PEDIATRIC EMERGENCY MEDICINE SECRETS

THIRD EDITION

QUESTIONS YOU WILL BE ASKED

TOP 100 SECRETS ■ KEY POINTS ■ WEB SITES

STEVEN M. SELBST MD

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SAUNDERS

PEDIATRIC EMERGENCY MEDICINE

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Third Edition

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To my wife, Andrea, for her endless love and support

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PREFACE

The Emergency Department (ED) is a stressful environment, and clinicians who treat children in the ED are constantly challenged. Every shift is demanding, as complex care must be delivered rapidly to patients with serious injuries and high-acuity illnesses. Important questions about diagnosis and management accompany almost every patient. Besides perplexing patients, senior physicians are frequently challenged by inquisitive trainees during bedside discussions. In a busy ED, clinicians need to find succinct, up-to-date information easily and quickly. This book addresses common and difficult questions about pediatric emergencies, and it offers sensible and evidence-based answers.

Pediatric Emergency Medicine Secrets, Third Edition, has six sections. As in previous editions, the first section addresses life-threatening conditions and immediate stabilization of children. The second section features common chief complaints that are frequently managed in the ED. Later sections focus on important medical emergencies, surgical emergencies, major and minor trauma, and environmental emergencies. Finally, questions and answers relating to special topics in pediatric emergency medicine (procedural sedation, bio-terrorism, patient safety, risk management, and the transport of children to specialized centers) are included.

All chapters in this third edition have been revised and renewed, with current references and updated information. There are dozens of new questions to inform, entertain, and test the readers. Many questions have accompanying references and relevant websites to allow further study. Classic photographs and radiographs enhance learning throughout the book. Each chapter features Key Points that highlight essential tips and pearls. The Top 100 Secrets have been revamped and summarize the most salient points of each chapter. Some questions in this book bring up amusing "fun trivia." Most questions address important clinical problems seen in the ED, and they provide valuable insight into diagnosis and management.

Pediatric Emergency Medicine Secrets is unique because of the question-and-answer format. I sincerely hope it will help those who are preparing for examinations or board certification. Most important, I hope this book helps clinicians on the "front line," as they provide emergency care to ill and injured children.

Steven M. Selbst, MD

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Dr. Kate Cronan, who co-edited the first two editions of *Pediatric Emergency Medicine Secrets*, chose to forgo this third edition. Kate is a wonderful colleague and a dear friend, and I am very thankful for all that she did to develop this book in the early stages. Fortunately, Kate continues to contribute as an author of several chapters in this edition.

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Steven M. Selbst, MD

TOP 100 SECRETS

These secrets are 100 of the key teaching points of *Pediatric Emergency Medicine Secrets*, 3rd edition. They summarize the basic concepts and principles and most salient details of pediatric emergency medicine.

1. Children commonly develop respiratory failure prior to cardiac arrest. Early intervention, before cardiac arrest, offers the best chance for successful outcome.
2. The poor prognosis for children in cardiopulmonary arrest probably reflects the terminal nature of asystole, the most common rhythm. Long-standing tissue hypoxemia and acidosis from antecedent prolonged respiratory insufficiency add to the poor prognosis.
3. The two-thumb method of chest compressions is preferred for newborns, with the depth of compression being one third of the anteroposterior diameter of the chest. Compression should be deep enough to generate a pulse.
4. Newborn infants do not tolerate cold, and hypothermia can prolong acidosis. Prevent heat loss as much as possible.
5. Respiratory failure may be present without respiratory distress. Work of breathing may appear normal in children with a reduced level of consciousness (ingestion, metabolic derangements, head trauma), neuromuscular dysfunction (muscle disease), or fatigue, despite the presence of significant hypoventilation.
6. Shock exists when the patient's metabolic demand exceeds the body's ability to deliver oxygen and nutrients. Early recognition is essential. The shock state often exists in the presence of a "normal" blood pressure.
7. Hypovolemia is the most common cause of shock in children, and this is managed with aggressive volume resuscitation. The greatest error that you can make in the treatment of shock is to use pressor agents to treat hypovolemia.
8. Do not withhold analgesia from a child with abdominal pain of unknown cause simply for fear of delaying diagnosis or causing misdiagnosis.
9. In children with abnormal mental status, do not minimize the extent of the child's illness by assuming the patient missed his nap that day or is difficult to wake up because it is late at night. Do not send the child home without seeing him or her awake and alert.
10. Suspect head trauma or toxicologic ingestion in children with altered mental status even when there is no history of either.
11. Check a rapid bedside glucose level for any young child with altered mental status if the cause is not immediately obvious.
12. Placing babies in the supine sleeping position has decreased the incidence of sudden infant death syndrome (SIDS).
13. A period of observation in the emergency department (ED) or admission to the hospital is necessary for patients with an apparent life-threatening event (ALTE).
14. Chest pain in children is rarely related to previously undiagnosed cardiac disease, but children with this symptom deserve careful evaluation. Pediatric chest pain is concerning when it is induced by exercise, associated with fever, or accompanied by an abnormal finding on physical examination.
15. A young child with persistent cough following a choking episode likely has an aspirated foreign body in one of the mainstem bronchi.

16. Crying and irritability in an infant may indicate a life-threatening condition, such as meningitis, or only colic. A careful history and physical examination are essential to detect treatable causes for crying in infancy.
17. A clear liquid diet is not necessary for most children with acute gastroenteritis. Breast milk, full-strength formula or milk, and age-appropriate foods are recommended for children with uncomplicated diarrhea.
18. Oral rehydration therapy (ORT), when properly administered, is as effective as intravenous (IV) rehydration in the majority of children with mild to moderate dehydration due to gastroenteritis. ORT takes less staff time and shortens length of stay in the ED. Offer oral electrolyte solution, 1 mL/kg for mild dehydration and 2 mL/kg for moderate dehydration, every 5 minutes.
19. "Button" batteries in the ear canal, nose, or esophagus can cause extensive caustic damage in a short period of time and must be removed promptly.
20. The height of the fever by itself is a poor predictor of serious bacterial illness. Clinical signs such as age of the child, appearance, and peripheral perfusion are better predictors of serious illness than the height of the fever.
21. Urinary tract infection is the most common bacterial infection in febrile infants less than 2 months old.
22. Ingestion of multiple magnets can result in serious complications including bowel perforation, volvulus, and death. Consider emergency removal of magnets in the gastrointestinal tract.
23. Avoid routine neuroimaging in children with headaches. Use specific "red flags" in the patient's history and physical examination to prompt computed tomography (CT) or magnetic resonance imaging (MRI) evaluation.
24. Urinary tract infection is the most common cause of hematuria and/or dysuria in children. Evaluate a child with reported hematuria: Obtain a urinalysis with microscopic examination and measure the child's blood pressure.
25. Measure the blood pressure in children with Henoch-Schönlein (HSP) purpura, as the presence of hypertension justifies hospital admission. Confirm elevated blood pressure readings with an appropriate-size cuff when the child is calm and not in pain.
26. Most neonates with unconjugated hyperbilirubinemia have physiologic or breast milk jaundice. Conjugated hyperbilirubinemia is pathologic at any age and requires further diagnostic studies and hospital admission. Jaundice in children older than 3 months is pathologic.
27. Slipped capital femoral epiphysis (SCFE) is an orthopedic emergency, and the patient (usually an obese young teenager) may present with hip pain or pain referred to the knee or thigh.
28. Transient synovitis is a common cause of limping and can mimic septic arthritis. However, patients with transient synovitis usually have little or no fever, appear well, and have less pain and limitation of motion than those with septic arthritis.
29. Neoplastic neck masses are generally painless, firm, fixed cervical masses. Neck masses that are tender, warm, and erythematous are more likely to have an infectious cause (cervical adenitis).
30. Stevens-Johnson syndrome and toxic epidermal necrolysis represent a spectrum of the same disease; erythema multiforme is a less serious condition.
31. A CT scan may help distinguish orbital cellulitis from preseptal cellulitis when the child has severe periorbital edema and eye examination is difficult. Orbital cellulitis is often associated with a sinus infection.

32. When evaluating a boy with abdominal pain, perform a genitourinary examination to look for scrotal involvement. Patients with testicular torsion can present with abdominal pain. A history of trauma often confuses the picture.

33. Children with group A β-hemolytic streptococcus (GABHS) pharyngitis usually do not have cough or coryza, but submandibular lymphadenopathy is frequently present.

34. Consider a lumbar puncture to rule out meningitis for any child with a stiff neck, fever, and ill appearance. However, young infants with meningitis may not have a stiff neck until the infection progresses.

35. Consider retropharyngeal abscess in infants with a stiff neck, fever, and ill appearance. Usually patients with this condition have fullness on one side of the neck and refuse lateral neck movement. A lateral neck radiograph is very helpful.

36. Laryngomalacia is the most common cause of stridor in infants. It is generally benign and resolves spontaneously as the child grows.

37. Bacterial tracheitis can mimic croup and epiglottitis. Patients present with stridor, fever, and toxicity that worsens over a few days.

38. Syncope in children is usually benign and neurocardiogenic in origin, but potentially serious causes must be eliminated with a thorough history, physical examination, and review of an electrocardiogram (ECG). Findings will guide further investigation.

39. Syncope is likely due to a cardiac abnormality if it is recurrent; occurs during infancy, when the patient is in a supine position, or with exertion; or is associated with chest pain or an injury related to the patient's fall. These findings require further testing, referral to a cardiologist, and possibly admission to the hospital.

40. Consider hospitalization of a patient with pelvic inflammatory disease in the following instances: pregnancy, unclear diagnosis, vomiting, peritoneal signs, a young teenager (age < 15 years), tubo-ovarian abscess present or suspected, failed outpatient treatment, or patient's inability to follow the outpatient regimen.

41. Vomiting that persists for more than 24 hours without the development of diarrhea may not be due to gastroenteritis. Consider obstruction and nongastrointestinal causes such as brain tumor, meningitis, and diabetic ketoacidosis.

42. Failure to administer epinephrine (or delayed administration) during anaphylaxis is common and is associated with severe and fatal reactions. Antihistamines and steroids have a limited role in the acute treatment of anaphylaxis.

43. If a cyanotic neonate presents to the ED in distress, begin a prostaglandin drip immediately, given concern for a possible ductal-dependent cardiac lesion.

44. Treat supraventricular tachycardia with vagal maneuvers (ice to the face for infants, Valsalva maneuver for older children), administer adenosine by rapid IV push and saline flush, or consider synchronized cardioversion.

45. Do not delay treatment of status epilepticus when IV access is unavailable; consider alternatives for administration of medication, such as intranasal, buccal, rectal, or even intraosseous routes.

46. The development of cerebral edema in patients with diabetic ketoacidosis is associated with severe acidosis, high initial blood urea nitrogen, low arterial carbon dioxide, inadequate increase in serum sodium concentration, age less than 3 years, and bicarbonate administration.

47. A quick estimate of degree of dehydration can be obtained by checking four examination findings: capillary refill at the fingertip greater than 2 seconds, ill general appearance, dry mucous membranes, and absence of tears. If two or more are found, the child is likely to be at least 5% dehydrated.

48. A “currant jelly” stool is a late (and uncommon) finding in intussusception, which implies that bowel necrosis has occurred.

49. An infant less than 1 month old with bilious vomiting has malrotation (with or without volvulus) until proved otherwise.

50. A history of recent vigorous activity may provide a clue to the diagnosis of ovarian torsion in a female with sudden onset of stabbing abdominal pain.

51. Patients with sickle cell disease are immunocompromised, and those with fever are at high risk for bacterial infection. Consider treatment with broad-spectrum antibiotics even if no source for fever is identified.

52. Anticipate tumor lysis syndrome in patients with newly diagnosed leukemia or lymphoma. Aggressive hydration with intravenous fluids is the mainstay of treatment, with the goal to protect the kidneys.

53. Admit pediatric patients to the hospital for treatment of a urinary tract infection (UTI) if you are dealing with any of the following: age less than 3 months, older child with ill appearance, dehydration, associated chronic disease such as diabetes mellitus or sickle cell disease, or a child who is vomiting and cannot tolerate oral medications.

54. Admit pediatric patients to the hospital for treatment of pneumonia if they are hypoxic, are toxic appearing, have failed outpatient treatment, have respiratory distress, show questionable adherence, are not tolerating oral fluids, have an associated serious condition (immunodeficiency, cardiopulmonary disease), have complications such as empyema, or have infection with MRSA (methicillin-resistant *Streptococcus aureus*) or other virulent organisms.

55. Characterization of a poisoned patient’s mentation, vital signs, pupil size and reactivity, and skin appearance are usually more useful to the clinician than urine drug screening.

56. Careful support to a poisoned patient’s airway and breathing, circulation, and neurologic function will help more patients than antidotal therapies.

57. The most significant risk factors for adolescent suicide include male sex, age above 16 years, previous suicide attempt, homosexual orientation, mood disorder, substance abuse, poor social support, and access to firearms or other lethal means.

58. When physically restraining an out-of-control child, explain the need to the patient, gather adequate personnel, avoid pressure on the patient’s throat or chest, avoid placement of the child in a prone position, and carefully monitor the restrained patient.

59. The management of bronchiolitis is mainly supportive. Most children do not respond to inhaled β-receptor agonists, and there is no evidence to support the use of corticosteroids or other treatments.

60. Steroids are useful in treating children with croup. There is no evidence that humidified air is beneficial.

61. If a child with a tracheostomy tube is in respiratory distress, assume the tube is obstructed or malpositioned. Immediately assess the patient’s airway and breathing, and be prepared to change the tracheostomy tube.

62. Dental abscesses are a common cause of facial swelling in young children and can usually be managed on an outpatient basis with oral antibiotics and follow-up with a dentist.

63. Pyloric stenosis is the most common surgically correctable cause of vomiting in infants. Consider this condition in babies 2 to 6 weeks old with *nonbilious* vomiting, usually described as “projectile.” Infants are often hungry after vomiting, unless they become dehydrated. Physical examination rarely reveals an “olive,” and ultrasound is the diagnostic test of choice.

64. Strokes are uncommon in the pediatric population, but a high index of suspicion is needed to avoid delay in diagnosis and treatment.

65. Cushing’s triad (bradycardia, hypertension, irregular respiration) indicates increased intracranial pressure—manage this with attention to the child’s airway, breathing, and

circulation. Rapid sequence induction (RSI) and endotracheal intubation allow for airway protection, and RSI limits further intracranial pressure (ICP) elevation during intubation. Consult neurosurgery immediately and begin treatment with pharmacologic agents such as mannitol or hypertonic saline. Reserve hyperventilation for cases of impending herniation.

66. Always consider infection with *Chlamydia* or *Neisseria gonorrhoeae* in neonates with conjunctivitis and purulent discharge.

67. Prompt irrigation of the eye with saline (or tap water if this is immediately available) limits damage from chemical injury to the eye.

68. Magnetic resonance imaging is the imaging test of choice for the diagnosis of osteomyelitis. Plain radiographs are usually normal in patients with this infection.

69. To manage bleeding after a tonsillectomy, attend to the child's airway, breathing, and circulation. Restore intravascular volume. If there is a clot in the posterior pharynx, leave it alone. Removal of this clot could lead to brisk bleeding and even death from aspiration. For severe active bleeding, tamponade the site with gauze and digital pressure until the ear, nose, and throat specialist arrives.

70. Children with facial edema may have nephrotic syndrome rather than an allergy. Check the child's urine for proteinuria and, if present, evaluate renal function, assess for infection, and measure the child's blood pressure.

71. For pediatric patients with abdominal injury, nonoperative management is usually appropriate. Clinical instability is the most important indication for an emergent laparotomy in a child with abdominal injury.

72. Prevention is more effective than medical intervention in decreasing morbidity and mortality rates from burn and smoke inhalation injury. Most children involved in fires die from the effects of smoke inhalation as opposed to burn injuries.

73. Most sexually abused children have a normal physical examination.

74. Rib fractures, metaphyseal chip fractures, spine and scapula fractures, and complex skull fractures have a high probability of being caused by child abuse.

75. Immediately reimplant an avulsed permanent tooth to preserve tooth viability. If this is not possible, temporarily store the tooth in cool milk, saliva, or saline until emergent dental consultation can be obtained.

76. The most common mechanism of pediatric elbow injury is a fall onto an outstretched hand. If there is swelling of the elbow on examination, or if elbow radiographs show a posterior fat pad or a displaced anterior fat pad, consider a supracondylar fracture, even if the fracture is not obvious.

77. Chemical burns and suspicion of globe perforation are true ophthalmologic emergencies that require immediate recognition and initiation of treatment. Both warrant emergent (same-day) ophthalmologic consultation or referral.

78. Pediatric patients with an eye injury may have a globe perforation, but the physical examination can be deceiving. Be suspicious if the mechanism suggests a penetrating foreign body (e.g., hammering or grinding metal) or if the pupil is shaped irregularly (e.g., teardrop pupil).

79. A young infant with a large nonfrontal scalp hematoma often has a skull fracture and may have an intracranial injury.

80. Children older than 2 years with a nonsevere mechanism of injury, normal mental status, no signs of basilar skull fracture, and no history of loss of consciousness, vomiting, or severe headache have an extremely low likelihood of a *clinically important* intracranial injury.

81. Topical anesthetics such as LET (lidocaine 4%, epinephrine 1:1000, tetracaine 0.5%) are very helpful in reducing pain associated with laceration repair and often preclude the need for injection with lidocaine.

82. Injured children are different from adults. They are more likely than adults to become hypothermic at the scene and during ED resuscitation. Owing to a flexible and less muscular chest wall, rib fractures and flail chest are less common in children, but forces are more easily transmitted to internal organs. Solid organs in the abdomen of children are disproportionately larger and more exposed than in adults. Children have larger heads relative to their bodies and are more likely to land on their heads when they fall; this situation also contributes to cervical spine injuries at a higher level (C2-C3) in children than adults.
83. Steroids are not recommended for most spinal cord injuries in children. Consult a neurosurgeon before administering steroids.
84. A hard cervical collar must be sized appropriately but does not provide complete neck immobilization.
85. Hematuria, a hallmark for genitourinary trauma, is absent with some pedicle and penetrating injuries. Contrast-enhanced CT scan is the diagnostic test of choice for *stable* patients with suspected renal injury.
86. Avulsion fractures of the ischial tuberosity or iliac spine may occur from sudden muscle contraction during vigorous running or jumping, with rapid acceleration or deceleration, or with a quick change of direction.
87. A child struck in the chest by a pitched baseball may develop commotio cordis and sudden cardiac arrest.
88. Second impact syndrome can be fatal and results from acute brain swelling when a second head injury occurs prior to full recovery from a concussion.
89. Tension pneumothorax is diagnosed clinically, without taking time for radiographs, in a child with respiratory distress or cardiovascular compromise.
90. Treatment for a brown recluse spider bite is supportive and not specific: Local wound care, analgesics, and tetanus immunization are mainstays of therapy. Extensive dermal injury may require skin grafting.
91. Observe all children in the ED after a submersion injury for at least 6 to 8 hours. Initially asymptomatic, alert patients may develop respiratory distress within a few hours of the submersion.
92. Most children with household electrical injuries are exposed to low voltage and can be discharged from the ED after brief observation. Admit patients to the hospital for cardiac monitoring if there is an abnormal electrocardiogram, past cardiac history, loss of consciousness, or injury involving greater than 240 volts.
93. The two priorities of treating heat stroke are eliminating hyperpyrexia and supporting the cardiovascular system. Bring the patient into a cool location and remove all clothing. Actively cool the patient by spraying him with lukewarm water, positioning fans to blow air across the body, and applying ice packs to the neck, groin, and axilla. IV hydration and diuresis are essential to treat myoglobinuria.
94. Manage frostbite with rapid rewarming of affected body parts in a bath of water (40° C to 42° C) and give narcotic analgesics while consulting surgical colleagues.
95. Suspect a biologic attack when there is an epidemic presentation in a relatively compressed time frame, especially when the disease is rare or not endemic to the area and when there are particularly high morbidity or mortality rates and more respiratory forms of disease than usual.
96. Automated external defibrillators (AEDs) can be used to treat cardiac rhythms amenable to shock in pediatric patients. AEDs equipped with a pediatric attenuator (which decreases the energy delivered) are preferred for children under 8 years of age. If one is not available, an AED without a dose attenuator results in minimal myocardial damage and good neurologic outcomes.

97. Essential information for a handoff in the ED includes relevant medical and surgical history, patient course and current condition, studies obtained and pending, suspected diagnosis, and anticipated disposition.

98. Parents *do not* have the right to refuse treatment for their child in the ED if a life-threatening situation exists and the emergency physician believes that it is unsafe for a patient to leave the ED to seek care elsewhere, if the patient or parent is under the influence of drugs or alcohol and cannot understand the risks and benefits of receiving or refusing care, and when child abuse is suspected.

99. Ideal staffing for procedural sedation and analgesia in the ED includes a physician experienced in pediatric advanced life support who will administer medications and closely observe the child's response. A second physician should perform the procedure while a nurse documents the patient's response to medications and is available to assist in suctioning and administering oxygen or reversal agents.

100. The Emergency Medical Treatment and Active Labor Act (EMTALA) dictates that referring clinicians must do everything possible to stabilize the patient's medical condition before transport. The receiving hospital must accept a patient for transport if space and appropriate level of care are available. The patient's ability to pay is not relevant to transfer.

CHILDHOOD RESUSCITATION

Allen L. Hsiao and M. Douglas Baker

1. What is the incidence of pediatric cardiopulmonary arrests?

Schoenfeld and Baker noted that 0.25% of patient visits to an urban emergency department involved management in the resuscitation room. A prospective study by Ong and associates found an overall annual incidence of cardiopulmonary arrests of 59.7 per million children, with the highest incidence, 175 per million children, noted in the youngest age group (under 4 years). For patients admitted to the hospital, arrests occur in about 0.7% to 3% of pediatric admissions and 1.8% to 5.5% of pediatric intensive care admissions. The estimates of incidence of pediatric cardiopulmonary arrests vary by geographic location of the patient population.

Ong ME, Stiell I, Osmond MH, et al: Etiology of pediatric out-of-hospital cardiac arrest by coroner's diagnosis. Resuscitation 2006;68:335-342.

Schoenfeld PS, Baker MD: Management of cardiopulmonary and trauma resuscitation in the pediatric emergency department. Pediatrics 1993;91:726-729.

Topjian AA, Berg RA, Nadkarni VM: Advances in recognition, resuscitation, and stabilization of the critically ill child. Pediatr Clin North Am 2013;60(3):605-620.

2. Is the pathophysiology of cardiopulmonary arrest in children similar to that in adults?

No. Cardiopulmonary arrests in children most commonly involve primary respiratory failure with subsequent cardiac arrest. Furthermore, cardiopulmonary arrests in children generally follow progressive deterioration and usually do not occur as sudden events. Exceptions to this statement include cases of sudden infant death syndrome (SIDS), major trauma, and certain primary cardiac events. Because arrest follows most often from primary respiratory failure in children, unlike in adult bystander cardiopulmonary resuscitation (CPR), in which only chest compressions are now emphasized, rescue breathing is still recommended during resuscitation of children.

3. What are the common causes of cardiopulmonary arrest in children?

Common causes of cardiopulmonary arrest in children are numerous, but most fit into the classifications of respiratory, infectious, cardiovascular, traumatic, and central nervous system (CNS) diseases (Table 1-1). Respiratory diseases and SIDS together consistently account for one third to two thirds of all pediatric cardiopulmonary arrests in published series.

4. What is the typical age distribution of pediatric cardiopulmonary arrests?

Almost regardless of the underlying disease, the age distribution of cardiopulmonary arrest in children is skewed toward infancy. In published series on childhood cardiopulmonary arrests, 56% (range, 43%-70%) of patients are younger than 1 year, 26% (range, 21%-30%) are between 1 and 4 years of age, and 18% (range, 6%-28%) are older than 4 years. For general emergency medicine practice settings, this finding is particularly important. Equipment and skills preparedness for this young age range are crucial to achieving best outcomes.

5. What are the outcomes of pediatric cardiopulmonary arrests?

The survival rates for children who experienced isolated respiratory arrest ranges from 73% to 97%, and survival rates for children who experienced full cardiopulmonary arrest ranges from 4% to 28%. One recent comprehensive review of 41 articles on pediatric arrest found that of 5363 out-of-hospital pediatric arrests, only 12.1% of patients survived until discharge and only 4% were neurologically intact. Another study on out-of-hospital pediatric cardiac arrests prospectively followed 474 patients and found that only 1.9% survived to discharge. A multicenter registry of 3419 in-hospital arrests found somewhat better outcomes: 27.9% survived until discharge, but only 19% had favorable neurologic outcomes.

The overall poor prognosis of full cardiopulmonary arrests probably reflects the terminal nature of asystole, which is often preceded by prolonged respiratory insufficiency and its

Table 1-1. Common Causes of Cardiopulmonary Arrest in Children

Respiratory	**Central Nervous System**
Pneumonia	Seizures, or complications thereof
Near drowning	Hydrocephalus, or shunt malfunction
Smoke inhalation	Tumor
Aspiration and obstruction	Meningitis
Apnea	
Hemorrhage	
Suffocation	**Other**
Bronchiolitis	Trauma
Cardiovascular	Sudden infant death syndrome
Congenital heart disease	Anaphylaxis
Congestive heart failure	Gastrointestinal hemorrhage
Pericarditis	Poisoning
Myocarditis	
Arrhythmia	
Septic shock	

resultant long-standing tissue hypoxemia and acidosis. This is one reason why initial management is directed toward improvement of oxygenation and ventilation.

Donoghue AJ, Nadkarni V, Berg RA, et al: Out-of-hospital pediatric cardiac arrest: An epidemiologic review and assessment of current knowledge. Ann Emerg Med 2005;46:512-522.

Lopez-Herce J, Garcia C, Dominguez P, et al: Outcome of out-of-hospital cardiorespiratory arrest in children. Pediatr Emerg Care 2005;21:807-815.

Matos RI, Watson RS, Nadkarni VM, et al: Duration of cardiopulmonary resuscitation and illness category impact survival and neurologic outcomes for in-hospital pediatric cardiac arrests. Circulation 2013;127(4):442-451.

Nadkarni VM, Larkin GL, Peberdy MA, et al: First documented rhythm and clinical outcome from in-hospital cardiac arrest among children and adults. JAMA 2006;295:50-57.

6. What are some prognostic factors for pediatric cardiopulmonary arrests?

Some factors that appear to be prognosticators of outcome for arrests include location (in or out of hospital), resuscitation at the scene, presenting rhythm, length of resuscitation, and whether drowning or trauma was involved.

For out-of-hospital arrests, bystander or paramedic initiation of resuscitation of witnessed arrest has repeatedly been found to improve survival as much as fourfold compared to initial resuscitation by physicians after patient arrival at the hospital.

Survival of patients presenting in ventricular fibrillation (VF) is much higher than among those in asystole, severe bradycardia, or pulseless electrical activity (PEA). Prolonged resuscitation over 20 minutes is often thought to be the strongest indicator of fatality, with chance of survival decreasing by 2.1% per minute in one large study of 3419 pediatric arrests. Overall, trauma- and submersion injury–associated arrests are associated with better survival rates compared with isolated cardiac-origin arrests (21.9% and 22.7% versus 1.1%, respectively). However, those with blunt traumas are about three times less likely to survive compared to those with penetrating traumas.

Outcomes can also vary greatly based on region; for instance, even in a relatively homogeneous society such as Japan, the 1-month survival rate ranged from 5.8% to more than double that rate, 12.2%. This variance is likely due to statistically different variations in factors

such as patient age, CPR initiation and type, emergency medical services (EMS) responsiveness, and their use of epinephrine and intubation.

De Maio VJ, Osmond MH, Stiell IG, et al: Epidemiology of out-of hospital pediatric cardiac arrest due to trauma. Prehosp Emerg Care 2012;16(2):230-236.

Okamoto Y, Kwami T, Kitamura T, et al: Regional variation in survival following pediatric out-of-hospital cardiac arrest. Circ J 2013;77(13):2596-2603. Epub 2013 Jul 4.

Scribano PV, Baker MD, Ludwig S: Factors influencing termination of resuscitative efforts in children: A comparison of pediatric emergency medicine and adult emergency medicine physicians. Pediatr Emerg Care 1997;13:320-324.

7. Does the initial approach to childhood resuscitation differ from that for adults?

Historically, the initial approach to adult resuscitation is similar to that for children: A (airway), B (breathing), C (circulation/compression), D (drugs), E (exposure). Attention to proper positioning, oxygenation, and ventilation comes first, and drug therapy comes last. However, in recent years, there has been strong interest in initiating chest compressions earlier, as each minute of delay may result in a 10% decreased chance of survival. The C (compression) A (airway) B (breathing) sequence is generally accepted, especially for adult patients. Because the majority of pediatric arrests are primarily respiratory in nature, adoption of CAB(DE) over ABC(DE) for pediatric patients is not widespread, despite one study showing a statistically significant 24-second advantage to chest compressions in CAB in simulated pediatric cardiac arrests.

Whether one does ABC or CAB, it is always advisable to preassign resuscitation duties to available staff. This preparation eliminates confusion during the heat of the action. Care should always be taken to protect the cervical spine (and spinal cord) during resuscitation, especially during manipulation of the neck and jaw. Some authors suggest that nearly simultaneous initiation of airway alignment and compressions may be the appropriate compromise for pediatric resuscitation.

Lubrano R, Cecchetti C, Bellelli E, et al: Comparison of times of intervention during pediatric CPR maneuvers using ABC and CAB sequences: A randomized trial. Resuscitation 2012;83(12):1473-1477. Epub 2012 May 8.

8. After establishing a clear chain of command and assigning specific duties to all members of the resuscitation team, what is the order of priorities?

The order of priorities is:

1. Identify the patient's level of responsiveness.
2. Properly position the patient on a firm surface, considering the potential for head or cervical spine injury.
3. Establish a patent airway.
4. Assure proper oxygenation and ventilation.
5. Attend to circulation.
6. Consider drug therapy.

9. What is the recommended way to establish a patent airway?

- The first attempt to establish airway patency should be through *proper airway positioning*. Often, this step alone will be effective. Because most airway obstruction is due to the effect of gravity on the mandibular block of soft tissues, it can be relieved by either a head-tilt chin-lift or jaw-thrust maneuver.
- Vomitus or other foreign material can also obstruct airways. Inspect the airway for these materials, and *suction early and frequently*.
- In selected patients with altered levels of consciousness, *nasopharyngeal or oropharyngeal airway stents* are useful. Semiconscious children generally tolerate nasopharyngeal airways better than oropharyngeal airways. Children, such as those in postictal states, who have sustained spontaneous respiratory effort but have upper airway obstruction due to poor muscle tone often benefit from the use of these devices.
- Although jumping straight to intubation is often tempting, proper positioning with *appropriately sized mask and bag-valve device* is often the most efficacious way to quickly intervene and immediately manage an airway during resuscitations.
- The *laryngeal mask airway* is a relatively new and underused supraglottic advanced airway device that may be a very useful tool to the experienced user in certain situations.

10. What is the recommended way to deliver supplemental oxygen to a child?

Supplemental oxygen can be delivered to a child by a variety of different means. For the sickest patients, oxygen should be delivered in the highest concentration and by the most direct method possible. Children who demonstrate spontaneous breathing might require less invasive means of administration of supplemental oxygen. Table 1-2 lists some different methods of oxygen delivery with their associated delivery capabilities.

Children without adequate spontaneous breathing effort require mechanical support. Different bag-valve-mask devices have different oxygen delivery capabilities. Self-inflating bag-valve devices are capable of delivering 60% to 90% oxygen, but non–self-inflating devices (anesthesia ventilation systems) deliver 100% oxygen to the patient. Endotracheal intubation offers the most secure and direct means of delivery of 100% oxygen to the patient.

American Heart Association: 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Part 10: Pediatric Basic and Advanced Life Support. Circulation 2010;122:S466-S515.

11. Which children require intubation?

Although the most obvious indication for endotracheal intubation is sustained apnea, a number of other indications exist:

- Inadequate CNS control of ventilation
- Functional or anatomic airway obstruction
- Strong potential for developing airway obstruction (e.g., inhalation airway burns, expanding airway hematoma)
- Loss of protective airway reflexes
- Excessive work of breathing, which might lead to fatigue and respiratory insufficiency
- Need for high airway pressures to maintain effective alveolar gas exchange
- Need for mechanical ventilatory support
- Potential occurrence of any of the preceding during patient transport

In many instances, bag-mask ventilation and bag–endotracheal tube ventilation are equally effective for the patient. In such circumstances, it is logical to employ the method that the rescuer is best able to deliver. One prospective study randomized the use of bag-mask ventilation and endotracheal intubation by paramedics in 830 out-of-hospital pediatric arrests. There was no significant difference in survival (30% versus 26%, respectively) or good neurologic outcome (23% versus 20%) between the two groups of children. Subsequent studies confirmed that bag-mask ventilation is preferred in the field given the high incidence of intubation-related complications in patients managed by prehospital providers in the United States. In Europe, where physicians, and not paramedics, manage patients at the scene, intubation is strongly preferred and complications are minimal.

DiRusso SM, Sullivan T, Risucci D, et al: Intubation of pediatric trauma patients in the field: Predictor of negative outcome despite risk stratification. J Trauma 2005;59:84-90.

Gausche M, Lewis RJ, Stratton SJ, et al: Effect of out-of-hospital pediatric endotracheal intubation on survival and neurological outcome: A controlled clinical trial. JAMA 2000;283:783-790.

Gerritse BM, Draaisma JM, Schalkwjk A, et al: Should EMS paramedics perform paediatric tracheal intubation in the field? Resuscitation 2008;79:225-229.

Martinon C, Duracher C, Blanot S, et al: Paediatric prehospital tracheal intubation: What makes different our practice across the ocean? Resuscitation 2010;81(5):634.

Table 1-2. Methods of Oxygen Delivery and Their Delivery Capabilities

Nasal cannula: 30-40% oxygen
Simple masks: 30-60% oxygen
Partial rebreather masks: 50-60% oxygen
Oxygen tents: 30-50% oxygen
Oxygen hoods: 80-90% oxygen
Nonrebreather masks: ~100% oxygen

12. When selecting an endotracheal tube (ETT), what sizing guidelines are suggested?

There are a number of ways to ensure selection of properly sized ETTs for children. The most often cited is the following age-based formula:

$$\text{ETT internal diameter (mm)} = (16 + \text{years of age})/4$$

Another "rule of thumb" is really a "rule of finger." Research has demonstrated that the width of the child's fifth fingernail is approximately equal to the outer width of the appropriately sized ETT. Most emergency physicians use uncuffed tubes for children younger than 10 years, because in these patients, the anatomic narrowing at the level of the cricoid cartilage provides a natural "cuff." However, in the in-hospital setting, a cuffed tube has been shown to be as safe as an uncuffed tube for infants beyond the newborn period. In some circumstances (e.g., poor lung compliance, high airway resistance, or a large glottic leak), a cuffed tube may be preferable.

American Heart Association: 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Part 10: Pediatric Basic and Advanced Life Support. Circulation 2010;122:S466-S515.

Key Points: How to Determine the Proper Placement of the Endotracheal Tube

1. Check to see that the tube is inserted at a depth that is three times the internal diameter of the ETT (from the point of the patient's central incisors).
2. Observe for symmetric chest expansion.
3. Auscultate for symmetric breath sounds.
4. Look for distention of the abdomen, indicating misplacement of the tube.
5. Measure end-tidal carbon dioxide using a colorimetric detector. In infants and children with a perfusing rhythm, a purple color on the device indicates a problem, whereas a yellow color implies that the tube is in the trachea.
6. Confirm tube placement with a chest radiograph.

13. How can I determine if the ETT is appropriately placed?

Proper depth for ETT insertion from the point of the patient's central incisors can be estimated to be three times the internal diameter of the ETT. Measurement of end-tidal carbon dioxide using a colorimetric detector, observation for symmetric chest expansion, and auscultation for symmetric breath sounds can help to ensure proper placement. Confirmation of placement is probably best determined with a chest radiograph. Prior to a chest radiograph, the colorimetric detector offers a rapid bedside determination to detect CO_2 to confirm ETT placement (Fig. 1-1).

14. What are the best methods to assess a child's circulatory status?

Assessment of a child's circulatory status should always include appraisal of the following:

- Skin and mucous membrane color
- Presence and quality of pulses
- Capillary refill
- Heart rate and blood pressure

Always keep in mind that in the instance of acute blood loss, the protective mechanisms of increased heart rate and increased vascular resistance maintain a child's blood pressure within a normal range in spite of losses as high as 25% of total body blood volume.

15. What is the pediatric assessment triangle (PAT)?

The PAT is a visual and auditory assessment tool developed for rapid standardized assessment of pediatric patients. As seasoned pediatricians know, you can often identify ill infants and children even without any equipment. The PAT emphasizes a quick evaluation of a patient in three main areas: (1) appearance, (2) work of breathing, and (3) circulation to skin, to form a general impression of the child's condition (Fig. 1-2). Based on an assessment of normal or abnormal, patients can be categorized in different physiologic categories ranging from "stable" to "respiratory distress" to "decompensated shock" and full "cardiopulmonary failure" (Table 1-3). The PAT is now widely accepted and taught to prehospital specialists in pediatric advanced life

Figure 1-1. Colorimetric device. In infants and children with a perfusing rhythm, a purple color on the device indicates a problem, whereas a yellow color implies that the tube is in the trachea.

support (PALS and APLS). To be clear, however, it is an initial quick assessment and not meant to take the place of ABCDEs.

Dieckmann RA, Brownstein D, Gausche-Hill M: The pediatric assessment triangle: A novel approach for the rapid evaluation of children. Pediatr Emerg Care 2010;26(4):312-315.

16. To whom and how should external cardiac compression be delivered?

Apply external cardiac compression to any child with ineffective pulses. A compressions-to-ventilations ratio of 30:2 is recommended for the lone rescuer performing CPR in infants and

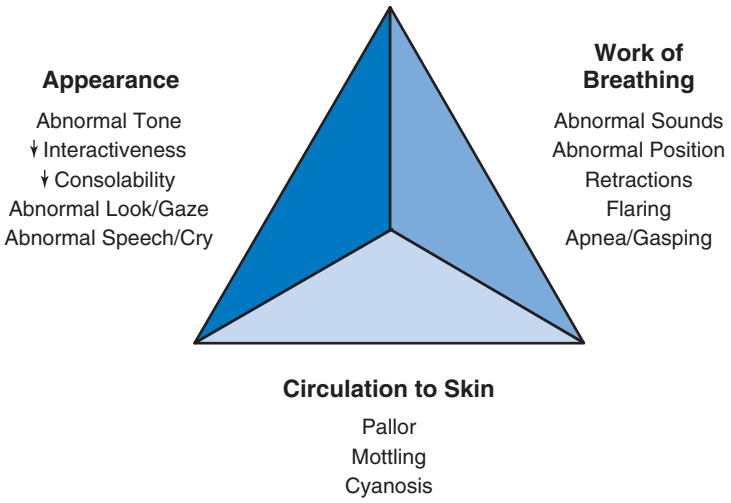

Figure 1-2. The pediatric assessment triangle. *(From Horeczko T, Enriquez B, McGrath NE, et al: The pediatric assessment triangle: Accuracy of its application by nurses in the triage of children. J Emerg Nurs 2013;39(2):182-189. Epub 2012 Jul 24.)*

Table 1-3. Pediatric Assessment Triangle and Patient General Impression

	STABLE	RESPIRATORY DISTRESS	RESPIRATORY FAILURE	SHOCK	CENTRAL NERVOUS SYSTEM/METABOLISM DISTURBANCE	CARDIOPULMONARY FAILURE
Appearance of patient	Normal	Normal/ abnormal	Abnormal	Normal/ abnormal	Abnormal	Abnormal
Work of breathing	Normal	Abnormal	Abnormal	Normal	Normal	Abnormal
Circulation to skin	Normal	Normal	Normal/ abnormal	Abnormal	Normal	Abnormal

Adapted from Dieckmann RA, Brownstein D, Gausche-Hill M (eds): Pediatric Education for Prehospital Professionals: PEPP Textbook. Sudbury, MA, Jones & Bartlett Publishers, 2000.

children. If two rescuers are performing CPR, a compressions-to-ventilations ratio of 15:2 is recommended. When a tracheal tube is placed, compressions should not be interrupted for ventilations.

It takes a number of compressions to raise coronary perfusion pressure, which drops with each pulse. Interruptions in chest compressions are associated with a decreased rate of return of spontaneous circulation. It is currently recommended that in infants, compressions be applied evenly over the lower half of the sternum. Deliver chest compressions at a rate of 100 per minute: "push fast" and "push hard." The two thumb–encircling hands technique may be preferred for two-rescuer CPR because it produces higher coronary perfusion pressure and more consistently results in appropriate depth of compression. But either a one- or two-hand technique can be used to perform chest compressions in children. For children and adolescents, compress the lower half of the sternum with the heel of one hand or with two hands, but do not press over the xiphoid process or ribs.

American Heart Association: 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Part 13: Pediatric Basic Life Support. Circulation 2010;122:S862-S875.

17. What are the golden rules of vascular access?

1. First attempt the technique that has yielded best personal success.
2. One small-gauge line beats none at all.

The messages are obvious. During resuscitation, procedures should be done by those most talented, and they should do what they do best. Although it is better to have large-gauge vascular access for resuscitation, small-gauge vascular access is adequate to deliver medications and slower infusions of fluids.

18. What are the options for vascular access in children?

There are many options for vascular access in children. Depending on the situation at hand, some might not be as available or achievable as others. Conditions permitting, *peripheral venous access* is generally preferred over other means. Antecubital, hand, wrist, foot, and ankle veins are the most popular access sites. Saphenous veins in the ankle are deep but often accessible. External jugular veins are also reliably accessible but require difficult positioning of the child to be successful. Scalp veins are potential sites of access in infants but might be difficult to access while managing the patient's airway.

Central access sites include bone marrow, femoral veins, and subclavian veins. Subclavian access should be attempted only by those skilled in the procedure. Consider intraosseous (IO) access early when venous access cannot be obtained, especially in the case of apnea and pulselessness in an infant.

19. Why does intraosseous infusion work?

The bone marrow serves as a "stiff" vascular bed. It is composed of interconnected sinusoids that are fed and drained by veins that traverse the cortex of the bone and connect with the central circulation. Fluids infused anywhere into the marrow cavity enter these vascular channels and find their way to the central venous system. In animal models, transit times from the tibia to the heart are short (less than 60 seconds). Numerous medications and fluids have been shown to be effective when administered via this route.

20. What are the do's and don'ts surrounding intraosseous (IO) infusion?

Although there is no age limit for use of IO infusion, it may be easier to accomplish in younger patients, whose bones are less calcified. Remember that IO infusion was developed in the 1930s as a technique of vascular access in adults. Numerous studies using adult patients have demonstrated a cumulative 98% success rate. Preferred sites of IO needle placement are the proximal tibia in children younger than 2 years and the distal tibia in those age 2 or older. The distal femur may also be used. Any intravenous (IV) fluid or medication can be safely and effectively administered via the IO route. Rates of infusion are limited by needle gauge and length. When infusion is delivered with pressure, flow rates of saline through 20-G needles have been measured as high as 25 mL/minute. Do not attempt IO infusion in a bone that is fractured or when previous attempts have punctured the bone.

