops to the desired range.³⁹ In most patients, hyperventilation lowers the ICP by 25%; if the patient does not rapidly respond, the prognosis for survival is generally poor.

Prolonged hyperventilation is not recommended because it may cause profound vasoconstriction and ischemia. This vasoconstriction worsens cerebral blood flow that is already severely compromised during the first 24 hours after TBI.¹¹ Hyperventilation should be viewed as a short-term lifesaving intervention and should be used only when a patient experiences an acute neurologic decline or demonstrates signs consistent with herniation.

Osmotic Agents. Additional therapy for increased ICP includes the use of osmotic diuretics, such as mannitol and hypertonic saline. With deepening coma, pupil inequality, or other deterioration of the neurologic examination, osmotic agents may be lifesaving.

Mannitol is the mainstay for control of elevated ICP acute severe TBI. The Brain Trauma Foundation and the European Brain Injury Consortium recommend mannitol as the osmotic drug of choice.^{40,41} However, little comparative data exist on mannitol and other ICP-lowering medications. A Cochrane database review concluded that mannitol may have a small beneficial effect compared to pentobarbital. ICP-directed therapy based on neurologic signs may also be beneficial. However, one study indicates that mannitol may be detrimental compared to hypertonic saline.⁴² Further research is needed on optimal osmotic therapy in severe head trauma.

Mannitol (0.25–1 g/kg) can effectively reduce cerebral edema by producing an osmotic gradient that prevents the movement of water from the vascular space into the cells during membrane pump failure and draws tissue water into the vascular space. This reduces brain volume and provides increased space for an expanding hematoma or brain swelling. The osmotic effects of mannitol occur within minutes and peak approximately 60 minutes after bolus administration. The ICP-lowering effects of a single bolus may last for 6 to 8 hours.⁴⁰ Mannitol has many other neuroprotective properties. It is an effective volume expander in the presence of hypovolemic hypotension and therefore may maintain systemic blood pressure required for adequate cerebral perfusion. It also promotes CBF by reducing blood viscosity and microcirculatory resistance. It is an effective free radical scavenger, reducing the concentration of oxygen free radicals that may promote cell membrane lipid peroxidation. However, mannitol can produce renal failure or hypotension if given in large doses. It may also induce a paradoxical effect of increased bleeding into a traumatic lesion by decompressing the tamponade effect

of a hematoma. Because of these and other potential problems, the use of mannitol should be reserved for head-injured patients with evidence of increasing ICP and neurologic deterioration.⁴⁰

Hypertonic saline (HTS) has been used for severe TBI since 1919.⁴³ Preclinical studies have demonstrated that HTS can significantly reduce ICP; however, fewer than 300 patients have been enrolled in all clinical trials of HTS. The interpretation of these clinical studies is complicated by variation in protocols, HTS concentration, and administration. Few studies have been prospective, randomized, and controlled. Potential adverse events associated with HTS include renal failure, central pontine myelinolysis, and rebound ICP elevation.⁴¹

Encouraging clinical data are available on hospitalized pediatric TBI patients treated with a continuous infusion of 3% normal saline for control of intracranial hypertension.⁴⁴ The clinical studies using HTS for acute resuscitation of severe TBI are conflicting. Using a post hoc analysis of adult trauma data, Vassar and colleagues⁴⁵ and Wade and colleagues⁴⁶ showed beneficial effects of HTS on patients with severe TBI. However, Cooper and colleagues⁴⁷ found no benefit for out-of-hospital use of HTS in reducing elevated ICP and improving CPP compared to lactated Ringer's in hypotensive patients who had a head injury. Morbidity and mortality outcome data were equal in both groups. In summary, clinical effectiveness data on the use of HTS in head-injured adults for acute treatment of increased ICP are inconclusive, but research on this topic is ongoing.

Barbiturates. Barbiturate therapy is occasionally used in severely head-injured patients to reduce cerebral metabolic demands of the injured brain tissue. Barbiturates also affect vascular tone and inhibit free radical-mediated cell membrane lipid peroxidation. The effects of barbiturates are delayed relative to other acute interventions for reducing ICP; therefore, they are rarely initiated in the emergency department. If other methods of reducing ICP have been unsuccessful, barbiturates may be added in the hemodynamically stable patient. *Pentobarbital* is the barbiturate most often used.⁴⁸

Steroids. Despite their popularity in the past, there is no benefit to giving steroids in head-injured patients. They do not lower ICP, and high-dose methylprednisolone in moderate and severe TBI is associated with increased mortality.⁴⁹

Hypothermia. Although hypothermia remains a significant area of research and promise for severe and moderate TBI patients, the available scientific evidence does not support improved mortality or morbidity with prophylactic hypothermia in adult patients.⁵⁰ In a meta-analysis performed by the Brain Trauma Foundation, duration of hypothermic treatment for more than 48 hours was associated with a reduction in mortality. This finding is significantly limited due to small sample sizes.⁵⁰

Cranial Decompression. Patients with signs of herniation who have not responded to other means of ICP reduction and who are rapidly deteriorating in the emergency department should be considered for placement of emergency burr holes. Emergency trephination has been described for centuries (Fig. 38-7). It is a blind invasive procedure, and the chances of localizing the expanding lesions are uncertain. In carefully considered patients, however, emergency cranial decompression may temporarily reverse or arrest the herniation syndrome, providing the time needed to prepare the patient for formal craniotomy.

Most patients who have been unconscious since an accident occurred—with erratic or absent respiratory effort, bilateral fixed and dilated pupils, no spontaneous eye movements, and decerebrate posturing—have sustained diffuse massive brain injury with no focal lesion amenable to emergency decompression. These patients probably do not benefit from emergent

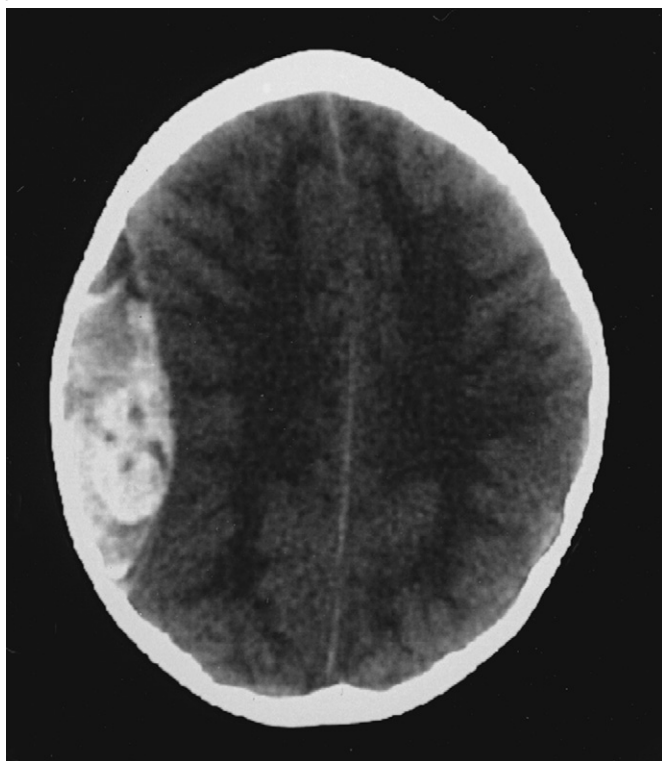


Figure 38-7. Non-contrast-enhanced computed tomography scan of acute epidural hematoma at the level of right midconvexity. There is an associated mass effect and moderate midline shift.

burr holes. Instead, these patients should undergo rapid CT scanning or formal surgical decompression.

Seizure Prophylaxis. Up to 12% of all patients who sustain blunt head trauma and 50% of those with penetrating head injury develop early post-traumatic seizures.⁵¹ Although the occurrence of seizures in the immediate posttrauma period has no predictive value for future epilepsy, early seizures can cause hypoxia, hypercarbia, release of excitatory neurotransmitters, and increased ICP, which can worsen secondary brain injury. Constantly firing neurons are soon depleted of their energy sources, and in the head trauma patient with compromised cerebral metabolism, uncontrolled seizures exacerbate the neurologic deficit.²⁴

Box 38-2 lists accepted indications for early anticonvulsant therapy after head trauma. If the patient is actively seizing, benzodiazepines are administered as effective, rapid-acting first-line anticonvulsants. Lorazepam (0.05–0.15 mg/kg IV over 2–5 minutes up to a total of 4 mg) has been found to be most effective at aborting status epilepticus.⁵¹ Diazepam (0.1 mg/kg, up to 5 mg IV, every 10 minutes up to a total of 20 mg) is an alternative. For long-term anticonvulsant activity, phenytoin (18–20 mg/kg IV) or fosphenytoin (15–18 phenytoin equivalents/kg) IV or IM can be given. Fosphenytoin has the advantages of rapid administration, a smaller volume of fluid for the dose delivered, and less hypotension than phenytoin, although its cost is much higher. In a Cochrane review, the use of antiepileptic drugs reduced the risk of early seizures by 66%.⁵¹ Early seizure prophylaxis does not prevent late post-traumatic seizures; the goal is to prevent additional insult to the damaged brain.^{52,53}

If the patient has been paralyzed to facilitate management, clinical manifestations of generalized seizures are obscured. Therefore, all paralyzed head-injured patients should have prophylactic anticonvulsant therapy in the acute phase. Con-

BOX 38-2

INDICATIONS FOR ACUTE SEIZURE PROPHYLAXIS IN SEVERE HEAD TRAUMA

- Depressed skull fracture
- Paralyzed and intubated patient
- Seizure at the time of injury
- Seizure at emergency department presentation
- Penetrating brain injury
- Severe head injury (Glasgow Coma Scale score ≤ 8)
- Acute subdural hematoma
- Acute epidural hematoma
- Acute intracranial hemorrhage
- Prior history of seizures

tinuous electroencephalographic monitoring is necessary for the ongoing assessment of seizure activity in paralyzed patients and, if available, should be initiated in the emergency department or the intensive care unit.

Antibiotic Prophylaxis. Infection may occur as a complication of penetrating head injury, open skull fractures, and complicated scalp lacerations. Prophylactic antibiotics may be used in these circumstances but are not recommended in patients with otorrhea or rhinorrhea from a basilar skull fracture.⁵⁴

Recombinant factor VIIa (rFVIIa) is a hemostatic agent that was originally developed to treat bleeding in hemophiliacs. Considerable interest has arisen regarding its potential use in intracerebral hemorrhage.^{55,56} A single, appropriate dose of rFVIIa for a 70-kg individual can exceed \$4500.⁵⁶ Experience from the Iraq war has produced conflicting results regarding its benefits in traumatic intracranial hemorrhage. Use of rFVIIa for traumatic head bleeds should be individualized and made in concert with an institution's treatment protocol.

Ancillary Evaluation

Laboratory Tests. The acute management of the severely head-injured patient is directed by physical examination and diagnostic imaging. Ancillary laboratory tests that may provide useful information in the subsequent management of the patient include a urine toxicology screen, blood alcohol level, complete blood count, electrolytes, glucose, and coagulation studies.

Neuroimaging. The advantages and indications for neuroimaging techniques in the acute evaluation of head injury are listed in Table 38-2. In the acute phase, the most useful imaging technique is a non-contrast-enhanced head CT scan. This scan delineates acute intra-axial and extra-axial bleeding, subarachnoid blood, cerebral swelling, ischemic infarction caused by hypoxia after trauma, evidence of increased ICP, and pneumocephalus. Emergency management decisions are strongly influenced by these acute CT scan findings. The bone windows of the CT scan can detect skull fractures (including basilar fractures); plain skull radiographs are not necessary in patients who undergo CT scanning.

Magnetic resonance imaging (MRI) is better than CT in detecting post-traumatic ischemic infarctions, subacute non-hemorrhagic lesions and contusions, axonal shear injury, and lesions in the brainstem or posterior fossa. Monitoring and managing patients in the MRI suite can be very difficult, especially in severe TBI patients who have other life-threatening injuries. MRI is not recommended as the first-line imaging modality for severe or moderate head injury.

Disposition

Consultation. All patients with severe head trauma require an imaging modality to determine the extent and nature of the brain injury and the necessity of neurosurgical intervention.