21. Has opinion about the use of intraosseous (IO) access in recent years changed?

Yes. Although IO needle placement should never be taken lightly, as it is painful and time-limited in its effectiveness, the literature increasingly supports its use as an important primary vascular access method in emergencies. Underutilization may be due to fears of complications such as needle damage (bending or breaking), fluid extravasation at the needle entry site, and puncture through both sides of the bone (it is important to note that IO infusion will not succeed with bones that have breaks or holes in them because fluid will extravasate through these openings). However, once placement is established, actual IO complications appear to be very rare.

Enhanced interest in the use of IO access is due, at least in part, to there now being several different IO battery-powered drill systems available. These drills are very effective: they are fast, use larger gauge needles that are less prone to bending, and do not require strong force to break the plane of the cortex. They also appear to gain access more quickly than traditional manual IO needles, on average within 67 seconds in one study of paramedics in prehospital scenarios.

Byars DV, Tsuchitani SN, Erwin E, et al: Evaluation of success rate and access time for an adult sternal intraosseous device deployed in the prehospital setting. Prehosp Disaster Med 2011;26(2):127-129.

Hansen M, Meckler G, Spiro D, Newgard C: Intraosseous line use, complications, and outcomes among a population-based cohort of children presenting to California hospitals. Pediatr Emerg Care 2011;27(10):928-932.

Voigt J, Waltzman M, Lottenberg L: Intraosseous vascular access for in-hospital emergency use: A systematic clinical review of the literature and analysis. Pediatr Emerg Care 2012;28(2):185-199.

22. What role does drug therapy play in pediatric resuscitation?

Drug therapy during resuscitation is reserved for patients who do not respond adequately to the ABCs. Other than oxygen, most pediatric resuscitations require few drugs. Other useful chemical agents include the following:

- Epinephrine (to increase heart rate, myocardial contractility, and systemic vascular resistance)
- Atropine (to increase heart rate in nonneonates)
- Dextrose (to increase glucose)
- Amiodarone or procainamide (to reverse ventricular arrhythmias)
- Naloxone (to reverse the effects of narcotics)
- Adenosine (to reverse supraventricular tachycardia)
- Dopamine (to increase vasoconstriction and blood pressure)
- Dobutamine (to increase myocardial contractility)
- Benzodiazepines (to achieve sedation and control seizures)

Keep in mind that administration of any of these drugs should never be considered as a first line of management for any situation. During resuscitation, drug therapy should always be preceded by another intervention. *Oxygenation and ventilation are always the first priorities for any seriously ill child.* Other appropriate supportive measures (e.g., chest compressions for pulselessness or fluid infusion for shock) should also precede administration of drugs during resuscitation. Note: There is insufficient evidence to support the routine use of atropine in pediatric cardiac arrest.

23. What are the PALS recommendations for pulseless arrest (PEA, asystole)?

- Give epinephrine for PEA or asystole intravascularly (IV or IO route) as a standard dose (0.01 mg/kg). This can be delivered as 0.1 mL/kg of a 1:10,000 solution of epinephrine. Use of vasopressin is considered *class indeterminate* (not enough evidence to recommend for or against) in pediatric arrests.
- If epinephrine for PEA or asystole is administered by the ETT, give as a higher dose (0.1 mg/kg). This dose can be delivered as 0.1 mL/kg of a 1:1000 solution of epinephrine. An IV or IO route of administration is preferred, however, if at all possible.
- Higher-dose epinephrine (0.1 mg/kg; 0.1 mL/kg of a 1:1000 solution) is not routinely recommended for subsequent doses of epinephrine given through an IV or IO route.

24. What are the PALS recommendations for bradycardia?

- Any intravascularly (IV or IO route) administered dose should be given as a standard dose (0.01 mg/kg). This dose is generally delivered as 0.1 mL/kg of a 1:10,000 solution of epinephrine.
- Any doses administered by the ETT should be given as higher doses (0.1 mg/kg). This can be delivered as 0.1 mL/kg of a 1:1000 solution of epinephrine.

American Heart Association: 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Part 10: Pediatric Basic and Advanced Life Support. Circulation 2010;122:S466-S515.

25. Which resuscitation drugs are effective when given via an ETT?

The preferred routes of drug delivery for infants and children in cardiac arrest are IV and IO. However, there are four "traditional" resuscitation drugs that are effective when administered through the ETT. Those four are *l*idocaine, *a*tropine, *n*aloxone, and *e*pinephrine. The acronym LANE is an easy way to remember them. Versed (midazolam) also is useful and is effective when administered via the ETT. Adding this drug to the list yields a different acronym: NAVEL. With the exception of epinephrine, endotracheal doses are the same as intravascular doses. All doses of epinephrine given through the ETT should be a higher dose (0.1 mg/kg).

American Heart Association: 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Part 10: Pediatric Basic and Advanced Life Support. Circulation 2010;122:S466-S515.

Key Points: Drugs That Can Be Given via the Endotracheal Route

1. Lidocaine
2. Atropine
3. Naloxone
4. Epinephrine
5. Versed

26. Are there minimum dosing requirements for any resuscitation drugs?

- **Atropine** (usual dose, 0.02 mg/kg) has a minimum dosing requirement for effective reversal of bradycardia. It appears that at doses lower than 0.1 mg, atropine exerts an effect that might actually worsen bradycardia. Thus, if its use is considered for reversal of bradycardia in a child who weighs less than 5 kg, a minimum of 0.1 mg should be administered.
- **Dopamine** also has different effects when administered at different doses. At lower doses (1-5 μg/kg/minute), dopaminergic effects are seen. When administered at these lower doses, dopamine tends to augment renal blood flow and enhance urinary output. During resuscitation, dopamine typically is used to bolster blood pressure through increased vasoconstriction. For that α-adrenergic effect, higher doses (10-20 μg/kg/minute) are required.

27. What are the recommendations for use of adenosine?

Adenosine is the drug of choice in the acute management of supraventricular tachycardia. It is a short-acting agent (half-life of approximately 10 seconds) that slows atrioventricular node conduction. The initial dose is 0.1 mg/kg, given as a rapid intravascular push with an immediate saline flush. If the drug is not given rapidly, its effectiveness is diminished. If the first dose is properly administered but ineffective, give a larger second dose of 0.2 mg/kg. Usual adult doses are 6 mg (first dose), followed by 12 mg (second dose). Expect that the first dose might be completely ineffective or only transiently effective. Administration of subsequent higher doses generally yields success.

Dixon J, Foster K, Wyllie J, Wren C: Guidelines and adenosine dosing in supraventricular tachycardia. Arch Dis Child 2005;90:1190-1191.

28. Does calcium have any usefulness in pediatric resuscitations?

The American Heart Association does not recommend the routine use of calcium in pediatric cardiac arrest. Although the use of calcium during resuscitation has declined considerably, there remain specific instances when it has significant value. Use calcium to remedy the following situations:

- Documented hypocalcemia
- Documented hyperkalemia
- Documented hypermagnesemia
- Calcium channel blocker excess

When administered, calcium should be infused slowly. Rapid infusion results in severe bradycardia. Take care to avoid back-to-back infusion of calcium and sodium bicarbonate–containing solutions. If mixed, these agents form calcium carbonate (chalk) in the IV tubing.

American Heart Association: 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Part 10: Pediatric Basic and Advanced Life Support. Circulation 2010;122:S466-S515.

29. Does sodium bicarbonate have a role in pediatric resuscitations?

Sodium bicarbonate is not recommended for routine use in pediatric resuscitations. Although it is a useful agent for the reversal of documented metabolic acidosis, it is effective only in the presence of adequate ventilation. When bicarbonate combines with hydrogen, it forms a complex molecule that splits into carbon dioxide and water. The carbon dioxide has only one route of exit, the respiratory tract. Without effective ventilation, this by-product is not removed and the buffering capacity of the bicarbonate is eliminated. A randomized, controlled trial found no benefit from sodium bicarbonate use in neonatal resuscitation. After provision of effective ventilation and chest compressions and administration of epinephrine, consider sodium bicarbonate for prolonged cardiac arrest.

American Heart Association: 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Part 10: Pediatric Basic and Advanced Life Support. Circulation 2010;122:S466-S515.

Lokesh L, Kumar P, Murki S, Narang A: A randomized controlled trial of sodium bicarbonate in neonatal resuscitation—Effect on immediate outcome. Resuscitation 2004;60:219-223.

30. Is there an easy method to calculate mixtures of constant infusions of drugs?

Several methods are used. Here is one easy method:

- For constant infusion of drugs (epinephrine, isoproterenol) beginning at *0.1 μg/kg/minute*: 0.6 times the weight (in kg) equals the number of milligrams of drug to add to enough water to make a total of 100 mL of solution. The resultant solution is then infused at a rate of 1 mL/hour, delivering 0.1 μg/kg/minute.
- For constant infusion of drugs (dopamine, dobutamine) beginning at *1 μg/kg/minute*: 6 times the weight (in kg) equals the number of milligrams of drug to add to enough water to make a total of 100 mL of solution. The resultant solution is then infused at a rate of 1 mL/hour, delivering 1 μg/kg/minute.

31. What role does defibrillation play in pediatric resuscitation?

Historically, pediatric resuscitation has focused on pulmonary causes; defibrillation is a relatively uncommon intervention in pediatric resuscitation. Although asystole remains the most commonly observed arrhythmia during pediatric cardiac arrests, recent research indicates that VF may occur much more frequently than originally thought. The National Registry of Cardiopulmonary Resuscitation, the largest inpatient pediatric cohort reported to date, found VF occurred in 14% of pediatric arrests. In that study, pediatric patients with VF had a higher survival rate (29%) than those with asystole (24%) or PEA (11%). A study of out-of-hospital pediatric arrests found VF as the presenting rhythm in 17.6% of cases, with children older than 7 years of age having the highest incidence (38/141, 27.0%). Survival of patients with VF was threefold greater (31.3% versus 10.7%) than in those without a shockable rhythm.

Smith BT, Rea TD, Eisenberg MS: Ventricular fibrillation in pediatric cardiac arrest. Acad Emerg Med 2006;13:525-529.

32. How is defibrillation best accomplished?

In any resuscitation, carefully check the rhythm after airway and breathing are established. Carefully confirm VF before defibrillation is attempted. Unmonitored defibrillation of a child is not recommended.

Defibrillation works by producing a mass polarization of myocardial cells with the intent of stimulating the return of a spontaneous sinus rhythm. Once VF is diagnosed, prepare the patient for defibrillation and correct acidosis and hypoxemia. High-amplitude (coarse) fibrillation is more easily reversed than low-amplitude (fine) fibrillation. Administration of epinephrine can help coarsen fibrillation.

Defibrillation is most effective with use of the largest paddle that makes complete contact with the chest wall. Using the larger (8-cm diameter) paddle lowers the intrathoracic impedance and increases the effectiveness of the defibrillation current.

Take care to use an appropriate interface between the paddles and the chest wall. Electrode cream, paste, or gel pads are preferred when using paddles. Do not use saline-soaked gauze pads, ultrasound gel, alcohol pads, or bare paddles. Whenever available and if time allows, place and use self-adhesive defibrillation pads instead of paddles, as they allow for safer and more efficient shock delivery and then can be used for cardiac pacing when appropriate.

Whether gel, paste, or pads are used, placement must be meticulous, because electrical bridging across the surface of the chest results in ineffective defibrillation and, possibly, skin burns. When attempting defibrillation, immediate CPR should follow the delivery of one shock, rather than delivery of up to three shocks before CPR. This recommendation is based on the fact that the first shock eliminates VF 85% of the time, and studies have shown long delays typically occur between shocks when automated external defibrillators (AEDs) are used.

For defibrillation of the pediatric patient use an initial dose of 2 to 4 J/kg.

American Heart Association: 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Part 10: Pediatric Basic and Advanced Life Support. Circulation 2010;122:S466-S515.

33. What treatment is recommended for VF or pulseless ventricular tachycardia (VT) if electric shock is not effective?

Amiodarone may be used for the treatment of shock-refractory or recurrent VF/pulseless VT in infants and children. If amiodarone is not available, consider the use of lidocaine. There are no pediatric data investigating the efficacy of lidocaine for shock-refractory VF/pulseless VT.

American Heart Association: 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Part 10: Pediatric Basic and Advanced Life Support. Circulation 2010;122:S466-S515.

34. Are AEDS useful for children with sudden collapse?

Initially AEDs were not recommended for use in children under 8 years of age; however, newer models of AEDs have been shown to reliably recognize shockable rhythms in children. PALS guidelines as of 2010 now recommend use of AEDs for children 1 year of age and older. An AED can also be used in infants younger than 1 year of age if an attenuator for it is available, or if there are no alternatives (i.e., no manual defibrillator is available).

American Heart Association: 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Part 10: Pediatric Basic and Advanced Life Support. Circulation 2010;122:S466-S515.

35. Is induced hypothermia useful in treating children with cardiac arrest?

There are no randomized studies on induced therapeutic hypothermia following cardiac arrest in pediatric patients. Therapeutic hypothermia (32° C to 34° C) may be beneficial for adolescents who remain comatose following resuscitation from sudden witnessed out-of-hospital VF cardiac arrest. Consider this for infants and children who remain comatose following resuscitation from cardiac arrest.

American Heart Association: 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Part 10: Pediatric Basic and Advanced Life Support. Circulation 2010;122:S466-S515.

36. Should families be cleared from the resuscitation room when treating children with cardiac arrest?

In general, family members should be offered the opportunity to be present during the resuscitation of their infant or child. Multiple studies indicate that parents prefer to be given the option to remain in the room. Many relatives believe their presence is helpful to the patient, and some studies show that being present during the resuscitation helped their adjustment to the family member's death.

CHAPTER 2

NEONATAL RESUSCITATION

Constance McAneney

1. What physiologic changes take place during the transition from intrauterine to extrauterine life?

The cardiopulmonary systems undergo a rapid change from fetal to extrauterine life. At birth the umbilical cord is clamped, and systemic vascular resistance rises. With the newborn's first breaths (increasing the neonate's Pa_{O_2} and pH), pulmonary vascular resistance decreases, thereby causing an increase in pulmonary blood flow. Blood flow through the foramen ovale and the ductus arteriosus reverses direction, and then these structures eventually close. The ductus arteriosus is usually closed functionally by 15 hours of age.

If the pulmonary vascular resistance does not fall adequately, a persistent right-to-left shunt will occur (persistent pulmonary hypertension). Inability to expand alveolar spaces can cause intrapulmonary shunting of blood (hypoxia). Disruption of fetal-maternal circulation (placenta previa, abruptio placentae) can result in acute blood loss and hypovolemia in the newly born infant.

Aronson PL, Alessandrini EA: Neonatal resuscitation. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010.

2. What preparation is necessary to have for the unexpected emergency department (ED) delivery?

Preparation is key, as most ED deliveries are "unexpected." A prearranged plan should be set in motion as soon as birth is imminent. That plan should include the assembly of personnel who are best able to take care of the newly born infant. A brief history should be obtained if possible, because it may affect the resuscitation. Most newborns who will need resuscitative interventions can be identified prior to birth. Equipment and medications specifically for a neonatal resuscitation should be kept in a designated tray so they are quickly available (Table 2-1). Periodic inspection of this equipment for proper functioning and expiration dates of medication should become part of the routine upkeep of the neonatal resuscitation tray.

3. What are the critical facts in the history that should be elicited, if possible, prior to delivery?

The standard maternal history is important but may need to wait until after delivery because of the imminent birth of the infant. Critical information may need to be narrowed to facts that may affect the immediate preparation (equipment and personnel) for the delivery.

It is important to ask if the expectant mother knows *if she is having twins*. Additional resuscitation equipment as well as personnel should then be quickly gathered. Ideally there should be a resuscitation area, equipment, and personnel for each expected newly born infant.

The *expected due date* is crucial to determine if the newly born infant will be premature and, if so, approximately how premature. Infants born at less than 36 weeks' gestation are more likely to be born "unexpectedly" and will more likely need resuscitation. Smaller caliber equipment will be needed.

The *color of the amniotic fluid* is important. If the fluid is meconium-stained (greenish), then one should anticipate a distressed newly born infant with or without airway obstruction from the meconium. The infant may require intubation with suctioning. Equipment should be available, and personnel should be aware of this clinical situation.

Hazinski MF (ed): Textbook of Pediatric Advanced Life Support. Dallas, American Heart Association, 2002.

4. How do you assess the condition of a newly born infant?

In assessing the newly born infant, three basic questions should be asked:

1. Is the newly born infant at term gestation?
2. Is the newly born infant crying or breathing?
3. Does the newly born infant have good muscle tone?

Table 2-1. Equipment and Drugs for the Neonatal Resuscitation

Equipment
Gowns, gloves, and masks
Warm towels and blankets
Bulb syringe
Meconium aspirator
Suction catheters (sizes 5-10 F)
Face masks (sizes premature, newborn, and infant)
Oral airways (sizes 000, 00, 0)
Anesthesia bag with manometer (preferably 500 mL, no larger than 750 mL)
Laryngoscope with straight blades (sizes 0 and 1)
Spare bulbs and batteries
Stethoscope
Endotracheal tubes (sizes 2.5, 3.0, 3.5, 4.0) and stylet
Tape
Umbilical catheters (3.5 and 5 F)
Umbilical catheter tray
Three-way stopcocks
Nasogastric feeding tubes (8 and 10 F)
Needles and syringes
Chest tubes (8 and 10 F)
Magill forceps
Radiant warmer
Cardiorespiratory monitor with electrocardiogram leads
Pulse oximeter with neonatal probes
Suction equipment
Oxygen source with flowmeter and tubing
End-tidal CO_2 detector
Laryngeal mask airway (optional)
Drugs
Epinephrine 1:10,000
Naloxone
Sodium bicarbonate
Dextrose in water 10%
Normal saline, Ringer's lactate
Resuscitation drug chart

If the answers to all of these questions are "yes," then the newly born can remain with the mother. The infant does not require routine suctioning of the nose and mouth. The baby should be dried and placed on the mother, skin-to-skin. Cover with dry linen and observe breathing, color, and activity. Delay cord clamping for at least 1 minute for newborns not requiring resuscitation.

Table 2-2. Apgar Score Chart

	Score		
SIGN	**0**	**1**	**2**
Heart rate	Absent	Slow (<100/min)	>100/min
Respirations	Absent	Slow, irregular	Good, crying
Muscle tone	Limp	Some flexion	Active motion
Reflex irritability (catheter in nares)	No response	Grimace	Cough, sneezes
Color	Blue or pale	Pink body with blue extremities	Completely pink

The newly born infant should be assessed and assigned an Apgar score at 1 minute and at 5 minutes of life (Table 2-2). The Apgar score assesses heart rate, respirations, muscle tone, reflex irritability, and color. It indicates how the infant is doing or the responsiveness to the resuscitation but is not an indicator to initiate resuscitation. If the Apgar score is less than 7 at 5 minutes, then the scoring continues every 5 minutes for 20 minutes. *Do not delay resuscitative efforts to obtain an Apgar score.*

If any answer to the three basic questions (term, breathing or crying, good tone) is "no," then be prepared to initiate action in one of these four categories:

1. Initial steps in stabilization (warm, clear airway, dry, stimulate)
2. Ventilation
3. Chest compressions
4. Administration of epinephrine and volume expansion

Within the first 60 seconds, or the "golden minute," the initial steps should be completed and ventilation (if warranted) begun.

Aronson PL, Alessandrini EA: Neonatal resuscitation. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010.

Kattwinkel J, Perlman JM, Aziz K, et al: Part 15: Neonatal resuscitation: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation 2010;122(18 Suppl 3):S909-S919.

Key Points: Recommendations for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care

1. Prevent hypothermia.
2. Intrapartum routine suctioning of the newborn's nose and mouth is not recommended.
3. Color of the newborn is no longer used as an indicator of oxygenation or effectiveness of resuscitation.
4. Begin resuscitation of infants (term or preterm) with air or blended oxygen with the goal of preductal Spo_2 norms.
5. The laryngeal mask may be used by trained providers when bag-mask ventilation is ineffective or attempts at endotracheal intubation have been unsuccessful.
6. The two-thumb method of chest compressions is the preferred method, with the depth of compression being one third of the anteroposterior diameter of the chest rather than a fixed depth.
7. An intraosseous needle can be used for access if the umbilical vein is not readily available.
8. Administer epinephrine if the heart rate remains at or under 60 beats per minute after 30 seconds of adequate ventilation and chest compressions.

5. What are the initial steps in *stabilization* (newly born is preterm, not crying or breathing, does not have good tone)?

Because newly born infants do not tolerate cold, and hypothermia can prolong acidosis, heat loss should be prevented as much as possible. Dry the infant of the amniotic fluid and place the

newborn in a slight Trendelenburg position, on his or her back with the neck slightly extended, under a prewarmed radiant warmer. Clean the airway, *if necessary*, with a bulb syringe or suction catheter. Do not suction more than 5 seconds and do not pass the tip farther than 5 cm. Vigorous suctioning will cause bradycardia. Therefore, if the amniotic fluid is clear and the newly born shows no signs of obstruction or need for positive-pressure ventilation, then suctioning, even with a bulb syringe, is not indicated. Usually by drying the infant, he or she is adequately stimulated to begin effective respirations. Avoid vigorous stimulation.

Meyer MP, Bold GT: Admission temperatures following radiant warmer or incubator transport for preterm infants <28 weeks: A randomised study. Arch Dis Child Fetal Neonatal Educ 2007;92: F295-F297.

6. How can a very low birth weight premature newborn infant (<1500 g) be kept warm in the ED?

Drying and swaddling, warming pads, increased environmental temperature, and covering with a blanket have been used to keep newborns warm. These techniques have not been evaluated in controlled trials and may not be enough to warm very small newborns. Very low birth weight infants may need additional warming techniques, such as covering the infant in plastic wrapping (food-grade, heat-resistant plastic) and placing him or her under radiant heat.

Vohra S, Roberts RS, Zhang B, et al: Heat loss prevention (HeLP) in the delivery room: A randomized controlled trial of polyethylene occlusive skin wrapping in very preterm infants. J Pediatr 2004;145:750-753.

7. When does the newly born infant need assistance with ventilation?

Approximately 10% of newly born infants require some form of assistance to begin breathing at birth, and 1% require extensive resuscitation measures. After the infant has been quickly assessed and found to have apnea or gasping respirations, initiate positive-pressure ventilation with air. Also initiate positive-pressure ventilation if the heart rate is less than 100 beats per minute, because bradycardia in a newborn is usually due to hypoxia.

Kattwinkel J, Perlman JM, Aziz K, et al: Part 15: Neonatal resuscitation: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation 2010;122(18 Suppl 3):S909-S919.

8. What is the best way to determine heart rate in the newly born infant?

The best way to determine the heart rate of the newly born is by auscultation of the precordial pulse. If a pulse is detected, then palpation of the umbilical pulse is more accurate than other sites and gives a rapid estimate of the pulse while other interventions may be taking place.

Owen CJ, Wyllie JP: Determination of heart rate in the baby at birth. Resuscitation 2004;60(2):213-217.

9. When is supplemental oxygen indicated?

Controversy, controversy, controversy! There are growing data in the literature that both not enough oxygen and too much oxygen, even brief exposure to excessive levels during resuscitation, are harmful to the newly born infant. The oxygen saturation level does not reach extrauterine values until several minutes after birth, which may result in the appearance of cyanosis. It has been shown that absence of cyanosis is also a poor indicator of oxygenation after birth. Place a pulse oximeter with a neonatal probe on the newly born infant in a preductal location (right wrist or right medial surface of the palm) if resuscitation is anticipated, when positive-pressure ventilation is initiated, when cyanosis persists, or when supplemental oxygen is administered. Initiate resuscitation with air or blended oxygen and titrate with the goal of an oxygen saturation in the interquartile range of preductal saturation percentages shown in Table 2-3.

Kattwinkel J, Perlman JM, Aziz K, et al: Part 15: Neonatal resuscitation: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation 2010;122(18 Suppl 3):S909-S919.

Rabi Y, Rabi D, Yee W: Room air resuscitation of the depressed newborn: A systematic review and meta-analysis. Resuscitation 2007;72:353-363.

Vento M, Asensi M, Sastre J, et al: Resuscitation with room air instead of 100% oxygen prevents oxidate stress in moderately asphyxiated term neonates. Pediatrics 2001;107(4):642-647.

Table 2-3. Target Preductal Spo_2 After Birth

TIME	Spo_2
1 min	60-65%
2 min	65-70%
3 min	70-75%
4 min	75-80%
5 min	80-85%
10 min	85-95%

10. What is the proper technique for assisting ventilations in the newly born infant?

The mask should fit around the nose and mouth but not cover the eyes or go below the chin. Assisted ventilations should be at a rate of 40 to 60 breaths per minute (30 breaths per minute when chest compressions are being performed). The initial breaths may require higher inflation pressures and longer inflation times. Monitor inflation pressure. An inflation pressure of 20 cm H_2O may be sufficient. Regardless, the effectiveness of the assisted ventilation is judged by the movement of the chest, adequacy of breath sounds, and the heart rate. Poor face mask technique, airway obstruction, movement of the infant, interventions such as removing wet blankets, and distraction of the resuscitator contribute to ineffective mask ventilation. Mask leak and airway obstruction, being the most common reasons, may go undetected unless CO_2 detectors of residual function monitors are used.

If the condition of the neonate does not improve, then reposition the head, check for patency of the airway, improve the seal of the mask on the face, and increase the inflating pressure of the bag.

Kattwinkel J, Perlman JM, Aziz K, et al: Part 15: Neonatal resuscitation: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation 2010;122(18 Suppl 3):S909-S919.

O'Donnell C, Schmolzer GM: Resuscitation of the fetus and newborn resuscitation of preterm infants. Clin Perinatol 2012;39:857-869.

11. What are the indications for tracheal intubation of the newly born infant?

Indications for tracheal intubation vary but are based on the degree of respiratory depression, the success of ventilation efforts, the presence of meconium, the degree of prematurity, and the skill of the health care provider. There is controversy at every turn. For instance, some neonatal experts feel that early intubation in infants younger than 28 weeks is indicated, and others suggest these infants can be handled with mask or nasal prong CPAP (continuous positive airway pressure).

Endotracheal intubation is indicated if a neonate:

- Has not responded to assisted ventilations with a bag-mask
- Is extremely low birth weight
- Requires chest compressions
- Needs tracheal administration of medications
- Has signs of respiratory depression with meconium
- Has special circumstances (diaphragmatic hernia)

12. How should endotracheal intubation of the newborn be performed?

Perform the tracheal intubation by the oral route, using an uncuffed endotracheal tube and a laryngoscope with a straight blade (size 0 for premature, size 1 for term). If a stylet is used, it should not protrude beyond the end of the tube. Cricoid pressure may be needed. After the endotracheal tube is passed through the vocal cords, check the position by observing symmetrical chest wall movement, listen for breath sounds at the axilla, and note the absence of breath sounds over the stomach. Confirm the absence of gastric inflation, watching for condensation in the endotracheal tube during exhalation, and note the improvement in heart

rate, color, and activity of the newborn. A prompt increase in heart rate is the best indicator that the tube is in the tracheobronchial tree and providing effective ventilation. Confirm tube placement with a CO_2 monitor. Exhaled CO_2 detection is effective for confirmation of endotracheal placement in infants, including very low birth weight infants. Confirmation of tube placement by radiograph is also recommended.

The guide for the proper size of the endotracheal tube follows:

$$\text{Endotracheal tube size} = \text{Gestational age in weeks}/10$$

The proper depth of insertion can be estimated by using the following calculation:

$$\text{Insertion depth at lip in cm} = \text{Weight in kg} + 6\ \text{cm}$$

Kattwinkel J, Perlman JM, Aziz K, et al: Part 15: Neonatal resuscitation: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation 2010;122(18 Suppl 3):S909-S919.

13. Are airway adjuncts useful or indicated in the management of the newly born?

Yes. CPAP is widely used in infants who are breathing but exhibiting increased respiratory effort. It has been studied in preterm infants and shown to decrease intubation rates, mechanical ventilation duration, and use of surfactant, but increases rates of pneumothorax. Local expertise and comfort should guide the use of CPAP.

Laryngeal mask airways (LMAs) are adjuncts to airway management and are generally used when tracheal intubation is unable to be attained or face mask ventilation is inadequate. The newly born infant over 2000 g or 34 or more weeks of gestation can be ventilated effectively with LMAs. They have not been studied in infants with meconium-stained fluid, during chest compressions, or for the administration of tracheal medications.

Kattwinkel J, Perlman JM, Aziz K, et al: Part 15: Neonatal resuscitation: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation 2010;122(18 Suppl 3):S909-S919.

Trevisanuto D, Micaglio M, Pitton M, et al: Laryngeal mask airway: Is the management of neonates requiring positive pressure ventilation at birth changing? Resuscitation 2004;62:151-157.

14. When are chest compressions indicated in the resuscitation of the newly born infant?

Effective ventilation usually restores vital signs to normal in a newborn, and chest compressions are generally not needed. Because chest compressions make effective ventilations more difficult and heart rate usually responds to assisted ventilation, chest compressions are not initiated until assisted ventilation has been started. The indications for the initiation of chest compressions during the resuscitation of the newly born infant are absent heart rate or heart rate less than 60 beats per minute despite adequate assisted ventilation with oxygen for 30 seconds.

Kattwinkel J, Perlman JM, Aziz K, et al: Part 15: Neonatal resuscitation: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation 2010;122(18 Suppl 3):S909-S919.

15. What is the proper technique for chest compressions in the newborn?

The two acceptable techniques for performing chest compressions are applying two thumbs superimposed or next to each other on the sternum with the fingers surrounding the chest, or two fingers placed on the sternum at a right angle to the chest with the other hand supporting the back. Data suggest that the two-thumb method may have the advantage of generating peak systolic and coronary perfusion, and it is preferred by providers. Placement on the chest is at the lower third of the sternum. The rate should be approximately 90 times per minute at a 3:1 ratio with assisted ventilations. Take care not to simultaneously provide a breath while compressing the chest. Compress the chest to one third of the anteroposterior diameter of the chest. Compressions must be adequate to generate a pulse. Reassess the heart rate every 30 seconds during this time and continue compressions until there is a spontaneous heart rate over 60 beats per minute. Avoid interruptions of chest compressions.

Kattwinkel J, Perlman JM, Aziz K, et al: Part 15: Neonatal resuscitation: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation 2010;122(18 Suppl 3):S909-S919.

16. How do I put all of this together?

Refer to the newborn resuscitation algorithm (Fig. 2-1).

Wyllie J, Perlman JM, Kattwinkel J, et al: Part 11: Neonatal resuscitation. 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Resuscitation 2010;81(Suppl 1):e260-e287.

17. How does the resuscitation of the newly born infant differ if meconium is present in the amniotic fluid?

Current recommendations no longer advise routine intrapartum oropharyngeal and nasopharyngeal suctioning for meconium-stained infants. Routine endotracheal intubation and direct tracheal suctioning of meconium-stained infants was also shown to be of no value in a randomized control trial. Perform endotracheal suction for nonvigorous or "depressed" infants (decreased tone, absent or depressed respirations, or a heart rate less than 100 beats per minute) with meconium-stained amniotic fluid. This is accomplished by suctioning while withdrawing the endotracheal tube from the airway. Repeat intubation with suctioning until no more meconium is suctioned. If the heart rate falls below 60 beats per minute, keep the endotracheal tube in place and initiate positive-pressure ventilation. Meconium-stained newborns who develop respiratory depression should receive tracheal suctioning prior to positive-pressure ventilation.

Vain NE, Szyld EG, Prudent LM, et al: Oropharyngeal and nasopharyngeal suctioning of meconium-stained neonates before delivery of their shoulders: Multicentre, randomised controlled trial. Lancet 2004;364:597-602.

Wiswell TE, Gannon CM, Jacob J, et al: Delivery room management of the apparently vigorous meconium stained neonate: Results of the multicenter, international collaborative trial. Pediatrics 2000;105:1-7.

18. What are the most common drugs used in neonatal resuscitation, and when are they indicated?

Drugs are rarely used in neonatal resuscitation, as most problems are improved by addressing airway, breathing, and circulation. Bradycardia in the newborn is usually due to inadequate lung inflation and hypoxemia, so adequate ventilation is most important.

Epinephrine is recommended when the heart rate remains below 60 beats per minute despite adequate ventilation with 100% oxygen and chest compressions for 30 seconds. Evidence from neonatal models shows increased diastolic and mean arterial pressures in response to epinephrine. The current recommended dose for epinephrine during neonatal resuscitation is 0.01 to 0.03 mg/kg of 1:10,000 concentration (0.1-0.3 mL/kg). High-dose epinephrine is not recommended for neonates because of the rare incidence of ventricular fibrillation and the theoretical risk of a hypertensive response, which could result in intraventricular hemorrhage. Use the intravenous route.

Atropine is a parasympathetic drug that decreases vagal tone and is not recommended in neonatal resuscitation. Bradycardia in the neonate is usually caused by hypoxia, and therefore atropine is unlikely to be beneficial.

Naloxone is a narcotic antagonist and is not indicated in the initial resuscitation of the newly born.

Volume expanders such as crystalloids (normal saline or Ringer's lactate) and colloids (blood) are indicated for signs of hypovolemia. Signs of hypovolemia in the neonate include pallor, weak pulses, and poor response to resuscitative efforts. The dose for volume expanders is 10 mL/kg, with reassessment after each dose. Isotonic crystalloids are the first choice among volume expanders. Red blood cells (O negative) are indicated in situations of large blood loss.

Aronson PL, Alessandrini EA: Neonatal resuscitation. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010.

Barber CA, Wyckoff MH: Use and efficacy of endotracheal versus intravenous epinephrine during neonatal cardiopulmonary resuscitation in the delivery room. Pediatrics 2006;118:1028-1034.

19. Where is the best site to obtain intravenous access?

The easiest and most direct access is the umbilical cord. Any medication, as well as volume expanders, can be given through the umbilical vein. Note that it is not recommended to administer resuscitative drugs via the umbilical artery. Peripheral veins in the extremities and

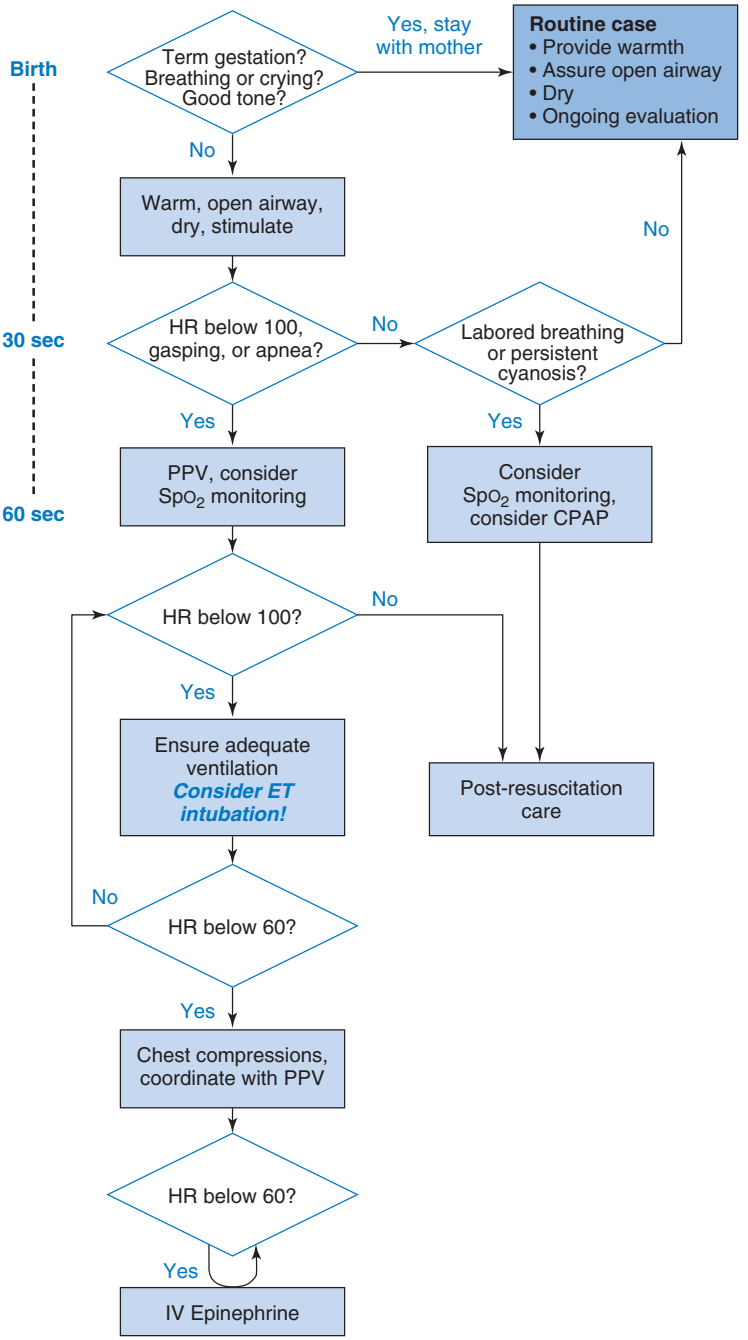

Figure 2-1. Newborn resuscitation algorithm. CPAP, continuous positive airway pressure; ET, endotracheal tube; HR, heart rate; IV, intravenous; PPV, positive pressure ventilation airway pressure. (*From Wyllie J, Perlman JM, Kattwinkel J, et al: Part 11: Neonatal resuscitation. 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Resuscitation 2010;81 (Suppl 1):e260-e287. © 2010 European Resuscitation Council, American Heart Association, Inc., and International Liaison Committee on Resuscitation.*)

the scalp can also be used but generally require more skill. Intraosseous lines can be used when no other access can be obtained. Drugs can also be given via the endotracheal tube. The preferred route of administration of epinephrine is intravenous. Sodium bicarbonate cannot be administered via the endotracheal tube.

20. Are there circumstances when resuscitation of the newly born infant may not be the appropriate action?

Because all ED deliveries are considered "unexpected" and there is no previous relationship with the delivering mother, conversations about withholding resuscitation are difficult at best. Antenatal information can be incomplete or inaccurate. In the ED, it may not be possible to gather this information quickly with precision and reliability. Guidelines should be developed after discussion with local resources, review of the most recent literature, and discussion with parents. Review the guidelines regularly and modify them on the basis of changes in resuscitation and neonatal intensive care practices. If gestational age, birth weight, or congenital anomalies are associated with almost certain death or high morbidity, resuscitation is not indicated. Examples include extreme prematurity (<23 weeks' gestation or birth weight <400 g), anencephaly, and major chromosomal abnormalities incompatible with life.

Kattwinkel J, Perlman JM, Aziz K, et al: Part 15: Neonatal resuscitation: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation 2010;122(18 Suppl 3):S909-S919.

21. When is it appropriate to discontinue the resuscitation of the newly born?

Stopping resuscitation of the newly born is obviously a very difficult decision. If no heart rate is detectable after 10 minutes of resuscitation, consider stopping the resuscitation. Cause of the arrest, gestation of the infant, presence of complications, and parents' expressed acceptability of morbidity risk will also play a part in the decision.

Kattwinkel J, Perlman JM, Aziz K, et al: Part 15: Neonatal resuscitation: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation 2010;122(18 Suppl 3):S909-S919.

22. What other major guideline changes and recommendations have been made by the 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care?

- Prevent hypothermia. Some studies have suggested that the asphyxiated infant, born at 36 weeks gestation or greater, with moderate to severe hypoxic-ischemic encephalopathy who underwent induced hypothermia had lower mortality risks and less neurodevelopmental disability than similar infants who were not cooled. However, induced hypothermia is recommended only for those infants who were at 36 or more weeks' gestation that meet the strict criteria of hypoxic-ischemic encephalopathy, and studied protocols should be followed.
- Intrapartum routine suctioning of the newborn's nose and mouth is not recommended.
- Color is no longer used as an indicator of oxygenation or effectiveness of resuscitation.
- 100% oxygen has been traditionally used during positive-pressure ventilation. Resuscitation of infants (term or preterm) should begin with air or blended oxygen with the goal of preductal Spo_2 norms.
- Pulse oximeters are a reliable indication of oxygenation as long as there is sufficient cardiac output and skin blood flow.
- A laryngeal mask may be used as an alternative airway method. The laryngeal mask may be used by trained providers when bag-mask ventilation is ineffective or attempts at endotracheal intubation have been unsuccessful.
- CO_2 detectors are effective methods of confirmation of endotracheal intubation in the newly born (term to very low birth weight infants).
- The two-thumb method of chest compressions is the preferred method, with the depth of compression being one third of the anteroposterior diameter of the chest rather than a fixed depth. Compression should be deep enough to generate a pulse.
- Administer epinephrine if the heart rate remains at or under 60 beats per minute after 30 seconds of adequate ventilation and chest compressions.

- Albumin-containing solutions are no longer the fluid of choice for initial volume expansion. Isotonic crystalloids are the first choice.
- Intraosseous access can be used if the umbilical vein is not readily available.
- Both simulations and debriefings of neonatal resuscitations can improve knowledge and skills and should be used for acquisition and maintenance of skills.

CHAPTER 3

RESPIRATORY FAILURE

Mark D. Joffe

1. How is respiratory failure defined? When does respiratory *distress* become respiratory *failure*?

A general definition of respiratory failure is inadequate oxygenation to meet metabolic needs or inadequate excretion of CO_2. Many specific definitions have been proposed, but the best clinicians individualize their decisions about therapy according to the particulars of the case. One definition of respiratory failure is P_{O_2} less than 60 mm Hg or O_2 saturation less than 93% or more than 60% oxygen, P_{CO_2} more than 60 mm Hg and rising, or clinical apnea.

2. Why is it so important to know about respiratory failure in children?

Children are at greater risk of respiratory failure than adults, so identifying those at risk, and intervening before respiratory failure occurs, is a critically important skill for pediatric clinicians. Respiratory symptoms are among the most common reasons children are taken to emergency departments (EDs), and respiratory diseases are the most frequent cause of cardiopulmonary arrest. The potential for progression of respiratory distress to respiratory failure necessitates prompt and careful evaluation of children with respiratory symptoms. Much of the morbidity and mortality risk from respiratory disease in children can be prevented by competent pediatric emergency care.

3. When should one anticipate respiratory failure?

Consider respiratory failure when there is:

- An increased respiratory rate, particularly with signs of distress (e.g., increased respiratory effort including nasal flaring, retractions, seesaw breathing, or grunting)
- An inadequate respiratory rate, effort, or chest excursion (e.g., diminished breath sounds or gasping), especially if mental status is depressed
- Cyanosis with abnormal breathing despite supplementary oxygen

American Heart Association: 2010 Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Part 14: Pediatric Advanced Life Support. Circulation 2010;122(18):877.

4. Are there different types of respiratory failure?

Some clinicians divide respiratory failure into two categories. The hypoxemic type is generally caused by mismatch of ventilation and perfusion in the lung. Hypoxemic respiratory failure from mismatch of ventilation to perfusion is often associated with normal or low P_{CO_2}. Other patients with respiratory failure have an overall decrease in alveolar ventilation that is usually the result of upper airway obstruction, neuromuscular disease, thoracic trauma, or muscle fatigue. These patients have increases in P_{CO_2} and relatively proportional decreases in P_{O_2}. The physiology in most children with respiratory failure is a combination of these two types, because one type often leads to the other. For instance, an infant with bronchiolitis initially may have hypoxemia from atelectasis and ventilation-perfusion mismatch, but may progress to inadequate alveolar ventilation when airway resistance is high and respiratory muscle fatigue supervenes.

5. Can respiratory failure be present without respiratory distress?

Absolutely. Children may hypoventilate because of reduced level of consciousness (ingestion, metabolic derangements, and head trauma) or neuromuscular dysfunction. After prolonged respiratory distress, children may become fatigued, and their work of breathing may appear normal in the presence of significant hypoventilation. Elevation of the P_{CO_2} from hypoventilation may signal worsening fatigue and impending respiratory arrest (Fig. 3-1).

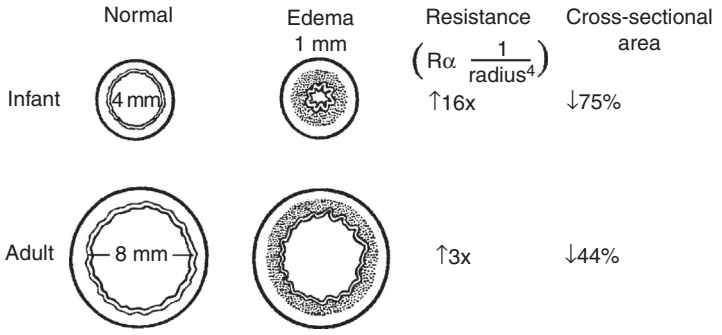

Figure 3-1. Comparison of the effects of 1 mm of mucosal edema on airway resistance in an infant versus an adult. Resistance is inversely proportional to the fourth power of the radius of the airway for laminar flow and the fifth power for turbulent flow. Airway resistance increases 16-fold in the infant for laminar flow. In a crying infant with turbulent airflow, resistance increases 32-fold. (*From Cote CJ, Todres ID: The pediatric airway. In Cote CJ, Ryan JF, Todres ID, Groudsouzian NG [eds]: A Practice of Anesthesia for Infants and Children, 2nd ed. Philadelphia, WB Saunders, 1993.*)

6. Why are children at greater risk for respiratory failure?

Infants and children require more oxygen per kilogram of body weight than adults. *Anatomic factors* put infants at particularly high risk for respiratory failure. Infants breathe almost exclusively through their noses, so nasal obstruction can cause significant respiratory signs and symptoms. The caliber of infant airways is small, so respiratory resistance is much higher, especially when there is inflammation of the respiratory tree. Alveoli have less collateral ventilation in infants. Thus, obstruction of small, peripheral airways is more likely to lead to atelectasis and hypoxemia. A compliant chest wall facilitates passage through the birth canal but leads to respiratory problems when airway resistance is increased. The diaphragm of infants is weaker and more easily fatigued compared with the diaphragm of older children and adults. Also, the inability of younger children to verbalize their symptoms may cause delayed presentations of significant respiratory problems.

7. How do I know which of the numerous children with respiratory symptoms will progress to respiratory failure?

Identifying patients with projected respiratory failure or impending respiratory failure is one of the most important skills for a pediatric clinician. A detailed history can give information about the vulnerability of the child to respiratory decompensation. Children who are very young, were born prematurely, have chronic pulmonary or cardiac diseases, or have immunodeficiencies are at particular risk. Recent medical advances, including the development of home nursing capabilities, have resulted in many "graduates" of intensive care nurseries living in our communities. EDs are confronted with these medically fragile children more than ever before.

Predicting the future is difficult, but most diseases have a typical natural history that should be considered. If a child is evaluated early in the course of respiratory infection, that child is very likely to worsen before improvement will be noted. Sometimes young children who look well require admission because they are vulnerable and are very early in the course of their respiratory illness. Children with significant respiratory effort who appear happy and playful and are maintaining their oxygen saturation and ventilation may worsen suddenly as they become fatigued, or as their disease process progresses.

Young children are more difficult to assess for respiratory problems. Histories are obtained secondhand, as the parent interprets behaviors and relays observations that have been made. A careful clinical assessment of risk factors, illness time course, and the current degree of respiratory distress is necessary to identify those patients most likely to develop respiratory failure.

8. How do I assess respiration in a baby who screams every time I approach him?

"Stranger anxiety" normally develops in the second half of the first year of life. The child's alertness to your presence is certainly a positive sign. Observing the child from across the room

provides valuable information. General appearance, state of hydration, respiratory rate, nasal flaring, retractions, paradoxical respirations, and grunting can be appreciated without close proximity to the child. In many cases, the child is more cooperative if your approach is delayed, slow, and accompanied by soothing speech.

9. Why does respiratory resistance increase so significantly with inflammation of the respiratory tree?

The resistance to laminar airflow through a tube is inversely related to the radius of that tube taken to the fourth power (Poiseuille's law). Modest decreases in the radius of the lumen of a small airway can lead to dramatic increases in respiratory resistance (see Fig. 3-1).

10. What are retractions and why do they occur?

Normally, inspiration is almost effortless. When airway resistance is high, a child must generate greater negative intrathoracic pressure to draw air into the lungs. That requires greater work of breathing, which can be seen as greater muscular activity of the neck, chest, and abdominal musculature. Flaring of the nostrils may also be noted when respiratory distress is severe. When intrathoracic pressure is very negative, parts of the chest retract inward. These retractions may be seen just below the costal margin (subcostal), just above the sternum (suprasternal), or between the ribs (intercostal). Retractions are a very important clinical finding even in the absence of wheezing or rales, because a child with impending or existing respiratory failure may have retractions without enough airflow to generate audible abnormal breath sounds.

11. What are paradoxical respirations? Why do they occur?

Infants who have increased airway resistance generate high negative intrathoracic pressures to inflate their lungs. As the diaphragm moves downward and the intrathoracic pressure becomes negative, the soft, cartilaginous bones and weak intercostal musculature cannot maintain the thoracic circumference. As the abdomen moves outward, the infant's compliant chest may collapse inward (rather than the normal expansion) on inspiration, hence the terms *paradoxical respirations*, *thoracoabdominal asynchrony*, or *seesaw breathing*. Paradoxical respirations are often a sign of impending respiratory failure.

12. How can one tell if the respiratory failure is caused by an upper airway disease or by a lower airway disease?

Distinguishing upper from lower respiratory disease is a difficult but important part of clinical evaluation. Many children have disease processes that involve the upper and lower respiratory tracts simultaneously, for example, bronchiolitis caused by respiratory syncytial virus. In general, respiratory sounds reflecting upper airway inflammation are most prominent during inspiration, when negative intraluminal pressure causes upper airway narrowing and turbulent airflow in the trachea. Conversely, intrathoracic (lower airway) processes produce wheezing and other sounds of airway obstruction primarily during expiration, when positive intrapleural pressure compresses intrathoracic airways. If breath sounds are more obstructed on inspiration, the upper airway is probably involved. Wheezing or other sounds more prominent during expiration suggests lower airway disease.

13. Is wheezing a reliable sign of severe lower airway disease?

No. Audible wheezing requires significant airflow through narrowed small airways. Children with severe obstruction of small airways may have so little airflow that audible wheezing is not present. These children usually have decreased inspiratory breath sounds and increased work of breathing. Administration of bronchodilators to these patients will often increase wheezing because there is more turbulent airflow as the narrowed airways open up.

14. After I examine a child, does pulse oximetry tell me anything I don't already know?

Yes. Hypoxemia is not always obvious from the physical examination. Most children who are hypoxemic from a respiratory illness have signs of respiratory distress, but in many cases mild to moderate hypoxemia is clinically inapparent. Hypoxemia is a less potent stimulator of the respiratory center than is hypercarbia. Thus, the increase in minute ventilation that occurs with mild hypoxemia is very modest and may be difficult to detect by physical examination. In patients with acute exacerbations of asthma there is very poor correlation between asthma score, a measure of respiratory distress, and oxygen saturation.

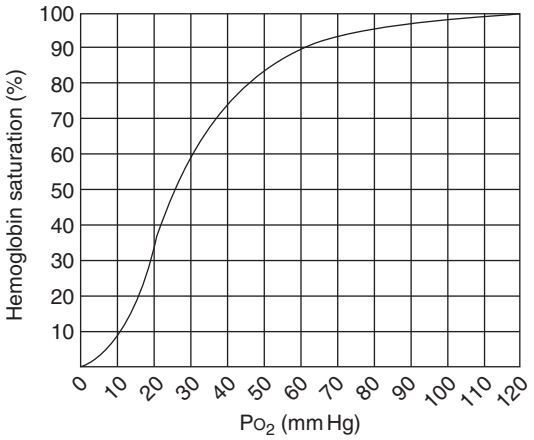

Figure 3-2. As Po_2 drops below 70 mm Hg, oxygen saturation declines more precipitously.

Also, cyanosis requires 3 to 5 g of unsaturated hemoglobin per deciliter to be visible. If a child has a total hemoglobin of 12 g/dL, cyanosis is not apparent until the oxygen saturation drops below 75%. Most clinicians believe it is useful to be aware of hypoxemia before it is severe enough to cause visible cyanosis (Fig. 3-2).

15. What causes pulse oximeter readings to be inaccurate?

The most common problem is the oximeter probe not consistently registering the pulsatile arterial flow through the skin. This is often caused by movement, poor perfusion of the skin, or bright ambient light. Careful attention to the graphical display of pulsatile flow on the pulse oximeter can almost always distinguish a falsely low saturation due to poor signal capture from true hypoxemia. Occasionally, hemoglobin is abnormal. Carboxyhemoglobinemia will cause a falsely elevated measurement of oxygen saturation by pulse oximetry; methemoglobinemia in significant concentrations will cause a modest lowering.

Lee WW, Mayberry K, Crapo R, et al: The accuracy of pulse oximetry in the emergency department. Am J Emerg Med 2000;18:427.

16. What is the lowest acceptable oxygen saturation for a child to be discharged?

There is often no simple answer to what appears to be a simple question. In healthy awake children a normal oxygen saturation is 97% to 99%. A saturation below 95% is greater than two standard deviations below the mean (−2 SD). For healthy young infants, −2 SD below the mean is 93%. At altitudes higher than 3000 m (10,000 ft), −2 SD is below 85%. For infants, most clinicians use a threshold of 92% to 94% as a criterion for discharge, though there is very little high-quality evidence for this. If an inpatient with bronchiolitis is in the resolution phase of illness, many institutions stop continuous pulse oximetry because the brief dips that are often noted prolong hospitalization without evidence of improved outcomes.

Unger S, Cunningham S: Effect of oxygen supplementation on length of stay in infants with acute viral bronchiolitis. Pediatrics 2008;121(3):470.

17. Are oxygen desaturations that only last for a few seconds significant?

Probably not. Pulse oximeters display an oxygen saturation that is an average over a period of time. The technology was first developed for intraoperative monitoring, when beat-to-beat saturation readings are desirable, not for use in EDs and offices to predict respiratory deterioration. Most instruments are programmed with short averaging times, typically 3 to 5 seconds. A longer averaging time will not detect brief episodes of hypoxemia but will display a number that better represents the general trend in oxygen saturation. A brief period of desaturation identified by a pulse oximeter with a short averaging time may signify a very transient physiologic event that does not reflect the general status of pulmonary function. Although controversial, most authorities do not believe brief episodes of mild desaturation lead to significant morbidity.

18. What about episodes of desaturation that occur only while the child is feeding or sleeping?

Oxygen saturation during feeding or while sleeping often dips below the baseline. Feeding and sleeping may be stress tests for desaturation. Normal children monitored during sleep have occasional oxygen saturation nadirs below 93%, and even dipping below 90% is not uncommon. In infants with respiratory illnesses who are asleep, there is no consensus, nor are there data, on whether an oxygen saturation of 90% to 92% for a brief period portends future deterioration or respiratory failure.

Urschitz MS, Wolf J, Von Einem V, et al: Reference values for nocturnal home pulse oximetry during sleep in primary school children. Chest 2003;123(1):96-102.

19. What's the big deal if the oxygen saturation is 2% to 3% below normal?

The relationship between P_{O_2} and saturation is not linear but sigmoidal. Although there is little difference in P_{O_2} between saturations of 99% and 96%, there is a much greater difference in P_{O_2} between 93% and 90%. Oxygen saturation and respiratory rate are objective measures of pulmonary function available to clinicians in most settings. Saturations below 93% in children with respiratory diseases may identify patients with pulmonary/airway inflammation that puts them at risk for developing respiratory failure. Transient periods of desaturation are common, especially in sleeping children with respiratory illnesses, and usually require no intervention. Consider oxygen therapy and closer monitoring if the desaturation is persistent (see Fig. 3-2).

20. Why do some patients with wheezing have a reduction in their oxygen saturation after bronchodilator treatment when they otherwise appear to be improving?

Albuterol and other bronchodilators are β-adrenergic agents that are somewhat β_2 selective. β_2-Receptor stimulation causes *vaso*dilation as well as *broncho*dilation. One explanation for decreases in oxygen saturation after treatment with bronchodilators is that pulmonary vasodilation results in increased perfusion of poorly ventilated areas of the lung and worsening of ventilation-perfusion matching. This is seen in a minority of patients; is usually a transient phenomenon; and is not a contraindication to continued, aggressive bronchodilator therapy in children with severe lower airway disease.

21. What is the value of a chest radiograph in evaluation of a child with suspected respiratory failure?

In previously healthy children, respiratory failure is generally preceded by clinically identifiable respiratory distress. Young children with fever and tachypnea may benefit from a chest radiograph, because bacterial pneumonia can be difficult to diagnose by history and physical examination alone. Foreign-body aspiration, pneumothorax/pneumomediastinum, and cardiac disease are a few of the diagnoses that can also be suspected on the basis of a chest radiograph. Children with minor respiratory illnesses generally do not need chest radiographs, even if they seek care in an ED. If there is concern about impending or existing respiratory failure, a chest radiograph can be very useful.

22. What is ARDS in children? Doesn't the "A" stand for *adult*?

Acute respiratory distress syndrome (ARDS) occurs in children, so the "A" was changed from *adult* to *acute*. It is a common cause of respiratory failure at all ages. ARDS is a diffuse pulmonary process that develops after lung injury. Some causes of ARDS that are treated in EDs include sepsis, hypotension, pneumonia, aspiration of gastric contents, smoke or other inhalation injury, near drowning, and chest trauma with pulmonary contusion. Chest radiographs may show diffuse, bilateral infiltrates that resemble left-sided congestive heart failure, but left atrial pressures must be normal for the diagnosis of ARDS. The diagnostic criteria for ARDS are as follows:

- Acute onset
- Severe hypoxemia ($P_{O_2} < 200$ mm Hg regardless of fraction of inspired oxygen and positive end-expiratory pressure)
- Diffuse bilateral infiltrates on chest radiography
- Normal left atrial pressure

Bernard GR: Acute respiratory distress syndrome: A historical perspective. Am J Respir Crit Care Med 2005;172:798.

23. Why are some patients who meet the definition for respiratory failure not intubated and mechanically ventilated to normalize their blood gases?

Children tolerate hypercarbia better than adults do. If oxygenation is adequate and hypercarbia is likely to be reversed in the near future, some intensivists permit the hypercarbia to persist for a period of time. So-called *permissive hypercarbia* reduces barotrauma to the lungs that results from positive-pressure and mechanical ventilation. In patients with reactive airway disease or asthma, positive-pressure ventilation is fraught with risk of pneumomediastinum and pneumothorax. Because there are effective medications to reverse airway obstruction in a relatively short period of time, some patients can be closely monitored without endotracheal intubation and mechanical ventilation, despite levels of CO_2 that define respiratory failure.

24. Is endotracheal intubation the only way to manage the airway when a child is in respiratory distress?

No. In fact, bag-valve-mask ventilation is adequate for many children with transient, reversible airway problems. Positioning the child with some extension of the neck and moving the mandible forward by lifting the angles of the jaw pulls the tongue off the posterior pharynx, often relieving airway obstruction. Oral airways (for unconscious patients) and nasal airways can be used to maintain the patency of the upper airway during bag-valve-mask ventilation. Provide to all children effective bag-valve-mask ventilation with 100% oxygen prior to intubation.

American Heart Association: 2010 Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Part 14: Pediatric Advanced Life Support. Circulation 2010;122(18):878.

Key Points: Indications for Endotracheal Intubation

1. Progressive respiratory exhaustion—unlikely to reverse quickly
2. Apnea, hypoventilation that requires mechanical ventilation
3. Need for airway protection (upper airway obstruction, loss of protective airway reflexes)
4. Shock
5. Airway access for pulmonary toilet

25. What are the indications to intubate the trachea of a child?

- Respiratory failure that is unlikely to be reversed quickly, especially if hypoxemia is present despite greater than 60% oxygen administration
- Apnea, hypoventilation, or progressive respiratory exhaustion that requires ongoing mechanical ventilation
- Need for airway protection for children who have upper airway obstruction or an inability to protect their airway from aspiration
- Desire to decrease the work of breathing for patients in shock (under normal circumstances, work of breathing requires less than 5% of the total energy expenditure, but with respiratory distress it can demand up to 50%; in a shock state, energy can be better utilized for other essential body functions)
- Therapeutic interventions, such as tracheal administration of medications and suctioning for pulmonary toilet (mechanical ventilation is also required)

Key Points: Steps to Perform Endotracheal Intubation

1. Preoxygenate with 100% oxygen by bag-valve-mask device.
2. Prepare equipment (e.g., suction, endotracheal tubes, laryngoscopes, monitors—electrocardiogram [ECG], pulse oximeter, end-tidal CO_2 detector or monitor).
3. Confirm functioning intravenous (IV) line.
4. Administer medications (atropine for younger children and those receiving succinylcholine, sedative, paralytic agent).
5. Intubate the trachea, observing the tube pass through the vocal cords.
6. Verify proper placement—auscultate the chest, check for CO_2 by capnometry, chest radiograph.
7. Secure the endotracheal tube.
8. Evacuate the stomach with a nasogastric or orogastric tube.

26. What are the steps for emergency endotracheal intubation of a child?

Begin bag-valve mask ventilation with 100% oxygen as soon as the need for positive-pressure ventilation is identified. Emergency endotracheal intubations are generally treated as "full-stomach" intubations. The steps for a rapid sequence intubation are as follows:

1. Preoxygenate with bag-valve-mask ventilation with 100% oxygen.
2. Prepare all equipment, including suction, endotracheal tubes, and laryngoscopes.
3. Make certain an IV catheter is functioning well.
4. Administer atropine (for younger children and those receiving succinylcholine), followed by a sedative agent and then a paralytic agent.
5. Perform laryngoscopy once paralysis is complete, and watch the endotracheal tube go through the vocal cords into the trachea.
6. Auscultate for equal breath sounds and check for the presence of CO_2 by capnometry. Observe symmetric chest expansion, misting in the tube with exhalation, and improvement in oxygen saturation by pulse oximetry, and listen for gurgling over the stomach, which suggests esophageal intubation.
7. Secure the tube with tape.
8. Evacuate the stomach with a nasogastric or orogastric tube.
9. Obtain a chest radiograph to check the position of the tube and adjust accordingly.

American Heart Association: 2010 Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Part 14: Pediatric Advanced Life Support. Circulation 2010;122(18):876-908.

King C, Rappaport LD. Emergent endotracheal intubation. In Henretig FM, King C (eds). Textbook of Pediatric Emergency Procedures 2nd Edition. Philadelphia, Wolters Kluwer/Lippincott, Williams & Wilkins 2008;146-190.

27. Why is cricoid pressure (Sellick maneuver) no longer recommended?

Cricoid pressure was recommended for rapid sequence intubation for many years as a means of preventing gaseous distention of the stomach from bag-valve-mask ventilation and passive regurgitation with aspiration during the intubation procedure. However, cricoid pressure can compress the trachea, making passage of the endotracheal tube more difficult. It often displaces the esophagus laterally, and there is little evidence that it decreases the risk of aspiration. The American Heart Association in their 2010 guidelines no longer recommends cricoid pressure for emergent intubations.

American Heart Association: 2010 Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Part 14: Pediatric Advanced Life Support. Circulation 2010;122(18):876-908.

Butler J, Sen A: Best evidence topic report. Cricoid pressure in emergency rapid sequence induction. Emerg Med J 2005;22(11):815-816.

28. Can a difficult endotracheal intubation be predicted?

Not always, so it is important to have people experienced with airway management available, especially when a difficult intubation is anticipated. The conditions found in Table 3-1 often result in difficult intubations.

Table 3-1. Conditions Found in Difficult Intubations

CONGENITAL	ACQUIRED
Micrognathia	Hoarseness/stridor/drooling
Macroglossia	Facial burns/singed facial hairs
Cleft or high arched palate	Facial fractures/oral trauma
Protruding upper incisors	Foreign body
Small mouth	
Limited mobility of temporomandibular joint	

29. What should you consider if a patient deteriorates after endotracheal intubation?

If an intubated patient's condition deteriorates after endotracheal intubation, consider DOPE:

- **D**isplacement of the endotracheal tube—no longer in the trachea
- **O**bstruction of the tube—perhaps by mucus
- **P**neumothorax
- **E**quipment failure—perhaps the ventilator is malfunctioning, or perhaps you are not actually delivering 100% oxygen as you thought

American Heart Association: 2010 Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Part 14: Pediatric Advanced Life Support. Circulation 2010;122(18):880.

CHAPTER 4

SHOCK

Samuel J. Prater and Brent R. King

1. What blood pressure defines shock in the pediatric patient?

Shock is not defined by the blood pressure or by any other vital sign. Shock exists when the patient's metabolic demand exceeds the body's ability to deliver oxygen and nutrients. This occurs most commonly when metabolic demand is normal or slightly elevated but delivery of oxygen and nutrients is dramatically reduced. Examples include excessive blood or fluid volume loss (hemorrhage or diarrhea), poor cardiac function, and sepsis. The shock state can and often does exist in the presence of a "normal" blood pressure.

Bell LM: Shock. In Fleisher GM, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 46-57.

Key Points: Definition of Shock

1. Shock is a condition in which the patient's metabolic requirements are unmet.
2. The shock state is a complex interplay between the physiologic insult and the host's response to that insult; both play a role.
3. In its earliest phase, shock might be recognized only by abnormal results of laboratory tests that measure tissue acid-base status (e.g., serum lactate). Overt clinical signs are seen as the shock state progresses.

2. How can shock be recognized?

To recognize shock, consider both the consequences of inadequate perfusion and the patient's compensatory mechanisms. The clinical manifestations of shock are those of *inadequate perfusion* and *compensation*. Inadequate perfusion of the brain results in an alteration in the child's level of consciousness. Inadequate perfusion of the kidneys results in decreased urine output.

As perfusion decreases, compensatory changes occur. These changes improve delivery of oxygen and nutrients and direct blood flow to the vital organs. The first compensatory mechanism is usually an *increased heart rate; tachycardia out of proportion to the child's clinical picture (i.e., fever, distress) should be a red flag.* Because cardiac output is equal to the rate multiplied by the stroke volume, an increased heart rate can maintain cardiac output in the face of decreased stroke volume. Additionally, peripheral vasoconstriction helps to maintain blood flow to the central organs and to the brain. The patient therefore has pale, cool extremities and a delayed capillary refilling time. This increased vascular tone also affects the measured blood pressure. The diastolic pressure is slightly elevated, so the difference between the systolic and diastolic pressures—the pulse pressure—is smaller. This is referred to as a "narrowed" pulse pressure.

To compensate for both the decreased oxygen delivery and the acidosis created by underperfusion of peripheral tissues, the respiratory rate increases. The blood pressure eventually falls, but this is a late, ominous finding and may signal that the shock state is irreversible.

3. What are the stages of shock?

Shock is a spectrum of illness, so any division into discrete segments is somewhat artificial. However, shock is usually divided into two stages: *compensated shock* and *uncompensated shock*.

4. What is compensated shock?

In early shock, various physiologic changes allow continued delivery of oxygen and nutrients to the heart, kidneys, brain, and other vital organs. Tachycardia is usually the first compensatory mechanism. The increased heart rate helps to maintain cardiac output in the face of low blood volume, excessive vasodilation, or pump failure. Increased vasomotor tone shunts blood away from the skin and the extremities to more vital organs. This is manifested as mottling and decreased capillary refill time. In compensated shock, the patient is able to continue to meet his or her metabolic demand, even if only marginally.

5. Are there exceptions to the compensatory mechanisms described earlier?

Yes. In *septic shock* the patient sometimes develops so-called warm shock or warm distributive shock. In this state the patient has flushed skin and bounding pulses associated with a hyperdynamic precordium. The capillary refill time can be normal or sometimes what is referred to as flash capillary refill. This state can be explained by the cascade of inflammatory mediators that are responsible for the condition called septic shock. Likewise, in *neurogenic shock*, loss of sympathetic tone can result in bradycardia in the face of profound hypotension.

6. What is uncompensated shock?

If the shock state progresses without interruption, the patient's compensatory mechanisms eventually fail. Hypoperfusion of organ systems causes acidosis and further release of inflammatory mediators. As blood flow to the brain decreases, the patient can become irritable or stuporous and eventually slips into coma. Likewise, decreased renal blood flow causes decreased urine output and finally results in anuria. The gastrointestinal tract is similarly affected, so the patient often has decreased bowel motility followed by distention and edema of the bowel wall. As tissue ischemia and acidosis progress, the inflammatory mediators cause diffuse vascular injury and capillary leakage. The pulmonary bed is especially sensitive to this type of injury. Damage to the pulmonary tissues exacerbates tissue hypoxemia. The ultimate result of progressive shock is multiorgan system failure and acute respiratory distress syndrome. At some point during this process the patient's blood pressure falls.

7. What are the types (or mechanisms) of shock?

There are multiple mechanisms for shock:

- **Hypovolemic shock:** Hypovolemia, such as might occur with blood loss, vomiting, and diarrhea, decreases perfusion to the tissues and leads to shock.
- **Distributive or vasodilatory shock:** This type of shock is the final common pathway of a variety of conditions that result in vasodilation. Neurogenic distributive shock is caused by a spinal cord injury that eliminates sympathetic innervation to the blood vessels, causing profound vasodilation and bradycardia. Accidental ingestion of vasodilating medications can also result in distributive shock. Anaphylaxis results in vasodilation, and although anaphylaxis has many other components, shock is a part of the clinical picture. Septic shock is largely distributive in nature but is a complex process (see later discussion).
- **Cardiogenic shock:** Pump failure is the primary mechanism for cardiogenic shock. Decreased myocardial contractility makes adequate delivery of oxygen and nutrients impossible. Because children are very dependent on a normal heart rate to produce an adequate cardiac output, drugs and other conditions that cause bradycardia can lead to shock. The patient will have evidence of congestive heart failure, such as rales on pulmonary auscultation, hepatomegaly, and peripheral edema. Viral myocarditis, hypertrophic cardiomyopathy, and certain myocardial depressant drugs can cause cardiogenic shock.
- **Septic shock:** Many consider septic shock to be another form of distributive shock. In septic shock, a stimulus causes the formation of inflammatory mediators that result in profound vasodilation and shock. However, some of these mediators also directly depress myocardial activity; thus, septic shock can have features of both distributive and cardiogenic shock.

Jones AE, Craddock PA, Tayal VS, Kline JA: Diagnostic accuracy of left ventricular function for identifying sepsis among emergency department patients with nontraumatic, symptomatic, undifferentiated hypotension. Shock 2005;24:513-517.

8. Is the pathophysiology of shock really that simple?

No, it is exceedingly complex. What we refer to as "shock" is the final common pathway for a variety of physiologic insults. Whether the process starts with acute blood loss or with an overwhelming infection, eventually the host mounts a response to the insult, and this response—at least in some cases—seems to contribute to the shock state.

9. What usually initiates septic shock?

The most common and potent initiator of the inflammatory cascade called septic shock is exposure to *endotoxin*. Endotoxin is the lipopolysaccharide (LPS) coat of gram-negative bacteria. Other bacterial and viral agents can also start this process. Examples include certain viral proteins and teichoic acid.

10. Is septic shock caused by gram-positive organisms different from septic shock caused by gram-negative organisms?

Yes, gram-negative septic shock is more severe. The expected mortality rate from gram-negative septic shock is 20% to 50%, but that from gram-positive septic shock is 10% to 20%.

11. How do proteins such as endotoxin cause septic shock?

This is a trick question. Endotoxin and the other bacterial and viral proteins do not actually cause septic shock. Instead, their presence in the body leads to a response from the host. This response involves a *cascade of inflammatory mediators*, called *cytokines*, that are responsible for most of the symptoms of shock.

12. Which of the cytokines is the most important?

Tumor necrosis factor was once considered to be the most important of the cytokines. Although its formation seems to initiate and propagate the remainder of the inflammatory cascade, recent research on the cytokine pathway reveals it to be a tightly regulated balance among proinflammatory cytokines, anti-inflammatory cytokines, and soluble inhibitors of proinflammatory cytokines.

Schulte W, Bernhagen J, Bucala R: Cytokines in sepsis: potent immunoregulators and potential therapeutic targets—An updated review. Mediators Inflamm 2013;2013:165974. Epub Jun 18, 2012.

13. Describe how the cascade of septic shock begins.

Most commonly, endotoxin (LPS) is bound by a plasma protein called LPS-binding protein (LBP). The LPS-LBP complex then binds to the CD14 receptor on the surfaces of macrophages. This process stimulates the formation of both tumor necrosis factor and interleukin. These two cytokines begin the cascade that leads to septic shock.

14. Which is more important in the management of the child with septic shock: aggressive resuscitation at the referring hospital or excellent care at the tertiary care center?

Although both are important, a recent study demonstrated that children in shock from sepsis who are aggressively resuscitated at the referring hospital have better outcomes than those who do not receive such care. Additionally, recent guidelines from the American College of Critical Care Medicine emphasize goal-directed fluid resuscitation, inotrope therapy, and antibiotic delivery within the first hour. These results support the notion that management of shock must begin as soon as it is recognized.

Brierley J, Carcillo JA, Choong K, et al: Clinical practice parameters for hemodynamic support of pediatric and neonatal septic shock: 2007 update from the American College of Critical Care Medicine. Crit Care Med 2009;37(2):666-688.

Han YY, Carcillo JA, Dragotta MA, et al: Early reversal of pediatric-neonatal septic shock by community physicians is associated with improved outcome. Pediatrics 2003;112:793-799.

15. What is the most common cause of shock in children?

Hypovolemia is the most common cause of shock in children.

16. What is the usual cause of hypovolemia?

Throughout most of the world, hypovolemia is caused by diarrheal illness. Millions of children die of hypovolemia caused by diarrhea each year.

17. What are some ways in which trauma can cause shock?

Posttraumatic hemorrhage is the most common way that trauma causes shock. Because young children have abdominal wall muscles that are poorly developed, they are at risk for liver and spleen injury after blunt abdominal trauma.

In addition to hemorrhagic shock, blunt chest trauma can result in a tension pneumothorax. Tension pneumothorax causes increased intrathoracic pressure, which in turn reduces venous return to the heart and causes shock. Similarly, blunt chest trauma can result in pericardial tamponade (essentially a form of restrictive cardiomyopathy).

Finally, cervical spine injury can result in neurogenic shock.

18. How does isolated head trauma cause shock?

In the absence of exsanguination from a large scalp laceration, it doesn't. If a patient has shock after what appears to be isolated head trauma without obvious exsanguination from a scalp wound, there must be another explanation for the shock.

Key Points: Etiology of Shock in Children

1. Hypovolemia (not enough circulating volume to deliver oxygen and nutrients)
2. Impaired cardiac function (ineffective pumping of the circulating volume)
3. Inappropriate vasodilation (the circulating volume exists primarily in the venous capacitance system and is unavailable to deliver oxygen and nutrients)

19. How is hemorrhage classified?

Hemorrhage is divided into four classes based on the amount of blood loss. Class I hemorrhage produces the least amount of blood loss, and class IV, the greatest.

20. How do the classes of hemorrhage relate to shock?

There is no direct relationship. In fact, a patient can experience class I hemorrhage without demonstrating signs of shock. However, as patients experience greater degrees of hemorrhage, they are more likely to have symptoms of shock.

21. What are the classes of hemorrhage?

- **Class I hemorrhage:** The patient has lost up to 15% of his or her blood volume. Otherwise healthy patients are likely to have minimal tachycardia and no other symptoms. Unless there is ongoing hemorrhage, the patient should require no treatment.
- **Class II hemorrhage:** The patient has lost 15% to 30% of his or her blood volume. Loss of this amount of blood stimulates the compensatory mechanisms usually associated with early, compensated shock. Tachycardia, increased respiratory rate, and narrowed pulse pressure are seen. Urine output is usually maintained, but the patient may have signs of early central nervous system impairment. Such signs may include fright or anxiety.
- **Class III hemorrhage:** The patient has lost 30% to 40% of his or her blood volume. This amount of blood loss is clearly associated with signs of compensated shock but may also be associated with uncompensated shock. Even healthy individuals may have a drop in systolic blood pressure with this degree of blood loss. Urine output is likely to be decreased, and the patient may be very anxious or confused.
- **Class IV hemorrhage:** This represents loss of more than 40% of the circulating blood volume. This degree of hemorrhage is uniformly fatal if untreated. The shock state may, in some cases, be irreversible. The patient has a markedly decreased blood pressure. He or she can be expected to have complete peripheral vasoconstriction, extreme tachycardia, and little or no urinary output. Mental status is very depressed, and the patient may be unconscious.

22. How can emergency physicians make a presumptive diagnosis of cardiogenic shock?

Recent studies have demonstrated that emergency physicians using bedside ultrasonography can correctly identify cardiac wall motion abnormalities as well as cardiac index. This technology makes it easy for the emergency physician to identify patients with abnormal cardiac function.

Dey I, Sprivulis P: Emergency physicians can reliably assess emergency department patient cardiac output using the USCOM continuous wave Doppler cardiac output monitor. Emerg Med Austr 2005;17:193-199.

Dinh VA, Ko HS, Rao R, et al: Measuring cardiac index with a focused cardiac ultrasound examination in the ED. Am J Emerg Med 2012;30(9):1845-1851.

23. How can neurogenic shock be distinguished from hemorrhagic shock?

The patient with hemorrhagic shock has a rapid and possibly irregular pulse, but the patient with neurogenic shock has a slow and regular pulse.

24. In general, what is the initial treatment for shock?

There is no single treatment for shock: Therapy is aimed at the cause. That being said, most types of shock respond well to fluid therapy, so if the cause cannot be identified, a single bolus of 20 mL/kg of either normal saline or lactated Ringer's solution may be helpful and, at worst, is unlikely to cause serious harm. Additionally, the patient should receive supplemental oxygen and may require assisted ventilation to ensure that oxygen delivery is maximized.

25. Which treatments should be avoided?

The greatest error that you can make in the treatment of shock is to use pressor agents to treat hypovolemia. Even in cases of distributive shock, fluids should be used for initial treatment. Note that excessive fluid administration can be harmful in cardiogenic shock; excessive crystalloid fluid can also be harmful in hemorrhagic shock.

26. What are the different types of common pressor agents used to manage shock?

Dopamine is an endogenous catecholamine that results in increased inotropy (contractility), increased chronotropy (heart rate), and vasodilation at low doses. At higher doses, dopamine results in vasoconstriction. Dopamine is often used first at low doses because it is tolerated well when given peripherally. *Dobutamine* is a synthetic catecholamine that results in increased inotropy and vasodilation. *Norepinephrine* is a potent central nervous system neurotransmitter that results in significant vasoconstriction and minimal effect on inotropy or chronotropy. *Epinephrine* is a natural hormone that results in increased inotropy and chronotropy. At lower doses epinephrine results in vasodilation, whereas higher infusion rates (>0.3 μg/kg/minute) result in systemic and pulmonary vasoconstriction (Table 4-1).

Matthew H, Trakas EV, Su E, et al: Advances in monitoring and management of shock. Pediatr Clin North Am 2013;60(3):641-654.

27. How should hypovolemic shock be treated?

Treat hypovolemia with volume. Initial therapy is usually with crystalloids. Acceptable crystalloids are lactated Ringer's solution and normal saline. Give boluses of 20 mL of fluid per kilogram of body weight, each given over 5 to 10 minutes. Initial volume resuscitation can require 40 to 60 mL/kg or more before considering other therapy.

28. How should cardiogenic shock be treated?

Cardiogenic shock should be treated with afterload reduction coupled with inotropic support. This can be achieved with low-dose epinephrine (0.05-0.3 μg/kg/minute) and the inodilators, milrinone or amrinone.

Mtaweh H, Trakas EV, Su E, et al: Advances in monitoring and management of shock. Pediatr Clin North Am 2013;60(3):641-654.

29. How should hemorrhagic shock be treated?

Compelling evidence exists in adult populations about massive transfusion protocols and balanced ratios of blood products in lieu of crystalloid resuscitation. Although little evidence exists in the pediatric literature, many trauma centers have adapted adult protocols to their pediatric patients. The tenets of these protocols involve minimizing crystalloid fluid and judicious transfusion of blood products in ratios that more closely mimic fresh whole blood. Undoubtedly some patients with mild blood loss can be managed with crystalloid, but patients with severe (class III and IV) hemorrhage will require blood replacement therapy.

Table 4-1. Common Pressor Agents Used to Manage Shock

	INOTROPE	CHRONOTROPE	VASODILATION	VASOCONSTRICTION
Dopamine	X	X	Low dose	Higher doses
Dobutamine	X		X	
Norepinephrine				X
Epinephrine	X	X	Low dose	Higher doses

Dehmer JJ, Adamson WT: Massive transfusion and blood product use in the pediatric trauma patient. Semin Pediatr Surg 2010;19(4):286-291.
Spinella PC, Holcomb JB: Resuscitation and transfusion principles for traumatic hemorrhagic shock. Blood Rev 2009;23(6):231-240.

30. What is the treatment for neurogenic shock?

In neurogenic shock, injury to the spinal cord results in decreased sympathetic input to the vascular system. Most of the patient's blood supply is left in the venous or capacitance system. *Fluid therapy* is an appropriate initial treatment, but pressor agents may also be needed. *Norepinephrine and phenylephrine* are powerful α-agonists and are often recommended for neurogenic shock in the setting of normal heart rate and cardiac output. Due to its inotropic properties *dopamine* is an excellent alternative to norepinephrine and phenylephrine when cardiac output and heart rate are inadequate. If the patient has profound bradycardia, *atropine* may be used to increase the heart rate and, therefore, the cardiac output.

Consortium for Spinal Cord Medicine: Early acute management in adults with spinal cord injury: A clinical practice guideline for health-care providers. Washington, DC, Paralyzed Veterans of America, 2008. Available from http://www.pva.org/site/DocServer/57462_PVA.pdf?docID=5181. Accessed August 6, 2013.
Stevens RD, Bhardwaj A, Kirsch JR, Mirski MA: Critical care and perioperative management in traumatic spinal cord injury. J Neurosurg Anesthesiol 2003;15(3):21-29.

31. How should the patient with septic shock be treated?

Treatment of septic shock is difficult and complex. Early recognition and aggressive resuscitation are critical to improving outcomes. Fluid therapy is usually employed first, but pressor agents are often required. Treatment with antibodies directed against the inflammatory mediators has not proved to be effective, but as our understanding of this complex process evolves, effective immunomodulator therapy may be developed. Interestingly, although antibiotics are needed to limit the infectious process, they alone are not sufficient treatment for septic shock. In certain cases, they may actually increase the antigen load in the system by destroying gram-negative organisms, which results in more LPS in the circulatory system. However, patients who do not receive adequate antibiotic therapy have a higher mortality rate than those receiving such therapy. Additionally, antibiotics alone may be insufficient for source control, and sometimes surgical control is needed to eradicate the source.

Brierley J, Carcillo JA, Choong K, et al: Clinical practice parameters for hemodynamic support of pediatric and neonatal septic shock: 2007 update from the American College of Critical Care Medicine. Crit Care Med 2009;37(2):666-688.

32. How should I choose an appropriate antibiotic for the patient in septic shock?

Septic shock is a severe and life-threatening condition and should be treated promptly. It may, however, be impossible to identify the causative organism in a timely fashion. Therefore, empirically administer antibiotic therapy. In most cases, it is prudent to choose broad-spectrum agents that are effective against a wide range of likely pathogens.

Dellinger RP, Levy MM, Rhodes A, et al, Surviving Sepsis Campaign Guidelines Committee including the Pediatric Subgroup: Surviving sepsis campaign: International guidelines for management of severe sepsis and septic shock: 2012. Crit Care Med 2013;
41(2):580-637.

33. How should I select initial pressor agents for patients in septic shock?

For patients with evidence of shock refractory to adequate fluid resuscitation, dopamine is started initially if the patient is normotensive. For patients who are hypotensive, norepinephrine is the agent of choice when systemic vascular resistance (SVR) is low (warm shock), and epinephrine (<0.3 μg/kg/minute) and dopamine (<10 μg/kg/minute) are the agents of choice when SVR is elevated (cold shock).

Brierley J, Carcillo JA, Choong K, et al: Clinical practice parameters for hemodynamic support of pediatric and neonatal septic shock: 2007 update from the American College of Critical Care Medicine. Crit Care Med 2009;37(2):666-688.

34. Under what circumstances might the drug phenylephrine be useful in the management of shock?

Phenylephrine causes vasoconstriction without causing excessive tachycardia. In the patient whose shock state is caused primarily by vasodilation, phenylephrine might be indicated.

35. What is early, goal-directed therapy?

Early, goal-directed therapy is a management scheme that has been most strongly promoted for children with septic shock. This therapy is an aggressive approach to improve physiologic indicators of perfusion and vital organ function in the first 6 hours. Physiologic indicators targeted during early, goal-directed therapy include the following:

1. Blood pressure (systolic pressure minimally 60 mm Hg for those <1 month of age, 70 mm Hg + [2 × age in years] in children 1 month to 10 years of age, 90 mm Hg in children 10 years of age or older)
2. Strong, distal pulses equal to central pulses
3. Skin perfusion (warm, with capillary refill <2 seconds)
4. Normal mental status
5. Urine output (≥1 mL/kg/hour, once effective circulating volume is restored)

Brierley J, Carcillo JA, Choong K, et al: Clinical practice parameters for hemodynamic support of pediatric and neonatal septic shock: 2007 update from the American College of Critical Care Medicine. Crit Care Med 2009;37(2):666-688.

Rivers E, Nguyen B, Havstad S, et al: Early goal-directed therapy in the treatment of severe sepsis and septic shock. N Engl J Med 2001;345:1368-1377.

36. How should anaphylactic shock be treated?

Most manifestations of anaphylaxis, including hypotension, respond well to intramuscular epinephrine. Epinephrine has both α- and β-receptor agonist effects, so it can effectively treat bronchospasm and hypotension.

American Heart Association: 2010 Guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. Part 12.2: Cardiac arrest associated with anaphylaxis. circulation 2010;122: S829-S861.

37. What is toxic shock syndrome (TSS)?

TSS is an illness characterized by fever, erythroderma, hypotension, and involvement of several other organ systems. It is caused by strains of *Staphylococcus aureus* that produce an exotoxin (TSST-1, enterotoxin B, enterotoxin C). It is most often associated with prolonged use of tampons, but young children (boys and girls) with open skin wounds or minor abrasions can also develop TSS.