Table 38-2 Comparison of Head Imaging Modalities

	COMPUTED TOMOGRAPHY SCANS	MAGNETIC RESONANCE IMAGING	ANGIOGRAPHY	SKULL RADIOGRAPHY
Advantages	Fast Patient accessible for monitoring Defines acute hemorrhages, mass effects, bone injuries, hydrocephalus, intraventricular blood, edema	Defines contusions and pericontusion edema, post-traumatic ischemic infarction, brainstem injuries	Helps localize acute traumatic lesions Defines vascular injuries, injuries to venous sinuses Detects mass effects	Readily available May help screen some patients for further imaging studies
Disadvantages	Artifacts arise from patient's movement, foreign bodies Streak artifacts may obscure brainstem or posterior fossa	Slow Patients not easily accessible for monitoring Does not define most acute hemorrhagic lesions Not useful for bone injuries	Does not define nature of acute lesion Does not detect infratentorial masses	Does not indicate presence or absence of intracranial injury
Indications	Acute severe head trauma Acute moderate head trauma Suspected depressed skull fracture High-risk minor head trauma Suspected child abuse in minor head trauma Deteriorating neurologic status	Persistent symptoms with postconcussive syndrome Suspected post-traumatic ischemic infarction Suspected contusions not seen on CT scan	Suspected vascular injury CT scan not available	CT scan may not be done Penetrating head trauma

CT, computed tomography.

Neurosurgical consultation should be obtained as soon as possible to help direct the patient's subsequent management.

Transfer. Severely head-injured patients require admission to an institution capable of intensive neurosurgical care and acute neurosurgical intervention. If this is not available at the receiving hospital, the patient should be transferred to an appropriate institution by the most expedient transport method available.

Priority Management. The hemodynamically unstable patient with multiple trauma that includes head injury presents difficult emergency management decisions. The emergency physician must decide on the sequence that best addresses the most life-threatening pathologic conditions while still preventing morbidity and mortality from other serious injury. If the patient requires immediate surgical intervention for a life-threatening chest or abdominal injury, complete evaluation of the head injury may be curtailed. Moreover, these patients are anesthetized for surgery, and any neurologic deterioration is not detected. Some patients may be too unstable to obtain even an abbreviated head CT scan before emergent surgical intervention for other life threats. In this circumstance, early neurosurgical and general surgical consultation should be coordinated by the emergency physician. Intraoperative ventriculostomies or bilateral trephinations may provide some temporary protection from increasing ICP while the patient undergoes surgical correction of the life-threatening injury. A CT scan can be performed after the primary life threats have been corrected.

Moderate Head Trauma

Approximately 10% of all patients with head injury have sustained moderate TBI, defined as a postresuscitation GCS of 9 to 13.¹ Moderate TBI is often caused by motor vehicle collisions. Most patients with moderate TBI do not die at the scene from their initial head injury and present to the emergency department for stabilization and evaluation.

Moderate TBI produces a number of physiologic abnormalities, including neuronal cell membrane dysfunction and a

mild, brief acidosis with no concurrent depletion of adenosine triphosphate. These changes are probably reversible and therefore may be amenable to acute intervention to correct or prevent progression. The neuropathology of moderate brain injury probably represents the front end of the spectrum of pathophysiology seen with severe head trauma. Because of this, patients must be vigilantly monitored to avoid hypoxia and hypotension and other secondary systemic insults that could worsen neurologic outcome.

Clinical Features and Acute Management

A wide variety of clinical presentations occur with moderate head injury. Patients often have experienced a change in consciousness at the time of injury, a progressive headache, post-traumatic seizures, vomiting, and post-traumatic amnesia. On emergency department presentation, patients are often confused or somnolent, but most can still follow commands. Focal neurologic deficits may be present. Many patients with moderate head trauma have concurrent serious facial injuries that may interfere with attempts at securing their airway. Other systemic trauma must also be ruled out.

An important clinical scenario in the spectrum of moderate head injury is that of the "talk and deteriorate" patient. These patients have a GCS score of 13 or greater on presentation but deteriorate to a status of a severe head injury (GCS \leq 8) within 48 hours.⁵⁷ Although this description can include patients with minor head trauma, most patients who talk and deteriorate present with GCS scores suggesting moderate head trauma. When the syndrome was first described by Reilly more than 30 years ago,⁵⁸ the incidence of death in head-injured patients who presented talking was estimated to be as high as 38%.⁵⁷⁻⁶⁰ However, CT scans were not readily available at that time, and the GCS was not in widespread use. With the current availability of early CT scanning, as well as rapid transport via emergency medical service, injuries are detected earlier, and the incidence of talk and deteriorate is now estimated to be 2.5 to 12%.^{57,61} In early descriptions of talk and deteriorate, most patients were found to have sustained subdural or epi-

dural hematomas. More recently, an equal number are found to have contusions with subsequent edema.^{57,61} Patients who talk and deteriorate are generally elders with higher injury severity scores.^{57,62} In addition, anticoagulation puts patients at increased risk.

Successful management of moderately head-injured patients involves close clinical observation for changing mental status or focal neurologic findings, early CT scanning, and aggressive neurosurgical intervention. When no neurosurgeon is available and the patient develops symptoms consistent with herniation syndrome not reversed by acute hyperventilation and mannitol, emergency department trephination and hematoma evacuation of any amenable lesion should be considered.

Because of the varied presentation of patients with moderate head trauma, the initial examination alone cannot accurately predict who will have surgically correctable intracranial lesions. Approximately 40% of moderately head-injured patients have an abnormal CT scan, and 10% lapse into coma.⁶³ A CT scan is essential in patients with moderate head trauma to avoid delayed diagnosis of traumatic mass lesions or diffuse injury. This is especially true for elders or patients on anticoagulation therapy.^{64,65} Skull radiographs may be useful if the patient has sustained a depressed skull fracture or a penetrating injury but are otherwise rarely helpful.

Disposition

All patients with moderate head injury should be admitted for observation, even with an initial apparently normal CT scan. Ninety percent of patients improve over the first few days after injury.^{24,63} Frequent neurologic checks should be initiated, and a repeated CT scan is indicated if the patient's condition deteriorates or fails to improve over the first 48 hours after trauma.

Complications

The overall mortality of patients with isolated moderate TBI is approximately 20%, but the morbidity is substantial. Most moderate TBI patients remain symptomatic for extended periods after head injury. At 3 months after trauma, up to 70% are unable to return to work, 90% have memory difficulties, and more than 90% complain of persistent headaches.^{63,66} Almost 50% are left with a long-term disability that interferes with their previous daily activities. In patients with persistent symptoms of headache, confusion, or memory difficulties, delayed MRI may define lesions in the regions related to cognition that cannot be seen on CT. Although not useful in the acute setting, MRI has prognostic value during subsequent care and assists in directing the future rehabilitation of these patients.

Minor Head Trauma

Minor TBI is a temporary and brief interruption of neurologic function after head trauma, which may involve a loss of consciousness. The neuropathology involved in producing signs and symptoms of minor TBI may remain at the neurobiochemical level, without damage to the microstructure.⁶⁷ Heightened ionic flux, surges in levels of glutamate transmitters, disruption of enzymatic pathways, and accumulation of lactate and nitric oxide have been reported in brain tissue after experimental minor TBI.¹⁴ Axonal stretching or twisting that may occur with some mechanisms producing minor TBI promote glutamine-induced neurotoxic cascades that may lead to the axonal damage typically described as diffuse axonal injury.⁶⁸

Traditionally, minor TBI was defined as injury producing a GCS score of 13 to 15. However, because many patients with a GCS of 13 have outcomes more consistent with moderate TBI, many authorities now classify minor TBI as that producing a GCS score of 14 or 15.⁶⁸ In fact, the GCS is not sensitive enough to be of prognostic usefulness in minor TBI; a perfect score of 15 in the emergency department does not take into account the level of alertness or neurologic status immediately after trauma or the presence or absence of focal neurologic injury.⁶⁸

From a practical standpoint, minor TBI is a clinical diagnosis. The diagnosis requires a credible mechanism of injury. In civilian life, most mechanisms that do not involve direct craniofacial impact cannot produce minor TBI. For example, belted drivers in low-velocity rear-end impact motor vehicle crashes are not subjected to high enough acceleration-deceleration or rotary forces to the head to reach the force threshold needed to produce cerebral injury unless there is also direct impact to the head against a stationary object.⁶⁸ It is therefore unlikely that a patient with a whiplash or "jolt" injury would also have minor TBI. Collision sports-related minor TBI can be caused by acceleration-deceleration. It is not known if the forces generated in blast injuries in wounded service personnel are large enough to cause minor TBI without direct head impact.

Clinical and Historical Features

By the time most patients with minor TBI reach the emergency department, their symptoms are resolving or have completely resolved. The most common complaint after minor TBI is headache. Other common problems are nausea and emesis. Patients may also report transient disorientation, confusion, or amnesia. There has been little research to correlate these symptoms with the presence of intracranial lesions in patients with minor TBI.

Clinical signs and symptoms of minor TBI include amnesia for the impact, a period of disordered awareness (with or without loss of consciousness), and a finite period of post-trauma amnesia.⁶⁸ Retrograde amnesia is impaired information retrieval and begins with and includes the traumatic event. In minor TBI, it generally lasts up to several minutes and then very rapidly resolves. Post-traumatic amnesia (PTA) is impairment of information encoding and therefore does not resolve. PTA is the period of time from the injury to the return of conscious recall: events during this interval are essentially lost to the patient. PTA is a better predictor of injury severity and eventual outcome than the duration of retrograde amnesia or GCS.⁶⁸

Patients with minor TBI often have balance deficits, impaired verbal memory, delayed language comprehension, and slowed speech.⁶⁹ These subtle findings can be overlooked unless a careful and complete neurologic and mental status examination is performed.

Approximately 5% of minor TBI patients presenting to the emergency department with a GCS of 15 have abnormal CT scans.⁷⁰ This estimate is probably inflated since at least 25% of minor TBI patients never seek medical care. The incidence of life-threatening lesions that require neurosurgical intervention following minor TBI is less than 1%⁶⁸; the goal of emergency evaluation and management of patients with minor TBI is to identify these high-risk patients. Further diagnostic workup hinges on risk stratification in patients with minor TBI (Box 38-3). The criteria are based on several large studies, but because of inconsistent methodology and reporting, they have limitations. For example, loss of consciousness (LOC) in minor head trauma has historically been considered a risk factor for

BOX 38-3

RISK STRATIFICATION IN PATIENTS WITH MINOR HEAD TRAUMA

High Risk

Focal neurologic findings
 Asymmetrical pupils
 Skull fracture on clinical examination
 Multiple trauma
 Serious, painful, distracting injuries
 External signs of trauma above the clavicles
 Initial Glasgow Coma Scale score of 14 or 15
 Loss of consciousness
 Post-traumatic confusion/amnesia
 Progressively worsening headache
 Vomiting
 Post-traumatic seizure
 History of bleeding disorder/anticoagulation
 Recent ingestion of intoxicants
 Unreliable/unknown history of injury
 Previous neurologic diagnosis
 Previous epilepsy
 Suspected child abuse
 Age >60 yr, <2 yr

Medium Risk

Initial Glasgow Coma Scale score of 15
 Brief loss of consciousness
 Post-traumatic amnesia
 Vomiting
 Headache
 Intoxication

Low Risk

Currently asymptomatic
 No other injuries
 No focal findings on examination
 Normal pupils
 No change in consciousness
 Intact orientation/memory
 Initial Glasgow Coma Scale score of 15
 Accurate history
 Trivial mechanism
 Injury >24 hr ago
 No or mild headache
 No vomiting
 No preexisting high-risk factors

significant injury, but the negative predictive value for LOC has not been determined, and many minor TBI patients may have sustained only brief or no LOC. Animal studies and anecdotal reports on humans suggest that the longer the duration of LOC, the more likely that an intracranial lesion exists; however, the actual correlation between the duration of the LOC and the incidence of intracranial lesions or injury severity has not been determined.^{69,70} Other high-risk minor head trauma criteria have also been proposed. The key for the emergency physician, however, is to determine the low-risk patient, and these criteria are less controversial.

Imaging Studies

A major and controversial decision regarding the emergency management of minor TBI is whether imaging studies should be performed. Several approaches have been described, but the research behind these suggestions remains confusing pri-

marily because of differences in study populations, definitions, and methods. Because they consult on a selected, more injured set of minor TBI patients, neurosurgeons often advocate liberal CT scanning of most patients with minor TBI with a history of LOC (duration not clearly defined) or with amnesia for the traumatic event.¹⁵ Others advocate only hospital observation because the yield of initial abnormal scans requiring acute neurosurgical intervention is low, but patients who do deteriorate after minor head trauma have substantial morbidity and mortality. If resources allow, prolonged emergency department observation may be practical in some circumstances. For example, intoxicated patients with minor TBI who otherwise fulfill low-risk criteria should undergo meticulous serial evaluations in the emergency department until clinical sobriety is achieved. In these patients, a CT scan may be unnecessary, and observation is beneficial.