38. How do the exotoxins cause shock?

TSST-1 and the other exotoxins are profound vasodilators, and their effect causes distributive shock. Additionally, vasodilation seems to cause rapid movement of fluids and serum proteins to the extravascular space, leading to intravascular volume depletion. Exotoxins also have a direct effect upon the heart that results in decreased myocardial function. Finally, many patients with TSS experience vomiting or diarrhea, which leads to further volume depletion.

39. Are there other forms of TSS?

Yes, a clinical syndrome very similar to TSS has developed in association with group A streptococcal infections (*Streptococcus pyogenes*). Group A streptococci produce exotoxins very similar to those produced by staphylococci. These toxins are called streptococcal pyogenic exotoxins (SPEs). There are three of these: SPEs A, B, and C. SPE A has long been associated with the clinical features of scarlet fever. Exactly why these toxins have recently become more virulent is unknown.

ABDOMINAL PAIN

Payal K. Gala and Jill C. Posner

1. Why is the evaluation of abdominal pain challenging in the pediatric patient?

The diagnosis of abdominal pain can often be determined by a good history and physical examination, but these can be difficult to obtain from infants and young children. In addition, infants with abdominal pain may present with nonspecific signs, such as irritability, poor feeding, and lethargy. Toddlers and young children may report pain but are unable to provide further details, such as the quality, severity, or location of the pain. The history and physical examination of a child require time and patience and may be more difficult to obtain. The differential diagnosis of abdominal pain in children must include both common diseases and a host of disorders unique to the pediatric patient, such as congenital anatomic abnormalities, Henoch-Schönlein purpura, abdominal migraines, and metabolic disorders.

Marin JR, Alpern ER: Abdominal pain in children. Emerg Med Clin North Am 2011;29:401-428.

2. How can I organize my approach to the *stable* patient?

The history and physical examination are the essential components of the evaluation, with judicious use of ancillary testing serving to confirm diagnosis. Elicit the nature of the pain if possible, such as its onset, quality, severity, location, and duration, as well as the presence of any associated symptoms. The differential diagnosis of abdominal pain in children is extensive (Table 5-1). The age of the patient and the most likely diagnoses in that age group can be used in concert with the history and physical examination to narrow the differential and guide further diagnostic testing.

3. How can I maximize the physical examination of the pediatric patient with abdominal pain?

In approaching any child with abdominal pain, one must take time to establish rapport. Essential information can be obtained before even touching the patient. Begin with observation, often best accomplished from outside the room. Notice the general appearance of the child. Is he lying still on the stretcher, suggesting peritonitis, or writhing with colicky pain? Is she running around the room playing with toys? Continue by examining the least threatening areas first and saving particularly invasive aspects of the examination for last (e.g., otoscopy). Distracting the child with a toy or by conversation will allow for a more reliable examination. Laying the child with his or her knees flexed may facilitate relaxation of the rectus muscles. The child's "help" can be elicited during the abdominal examination by allowing him to place his hands on top of the examiner's during palpation. Apply gentle pressure beginning in a location away from the area identified by the child as the most painful. To assess for peritoneal signs, ask the child to hop up and down; or ask parents to gently bounce a baby in their lap to elicit presence of pain. If a digital rectal examination is deemed necessary, insert a small finger into the rectal vault.

Marin JR, Alpern ER: Abdominal pain in children. Emerg Med Clin North Am 2011;29:401-428.

4. What are the life-threatening causes of abdominal pain?

- Appendicitis
- Intussusception
- Incarcerated hernia
- Trauma (accidental or inflicted injury)
- Tumors
- Sepsis
- Malrotation/volvulus
- Ectopic pregnancy

- Diabetic ketoacidosis
- Intra-abdominal abscess (pelvic inflammatory disease, inflammatory bowel disease)
- Hemolytic uremic syndrome
- Intestinal obstruction
- Pancreatitis
- Megacolon
- Metabolic acidosis/inborn error of metabolism
- Aortic aneurysm
- Toxic ingestion (iron, lead, aspirin)

Table 5-1. Causes of Acute Abdominal Pain

INFANCY (<2 YEARS OF AGE)	PRESCHOOL AGE (2-5 YEARS OF AGE)	SCHOOL AGE (>5 YEARS OF AGE)	ADOLESCENT
Common			
Colic (age < 3 mo), GERD, acute gastroenteritis, "viral syndromes"	Acute gastroenteritis, UTI, trauma, appendicitis, pneumonia, asthma, sickling syndromes, "viral syndromes," constipation	Acute gastroenteritis, trauma, appendicitis, UTI, functional abdominal pain, sickling syndromes, constipation, "viral syndromes"	Acute gastroenteritis, gastritis, colitis, GERD, trauma, constipation, appendicitis, pelvic inflammatory disease, UTI, pneumonia, asthma, "viral syndromes," dysmenorrhea, epididymitis, lactose intolerance, sickling syndromes, mittelschmerz
Less Common			
Trauma (possible child abuse), intussusception, incarcerated hernia, sickling syndromes, milk protein allergy	Meckel's diverticulum, Henoch-Schönlein purpura, toxin, cystic fibrosis, intussusception, nephrotic syndrome	Pneumonia, asthma, cystic fibrosis, inflammatory bowel disease, peptic ulcer disease, cholecystitis, pancreatic disease, diabetes mellitus, collagen vascular disease, testicular torsion	Ectopic pregnancy, testicular torsion, ovarian torsion, renal calculi, peptic ulcer disease, hepatitis, cholecystitis or pancreatic disease, meconium-ileus (cystic fibrosis), collagen vascular disease, inflammatory bowel disease, toxin
Very Uncommon or Rare			
Appendicitis, volvulus, tumors (e.g., Wilms' tumor), toxin (heavy metal, lead), malabsorptive syndromes	Incarcerated hernia, neoplasm, hemolytic uremic syndrome, rheumatic fever, myocarditis, pericarditis, hepatitis, inflammatory bowel disease, choledochal cyst, hemolytic anemia, diabetes mellitus, porphyria	Rheumatic fever, toxin, renal calculi, tumor, ovarian torsion, meconium-ileus (cystic fibrosis), intussusception	Rheumatic fever, tumor, abdominal abscess

GERD, gastroesophageal reflux disease; UTI, urinary tract infection.

Source: Neuman MI: Pain—Abdomen. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, p 422.

5. What are some extra-abdominal causes of abdominal pain?

Pain originating at sites distant from the abdomen can manifest as abdominal pain. In processes such as lower lobe pneumonia, afferent nerves from the parietal pleura share central pathways with those that originate from the abdominal wall. Similarly, scrotal pain may be referred to the abdomen. Remember to always perform a testicular examination on all males with abdominal pain! Other illnesses that are associated with abdominal pain include streptococcal pharyngitis, diabetes mellitus, testicular disease, sickle cell disease with vaso-occlusive crisis, lead toxicity, and porphyria. Physical examination may identify one of these diseases as the cause of abdominal pain.

Marin JR, Alpern ER: Abdominal pain in children. Emerg Med Clin North Am 2011;29:401-428.

Key Points: Problems in Diagnosing Pediatric Abdominal Pain

1. History may be limited; physical examination may be difficult.
2. Plain films are often not diagnostic.

6. Does the use of intravenous (IV) analgesia affect diagnostic accuracy in children with *acute* abdominal pain of unknown cause?

Multiple studies with adults and children have shown that giving IV narcotics at adequate doses to decrease pain does not adversely affect the physical examination or diagnostic accuracy in patients with abdominal pain of unknown cause. In fact, pain control might facilitate localization of the origin of the pain, thereby improving the diagnostic accuracy of the physical examination. Therefore, avoid delaying administration of proper analgesia simply because the diagnosis is unknown or the child is awaiting examination by a consultant.

Anderson M, Collins E: Analgesia for children with acute abdominal pain and diagnostic accuracy. Arch Dis Child 2008;93:995-997.

Green R, Bulloch B, Kabani A, et al: Early analgesia for children with acute abdominal pain. Pediatrics 2005;116:978-983.

7. Which blood tests may be useful in the evaluation of abdominal pain?

Patients who have experienced persistent vomiting and appear dehydrated may have electrolyte abnormalities. In young infants, patients who will undergo surgery, or patients who are more severely ill, it may be more important to obtain blood studies to evaluate abdominal pain. The benefit of the complete blood count (CBC) remains controversial. Although an elevated white blood cell count is common in appendicitis, this finding is neither sensitive nor specific. The absolute neutrophil count (ANC) and C-reactive protein test (CRP) may also be helpful in diagnosing appendicitis. The erythrocyte sedimentation rate (ESR) may be helpful in the diagnosis of inflammatory bowel disease. Measurement of liver aminotransferase values is useful in patients with scleral icterus or right-upper-quadrant tenderness. An elevation of one or both of serum amylase and lipase in the appropriate clinical context supports the diagnosis of pancreatitis.

Kwan KY, Nager AL: Diagnosing pediatric appendicitis: Usefulness of laboratory markers. Am J Emerg Med 2010;28:1009-1015.

Marin JR, Alpern ER: Abdominal pain in children. Emerg Med Clin North Am 2011;29(2):401-428.

8. What other laboratory testing may be helpful in certain patients?

A urinalysis can be helpful in diagnosing urinary tract infections and renal calculi but can be misleading if pyuria is due to cervicitis or bladder/ureteral irritation from an adjacent inflamed appendix. A urine pregnancy test is indicated in postpubertal females with abdominal pain. Additionally, include diagnostic testing for gonorrhea and chlamydia infection when indicated.

9. What are the two most common causes of acute abdominal emergencies in children?

Appendicitis is the most common cause in the United States, followed by intussusception.

Pollack ES: Pediatric abdominal surgical emergencies. Pediatr Ann 2003;25:448-457.

10. What is the typical presentation of appendicitis in children?

Classically, periumbilical abdominal pain precedes the onset of vomiting and is associated with low-grade fever, nausea/vomiting, and anorexia. As the inflammation of the appendix advances

and touches the adjacent peritoneum, the pain localizes to the right lower quadrant at McBurney's point. However, the clinical course often does not follow the "textbook" description, so the diagnosis can be difficult to make and the physician must maintain a high index of suspicion. Remember that history of abdominal pain preceding vomiting is a clue to differentiate appendicitis from acute gastroenteritis.

D'Agostino J: Common abdominal emergencies in children. Pediatric Emerg Med 2002;20:139-153.
Marin JR, Alpern ER: Abdominal pain in children. Emerg Med Clin North Am 2011;29:401-428.

11. What are some of the pitfalls in diagnosing appendicitis?

The presentation of a child with appendicitis may not be "textbook." The absence of fever or anorexia, pain in an atypical location, the presence of diarrhea, prolonged symptoms, and normal laboratory values can occur in patients with appendicitis. An appendix that is located in the lateral gutter can cause flank pain and lateral abdominal tenderness; an appendix that lies toward the left may produce hypogastric tenderness; and a retrocecal appendix may cause back or pelvic pain or pain elicited only on deep palpation. Although vomiting occurs more commonly, diarrhea may result from direct sigmoid irritation from the adjacent low-lying pelvic appendix. Similarly, bladder or ureteral irritation may result in dysuria and pyuria, confusing the diagnosis for a urine infection.

12. What are the test characteristics of ultrasound and computed tomography (CT) for the evaluation of children with suspected appendicitis?

Ultrasonography is beneficial because it does not expose the child to ionizing radiation. However, it is operator dependent and may have limited use in obese children. Ultrasound can reach sensitivities of 90% and specificities of 97% for appendicitis with an experienced operator. However, if ultrasound does not visualize the appendix, appendicitis cannot be ruled out. Then, CT scan may be helpful (or perhaps MRI–see Question 14). CT is less operator dependent than ultrasonography, may provide several alternate diagnoses, and has excellent reported test characteristics in children. The sensitivity is up to 97% and specificity is 97%.

Doria AS, Moineddin R, Kellenberger CJ, et al: US or CT for diagnosis of appendicitis in children and adults? A metaanalysis. Radiol 2006;241:83-94.
Krishnamoorthi R, Ramarajan N, Wang N, et al: Effectiveness of a staged US and CT protocol for the diagnosis of pediatric appendicitis: reducing radiation exposure in the age of ALARA. Radiol 2011;259:231-239.

13. Is oral and IV contrast necessary in CT for the evaluation of children with suspected appendicitis?

CT for appendicitis requires IV contrast to detect inflammation due to appendicitis or various other causes, but oral contrast is not necessary for the diagnosis of appendicitis. There is an increasing body of evidence that shows that there is no diagnostic compromise in those children who undergo CT without oral contrast for suspected appendicitis. In fact, diagnostic performance of CT without oral contrast has been found to be equivalent or better compared to CT with oral contrast. There is a high percentage of patients that do take oral contrast for whom the contrast does not even reach the point of interest, the terminal ileum, prior to the CT. In addition, delayed diagnostic evaluation, frequency of emesis after contrast bolus, and the need for a nasogastric tube to tolerate the bolus all limit the efficacy of oral contrast for CT.

Anderson BA, Salem L, Flum DR: A systematic review of whether oral contrast is necessary for the computed tomography diagnosis of appendicitis in adults. Am J Surg 2005;190:474-478.
Laituri, Fraser JD, Aguayo P, et al: The lack of efficacy for oral contrast in the diagnosis of appendicitis by computed tomography. J Surg Res 2011;170:100-103.

14. Is magnetic resonance imaging (MRI) useful in the evaluation of children with suspected appendicitis?

MRI has been suggested as an alternative to CT in children suspected of appendicitis with an inconclusive ultrasound to avoid the detrimental effects of radiation. It has been shown that the sensitivity of MRI without contrast in diagnosing appendicitis has been up to 100%, with a specificity of 96%, a positive predictive value of 88%, and a negative predictive value of 100%.

This diagnostic performance, in addition to its lack of radiation, makes MRI attractive as a potential alternative to CT for the diagnosis of appendicitis.

Apelsund G, Fingeret A, Gross E, et al: Ultrasonography/MRI versus CT for diagnosing appendicitis. Pediatrics 2014;133:1-8.

Herliczek TW, Swenson DW, Mayo-Smith WW: Utility of MRI after inconclusive ultrasound in pediatric patients with suspected appendicitis: Retrospective review of 60 consecutive patients. AJR Am J Roentgenol 2013;200:969-973.

Moore MM, Gustas CN, Choudhary AK, et al: MRI for clinically suspected pediatric appendicitis: An implemented program. Pediatr Radiol 2012;42:1056-1063.

15. Which patients are more likely to develop appendiceal perforation?

Young children, those with atypical presentations, and those who present early in their clinical course are at the highest risk.

16. What is the "classic triad" of intussusception? Does it occur in most patients?

Intussusception occurs when a portion of the bowel, usually the distal ileum, telescopes into an adjacent segment of bowel. This effectively leads to intestinal obstruction followed by venous congestion and, finally, arterial insufficiency. The classic triad of pain, currant-jelly stool, and abdominal mass on palpation is present in only 20% to 25% of children with intussusception.

17. In which patients should the diagnosis of intussusception be considered?

In the classic description of intussusception, a child age 3 months to 3 years presents with the legs intermittently drawn up to the chest while crying, bloody stools, vomiting, and a sausage-shaped abdominal mass. Unfortunately, the classic presentation is not common, so a high index of suspicion should be maintained for those children presenting with intermittent colicky abdominal pain and vomiting. Some children present with lethargy or a change in mental status. The goal is to diagnose and treat intussusception prior to the evolution of "currant-jelly stools," an indicator that significant bowel ischemia has occurred.

Marin JR, Alpern ER: Abdominal pain in children. Emerg Med Clin North Am 2011;29:401-428.

18. What imaging modalities are commonly used in children to confirm or rule out intussusception?

- **Plain radiographs** lack sensitivity and many false-negative results occur, so normal plain films should not exclude the diagnosis of intussusception. Later in the disease, up to 60% may show absence of air in the right upper and lower quadrants and evidence of soft tissue density.
- **Ultrasonography** has the advantage of being relatively fast, noninvasive, and without exposure to ionizing radiation. Although it may be operator dependent, in experienced hands the sensitivity (98-100%) and specificity (88-100%) are high. It is important to note that if intussusception is detected on ultrasound, air or barium contrast enema under fluoroscopy is still indicated for treatment.
- **Contrast enema** has long been the standard for diagnosis and treatment of intussusception. Barium or air is introduced under pressure into the bowel via a tube in the rectum during fluoroscopy. Air is safer, cheaper, and more effective at reduction and poses less risk if there is bowel perforation.

Byrne AT, Goeghegan T, Govender P, et al: The imaging of intussusception. Clin Radiol 2005;60:39-46.

Marin JR, Alpern ER: Abdominal pain in children. Emerg Med Clin North Am 2011;29:401-428.

19. What abnormalities may appear on plain radiographs in children with abdominal pain?

The plain film should be assessed for "bones, stones, masses, and gas." An appendicolith is present in only about 5% to 15% of patients with appendicitis. Other findings in appendicitis may include sentinel loop, air-fluid levels, fecolith, mass in right lower quadrant, and indistinct psoas margins with scoliosis toward the right. Rarely, a perforated appendix may produce pneumoperitoneum. Some renal calculi can be visualized on plain radiographs of the abdomen. The invaginating bowel of intussusception may be apparent as an intraluminal density, but the

more common finding is a paucity of air in the right upper and lower quadrants. Multiple stacked, dilated loops of bowel with air-fluid levels and the absence of distal air may signify intestinal obstruction. Abdominal radiographs may show evidence of constipation, previously unsuspected, or foreign bodies (FBs) such as ingested magnets.

Marin JR, Alpern ER: Abdominal pain in children. Emerg Med Clin North Am 2011; 29:401–428.

20. Are there any other useful radiologic studies for children with abdominal pain?

Ultrasound is an integral component of the workup for pyloric stenosis and for renal calculi and can be useful in evaluating the postpubertal female with possible ovarian or uterine disease, or the child with suspected appendicitis. The CT scan is useful for detecting renal calculi and intra-abdominal infections, including appendicitis. However, it is important to remember that CT exposes children to a substantial amount of radiation. An upper gastrointestinal (GI) series with small bowel follow-through is used to detect intestinal malrotation. Failure of the C-loop of the duodenum to cross the midline and an abnormal location of the cecum signify a malrotation is likely.

21. What is the management of children who ingest foreign bodies?

Children commonly ingest FBs due to their natural curiosity and play. Most ingested FBs pass spontaneously if they move beyond the gastroesophageal junction, are less than 5 cm in length, and are not very sharp (sewing needles). Most sharp objects such as tacks, screws, and staples will safely pass surrounded by stool. Frequent radiographs for asymptomatic children are unnecessary for commonly ingested FBs.

However, a heightened measure of caution should be exercised when the FB is a button battery or a magnet. Button battery and magnet ingestions are on the rise as a result of their incorporation into many childhood toys. Button batteries are also present in small electronics, hearing aids, and musical greeting cards. Of particular concern are lithium batteries, which have an external current that can damage surrounding tissue. Button batteries can become lodged against mucosa in the nose or esophagus and have the potential to cause necrosis, perforation, and life-threatening GI bleed. These nasal and esophageal button batteries should be removed immediately. Batteries in the stomach usually pass spontaneously without causing abdominal pain.

A single magnet may cause little problem, but if more than one magnet is ingested, their attraction across bowel wall can cause necrosis leading to obstruction, volvulus, or perforation. These patients can present with abdominal pain, vomiting, constipation, and peritoneal irritation. It is important to remember that on radiography, two attached magnets can appear as one on film. The ingestion of a single magnet should be managed by a two-view radiograph, and if the patient is asymptomatic, he or she should be discharged with close follow-up in 3 days. A repeat radiograph should be considered to ensure movement along the GI tract. Adequate discharge instructions should be given for symptoms suggestive of perforation or obstruction. Children who ingest multiple magnets or one magnet in addition to a metallic object or have signs of intestinal obstruction with single magnet ingestion should be evaluated by a surgeon for endoscopic or operative removal because of the risks discussed.

Schunk JE: Foreign body—Ingestion/aspiration. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 767-768, 777-779.

Tavarez MM, Saladino RA, Gaines BA, Manole MD: Prevalence, clinical features and management of pediatric magnetic foreign body ingestions. J Emerg Med 2013;44:261-268.

22. A 17-year-old female presents with right-upper-quadrant abdominal pain and low-grade fever. She denies vomiting, diarrhea, dysuria, or vaginal discharge. The physical examination is remarkable for mild right-upper-quadrant tenderness without peritoneal signs. She is anicteric. The pelvic examination does not reveal discharge, cervical motion, or adnexal tenderness. What is the most likely diagnosis?

Fitz-Hugh-Curtis syndrome occurs in 5% to 10% of patients with chlamydial or gonococcal pelvic inflammatory disease. It is theorized that seeding of the peritoneal cavity occurs as the organism ascends the female genital tract. It then tracks along the paracolic gutter, reaches the liver, and causes inflammation of its capsule. There have been several reported cases of

perihepatitis in men, thus precipitating the emergence of alternative hypotheses for its pathophysiology: lymphatic or hematogenous spread. In affected women, the pelvic examination may be normal and cervical cultures may not isolate an organism. Hepatic aminotransferase values are normal or transiently mildly elevated. In most cases, the diagnosis is inferred as the symptoms abate with antibiotic therapy. Definitive diagnosis can only be made laparoscopically.

Peter NG, Clark LR, Jaeger JR: Fitz-Hugh-Curtis syndrome: A diagnosis to consider in women with right upper quadrant pain. Cleve Clin J Med 2004;71:233-239.

23. Describe the management of an acute abdominal emergency.

The immediate management should begin with a careful assessment of the patient's airway, breathing, and circulation, particularly in an unstable patient. Intravascular access should be obtained and fluid resuscitation initiated with IV normal saline (20 mL/kg). Laboratory studies, including a blood glucose level, may be sent with blood obtained during the IV placement. Promptly administer broad-spectrum antibiotics, including anaerobic coverage, if there is a strong possibility of sepsis, and obtain surgical consultation as early as possible if there is a suspicion of a surgical emergency.

McCollough M, Sharieff GO: Abdominal surgical emergencies in infants and young children. Emerg Med Clin North Am 2003;21:909-935.

24. What is the most common cause of recurrent abdominal pain?

Although numerous organic causes are possible, up to 30% of patients will have functional pain with diagnoses such as irritable bowel syndrome and abdominal migraines. In the functional abdominal pain syndrome, the pain is generally episodic, is periumbilical, rarely occurs during sleep, and rarely is associated with eating or activities. There are no signs of systemic illness, such as fever, diarrhea, vomiting, rash, or joint pain. The child's growth and development are normal. The physical examination is usually unremarkable, with the exception of mild midabdominal tenderness without peritoneal signs.

25. How should the diagnosis of functional abdominal pain be addressed in the emergency department (ED)?

The diagnosis of functional abdominal pain is usually evident following the completion of the history and physical examination. Failure to mention this diagnosis early or obtaining unnecessary studies to appease an anxious family may only result in the parents feeling that "the right" diagnostic test has yet to be performed. The parents and child should be reassured that stress-related abdominal pain is real pain and not due to the child's "faking it." They should be encouraged to continue their normal activities (e.g., school attendance) and seek psychological services. Finally, the emergency physician should provide careful instructions on the symptoms that should prompt an immediate return to the ED and should encourage follow-up with the child's primary care provider.

Key Points: Abdominal Emergencies

1. Children may not have "classic" features of appendicitis or intussusception.
2. CBC is not specific for appendicitis.
3. Analgesia should not be withheld from a child with abdominal pain of unknown cause simply for fear of delaying diagnosis or causing misdiagnosis.
4. Children are at particularly high risk of ruptured appendix, with the youngest children possessing the highest risk.

Acknowledgment

The authors wish to thank Dr. Reza Dougherty for his contributions to this chapter in the previous edition.

CHAPTER 6

ALTERED MENTAL STATUS

Douglas S. Nelson

1. How can altered mental status (AMS) present in an infant?

It may present as a combination of excessive crying, irritability, poor feeding, or sleeping more or less than usual.

2. An afebrile 3-year-old looking acutely intoxicated is brought in by his grandmother. Blood and urine toxicologic screens are negative, as are complete blood count (CBC), electrolytes, and brain computed tomography (CT). What is the likely diagnosis?

Toxic ingestion, despite the lack of a positive finding on toxicologic screening. Many substances that affect the function of the human central nervous system (CNS) are not detected on these screens, which focus on drugs of abuse. Those causing AMS with miosis include clonidine, organophosphates, and tetrahydrozoline. Mydriasis and AMS are found with other toxins: carbon monoxide, cyanide, methemoglobinemia, lysergic acid diethylamide (LSD), and γ-hydroxybutyrate (GHB). If someone in the home is using nicotine patch or e-cigarette products, suspect acute nicotine poisoning.

Connolly G, Richter P, Aleguas A Jr., et al: Unintentional child poisonings through ingestion of conventional and novel tobacco products. Pediatrics 2010;125:896.

Wang GS, Deakyne S, Bajaj L, et al: The limited utility of screening laboratory tests and electrocardiograms in the management of unintentional asymptomatic pediatric ingestions. J Emerg Med 2013;45(1):34-38.

Key Points: When to Suspect Toxic Ingestion in a Child with Altered Mental Status and No History of Ingestion

1. No history or physical examination findings of head trauma
2. Sudden onset of symptoms
3. Large number of children, including visitors, in the home
4. Previous ingestions by the patient or their siblings
5. Presence in the home of a sibling on multiple medications for behavior control or seizures

3. What scales are in use to quantify AMS?

The level of consciousness of a neurologically impaired patient may initially be evaluated by using a simple AVPU scale, representing four major levels of alertness: alert (A), responsive to verbal stimuli (V), responsive to painful stimuli (P), and unresponsive (U).

A more widely used measurement of consciousness is the Glasgow Coma Scale (GCS). Patients are graded on three areas of neurologic function: eye opening, motor responses, and verbal responsiveness. These numeric scores are added to determine the GCS score. A GCS score of 3 is the minimum score possible and represents complete unresponsiveness, and a GCS score of 15 is assigned to fully alert patients. Details of the scores assigned are listed, for children above 2 years of age and below, in Table 6-1 and Table 6-2.

4. Why should I assign a GCS score to every patient with AMS?

There are several good reasons to use a standard quantifiable mental status scale. It allows evaluation of a patient's changing neurologic status over time and the recording of this information in the medical record. The effect of medical interventions may then be more easily assessed. The use of accepted scoring systems also facilitates communication with consultants, such as neurologists and neurosurgeons.

Table 6-1. Glasgow Coma Scale

SCORE VALUE	EYE OPENING	BEST MOTOR RESPONSE	BEST VERBAL RESPONSE
6		Obeys verbal command	
5		Localizes to pain	Oriented, converses
4	Spontaneous	Flexion withdrawal	Disoriented, converses
3	To speech	Flexion decorticate	Inappropriate words
2	To pain	Extension decerebrate	Incomprehensible sounds
1	None	None	None

Table 6-2. Pediatric Glasgow Coma Scale (Use for Patients under 2 Years of Age)

SCORE VALUE	EYE OPENING	BEST MOTOR RESPONSE	BEST VERBAL RESPONSE
6		Normal spontaneous movement	
5		Withdraws to touch	Coos, babbles
4	Spontaneous	Withdraws to pain	Irritable, cries
3	To speech	Abnormal flexion	Cries to pain
2	To pain	Abnormal extension	Moans to pain
1	None	None	None

Table 6-3. "Immunization" Mnemonic Clues to Causes of Altered Mental Status

DPT	Dehydration, poisoning, trauma
OPV	Occult trauma, postictal or postanoxia, ventriculoperitoneal shunt
HIB	Hypoxia or hyperthermia, intussusception, brain masses
MMR	Meningitis or encephalitis, metabolic, Reye's syndrome + other rarities

5. What do the letters DPT, OPV, HIB, and MMR stand for with respect to AMS?

Although these letters represent abbreviations for several childhood immunizations, they also are useful as a mnemonic to recall common causes of abnormal mental status (Table 6-3).

Schunk JE: The pediatric patient with altered level of consciousness: Remember your "immunizations." J Emerg Nurs 1992;18:419-421.

6. When should I consider obtaining a CT scan on a child with AMS?

Consider CT if there is any history of trauma, any focal or lateralizing signs on physical examination, or any suspicion of physical abuse.

7. Can children presenting with abnormal mental status just be observed?

The only common situation when a child with AMS can be observed is when they are in the postictal period from a recent seizure. Otherwise, children presenting with AMS due to

trauma, illness, known toxic ingestions, or undefined reasons almost always need laboratory or radiographic studies to be performed. A recent large study by the Pediatric Emergency Care Applied Research Network (PECARN) identified children at low risk for clinically important traumatic brain injury (ciTBI), but children with GCS scores of 14 or less, or any other signs of AMS, were in the group at high risk of ciTBI, and CT scanning was recommended.

Kuppermann N, Homes JF, Dayan PS, et al: Identification of children at very low risk of clinically-important brain injuries after head trauma: A prospective cohort study. Lancet 2009;374(9696):1160-1170.

8. A teenager is brought to the emergency department (ED) from a party by friends. He is comatose and has profound respiratory depression with no history of head trauma. You intubate him, order a toxicologic screen, and obtain a CT scan, which are both normal. Upon returning from the scanner, the patient sits up, rips the endotracheal tube out of his mouth, and wants to leave. What was the most likely cause of his problem?

At the party he drank GHB. CNS effects of GHB include drowsiness, ataxia, confusion, hallucinations, amnesia, incontinence, seizures, and coma. Mydriasis and nystagmus may be present, accompanied by respiratory depression, bradycardia, and hypotension.

Recovery from GHB intoxication is usually rapid, within several hours after ingestion. Routine toxicologic screens miss the presence of this club drug, which should now be considered in the differential diagnosis of all teens presenting with abnormal mental status. Toddlers have presented with AMS after ingesting toys containing a GHB precursor, 1,4-butanediol.

Galicia M, Nogue S, Miro O: Liquid ecstasy intoxication: Clinical features of 505 consecutive emergency department patients. Emerg Med J 2011;28(6):462-466.

Key Points: "Club Drugs" That Most Commonly Cause Altered Mental Status in Teens

1. Flunitrazepam (Rohipnol, "roofies")
2. Gamma-hydroxybutyrate (GHB, "liquid ecstacy," "liquid X," "lollypops")
3. Gamma-butyrolactone ("GBL," "Blue Nitro," "Firewater," "G3," "Gamma G")
4. Ketamine ("vitamin K")
5. Lysergic acid diethylamide (LSD, "acid")
6. Methamphetamine ("meth," "crystal")
7. Methylenedioxymethamphetamine (MDMA, "ecstasy," "X," "XTC")

9. An 11-year-old male born at 28 weeks' gestation with a history of cerebral palsy and shunt-dependent hydrocephalus is brought to the ED because of excessive somnolence and bradycardia. He has an intrathecal baclofen pump, has a ventriculoperitoneal shunt, and is afebrile and bradycardic, with normal blood pressure (BP). What is causing his AMS?

As more extremely premature infants survive, increasing numbers of individuals live with implanted medical devices such as cerebrospinal fluid (CSF) shunts and intrathecal baclofen pumps. A brain CT showed that this patient's shunt was functioning. Query of the patient's baclofen pump revealed a malfunction, and the patient was admitted to the hospital with presumed baclofen toxicity. Any child with an intracranial CSF shunt or a baclofen pump and AMS has a shunt or pump problem until proven otherwise.

Yeh RN, Nypaver MM, Deegan TJ, Ayyangar R: Baclofen toxicity in an 8-year-old with an intrathecal baclofen pump. J Emerg Med 2004;26:163-167.

10. A 6-month-old infant is brought in by her mother after being left alone with the mother's boyfriend. She was well yesterday, but today will not feed and is sleepier than normal. She has no fever, congestion, vomiting, or diarrhea. The physical examination is normal except that the child seems more difficult to arouse than usual. What possible causes should be considered?

Physical abuse is most likely, most commonly of the "shaken baby" type, when the whipping motion of an infant's head causes tearing of cortical bridging veins between the dura and arachnoid veins, leading to subdural hematoma formation. These hematomas can occur

bilaterally and are 5 to 10 times more common than epidural bleeding. Subdural hematomas may occur on a chronic basis in young abused children and are associated with skull fractures in 30% of cases. Neuroimaging classically reveals crescent-shaped lesions between the brain and skull. Skeletal surveys performed on these children typically show fractures in various stages of healing.

11. Magnetic resonance imaging (MRI) provides a sharper, more detailed picture of the brain than does CT. Why, then, is CT usually performed first in a patient presenting with abnormal mental status?

MRI scans are more costly, take longer to obtain, and are generally more difficult to arrange than CT scans. CT images show most structural lesions that may present with AMS, such as tumors or hemorrhage. Young children may require sedation for MRI, but not for a quick head CT.

12. What are clues that a child may be "faking" an AMS?

Malingering should be suspected if the patient has a psychiatric history or falls down from "spells" without ever being injured. On physical examination, when you lift the patient's hand to a position directly over his or her face and let go, the patient will not hit himself or herself in the face unless there has been an alteration in mental status. In very convincing cases, it may be necessary to perform electroencephalography to prove this suspicion.

13. A 3-year-old presents one winter day with sleepiness and vomiting. Other family members feel unwell but are less ill. No history of ingestion or head trauma is present. Physical examination reveals a well-appearing but drowsy child with mild tachypnea and tachycardia. The examination is otherwise normal; oxygen saturation is 100% in room air. Electrolytes, CBC, and blood gas samples are obtained. What test comes back with abnormal results?

The blood gas, which shows a carboxyhemoglobin level of 20%. This level indicates that the patient and presumably his or her family are being poisoned by carbon monoxide. This cause of abnormal mental status is seen most often in early winter, as families turn on their furnaces for the first time since the previous heating season, or in electrical blackouts with home generator use. Treatment usually consists of administering 100% oxygen via a rebreather face mask. Severe cases, such as in children rescued from house fires, may require endotracheal intubation and hyperbaric oxygen. Pulse oximetry often reads 100%, as carboxyhemoglobin is misread as oxygenated hemoglobin.

Cho CH, Chiu NC, Ho CS, Peng CC: Carbon monoxide poisoning in children. Pediatr Neonatol 2008;49 (4):121-125.

Fife CE, Smith LA, Maus EA, et al: Dying to play video games: Carbon monoxide poisoning from electrical generators used after hurricane Ike. Pediatrics 2009;123(6):e1035-e1038.

14. If the physical examination of a patient with AMS does not reveal the source of neurologic disability, what laboratory tests should I consider?

Glucose is the most important laboratory test to check, and one of the fastest and easiest. In many institutions, a rapid bedside test can be done to provide a value in minutes. Consider also a toxicologic screen, electrolytes, or head CT.

15. What subtle signs may represent seizure activity as the cause of AMS?

Suspect seizure if the patient is dazed or confused and exhibits staring, swallowing, eye blinking, lip quivering, nystagmus, and automatisms (motor actions performed without conscious intent). The degree of neurologic impairment can fluctuate over time.

Benson PJ, Klein EJ: New-onset absence status epilepsy presenting as altered mental status in a pediatric patient. Ann Emerg Med 2001;37:402-405.

16. What causes of abnormal mental status are particularly life-threatening?

The list includes epidural hematoma, cerebral edema, brain neoplasms, cerebral infarctions, CSF shunt malfunction, meningitis, encephalitis, toxic ingestions, carbon monoxide poisoning, hypotension, hypoxia, and sepsis.

Nelson DS: Coma and altered level of consciousness. In Fleisher G, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 176-186.

17. A 4-year-old boy is brought to the ED because of sleepiness, vomiting, and "not acting right." History is positive for birth at 30 weeks' gestation but is negative for head trauma, fever, toxic ingestion, or other signs of illness. On examination, the patient is afebrile, with a heart rate of 120 beats per minute (BPM), BP of 170/100 mm Hg (quite high for the patient's age), and a respiratory rate of 36 breaths per minute. The patient is difficult to arouse and cries when awake. Lumbar puncture (LP) is not performed because of concerns of increased intracranial pressure. A CT scan is read as normal, but MRI reveals subtle occipital abnormalities. What problem caused the patient's abnormal mental status?

The patient has hypertensive encephalopathy due to renal artery stenosis caused by an umbilical artery catheter used during his postnatal neonatal intensive care unit stay. The MRI findings are consistent with posterior leukoencephalopathy syndrome. Clinical findings may include AMS, seizures, headache, and blindness. Appropriate treatment of hypertension results in resolution of neurologic and MRI abnormalities. BP values obtained in the ED setting are often elevated due to patient anxiety/crying, the use of electronic BP machines, or wrong cuff size, and as a result practitioners in that setting tend to discount high BPs. Sometimes, however, they are real and are actually the root of the problem.

Singhi P, Subramanian C, Jain V, et al: Reversible brain lesions in childhood hypertension. Acta Paediatr 2002;91:1005-1007.

18. A 12-year-old boy with a history of sinus and ear infections is brought to the ED by his parents, who are concerned by his diminished responsiveness. The previous evening, he had reported a headache and seemed to have a tactile fever. On examination, he is somnolent with slow reaction times, but no focal neurologic findings. Why is he ill?

The patient has a subdural abscess from long-standing sinusitis. Intracranial complications of sinusitis are rare but possible in childhood. When pus-filled sinuses decompress into the cranial vault, minimal facial tenderness may be present. A high index of suspicion is needed in patients with a history or symptoms of sinus infections. Brain and sinus CT scans will reveal the cause of the patient's mental status.

Calik M, Iscan A, Abuhandan M, et al: Masked subdural empyema secondary to frontal sinusitis. Am J Emerg Med 2012;30(8):1657.

Hicks CW, Weber JG, Reid JR, Moodley M: Identifying and managing intracranial complications of sinusitis in children: A retrospective series. Pediatr Infect Dis J 2011;30(3):222-226.

19. A 7-year-old girl who has an unremarkable recent medical history is brought to the ED because she is much sleepier than usual. Examination shows an afebrile, well-nourished, well-developed child who prefers to sleep. When awakened, the patient seems confused, and "not all there." CBC, electrolytes, blood gas, urinalysis, toxicologic screen, and noncontrast head CT are all normal. What tests are indicated? What did they show in this patient?

The patient needs an LP and an MRI to check for acute disseminated encephalomyelitis (ADEM). ADEM is an immune-mediated inflammatory process that may appear following viral infection, after vaccination, or spontaneously. This autoimmune demyelination process produces AMS characterized by irritability, sleepiness, confusion, obtundation, seizures, and coma. CSF findings are variable but usually include moderate leukocytosis with a lymphocytic or monocytic predominance, although they can be normal in 20% of cases, and MRI shows white matter lesions.

Elhassanien AF, Aziz HA: Acute demyelinating encephalomyelitis: Clinical characteristics and outcome. J Pediatr Neurosci 2013;8(1):26-30.

Fridinger SE, Alper G: Defining encephalopathy in acute disseminated encephalomyelitis. J Child Neurol 2013;29(6):751-755.

Key Points: Tips for Performing Lumbar Punctures in Older Children

1. Provide anxiolysis with intranasal midazolam (0.4 mg/kg, maximum 10 mg).
2. Use ethyl chloride spray (or ice held against the skin) to numb the site where you will inject lidocaine.
3. Anesthetize the L4-L5 space with buffered 1% lidocaine (not from the LP kit) using small needles before donning sterile gloves and opening the LP tray.
4. Try positioning the patient sitting up, leaning over pillows, and being stabilized by ED staff.
5. Use video content on a tablet or smartphone to distract the patient, who can use earphones.

20. An 8-year-old girl from a rural area is brought into the ED agitated and confused. She has had a low-grade fever and malaise for 48 hours. She has no history of head trauma or overseas travel, but she received a new puppy 3 months ago. CT scan is abnormal, with hypodensities in the basal ganglia bilaterally. What unusual illness would be of most concern?

Rabies. Younger domestic animals are more susceptible to acquiring the disease from infected wild animal hosts (skunks, raccoons) via bites or inadvertent sharing of outdoor water bowls. Also consider the disease in a patient with a travel history to areas of the world where the disease is endemic, such as India and the Philippines.

CHAPTER 7

APNEA, SUDDEN INFANT DEATH SYNDROME, AND APPARENT LIFE-THREATENING EVENTS

Andrew D. DePiero

1. **What is the definition of apnea?**
   Apnea is a pause in respiratory airflow. Apnea is usually defined by a respiratory pause greater than 20 seconds or a respiratory pause associated with bradycardia or oxygen desaturation.

2. **What is the difference between apnea of prematurity and apnea of infancy?**
   Apnea of infancy is defined as apnea that occurs after the baby has reached 37 weeks of gestation. Apnea of prematurity is related to the underdeveloped respiratory and neurologic systems.

3. **What is the difference between obstructive apnea and central apnea?**
   Obstructive apnea is related to an obstruction of the upper airway. Respiratory movements continue during attempts to relieve the obstruction. Central apnea is a dysfunction of the neurologic centers that regulate breathing. During central apnea, all respiratory efforts cease. Occasionally, both central and obstructive components are present. This is classified as mixed apnea.

4. **What is periodic breathing?**
   Periodic breathing describes a pattern of a short respiratory pause followed by an increase in respiratory rate. Periodic breathing occurs in cycles and is a normal pattern in infants. During these episodes, babies are neither plethoric nor cyanotic.

5. **What are some of the pathophysiologic factors contributing to apnea in young infants?**
   - **Hypoxic drive:** In the neonate, hypoxia results in a brief increase in respiratory rate followed by depressed respiratory drive and apnea. Mild hypoxemia during sleep can cause periodic breathing or apnea, and hypoxemia during sleep may not cause arousal.
   - **Effects of feeding:** Difficulty with coordination of sucking and breathing can result in hypoxemia. The presence of an accentuated laryngeal chemoreflex can cause apnea and bradycardia if regurgitation occurs while the infant is hypoxic.
   - **Metabolic abnormalities:** Apnea can develop in newborns and young infants as the result of hypoglycemia or anemia.
   - **Mechanical factors:** Because of the pliable thoracic cage and fatigability of the diaphragmatic muscle, attempts to increase minute ventilation by increasing tidal volume can increase the work of breathing. Thus, the infant in respiratory distress is more susceptible to respiratory failure and apnea.

6. **What are some of the underlying causes of apnea?**
   - **Central nervous system:** seizure activity, breath-holding
   - **Infection:** meningitis, bronchiolitis (in premature infants or infants with lung or heart disease), sepsis, croup, infant botulism, pertussis
   - **Cardiac:** arrhythmia
   - **Gastrointestinal:** gastroesophageal reflux (GER)

- **Metabolic:** hypoglycemia, inborn errors of metabolism, toxin (ingestion)
- **Trauma:** accidental or inflicted head or blunt abdominal injury
- **Prematurity**
- **Idiopathic/unknown cause**

Arens R, Gozal D, Williams JC, et al: Recurrent apparent life-threatening events during infancy: A manifestation of inborn errors of metabolism. J Pediatr 1993;123:415-418.

Bloch-Salisbury E, Hall MH, Sharma P, et al: Heritability of apnea of prematurity: A retrospective twin study. Pediatrics 2010;126(4):e779-e787.

7. What is sudden infant death syndrome (SIDS)? How is it diagnosed?

SIDS is defined as "the sudden death of an infant under 1 year of age that remains unexplained after a thorough case investigation, including performance of a complete autopsy, examination of the death scene, and review of the clinical history."

Moon RY: American Academy of Pediatrics Policy Statement. SIDS and other sleep related infant deaths. Expansion of recommendations for a safe infant sleeping environment. Task Force on Sudden Infant Death Syndrome. Pediatrics 2011;128(5):e1341-e1367.