The most practical approach regarding imaging in the emergency department patient is probably selective CT scanning or observation based on risk stratification of the minor TBI patient. If the low-risk patient is fully awake and not intoxicated, has no focal neurologic findings, has no clinical evidence of skull fracture, and can be kept under competent observation for 12 to 24 hours, neuroimaging is usually not indicated. Patients with moderate-risk minor head trauma (see Box 38-3) should probably undergo CT scanning or prolonged emergency department observation. Studies have prospectively identified and validated high-risk criteria for adult patients with minor head trauma that correlate with increased likelihood of intracranial lesions. These include the presence of a headache, vomiting, age older than 60 years, drug or alcohol intoxication, short-term memory deficits, external signs of trauma above the clavicles, and post-traumatic seizures.^{71,72} A CT scan should therefore be considered for patients with these high-risk findings as well as the other criteria listed in Box 38-3.

Skull radiography after head trauma in adults has been largely replaced by more sophisticated imaging, when imaging is performed. Facial, scalp, or external signs of head trauma by themselves do not predict TBI and are not indications for screening skull radiographs. The presence of a skull fracture suggests significant impact to the head, and therefore an increased likelihood of TBI, but the absence of a skull fracture does not rule out TBI. Patients with clinical signs of skull fracture have a substantially increased incidence of intracranial lesions associated with their minor head trauma. When the clinical examination shows evidence of skull fractures, CT scanning should be obtained, forgoing plain radiographs.

Although CT scanning is extremely sensitive for acute blood, MRI is more sensitive than CT for detecting diffuse axonal injury, ischemia after TBI, and some hemorrhagic lesions, especially those located at the base of the skull or in the posterior fossa. Functional imaging, such as positron emission tomography (PET), can provide information on the metabolic and neurochemical state of the injured brain. Many studies suggest that significant long-term neuropsychiatric sequelae after minor head trauma can occur despite an initial negative head CT scan, and these may be related to lesions seen initially only by MRI or functional imaging.¹⁴ Functional imaging is not currently within the scope of emergency assessment of minor TBI patients, but it can direct rehabilitation strategies for the small subset of patients who suffer significant morbidity after minor TBI.

Ancillary Studies

No routine laboratory tests are needed for patients with isolated minor head trauma. A urine toxicology screen and blood

alcohol level may be useful in interpreting the patient's mental status. Alcohol can affect the GCS, but this effect is not observed until the blood alcohol concentration is greater than 200 mg/dL; until that level, changes in mental status cannot be explained solely by acute alcohol intoxication.⁶⁸

A number of CNS biomarkers, such as S-100B, neuron-specific enolase, myelin basic protein, cleaved tau, and creatine kinase isoenzyme BB, have been investigated in minor TBI. None of these have been shown to strongly correlate with long-term outcome, and only S-100 B predicts abnormal CT findings in minor TBI. However, S-100B appears to lack CNS specificity and is often elevated in multiple trauma patients with no head injury.¹⁴ To date, serum biomarkers have lacked the precision needed for meaningful application in the emergency setting.

Disposition

Most patients with low-risk minor TBI can be discharged from the emergency department after a normal examination and a reasonable period of observation (i.e., 4–6 hours).²⁸ If the emergency physician decides that the patient with moderate- or high-risk minor TBI can be sent home, an appropriate early follow-up should be arranged. Patients should be discharged with instructions describing the signs and symptoms of delayed complications of head injury, should have access to a telephone, and should be monitored in the acute post-trauma period by a responsible, sober adult. If any doubt exists regarding the safety of the discharged patient with minor head injury, a brief inpatient observation period (i.e., 12–24 hours) is advisable.

If a patient with minor head trauma returns to the emergency department because of persistent symptoms, delayed complications of minor head injury should be sought. If a CT scan was not performed, the intensity of symptoms may guide the decision to obtain a CT scan at the second visit. If a negative scan was initially obtained, the decision to rescan is more complex. The literature about repeat scanning in minor TBI is scant; however, in one systematic review of severe and moderate TBI, progression of lesions most commonly correlated with overall injury severity and the patients' use of anticoagulants.⁷³

Concussion. A concussion (or complicated minor TBI) is a type of minor TBI usually caused by acceleration-deceleration or rotational injury to a freely mobile head, and it is most commonly associated with collision sports.⁷⁴ The injury results in distortion of axons, vasculature, and brain neuroanatomy. As in other types of minor TBI, acute CT or MRI abnormalities are not usually found, but functional imaging (i.e., PET) reveals abnormalities with glucose uptake and blood flow.^{14,74,75} Levels of neurotransmitters remain elevated, and a hypermetabolic state may persist in the brain for several days to weeks after the initial injury.

Like patients with minor TBI, concussed patients frequently complain of headaches, dizziness, confusion, and amnesia for the traumatic event and have nonfocal neurologic examinations. Patients with concussion have more severe and persistent symptoms than those with uncomplicated minor TBI. In young children, acute symptoms of concussion differ from those in adults and may include restlessness, lethargy, confusion, or irritability. On presentation, concussed children may be vomiting, tachycardic, or appear pale. These signs and symptoms are usually completely resolved by 6 hours.

Approximately 300,000 yearly sports-related concussions are reported to the Centers for Disease Control and Prevention.⁷⁶ A study of concussed football players showed acute symptoms

lasting at least 5 days, cognitive impairments lasting 5 to 7 days, and balance deficits lasting 3 to 5 days after concussion; 91% were back to their preinjury baseline by 7 days, but some had deficits on verbal fluency tests as long as 90 days after injury.⁷⁶

The demonstrated period of neurodysfunction and the delayed return to cognitive and physical baseline that follows concussive impact have led to the development of several scoring systems to grade severity of concussions with the goal of determining when it is safe for an athlete to return to play. No single set of guidelines has emerged that has been universally accepted, but all are predicated on the concern about a period of vulnerability following impact. Football players sustaining concussion appear susceptible to an additional concussion, with the majority of reinjury occurring within 10 days after the first injury. This is variably attributed to balance defects, delayed reflexes, delayed speed of information processing, or simply because of continued exposure to collision sports.^{75,76}

The *second impact syndrome* (SIS) occurs when an athlete sustains a second concussion before being completely asymptomatic from the first and then experiences a rapid, usually fatal, neurologic decline. It is postulated that persistent neurochemical disruptions and altered autoregulatory mechanisms after a first injury make the brain particularly vulnerable to marked brain swelling and subsequent herniation after a seemingly minor second impact. Although the existence and frequency of the SIS are controversial and debated in the sports medicine literature, its serious implications affect subsequent management decisions regarding head-injured athletes and others with concussion.^{67,74,75} All current recommendations for return to play after a sports-related concussion state that players with concussion should not return to play for at least 1 week after they have become asymptomatic. This is usually increased to at least a symptom-free month if an LOC or prolonged post-traumatic amnesia occurred at the time of concussion.

Almost all patients with minor TBI will have rapid and complete resolution of their symptoms,⁷⁷ and there is currently no good evidence that uncomplicated minor TBI leads to long-term sequela.⁶⁸ However, a subset of patients with concussion report symptoms that persist for long periods after trauma. These persistent symptoms are called the postconcussive syndrome (PCS). The incidence of PCS is reported to be 10 to 25%,^{14,68,78} but these estimates are based on inpatient studies and are therefore likely to be high.

The most common delayed or persistent postconcussive complaints are headache, sensory sensitivity, memory or concentration difficulties, irritability, sleep disturbances, and depression. It was long believed that persistent postconcussive symptoms were psychosomatic, litigious, or factitious, and it is likely that the manifestation and expression of PCS depend on factors in addition to injury severity, such as preinjury physical and mental health.⁶⁸ However, the presence of abnormalities on functional imaging and with sophisticated neuropsychometric testing suggests a pathophysiologic basis for PCS.^{14,79} Studies of concussed athletes (for whom preinjury baseline data are available) show that the cognitive domain most frequently involved in PCS is memory.¹⁴ Dizziness occurring early after trauma is associated with a prolonged PCS.⁷²

Management and Disposition

The management decisions faced by the emergency physician are the same as those addressed when evaluating all patients with apparently minor TBI: extent of workup to initiate in the

emergency department and whether the patient can be safely discharged home. Emergency department patients who have a sports-related concussion should probably not be allowed to return to play from the emergency department; current recommendations are for a gradual progressive return to play.¹³ Many authorities suggest follow-up at 1 week to determine the duration of symptoms and when the patient can safely return to sports. The possibility of PCS should be considered in all concussed patients, regardless of their initially benign presentation, and it may be prudent to suggest scheduled primary care follow-up for reassessment if symptoms persist.

■ PEDIATRIC HEAD INJURIES

Epidemiology

Approximately 650,000 children ages 0 to 19 years are emergently evaluated yearly in the United States for head trauma, with approximately 65,000 hospital admissions and 7500 fatalities.¹ TBI accounts for the largest source of childhood mortality and morbidity after trauma. In children, transportation-related injuries or falls account for most head trauma. Child abuse is a common etiology of head injury in young children. Duhaime and colleagues⁸⁰ found that nearly 25% of head-injured children younger than 2 years of age had inflicted injuries. In head-injured children younger than 1 year of age, as many as 66% of all injuries and 95% of severe injuries may be nonaccidental.⁸¹

Pathophysiology

Until the cranial sutures close, children's skulls are more distensible than those of adults. As a result, young children may often sustain less TBI after head trauma than adults with comparable nonfatal mechanisms of injury.⁸² However, children appear to have an age-dependent brain vulnerability. Very young children (younger than 1 year) have higher mortality after head trauma than older children with the same severity of injury.⁸² Many factors contribute to this. Medical attention is often delayed in children with nonaccidental injuries. Because of limited language and comprehension, an accurate formal neurologic examination in young children is sometimes difficult. Medical personnel tend to underestimate the extent of the injuries in small children and are often reluctant to initiate invasive procedures that may be necessary to aid in the diagnostic workup, such as IV access for sedation in CT scanning.

The types of TBI sustained after head trauma in children differ from those in adults. Children have fewer traumatic mass lesions (with the exception of subdural hematomas in the very young), fewer hemorrhagic contusions, more diffuse brain swelling, and more diffuse axonal injury.⁸³ Of head-injured patients younger than 20 years of age who talk and deteriorate, 39% have brain swelling only (i.e., no mass lesions), whereas 87% of patients older than 40 who talk and deteriorate have mass lesions.⁶⁰

Clinical Features

As with adults, an accurate description of the mechanism of injury, the appearance of the child immediately before and after the injury, and subsequent events can provide useful information to assist in the evaluation and management of the acutely head-injured child.

In principle, the acute neurologic assessment of head-injured children is the same as that of adults. Pupillary respon-

siveness and size, corneal reflexes, the presence of a gag or cough reflex, and spontaneous motor movements provide baseline neurologic information that can be followed over time. Additional neurologic assessment can be more challenging. Because of its reliance on developed language skills and the patient's attention and cooperation, the GCS is difficult to apply to children younger than 5 years of age. Modified scales have been developed, but none has been rigorously validated; as yet, no universally accepted coma scale exists for children.⁸² Mental status changes, which may be the first symptom of head injury, are difficult to evaluate in children, and often the head-injured toddler has no deficit except irritability.

Children with severe or moderate head injury are clinically similar to adults with these degrees of injury, although children have an increased incidence of post-traumatic seizures after severe head injury. Infants appear to be at especially high risk for post-traumatic seizures.⁸⁴ Overall, up to 6% of all head-injured children and 35% of severely head-injured children have early (i.e., within the first week) post-traumatic seizures.⁸² Most seizures occur within the first 24 hours and do not predict seizures later in the post-traumatic period. However, early post-traumatic seizures can exacerbate secondary brain injury. Although prophylactic use of anticonvulsants is not recommended to prevent late post-traumatic seizures, acute anticonvulsant prophylaxis is a treatment option in severely head-injured children to prevent early post-traumatic seizures.⁴⁴

Children who sustain minor head trauma often have more pronounced physical signs and symptoms than adults. Despite apparently trivial trauma, children may appear pale, be lethargic, and have frequent emesis and complaints of headaches and dizziness. Concussive injuries in children produce two unique clinical circumstances. Many children experience a brief *impact seizure* at the time of relatively minor head injury. By the time the child is evaluated, he or she is at baseline neurologic function. Impact seizures do not appear to predict subsequent early post-traumatic seizures. However, a post-traumatic impact seizure may confuse the initial assessment of the severity of the head injury and prompt a more aggressive workup than needed. *Postconcussive blindness*, another serious complication of concussive injuries in children, is usually associated with impact to the back of the head. Children experience a temporary loss of vision that can persist from minutes to hours before normal vision returns.

The clinical presentation of post-traumatic *intracranial lesions* in infants can be extremely subtle, especially in those younger than age 6 months. Often, these lesions are associated with scalp injuries and skull fractures but no other symptoms.^{82,85} Toddlers are also difficult to assess; the only symptom of an intracranial injury may be irritability. Recent guidelines for the management of head injury in children younger than 2 years of age allow risk stratification and are summarized in [Box 38-4](#).^{85,86} Recommendations are also offered regarding the emergency department workup in these very young children.

Inflicted head injury is the most common cause of head injury deaths in infants.⁸⁷ Child abuse must be considered in all children with unexplained head injuries or injuries not consistent with the history provided. For example, children who experience low-energy mechanisms of injury (i.e., a short-distance fall) seldom sustain skull fractures; a finding of a fracture should therefore prompt consideration of abuse.⁸⁸ The *shaken baby syndrome* involves an acceleration-deceleration brain injury, with the moving brain striking against the interior of the skull. This occurs most often in children younger than age 1 year. Patients present with a broad range of symptoms,

BOX 38-4

MINOR HEAD TRAUMA RISK STRATIFICATION IN CHILDREN YOUNGER THAN AGE 2 YEARS^{85,86}**High Risk: CT Recommended for All**

Decreased mental status
Focal neurologic findings
Signs of depressed or basilar skull fractures
Acute skull fracture by clinical examination or skull radiographs (if already obtained)
Irritability
Bulging fontanel
Seizure
Vomiting (5 or more times)
Age <3 months
LOC >1 min

Intermediate Risk*CT scan or observation recommended*

3 or 4 episodes of vomiting
Transient LOC (<1 min)
History of lethargy or irritability, now resolved
Behavior not at baseline
Nonacute skull fracture (injury more than 24 hr old)

Either CT or SR or observation recommended

High-force mechanism
Fall onto a hard surface
Scalp hematoma
Unwitnessed trauma
Vague history with physical signs of trauma

Low Risk: Observation Recommended

Low-energy mechanism with no signs and symptoms 2 hr after trauma

CT, computed tomography; LOC, loss of consciousness; SR, skull radiograph.

from nonspecific complaints to seizures and coma. Classically described findings include retinal hemorrhages, subdural hematomas, subarachnoid blood, and no signs of external trauma.⁸⁸

Diagnosis and Management

As with adults, pediatric head-injured patients should be assessed for other major trauma and assumed to have cervical spine injury until proved otherwise. Goals of emergency department management are also generally the same: to prevent secondary injury and systemic insults, to prevent increasing ICP, and to detect traumatic mass lesions requiring emergency surgery.

Children with severe TBI who are transported directly to a trauma center have better outcomes than those transported to nontrauma hospitals.⁴⁴ It is essential that hypoxia be avoided to decrease the likelihood of secondary brain injury. Hypoxia and hypoventilation are common in pediatric head-injured patients, and they develop more rapidly in children than in adults. In children with GCS less than 8, airway management should be aggressive to prevent hypoxia, aspiration, and hypercarbia and to allow mild hyperventilation if the patient has signs of herniation. There is no evidence to support the advantage of out-of-hospital intubation over bag-valve-mask ventilation of pediatric patients with TBI; scene time should not be prolonged with multiple attempts at endotracheal intubation.⁴⁴

Hypotension should be identified and corrected as soon as possible to ensure adequate cerebral perfusion pressure. There

is no evidence to suggest that aggressive fluid resuscitation exacerbates cerebral edema in head-injured children. Occult blood loss from multiple trauma should be considered as a possible cause of hypoperfusion. Spinal injury causing shock should also be considered. In children, unlike adults, hypovolemic hypotension can occur because of head trauma. Hypotension from intracranial bleeding can occur in children younger than 1 year of age with a large linear skull fracture and an underlying large epidural hematoma.⁸⁷ The intracranial blood can seep through the fracture and produce a large galeal or subperiosteal hematoma. Hypotension from intracranial bleeding can also occur in a child with hydrocephalus and a functioning shunt. Blood may accumulate without much evidence of increased ICP. Scalp lacerations can also produce significant hemorrhage and subsequent hypotension.

Up to 80% of children with severe head trauma have elevated ICP.⁸² In infants, a bulging fontanel suggests elevated ICP. Other signs of elevated ICP include bradycardia, papilledema, declining level of consciousness, and seizures. When increased ICP is indicated by physical examination, methods to reduce ICP should be initiated. As with adults, acute hyperventilation has immediate effects but is never indicated for prophylaxis or for prolonged management of increased ICP.⁴⁴ Hyperosmolar therapy is effective at reducing ICP. Although efficacy studies were based on adults, mannitol at doses of 0.25 to 0.5 g/kg has become a mainstay in the treatment of elevated ICP in children with severe TBI. Several studies support the effectiveness of hypertonic saline in lieu of mannitol for elevated ICP in pediatric head-injured patients. Treatment is with a continuous infusion of 3% normal saline at between 0.1 and 1.0 mL/kg body weight, titrated to effect.¹⁸ Severely head-injured children are less likely to have a surgically amenable lesion than adults. Because diffuse brain swelling is the most common finding in severely head-injured children, emergency burr holes are generally ineffective.

When considering minor head injury in children, it is important to differentiate children younger than age 2 years and older children. Children younger than age 2 years with traumatic brain injuries are often difficult to assess and may have subtle clinical findings. In general, the literature supports the conclusion that younger patients are at higher risk for intracranial injury.⁸⁹ One clinical sign of potential brain injury in children younger than age 2 years is the presence of a scalp hematoma, especially a large parietal scalp hematoma.¹ In an observational cohort study involving children with low risk for brain injuries, scalp hematomas were present in 93% of children 2 years old or younger who had brain injuries.⁹⁰

The CT scan is the diagnostic imaging modality of choice in the evaluation of moderate or severe pediatric TBI. It should also be strongly considered in pediatric patients with high-risk minor TBI.^{85,86,90} However, potential risks are associated with childhood exposure to radiation. Children are more sensitive to radiation than adults because of rapidly dividing cells occurring during growth. The younger the child, the more potential risk. Increased rates of cancer and decreased cognitive performance and academic development have been associated with radiation doses similar to that of a single head CT scan.⁸⁵ An additional safety concern is the frequent need to sedate young children in order to obtain an adequate imaging study. In these circumstances, the risks of radiation exposure and sedation should be weighed against the likelihood of an intracranial lesion in the child with minor head trauma.

Many attempts have been made to derive clinical prediction rules for high-risk minor TBI in children. However, unless there is both high sensitivity and high specificity, such rules may actually result in an increased number of CT scans being

performed. Clinical predictors for increased risk of intracranial lesions in children with minor TBI have been described and can help with risk stratification. A meta-analysis including more than 22,000 children suggested that the presence of a skull fracture, focal neurologic signs, documented loss of consciousness, and a presenting GCS score of less than 15 were statistically associated with TBI.^{91,92} Many authorities also include a history of protracted vomiting, abnormal mental status or lethargy, obvious scalp hematomas in children 2 years old or younger, and progressively worsening headache.⁸⁵

The use of skull radiographs in the diagnostic workup of head-injured children is controversial but may be appropriate in some circumstances. As with adults, when a CT scan is indicated, skull radiographs are not necessary. Up to 11% of children younger than age 2 years will sustain a skull fracture associated with head trauma, and 15 to 30% of these will have TBI. Therefore, in children younger than age 2 years, a skull fracture is a predictor of TBI.⁹³ The presence of a skull fracture in children significantly increases the likelihood of intracranial pathology; conversely, a negative radiographic skull screen does not guarantee the absence of TBI. Parietal skull fractures are the most common. Often, fractures occur in infants who sustain relatively minor head injury. Skull films may be useful as a screening tool in determining the need for a CT scan, especially in children 2 years old or younger whose neurologic examination is difficult to obtain and interpret. In alert children younger than age 2 years with minor head injury, a low-risk history, a normal physical examination that includes a normal neurologic and mental status examination appropriate for age, and a scalp hematoma, skull radiographs may be a useful screen.⁸⁵ If the skull radiograph is negative for a fracture, a CT scan may be unnecessary. If the skull radiograph shows a fracture, CT imaging is indicated.

In older children, skull radiographs are rarely useful unless a specific lesion is suspected, such as a depressed skull fracture or a penetrating foreign object. Skull fractures are more clinically significant in children than in adults. Fractures, especially complex stellate or multiple injuries, are often seen in abused children, and skull films should be obtained if abuse is suspected. *Ping-pong fractures* occur with concentrated forces that indent the skull. These fractures are unique to infants and appear as multiple indentations in the skull with no significant bone discontinuity. Skull fractures are common in children who have sustained deep scalp lacerations or who have a large scalp hematoma.

Leptomeningeal cysts or *growing skull fractures* are delayed complications of linear skull fractures in infancy. If a tear in the dura accompanies the linear fracture, the meninges may fill with CSF and prolapse through the fracture margins, thus preventing fracture healing.⁸⁵ These cysts can grow in size and have the potential to cause a mass effect. If a linear fracture is found by skull radiography, close follow-up is indicated to assess for this delayed complication.

Overall, children who sustain severe head injury have lower mortality and a better neurologic outcome than comparably injured adults. This is probably because of the neuroplasticity of the young brain; however, in children younger than age 2 years, the prognosis after severe head injury is poor.⁸⁹ Very young children have immature cerebrovascular autoregulation, which increases the risk of cerebral edema formation. The immature brain has increased susceptibility to permanent injury because of incomplete myelination.

The emergency evaluation of children with minor head injury is especially challenging, given their potentially dramatic presentation and the added difficulty of obtaining an accurate neurologic assessment. Evidence-based consensus guidelines have been proposed for the evaluation and manage-

BOX 38-5**RECOMMENDATIONS FOR MANAGEMENT OF MINOR CLOSED-HEAD TRAUMA IN CHILDREN****No LOC, Presenting GCS Score of 15**

Thorough history and PE

If low-risk traumatic mechanism and classification (see Box 38-3) and normal examination, observation (up to 24 hr) by competent caregiver

Brief LOC (<1 Min), Presenting GCS Score of 15

Thorough history and PE

If low-risk traumatic mechanism and classification (see Box 38-3) and normal examination, observation (up to 24 hr) by competent caregiver

Consider CT scanning if history and/or examination suggest high-risk minor head trauma (see Box 38-3). Skull films should be considered only if CT scanning not available

Presenting GCS Score of 14

Strong consideration for CT scanning

Imaging Recommended

Children <3 months old

Consider CT scanning unless asymptomatic, low-risk history and PE (see Box 38-3), no scalp hematoma, and trivial traumatic mechanism

Children 3 months to 2 years

Normal neurologic examination, no symptoms, no scalp hematoma: no imaging

Normal neurologic examination, no symptoms, scalp hematoma: skull radiographs

If skull films positive for fracture, follow with CT scanning
Abnormal neurologic examination, moderate- or high-risk history or PE findings (see Box 38-3): CT scanning

Children >2 years

Normal neurologic examination, no symptoms, no scalp hematoma: no imaging

Normal neurologic examination, symptoms, low- or moderate-risk history or PE findings (see Box 38-3): consideration of CT scanning

Abnormal neurologic examination and/or high-risk history or PE findings (see Box 38-3): CT scanning

CT, computed tomography; GCS, Glasgow Coma Scale; LOC, loss of consciousness; PE, physical examination.

ment of minor head injury in children.^{85,86} Disposition of pediatric patients with minor head injury is summarized in Box 38-5. Parents should be educated about the warning signs and symptoms of delayed complications of minor head trauma.

■ PENETRATING HEAD INJURIES**Epidemiology**

Penetrating brain injury (PBI) occurs at a rate of 12 per 100,000 population and can be sustained by missile injuries or impalement.⁹⁴ The United States has the highest penetrating head injury rate among developed countries in the world, with the most common cause being gunshot wounds (GSWs). These dramatic injuries are increasing in frequency, and the neuroscientific understanding of the complicated cerebral events that occur with penetrating head injury does not yet equal our understanding of the pathophysiology of blunt injuries.