Willinger M, James LS, Catz C: Defining the sudden infant death syndrome (SIDS): Deliberations of an expert panel convened by the National Institute of Child Health and Human Development. Pediatr Pathol 1991;11:677-684.

Key Points: Sudden Infant Death Syndrome

1. SIDS requires a thorough investigation including an autopsy and an examination of the death scene.
2. Placing babies in the supine sleeping position has decreased the incidence of SIDS.
3. An apparent life-threatening event (ALTE) is not predictive of SIDS.
4. Home monitoring after an ALTE does not decrease the incidence of SIDS.

8. What is SUID?

"Sudden unexpected infant death" is a term used to describe all unexpected infant deaths. SUIDs are divided into those that are explained and those that are not explained. Unexplained infant deaths include cases of SIDS and other deaths that occur under unclear circumstances. Explained SUIDs would include, among other causes, ingestions, infections, and accidental suffocation.

9. What are the risk factors for SIDS?

Maternal risk factors for SIDS include young age, smoking during pregnancy, and lack of prenatal care. Other risk factors are prone sleeping position, sleeping on a soft surface, overheating, prematurity, smoking in the home, and low birth weight.

Prone and side sleep positions are associated with an increase in rebreathing of expired gases. Sleep in the prone position can also increase the risk of overheating by decreasing the rate of heat loss. The prone sleep position is thought to alter the autonomic control of the infant's cardiovascular system during sleep. This can result in decreased cerebral oxygenation.

Task Force on Sudden Infant Death Syndrome (SIDS) and Other Sleep-Related Infant Deaths: Expansion of recommendations for a safe infant sleeping environment. Pediatrics 2011;128:1030.

10. Why has there been a decline in the incidence of SIDS?

The recommendation for the supine sleeping position for babies has correlated with a significant decrease in the number of SIDS cases. The explanation for this is unclear. Changes to the sleep environment (firm surface, removing bumper pads, avoidance of overheating/excessive clothing) have also led to a reduction in sleep-related deaths.

11. What is an apparent life-threatening event (ALTE)? Is it related to SIDS?

An ALTE is an event that is "frightening to the observer and that is characterized by some combination of apnea (central or occasionally obstructive), change in color (usually cyanotic or pallid, but occasionally erythematous or plethoric), marked change in muscle tone (usually

limpness), choking or gagging. In some cases, the observer feels the infant has died." Although the cause of an ALTE is frequently elusive, most infants who die from SIDS have not previously had such an event.

National Institutes of Health Consensus Development Conference on Infantile Apnea and Home Monitoring, Sept 29 to Oct 1, 1986. Pediatrics. 1987;79(2):292–299.

12. How should the evaluation begin for an infant after an ALTE?

The evaluation should start with a cardiorespiratory assessment. Most patients are clinically stable after their episode. The evaluation should then focus on two issues:

1. Did a clinically significant episode occur?
2. Is there an underlying condition that requires emergent or urgent evaluation or treatment?

13. How should the history be obtained?

It is essential to question firsthand observers of the apneic event as objectively as possible. This can be challenging considering the stressful nature of the event. Questions such as, "What did the baby look like?" "What color was she?" "Was he awake?" "Did he appear frightened?" and "Then what did you do?" may help the historian relate his or her observations to you more objectively. Information from prehospital personnel is useful as well.

14. Are there specific details that must be included in the history?

Yes. Ask these questions to ensure a thorough history:

- Where did the event take place: in the infant's crib, in another bed, in a car seat?
- How long did the episode last?
- Was the infant awake or asleep?
- Was there a change in the baby's color?
- Was there a change in tone or posture, or were there abnormal movements?
- Did the child require resuscitation, and, if so, how did he respond?
- When was the infant last fed?

15. Which other questions should be asked in the history?

- **History of present illness:** Prior to the event, was the child well? Are there symptoms of other illnesses, specifically changes in behavior, activity, or appetite? Does the baby have GER? Has there been a recent illness, fever, cough, or cold? Has the infant recently received an immunization?
- **Past medical history:** Have there been similar episodes in the past? Were there problems with pregnancy, labor, or delivery? What was the child's birth weight?
- **Family history:** Is there any family history of seizures, infant deaths, or serious illnesses in young family members?
- **Social history:** Who was watching the child at the time of the episode? Are there medications or other toxins accessible to the child?

16. Can a child with normal findings on physical examination have had a significant apneic episode?

Yes. After careful examination, the infant's heart rate, respiratory rate, blood pressure, pulse oximetry, and rectal temperature can be entirely normal—even though the baby "looked dead" at the time of the episode. The remaining physical examination may also be unremarkable. Therefore, you must obtain a careful history to guide further evaluation.

17. Which physical findings indicate a specific cause for an apneic episode?

Fever or hypothermia suggests the possibility of infection. Tachypnea may be the result of respiratory disease or metabolic acidosis. A young infant with cough or wheezing may have bronchiolitis. A child in shock may have sepsis or hypovolemia from an occult injury. Depressed mental status, bulging fontanel, and papilledema are consistent with central nervous system infection or injury. An infant with dysmorphic features may have an underlying congenital abnormality as the cause of apnea.

18. What laboratory tests should be ordered after an ALTE?

There is no standard diagnostic evaluation for infants after an ALTE. The diagnostic evaluation must be guided by the history and physical examination. Positive findings in either the history or physical examination are correlated with establishing an underlying cause for the ALTE. An unremarkable history and normal physical examination do not rule out significant

disease. For example, significant head trauma has been found in infants without physical examination findings.

Brand DA, Altman RL, Purtill K, et al: Yield of diagnostic testing in infants who have had an apparent life-threatening event. Pediatrics 2005;115:885-893.

Morris MW, Smith S, Cressman J, Ancheta J: Evaluation of infants with subdural hematoma who lack external evidence of abuse. Pediatrics 2000;105(3 Pt 1):549-553.

19. What is the relationship between respiratory syncytial virus (RSV) and apnea?

RSV can lead to apnea due to respiratory failure from bronchiolitis or from RSV infection without bronchiolitis. The incidence of apnea from RSV ranges from 1.2% to 23.8%, depending on the study. Patients at high risk for apnea in the hospital are those who were born at full term but are less than a month old, those who were premature at birth and for those who are less than 48 weeks after conception, and those who had a witnessed apnea episode at home. In the emergency department (ED), young age and presentation with apnea have been identified as risk factors for apnea. Most patients with apnea and RSV infection are younger than 2 months of age and have been ill for less than 5 days.

Arms JL, Ortega H, Reid S: Chronological and clinical characteristics of apnea associated with respiratory syncytial virus infection: A retrospective case series. Clin Pediatr 2008;47(9):953-958.

Ralston S, Hill V: Incidence of apnea in infants hospitalized with respiratory syncytial virus bronchiolitis: A systematic review. J Pediatr 2009;155(5):728-733.

Willwerth BM, Harper MB, Greenes DS: Identifying hospitalized infants who have bronchiolitis and are at high risk for apnea. Ann Emerg Med 2006;48(4):441-447.

20. What is the relationship between GER and an ALTE?

GER is common in infants. Classic teaching suggested that GER is a common cause of ALTE. However, the temporal nature of the relationship has come into question. Studies have shown that apnea precedes the reflux in most cases. The benefit of treating patients found to have GER after an ALTE is unclear.

Arad-Cohen N, Cohen A, Tirosh E: The relationship between gastroesophageal reflux and apnea in infants. J Pediatr 2000;137(3):321-326.

Mousa H, Woodley FW, Metheney M, Hayes J: Testing the association between gastroesophageal reflux and apnea in infants. J Pediatr Gastroenterol Nutr 2005;41(2):169-177.

21. When should an infant who has had an ALTE be admitted to the hospital?

The criteria for admission of a child who had an ALTE are subject to much interpretation. In cases in which you are convinced that a significant event did not occur, discharge may be reasonable after a period of observation in the ED. If a significant event did occur, hospital admission is advised. Some patients are considered low risk and can potentially be discharged from the ED, but the data for these patients are somewhat limited. Predictors of significant intervention during hospitalization include prematurity, an abnormal finding on the physical examination, color change to cyanosis, absence of symptoms of upper respiratory tract infection, and absence of choking. Patients without these risk factors could be considered for outpatient management.

Fu LY, Moon RY: ALTE: An update. Pediatr Rev 2012;33(8):361-369.

Mittal MK, Sun G, Baren JM: A clinical decision rule to identify infants with apparent life-threatening event who can be safely discharged from the emergency department. Pediatr Emerg Care 2012;28 (7):599-605.

Key Points: Approach to the Infant with an Apparent Life-Threatening Event

1. Perform a cardiopulmonary assessment.
2. Determine the clinical significance of the episode.
3. Obtain a detailed history from observers and emergency medical services personnel.
4. The history and physical examination should guide any diagnostic testing.
5. A period of observation in the ED or admission to the hospital is necessary.

22. Should a home cardiorespiratory monitor be recommended for patients after an ALTE?

Not usually. Home cardiorespiratory monitoring has never been proved to prevent SIDS, nor has the practice of home monitoring changed the incidence of SIDS. Apnea was previously believed to be related to SIDS, and the theory was that monitoring for apnea could prevent SIDS. Neither apnea or ALTEs have been shown to cause SIDS. The Committee on Fetus and Newborn from the American Academy of Pediatrics recommends that monitoring should not be prescribed to prevent SIDS.

American Academy of Pediatrics: Policy statement. Apnea, sudden infant death syndrome and home monitoring. Committee on Fetus and Newborn. Pediatrics 2003;111:914-917. Available at http://aappolicy.aappublications.org/cgi/content/full/pediatrics;111/4/914.

Moon RY: American Academy of Pediatrics Policy Statement. SIDS and Other Sleep-Related Infant Deaths: Expansion of Recommendations for a Safe Infant Sleeping Environment. Task Force on Sudden Infant Death Syndrome. Pediatrics 2011;128(5):e1341-e1367.

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CHEST PAIN

Steven M. Selbst

CHAPTER 8

1. How common is chest pain in children?

Chest pain is a common pediatric complaint. It is not nearly as frequent as abdominal pain or headache, but it is perhaps the third leading pain syndrome in children. Chest pain has been reported to occur in 0.3% to 0.6% of visits to a pediatric emergency department (ED).

Massin MM, Bourguignont A, Coremans C, et al: Chest pain in pediatric patients presenting to an emergency department or to a cardiac clinic. Clin Pediatr 2004;43:231-238.

Selbst SM, Ruddy R, Clark BJ, et al: Pediatric chest pain—A prospective study. Pediatrics 1988;82:319-323.

2. What is the peak age for pediatric chest pain?

Most studies report the mean age of children with chest pain to be 12 to 14 years. It affects children of all ages, and half of children with chest pain are younger than 12 years of age.

3. How does the cause of chest pain in children differ from that in adults?

Children with chest pain are far less likely to have a cardiac cause for their pain. Most children with chest pain have a self-limited, "benign" cause.

Drossner DM, Hirsh DA, Sturm JJ, et al: Cardiac disease in pediatric patients presenting to a pediatric ED with chest pain. Am J Emerg Med 2011;29:632-638.

4. Which diagnoses are most common in children who present to an ED with chest pain?

In many studies, up to 45% of cases of chest pain in children are labeled "idiopathic." That is, after a careful history and physical examination, the cause is still uncertain. When a diagnosis can be found, musculoskeletal injury is most common. Active children frequently strain chest wall muscles while carrying heavy books, exercising, or engaging in rough play. Many other children suffer chest pain from a direct blow to the chest that results in a mild contusion or, in rare cases, a rib fracture. Costochondritis accounts for about 10% to 20% of cases of chest pain. This musculoskeletal disorder produces tenderness over the costochondral junctions and is often bilateral. It is exaggerated by physical activity or breathing. Musculoskeletal pain is often reproducible by palpation of the chest wall or moving the arms and chest through a variety of positions. Table 8-1 lists the most common causes for pediatric chest pain.

Gokhale J, Selbst SM: Chest pain and chest wall deformity. Pediatr Clin North Am 2009;56(1):49-65.

Selbst SM: Approach to the child with chest pain. Pediatr Clin North Am 2010;57(6):1221-1234.

5. Is cardiac disease more likely if a child with chest pain is evaluated in a cardiology clinic, rather than the ED?

No. Cardiac disease as a cause of chest pain is still rare. In one study, it accounted for 1% of cases referred to a pediatric cardiology clinic. It is possible that some serious conditions were screened out in the ED and did not get referred to the cardiology clinic in that study.

Danduran MJ, Sheridan DC, Frommett PC: Chest pain characteristics of children/adolescents. Pediatr Cardiol 2008;29:775-781.

Friedman KG, Kane DA, Rathod RH, et al: Management of pediatric chest pain using a standardized assessment and management plan. Pediatrics 2011;128:239-245.

Saleeb SF, Li WY, Warren SZ, et al: Effectiveness of screening for life threatening chest pain in children. Pediatrics 2011;128:e1062-e1068.

6. How is the cause of chest pain related to the child's age?

Young children are more likely to have chest pain related to a cardiorespiratory condition (cough, asthma, pneumonia, or heart disease). Children over the age of 12 years are more likely to have a psychogenic disturbance as the cause of their pain.

Table 8-1. Most Common Causes of Pediatric Chest Pain

Idiopathic Causes
Musculoskeletal conditions
Chest wall strain
Costochondritis
Direct trauma
Respiratory Conditions
Asthma
Cough
Pneumonia
Gastrointestinal Problems
Esophagitis
Esophageal foreign body
Psychogenic Causes (Stress Related)
Cardiac Disease
Myocarditis

7. What common gastrointestinal condition causes chest pain?

Gastroesophageal reflux, which is very common in children and accounts for at least 7% of instances of pediatric chest pain. Some feel it is underdiagnosed. The pain is usually worse in the recumbent position. History may reveal that the pain is "burning" in quality and may have developed after eating spicy foods. A trial of antacids is often diagnostic and therapeutic.

Key Points: Most Common Causes of Childhood Chest Pain

1. Musculoskeletal pain
2. Idiopathic cause
3. Pulmonary conditions
4. Psychological cause
5. Trauma
6. Gastrointestinal problems
7. Cardiac disease
8. Sickle cell disease

8. What is precordial catch syndrome?

In 1955, Miller and Texidor described a syndrome of left-sided chest pain that is brief (less than a 5-minute duration) and sporadic. This pain may recur frequently for a few hours in some individuals and then remain absent for several months. The pain seems to be associated with a slouched posture or bending and is not related to exercise. It is usually relieved when the individual takes a few shallow breaths, or sometimes one deep breath, and assumes a straightened posture. It is believed that the pain arises from the parietal pleura or from pressure on an intercostal nerve, but the cause remains unclear. Some refer to this pain syndrome as "Texidor's twinge" or a "stitch in the side."

Gumbiner CH: Precordial catch syndrome. South Med J 2003;96:38-41.

9. What is "slipping rib syndrome"?

This is a rare sprain disorder caused by trauma to the costal cartilage of the eighth, ninth, and tenth ribs, which do not attach to the sternum. Children with slipping rib syndrome report pain under the ribs or in the upper abdominal quadrants. They also hear a clicking or popping sound when they lift objects, flex the trunk, or even walk. It is believed that the pain is caused by one of the ribs hooking under the rib above and irritating the intercostal nerves. The pain can be duplicated and the syndrome confirmed by performing the "hooking maneuver," whereby the affected rib margin is grasped and then pulled anteriorly. Intercostal block has been

tried for pain relief. Surgery to resect the involved costal cartilage may provide long-term relief, though most patients are treated satisfactorily with oral analgesics.

Mooney DP, Shorter NA: Slipping rib syndrome in childhood. J Pediatr Surg 2002; 132:1081-1082.

10. How can ingestion of tetracycline lead to chest pain?

Tetracycline, doxycycline, and other pill medications can cause acute esophagitis (pill-induced esophagitis). The pain is especially likely if the patient takes the medication with a minimal amount of water and then lies down. A history of esophageal dysmotility or stricture makes the pain more likely. However, many healthy teenagers also report this pain. Because of the pH of the drug, doxycycline produces an acidic solution or gel as it dissolves, and thus it is caustic when it remains in the esophagus.

11. How can I diagnose pill-induced esophagitis?

The diagnosis is made by taking a careful history. Physical examination generally is unremarkable. These medications are often taken by adolescents for treatment of acne, and because they are used long-term, teens may fail to reveal that they take the medication unless asked specifically. Some physicians prefer to perform an endoscopic evaluation to document esophageal ulcers (midesophageal ulcers are most common, as the tablets are most likely to lodge in that region). Others omit endoscopy and, instead, discontinue use of the tetracycline medications and treat with sucralfate. This approach can be both diagnostic and therapeutic, if the patient responds well.

Palmer KM, Selbst SM, Shaffer S, et al: Pediatric chest pain induced by tetracycline ingestion. Pediatr Emerg Care 1999;15:200-201.

12. When should a pneumothorax be suspected in a child with chest pain?

Suspect a pneumothorax if a child develops acute onset of sharp chest pain associated with some degree of respiratory distress. The pain is usually worsened by inspiration and may radiate to the shoulder, neck, or even the abdomen. Children with this condition do not have long-standing pain, and almost all present for care within 48 hours of developing the pneumothorax. The patient will usually have dyspnea, tachycardia, and, perhaps, decreased breath sounds on the affected side, or even cyanosis. However, these signs and symptoms depend on the size of the pneumothorax and whether it is under tension (Fig. 8-1). A small pneumothorax may produce minimal findings on examination.

History of trauma may increase your suspicion of pneumothorax, but many cases occur spontaneously or with exercise or cough. In those cases, a small, unrecognized subpleural bleb ruptures, leading to the air leak. Some underlying conditions increase the risk of "spontaneous" pneumothorax. Patients with asthma, cystic fibrosis, and Marfan's syndrome are particularly prone to chest pain secondary to pneumothorax. Also, several cases have been reported in teenagers who smoke crack cocaine.

Gokhale J, Selbst SM: Chest pain and chest wall deformity. Pediatr Clin North Am 2009;56(1):49-65.
Rafailov AS, Chao JH: Spontaneous pneumomediastinum. Pediatr Emerg Care 2010;26:588-591.

13. How can anxiety or emotional stress lead to chest pain in children?

The relationship of pain to emotional stress is not quite clear. However, we assume children get headaches and abdominal pain from stress. It is reasonable to conclude that chest pain can also be related to stress. Studies have shown that stress (psychogenic pain) is the cause in about 10% of children with chest pain who present to a pediatric ED. Possible stressors include school failure, recent death or illness in the family, recent loss of friends from moving to a new city or school, and school phobia. It is important to consider stress as a cause of chest pain in all children who present with the complaint. This should not be a diagnosis of exclusion, but if significant stress is temporally related to the pain, it is a reasonable diagnosis.

14. Why do some adolescents who present with hyperventilation also have chest pain?

Hyperventilation may be associated with psychogenic chest pain and may lead to pain by producing a hypocapnic alkalosis. Prolonged hyperventilation can lead to coronary artery vasoconstriction and chest pain. Also, stomach distention due to concomitant aerophagia can occur with hyperventilation and produce chest pain. Lightheadedness, headache, and paresthesias may also be found.

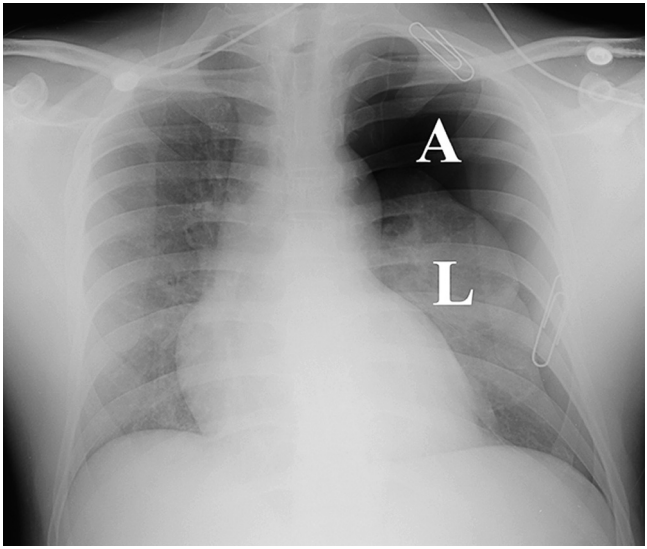

Figure 8-1. Tension pneumothorax on a frontal chest radiograph. Note the thin, sharply defined visceral pleural "white" line between radiolucent lung (*L*) and radiolucent "black" free air (*A*) in the peripheral pleural space in addition to rightward mediastinal shift and inferior left hemidiaphragmatic shift due to air under tension. *(Reproduced from Torigian DA, Miller WT Jr: Pleural diseases. In Pretorius ES, Solomon JA [eds]: Radiology Secrets, 2nd ed. Philadelphia, Mosby, 2006, Fig. 66-3, p 541.)*

Sawni A, Wright K, Ragothaman R: Hyperventilation and chest pain in an adolescent female: Index of suspicion. Clin Pediatr 2004;43:663-666.

15. Which children with chest pain deserve an evaluation with an electrocardiogram (ECG) and chest radiograph?

1. Those with worrisome historical features:
   - Acute onset of pain
   - Chest pain associated with exercise
   - Associated syncope, dizziness, palpitations
   - History of heart disease
   - History of a condition that can affect the heart or lungs—diabetes mellitus, Kawasaki disease, Marfan's syndrome, asthma, anemia, systemic lupus erythematosus
   - History of sickle cell disease
   - Use of cocaine
   - Trauma
   - Foreign-body ingestion
   - Fever
2. Those with an abnormal physical examination:
   - Respiratory distress
   - Decreased or abnormal breath sounds
   - Cardiac findings (murmur, rub, click, arrhythmia)
   - Fever
   - Trauma
   - Palpation of subcutaneous air

16. Which children with chest pain do *not* need extensive evaluation with laboratory studies in the ED?

Those with chronic pain (and none of the worrisome features mentioned previously) do not necessarily need laboratory studies. Management must be individualized, but most patients

can be managed with analgesics, reassurance that cardiac disease is unlikely, and close follow-up. If the history and physical examination do not suggest a cause for the pain, it is unlikely that laboratory tests will be helpful.

17. Why is chest pain of acute onset more worrisome?

Children with sudden onset of pain (within 48 hours of presentation) are more likely to have an organic cause for the pain. It is not necessarily a serious cause, but pneumonia, asthma, trauma, and pneumothorax are more likely.

Selbst SM, Ruddy R, Clark BJ, et al: Pediatric chest pain—A prospective study. Pediatrics 1988;82:319-323.

18. Why is fever associated with chest pain of concern?

Fever is much more likely to be associated with pneumonia, myocarditis, or pericarditis. Endocarditis is a very rare condition in otherwise healthy adolescents, but it should also be considered.

Key Points: Fever and Chest Pain

1. Consider pneumonia, myocarditis, and pericarditis.
2. Listen for decreased breath sounds (pneumonia).
3. Listen for distant heart sounds (pericarditis, myocarditis).
4. Obtain a chest radiograph.
5. Consider an electrocardiogram if pneumonia is excluded.

19. Which cardiac conditions can cause chest pain in children?

- **Arrhythmia:** Supraventricular tachycardia (SVT), ventricular tachycardia
- **Infection:** Myocarditis, pericarditis
- **Structural abnormalities:** Hypertrophic cardiomyopathy, aortic valve stenosis, anomalous coronary arteries
- **Coronary artery disease (CAD; ischemia or infarction):** Kawasaki disease, long-standing diabetes mellitus, familial hypercholesterolemia, or lipidemia

Drossner DM, Hirsh DA, Sturm JJ, et al: Cardiac disease in pediatric patients presenting to a pediatric ED with chest pain. Am J Emerg Med 2011;29:632-638.

20. How common is cardiac chest pain in children?

Pediatric chest pain is rarely due to cardiac disease. Studies have found that only about 0.6% to 4% of children who present to a pediatric ED with chest pain have a cardiac cause for their pain, and some of these children had known heart disease at the time of presentation. In the Drossner study, arrhythmia was the most common cardiac cause for cardiac-related chest pain, followed by infections (pericarditis, myocarditis).

Drossner DM, Hirsh DA, Sturm JJ, et al: Cardiac disease in pediatric patients presenting to a pediatric ED with chest pain. Am J Emerg Med 2011;29:632-638.

Selbst SM, Ruddy R, Clark BJ, et al: Pediatric chest pain—A prospective study. Pediatrics 1988;82:319-323.

21. Which arrhythmias can possibly cause chest pain in children?

Consider the possibility of an arrhythmia such as SVT as a cause of chest pain in an older child who reports having palpitations. Ventricular tachycardia and premature ventricular contractions are rare in children but may also cause sharp chest pain and an irregular cardiac rhythm. They may be found in children taking various medications or drugs such as cocaine.

22. When should I be concerned about a cardiac infection as the cause of chest pain?

Children with infections such as myocarditis or pericarditis can present with chest pain and usually have fever. Those with pericarditis may report a sharp, stabbing midsternal pain that is somewhat relieved when the patient sits up and leans forward. Distant heart sounds, neck vein distention, and a friction rub may be found. Viral myocarditis is more common and usually presents with low-grade fever and dull substernal chest pain. There is often respiratory distress as the infection progresses, and there may be muffled heart sounds or a gallop rhythm heard. Tachypnea is common, and tachycardia out of proportion to the degree of fever is

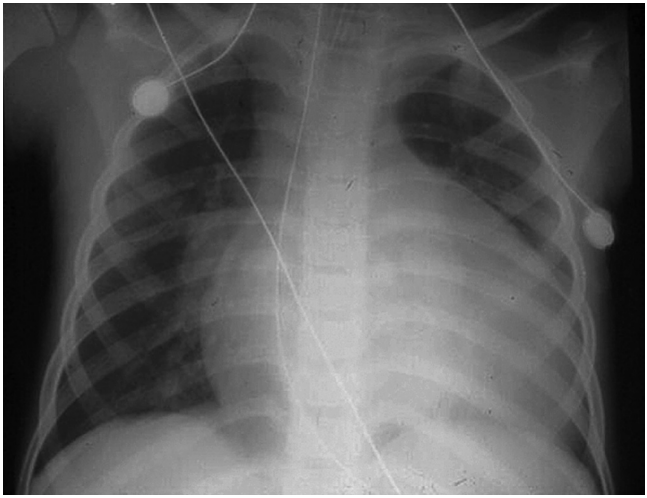

Figure 8-2. Radiograph of large heart from myocarditis.

characteristic. Tachycardia and hypotension may be worse with standing, and this may not resolve after fluid resuscitation. A chest radiograph often reveals cardiomegaly (Fig. 8-2).

Durani Y, Egan M, Baffa G, et al: Pediatric myocarditis: Presenting clinical characteristics. Am J Emerg Med 2009;27:942-947.

Freedman SB, Haladyn JK, Floh A, et al: Pediatric myocarditis: Emergency department clinical findings and diagnostic evaluation. Pediatrics 2007;120:1278-1285.

23. Which cardiac conditions can lead to ischemia and chest pain?

Some rare conditions may lead to ischemic myocardial dysfunction in children. For instance, hypertrophic cardiomyopathy can cause pain, especially with exercise. Aortic valve stenosis can also cause ischemia and chest pain, and those who had cardiac surgery for repair of transposition of the great vessels are at risk for subsequent myocardial infarction. Finally, those with underlying problems of the coronary arteries may develop chest pain. This is not common in children, but some children with an anomalous left coronary artery, thrombophilia, familial hypercholesterolemia, and long-standing diabetes mellitus may have CAD, and children who had Kawasaki disease in the past may have persistent coronary artery aneurysms that can produce symptoms long after the initial illness.

Drossner DM, Hirsh DA, Sturm JJ, et al: Cardiac disease in pediatric patients presenting to a pediatric ED with chest pain. Am J Emerg Med 2011;29:632-638.

Lane JR, Ben-Scachar G: Myocardial infarction in healthy adolescents. Pediatrics 2007;120:e938-e943.

Madhok AB, Boxer R, Green S: An adolescent with chest pain—Sequela of Kawasaki disease. Pediatr Emerg Care 2004;20:765-768.

Mahle WT, Campbell RM, Favaloro-Sabatier J: Myocardial infarction in adolescents. J Pediatr 2007;151:150-154.

Key Points: Cardiac Causes of Chest Pain

1. CAD: arteritis from Kawasaki disease, long-standing diabetes mellitus
2. Arrhythmias: SVT, premature ventricular contractions, ventricular tachycardia
3. Structural lesions: hypertrophic cardiomyopathy, aortic valve stenosis
4. Infections: myocarditis, pericarditis, endocarditis

24. What are the concerns in children with Marfan's syndrome who report chest pain?

These children may report chest pain due to rupture of an aortic aneurysm, which can be fatal. They are also at risk for spontaneous pneumothorax.

25. Why is chest pain associated with exercise of concern?

Young children with sudden death often had preceding chest pain with exercise. Pain related to serious conditions such as myocarditis, CAD, and hypertrophic cardiomyopathy is worsened by exertion. Such patients should be referred to a cardiologist for further testing, including echocardiography, Holter monitoring, or possibly exercise stress tests. Exercise-induced asthma is another condition in which chest pain is precipitated or worsened by exercise.

Danduran MJ, Sheridan DC, Frommett PC: Chest pain characteristics of children/adolescents. Pediatr Cardiol 2008;29:775-781.

26. What is Tietze's syndrome?

This is a rare condition of unknown etiology that causes sternal chest pain. Physical examination may reveal tender, spindle-shaped swelling at the sternochondral junctions. The condition can last for months.

27. Why do children with asthma report chest pain?

Some possible causes include anxiety and overuse of chest wall muscles due to respiratory distress. Some have an associated pneumonia that could lead to diaphragmatic irritation. Others may develop a pneumothorax or pneumomediastinum.

28. What cause should be considered in an afebrile, previously well toddler with no injury who reports sudden midsternal chest pain?

Foreign body in the esophagus! Many young children ingest coins, and most are asymptomatic. However, if the coin lodges in the esophagus (especially in the upper esophagus), the child may report sudden chest pain. Dysphagia may also be present. A chest radiograph or a metal detector usually confirms the diagnosis.

29. When would you suspect hypertrophic cardiomyopathy in a child with chest pain?

There may be a positive family history for this condition. It is generally inherited as an autosomal dominant disorder. A murmur may be heard best when the child is standing or performing a Valsalva maneuver. These positions and exercise may exaggerate the chest pain. Squatting or lying supine minimizes the obstruction and reduces the murmur. Shortness of breath is the most common symptom. This condition is concerning because it is among the leading causes of sudden death among young athletes.

Maron BJ, Doerer JJ, Haas TS, et al: Sudden deaths in young competitive athletes; analysis of 1866 deaths in the US. Circulation 2009;119:1085-1092.

30. Does mitral valve prolapse (MVP) cause chest pain in children?

This is uncertain. Some postulate that MVP causes papillary muscle or left ventricular endocardial ischemia. However, in many cases, chest pain can be attributed to other causes, even in children with MVP. MVP is no more common in children with chest pain than in the general population. About 6% to 8% of children in the United States are believed to have MVP.

31. Name a cutaneous condition that is associated with chest pain.

Herpes zoster infection (Fig. 8-3). Shingles is often associated with distressing chest pain when the lesions involve a chest wall dermatome. The chest pain can sometimes precede the vesicular rash by several days.

32. What is the "devil's grip"?

This unusual condition, also known as *pleurodynia*, is characterized by paroxysms of sharp pain in the abdomen or thorax. It is caused by coxsackievirus and may occur in mini-epidemics.

33. What are possible causes of chest pain in a teenage girl with systemic lupus erythematosus?

These patients are at risk for pericarditis, myocarditis, endocarditis, pneumonia, pleural effusion, and myocardial ischemia if they have renal disease and hypertension.

34. Should pulmonary embolism be considered as a cause of chest pain in a child?

Pulmonary embolism is a rare problem in children. Consider it in a patient with minor trauma to a lower extremity (usually a male) or any patient with increased likelihood of clotting or a

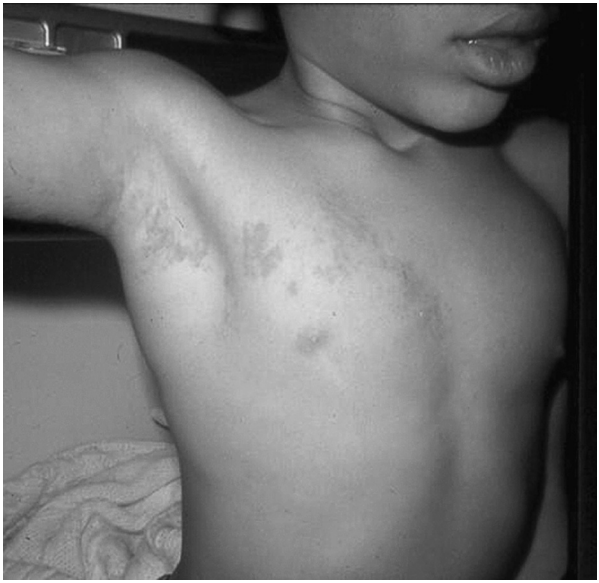

Figure 8-3. Herpes zoster infection.

hypercoagulable state (e.g., nephrotic syndrome). It is very rarely reported in teenage girls who are obese (BMI > 25), have deep vein thrombosis or systemic lupus erythematosis, or use oral contraceptives and in those who have recently had elective abortions. A patient with a pulmonary embolism usually has accompanying shortness of breath, pleuritic chest pain, fever, cough, and hemoptysis. The finding of a swollen, tender lower extremity on physical examination increases concern about venous thrombosis and a pulmonary embolism.

Agha BS, Sturm JJ, Simon HK, et al: Pulmonary embolism in the pediatric emergency department. Pediatrics 2013;132:663-667.

Rajpurkar M, Warrier I, Chitiur M, et al: Pulmonary embolism—Experience at a single children's hospital. Thromb Res 2007;119:699-703.

35. In children with chest pain and sickle cell disease, what are the likely causes?

Vaso-occlusive crisis may be the cause of pain. However, consider acute chest syndrome in these patients. Pneumonia due to an encapsulated organism is also of concern. Finally, ischemia due to a heart weakened by chronic anemia is possible. All such patients deserve a chest radiograph and perhaps an electrocardiogram as part of their evaluation.

36. Chest pain associated with a "crunching" sound on chest examination is found with which condition?

Pneumomediastinum. Free mediastinal air and subcutaneous emphysema are complications of asthma exacerbation. It may also occur as a result of trauma, or spontaneously. Some children with this finding have associated dyspnea and cough. Crepitation is often noted in the suprasternal notch and may extend to the neck, axilla, and face. A chest radiograph confirms the diagnosis. The condition usually has a good outcome and resolves within a few days. Hospital admission and treatment of the underlying cause (i.e., asthma) are recommended.

Bullaro FM, Bartoletti SC: Spontaneous pneumomediastinum in children—A literature review. Pediatr Emerg Care 2007;23:28-30.

Rafailov AS, Chao JH: Spontaneous pneumomediastinum. Pediatr Emerg Care 2010;26:588-591.

37. Can chest pain in teenagers be caused by toxins?

Yes. Adolescents frequently use illicit drugs, and cocaine can cause chest pain and serious disease. Numerous cases of cocaine-related myocardial infarction (MI) have been reported in

young patients. Patients who abuse cocaine may complain of "chest tightness," "palpitations," and "pressure," and they may have associated nausea, vomiting, and anxiety. In 2005, there were almost one half million visits to the ED (all ages) related to cocaine, and most of these patients had chest pain. Physical examination reveals tachycardia, tachypnea, and hypertension. Cocaine can cause coronary artery vasospasm. The incidence of MI with cocaine abuse is as high as 6% in adults, but the incidence in adolescents is unknown. Some adolescents who snort cocaine may develop a pneumothorax or pneumomediastinum.

Methamphetamines can also cause chest pain. Consider screening for illicit drugs in all teens with chest pain. An ECG may show ischemia or arrhythmia. Serum cardiac enzymes (troponins) may be elevated, indicating evidence of an MI.

McCord J, Jneid H, Hollander J, et al: Management of cocaine associated chest pain and myocardial infarction. A scientific statement from the American Heart Association Acute Cardiac Care Committee of the Council on Clinical Cardiology. Circulation 2008;117:1897-1907.

Selbst SM, Palermo R, Durani Y, et al: Adolescent chest pain—Is it the heart? Clin Pediatr Emerg Med 2011;12:289-300.

38. When should referral to a cardiologist be considered for a child with chest pain?

Chest pain associated with exercise (during or immediately after) requires an evaluation by a cardiologist. If the pediatric patient has pain associated with syncope, dizziness, or palpitations, evaluation by a cardiologist is also reasonable. Furthermore, if the child has a history of underlying heart disease or cardiac arrhythmia or has a previous condition that subjects him to heart disease (i.e., long-standing diabetes mellitus, Kawasaki disease), referral to a cardiologist seems prudent. It is also recommended to have a cardiologist evaluate the child if there is a history of premature sudden death in the family or if the child has hypertension or features of Marfan's syndrome.

Saleeb SF, Li WY, Warren SZ, et al: Effectiveness of screening for life threatening chest pain in children. Pediatrics 2011;128:e1062-e1068.

Selbst SM: Approach to the child with chest pain. Pediatr Clin North Am 2010;57(6):1221-1234.

CHAPTER 9

COUGH

Christopher King

1. Describe the cough reflex.

Stimuli that induce cough can be mechanical (e.g., foreign body, dust), chemical (e.g., capsaicin, acetic acid), or inflammatory mediators (e.g., histamine, bradykinin, prostaglandin E_2). Such stimuli interact with cough receptors in the upper and lower airways that cause production of mediators, such as tachykinins and neurokinin A. Stimulation of the cough receptors is transmitted via the vagus nerve to the "cough center" of the brainstem (probably located in the pons). The efferent limb of the cough pathway is poorly understood but is known to include the spinal cord from C3 to S2, spinal nerves, and the recurrent laryngeal nerve (for glottic closure). Activation of the efferent pathway causes constriction of the expiratory muscles, which produces increased airway pressure against the closed glottis. The airway also narrows slightly. When the glottis is suddenly opened, air is expelled at a high velocity, clearing the airway of secretions and foreign material.

2. How is a cough suppressed?

As with respiration, cough is largely involuntary but can be suppressed voluntarily to a certain extent (cortical modulation). Opioids that suppress cough (e.g., morphine) are believed to produce this effect by acting centrally on the cough center. Inhalation of nebulized lidocaine can suppress cough by affecting cough receptors in the airway.

3. Is the cough reflex mediated by the same pathway that produces bronchospasm?

There has been a widely held belief that airway hypersensitivity (asthma/bronchospasm) and cough are manifestations of the same physiologic mechanism. By this thinking, persistent coughing, like wheezing, is a common presentation of asthma. This view has led, in part, to the practice of diagnosing children with persistent coughing as being asthmatic ("cough-variant" asthma), even when these patients have no other evidence of bronchospasm (dyspnea, exercise intolerance, abnormal spirometry findings). However, evidence suggests that although airway hypersensitivity and cough receptor sensitivity can be *triggered* by the same stimuli, this probably occurs via two *different* pathways. For most children, it appears that persistent coughing results solely from increased sensitivity of cough receptors in the upper and lower airways and does not represent true asthma/bronchospasm (see also Questions 7 and 8).

Chang AB: Cough, cough receptors, and asthma in children. Pediatr Pulmonol 1999;28:59-70.

4. How much coughing is normal?

Previously, one of the primary obstacles in studying cough in children was that cough frequency was difficult to measure precisely, as reproducibility of caretaker questionnaires was relatively poor. The use of "cough meters" that children being studied would wear continuously over a given period of observation provided a more accurate count of coughing episodes. Interestingly, when cough meters were used in healthy children with no symptoms of respiratory illness, the range of cough episodes over 24 hours was 1 to 34 in one study and 0 to 141 in another study. Clearly, the frequency of coughing episodes varies widely even among children without respiratory problems. This occurrence of cough in healthy children has been referred to as "expected cough," and the normal variability within the population should be kept in mind when evaluating a pediatric patient with cough.

Chang AB, Phelan PD, Robertson CF, et al: Frequency and perception of cough severity. J Paediatr Child Health 2001;37:142-145.

Munyard P, Bush A: How much coughing is normal? Arch Dis Child 1996;74:531-534.

5. What is the most common cause of acute cough?

Cough in children has been characterized based on duration as acute (<2 weeks), protracted acute (2 to 4 weeks), and chronic (>4 weeks). Although the differential diagnosis of acute

Table 9-1. Differential Diagnosis of Persistent Cough by Age

Infancy (<1 year of age)
Infection: viral, bacterial, chlamydial, pertussis
Anatomic abnormalities: tracheomalacia, vascular rings
Cystic fibrosis
Bronchopulmonary dysplasia (premature infants)
Preschool (1-5 years of age)
Asthma
Infection: viral, bacterial, chlamydial, pertussis
Foreign-body aspiration
Cystic fibrosis
School Age (>5-18 years of age)
Asthma
Infection: viral, bacterial, chlamydial, pertussis
Smoking
Psychogenic causes (habit cough)

cough in children is relatively broad (Table 9-1), the most common cause is probably postviral or inflammatory cough, which has been variably called *nonspecific cough*, *isolated cough*, or *cough illness*. Children who develop a viral upper respiratory tract infection may continue to cough long after other viral symptoms (e.g., rhinitis, fever) have subsided. Patients often have a persistent dry cough (particularly at night) but do not have wheezing, chest tightness, dyspnea on exertion, or other symptoms of bronchospasm. Worry about the duration of cough after several days commonly prompts caretakers to seek medical attention. When symptoms of a preceding upper respiratory illness are elicited in a child with a persistent dry cough and no other associated symptoms (chest pain, rhinitis, recurrent fevers), reassurance that the coughing will most likely subside spontaneously is generally appropriate. However, for children who continue to cough beyond 4 weeks, referral to a specialist may be warranted.

Hay AD, Wilson AD: The natural history of acute cough in children aged 0 to 4 years in primary care: A systematic review. Br J Gen Pract 2002;52:401-409.

Hay AD, Wilson A, Fahey T, et al: The duration of acute cough in pre-school children presenting to primary care: A prospective cohort study. Fam Pract 2003;20:696-705.

6. Is gastroesophageal reflux disease (GERD) known to commonly cause chronic cough in children?

No. In fact, the "big three" causes of chronic cough in adults—asthma, GERD, and postnasal drip—all appear to be relatively uncommon causes of chronic cough in pediatric patients. Specifically, there is a paucity of useful data relating to a cause-and-effect relationship between GERD and chronic cough in children. A few prior case studies purportedly showed a causal link between these two conditions, but prospective data have failed to bear this out, and currently there is no convincing evidence that GERD is a common cause of chronic cough in children.

Chang AB: Pediatric cough: Children are not miniature adults. Lung 2010;188(Suppl 1):S33-S40.

Rudolph CD: Supraesophageal complications of gastroesophageal reflux in children: Challenges in diagnosis and treatment. Am J Med 2003;115(Suppl 3A):150S-156S.