Civilian GSWs to the head account for approximately 21,000 deaths per year, and up to 66% of all patients who sustain a GSW to the head are dead at the scene.⁹⁵ Overall, the mortality caused by a GSW to the head is estimated to be 90%.⁹⁶ If the patient is hemodynamically stable, has not sustained secondary systemic insults such as hypoxia or hypotension, has no expanding mass lesions from the missile injury, and has not ingested intoxicants that may interfere with assessment, prognosis after a GSW to the head can be predicted by the presenting GCS and pupillary responsiveness.⁹⁷ If the presenting GCS is less than 5, mortality approaches 100%. If the presenting GCS is greater than 8 and the pupils are reactive, survival approaches 75%. Survivors of GSW to the head tend to do well, with up to 60% returning to their former employment.⁹⁷

Pathophysiology

Missile injuries to the head can result in several different patterns of damage. *Tangential wounds* are caused by an impact that occurs at an oblique angle to the skull. If the missile has high velocity but low energy, it can travel around the skull under the scalp without passing through the skull. Intracranial damage, primarily cortical contusions, can occur at the initial site of impact because of pressure waves generated by the impact. In one study, 24% of patients with tangential GSWs also had intracranial hemorrhage, and 16% sustained skull fractures.⁹⁸ *Perforating wounds* are usually caused by high-velocity projectiles, which cause through-and-through injuries of the brain with an entrance and an exit wound. This type of injury is largely discussed within the context of military GSWs to the head. In cases of complete traversal (through-and-through) GSW to the head, the entrance wound is usually smaller than the exit wound.

Penetrating missile wounds are produced with moderate- to high-velocity projectiles discharged at close range. The majority of the civilian PBI literature deals with penetrating missile wounds.⁹⁶ The penetrating object may travel through the entire skull, bounce off the opposite inner table of the skull and ricochet within the brain, or stop somewhere within the cranial cavity. Bullets that penetrate the skull do not travel in a straight path. The wounding capacity of a firearm is related to the kinetic energy of its missile on impact and how much energy is dissipated in the tissues. Low-velocity missiles tend to be deflected by intracranial structures. The final track is therefore erratic and occasionally bears no relation to the exit or entrance site of the missile. High-velocity missiles can project straight through the tissues and easily fracture bones. Flight stability and the angle at which the bullet strikes its target affect the path through the brain. Within tissue, destabilizing motions include deviation of the longitudinal axis of the bullet from a straight line (yaw), forward rotation of the bullet around its center of mass (tumbling), and oscillatory motion of the bullet axis around its center of mass (rotation). As the bullet passes through the brain, a tissue cavity is created. This cavity can be as much as 10 times the diameter of the missile. A percussion shock wave is also created, lasting 2 msec but causing little tissue destruction.⁹⁶

The morbidity and mortality from missile injuries to the head depend on the intracranial path, speed of entry, and the size and type of the penetrating object. Projectiles that cross the midline or the geographic center of the brain, pass through the ventricles, or come to rest in the posterior fossa are associated with extremely high mortality.⁹⁴ High-velocity wounds are associated with greater mortality than low-velocity injuries. Large missiles or missiles that fragment within the cranial vault are usually fatal. The design of the bullet and its fragmentation potential (capacity to deform or fragment) also con-

tribute to final tissue destruction and patients' morbidity and mortality.

Many GSWs to the head are intentionally self-inflicted injuries. The percentage of penetrating head injuries caused by self-inflicted GSWs ranges from 13 to 88%.⁹⁷ Characteristics of self-inflicted GSWs include injury on the dominant side, powder burns at the entrance site, and large stellate scalp lacerations caused by dissection of the subgaleal layer by exploding gases released close to the scalp. In suicide attempts, GSWs to the head tend to traverse the midline in the coronal plane and often involve major vascular structures. If the self-inflicted GSW has an entrance through the mouth, injury to the hard palate may occur with potential upper airway compromise. The careful aim and close range of self-inflicted GSWs to the head make these injuries particularly devastating; mortality is higher than with non-self-inflicted penetrating injuries, and odds ratios of death vary between 1.63 and 5.83.⁹⁷

Clinical Features

Physical assessment of the patient with a missile wound to the head focuses on the presenting GCS and pupillary responsiveness. In addition to the physical damage to brain tissue caused by the penetrating injury, other devastating physiologic changes occur immediately after injury. ICP increases, the BBB breaks down, CBF is altered, and cerebral edema develops. Cerebral autoregulation is lost, and CPP may fall.

Management

The emergency department management is directed at reducing the occurrence of the secondary systemic insults of hypoxia and ischemia, with emergent intervention if signs of herniation syndrome develop and the patient is viable. Management should be aggressive until the prognosis can be established by examination and neuroimaging data. When penetration of the cranial vault is established, the patient should be intubated. If the physician waits for coma before intubating the patient, mortality approaches 100%.⁹⁹

Emergency treatment should include IV antibiotics because penetrating missiles are contaminated with skin, bone, and hair. Tissue contamination may be widespread because of the cavitation caused by the missile as it passes through the brain.^{100,101} Approximately 90% of all CNS infections associated with penetrating TBI injuries occur within 6 weeks. Most neurosurgeons do not automatically débride bone and missile fragments due to a growing body of literature that shows no evidence that removal of these foreign bodies decreases infection rate.¹⁵

Between 30 and 50% of patients with PBI develop seizures; 10% of these occur in the first week.¹⁵ Anticonvulsants should be given in the acute setting to prevent early post-traumatic seizures in the patient with PBI, especially if the patient is to be transported to another institution after acute stabilization. Anticonvulsants should not be given beyond the first week after PBI because this has not been shown to prevent the development of late seizures.

Skull radiographs may be useful in determining the number of penetrating fragments and their track. A CT scan defines the precise location of the missile, its intracranial path, the presence of bone or missile fragments, extra-axial or intracerebral blood collections or other traumatic lesions, and pneumocephalus. CT scanning is the radiologic test of choice for PBI.¹⁰¹ Pneumocephalus is often associated with missile wounds that penetrate the sinuses but can be caused by free air sucked into the penetration cavity behind the projectile.

When a penetrating head injury is caused by *impalement*, the penetrating object should be left in place to be removed at surgery. A skull radiograph shows the size of the object, the angle of impalement, and the depth of penetration. Angiography may be indicated to better discern location referable to key vascular structures.

■ COMPLICATIONS AFTER HEAD INJURY

Neurologic Complications

Seizures

Post-traumatic seizures are relatively common in the acute or subacute period. Acute post-traumatic seizures are usually brief and are probably caused by transient mechanical and neurochemical changes within the brain. After the acute seizure, the patient often has no additional seizure activity. In the subacute period, 24 to 48 hours after trauma, seizures are caused by worsening cerebral edema, small hemorrhages, or penetrating injuries. Post-traumatic seizures are common in children and can be precipitated by relatively minor head injury.⁸⁴ Acute post-traumatic seizure prophylaxis in the emergency department is recommended for some head-injured patients even if they have not had a seizure (see Box 38-2).^{53,102} This is especially important in patients who will have neuromuscular blockade to facilitate management or transfer because the clinical manifestations of seizures are lost in these patients. Phenytoin (18–20 mg/kg) is used as a first-line agent for prophylaxis. The decision to maintain the head-injured patient on long-term anticonvulsant therapy during the recovery period depends on the patient's subsequent course. Long-term seizure prophylaxis is not indicated for all patients who have had post-traumatic seizures in the acute or subacute period. The utility of prophylactic anticonvulsants to prevent late post-traumatic seizures has not been proved, and their use is not recommended.¹⁰²

Central Nervous System Infections

Meningitis after Basilar Fractures. Post-traumatic meningitis is caused by a variety of microbes, depending on the portal of bacterial entry. Patients present with typical signs and symptoms of meningitis, including fever, altered mental status, and occasional focal neurologic signs. In patients with a CSF leak after basilar fracture, early meningitis (i.e., within 3 days of injury) is usually caused by pneumococci. Ceftriaxone or cefotaxime is a reasonable antibiotic choice with the addition of vancomycin. Gram-negative organisms often cause meningitis that develops more than 3 days after trauma.¹⁰³ A third-generation cephalosporin, with nafcillin or vancomycin added to ensure coverage of *Staphylococcus aureus*, should be started. In children, post-traumatic meningitis may be caused by *Haemophilus influenzae*. Prophylactic antibiotics are not currently recommended in the acute setting in patients with CSF leaks caused by basilar skull fractures.¹⁰³

Brain Abscess. Brain abscesses develop infrequently after penetrating missile injuries to the head. Abscesses can also develop after open depressed skull fractures if bone fragments are not removed or as a postoperative complication. Post-traumatic CSF fistulae and fractures that disrupt air-filled sinuses predispose to the formation of brain abscesses. Clinical manifestations include headaches, nausea, vomiting, declining mental status, signs of increased ICP, or new focal neurologic findings in patients who had been improving after trauma. Occasionally, nuchal rigidity, hemiparesis, or seizures may be present. Systemic signs are often subtle, and CSF leukocytosis may be absent.

Contrast-enhanced CT scanning makes the diagnosis of brain abscess. A ring pattern with a low-density center is characteristic. The enhanced ring represents surrounding altered vascular permeability and therefore is also seen in the cerebritis stage early in abscess formation. Lumbar puncture is often not helpful and should not be performed in the patient with signs of increased ICP (e.g., headache, vomiting, and papilledema).

The treatment of brain abscess is usually operative drainage. The patient with cerebritis may respond to IV antibiotics but requires close monitoring with repeated CT scans. Common organisms isolated from post-traumatic abscesses are *S. aureus* and gram-negative aerobes.¹⁰³

Cranial Osteomyelitis. Cranial osteomyelitis can occur after penetrating injury to the skull. The clinical manifestations include pain, tenderness, swelling, and warmth at the infected site. More than 50% of cases are obvious on plain skull radiographs.¹⁰³ Technetium bone scans can help in the diagnosis when the skull radiographs are negative, but false-positive bone scans occur in patients with previous trauma or craniotomy. Adding a gallium scan helps to differentiate infection from other causes of a positive technetium scan. Patients with post-traumatic cranial osteomyelitis require surgical débridement and removal of the infected bone. Antibiotic choice is determined by culture results. If systemic symptoms are present, an underlying subdural or epidural empyema is often present.

Medical Complications

Disseminated Intravascular Coagulation

The injured brain is a source of tissue thromboplastin that activates the extrinsic clotting system. Disseminated intravascular coagulation (DIC) can develop within hours after any injury disrupting brain tissue. It has been detected in nearly all patients with severe TBI.⁶⁵ DIC increases morbidity and mortality after severe head trauma as well as the risk of delayed intracranial hemorrhage. If a stable patient with DIC suddenly deteriorates, a repeat CT scan should be obtained to rule out hemorrhage.

The extent of tissue destruction determines the degree of DIC that develops. The diagnosis is based on abnormalities in international normalized ratio, prothrombin and partial thromboplastin time, platelets, plasma fibrinogen levels, and fibrin degradation products. Patients with coagulopathy or abnormal platelet function require interventions to correct these.

Neurogenic Pulmonary Edema

Neurogenic pulmonary edema can develop from minutes to days after head trauma. This noncardiac pulmonary edema probably results from altered hydrostatic forces and microvascular permeability directly caused by brain injury. Lowering the ICP appears to reverse the neurogenic stimulation that causes this edema.¹⁰⁴

Cardiac Dysfunction

A variety of cardiac rhythm, rate, and conduction abnormalities are detected after head injury. These abnormalities can be life threatening and require aggressive therapy. In addition, adequate cardiac output is essential in head-injured patients to ensure cerebral perfusion. Many head-injured patients with cardiac dysfunction have concurrent myocardial injury from underlying disease or from chest injury. However, brain injury

can cause primary cardiac dysfunction. Cardiac rhythm abnormalities have been reported in up to 70% of all patients with subarachnoid hemorrhage (SAH) and more than 50% of all patients with intracranial hemorrhage.²⁸ In SAH, the cardiac dysrhythmias may result from autonomic nervous system dysfunction that subsequently affects ventricular polarization. High levels of circulating catecholamines have been measured in head-injured patients, with increased sympathetic nervous system activation.²⁸

The most common cardiac dysrhythmia after head injury is *supraventricular tachycardia*, but many other rhythms have been observed. Findings on the electrocardiogram include diffuse large upright or inverted T waves, prolonged QT intervals, ST segment depression or elevation, and U waves. The primary goal in the emergency management of cardiac dysfunction after head trauma is ensuring adequate tissue perfusion and avoiding hypoxia. Dysrhythmias in head-injured patients often resolve as ICP is reduced.²⁸ Standard advanced cardiac life support protocols should then be used because concurrent cardiac injury may also be present in multiply traumatized patients.

■ SPECIFIC INJURIES

Scalp Wounds

Scalp lacerations are extremely common after head injury and may be a source of significant bleeding because hemostasis may be difficult to achieve. Methods include direct digital compression of the bleeding vessel against the skull, infiltration of the wound edges with lidocaine with epinephrine, and ligation of identified bleeding vessels. If the galea is lacerated, it can be pulled up with a clamp and its edges folded over the lacerated skin edges to tamponade the bleeding vessels. Raney scalp clips applied to the edges of the wound are also effective. In stable patients, quick closure of the wound, after proper débridement and irrigation, is the most effective way to stop a bleeding scalp laceration and prevent the tissue crush injury that may occur if other compressive methods are used for too long. Obviously, in unstable patients, higher priority interventions take precedence over wound care.

When hemostasis is obtained, the wound should be irrigated to rinse away any debris. The emissary vessels of the subgaleal layer of the scalp drain directly into the diploe veins of the skull. These in turn drain into the venous sinuses. Contaminated or infected scalp wounds therefore have the potential to cause serious intracranial infections. Blood clots and other debris should be removed and the galea and underlying cranium palpated to detect any remaining debris, disruptions, or bone step-offs. Shear injuries to the scalp may deposit contaminants at sites distant from the apparent injury. The complexity of stellate lacerations often interferes with thorough inspection and débridement; stellate lacerations are particularly susceptible to infection. Digital exploration of a scalp wound should be carefully performed; if it is done too vigorously, comminuted or depressed bone pieces may be depressed further, and the examiner's finger is at risk for laceration.

It is easy to confuse a disruption in the galea or a tear in the periosteum with a skull fracture. The base of the laceration should therefore be directly visualized. Clipping away a small area of hair parallel to the edges of the wound may facilitate this. Alternatively, an antibiotic ointment can be applied to the hair immediately surrounding the wound and used to plaster the hair away from the injury site. If the laceration begins on the forehead and extends upward beyond the hairline, surrounding hair should not be removed. Removal obliterates a

useful landmark for cosmetic closure and may result in malalignment of the two laceration edges. If hair is accidentally embedded within the repaired laceration, it can delay healing by producing an inflammatory reaction or by serving as a nidus of infection.

Disruption of the galea usually results in a gaping scalp laceration. Large lacerations of the galea must be closed to prevent the edges of the wound from pulling apart as the muscles within the galea contract. The skin, dermis, and galea can usually be repaired in a single layer with interrupted or vertical mattress sutures of 3-0 nylon or polypropylene.¹⁰⁵ In scalp lacerations in which the galea is not involved, staples can be used in the repair.

Because of the rich blood supply of the scalp, even very large scalp avulsions can survive. If the avulsion remains attached to the rest of the scalp by a tissue bridge, it should be reattached to the surrounding tissue. If the avulsion is completely detached from the scalp, it should be treated as any other amputated part and reimplanted as soon as possible.

Scalp abrasions are often contaminated with pieces of dirt or other debris. The wound should be cleaned as thoroughly as possible and inspected for puncture wounds or other areas that penetrate beyond the superficial layers of the skin to ensure the removal of unsuspected foreign bodies. A careful inspection often reveals a small scalp laceration within the abraded area. Antibiotics are usually not needed for carefully managed scalp wounds because rapid healing is facilitated by the rich blood supply of the scalp.

Skull Fractures

Clinical Assessment and Significance

Skull fractures are local injuries caused by direct impact to the skull. The presence of a skull fracture does not always indicate underlying brain injury. However, the force required to fracture the skull is substantial, and all cases of skull fracture must be carefully evaluated to ensure that no additional injury is present. With increasing severity of head injury the likelihood of skull fracture increases, and the presence of a skull fracture after trauma increases the likelihood of having a TBI. It is often difficult to predict the presence of a skull fracture by clinical examination, and if this can be done, it is likely that substantial underlying brain injury is also present. The pattern, extent, and type of skull fracture depend on the force of the impact applied and the ratio of the impact force to the impact area. The fracture usually starts at the point of maximum impact.

Clinically significant skull fractures result in intracranial air and pass through an air-filled space (e.g., sinus), are associated with an overlying scalp laceration (open skull fracture), are depressed below the level of the skull's inner table, or overlie a major dural venous sinus or the middle meningeal artery.¹⁰⁶

Plain radiographs are most useful in demonstrating a depressed skull fracture, the depth and extent of a penetrating injury, or the presence of an intracranial foreign body. A CT scan with bone windows also demonstrates these findings; therefore, patients undergoing CT do not require skull radiographs.

Linear Fractures

A linear skull fracture is a single fracture that goes through the entire thickness of the skull. Linear skull fractures are clinically important if they cross the middle meningeal groove or

major venous dural sinuses; they can disrupt these vascular structures and cause the formation of epidural hematomas. Most other linear skull fractures are not clinically significant.

It is sometimes difficult to distinguish linear skull fractures demonstrated on radiographs from cranial sutures. In general, fractures are more lucent than vascular grooves and sutures. Sutures are usually less than 2 mm wide in adults; fractures are often 3 mm or greater in overall width and tend to be widest in the midportion and narrow at each end.¹⁰⁶ Linear fractures are most common in the temporoparietal, frontal, and occipital regions of the skull and can usually be visualized on more than one radiographic view. In children, skull fractures heal within 3 to 6 months; in adults, complete healing may take up to 3 years.¹⁰⁶

Sutural diastasis is the traumatic disruption of a cranial suture. In adults, sutural diastasis often involves the coronal or lambdoid sutures. Sutural diastasis usually occurs when a linear fracture extends into the suture line, and it is rare after sutures have undergone bone fusion.

Comminuted skull fractures are multiple linear fractures that radiate from the impact site. Usually, this injury suggests a more severe blow to the head than that producing a single linear fracture.

A linear *vault* fracture substantially increases the risk of intracranial injury. If any skull fracture is detected, a CT scan should be obtained, and the patient should be carefully observed for delayed complications of head trauma.

Depressed Fractures

Depressed skull fractures are clinically important because they predispose to significant underlying brain injury and to complications of head trauma, such as infection and seizures. When a depressed fracture occurs, traumatic impact drives the bone piece below the plane of the skull. The edges of the depressed portion of skull may become locked underneath the adjacent intact bone and fail to rebound into their previous position. As a result, the depressed piece of bone can penetrate tissue and lacerate the dura. Depressed skull fractures are usually caused by direct impact injury with small blunt objects, such as a hammer or a baseball bat. Most depressed skull fractures occur over the parietal or temporal regions. If the free piece of bone is depressed deeper than the adjacent inner table of the skull, most neurosurgeons consider this injury significant enough to require surgical elevation.

On skull radiographs, depressed fractures may be difficult to visualize. The free piece of bone demonstrates increased or double density because it often overlaps the nonfractured bone or it is viewed relatively rotated from the rest of the adjacent cranium. Tangential views of the skull may increase the ability to visualize the fracture.

Depressed skull fractures can often be felt with palpation of the skull beneath a scalp laceration. This examination should be done cautiously to avoid driving a depressed bone fragment deeper into the cranial tissue. The clinical examination for a depressed skull fracture may be misleading. The mobility of the scalp can result in nonalignment of the fracture with an overlying scalp laceration. As a result, the skull underlying the laceration may be normal, with the depressed area several centimeters away. Scalp swelling may also interfere with physical examination findings and hide any palpable bone defects. The signs and symptoms of a depressed skull fracture depend on the depth of depression of the free bone piece. Approximately 25% of patients sustaining a depressed skull fracture report LOC.¹⁰⁶ Neurologic deficits may be present, depending on the extent of underlying brain tissue injury.

A CT scan is indicated for patients with a history or physical examination that suggests a depressed skull fracture. The CT scan should include bone windows to determine the depth of depression and the presence of concurrent traumatic intracranial lesions. Patients with depressed skull fractures should be admitted for continued observation by a neurosurgeon.

Depressed skull fractures may increase the risk for developing seizures. Emergency department patients suspected of having a depressed skull fracture warrant prophylaxis for post-traumatic seizures, especially if they have an altered level of consciousness or require chemical paralysis. Depressed skull fractures may also increase the risk for meningitis.¹⁰⁶

Basilar Fractures

Basilar fractures are linear fractures at the base of the skull. The fracture usually occurs through the temporal bone, with bleeding into the middle ear producing hemotympanum. Often, the fracture has caused a dural tear, which produces a communication between the subarachnoid space, the paranasal sinuses, and the middle ear. This offers a route for the introduction of infection into the cranial cavity and is suggested by a CSF leak.¹⁰³ As with linear skull fractures, a basilar fracture is not always associated with significant underlying brain injury; these fractures are the result of considerable impact force, however, and TBI must be ruled out.

Basilar fractures can compress and entrap the cranial nerves that pass through the basal foramina, can dislocate the bones of the auricular chain, and can disrupt the otic canal or cavernous sinuses, with subsequent injury to cranial nerves III, IV, and V. Fractures of the sphenoid bone can disrupt the intracavernous internal carotid artery, creating the potential for the formation of pseudoaneurysms or carotid venous fistulae. The diagnosis of a basilar skull fracture is based on associated clinical signs and symptoms (see [Box 38-1](#)).

Skull radiographs do not detect basilar fractures well. All patients with clinical evidence suggesting a basilar skull fracture should have a CT scan to define the fracture and to rule out concurrent intracranial pathology, and they should be admitted for observation. Because the basilar skull fracture may afford an entrance for bacteria, antibiotics are often considered. However, most CSF leaks resolve spontaneously with no complications in 1 week, and antibiotics generally are not given prophylactically during the first week of CSF rhinorrhea. If a patient with a previously diagnosed CSF leak returns to the emergency department with fever, the diagnosis of meningitis should be strongly suspected and appropriate workup (i.e., lumbar puncture) and antibiotic treatment initiated immediately. A rare but significant complication of basilar skull fracture is traumatic carotid cavernous fistula (TCCF). In a retrospective review, the overall incidence of TCCF in basilar skull fractures was 3.8%, with middle fossa basilar skull fractures having the highest association.¹⁰⁷

Open Fractures

A skull fracture is open when a scalp laceration overlies a fracture. If the fracture has disrupted the dura, a communication exists between the external environment and the brain. A fracture that disrupts the paranasal sinuses or the middle ear structures is also considered open. An open skull fracture requires careful irrigation and débridement. Blind probing of the wound should be avoided because it can introduce contaminants into the wound and can further depress comminuted fracture pieces.

Diffuse Axonal Injury

Prolonged traumatic coma not caused by mass lesions, ischemic insult, or nontraumatic causes of coma is thought to result from diffuse axonal injury (DAI). DAI is a pathologic process in which axons are stretched and twisted by the same shear and tensile biomechanical forces that produce concussion. Effected axons are dispersed between areas of undamaged cells. Badly damaged axons become edematous and eventually begin to separate from each other, causing widespread disruption of cortical physiology and microanatomy in the white matter of the brain and brainstem. Complete separation of axons does not always cause cell death; acute uncoupling of cerebral blood flow and metabolism⁷⁰ and apoptosis are thought to be the primary factors linked to axonal cell death after DAI. Recovery depends on the reversal or correction of structural and physiologic abnormalities.¹⁴

DAI is thought to be the cause of persistent traumatic coma that begins immediately at the time of trauma; however, some patients with DAI may recover consciousness briefly before lapsing into prolonged coma. No specific acute focal traumatic lesions are noted on a head CT scan. MRI may be more sensitive in detecting subtle injury in DAI, but it is often not practical to perform MRI on critically injured patients. Occasionally, small petechial hemorrhages in proximity to the third ventricle and within the white matter of the corpus callosum or within the internal capsule of the brainstem are detected. DAI is the most common CT finding after severe head trauma, estimated to occur in 50% of all comatose head-injured patients.¹⁰⁸

Because clinical diagnostic studies cannot predict the extent of the axonal damage, the severity of the injury is determined by the clinical course. Patients with *mild* DAI are in coma for 6 to 24 hours. Approximately one third of patients with mild DAI demonstrate decorticate or decerebrate posturing, but by 24 hours they are following commands.¹⁰⁸ The mortality in this group is 15% and is associated with infectious complications or concurrent intracranial injuries. Most patients who recover have mild or no permanent disabilities.