7. Is cough-variant asthma underdiagnosed?

No. In fact, the opposite is more likely the case. In the past, children with persistent cough accompanied by signs of true bronchospasm such as exercise intolerance, but without wheezing, often were not diagnosed with asthma. The syndrome of cough and acute exertional dyspnea was first identified in a series of adult patients in 1975, and the term *cough-variant asthma* was coined in 1979. Before that time, cough-variant asthma was certainly underdiagnosed. Now, however, the diagnosis of asthma in children with isolated cough has

been widely embraced. In fact, some have suggested that recent increases in the prevalence of asthma are largely due to expanding the diagnostic criteria to include children with isolated cough. This has occurred despite evidence that many of these patients do not respond to conventional asthma therapy (see Question 8). It would appear that the pendulum has swung too far, and we are currently overdiagnosing and overtreating asthma in patients with other causes of cough.

Benedictis FM, Selvaggio D, Benedictis D: Cough, wheezing and asthma in children: Lesson from the past. Pediatr Allergy Immunol 2004;15:386-393.

Lougheed MD, Turcotte SE, Fisher T: Cough variant asthma: Lessons learned from deep inspirations. Lung 2012;190:17-22.

Key Points: Etiology of Cough in Children

1. Most children with persistent cough have a postviral or inflammatory cough that does not respond to any standard therapies but resolves spontaneously over a period of weeks.
2. Cough-variant asthma is very unlikely to be the appropriate diagnosis for a child with a persistent cough who does not have other signs of asthma (e.g., exertional dyspnea) and does not respond to an appropriate course of treatment for asthma.
3. Passive cigarette or cigar smoke aggravates coughing in children.
4. Persistent cough in a young child after a severe choking episode may represent aspiration of a foreign body into a mainstem bronchus.
5. An infant with a "staccato" cough (repetitive, dry, "rapid-fire" with inspiration between each single cough) may have *Chlamydia trachomatis* pneumonia, especially when conjunctivitis is also present.
6. A child with violent coughing fits ("paroxysmal" cough) followed by an inspiratory "whoop" and post-tussive emesis and a normal chest x-ray appearance can be clinically diagnosed as having pertussis.

Key Points: Management of Cough in Children

1. For most children with isolated dry cough lasting less than 4 weeks who have no evidence of pneumonia, treatment with antibiotics is not indicated.
2. For children with a "wet cough" lasting longer than 10 days, a course of antibiotics (typically a macrolide) may be warranted.
3. Children with a cough lasting longer than 4 weeks may require referral to a specialist.
4. An appropriate course of a bronchodilator may be prescribed if cough-variant asthma is suspected, but if the child fails to respond, the treatment should be discontinued rather than escalated.

8. Do children with persistent cough respond to standard therapy for asthma?

Given the caveats described in the previous question, it is safe to say that children with true cough-variant asthma should respond to treatment with bronchodilators or steroids within hours to a few days. However, these patients represent a relatively small percentage of the children presenting to the emergency department (ED) with persistent cough. Most will have postviral cough, which does *not* respond to conventional asthma therapy. Therefore, unless a child with persistent cough has a history that suggests a true bronchospastic cause (severe cough and dyspnea occurring consistently with exertion), it is not generally wise to make a de novo diagnosis of cough-variant asthma and initiate bronchodilator or steroid treatment. Conversely, if a child has been receiving conventional asthma therapy for several days to weeks and coughing has not decreased, the patient is very unlikely to have cough-variant asthma and the asthma treatments can be stopped.

Thompson F, Masters IB, Chang AB: Persistent cough in children and the overuse of medications. J Paediatr Child Health 2002;38:578-581.

9. Does cough due to acute bronchitis respond to treatment with antibiotics?

Acute bronchitis seems to be a diagnosis in search of significance. Most clinicians use the term *acute bronchitis* when referring to a patient who has a *productive* cough without evidence of pneumonia. Yet if the diagnostic criteria are poorly defined in adults (how much sputum?

how often?), the situation with children is worse. The primary issue with children is that they rarely cough up sputum and instead simply swallow it. Some have used the term *wet* (or "moist") cough to identify a productive cough in children, and there is some evidence indicating that a chronic wet cough is more likely to be associated with a bacterial cause in pediatric patients. Two older studies with poor data quality found that children with "severe" cough lasting longer than 10 days improved more rapidly after a course of antibiotics and were less likely to develop subsequent pneumonia. Unfortunately, no subsequent randomized controlled trials have been performed to confirm these findings, and therefore definitive guidance on this issue is lacking. For children with persistent wet cough lasting longer than 10 days, a course of antibiotics may be warranted. However, for the more common presentation of a child with "acute bronchitis" who has an isolated dry cough lasting days to a few weeks, antibiotics are not indicated.

Bialy L, Domino FJ, Chang AB, et al: The Cochrane library and chronic cough in children: An umbrella review. Evidence-based child health. Cochrane Rev J 2006;1:736-742.

O'Brien KL, Dowell SF, Schwartz B, et al: Cough illness/bronchitis—Principles of judicious use of antimicrobial agents. Pediatrics 1998;101:178-181.

Wurzel DF, Marchant JM, Clark JE, et al: Wet cough in children: Infective and inflammatory characteristics in broncho-alveolar lavage fluid. Pediatr Pulmonol 2014;49(6):561-568. Epub Jun 20, 2013.

10. What is a staccato cough?

A "staccato" cough is the classic finding in an infant with *C. trachomatis* pneumonia. It is a repetitive, dry, "rapid-fire" cough with inspiration between each single cough. Typically presenting as an insidious, afebrile pneumonitis syndrome in infants between the ages of about 3 weeks and 3 months, *C. trachomatis* pneumonia causes progressively worsening respiratory symptoms with chest x-ray findings of bilateral pulmonary infiltrates and air-trapping. Diffuse crackles without wheezing are usually heard on chest auscultation. Conjunctivitis is present in about half of cases, and laboratory results may demonstrate a peripheral eosinophilia. Very young infants can have more severe disease with episodes of apnea, but overall prognosis of this condition in pediatric patients is good. In most cases, first-line antibiotic therapy for *C. trachomatis* pneumonia is oral erythromycin, but it should be remembered that this treatment has been associated with pyloric stenosis in infants younger than 4 to 6 weeks of age. Because the benefits of treatment outweigh the risks in this circumstance, caretakers should be apprised of the symptoms of pyloric stenosis and the need for reevaluation should these symptoms occur.

Darville T: Chlamydia trachomatis infections in neonates and young children. Semin Pediatr Infect Dis 2005;16:235-244.

Miller KE: Diagnosis and treatment of Chlamydia trachomatis infection. Am Fam Physician 2006;73:1411-1416.

11. What is a paroxysmal cough?

Pertussis (whooping cough) is characterized by violent paroxysms of coughing followed by an inspiratory "whoop" as well as post-tussive emesis. There are four classic stages of clinical pertussis in children—the *incubation stage*, which is asymptomatic and lasts 7 to 10 days; the *catarrhal stage*, which resembles a viral upper respiratory infection and lasts 1 to 2 weeks; the *paroxysmal stage*, during which the coughing paroxysms and whoop occur and which lasts 1 to 6 weeks; and the *convalescent stage*, a period of gradual recovery with possible recurrent exacerbations lasting 2 to 12 weeks. The protracted course of pertussis has led to it being called the "hundred day cough." Pertussis is highly contagious from the onset of the catarrhal stage though about 3 weeks into the paroxysmal stage. It is important to note that infants, the group at greatest risk for serious illness or even death, may not have paroxysmal cough (or any cough at all) and may instead present only with poor feeding, apnea, and bradycardia. Because of factors that include waning immunity from the vaccine (rarely lasting longer than 12 years) resulting in limited herd immunity, low immunization rates in some populations, and vulnerability of infants because of poor passive immunity from mothers, pertussis is the only vaccine-preventable disease with increasing incidence and mortality risk in the United States. Presumptive diagnosis is made based on clinical findings and can be confirmed with culture, polymerase chain reaction, and serologic tests. Early treatment (before the

catarrhal phase) with macrolide antibiotics may decrease symptoms, and treatment at any time within 21 days of the onset of cough will decrease transmission.

Frumkin K: Pertussis and persistent cough: Practical, clinical and epidemiologic issues. J Emerg Med 2013;44:889-895.

Wang K, Harnden A: Pertussis-induced cough. Pulm Pharmacol Ther 2011;24:304-307.

12. Do commonly used cough medications work?

In a word, no. Numerous studies have addressed this question, and the bulk of evidence indicates that the commonly used pediatric cough preparations are no more effective in suppressing cough than placebo. These medications include dextromethorphan, guaifenesin, and codeine. Furthermore, many of the pediatric cough preparations also contain potentially dangerous agents such as decongestants that, if used in excessive doses, can cause cardiac arrhythmias in children. In 2008, the Food and Drug Administration issued a statement that strongly condemned the use of over-the-counter cough and cold medications in children under 4 years of age, and manufacturers voluntarily complied by adding "do not use" labeling for this population. However, we continue to spend over $2 billion annually in the United States for pediatric cough and cold preparations that likely have no beneficial effect—another testament to the power of advertising and its influence on medicine.

Schroeder K, Fahey T: Over-the-counter medications for acute cough in children and adults in ambulatory settings. Cochrane Database Syst Rev 2004;(4):CD001831; update 2008;(1):CD001831.

Sharfstein JM, North M, Serwint JR: Over the counter but no longer under the radar—Pediatric cough and cold medications. N Engl J Med 2007;357:2321-2324.

13. What problem should come to mind if a toddler presents with a persistent, nagging cough after a severe choking spell?

This is the classic history for foreign-body aspiration. Caretakers of a young child who has ongoing episodes of moderate to severe coughing should be questioned about the occurrence of a choking spell. The child typically has a small toy or bead in his or her mouth and takes a breath, and the object enters the trachea, at which time a severe choking spell occurs. If the object is about the size of the trachea, and it cannot be expelled with coughing, the child will quickly develop signs of severe upper airway obstruction. Yet if the object is smaller than the diameter of one of the mainstem bronchi, it may lodge there so that the child recovers with only a persistent cough. A patient with normal lungs will have adequate oxygenation and ventilation despite even complete occlusion of one mainstem bronchus. Expiratory chest radiographs may show decreased "deflation" of the affected lung. If an adequate expiratory x-ray study cannot be obtained, bilateral decubitus radiographs of the chest may show the same finding. However, if the story is classic, it is wise to consult an otolaryngologist or pulmonologist (for possible bronchoscopy) even if the chest x-ray appearance is normal.

14. What is habitual cough?

Habitual cough (also called habit, psychogenic, or tic cough) is a type of conversion reaction, that is, a symptom that occurs typically in response to stressors with no underlying disease process. Other symptoms that are seen with conversion reactions include abdominal pain, headache, or difficulty breathing. Because such symptoms are often persistent, the patient may undergo a series of extensive yet unrevealing medical evaluations. Often, there are issues of secondary gain—attention-seeking, school avoidance, preventing caretakers from fighting—that may only be reinforced by repetitive medical tests. Fortunately, habitual cough is one of the more straightforward conversion reactions to manage in the ED, because unlike symptoms such as difficulty breathing or severe headache, cough in an otherwise well-appearing child with a normal examination is unlikely to be dangerous. Consequently, a patient with habitual cough can be safely discharged, after a specific plan for outpatient follow-up has been formulated, without significant concern that the child is being sent home with a potentially serious condition.

de Jongste JC, Shields MD: Chronic cough in children. Thorax 2003;58:998-1003.

Ramanuja S, Kelkar P: Habit cough. Ann Allergy Asthma Immunol 2008;102:91-95.

15. What are the characteristics of a patient with habitual cough?

One of the most striking aspects of this condition is often the disparity between the reaction of the caretakers and the reaction of the child—the caretakers are often extremely upset and

anxious, whereas the child appears unconcerned ("la belle indifference"). Caretakers are commonly "at the end of their rope" and are coming to the ED for a second or third opinion. The child may already have had one or more normal chest radiographs or blood test results obtained by other health care providers. A patient with habitual cough is typically older (school-aged or adolescent), has missed many days of school, and has a completely normal physical examination. Aspiration of a foreign body can generally be excluded because the patient is old enough to deny it. The cough itself will often sound like a "honk"—harsh, loud, and impossible to ignore.

Bush A: Paediatric problems of cough. Pulm Pharm Ther 2002;15:309-315.

16. How is habitual cough managed in the ED?

As with most other types of conversion reaction, habitual cough cannot always be diagnosed definitively in the ED. It is generally a diagnosis of exclusion made after other possible conditions (e.g., pneumonia, bronchospasm) have been ruled out, and may require additional outpatient testing by the primary pediatrician or specialist. If suspicion of habitual cough is high, it may be appropriate to have a preliminary discussion of this possibility with caretakers, although it is not uncommon for their reaction to be somewhat defensive ("Are you saying this is all in her head?"). Establishing rapport by demonstrating genuine concern for the overall well-being of the child while carefully avoiding any language that could be construed as pejorative is essential. When emotions are high, it may be better to leave a detailed discussion of this diagnosis to the primary pediatrician or specialist after potentially serious causes have been excluded in the ED.

17. What is one thing caretakers should always do if their child has a persistent cough?

They should stop smoking and forbid others from smoking in the house where the child resides. Cessation of smoking by caretakers has been shown to be a successful therapy for children with chronic cough.

18. What is the strangest cause of persistent cough?

Although this is certainly open to debate, perhaps the oddest cause of persistent cough is an eyelash stuck in the child's ear. Curiously, stimulation of the auricular branch of the vagus nerve by an eyelash in the external auditory canal can repetitively provoke the cough reflex. Unlike other otic foreign bodies, an eyelash does not cause pain or diminish hearing and may therefore go undetected for an extended time. Furthermore, it may not be immediately obvious on otoscopy. Consequently, it is wise to carefully examine the ears of a child with persistent coughing in hopes of finding this elusive yet easily remedied cause of a troublesome symptom.

CHAPTER 10

CRYING AND IRRITABILITY IN THE YOUNG CHILD

Robert G. Bolte

1. Why is an organized approach important to the evaluation of crying in an infant?

The cause of crying in the nonverbal and frequently uncooperative infant is often obscure. A well-organized approach is critical because the differential diagnosis is vast, ranging from a normal physiologic or temperamental response to a life-threatening medical or surgical emergency. Finding the right answer with a reasonable utilization of resources is a big part of the "art" of pediatrics (Table 10-1).

Bolte R: The crying child, Part 1: Potential causes. Contemp Pediatr 2007;24(5):75-81.
Bolte R: The crying child, Part 2: Evaluation and management. Contemp Pediatr 2007;24(6):90-95.

2. I work in an emergency department (ED) setting. Why do I have to know anything about colic?

Three reasons:

- It's a commonly seen problem, even in an ED setting, representing a significant cause of parental anxiety and frustration.
- Never forget that persistent crying is often a trigger for abusive injury.
- If you know what colic *is*, then you're less likely to misdiagnose more serious problems by assuming that the diagnosis is "only colic."

3. Describe the clinical features of the infant colic syndrome.

Cyclic discrete periods of intractable crying, usually on a daily basis, with onset at 1 to 4 weeks of age and spontaneous improvement by 3 to 4 months of age. The classic definition of infantile colic was first described by Wessel in 1954 as "crying lasting more than 3 hours per day, 3 days per week, and continuing more than 3 weeks in infants less than 3 months of age." The crying is not relieved by normal parental interventions, such as feeding, burping, changing diapers, providing a pacifier, or rocking. Vomiting, diarrhea, or poor weight gain are *not* features of the infant colic syndrome. If they are present, consider another diagnosis.

Barr RG: Changing our understanding of infant colic. Arch Pediatr Adolesc Med 2002;156:1172-1174.
Clifford TJ, Campbell MK, Speechley KN, Gorodzinsky F: Sequelae of infant colic: Evidence of transient infant distress and absence of lasting effects on maternal health. Arch Pediatr Adolesc Med 2002;156:1183-1188.

4. Is there a "normal" amount of inconsolable crying in a young infant?

Brazelton's data on how much time a *normal* infant spends crying *inconsolably* shows that the median daily amount of inconsolable crying at 2 weeks of age is 1 hour and 45 minutes. This *increases to 2 hours and 45 minutes at 6 weeks of age* and thankfully decreases to less than an hour by 12 weeks of age. Inconsolable crying usually occurs in the evening (between 3 PM and 11 PM) when most busy parents are frantically attempting to spend precious quality time with their new baby. Sharing this information with prospective parents may be the most effective argument for birth control that you can deliver.

Brazelton TB: Crying in infancy. Pediatrics 1962;29:579-588.

5. Are there any effective treatments for colic?

There's no magic remedy, but communicating empathy is crucial. Also, providing the family with accurate information on "normal" amounts of inconsolable crying and when they can expect improvement is always of value. Swaddling and decreasing environmental stimuli (light, noise) is reasonable advice for parents. A behavioral-modification approach described by

Table 10-1. Relatively Frequent Serious Conditions Associated with Intractable Crying That Must Be Excluded Prior to Emergency Department Discharge

CONDITION	CLINICAL CORRELATES
Meningitis/ encephalitis	Lethargy, vomiting, paradoxical irritability, fever
Sepsis	Lethargy, poor perfusion, fever, petechiae
Septic hip	Pain with range of motion/abnormal positioning of hip
Battered child syndrome	Bruising, bony tenderness, incompatible history
Shaken baby syndrome	Lethargy, full fontanel, retinal hemorrhages
Intussusception	Paroxysmal abdominal pain, lethargy, bloody stool, abdominal mass; rectal examination may be abnormal
Volvulus	Bilious vomiting, abdominal tenderness
Appendicitis	Abnormal examination of abdomen
Incarcerated inguinal hernia	Abnormal examination of inguinal region
Hemolytic uremic syndrome	Bloody diarrhea, hematuria/proteinuria, hemolytic anemia, thrombocytopenia, azotemia
Hypoxemia	Tachypnea, retractions, nasal flaring, wheezing, cyanosis
Hair encirclement	Abnormal examination of digits, genitalia, or uvula
Testicular torsion	Abnormal examination of scrotum
Supraventricular tachycardia	Nonvariable heart rate > 220 beats per minute
Infant botulism	Constipation, hypotonia, weak cry

Taubman has been shown to be helpful. Weizman and colleagues have reported that commercially available chamomile tea (not home-grown concoctions, which are potentially dangerous) may be useful. Simethicone (Mylicon; 0.3 mL given prior to each feeding) is basically a high-grade placebo. Simethicone is generally nontoxic, but avoid this medication if the infant is being treated with levothyroxine for hypothyroidism. Generally avoid recommending formula changes (likely effective only in a subset of infants requiring hydrolysate formula) or medications with potentially toxic side effects, such as paregoric, dicyclomine (Bentyl), or hyoscyamine (Levsin). There are several good sources for useful parent educational materials.

Patient information: Colic (the basics, beyond the basics). UpToDate, 2013. Available at www.uptodate.com.

Taubman B: Parental counseling compared with elimination of cow's milk or soy milk protein for the treatment of infant colic syndrome: A randomized trial. Pediatrics 1988;81: 756-761.

Weizman Z, Alkrinawi S, Goldfarb D, et al: Efficacy of herbal tea preparation in infantile colic. J Pediatr 1993;122:650-652.

6. What is a common medical problem in young infants that mimics colic?

Esophagitis, secondary to gastroesophageal reflux (GER). GER is a ubiquitous and generally benign condition in young infants ("the happy spitter"). Esophagitis is a common problem secondary to GER, often mimicking colic. The infant with esophagitis often presents to the ED during the first few months of life with intractable crying as the chief complaint.

There is usually a history of frequent nonforceful, *nonbilious* regurgitation following feedings. The infant with an irritated esophagus may take the first few sips of the feeding avidly but then turns away, crying in pain. Occasionally the infant with esophagitis exhibits a torticollis-like positioning of the neck (Sandifer syndrome).

Nelson SP, Chen EH, Syniar GM, et al: Prevalence of symptoms of gastroesophageal reflux during infancy: a pediatric practice-based survey. Pediatric Practice Research Group. Arch Pediatr Adolesc Med 1997;151:569-572.

Nelson SP, Chen EH, Syniar GM, et al: One-year follow-up of symptoms of gastroesophageal reflux during infancy. Pediatric Practice Research Group. Pediatrics 1998;102:e67.

7. What advice can you give parents in the ED to reduce crying from GER?

In the otherwise well-appearing infant with a normal growth rate, reasonable recommendations include upright positioning with feeding and thickening the formula. Outpatient treatment with medications can be considered (antacids, ranitidine, or omeprazole) with follow-up by the primary medical care provider. In infants with more serious associated problems, such as poor growth or significant chronic respiratory issues, consider pediatric gastroenterology referral.

Vandernplas Y, Rodolph CD, Di Lorenzo C, et al: Pediatric gastroesophageal reflux clinical practice guidelines: Joint recommendations of the North American Society for Pediatric Gastroenterology, Hepatology, and Nutrition and the European Society for Pediatric Gastroenterology, Hepatology, and Nutrition. J Pediatr Gastroenterol Nutr 2009;49:498-547.

8. A previously well 4-month-old presents with acute onset of intractable crying for the past 4 hours. The child is afebrile, and the general examination is otherwise normal. What diagnoses do you need to consider?

Abusive injury needs to be strongly considered in your differential diagnosis. Whenever crying is the chief complaint, a meticulous examination of the skin is mandatory and could ultimately be life-saving. A "secret" of examination is to always evaluate for trauma to the frenulum of the lips, which is associated with abusive injury.

Also consider intussusception, which is both life-threatening and relatively common.

Corneal abrasion is another possibility. In a prospective study by Poole, 21% of afebrile infants younger than 6 months of age presenting to an ED with a chief complaint of crying had corneal abrasions. In a more recent retrospective review by Freedman and associates of 237 afebrile infants less than 1 year of age presenting to a pediatric ED with a chief complaint of crying, they didn't find a single case of corneal abrasion. However, they only looked for it once. A reasonable approach may be to perform a corneal examination with fluorescein in the well-appearing infant with *acute* onset of *persistent* crying. A positive examination does not by itself exclude other potentially more serious diagnoses. However, if this *is* the correct diagnosis, all symptoms of crying and fussiness should be resolved with the administration of a topical anesthetic used during the examination.

Urinary tract infection (UTI) is also a significant consideration, even in the *afebrile* infant. The Freedman and associates study (which excluded febrile children) found an overall UTI rate of 3% in afebrile infants presenting with crying as the chief complaint. The UTI rate reached 10% in infants less than 4 weeks of age. A very important "secret" is to obtain a catheterized urine specimen in the infant with "atypical" colic or whose crying persists in the ED, even if there is no fever history. Also be aware that there is a high rate of false-negative urinalyses in children less than 1 year of age, so obtaining a culture is mandatory.

Freedman SB, Al-Harthy N, Thull-Freedman J: The crying infant: Diagnostic testing and frequency of serious underlying disease. Pediatrics 2009;123:841-848.

Poole SR: The infant with acute, unexplained, excessive crying. Pediatrics 1991;88: 450-455.

Rittichier KK, Roback MG, Bassett KE: Are signs and symptoms associated with persistent corneal abrasions in children? Arch Pediatr Adolesc Med 2000;154:370-374.

9. Why is it a very good idea to look closely at the fingers and toes of a baby presenting with acute onset of intractable crying?

You might find a hair tourniquet. The hair tourniquet occurs when a hair takes a somewhat mysterious but definitely twisted journey, ultimately causing constricting injury to an appendage. The vast majority of cases involve fingers or toes (Fig. 10-1). Rarely the penis, clitoris, or even the uvula may be involved. Although easily diagnosed by meticulous

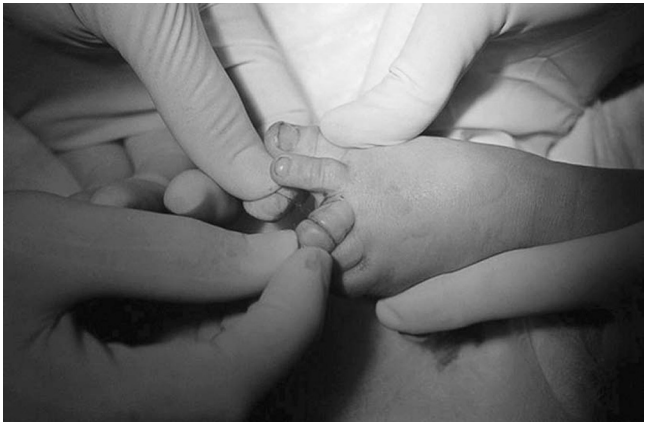

Figure 10-1. Hair tourniquet.

physical examination, treatment can be problematic. Treatment options include grasping the loose end of the hair and "unwinding" it, placing a needle or scalpel under the hairs and incising them, or chemical hair removal application (if there is no skin breakdown). If the hair tourniquet has penetrated into the soft tissues, then skin incision or surgical consultation may be necessary. One "secret" in the management of the hair tourniquet is the paramount importance of close follow-up. Despite your (or your consultant's) best efforts, all the encircling hairs may not be completely removed. This is especially an issue when the hair tourniquet has penetrated more deeply into the soft tissue and visualization becomes problematic. The family needs to explicitly understand the importance of returning to the ED if there is increasing swelling, discoloration, or pain in the appendage after discharge. The hair tourniquet syndrome is typically not an abusive injury.

Loiselle J, Cronan KM: Hair tourniquet removal. In Henretig FM, King C (eds): Textbook of Pediatric Emergency Procedures, 2nd ed. Philadelphia, Lippincott Williams & Wilkins, 2008.

O'Gorman A, Ratnapalan S: Hair tourniquet management. Pediatr Emerg Care 2011; 27:203-204.

Sytwestrzak MS, Fischer BF, Fischer H: Recurrent clitoral tourniquet syndrome. Pediatrics 2000;105:866.

10. A nontoxic-appearing 5-week-old presents with fever and crying. The mother mentions that the crying increases during diaper changes. The cerebrospinal fluid and urinalysis results appear normal, a blood culture is pending, and you've initiated parenteral antibiotics. What else should be considered?

A septic hip. The evaluation and "processing" of the febrile child younger than 8 to 12 weeks of age can become so rote that we sometimes forget to perform a thorough physical examination, *including the hips*. A septic hip can occur in this age range and is a surgical emergency with devastating consequences if not recognized promptly. The infant may exhibit "pseudo-paralysis" of the limb, with a preference for abduction and external rotation of the hip. Crying may increase at diaper changes as the parent inadvertently manipulates the hips. The peripheral white blood cell count is often normal. C-reactive protein and sedimentation rate are generally elevated.

11. An 8-month-old white female presents with crying and a history of fever. In the ED, she is febrile but nontoxic in appearance, and the general physical examination is normal. What is the most likely "treatable" diagnosis?

Far and away, it is UTI, unless you consider viral illness "treatable." In a 1998 ED study by Shaw and associates, 16% of febrile white females younger than 2 years of age had a UTI—even when "otitis media, viral gastroenteritis, or upper respiratory infection" had been diagnosed. In the same cohort, if isolated fever was the only presenting symptom, an

impressive 31% had a UTI. The rate of UTIs in young Hispanic girls is probably comparable to that in whites; however, it is much lower than the rate in young African-American females.

Shaw KN, Gorelick M, McGowan KL, et al: Prevalence of urinary tract infection in febrile young children in the emergency department. Pediatrics 1998;102:e16.

12. Are there any cardiac causes that present with crying as a chief complaint?

Somewhat surprisingly, supraventricular tachycardia was the ultimate diagnosis in 4% of the afebrile infants who presented with "excessive crying" in Poole's 1991 ED study. Keep this pearl tucked away: If there is a history suggestive of paroxysmal tachycardia, remember that in the asymptomatic period, a delta wave (widened QRS with a slurred upstroke seen on 12-lead electrocardiogram) can give you the diagnosis.

Poole SR: The infant with acute, unexplained, excessive crying. Pediatrics 1991;88:450-455.

Key Points: Emergency Department Evaluation of the Crying Infant

1. An organized approach is vital to appropriate diagnosis in the ED.
2. Routine use of "screening" laboratory studies and radiographs, not guided by history and physical examination findings, is generally not helpful.
3. A thoughtful history and a thorough physical examination are the cornerstones of diagnosis.
4. Knowledge of the natural history of colic can be an extremely useful tool in generating a differential diagnosis.
5. Awareness of potential life-threatening conditions with crying as a presenting symptom (e.g., abusive injury, meningitis, shaken baby syndrome, intussusception) is paramount.

13. Name three relatively common life-threatening surgical emergencies of infancy that you would expect to present with crying but in which crying sometimes is absent.

- **Intussusception:** Intussusception generally presents with paroxysmal crying/irritability, vomiting, and later bloody stool, but a significant subset of patients present with *isolated lethargy*. These usually afebrile infants may be misdiagnosed as having sepsis, a toxic ingestion, or a closed head injury. Intussusception generally presents in the 2-month to 2-year age range, with a peak at about 9 months of age. In the lethargic infant, always palpate for abdominal masses, and if the diagnosis of intussusception is in question, include a rectal examination. You may be surprised to find blood on the examining finger, although a negative finding does not exclude the diagnosis (occult blood is found in only 16% at the time of presentation).
- **Midgut volvulus:** Although crying may be associated with this diagnosis, many infants initially presenting with midgut volvulus are surprisingly nontoxic and calm in appearance, with a benign abdominal examination *until* the gut begins to infarct. Eighty percent of patients present in the first 4 weeks of life, and the cardinal diagnostic sign is *bilious* vomiting. If this diagnosis is suspected, an *emergent* upper gastrointestinal study is mandatory.
- **Shaken baby syndrome:** Although this syndrome may present with crying and irritability, many infants have a chief complaint of listlessness or lethargy. In children under 2 years of age, your threshold for computed tomography of the head should be low. Although most infants with shaken baby syndrome do *not* have external signs of trauma, *any* facial bruising or intraoral trauma should be a major red flag. Retinal hemorrhage is generally the only physical finding (but unless the child is severely obtunded, it is problematic to diagnose). In a 1999 study by Jenny and colleagues, physicians were most likely to miss this diagnosis if the family was white and both parents lived at home. In this same study, the most frequent misdiagnoses were viral gastroenteritis (persistent vomiting), *accidental* head trauma, rule-out sepsis, and colic.

Jenny C, Hymel KP, Ritzen A, et al: Analysis of missed cases of abusive head trauma. JAMA 1999;281:621-626.

Reijneveld SA, van der Wal MF, Brugman E, et al: Infant crying and abuse. Lancet 2004;364:1340-1342.

14. Are routine screening laboratory and radiologic tests generally useful in the ED evaluation of the infant with a chief complaint of crying?

The short answer is "no." Routinely obtaining a panel of laboratory or x-ray studies as a diagnostic screen *seldom* adds to the evaluation (except for the expense). Generally the diagnosis lies in a thoughtful history and a thorough physical examination. In *selected* cases, specific laboratory tests or selected studies can certainly be of value (most commonly a catheterized urine specimen and occasionally a corneal examination with fluorescein or a rectal examination for blood). If the infant is ill-appearing or the crying is persistent throughout the ED evaluation, hospitalization should be strongly considered. In these infants, additional testing may have critical importance (lumbar puncture in the child with meningitis, computed tomography of the head in shaken baby syndrome, air enema with intussusception, toxicology screen in methamphetamine exposure).

15. A 3-month-old infant is brought to the ED with a weak cry. The mother reports the baby has been crying or whimpering all day and she describes it as a "pathetic" cry. She also reports the baby is not feeding well and seems to have a weak suck. The mother denies any fever and there is no vomiting or diarrhea. In fact, the baby has not had a stool for a few days. On examination the vital signs are normal and the baby is alert, not toxic, staring at you. But the cry does sound weak, and the baby seems to be hypotonic. What is the most likely diagnosis?

Infant botulism is likely. Sepsis is a consideration, but with no fever, normal vital signs, and an alert baby, this is less likely. Poor suck, hypotonia, and constipation are common findings with infant botulism. However, the weak cry is often what the parents notice first, and this is what precipitates a visit to the ED.

Pifko E, Price A, Sterner S: Infant botulism and indications for administration of botulism immune globulin. Pediatr Emerg Care 2014;30(2):120-124.

CHAPTER 11

DIARRHEA

Linda D. Arnold

1. What is diarrhea?

Diarrhea is caused by a disturbance of the mechanisms regulating intestinal fluid and electrolyte transport, resulting in a decrease in the consistency, or an increase in the frequency, of stool. Marked variations in "normal" number, volume, and consistency of stools exist between individuals. Breastfed babies, for example, may stool more than seven times a day. An increase in stool quantity, or more than 10 stools per day, constitutes diarrhea in infants. Diarrhea in older children, teens, and adults is characterized as more than three stools per day.

2. Hasn't the rotavirus vaccine eliminated most diarrhea?

Although rotavirus infection rates in the United States have declined dramatically in recent years, acute gastroenteritis is still common among young children, who average two diarrheal illnesses per year. Gastroenteritis has recently been estimated to account for more than 80,000 hospitalizations, 1 million emergency department (ED) visits, and 2 million office visits annually for children under age 5.

Payne DC, Vinje J, Szilagi PG, et al: Norovirus and medically attended gastroenteritis in U.S. children. N Engl J Med 2013;368:1121-1130.

3. What are the major causes of acute diarrhea?

Diarrhea of less than a 2-week duration is classified as "acute" and, in the majority of cases, results from enteric infection due to viral pathogens. Norovirus is now the most common pathogen detected in both outbreaks and endemic cases of acute gastroenteritis. Other viruses, bacterial pathogens, food-borne toxins, and parasites also play a role. Acute diarrhea may be due to sorbitol or carbohydrate malabsorption, or the initial manifestation of milk or soy protein intolerance. Extraintestinal infections, such as otitis media, urinary tract infections, and appendicitis, can also cause diarrhea.

4. How about persistent diarrhea—what is the differential diagnosis?

- Persistent diarrhea in infants may result from infection, protein intolerance, malnutrition, anatomic anomalies, or metabolic disorders such as cystic fibrosis or enzyme and transport defects.
- In older infants and toddlers, nonspecific "toddler's diarrhea," postinfectious diarrhea, and protein intolerance are all common. Diarrhea in this age group may also be caused by *Giardia*, celiac sprue, sucrase-isomaltase deficiency, and Hirschsprung's enterocolitis.
- For school-aged children and adolescents, think about giardiasis, celiac disease, irritable bowel, lactose intolerance, and inflammatory bowel disease (IBD). Consider laxative abuse in teens.

5. What should the history focus on?

Detailed questions about type and duration of symptoms, weight loss, quantity of oral intake, number of episodes of emesis and diarrhea, and frequency of urine output will help determine the degree of dehydration. Ask about recent trips and possible consumption of unpasteurized milk or fruit juice or improperly cooked meat or poultry, which may contain *Escherichia coli*, *Salmonella*, or parasites. Children are not known for good hand hygiene and may become infected at day care, during visits to farms or petting zoos, or from contact with infected pets. One percent of healthy cows carry *E. coli* 0157:H7, which can be transmitted by direct contact, through contact with feces, or by ingesting infected food. *Salmonella* can be acquired from chickens and ducks, cows, pet birds, frogs, turtles, lizards, iguanas, furry little hamsters, and the fresh or frozen rodents fed to snakes. Many wild and domestic birds, farm animals, and young cats and dogs harbor *Campylobacter*. *Giardia* is the leading cause of diarrhea from untreated drinking water or recreational waters contaminated with sewage or human or animal feces.

Cryptosporidium is very chlorine resistant, and large outbreaks have been traced to swimming pools.

6. Why is a good travel history so important?

Domestic travel involving hiking, camping, or visits to remote areas may expose children to pathogens in untreated drinking water. Among children traveling internationally, 28% develop diarrhea; information about relevant pathogens and potential exposures is an important part of the evaluation. Ninety percent of diarrheal outbreaks on cruise ships are caused by norovirus, which is highly contagious, survives for long periods on many surfaces, and is resistant to alcohol-based disinfectants and many detergents. Enterotoxigenic *E. coli* is a less frequent cause of cruise ship outbreaks. Children visiting foreign relatives are at greatest risk of acquiring parasitic and bacterial infections from infected food or water. Compared to "tourists," children visiting family are much younger, stay longer, and are more likely to travel to disease-endemic areas. Conversely, they are less likely to receive pretravel health advice, immunizations, or prophylaxis.

Hagmann S, Neugebauer R, Schwartz E, et al: Illness in children after international travel: Analysis from the GeoSentinel Surveillance Network. Pediatrics 2010;125:e1072-e1080.

7. Can specific signs or symptoms help identify a cause?

Yes, sometimes:

- Viral pathogens tend to target the proximal small intestine. Onset of illness is abrupt and duration limited. Patients often present with emesis and diarrhea, and respiratory symptoms are common.
- Bacterial pathogens induce colonic inflammation, causing bloody or mucoid stools, cramping abdominal pain, fever, and tenesmus. Bacterial toxins may produce a watery stool.
- Food poisoning is characterized by abrupt onset of vomiting after a meal, followed by diarrhea. Norovirus is responsible half the time, and bacteria or bacterial toxins are the cause in 40% of cases.
- Increased flatus and bloating may be seen with *Giardia* infection or lactose intolerance.
- Irritable bowel syndrome is characterized by cramping pain and frequent, small-volume, liquid stools alternating with constipation; physical and emotional stress may exacerbate the condition.

8. How can I assess hydration status?

Physical findings are detectable when children have a fluid deficit equivalent to 3% or more of body weight. General appearance, capillary refill longer than 2 seconds, dry mucous membranes, and reduced tears are all significantly associated with dehydration. Tachycardia, decreased skin elasticity, sunken eyes, and weight loss are also important signs. Decreased urine output is an early finding in dehydration; reliance on this sign alone may lead to overdiagnosis of dehydration.

Gorelick MH, Shaw KN, Murphy KO: Validity and reliability of clinical signs in the diagnosis of dehydration in children. Pediatrics 1997;99(5):e6.

9. What else should I look for on physical examination?

Children with uncomplicated gastroenteritis tend to have mild diffuse abdominal tenderness and active bowel sounds. Localized or rebound tenderness and absent or high-pitched bowel sounds indicate a possible surgical process or bowel obstruction. Palpation of a mass or a discrete loop of bowel suggests constipation, intussusception, or IBD; children with IBD may also have perianal tags, fissures, or abscesses. Increased anal tone and explosive stools should raise concerns of Hirschsprung's enterocolitis, and pallor and decreased urine output in the setting of bloody diarrhea are suggestive of hemolytic uremic syndrome (HUS). Children with protuberant abdomens and wasting of the buttocks and extremities should be evaluated for giardiasis, celiac sprue, and cystic fibrosis.

10. Why is it sometimes helpful to inspect the stool?

Although the presence of blood or mucus in stool suggests an inflammatory cause, stools described as "bloody" often contain visible remnants of red foods, rather than actual blood.

Watery, explosive stools with a foul vinegar-like odor suggest carbohydrate malabsorption. Bulky, foul-smelling stools are seen in fat malabsorption. Consider celiac disease, cystic fibrosis, pancreatic insufficiency, and bile salt malabsorption in the presence of steatorrhea. Undigested

cellulose particles are often visible in the stools of children with toddler's diarrhea. Parental fixation notwithstanding, the color of a child's stool is generally not important.

11. What foods can mimic hematochezia?

Children who have ingested red fruit juices, candies, gelatin, popsicles, Kool-Aid, tomatoes, beets, plums, watermelon, or cranberries may have red stools. Tarry-looking stools may follow consumption of bismuth-containing antidiarrheal products, iron, black licorice, blueberries, spinach, purple grapes, chocolate, or grape juice. False-positive results on hemoccult testing can occur in children who have ingested red meat, cherries, tomato skin, or iron supplements.

12. Name the common infectious causes of diarrhea.

- **Viral:** norovirus, rotavirus, enteric adenovirus, astrovirus, calicivirus
- **Bacterial:** *Salmonella, Shigella, Campylobacter, Yersinia* spp., *E. coli, Clostridium difficile*
- **Preformed bacterial toxins:** *Bacillus cereus, Clostridium perfringens, Staphylococcus aureus*
- **Parasitic:** *Giardia* spp., *Cryptosporidium* spp., *Entamoeba histolytica, Strongyloides* spp., *Microsporidium* spp.

13. What complications of infectious diarrhea should I be concerned about?

Both *Shigella* spp. and *E. coli* can induce seizures via high fever or toxin elaboration.

Salmonella spp. cause bacteremia in approximately 6% of those infected, with rates of 11% to 45% reported in infants and neonates. Immunocompromised patients and those with hematologic disorders are at increased risk for developing salmonella osteomyelitis. Giardiasis, like celiac sprue and IBD, often causes failure to thrive. *C. difficile* cytotoxin release may lead to life-threatening pseudomembranous colitis. Ten percent of patients with *E. coli* O157:H7 infection develop HUS or thrombotic thrombocytopenic purpura as a result of cytotoxin-induced endothelial injury.

14. Which conditions may be mistaken for infectious diarrhea?

- Chronic nonspecific (toddler's) diarrhea
- Encopresis (constipation with overflow incontinence)
- Sorbitol-induced diarrhea
- Milk protein allergy
- Lactose intolerance
- Laxative abuse

15. What method of rehydration is best for children with dehydration from diarrheal illness?

Oral rehydration therapy (ORT) with an appropriate oral rehydration solution (ORS) is recommended for all children with uncomplicated gastroenteritis and mild-to-moderate dehydration, though pediatric emergency medicine providers have been slow to adopt it. ORT is as effective as intravenous (IV) hydration, takes less time to initiate, requires less staff time, and is preferred by families.

IV hydration is indicated for children with complicated illness, moderate-to-severe dehydration, shock, persistent vomiting, intractable diarrhea, or mental status changes.

Spandorfer PR, Alessandrini EA, Joffe MD, et al: Oral versus intravenous rehydration of moderately dehydrated children: A randomized, controlled trial. Pediatrics 2005;115:295-301.

Key Points: Oral Rehydration Therapy

1. ORT is recommended for mild to moderate dehydration.
2. Use a low-osmolarity ORS.
3. Give frequent, small aliquots via 5-mL syringe or spoon.
4. Give 50 to 100 mL/kg body weight over 2 to 4 hours for rehydration.
5. Replace ongoing losses: 10 mL/kg per stool; 2 mL/kg per emesis.
6. Consider slow, continuous nasogastric administration of ORS for persistent emesis.

16. Are antimotility agents safe for children?

Pharmacologic antimotility agents are not recommended for use in children because of limited testing and potential side effects. Bismuth-containing compounds pose a risk of salicylate

absorption and toxicity at high doses, and bismuth encephalopathy has been reported in patients with renal insufficiency. Opiates, such as Lomotil, and synthetic opiates, such as Imodium, can cause central nervous system–induced sedation, respiratory depression, and gastrointestinal ileus. Gut stasis following use of these agents may lead to invasion of the bowel wall by infectious organisms, leading to worsening infection. Lomotil, in particular, should be avoided because it contains atropine. Dysentery is an absolute contraindication to the use of antimotility agents.

17. When are antibiotics recommended?

Most diarrhea is caused by viruses. Although antibiotics may be required to treat parasitic infections, or prescribed for specific bacterial pathogens in selected populations, results of stool studies are seldom available at the time of ED evaluation. Empiric antibiotic treatment is seldom recommended for suspected bacterial enteritis, owing to limited effectiveness in shortening the duration of illness, role in increasing antibiotic resistance, and controversy surrounding the potential for increased risk of HUS, resulting from acute toxin release following antibiotic-induced injury to bacterial cell membranes. As a rule, antibiotics should be administered only to children with signs of sepsis, bacteremia, or extraintestinal spread of infection. When *Salmonella* gastroenteritis is suspected or confirmed, antibiotics are indicated for infants, and for children who are toxic appearing, asplenic, immunocompromised, or malnourished. Empiric therapy and infectious disease (ID) consultation are warranted when the history and clinical presentation suggest typhoid fever, dysentery, or severe colitis, particularly among recent travelers.

Key Points: Acute Gastroenteritis

1. The majority of cases of acute gastroenteritis are self-limited and require no specific therapy.
2. Morbidity and death due to diarrheal diseases in children result mostly from dehydration.
3. Infants and malnourished children have the greatest risk of sequelae from diarrheal illness.
4. Use of antidiarrheal agents is not recommended for children.
5. Proper handwashing is extremely effective in preventing transmission of infectious agents.

18. When is diarrhea a true emergency?

Urgent intervention is required for moderate-to-severe dehydration or when a surgical cause is suspected. Although most dehydration can be easily treated with oral or IV fluids, hypernatremic dehydration must be managed carefully, as cerebral edema may develop with rapid rehydration. Infants with infectious or allergic enteritis may present in compensated or uncompensated shock. Also, appendicitis can present with diarrhea secondary to cecal inflammation. Intussusception, characterized by severe episodic abdominal pain and lethargy, may also cause vomiting or passage of what appears to be bloody diarrhea. In addition, mortality rates for Hirschsprung's enterocolitis are high; prompt decompression via rectal tube is imperative, pending definitive treatment. Finally, fluids must be managed carefully in children with HUS, who frequently present with dehydration and acute renal failure, often complicated by hyperkalemia, hypertension, anemia, and thrombocytopenia. Although 85% of affected children recover fully, half require dialysis and mortality rates average 5%.

Scheiring J, Andreoli SP, Zimmerhackl LB: Treatment and outcome of Shiga-toxin-associated hemolytic uremic syndrome (HUS). Pediatr Nephrol 2008;23(10):1749-1760.

19. When is testing indicated in children with diarrhea?

The use of clinical and historical criteria (fever, visible blood or mucus, absence of vomiting, and stool frequency), combined with selective application of screening tests or stool cultures, offers a cost-effective means of identifying patients with bacterial pathogens. A higher index of suspicion is required for infants, among whom sequelae are more common. Children with persistent diarrhea or evidence of a noninfectious cause should be screened for conditions such as IBD, celiac disease, and carbohydrate intolerance. *Giardia* antigen or ova and parasite specimens should be sent and celiac antibody testing should be performed in children with persistent diarrhea and failure to thrive.

20. What instructions should parents be given about diet?

Breast milk and formula provide superior nutrition to clear liquids and should be offered throughout the course of an uncomplicated illness, if tolerated. When clear liquids are given for

rehydration, families should be instructed to use one of the commercially available glucose-electrolyte–containing solutions. Apple juice, soda, and sports beverages should be avoided; their hypertonicity and high carbohydrate load can cause diarrhea to worsen. The traditional BRAT diet (bananas, rice, applesauce, and toast) is well tolerated but fails to provide adequate calories, protein, fat, and fiber. Children should resume a regular diet, with age-appropriate foods and full-strength milk or formula, as soon as possible.

21. Shouldn't children with diarrhea avoid milk products?

A lactose-free diet is often recommended, but rarely required, during episodes of acute gastroenteritis. Patients with severe enteritis may have damage to the brush-border membrane of the small intestine, resulting in temporary decreases in lactase levels. Despite this, the majority of infants and children show no clinical signs of malabsorption when fed full-strength milk or formula. Formula-fed infants with persistent watery, explosive diarrhea may benefit from a disaccharide-free formula such as Prosobee or Isomil-DF until the gut recovers. Older children with these symptoms may consider avoiding lactose-containing products for a couple of weeks.

Key Points: Managing Fluids and Nutrition

1. Mothers should be encouraged to continue breastfeeding.
2. Use of special or dilute formulas is generally not necessary.
3. Oral rehydration with hypotonic glucose-electrolyte solutions is the therapy of choice for children with mild-to-moderate dehydration.
4. IV fluids are indicated in cases of moderate-to-severe dehydration or when oral fluids are not tolerated. Hypovolemic shock requires rapid administration of 20 mL/kg boluses of isotonic crystalloid, and frequent reassessments, until mental status and perfusion improve.
5. Rapid return to age-appropriate feeding patterns is indicated once children are rehydrated.

22. What is the best way to prevent diarrhea in children?

Multiple studies in both developed and developing countries, set in homes and institutions, have concluded that frequent handwashing is very effective in decreasing the frequency of diarrheal illness in children. Studies in day care centers show up to 50% decreases in episodes of diarrhea following training and education of children and child care providers.

EAR PAIN

Shabana Yusuf and Joan E. Shook

CHAPTER 12

1. What is the most common cause of ear pain in the young child?

Ear pain (otalgia) is a common complaint in the pediatric emergency department (ED). Although the differential diagnosis for otalgia is lengthy, the most common cause of ear pain is acute otitis media (AOM). Other otogenic causes include otitis externa and foreign bodies. Nonotogenic causes include dental caries or abscess, pharyngitis, and cervical lymphadenitis.

AOM is caused by an acute bacterial (or occasionally viral) infection resulting in inflammation of the middle ear that is usually associated with pain and fever. The peak age for AOM is 3 to 16 months of age, but it may occur at any time.

Arnett AM: Pain—Earache. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 455-460.

Elden LM, Potsic W: Otolaryngologic emergencies. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 1545-1559.

2. What are the diagnostic criteria for otitis media?

- Recent, usually abrupt onset of signs and symptoms of middle ear infection (such as pain, irritability, fever, otorrhea)
- Presence of middle ear effusion
- Signs of middle ear inflammation

Lieberthal AS, Carroll AE, Chonmaitree T, et al: Clinical practice guideline: The diagnosis and management of acute otitis media. Pediatrics 2013;131:e964-e999.

3. What signs or symptoms suggest middle ear inflammation?

New onset of ear pain and bulging or redness of the tympanic membrane (TM) with impaired mobility.

Conover K: Earache. Emerg Med Clin North Am 2013;31:413-442.

Lieberthal AS, Carroll AE, Chonmaitree T, et al: Clinical practice guideline: The diagnosis and management of acute otitis media. Pediatrics 2013;131:e964-e999.

Pelton SI: Otitis media: Re-evaluation of diagnosis and treatment in the era of antimicrobial resistance, pneumococcal conjugate vaccine, and evolving morbidity. Pediatr Clin North Am 2005;52:711-728.

4. What are other presenting signs and symptoms of otitis media?

Other signs and symptoms of otitis are hearing loss, vertigo, tinnitus, postauricular swelling, facial paralysis, and conjunctivitis. The association of otitis media and conjunctivitis is referred to as the otitis-conjunctivitis syndrome. This is usually caused by nontypeable *H. Influenzae*.

Bodor EF: Conjunctivitis-otitis syndrome. Pediatrics 1982;69:695-698.

Klein JO, Bluestone CD: Otitis media. In Feigin RD, Cherry JD (eds): Textbook of Pediatric Infectious Diseases, 6th ed. Philadelphia, WB Saunders, 2009, pp 216-237.

5. What are the most common bacterial pathogens that cause otitis media?

Streptococcus pneumoniae, nontypable *Haemophilus influenzae*, and *Moraxella catarrhalis* are the most common bacterial organisms that cause otitis media. *Streptococcus pyogenes*, *Staphylococcus aureus*, *Mycoplasma pneumoniae*, and gram-negative bacilli are occasionally seen. The use of the conjugate pneumococcal vaccine has decreased the number of episodes from *S. pneumoniae*, but a proportionate increase in incidence from *H. influenzae* and *M. cattarrhalis* has been seen.

Klein JO, Bluestone CD: Otitis media. In Feigin RD, Cherry JD (eds): Textbook of Pediatric Infectious Diseases, 6th ed. Philadelphia, WB Saunders, 2009, pp 216-237.

6. Describe the initial management of acute otitis media.

The initial management of otitis media can be either observation with close follow-up or antibiotic treatment. The option of watchful waiting can be done in children older than 6 months with unilateral otitis media with mild symptoms and without ottorhea. Pain associated with the infection can be managed with either acetaminophen or ibuprofen. Often a few drops of analgesic ear drops (e.g., Auralgan otic solution), placed in the ear canal until it is filled, seems to relieve pain promptly. Evidence for this may be lacking. If symptoms persist or get worse in the 48 to 72 hours after diagnosis, begin antibiotics.

Foxlee R, Johansson A, Wejfalk J, et al: Topical analgesia for acute otitis media. Cochrane Database Syst Rev 2006;(3):CD005657.

Lieberthal AS, Carroll AE, Chonmaitree T, et al: Clinical practice guideline: The diagnosis and management of acute otitis media. Pediatrics 2013;131:e964-e999.

7. Which antibiotics best treat bacterial otitis media?

The first-line drug in the treatment of otitis media is amoxicillin dosed at 80 to 90 mg/kg/day in two divided doses for 10 days. The second-line drug for otitis media is amoxicillin-clavulanate. In patients who do not have type 1 allergic reaction to penicillin, cefdinir, cefpodoxime, cefuroxime, or clindamycin can also be used. In patients with a history of type 1 allergic reaction to penicillin, azithromycin, clarithromycin, or trimethoprim-sulfamethoxazole can be used. If the patient is vomiting or is noncompliant with medications, a single dose of ceftriaxone (50 mg/kg) is also effective. A second dose of ceftriaxone can be given in 48 hours if symptoms have not resolved. Infants younger than 4 weeks old are usually treated for otitis media with intravenous (IV) medications (ampicillin and gentamicin).

Elden LM, Potsic W: Otolaryngologic emergencies. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 1545-1559.

Klein JO, Pelton S: Epidemiology, pathogenesis, diagnosis and complications of acute otitis media. UpToDate, Jan 31, 2014.

Klein JO, Bluestone CD: Otitis media. In Feigin RD, Cherry JD (eds): Textbook of Pediatric Infectious Diseases, 6th ed. Philadelphia, WB Saunders, 2009, pp 216-237.

8. What is otitis media with effusion and how is it treated?

Otitis media with effusion is a collection of fluid in the middle ear without signs and symptoms of acute infection. On examination, bulging of the TM, limited or absent mobility of the TM with pneumatic otoscopy, air-fluid level behind the TM, or otorrhea is seen. An initial course of antibiotics for 10 days with activity against β-lactamase-producing organisms is recommended. If effusion does not resolve, consider referral to an otolaryngologist for tympanostomy tubes.

Klein JO, Bluestone CD: Otitis media. In Feigin RD, Cherry JD (eds): Textbook of Pediatric Infectious Diseases, 6th ed. Philadelphia, WB Saunders, 2009, pp 216-237.

Lieberthal AS, Carroll AE, Chonmaitree T, et al: Clinical practice guideline: The diagnosis and management of acute otitis media. Pediatrics 2013;131:e964-e999.

9. What is the role of vaccines in preventing otitis media?

General usage of the pneumococcal conjugate vaccine has resulted in a reduction of otitis media by 6% to 7% and tube insertions by 24%. A 13-valent pneumococcal vaccine was introduced in the United States in 2010, and a 10-valent pneumococcal vaccine has been licensed in Europe. There is a potential of these vaccines to significantly reduce the incidence of otitis media. Viral vaccines have also been shown to decrease AOM. Influenza virus vaccine reduces the incidence of AOM in vaccinated children.

Lieberthal AS, Carroll AE, Chonmaitree T, et al: Clinical practice guideline: The diagnosis and management of acute otitis media. Pediatrics 2013;131:e964-e999.

Rovers M, Schilder AGM, Zielhuis GA, Rosenfeld R: Otitis media. Lancet 2004;363:465-473.

Vergison A, Dagan R, Aguedas A, et al: Otitis media and its consequences: Beyond the earache. Lancet Infect Dis 2010;10:195-203.

10. Is antibiotic prophylaxis indicated for prevention of otitis media?

Antibiotic prophylaxis can prevent otitis media, but 9 months of antibiotics is needed in order to prevent a single episode of AOM. This is not practical for most patients given the length of treatment, drug side effects, and increased bacterial resistance.

Rovers M, Schilder AGM, Zielhuis GA, Rosenfeld R: Otitis media. Lancet 2004;363:465-473.

11. What is the role of surgery in the management of otitis media?

The decision to place tympanostomy tubes depends largely on whether the child has associated symptoms such as hearing loss, thus placing the child at risk developmentally. For initial surgery, myringotomy with tympanostomy without adenoidectomy is recommended unless nasal obstruction is present. Tonsillectomy is not recommended to treat otitis media with effusion.

Rovers M, Schilder AGM, Zielhuis GA, Rosenfeld R: Otitis media. Lancet 2004;363:465-473.

12. What are the complications of otitis media?

The complications of otitis media include perforation of the TM, cholesteatoma, adhesive otitis media, tympanosclerosis, mastoiditis, petrositis, labyrinthitis, meningitis, facial paralysis, and suppurative complications of the brain.

Klein JO, Bluestone CD: Otitis media. In Feigin RD, Cherry JD (eds): Textbook of Pediatric Infectious Diseases, 6th ed. Philadelphia, WB Saunders, 2009, pp 216-237.

13. What is bullous myringitis? How is the diagnosis made?

Bullous myringitis is an infection of the TM with intensely painful bulla formation on the surface. The diagnosis is made easily by otoscopy. The cause is usually viral, but it can be caused by *S. pneumoniae*, *H. influenzae*, and *Mycoplasma*. Treatment is supportive with analgesics, and the same antibiotics are used as for otitis media without bullae.

Elden LM, Potsic W: Otolaryngologic emergencies. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 1545-1559.

14. You are evaluating a patient who presents with ear pain. On taking the history, you learn that the illness began with itchiness of the ear canal, which has become increasingly severe and evolved into pain. What is the most likely diagnosis in this child?

This presentation is classic for otitis externa (swimmer's ear). Otitis externa is an inflammatory process affecting the external auditory canal. It can be localized or diffuse and is most commonly seen during the summer months. Other predisposing factors include moisture retention due to a tortuous narrow canal or obstructive cerumen, loss of acidic environment due to inadequate cerumen lavage, exposure to an alkaline substance, or interruption of the epithelial lining of the canal because of trauma or dermatitis.

Physical examination of these patients is often revealing. The pain can be elicited by pushing on the tragus or by traction of the pinna or moving the jaw from side to side. The most common causative organisms are *Pseudomonas* sp., *Staphylococcus*, and *Streptococcus*. *Proteus* spp. may also be seen in association with *Pseudomonas*.

Dolitsky JN: Otalgia. In Bluestone CD, Stool SE, Kenna MA (eds): Pediatric Otolaryngology, Vol. I, 4th ed. Philadelphia, WB Saunders, 2003, pp 287-295.

Shah RK, Blevins N: Otalgia. Otolaryngol Clin North Am 2003;36:1137-1151.

15. How is otitis externa treated?

Treatment includes avoidance of swimming, pain control with analgesics, ear drops with a combination of antibiotic-corticosteroid preparation (e.g., cortisporin), or antibiotic drops alone (e.g., ofloxacin otic solution 0.3%, a good choice because it covers *Pseudomonas* sp. well). A wick placed in the external auditory canal may be needed if severe edema of the canal is present. In severe infection, oral antibiotics may be necessary.

Dolitsky JN: Otalgia. In Bluestone CD, Stool SE, Kenna MA (eds): Pediatric Otolaryngology, Vol. I, 4th ed. Philadelphia, WB Saunders, 2003, pp 287-295.

Long M: Otitis externa. Pediatr Rev 2013;34:143-144.

16. What is malignant otitis externa?

This condition is a severe form of otitis externa that is not responsive to conventional therapy. It is caused by *P. aeruginosa* and involves the bone and marrow of the skull base, causing osteomyelitis. Malignant otitis externa may also result in chondritis and facial nerve paralysis. Other complications include stenosis of the canal and permanent hearing loss. It is rare in children but can be seen in patients with diabetes mellitus or those who are immuno-

compromised. Both computed tomography (CT) scan and nuclear imaging are helpful in making the diagnosis. Treatment includes IV antibiotics and removal of granulation tissue.
Shah RK, Blevins N: Otalgia. Otolaryngol Clin North Am 2003;36:1137-1151.

17. A toddler presents with bruising to the internal surface of the pinna. According to the mother, he is a very active child and falls frequently. What diagnosis should you consider?

Bruising to the internal surface of the pinna may result from a direct blow ("boxing") to the ear. You therefore must consider the possibility of child abuse. The unexplained presence of hemotympanum and the perforation of the TM may also suggest child abuse, because they can result from a direct blow to the ear.
Feldman KW: Patterned abusive bruises of the buttocks and pinnae. Pediatrics 1990;4:633-636.
Manning SC, Casselbrant M, Lammers D: Otolaryngologic manifestations of child abuse. Int J Pediatr Otorhinolaryngol 1990;20:7-16.
Obiako MN: Ear drum perforation as evidence of child abuse. Child Abuse Negl 1987;11:149-151.

18. Infections at the site of ear piercing are often extremely painful and cause some concern. What organisms should you consider covering when you choose an antibiotic?

Cellulitis or perichondritis of the pinna can cause otalgia and are usually caused by *S. aureus* or *Pseudomonas* sp. These infections are managed first with removal of the piercing. Infection of the cartilage can be serious, so IV antibiotics are indicated and incision and drainage of abscesses may be needed.
Arnett AM: Pain—Earache. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 455-460.
Shah RK, Blevins N: Otalgia. Otolaryngol Clin North Am 2003;36:1137-1151.

19. You are examining a child who came to the ED because of fever and ear pain. You notice that his ear is red and swollen and seems to be protruding from the side of his head. What diagnosis is most likely?

This child likely has mastoiditis, a relatively uncommon complication of otitis media. On physical examination the posterior auricular area is erythematous and very tender. It is important to visualize the TM, because severe otitis externa also can present with postauricular erythema, swelling, and protrusion of the pinna. In mastoiditis, the TM is erythematous and bulging, although with otitis externa, the TM is usually normal. CT can help differentiate between mastoiditis and severe otitis externa when a complete physical examination is not possible. Petrositis, which is the infection of the petrous part of the temporal bone, is rare. Gradenigo syndrome or petrositis presents as a triad of deep facial pain, otitis media, and ipsilateral abducens nerve paralysis.
Conover K: Earache. Emerg Med Clin North Am 2013;31:413-442.
Elden LM, Potsic W: Otolaryngologic emergencies. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 1545-1559.
Klein JO, Bluestone CD: Otitis media. In Feigin RD, Cherry JD (eds): Textbook of Pediatric Infectious Diseases, 6th ed. Philadelphia, WB Saunders, 2009, pp 216-237.

20. How is mastoiditis treated?

Children with mastoiditis must be evaluated by an otolaryngologist and admitted to the hospital for IV antibiotics. Because the most common organisms in acute mastoiditis are *Staphylococcus* spp., *Streptococcus* spp., and *H. influenzae*, treat the patient with clindamycin or vancomycin and cefotaxime. Operative intervention in the form of mastoidectomy may be required in children with complicated infection or evidence of abscess formation.
Klein JO, Bluestone CD: Otitis media. In Feigin RD, Cherry JD (eds): Textbook of Pediatric Infectious Diseases, 6th ed. Philadelphia, WB Saunders, 2009, pp 216-237.

21. What are the potential complications of mastoiditis?

Complications of mastoiditis can be intratemporal or intracranial. Intratemporal complications include facial paralysis, labyrinthitis, and petrositis. Intracranial complications include meningitis, lateral sinus thrombosis, and epidural, subdural, or brain abscesses.

Klein JO, Bluestone CD: Otitis media. In Feigin RD, Cherry JD (eds): Textbook of Pediatric Infectious Diseases, 6th ed. Philadelphia, WB Saunders, 2009, pp 216-237.

22. The presence of a foreign body in the external auditory canal can cause severe pain. What is the best method for removing the foreign body?

Occasionally a child places a foreign body in the external canal and presents with symptoms such as hearing loss or a sensation of fullness, as well as ear pain. Symptoms at presentation vary depending on the nature of the foreign material and the length of time that it has been present. Objects can be removed by using alligator forceps, curettes, right-angle hooks, Baron suction devices, or irrigation with warm water. If the foreign body is a bean or other vegetable material, do not irrigate with water as the foreign body may swell. A foreign object that is lodged tightly in the canal may require removal with sedation or even general anesthesia using an operating microscope.

Canal wall lacerations are present 50% of the time after a foreign-body removal. It is recommended that after the object is recovered, the patient be treated with topical antibiotic or steroid drops to prevent the development of otitis externa.

Bressler K, Shelton C: Ear foreign-body removal: A review of 98 consecutive cases. Laryngoscope 1993;103:367-370.

23. You have just examined a child who has a hearing aid battery in her external auditory canal. You have not been successful in your attempts to remove it with forceps. What should your next step be?

Hearing aid batteries (or button batteries) can cause extensive caustic skin and bony damage in a short period of time. Removal of this foreign object is an otologic emergency and should be performed as soon as possible after detection. If you are unable to remove the battery, call an otolaryngologist for removal in the operating room.

Bressler K, Shelton C: Ear foreign-body removal: A review of 98 consecutive cases. Laryngoscope 1993;103:367-370.

24. A child presents with severe ear pain of sudden onset. You determine there is a live insect, most likely a roach, moving in the ear canal. How should you manage this patient to relieve pain quickly?

Before attempting to remove the insect, immediately place microscopic immersion oil, mineral oil, 1% lidocaine, 2% lidocaine, or viscous lidocaine in the ear canal. This thick viscous solution will "paralyze" the insect and relieve pain promptly. The foreign body can then be removed with an ear curette or by flushing the canal with water.

Leffler S, Cheney P, Tandberg D: Chemical immobilization and killing of intra-aural roaches: An in vitro comparative study. Ann Emerg Med 1993;22:1795-1798.

Su E: External auditory canal foreign bodies. In Dieckmann RA, Fiser DH, Selbst SM (eds): Illustrated Textbook of Pediatric Emergency and Critical Care Procedures. St. Louis, Mosby, 1997, pp 712-713.

25. What is middle ear barotrauma?

This is injury to the TM from a sudden change in pressure due to diving, flying, or blast injuries, and it can cause severe ear pain. On physical examination, the TM appears swollen and blue. Treatment for this condition is supportive with analgesics. Refer the patient to an otolaryngologist if he has dizziness or hearing loss, which may indicate a perilymphatic fistula.

Elden LM, Potsic W: Otolaryngologic emergencies. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 1545-1559.

Vernick DM: Ear barotrauma. UpToDate, Feb 3, 2014.

26. You are confronted with a patient who reports ear pain, and yet examination of the ear is normal. How can this phenomenon be explained?

Otalgia can be the result of referred pain from distant sites secondary to inflammatory processes, tumors, or trauma. Nonotogenic otalgia is most often of dental origin; however, pain that is perceived in either the external or the middle ear can be referred via cranial nerves V, VI, IX, and X and cervical nerves C2 and C3. These nerves supply the nasal sinus area, oral cavity and teeth, oropharynx, hypopharynx, larynx, and upper esophagus. Pain can also be psychogenic when no other cause is found.

Arnett AM: Pain—Earache. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 455-460.

Shah RK, Blevins N: Otalgia. Otolaryngol Clin North Am 2003;36:1137-1151.

27. Name some nonotogenic causes of pain in the ear. Name the nerve that carries the sensation.

- **Cranial nerve V:** Disturbances in the oral cavity, including stomatitis, gingivitis, lymphadenitis, parotiditis, trauma, and infections of the tongue; dental conditions, including eruption, impaction, trauma, caries, and abscess
- **Cranial nerve VI:** Bell's palsy, herpes zoster, tumors
- **Cranial nerve IX:** Tonsillitis and retropharyngeal abscess, adenotonsillectomy, nasopharyngeal or oropharyngeal tumors
- **Cranial nerve X:** Lesions at the base of the tongue, trachea, larynx, and esophagus; otalgia can be a manifestation of gastroesophageal reflux in infants and children
- **Upper cervical nerves:** c-spine injuries, arthritis, or disc disease

Arnett AM: Pain—Earache. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 455-460.

28. What is Ramsay-Hunt syndrome?

This is the eponym applied to herpes zoster otitis or viral polycranial neuropathy affecting the ear. It presents with severe otalgia, facial palsy, and vesicles on the pinna, external auditory canal, and TM. All cranial nerves can be affected, and other symptoms include hearing loss, vertigo, nausea, vomiting, and dizziness. Treatment is with acyclovir, systemic steroids, and analgesics.

Arnett AM: Pain—Earache. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 455-460.
Shah RK, Blevins N: Otalgia. Otolaryngol Clin North Am 2003;36:1137-1151.

29. Temporomandibular joint (TMJ) dysfunction is among the most common causes of referred otalgia. How can I confirm this as a source of ear pain?

To confirm the diagnosis of TMJ dysfunction, carefully palpate the TMJ externally by placing the fingers just anterior to the tragus and having the patient open and close the mouth. The patient with this condition will report otalgia during attempted occlusion. The pain may be caused by nerve irritation, muscle spasm, or degenerative change in the joint. TMJ dysfunction can occur in children who have bruxism, frequently clench their teeth, indulge in frequent gum chewing, or have malocclusion. Tympanometry in these patients is normal. Treatment is directed at reducing the inflammation and pain with heat therapy, soft diet, analgesia, physiotherapy, occlusal splints, and rarely surgery.

Bodner L, Miller VJ: Temporomandibular joint dysfunction in children: Evaluation of treatment. Int J Pediatr Otorhinolaryngol 1998;44:133-137.
Shah RK, Blevins N: Otalgia. Otolaryngol Clin North Am 2003;36:1137-1151.

Key Points: Ear Pain

1. Ear pain in the child is most commonly due to AOM.
2. To diagnose AOM, one must demonstrate the presence of fluid in the middle ear by pneumatic otoscopy.
3. If the examination of the ear is normal, consider secondary (referred) causes of otalgia, including TMJ syndrome, dental disease, pharyngitis, sinusitis, and gastroesophageal reflux.
4. Hemotympanum is a rare finding that can result from a basilar skull fracture, barotrauma from scuba diving, a turbulent airplane flight, or a direct blow to the ear. In the absence of a history, consider child abuse.

FEVER

Jeffrey R. Avner

CHAPTER 13

1. What percentage of visits to the emergency department (ED) is for the evaluation of fever?

Fever is one of the most common presenting symptoms at the pediatric ED, representing 15% to 30% of all visits. During the first 2 years of life, a child typically has 4 to 6 episodes of febrile illness.

2. What temperature is considered normal?

There is no single value that represents a "normal" temperature. This is largely because normal body temperature varies with age, time of day, physical activity, and environmental conditions. This individual variability limits the application of mean body temperature values derived from population studies. Thus, no single temperature should be used as the upper limit of normal. Rather, normal temperature is best described as a range of values for each individual. Although only a rough guide, some consider abnormal temperature to be higher than 38.0° to 38.2° C (rectally) in an infant and higher than 37.2° to 37.7° C (orally) in an older child or adult.

3. Who is credited with the first systematic measurement of body temperature?

In the 1860s, German physician Carl Wunderlich used foot-long thermometers to record over 1 million axillary temperature readings from 25,000 patients. He identified 37.0° C (98.6° F) as the mean temperature of healthy adults.

4. What are the differences in measurement among rectal, oral, tympanic, axillary, and skin temperature readings?

The core body temperature is best measured with an esophageal or nasopharyngeal probe, but this is difficult in the setting of an ED. Therefore, rectal temperature is used as the standard measurement to indirectly measure core body temperature. Oral measurement in the sublingual pocket is estimated to be 0.5° to 1° C lower than rectal temperature. Infrared tympanic thermometry is a popular method used in EDs, and studies show that, at least in older children and adults, the results are reasonably accurate. Axillary and infrared skin thermometers do not reliably predict rectal temperature and therefore are of limited value.

Craig JV, Lancaster GA, Williamson PR, et al: Temperature measured at the axilla compared with rectum in children and young people: Systemic review. BMJ 2000;320:1174-1178.

Paes BF, Vermeulen K, Brohet RM, et al: Accuracy of tympanic and infrared skin thermometers in children. Arch Dis Child 2010;95(12):974-978.

5. Can parents detect fever subjectively?

Touch is usually the first method parents use to detect fever. The forehead is the most common site used, and there is no difference in detection when multiple sites are palpated. Parents are usually able to detect if their child is "burning up" with fever. In general, parents are both moderately sensitive (75-89%) and specific (56-85%) in detecting the presence of fever subjectively in children. However, because the positive predictive value of "fever to touch" is 57% to 63%, maternal report of fever should always be confirmed by thermometry. Additionally, parents may not be as accurate in estimating the height of fever. In young infants (less than 2 months old), when the height of the fever is crucial for determining management, always obtain a rectal temperature.

Okposio M, Abhulimhen-Iyoha B: Accuracy of mother's touch in assessing the presence of fever in children. Niger J Paediatr 2012;39(2):56-59.

Teng CL, Ng CJ, Nik-Sherina H, et al: The accuracy of mother's touch to detect fever in children: A systematic review. J Trop Pediatr 2008;54(1):70-73.

6. Does body temperature vary during the day?

The variation of body temperature in typical circadian rhythm becomes established by 2 years of age. Peak temperature typically occurs in the late afternoon (5:00-7:00 PM), and the trough occurs in the early morning (2:00-6:00 AM). Daily temperature variation ranges from 0.1° to 1.3° C.

7. How does the body produce fever?

With infection, exogenous pyrogens (microbial products such as endotoxins) produced by various infectious organisms act on host inflammatory cells (especially monocytes and macrophages) to produce numerous cytokines, including endogenous pyrogens (interleukin, tumor necrosis factor, interferon). These circulating pyrogens act on the preoptic area of the anterior hypothalamus, which produces prostaglandin E_2 and causes fever.

8. Where is the core body temperature set?

Core body temperature is set in the anterior hypothalamus. Variation in body temperature is detected by thermosensitive neurons in the preoptic nucleus, which then directs autonomic changes in sweat glands, blood vessels, somatic neurons, and skeletal muscles.

9. How does the body regulate temperature?

Thermoregulation is controlled by a variety of physiologic and behavioral mechanisms under the direction of the hypothalamus. Elevation of body temperature occurs primarily through metabolic activity associated with increased cell metabolism, increased muscle activity, and involuntary shivering. Body temperature is decreased primarily through vasodilatation, which thereby increases heat loss by conduction, convection, or radiation through the skin. In addition, sweating and cold preference behavior (e.g., removing clothes) help dissipate the heat.

10. Is fever beneficial or harmful?

Whether fever is a friend or foe is a question for the ages. Fever has some physiologic benefits for the host, including an enhancement of both cellular and humoral immune responses as well as direct antimicrobial activity. On the other hand, fever makes the child uncomfortable and leads to a significant increase in metabolic activity (about 10% per Celsius degree), which increases oxygen consumption, carbon dioxide production, and insensible water loss. Whether the benefits outweigh the metabolic costs is debatable. Regardless, the presence of fever alerts the parents and clinicians that the child is in the process of fighting disease and, as such, can serve as an invaluable diagnostic aid.

11. What is "fever phobia"?

Barton Schmitt coined the term in the early 1980s in reference to parents' excessive concern about low-grade fever. Nowadays, the term *fever phobia* is often used to describe heightened anxiety about the presence of fever in the child that can lead to aggressive antipyretic use, unnecessary ED visits, laboratory testing, or empiric antibiotic treatment. Of note, both parents and providers may have "fever phobia."

Avner JR: Acute fever. Pediatr Rev 2009;30(1):5-13.

Poirier MP, Collins EP, McGuire E: Fever phobia: A survey of caregivers of children seen in a pediatric emergency department. Clin Pediatr 2010;49(6):530-534.

12. Does bundling cause fever in infants?

Overbundling of young infants, especially during the summer months, is known to increase measured temperatures. If an infant is heavily bundled or in a particularly hot environment, a 30- to 60-minute period of equilibration should be followed by a repeat temperature determination. This issue is particularly important in the evaluation of young febrile infants, because the height of the fever by itself is often a determinant for proceeding with an evaluation for sepsis.

13. What did Hippocrates feel was the role of fever?

Hippocrates, the father of medicine, felt that fever helped balance the four humors (blood, yellow bile, black bile, and phlegm), which were believed to cause disease when out of balance. At the current time, this theory remains unproved!

14. Who started the belief that high fever caused death?

Dr. Claude Bernard, the father of human physiology, heated animals in a makeshift oven and found that sustained temperatures over 106° F (41.1° C) was fatal. This report published

in 1876 in *Lecons sur la Chaleur Animale* (Lessons from Animal Heat) began the erroneous concern that high fever is inherently dangerous. Of note, his findings were the result of hyperthermia, *not* fever due to the elevation of the hypothalamic set point.

15. Does high fever cause brain damage?

Although this concern is a cornerstone of "fever phobia" by parents, this fear is unfounded. There is no evidence that children with fever, *as opposed to hyperthermia*, are at increased risk of serious sequelae. Deleterious effects of temperatures higher than 42° C are based on in vitro effects on enzyme systems and not clinical studies. When brain damage does occur in association with fever, it is usually due to sequelae of the underlying disease (such as meningitis) rather than the fever itself.

Section on Clinical Pharmacology and Therapeutics; Committee on Drugs, Sullivan JE, Farrar HC: Fever and antipyretic use in children. Pediatrics 2011;127(3):580-587.

16. Does fever trigger seizures?

Fever lowers the seizure threshold in children with an underlying seizure disorder and may precipitate a seizure in children (6 months to 6 years old) who are susceptible to simple febrile seizures. Fever, by itself, in the absence of predisposing factors, does not cause seizures.

17. Does teething cause fever?

Although some studies show a mild temperature elevation associated with teething, significant fever (temperature > 38.5° C) has never been shown to be associated with tooth emergence.

Ramos-Jorge J, Pordeus IA, Ramos-Jorge ML, Paiva SM: Prospective longitudinal study of signs and symptoms associated with primary tooth eruption. Pediatrics 2011;128(3):471-476.

18. When sponging a child for treatment of fever, what is the best temperature for the water?

The value of sponging to decrease temperature is controversial. Sponging uses evaporation to help cool the child. Sponging should be used in combination with an antipyretic and may take up to 20 minutes to be effective. Use tepid or lukewarm water so that the child remains comfortable and does not shiver. Never use cold water or isopropyl alcohol, as they cause excessive vasoconstriction and shivering. In addition, isopropyl alcohol can be toxic through skin absorption.

19. Why is the use of rubbing alcohol on children harmful?

Topical application of isopropyl alcohol can cause severe intoxication in children due to skin absorption or inhalation exposure. Furthermore, the resultant cooling effect on the skin causes peripheral vasoconstriction and limits the child's ability to dissipate internal heat.

20. Does fever reduction decrease the sequelae from a febrile illness, including febrile seizures?

No. There is no evidence that reducing fever, in an otherwise healthy child, reduces the morbidity or mortality risks from a febrile illness. In fact, there is some evidence that antipyresis actually prolongs the underlying infection, albeit at the expense of child discomfort. However, for children who are chronically ill or have limited metabolic reserve, the additional metabolic demand of the febrile state might be poorly tolerated. There is also no evidence that antipyretic therapy decreases the recurrence of febrile seizures.

Section on Clinical Pharmacology and Therapeutics; Committee on Drugs, Sullivan JE, Farrar HC: Fever and antipyretic use in children. Pediatrics 2011;127(3):580-577.

21. Does the response of fever to an antipyretic predict a more benign illness?

No. Fever response to antipyretics is not clinically useful in differentiating children with serious bacterial illness from those with a more benign cause. Most children, regardless of the underlying cause of their fever, experience some temperature decline with antipyretics, although rarely do they become afebrile. However, a child who has a serious illness often continues to appear ill after fever is reduced, whereas the appearance of a child who has a benign illness usually improves. The decision to perform additional diagnostic tests, such as a complete blood count (CBC) or lumbar puncture, should be determined based on clinical grounds and not the degree of defervescence.

22. Does the height of the fever indicate serious bacterial illness?

Most studies show limited usefulness of the height of fever as a predictor of serious bacterial illness. In an otherwise healthy child even temperatures higher than 39° C have relatively

low sensitivity (10-14%) and predictive value (4-40%) for serious illness in infants over 6 months and limited usefulness in infants 3 to 6 months. Other clinical signs such as age, appearance, and peripheral perfusion are better predictors. Thus, it appears that the presence or absence of fever is what is most important. This does not mean that a thorough evaluation of a child with high fever is unnecessary; rather, *any child with fever, regardless of the height of the temperature, should receive a thorough evaluation.*

National Institute for Health and Care Excellence: Assessment and initial management of feverish illness in children younger than 5 years: Summary of NICE guidance. BMJ 2013;346:f3764.

23. What are the normal increases in heart rate and respiratory rate with each degree rise in body temperature?

Fever is associated with an increase in heart rate of about 10 to 15 beats per minute and an increase in respiratory rate of about 3 to 5 breaths per minute for each rise of a Celsius degree.

24. Is alternating acetaminophen and ibuprofen more effective for fever reduction?

Although a common practice among pediatricians, there is currently limited scientific evidence that this combination of antipyretics has greater efficacy than either agent used alone. Although there is some evidence that the antipyretic combination results in slightly lower temperatures at 4 to 6 hours, there is no evidence in overall improvement in other clinical outcomes. Furthermore, this practice may, in fact, increase fever phobia and potential toxicity from incorrect dosing.

Section on Clinical Pharmacology and Therapeutics; Committee on Drugs, Sullivan JE, Farrar HC: Fever and antipyretic use in children. Pediatrics 2011;127(3):580-587.

Paul IM, Sturgis SA, Yang C, et al: Efficacy of standard doses of Ibuprofen alone, alternating, and combined with acetaminophen for the treatment of febrile children. Clin Ther 2010;32(14):2433-2440.

25. What are the risk factors to consider in evaluating a febrile child?

Children who have either immature or specific impairment of immunologic function are at higher risk of bacteremia and serious bacterial illness. Therefore, very young infants (less than 2 months), children with immune compromise (e.g., HIV [human immunodeficiency virus] infection, sickle cell disease), and children receiving immunosuppressive medication (such as chemotherapy and steroids) may require a blood culture and empiric antibiotic treatment as part of their management.

26. Why do we treat infants less than 2 months old with fever differently than the older child?

There are three main reasons: (1) the risk of serious bacterial illness in this age group is relatively high (approximately 10%), (2) young infants have immature immune responses that may not be able to contain infection, and (3) clinical appearance is difficult to interpret. At this age, the ability of a child to interact in an interpretable social manner is inconsistent. For example, a social smile is inconsistent, if not absent, in a 1-month-old.

27. What are the common pathogens that cause serious bacterial illness in febrile infants?

In infants less than 1 month old, maternal organisms, acquired perinatally, predominate: gram-negative enteric organisms (*Escherichia coli*), group B streptococcus, *Staphylococcus aureus*, and *Listeria*. In infants older than 6 weeks, community-acquired organisms predominate, in particular pneumococcus and meningococcus. Infants 4 to 6 weeks old are infected by pathogens from either age group. Immunizations have decreased the incidence of pneumococcus and virtually eliminated *Haemophilus influenzae* type b.

28. What are low-risk criteria for the management of febrile infants?

In an attempt to avoid routine hospitalization of all febrile young infants, many investigators have sought to devise clinical and laboratory criteria that would identify a subset of febrile infants at "low risk" of having bacterial disease as a cause of their fever. Three major prospective studies have established somewhat different low-risk criteria (Table 13-1).

29. What is a "sepsis workup"?

A "sepsis workup" is typically considered an evaluation of certain body fluids for bacterial infection. It usually includes a CBC; urinalysis; lumbar puncture; and cultures of blood, urine, and spinal fluid. A "*septic* workup" is a test performed with a dirty needle.

Table 13-1. Low-Risk Criteria for the Management of Febrile Infants

VARIABLE	BOSTON	PHILADELPHIA	ROCHESTER
Age (d)	28-89	29-56	0-60
Temperature (° C)	≥38	≥38.2	≥38
WBC count	<20,000	<15,000	5000-15,000
Urinalysis	<10 WBCs/HPF	<10 WBCs/HPF and no bacteria	<10 WBCs/HPF
CXR	No infiltrate	No infiltrate	Not required
CSF (WBCs/μL)	<10	<8	Not required
Other		Band/total neutrophilis < 0.2	Absolute band count ≤ 1500
Sensitivity (%)	Not listed	100	92.4
Negative predictive value (%)	94.6	100	98.9

CSF, cerebrospinal fluid; CXR, chest x-ray study; HPF, high-power field; WBC, white blood cell.
Data obtained from Baker MD, Bell LM, Avner JR: Outpatient management without antibiotics of fever in selected infants. N Engl J Med 1993;329:1437-1441; Baskin MN, O'Rourke EJ, Fleisher GR: Outpatient treatment of febrile infants 28 to 89 days of age with intramuscular administration of ceftriaxone. J Pediatr 1992;120:22-27; and Jaskiewicz JA, McCarthy CA, Richardson AC, et al: Febrile infants at low risk for serious bacterial infection—An appraisal of the Rochester Criteria and implications for management. Pediatrics 1994;94:390-396.

30. Do all febrile infants need a sepsis workup and admission?

Although there is no absolute consensus, most agree that febrile infants less than 1 month of age who are evaluated in the ED require an evaluation for sepsis. Many also feel that the same evaluation applies to infants 1 to 2 months old. However, some clinicians withhold a lumbar puncture in well-appearing febrile infants. Infants younger than 1 month and those infants at high risk should be hospitalized pending culture results. Infants 1 to 2 months old who are at low risk (see Table 13-1) for serious bacterial illness may be managed as outpatients if follow-up is assured. This approach may be modified for febrile infants evaluated in office practices.

Byington CL, Reynolds CC, Korgenski K, et al: Costs and infant outcomes after implementation of a care process model for febrile infants. Pediatrics 2012;130(1):e16-e24.
Huppler AR, Eickhoff JC, Wald ER: Performance of low-risk criteria in the evaluation of young infants with fever: Review of the literature. Pediatrics 2010;125(2):228-233.

31. Do young febrile infants with respiratory syncytial virus (RSV) bronchiolitis need a sepsis workup?

A recent multicenter study of over 1200 infants (22% with RSV) found that febrile infants (<60 days old) with RSV were at significantly lower risk of serious bacterial illness than RSV-negative infants, although the rate of certain infections, especially urinary tract infections, remained appreciable. However, in febrile infants less than 28 days old, the risk of serious bacterial illness remained "substantial" (about 13%) and was *not* altered by the presence of RSV infection.

Levine DA, Platt SL, Dayan PS, et al: Risk of serious bacterial infection in young febrile infants with respiratory syncytial virus infections. Pediatrics 2004;113:1728-1734.

Key Points: The Febrile Infant

1. In febrile infants less than 28 days old, the risk of serious bacterial illness is substantial (about 10%) and is not altered by the presence of RSV infection.
2. Urinary tract infection is the most common bacterial infection in febrile infants less than 2 months old.
3. For well-appearing febrile infants 4 to 8 weeks old, a variety of testing and management strategies are available.

32. What is occult bacteremia?

Occult means "mysterious" or "hidden." Bacteremia means bacteria in the blood. Simply put, occult bacteremia is unsuspected bacteria in the blood. This entity is used to describe a subset of febrile children (usually 3-36 months old) who are well-appearing and have no focus of infection but, nevertheless, have bacteremia. Note that the entity of occult bacteremia does *not* apply to a child who is ill-appearing or has an obvious focus of infection on physical examination.