Moderate DAI is the most common clinical picture. Patients with moderate DAI are in coma for longer than 24 hours. Often, they are victims of falls or vehicular crashes and have associated basilar skull fractures. Patients may exhibit transient decortication or decerebration but eventually recover purposeful movements. On awakening, patients have prolonged severe post-traumatic amnesia and moderate to severe persistent cognitive deficits. Almost 25% die of complications of prolonged coma.¹⁰⁶

Severe DAI is almost always caused by vehicular crashes. Patients remain in coma for prolonged periods and demonstrate persistent brainstem dysfunction (posturing) and autonomic dysfunction (e.g., hypertension and hyperpyrexia). Diffuse brain swelling subsequent to injury causes intracranial hypertension. Herniation syndrome can occur if elevated ICP does not respond to medical or surgical intervention. Some patients with severe DAI eventually awaken but are severely disabled. Some patients remain in a persistent vegetative state, but most with severe DAI die from their head injury. All patients with DAI present identically in coma. No early clinical predictor differentiates patients with mild, moderate, or severe DAI.

Contusions

Contusions are bruises on the surface of the brain, usually caused by impact injury. Most often, contusions occur at the poles and the inferior surfaces of the frontal and temporal lobes where the brain comes into contact with bone protuber-

ances in the base of the skull. If the contusion occurs on the same side as the impact injury, it is a *coup injury*; if it occurs on the opposite side, the contusion is a *contrecoup injury*. Contusions also often develop in the brain tissue that underlies a depressed skull fracture. Multiple areas of contused tissue may be produced with a single impact, often in association with other intracranial injuries.

Contusions are produced when parenchymal blood vessels are damaged, resulting in scattered areas of petechial hemorrhage and subsequent edema. Contusions develop in the gray matter on the surface of the brain and taper into the white matter. Often, subarachnoid blood is found overlying the involved gyrus. With time, the associated hemorrhages and edema of a contusion can become widespread and serve as a nidus for hemorrhage or swelling, thus producing a local mass effect. Compression of the underlying tissue can cause local areas of ischemia, and tissue infarction is possible if the compression is significant and unrelieved. Eventually, these ischemic areas become necrotic, and cystic cavities form within them.

The clinical presentation of patients with contusions is frequently delayed. They may have sustained only a brief LOC, but the duration of post-traumatic confusion and obtundation may be prolonged. If contusions occur near the sensorimotor cortex, focal neurologic deficits may be present. Many patients with significant contusions make uneventful recoveries, but contusions may cause significant neurologic problems, including increased ICP, post-traumatic seizures, and focal deficits.

Non-contrast-enhanced CT is the best diagnostic test to discover contusions in the early post-traumatic period. These appear heterogeneous and irregular because of mixed regions of hemorrhage, necrosis, and infarction. Often, the surrounding edematous tissue appears hypodense. By post-trauma days 3 and 4, the blood located within the contusions has begun to degrade, and MRI becomes more useful.

Epidural Hematoma

Epidural hematomas (EDHs) are blood clots that form between the inner table of the skull and the dura. Most EDHs are caused by direct impact injury that causes a forceful deformity of the skull. Often, a fracture occurs across the middle meningeal artery, vein, or a dural sinus. The temporoparietal region is the most likely site for an EDH.²⁴ The high arterial pressure of the bleeding vessel dissects the dura away from the skull, permitting the formation of the hematoma.

An EDH is usually unilateral, and 20% of patients have other intracranial lesions, usually subdural hematomas or contusions.²⁴ The deterioration of a patient who has an EDH from arterial bleeding can be rapid and dramatic. Because of their rapid formation, EDHs from arterial bleeding are usually detected within hours after injury and often earlier in children. EDHs that develop from a dural sinus tear develop more slowly, and clinical manifestations may be delayed, with resultant delays in detection.

EDH is primarily a disease of the young and accounts for 0.5 to 1% of all patients who have experienced TBI.²⁴ EDHs are rare in elders and children younger than 2 years of age because of the close attachment of the dura to the skull in both patient populations. The classic presentation of an EDH is described as head trauma producing a decreased level of consciousness followed by a "lucid" interval. Although the patient's consciousness is less decreased during the lucid interval, a completely normal mental status may not return before a second episode of decreased consciousness occurs. The lucid interval is not pathognomonic for an EDH and occurs in patients who sustain other expanding mass lesions. In fact, only approx-

imately 30% of patients with EDHs present classically.¹⁰⁹ The development of symptoms and signs of EDH is entirely dependent on how quickly the EDH is developing within the cranial vault. Patients with an EDH often complain of a severe headache, sleepiness, dizziness, nausea, and vomiting. A small EDH may remain asymptomatic, but this is rare.¹⁰⁹

If the patient is not in coma when the diagnosis of EDH is established and if the condition is rapidly treated, the mortality is 5 to 10%. If the patient is in coma, the mortality from EDH is approximately 20%. If the EDH is rapidly detected and evacuated, the functional outcome is excellent.

On CT scan, an EDH appears hyperdense, biconvex, ovoid, and lenticular. The EDH does not usually extend beyond the dural attachments at the suture lines. The margins are sharply defined, and the hematoma usually bulges inward toward the brain (Fig. 38-7). EDHs of mixed density on CT may be actively bleeding.

A posterior fossa EDH is the most common traumatic mass lesion of the posterior fossa and accounts for 5% of EDHs.²⁴ Direct occipital trauma resulting in a skull fracture that disrupts a venous sinus is the usual cause, and most patients have external evidence of occipital injury. Most patients become symptomatic within 24 hours after injury, with complaints of headache, nausea, vomiting, and nuchal rigidity. Most patients eventually have a decreased level of consciousness. On CT scan, a posterior fossa EDH looks similar to other EDHs, but it may cross the midline and extend above the tentorium to the supratentorial compartment (Fig. 38-8). Mortality approaches 26%.²⁴

Recent studies have investigated the indications for immediate operative intervention for EDHs. Epidurals greater than

30 cm³ in volume should be evacuated surgically, regardless of the patient's GCS score. Furthermore, it is strongly recommended that comatose patients with an acute EDH and anisocoria on pupillary examination undergo surgical evacuation as soon as possible.¹¹⁰

Subdural Hematoma

Subdural hematomas (SDHs) are blood clots that form between the dura and the brain. Usually, they are caused by the movement of the brain relative to the skull, as seen in acceleration-deceleration injuries. These hematomas are common in patients with brain atrophy, such as alcoholic or elder patients. In these patients, the superficial bridging vessels traverse greater distances than in patients with no atrophy. As a result, the vessels are more likely to rupture with rapid movement of the head. Once they are ruptured, blood can fill the potential space between the dura and arachnoid.

SDHs are more common than EDHs, occurring in up to 30% of patients with severe head trauma.¹¹¹ The slow bleeding of venous structures delays the development of clinical signs and symptoms. As a result, the hematoma compresses the underlying brain tissue for prolonged periods and can cause significant tissue ischemia and damage.

The patient's clinical presentation depends on the amount of brain injury sustained at the time of trauma and the rate of SDH expansion. If the patient with an SDH was rendered unconscious at the time of trauma, the prognosis is poor; these patients often have concurrent DAI. The signs and symptoms after injury that produce an SDH are initially related to the other intracranial injuries that may have been sustained and then to the slow expansion of the SDH.

SDHs are classified by the time to clinical presentation. *Acute* SDHs are symptomatic within 24 hours after trauma. Patients with acute SDHs often have a decreased level of consciousness. Most patients with an SDH present with a GCS score less than 8. Approximately 12 to 38% of patients will have a lucid period at some point in their presentation. The overall mortality of patients who have an SDH and require surgical intervention is between 40 and 60%.¹¹¹

Because of associated brain injury caused by the SDH, the delay in clinical signs and symptoms, and the more advanced mean age of the at-risk population, the mortality associated with SDH is much higher than that associated with EDH. Pupil inequality, motor deficit, and other signs consistent with increased brain swelling may be present on the initial examination. If the patient is deeply comatose at presentation with flaccidity and without signs of brainstem activity, supportive care should be considered in the emergency department. Subsequent management decisions should be discussed with the patient's family and the attending neurosurgeon.

If the SDH is very small (only a few millimeters thick at its widest point on CT scan), some neurosurgeons may choose careful observation for these patients. Even a small SDH may be accompanied by extensive brain tissue damage that can cause an increase in ICP sufficient to precipitate a herniation syndrome. Current consensus guidelines recommend that acute SDHs with a thickness greater than 10 mm or a midline shift of more than 5 mm on CT should be evacuated surgically, regardless of the patient's GCS score.¹¹¹ Research indicates that the longer the time between clinical worsening and operative treatment, the worse the patient's clinical outcome.¹¹¹ In these cases, surgical evacuation should be performed as soon as possible.

Unlike EDHs, SDHs often extend beyond the suture lines (Fig. 38-9). An SDH may follow the contour of the tentorium and be detected within the interhemispheric fissure (Fig. 38-

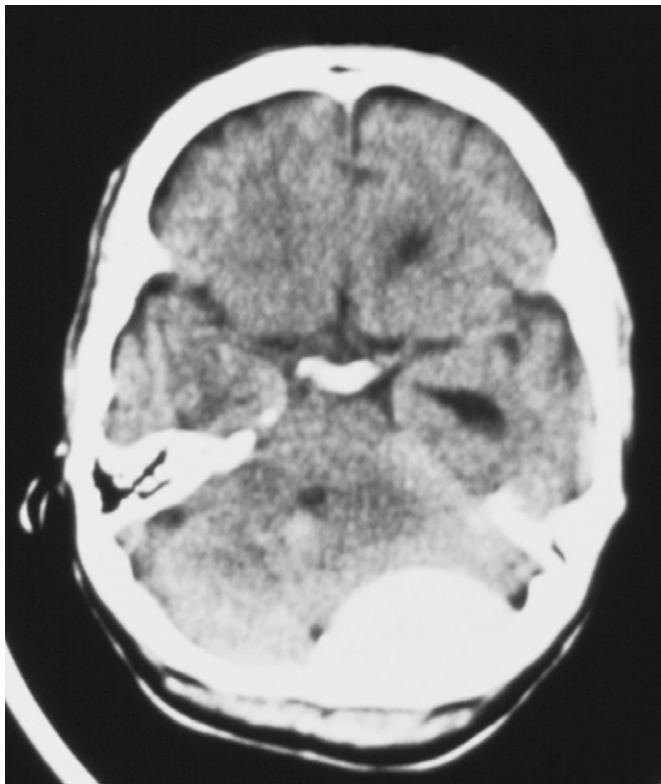


Figure 38-8. Non-contrast-enhanced computed tomography scan of large left posterior fossa, epidural hematoma. The size of the lesion at this high level suggests that it crosses into the supratentorial compartment. This lesion is often associated with occipital bone fracture that disrupts transverse sinus.

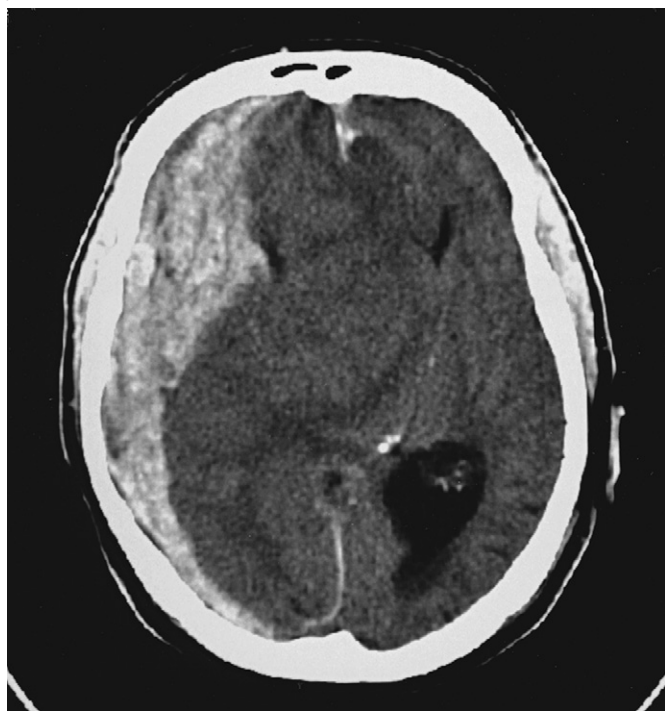


Figure 38-9. Non-contrast-enhanced computed tomography scan of acute right temporal subdural hematoma. There is acute bleeding as well as delayed bleeding, which explains the mixed density. Mass effect is large, with midline shift measuring approximately 2.7 cm right to left. The right lateral ventricle has been obliterated.

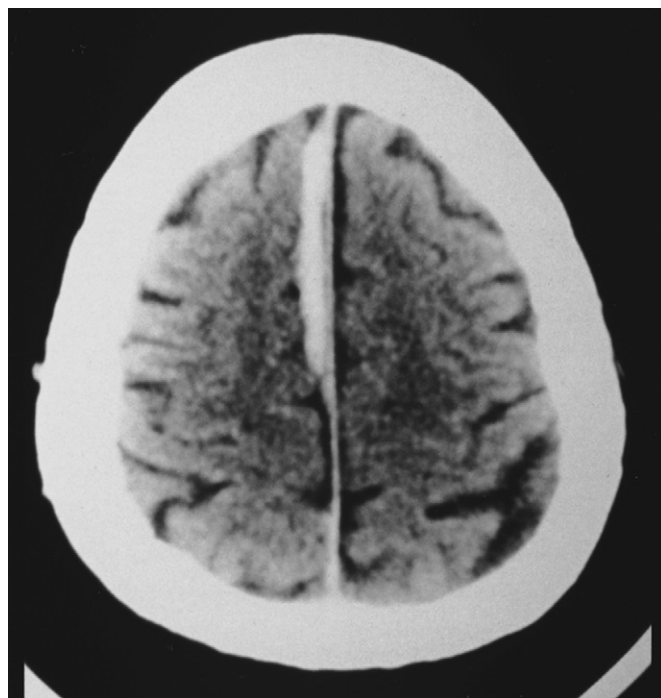


Figure 38-10. Non-contrast-enhanced computed tomography scan of intrahemispheric acute subdural hematoma.

10). Many patients with an acute SDH also show CT evidence of intracerebral lesions contralateral to the SDH.

A *subacute* SDH is symptomatic between 24 hours and 2 weeks after injury. It may appear hypodense or isodense on CT scans. Contrast increases detection of isodense lesions. Patients complain of a headache, altered mental status, muscle weakness, or frank paralysis. Most patients with subacute SDH require surgical evacuation of the lesion.

A *chronic* SDH becomes symptomatic 2 weeks or more after trauma. The signs and symptoms may be very subtle or non-specific, but many patients demonstrate unilateral weakness or hemiparesis.²⁴ Most report an altered level of consciousness, but some patients are unable to recall their head injury or describe only a minor injury. In 20% of cases, chronic subdurals are bilateral.²⁴ A chronic SDH may have initially been a small asymptomatic SDH that eventually expanded due to a combination of recurrent hemorrhage and escape of plasma into the hematoma. At some point, a critical mass is reached, and the chronic SDH becomes symptomatic. On CT scan, a chronic SDH may appear isodense or hypodense to brain parenchyma. In these cases, indirect evidence of the lesion includes a midline shift, effacement of the ipsilateral cortical sulci, and ventricular compression. Contrast may increase the likelihood of identifying a chronic SDH that has become isodense. On CT scan, blood of various ages is seen as a mixed-density lesion. On MRI, a chronic SDH appears hyperdense. The treatment of chronic SDHs is controversial. If they become symptomatic, chronic SDHs require surgical evacuation. Most patients have a good outcome after surgery. Overall, the mortality from surgically drained chronic SDH approaches 4%, with decreased survival in elders.²⁴

The prognosis of SDH does not entirely depend on the size of the hematoma but, rather, on the degree of brain injury caused by the pressure of the expanding hematoma on underlying tissue or by other intracranial injury caused by the initial

impact. Mortality is highest in older people, patients who have a GCS of 8 or less, and in those with signs of acute herniation syndrome on initial emergency department presentation. Posterior fossa SDHs make up less than 1% of all reported SDHs. They are caused by occipital trauma that tears bridging vessels or venous sinuses. Clinical manifestations of posterior SDH vary but usually include nausea, vomiting, headache, and decreased level of consciousness. Occasionally, cranial nerve palsies may be found, as well as nuchal rigidity, cerebellar signs and symptoms, and papilledema. On a CT scan, a posterior fossa SDH does not cross the midline or extend above the tentorium. The outcome of a posterior SDH is very poor.

In children, the presence of an SDH should prompt consideration of child abuse. Many types of injury can produce SDH in children, but the infant who is repeatedly and forcibly shaken is especially susceptible. Infants may have SDH because of birth trauma. In these cases, the initial clinical manifestation may be a generalized seizure within the first 6 months of life. On examination, the infant may have a bulging fontanel or an enlarged head circumference. A careful history may elicit long-standing constitutional symptoms, such as failure to thrive or lethargy.

Subdural Hygroma

A subdural hygroma (SDHG) is a collection of clear, xanthochromic blood-tinged fluid in the dural space. The pathogenesis of an SDHG is not certain. It may result from a tear in the arachnoid that permits CSF to escape into the dural space or effusions from injured vessels through areas of abnormal permeability in the meninges or in the underlying parenchyma. They may accumulate immediately after trauma or in a delayed manner. Clinically, an SDHG cannot be distinguished from other mass lesions. Often, patients have a decreased level of consciousness or focal motor deficits. They may complain of headaches, nausea, and vomiting. The ICP can increase because of the mass effect, and signs of increased ICP may be present on examination.

On CT scans, SDHGs appear crescent shaped in the extra-axial space. The CT density is the same as that of CSF. Bilateral SDHGs are common. If SDHGs are asymptomatic, observation is reasonable management. Otherwise, they must be surgically evacuated. Mortality varies from 12 to 55 % and appears to depend on the severity of other intracranial injury.

Traumatic Subarachnoid Hemorrhage

Traumatic subarachnoid hemorrhage (TSAH) is defined as blood within the CSF and meningeal intima and probably results from tears of small subarachnoid vessels. TSAH is detected on the first CT scan in up to 33% of patients with severe TBI and has an incidence of 44% in all cases of severe head trauma. It is therefore the most common CT scan abnormality seen after head injury. Data from the National Traumatic Coma Data Bank demonstrate a 60% unfavorable outcome in severely brain-injured patients in the presence of TSAH compared with a 30% unfavorable outcome if no TSAH occurs.¹¹² An increased incidence of skull fractures and contusions is found in patients with TSAH compared with patients with no TSAH. The amount of blood within the TSAH correlates directly with the outcome and inversely with the presenting GCS.

Patients may complain of headache and photophobia. A non-contrast CT scan makes the diagnosis, with increased density noted within the basilar cisterns. Blood can also be seen within the interhemispheric fissures and sulci.

TSAH with no other brain injury does not generally carry a poor prognosis. The most serious complication of TSAH is worsening of cerebral vasospasm, which may be severe enough to induce cerebral ischemia. Post-traumatic vasospasm is common, occurring approximately 48 hours after injury and persisting for up to 2 weeks. Calcium channel blockers (e.g., nimodipine and nicardipine) have been used in the acute intensive care unit setting to prevent or reduce vasospasm after TSAH. Although a radiographic reduction of vasospasm is not consistently seen, the overall outcome of patients treated with these agents seems to be improved compared with no treatment.¹¹³

Intracerebral Hematoma

Intracerebral hematomas (ICHs) are formed deep within the brain tissue and are usually caused by shearing or tensile forces that mechanically stretch and tear deep small-caliber arterioles as the brain is propelled against irregular surfaces in the cranial vault. Resulting small petechial hemorrhages subsequently coalesce to form ICHs. Approximately 85% are in the frontal and temporal lobes. They are often found in the presence of extra-axial hematomas, and in many patients multiple ICHs are present.¹⁰⁹ Isolated ICHs may be detected in as many as 12% of all patients with severe head trauma.

The clinical effects of ICH depend on size, location, and whether the bleeding is continuing. ICHs have been reported with all degrees of severity of head trauma. More than 50% of patients with ICH sustain LOC at the time of impact. The patient's subsequent level of consciousness depends on the severity of the impact and coexisting lesions. Combined with contusions, other concurrent lesions, and subsequent perilesion edema, an ICH can produce substantial mass effects and precipitate a herniation syndrome (Fig. 38-11).

An ICH may be detected on the first CT scan immediately after injury but often is not seen for several hours or days. Unlike contusions, ICHs are usually deep in the brain tissue and often become well demarcated over time. On CT scan, an ICH appears as a well-defined hyperdense homogeneous area of hemorrhage (Fig. 38-12).



Figure 38-11. Non-contrast-enhanced computed tomography scan of intracerebral hematoma and contusion in the left occipital region. The scan also shows layering of tentorial subdural hematoma. Mass effect and early uncus herniation are visible as well.



Figure 38-12. Non-contrast-enhanced computed tomography scan of right occipital and temporal intracerebral hematomas, surrounded by mild edema and hemorrhagic contusion. Small intrahemispheric subdural hematoma is visible in posterior interhemispheric fissure. Midline shift is obvious. Ventriculostomy has been placed and is visible as high-density image within ventricles.

Many patients with an ICH require emergent intervention or surgery to control elevated ICP. Mortality is low in patients who are conscious before surgery; in unconscious patients, mortality approaches 45%.^{114,115} ICHs that bleed into the ventricles or cerebellum also carry a high mortality rate.

Traumatic Intracerebellar Hematoma

Primary traumatic intracerebellar hematomas are rare but can occur after a direct blow to the occipital area. Often, these patients also have a skull fracture or a posterior fossa SDH. Supratentorial contrecoup hematomas and contusions are also common associated findings.

The clinical presentation of an isolated traumatic cerebellar hematoma is similar to that of other posterior lesions. When other traumatic lesions are present, the picture may be quite confusing. The acute management should first address the most clinically significant lesion. The mortality from isolated traumatic intracerebellar hematoma is very high.

Acknowledgments

The authors thank Emergency Medicine Clinics for allowing us to use certain portions of a recent article in this chapter.¹¹⁶

KEY CONCEPTS

Severe and Moderate Head Injuries

- All patients with severe or moderate head injury require serial neurologic examinations while in the emergency department to allow early detection of herniation syndrome related to expanding traumatic mass lesions or increasing cerebral edema. A non-contrast CT scan should be performed on all moderate and severe head-injured patients.
- Acute herniation syndrome manifested by neurologic deterioration should initially be managed with short-term hyperventilation, to a P_{CO_2} of 30 to 35 mm Hg, with monitoring and then surgical intervention as soon as possible. Long-term hyperventilation is not indicated. Mannitol or hypertonic saline should be used only in patients with increasing ICPs or acute neurologic deterioration.
- Secondary systemic insults such as hypoxia and hypotension worsen neurologic outcome after severe and moderate head trauma and should be corrected as soon as detected in the out-of-hospital or emergency department setting.
- For adult patients, hypotension in the presence of isolated severe head injury is a preterminal event. Hypotension usually results from comorbidity, and its cause should be sought and treated.
- The GCS is a useful clinical tool for following head-injured patients' neurologic status, but because of its limitations, the initial GCS in the emergency department cannot reliably predict prognosis after acute head injury.
- Head-injured patients who have been chemically paralyzed do not have clinical manifestations of seizures; anticonvulsants should be given prophylactically.
- Most "talk and deteriorate" patients who present with moderate head injury have subdural or epidural hematomas. Early detection, CT scan, and expedient surgical intervention are the keys to a good outcome.
- Caution should be given to out-of-hospital RSI intubations in severe head injury if the intubations may be prolonged or significantly challenging. Alternative airway management should be considered.

Minor Head Trauma

- Risk stratification of patients with minor head injury into low-risk and high-risk categories can help direct the emergency physician to an appropriate diagnostic workup.

- The decision to perform CT scans on patients with minor head trauma should be individualized but based on consideration of high- and moderate-risk criteria.
- Alcohol can affect the GCS and significantly obscure the neurologic examination. Intoxicated patients should be considered at high risk.
- Most patients with minor head trauma can be discharged from the emergency department after a period of observation but require a competent observer.
- Patients sustaining a concussion are at risk for prolonged and substantial morbidity. Athletes should not be allowed to return immediately to sports activities because of the potential risk of second impact syndrome. All current recommendations for return to play after a sports-related concussion state that players with concussion should not return to play for at least 1 week after they have become asymptomatic. This period is usually increased to at least a symptom-free month if an LOC or prolonged post-traumatic amnesia occurred at the time of concussion.

Pediatric Head Injuries

- Children with severe head trauma have fewer intracranial lesions than adults but more edema. In children, increasing edema alone can cause talk and deteriorate or other significant neurologic decline.
- Skull fractures have more clinical significance in children than in adults.
- In children, unlike adults, hypovolemic hypotension can occur because of head injury, especially in those younger than 1 year of age.
- In very young children, head injury often occurs from nonaccidental causes. Child abuse should be suspected in young children with head trauma, especially those younger than 2 years of age.

Penetrating Head Injuries

- Tangential gunshot wounds are associated with a high frequency of intracranial traumatic lesions; CT scanning should be performed.
- Anticonvulsant prophylaxis and antibiotics should be given to a patient with penetrating head injuries.
- The clinical outcome after gunshot wounds to the head can be predicted by the initial clinical presentation and the missile path through the brain.