33. Has universal pneumococcal vaccine (PCV) use affected the incidence of occult bacteremia?

Prior to universal pneumococcal vaccination, the overall rate of occult bacteremia was about 1.5%; *Streptococcus pneumoniae* was responsible for 85% to 90% of cases, with the remainder shared among *Neisseria meningitidis*, group A streptococci, *Salmonella* spp., and *S. aureus*. Studies after the use of the heptavalent pneumococcal vaccine demonstrated a reduction in the overall rate of bacteremia to less than 0.5%. Pneumococcal occult bacteremia has been reduced to less than 0.2% in vaccinated healthy children but remains at about 1.5% in unimmunized children.

Avner JR, Baker MD: Occult bacteremia in the post-pneumococcal conjugate vaccine era: Does the blood culture stop here? Acad Emerg Med 2009;16(3):258-260.

Wilkinson M, Bulloch B, Smith M: Prevalence of occult bacteremia in children aged 3 to 36 months presenting to the emergency department with fever in the postpneumococcal conjugate vaccine era. Acad Emerg Med 2009;16(3):220-225.

34. Has the change in incidence of occult bacteremia changed physician practice?

Yes. Since the use of the PCV-7 vaccine, fewer CBCs were ordered as part of the management of febrile 2- to 24-month-old children who had no source of infection. Additionally, the likelihood that physicians ordered neither a CBC nor a urinalysis increased.

Simon AE, Lukacs SL, Mendola P: National trends in emergency department use of urinalysis, complete blood count, and blood culture for fever without a source among children aged 2 to 24 months in the pneumococcal conjugate vaccine 7 era. Pediatr Emerg Care 2013;29(5):560-567.

35. Are biomarkers useful for predicting serious bacterial illness in otherwise healthy febrile children?

The peripheral white blood cell (WBC) count is often used as a nonspecific marker, but it provides limited diagnostic value in identifying serious illness in children. A recent review of children younger than 3 years with fever without a source showed that procalcitonin levels (>0.5 ng/mL) were better predictors of serious bacterial infection (sensitivity 83%, specificity 69%) than peripheral WBC count or C-reactive protein. However, it is not clear whether the infections could have been identified by other, less invasive testing (e.g., chest radiograph, urinalysis). Furthermore, the availability of procalcitonin testing is variable.

Van den Bruel A, Thompson MJ, Haj-Hassan T, et al: Diagnostic value of laboratory tests in identifying serious infections in febrile children: Systematic review. BMJ 2011;342:d3082.

Key Points: The Febrile Child

1. Clinical signs such as age, appearance, and peripheral perfusion are better predictors of serious illness than the height of fever.
2. The presence or absence of fever, rather than a specific value, is what is most important for determining a management strategy.
3. A child who has a serious illness often continues to appear ill after the fever is reduced, whereas the appearance of a child who has a benign illness usually improves.
4. The incidence of occult bacteremia has declined to less than 0.5% overall since the universal use of pneumococcal vaccination.

36. Should the decision on whether to perform a lumbar puncture on a febrile child be guided by the height of the fever or the peripheral WBC count?

No! Neither of these measures is sensitive enough at any threshold to predict meningitis. The criteria for lumbar puncture should be based on history and physical examination findings rather than nonspecific laboratory tests.

37. Why is the presence of a petechial rash in a febrile child of concern?
A petechial rash, especially if associated with fever, may be an early sign of infection with an invasive bacterial organism, especially *N. meningitidis*. Early identification of a child with meningococcemia is essential, because the disease can progress rapidly and has a high morbidity rate. Other causes for fever and petechiae include *S. pneumoniae*, group A streptococcus, *S. aureus*, *E. coli*, and *Rickettsia*. The incidence of invasive bacterial disease has been estimated to be as high as 20% in children who are hospitalized but much lower for patients presenting to the ED.

38. What evaluation is necessary in managing the child with fever and petechiae?
If the child is ill-appearing or immunocompromised, a complete evaluation for sepsis is necessary. Hospitalization and empiric antibiotics are essential. For children who are well-appearing and have no clear cause for the petechiae a CBC and blood culture should be considered, as well as a rapid streptococcal antigen test if there is pharyngitis. Additional studies such as coagulation tests and lumbar puncture, hospitalization, and need for empiric antibiotics are somewhat controversial. Management must be individualized. Most physicians admit and treat with parenteral antibiotics any child who is young (<12 months) or has an elevated (or low) WBC count. For children who have normal laboratory tests, outpatient management with close follow-up is an option. The literature on outpatient antibiotic treatment in meningococcal disease is limited.

39. How long must fever be present to be considered a fever of unknown origin (FUO)?
The definition of FUO has changed over recent years. FUO was applied to any febrile illness with an unexplained, persistent temperature higher than 38.5° C for 3 or more weeks. In light of more sophisticated diagnostic techniques, many investigators have shortened the minimum duration of fever to 7 days before labeling the condition as FUO.

40. What are the differences among intermittent fever, spiking fever, remittent fever, sustained fever, and relapsing fever?
Intermittent fever refers to a pattern in which the temperature returns to normal at least once a day. *Hectic* or *spiking* fever has a high peak and quick defervescence. In *remittent* fever, the temperature fluctuates but always remains elevated. *Sustained* fevers remain persistent with little fluctuation. In *relapsing* fever, the temperature may return to normal for as much as a day or more before fever returns.

Lorin MI, Feigin RD: Fever of unknown origin. In McMillan JA (ed): Oski's Pediatrics, 3rd ed. Philadelphia, Lippincott Williams & Wilkins, 1999, pp 844-848.

41. What is the leading cause of FUO?
In pediatrics, almost half of the cases of FUO are due to infectious diseases (usually respiratory tract infections, followed by infections of the urinary tract, skeleton, and central nervous system).

42. How often does FUO have no diagnosis or resolve spontaneously?
Almost 25% of children with FUO are either undiagnosed or have spontaneous resolution.

43. What is the difference between fever and hyperthermia?
Fever is caused by a rise in the hypothalamic set point, which is usually the result of the triggering of several pyrogenic cytokines. *Hyperthermia* is often used to describe a condition in which the thermoregulatory system is either dysfunctional or simply overwhelmed by a variety of internal or external factors. Hyperthermia may result from disorders of excessive heat production (exertional heatstroke, thyrotoxicosis, cocaine intoxication), disorders of diminished heat dissipation (classic heatstroke, severe dehydration, autonomic dysfunction), or disorders of hypothalamic function (cerebrovascular accidents, trauma).

44. How do you decrease body temperature in children with heatstroke?
Management of heatstroke begins with aggressive attempts at cooling, such as ice packs, fanning, ice water immersions, and, occasionally, cool IV (intravenous) fluids, until the core temperature drops below 39° C. Avoid antipyretics and alcohol sponge baths. Further management includes airway and cardiovascular support, IV rehydration, and pressor support if necessary.

45. What kind of fever is associated with bizarre movements in response to disco music?
Saturday Night Fever was first identified in the 1970s in a group of adolescents wearing unbuttoned silk shirts, flared pants, and platform shoes.

CHAPTER 14

FOREIGN BODIES IN CHILDREN

Jeff E. Schunk

1. Where would you expect to find a foreign body in a child?

Just about anywhere. During normal play and exploration, children place objects anywhere they will fit. The nose is the most common site for a retained foreign body. The mouth is a common portal allowing for potential ingestion or aspiration. Ears complete the common location list, and rarely, objects are placed in the vagina, rectum, or urethra.

2. Why are children prone to foreign bodies?

Developmental issues, supervisory issues, and innate curiosity all play a role. The 1- to 4-year-old age group is particularly predisposed. This age group is notorious for nasal foreign bodies, aspirated foreign bodies, and impacted, nonfood esophageal foreign bodies. Developmental and behavioral health issues may play a role in the older child and adolescent.

3. What are common symptoms of an impacted esophageal foreign body?

About half of children with an impacted esophageal foreign body will be asymptomatic on presentation. Foreign body sensation, gagging, vomiting, drooling, dysphagia, or refusal to eat or drink may occur. Respiratory symptoms, such as choking and coughing, may occur at the time of ingestion. Later, respiratory symptoms such as wheezing or stridor occur occasionally owing to indirect effects on the airway.

Little DC, Shah SR, St. Peter SD, et al: Esophageal foreign bodies in the pediatric population: Our first 500 cases. J Pediatr Surg 2006;41:914-918.

Louie JP, Alpern ER, Windreich RM: Witnessed and unwitnessed esophageal foreign bodies in children. Pediatr Emerg Care 2005;21:582-585.

4. Where do objects impact in the esophagus?

Impaction at the level of the cricopharyngeus muscle is most common, followed by the lower esophageal sphincter and, less likely, the level of the aortic arch. Patients with a history of congenital esophageal abnormalities or acquired strictures will have objects (usually meaty foodstuffs) impact at the area of anatomic narrowing.

Little DC, Shah SR, St Peter SD, et al: Esophageal foreign bodies in the pediatric population: Our first 500 cases. J Pediatr Surg 2006;41:914-918.

Louie JP, Alpern ER, Windreich RM: Witnessed and unwitnessed esophageal foreign bodies in children. Pediatr Emerg Care 2005;21:582-585.

Waltzman ML, Baskin M, Wypij D, et al: A randomized clinical trial of the management of esophageal coins in children. Pediatrics 2005;116:614-619.

5. What is the most common esophageal foreign body?

Coins are the most common, with pennies being the most common coin.

Little DC, Shah SR, St Peter SD, et al: Esophageal foreign bodies in the pediatric population: Our first 500 cases. J Pediatr Surg 2006;41:914-918.

6. Is there a role for "expectant observation" with an impacted esophageal coin?

Spontaneous passage of impacted esophageal foreign bodies, and particularly coins, is more likely from the lower esophageal sphincter but can occur from any typical (and nonpathologic) level of impaction (Fig. 14-1). Spontaneous passage rates are about 30%. It is reasonable to wait 24 hours from the time of ingestion for spontaneous passage if the child is asymptomatic, while allowing them to drink and eat. Obtain follow-up radiographs to confirm passage from the esophagus. Patients should return to the emergency department (ED) if they develop symptoms of esophageal obstruction or respiratory distress.

Sharieff GQ, Brousseau TJ, Bradshaw JA, Shad JA: Acute esophageal coin ingestions: Is immediate removal necessary? Pediatr Radiol 2003;33(12):859-863.

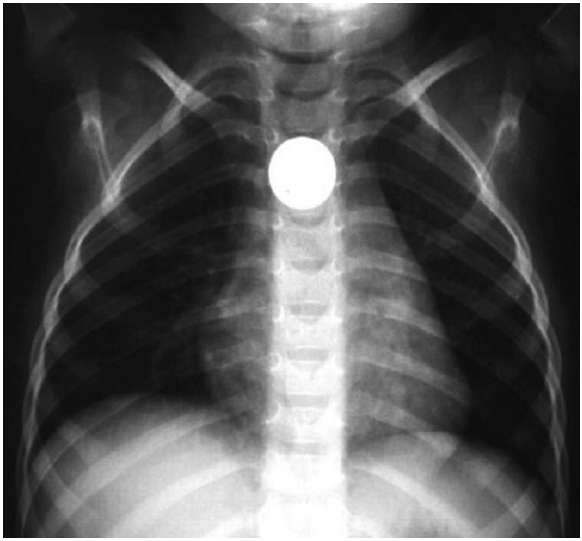

Figure 14-1. This radiograph shows the typical location of an impacted esophageal coin at the thoracic inlet.

Soprano JV, Fleisher GR, Mandl KD: The spontaneous passage of esophageal coins in children. Arch Pediatr Adolesc Med 1999;153:1073.
Waltzman ML: Management of esophageal coins. Pediatr Emerg Care 2006;22:367-369.
Waltzman ML, Baskin M, Wypij D, et al: A randomized clinical trial of the management of esophageal coins in children. Pediatrics 2005;116:614-619.

7. Can passage of a coin in the esophagus be "encouraged" with medication or otherwise?

Studies using intravenous (IV) glucagon have not shown efficacy in promoting passage above baseline rates. For an asymptomatic child with an impacted coin of short duration (less than a day), some clinicians offer a cracker, a small piece of bread, or soda to promote passage, recognizing the potential impact on NPO (nothing by mouth) status if passage fails.

Gilger MA, Jain AK, McOmber ME: Foreign bodies of the esophagus and gastrointestinal tract in children. UpToDate, July 2013. Available from http://www.uptodate.com.

8. How should impacted esophageal foreign bodies be removed?

Removal methods for impacted esophageal foreign bodies vary by local or regional practice. Flexible endoscopy or rigid endoscopy is the preferred method for many. This may be used for all types of foreign bodies. For coins, other removal methods include use of a bougie dilator to push the coin into the stomach, a Foley catheter with fluoroscopic guidance to either advance the coin or pull it into the oropharynx, and the penny pincher technique, in which endoscopic forceps within a red rubber catheter are directed to grasp the coin under fluoroscopic guidance.

Arms JL, Mackenberg-Mohn MD, Bowen MV, et al: Safety and efficacy of a protocol using bougienage or endoscopy for the management of coins acutely lodged in the esophagus: A large case series. Ann Emerg Med 2008;51:367-372.
Gilger MA, Jain AK, McOmber ME: Foreign bodies of the esophagus and gastrointestinal tract in children. UpToDate, July 2013. Available from http://www.uptodate.com.
Little DC, Shah SR, St. Peter SD, et al: Esophageal foreign bodies in the pediatric population: Our first 500 cases. J Pediatr Surg 2006;41:914-918.
Soprano JV, Mandl KD: Four strategies for the management of esophageal coins in children. Pediatrics 2000;105:e5.

9. How can one determine if a round, metallic impacted esophageal foreign body is a coin or a disc battery?

Disc ("button") batteries will display a double ring near the outer border (Fig. 14-2).

Sinclair K, Hill ID: Button and cylindrical battery ingestion. UpToDate, Nov. 2012. Available from http://www.uptodate.com.

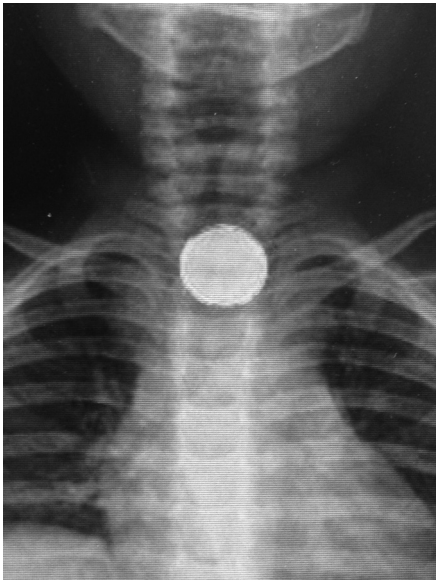

Figure 14-2. Disc battery in esophagus demonstrates double ring sign. Patient had a few days of refusal to eat and then respiratory symptoms prompting chest radiograph. Esophagogram demonstrated esophageal injury and evidence of leak into airway. Bronchoscopy visualized airway swelling and inflammation only.

10. How do batteries cause injury so quickly when impacted?

The moist environment of the esophagus in combination with the flow of electric current causes hydrolysis and hydroxide buildup and local corrosive injury. Leaking of battery contents occasionally plays a role after exposure to stomach acid. Arrange for prompt removal of disc batteries lodged in the esophagus; do not delay removal while waiting for spontaneous passage.

Sinclair K, Hill ID: Button and cylindrical battery ingestion. UpToDate, Nov. 2012. Available from http://www.uptodate.com.

11. Do foreign bodies beyond the esophagus pose a health risk?

Most ingested foreign bodies are blunt and relatively small, so passage through the remainder of the gastrointestinal (GI) tract is uneventful (Fig. 14-3). Ingestion of multiple magnets is an important exception to that rule, as they may cause serious complications in the intestines. Small (<5 cm) sharp objects usually pose minimal risk. Disc batteries typically pass without complication once beyond the esophagus. After a disc battery ingestion, follow-up radiographs are recommended (a) if the patient develops symptoms, (b) for children less than 6 years old with a 15 mm or larger battery, and (c) weekly if the battery is not seen in the stool.

Hussain SZ, Bousvaros A, Gilger M, et al: Management of ingested magnets in children. J Pediatr Gastroenterol Nutr 2012;55:239.

Litovitz T, Whitaker N, Clark L, et al: Emerging battery-ingestion hazard: Clinical implications. Pediatrics 2010;125:1168-1177.

Sinclair K, Hill ID: Button and cylindrical battery ingestion. UpToDate, Nov. 2012. Available from http://www.uptodate.com.

U.S. Consumer Product Safety Commission: CPSC warns high powered magnets and children make a deadly mix. Nov. 10, 2011. Available at http://www.cpsc.gov/Newsroom/News-Releases/2012/CPSC-Warns-High-Powered-Magnets-and-Children-Make-a-Deadly-Mix/.

12. Is there a hotline to help with management of ingested batteries?

Call the National Battery Ingestion Hotline at (202) 625-3333, or call your local poison control center at (800) 222-1222.

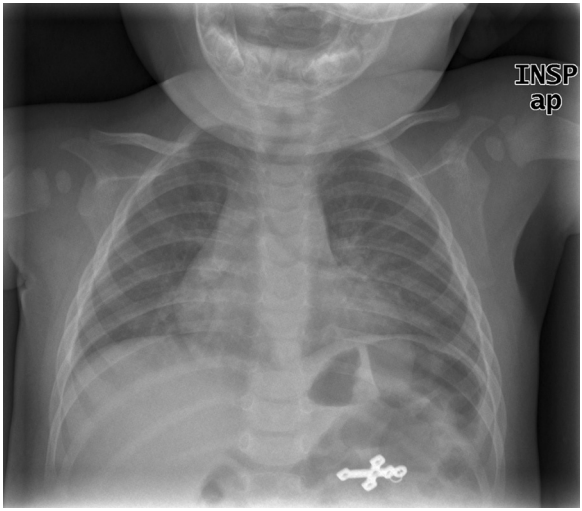

Figure 14-3. Ingested foreign body in stomach. The foreign body passed through the remainder of the gastrointestinal tract without problems.

13. Should sharp foreign objects be removed from the stomach?

Most small, sharp foreign bodies (e.g., small nails, pins, tacks, staples) are well tolerated, though some authors suggest removal if discovered in the stomach. Objects that are longer than 5 cm may not negotiate the tighter bends in the GI tract. Consult a specialist to remove these larger sharp objects from the stomach.

Gilger MA, Jain AK, McOmber ME: Foreign bodies of the esophagus and gastrointestinal tract in children. UpToDate, July 2013. Available from http://www.uptodate.com.

14. Do some blunt objects require removal from the stomach?

Yes, especially for ingestion of multiple magnets. Consider emergency removal of multiple magnets in the stomach, because they may travel at different rates through the GI tract and then magnetically attach to each other with bowel walls between. Ingestion of multiple magnets has resulted in serious complications including bowel perforation, volvulus, and death. Also, arrange for removal of large batteries ($\geq$15 mm) in young children (<6 years) that have not passed from the stomach in 48 hours or quarters that fail to pass from the stomach within 3 to 4 weeks.

Hussain SZ, Bousvaros A, Gilger M, et al: Management of ingested magnets in children. J Pediatr Gastroenterol Nutr 2012;55:239.

Litovitz T, Whitaker N, Clark L, et al: Emerging battery-ingestion hazard: Clinical implications. Pediatrics 2010;125:1168-1177.

U.S. Consumer Product Safety Commission: CPSC warns high powered magnets and children make a deadly mix. Nov. 10, 2011. Available at http://www.cpsc.gov/Newsroom/News-Releases/2012/CPSC-Warns-High-Powered-Magnets-and-Children-Make-a-Deadly-Mix/.

15. How often is the classic clinical triad of cough, wheezing, and unilateral decreased breath sounds present with a foreign body aspiration?

At most, one third of children with an aspirated foreign body have this classic triad. The longer the foreign body has been in place, the more likely this triad will be present and the more prominent the symptoms. Children with foreign body aspirations commonly present to the ED without symptoms, and about 20% have normal physical examinations.

Even L, Heno N, Talmon Y, et al: Diagnostic evaluation of foreign body aspiration in children: A prospective study. J Pediatr Surg 2005;40:1122-1127.

Hui H, Na L, Zhijun C, et al: Therapeutic experience from 1428 patients with pediatric tracheobronchial foreign body. J Pediatr Surg 2008;43:718-721.

16. What foreign bodies do children usually aspirate?

Children aspirate a large variety of materials, but foodstuffs predominate. Nuts (especially peanuts) are the most common, followed by apples, carrots, seeds, and popcorn. Radiopaque objects account for less than 15% of the cases. Given the creativity of the pediatric population, they may aspirate lint, chalk, crayon pieces, pen springs or caps, shell casings, toy parts, gum wrappers, earrings, bracelets, etc.

Black RE, Johnson DG, Matlak ME: Bronchoscopic removal of aspirated foreign bodies in children. J Pediatr Surg 1994;29:682-684.

Hui H, Na L, Zhijun C, et al: Therapeutic experience from 1428 patients with pediatric tracheobronchial foreign body. J Pediatr Surg 2008;43:718-721.

17. What are the signs and symptoms of an aspirated foreign body?

- Respiratory distress
- Decreased breath sounds
- Stridor
- Wheezing
- Crackles
- Cough
- Foreign body sensation
- No signs or symptoms

Even L, Heno N, Talmon Y, et al: Diagnostic evaluation of foreign body aspiration in children: A prospective study. J Pediatr Surg 2005;40:1122-1127.

Hui H, Na L, Zhijun C, et al: Therapeutic experience from 1428 patients with pediatric tracheobronchial foreign body. J Pediatr Surg 2008;43:718-721.

18. Do the signs of an aspirated foreign body differ from other respiratory conditions?

Unfortunately, there is considerable overlap in both signs and symptoms with very common conditions such as upper respiratory infection, bronchiolitis, asthma, and pneumonia. Always inquire about the possibility of aspiration or a recent choking episode when confronted with a child with acute or chronic respiratory symptoms. Ask specifically about high-risk foods (e.g., nuts, carrots, apples, seeds, popcorn). Studies demonstrate that a history of choking is present up to 85% of the time in cases of proven tracheobronchial foreign bodies.

19. How good is a plain film at screening for an aspirated foreign body?

In the uncommon instances of an aspirated radiopaque foreign body, the radiograph is an excellent test. For a radiolucent foreign body, the chest radiograph is a poor screen. Inspiratory and expiratory views improve sensitivity and may demonstrate air trapping as indirect evidence of an aspirated foreign body (Fig. 14-4). Overall sensitivity of these views is about 50%, and

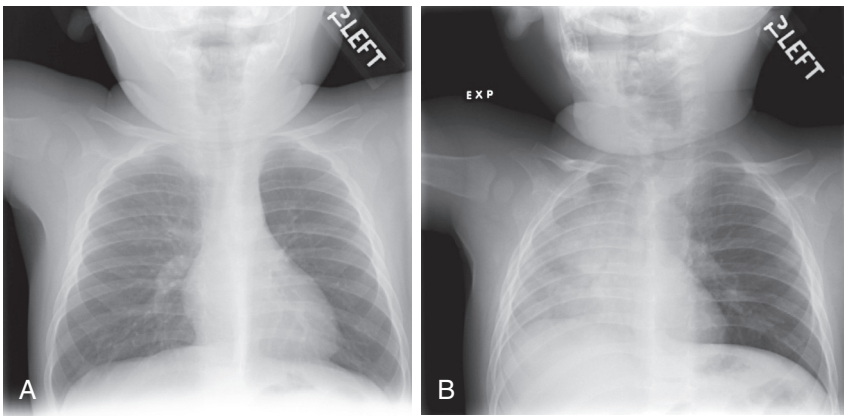

Figure 14-4. **A,** Inspiratory chest radiograph in a child who choked on popcorn. **B,** Expiratory chest radiograph of same child seen in **A** who choked on popcorn. The left lung fails to deflate with expiration, providing indirect evidence of retained foreign body.

unfortunately radiographic findings overlap with other common pediatric respiratory conditions. Right and left lateral decubitus views (Fig. 14-5A and B) also provide an alternative, especially in the younger child. Both views are performed during inspiration, and normally the dependent (downside) lung will be relatively deflated. Failure of the dependent lung to show deflation suggests a retained foreign body (Fig. 14-5B). Given the overall lack of sensitivity of plain films, when the history suggests an aspiration event, consider bronchoscopy (Fig. 14-6).

Hui H, Na L, Zhijun C, et al: Therapeutic experience from 1428 patients with pediatric tracheobronchial foreign body. J Pediatr Surg 2008;43:718-721.

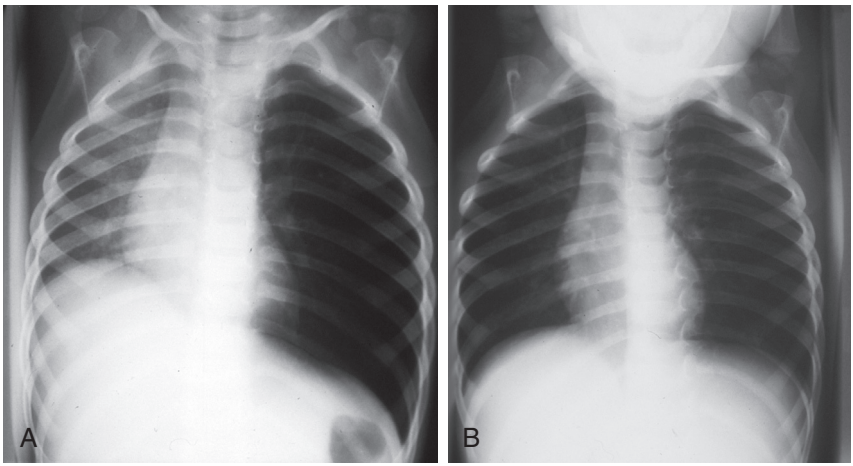

Figure 14-5. **A,** Right lateral decubitus chest radiograph. Normal relative deflation of dependent lung (*right*). **B,** Left lateral decubitus chest radiograph. Left lung does not show typical deflation of dependent lung. Foreign body is on left side.

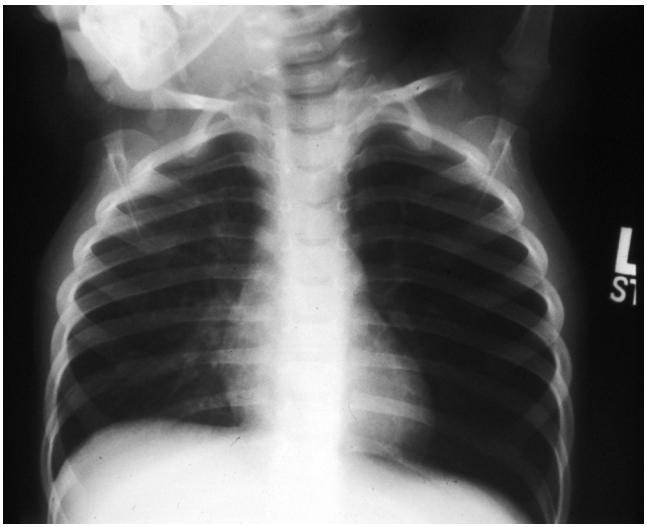

Figure 14-6. Inspiratory chest radiograph of the same patient shown in Figure 14-5A and B.

20. Are there other imaging alternatives to detect aspirated foreign bodies?

Fluoroscopy may provide additional utility, demonstrating an area of air trapping not seen on plain images. Computed tomography (CT) scans have been shown to demonstrate aspirated foreign bodies that are not evident on plain films. The overall role of CT scans in the evaluation is uncertain.

Black RE, Johnson DG, Matlak ME: Bronchoscopic removal of aspirated foreign bodies in children. J Pediatr Surg 1994;29:682-684.

Even L, Heno N, Talmon Y, et al: Diagnostic evaluation of foreign body aspiration in children: A prospective study. J Pediatr Surg 2005;40:1122-1127.

Hui H, Na L, Zhijun C, et al: Therapeutic experience from 1428 patients with pediatric tracheobronchial foreign body. J Pediatr Surg 2008;43:718-721.

21. A teenage boy presents to the ED after a choking episode while drinking beer. He reports that he removed the pull tab of a beer can and placed it inside the can. He believes he may have then swallowed or aspirated the pull tab while gulping beer. What study is best to determine the location of the pull tab?

CT of the chest may be needed in this case. Plain radiographs generally do not show an aluminum pull tab of a beer or soda can.

Bradburn DM, Carr HF, Renwick I: Radiographs and aluminum: A pitfall for the unwary. BMJ 1994;308:1226.

22. What is the appropriate management of a child with an aspirated foreign body?

Make the patient NPO, and admit him to a hospital where appropriate expertise is available for removal of the foreign body.

23. What foreign bodies end up in the ear or nose?

Just about anything that will fit in the nose could end up there, including beads, beans, peas, corn kernels, raisins, small toys or toy parts, pebbles, and erasers. Foreign bodies in the ear include the same, as well as jewelry, and one additional item: bugs (usually roaches).

24. Can a "kiss" help get a foreign body out of the nose?

Yes. In the ED, instruct the parent to give the child a quick, well-sealed, mouth-to-mouth breath (short and sharp puff) while occluding the unaffected nostril to help advance the foreign body out of the nose. Repeat this up to five times if necessary. The literature refers to this as a "mother's kiss" or "parent's kiss", and the technique is reportedly quite successful. Other low-tech methods for removal of a foreign body include encouraging the child (if old enough) to blow out through the nose while the unaffected nostril is occluded. This may advance the foreign body—but the family has probably tried this at home.

Taylor C, Acheson J, Coats TJ: Nasal foreign bodies in children: Kissing it better. Emerg Med J 2010;27:712-713.

25. What is the best method for nasal foreign body removal?

There is no best method, because the nature of the foreign body, skill of the provider, and available equipment all play a role. One can use a bag-mask apparatus over the mouth: While occluding the uninvolved nostril, squeeze the bag-mask to increase the pressure and expel the foreign body from the nose, similar to the kiss technique described earlier. Use of a vasoconstrictor may enhance the success of removal methods. Removal with alligator forceps or rolling the object out of the nostril with an ear curette under direct visualization is another alternative. Some objects are hard to grasp or difficult to get "behind," and the mucus-laden nasal environment can hinder the process. One can also pass a small Foley catheter beyond the foreign body, inflate the balloon (test prior to using to determine the size desired), and pull the catheter forward (out), bringing the foreign body with it. There is also a commercially available extractor with a balloon designed for this purpose.

Kiger JR, Brenkert TE, Losek JD: Nasal foreign body removal in children. Pediatr Emerg Care 2008;24: 785-789.

26. What are the clinical pearls regarding removal of foreign bodies from the ear?

Use of alligator forceps under direct visualization is effective to remove foreign bodies from the ear canal. Also, irrigation is a useful technique, but do not irrigate if the foreign object could expand with liquid (e.g., peas, beans). Ear foreign body removal is a more delicate procedure (the inner portion of the external canal is very sensitive) than removal of nasal foreign bodies, so sedation is frequently needed. For a live bug moving in the ear canal, it

is best to immediately drown the insect with something inert like mineral oil and then remove it. Or try spraying 1% lidocaine into the canal to hasten the insect's exit.

27. Should a child be sedated for removal of a foreign body from the ear or nose?

This choice depends on your level of comfort, but in general, nasal foreign bodies can be removed without sedation. However, the inner one third of the external auditory canal is exquisitely sensitive, and sedation may be needed for difficult foreign bodies in the ear.

28. Which patients with an ear foreign body should be referred?

Refer patients to a specialist if there is swelling or a macerated canal (from previous removal attempts), a very tight foreign body, a foreign body against the tympanic membrane, concern for damage to the tympanic membrane, or a disc battery in the ear canal or when adequate equipment and lighting are not available.

Key Points: Foreign Bodies

1. Remove disc batteries impacted in the esophagus emergently.
2. Ingestion of multiple magnets poses significant risk of intestinal complications.
3. Many children with impacted esophageal foreign bodies are asymptomatic.
4. Signs and symptoms often fail to diagnose an aspirated foreign body; place increased emphasis on a history of choking.
5. Plain radiographs are a poor screening test for an aspirated foreign body; when the history is suggestive, further studies or bronchoscopy is indicated.

CHAPTER 15

HEADACHE

Robert D. Schremmer and Joan E. Giovanni

1. Do children get migraine headaches?

Yes. Migraine is one of the most common causes of headache in children. The reported prevalence is 1% to 3% in children 3 to 7 years of age, and 8% to 23% in adolescents. The variation in prevalence between studies is due primarily to differences in diagnostic criteria used and the age range of children studied. Males tend to have more migraines in younger childhood, with females having more frequent headaches after puberty.

Hershey AD: Current approaches to the diagnosis and management of paediatric migraine, Lancet Neurol 9:190–204, 2010.

2. Are the International Headache Society (IHS) criteria for the diagnosis of migraine headache applicable to children?

The IHS criteria were developed primarily for the diagnosis of headache in adults. The second edition of the International Classification of Headache Disorders (ICHD-II), published in 2004 (revised in 2005), reduces the duration requirements of the headache from 4 to 72 hours in adults to 1 to 72 hours in children under the age of 15 years. It also acknowledges that photophobia and phonophobia must be inferred from behavior in young children. The IHS guidelines do not account for other possible symptomatic differences, including intensity and location of pain. Pediatric migraines are often bilateral. A higher incidence of either photophobia or phonophobia alone also occurs, especially in younger children.

3. List the criteria for pediatric migraine *without* aura.

A. At least five attacks fulfilling criteria B–D
B. Duration of 1 to 72 hours (untreated or unsuccessfully treated)
C. Headache has at least two of the following:
- Bilateral (frontal/temporal) or unilateral location
- Pulsatile quality
- Headache intensity moderate or severe
- Aggravation by or causing avoidance of routine physical activity

D. During headache at least one of the following occurs:
- Nausea and/or vomiting
- Photophobia and/or phonophobia (which may be inferred from behavior)

E. Not attributed to another disorder

Headache Classification Subcommittee of the International Headache Society: The International Classification of Headache Disorders, 2nd ed. Cephalalgia 2004;24(Suppl 1):9-160.

4. List the criteria for pediatric migraine headache *with* aura.

A. At least two attacks fulfilling criteria B–D
B. At least one of the following:
- Fully reversible visual symptoms including positive features (e.g., flickering lights, spots, or lines) and/or negative features (i.e., loss of vision)
- Fully reversible sensory symptoms including positive features (i.e., pins and needles) and/or negative features (i.e., numbness)
- Fully reversible dysphasic speech disturbance

C. At least two of the following:
- Homonymous visual symptoms and/or unilateral sensory symptoms
- At least one aura symptom develops gradually over 5 minutes or longer and/or different aura symptoms occur in succession over 5 minutes or longer
- Each symptom lasts 5 minutes or longer and less than 60 minutes

D. Headache fulfilling criteria B–D for migraine that begins during aura or follows aura within 60 minutes
E. Not attributed to another disorder

Headache Classification Subcommittee of the International Headache Society: The International Classification of Headache Disorders, 2nd ed. Cephalalgia 2004;24(Suppl 1):9-160.

5. List some common triggers for migraine in children.

Stress, lack of sleep, changes in normal eating patterns, weather changes, and some medications (including asthma treatments and stimulants) are common triggers. Certain foods containing nitrates, caffeine, tyramine, glutamate, or salt are potential dietary triggers. Eye strain, cold foods, and high altitude are less common triggers.

6. Do children get tension-type headaches (TTHs)?

Yes. TTHs are very common in children. Their prevalence ranges from 10% to 25%, but one study reported a prevalence of 73% in Brazilian children and adolescents ages 10 to 18 years. The ICHD-II lists three subtypes of TTH: (1) infrequent episodic (headaches on <1 day per month), (2) frequent episodic (headaches on 1-14 days per month), and (3) chronic (headaches on ≥ 15 days per month). These headaches are characterized by mild to moderate pressing or tightening pain that is nonpulsatile and typically last 4 to 6 hours. The pain is usually bilateral and not aggravated by routine activity. Nausea and vomiting do not occur, but phonophobia or photophobia may be present. Clinical features are the same in children and adults. It is common for children to continue with normal activities despite the headache.

Anttila P: Tension-type headache in children and adolescents. Curr Pain Headache Rep 2004;8:500-504.
Parisi P, Papetti L, Spalice A, et al: Tension-type headache in pediatric age. Acta Paediatr 2011;100:491-495.

7. Do psychological stress factors contribute to TTH in children?

Anxiety and stress factors are often present in children and adolescents with TTH. Divorced parents, fewer peer relations, and unhappy family atmosphere are all associated with childhood TTH. Children with episodic TTH are more likely to report other somatic complaints as well as family problems than children who do not suffer from headaches. TTHs often start in the afternoon while at school and may be absent during extended school vacations. Pediatric patients with chronic diseases and those who experience stressful family events have an increased risk for chronic TTH. In fact, over 50% of children with chronic TTH have had predisposing physical or emotional stress factors.

8. Can children suffer from both migraines and TTHs?

Distinguishing between migraines and TTHs in children can be challenging because of frequent overlapping symptoms and the inherent difficulties with history and physical examination in younger children. Some researchers have postulated that episodic TTH and migraine may fall along the same continuum of disorder. Long-term outpatient follow-up studies have also reported that TTH may develop into migraine over time and vice versa. However, there seems to be a smaller genetic effect on TTH than migraine in other studies, which may suggest they are distinct conditions and not on a continuum.

Anttila P: Tension-type headache in childhood and adolescence. Lancet 2006;5:268-274.

9. How will I recognize the child with a brain tumor headache?

Brain tumors are an uncommon but feared cause of headaches in children. Certain features help to distinguish a headache that is caused by a tumor or other intracranial mass (Fig. 15-1):

- Location: occipital headaches or headaches localized to the nape of the neck
- Timing: headaches that awaken the child from sleep or that are worse in the morning and improve on rising
- Positioning: headaches that worsen with body movement, especially bending forward, and improve with change in position
- Change over time: headaches that change in frequency, severity, or other characteristics
- Worsening with sneezing, coughing, or straining
- Headaches associated with blurred vision
- Headaches associated with neurologic signs or symptoms
- History of a syndrome associated with tumors (neurofibromatosis, tuberous sclerosis)

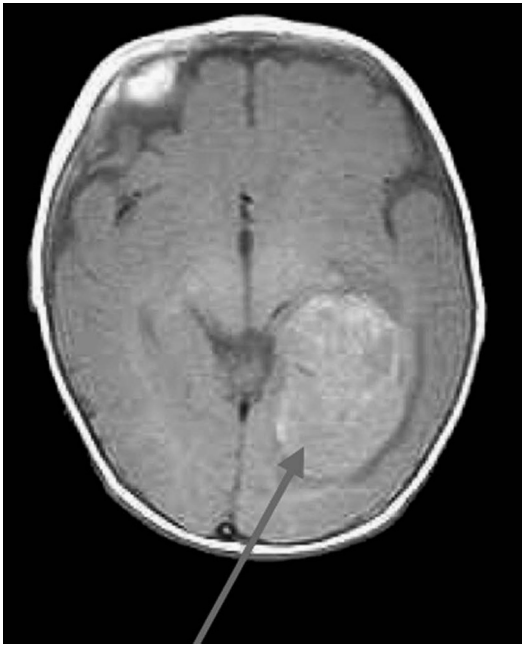

Figure 15-1. Computed tomography scan showing brain tumor (*arrow*).

Blume HK: Pediatric headache: a review, Pediatr Rev 33:562–576, 2012.
Honig PJ, Charney EB: Children with brain tumor headaches. Am J Dis Child 1982;136:121-124.

Key Points: Brain Tumor Headaches

1. Usually occipital
2. Worse with coughing, sneezing, bending forward
3. Worse in the early morning
4. Change in character over time
5. Accompanied by neurologic signs or symptoms

10. How common are chronic daily headaches (CDHs) in children?

CDHs encompass a group of headaches not specifically described by the ICHD-II, although three of the four types in a proposed classification are included in the 2004 IHS revisions:

- Transformed migraine: daily headache with average duration of 4 hours for more than a month, and a history of increased headache frequency with decreased migraine symptoms
- Chronic TTH (ICHD-II 2.3): history of episodic TTH in the past with evolution to daily headaches
- New daily persistent headache (ICHD-II 4.8): acute onset of headache on more than 15 days per month lasting at least 4 hours and without history of migraine or TTHs in the past
- Hemicrania continua (ICHD-II 4.7): unilateral, continuous headache of moderate severity known for its absolute relief with indomethacin

The prevalence of CDH varies between studies, affecting 1% to 6% of children and adolescents. CDH occurs more commonly in females, who have a prevalence rate two to three times higher than in males. Psychiatric comorbid conditions including sleep, anxiety,

and mood disorders and other pain syndromes is high, which increases the difficulty of treatment. Transformed migraine is the most common type.

Seshia SS, Wang SJ, Abu-Arafeh I, et al: Chronic daily headache in children and adolescents: A multifaceted syndrome. Can J Neurol Sci 2010;37:769-778.

11. Describe the most important aspects of the physical examination for children with headaches.

Pay special attention to vital signs such as heart rate and blood pressure, weight and height, and, if appropriate, head circumference. The neurologic examination should focus on mental status, speech, strength, sensation, gait, coordination, cranial nerve function, and funduscopic examination. Look for signs of trauma, and examine the skin for evidence of a neurocutaneous disease. Multiple café-au-lait lesions suggest neurofibromatosis and possible intracranial tumor.

Özge A, Termine C, Antonaci F, et al: Overview of diagnosis and management of pediatric headache. Part I: diagnosis, J Headache Pain 12:13–23, 2011.

12. What is the role of emergent neuroimaging in children with headache?

Although most headaches in children are benign, the use of neuroimaging for the evaluation of headache in the emergency department has increased dramatically in the past 15 years. Neuroimaging is useful in identifying children with space-occupying or surgical lesions. An important concern is brain tumor, which, although rare, represents the largest group of solid neoplasms in children. Indications for neuroimaging are as follows:

- Headache associated with abnormal neurologic findings, especially papilledema, nystagmus, mental status changes, or gait or motor disturbances
- Seizures
- Persistent headaches not associated with a family history of migraine
- Headaches that awaken a child from sleep or are present immediately on awakening
- Persistent headache associated with episodes of confusion, disorientation, or vomiting
- New severe headache or worsening of a previously stable headache
- Family history or medical history of disorders predisposing one to central nervous system (CNS) lesions (such as neurofibromatosis)
- Clinical or laboratory findings suggestive of CNS involvement

A practice parameter on the evaluation of pediatric patients with recurrent headaches written by the American Academy of Neurology discusses the issue of neuroimaging for recurrent headache. The parameter concludes that routine neuroimaging should be avoided in children with a normal neurologic examination. It should be considered, however, in patients with headache who have a history of seizures, an abnormal neurologic examination, a recent change in type of headache experienced, or characteristics implying neurologic dysfunction.

Alexiou GA, Argyropoulou MI: Neuroimaging in childhood headache: A systematic review. Pediatr Radiol 2013;43:777-784.

Gilbert JW, Johnson KM, Larkin GL, Moore CL: Atraumatic headache in US emergency departments: Recent trends in CT/MRI utilization and factors associated with severe intracranial pathology. Emerg Med J 2012;29:576-581.

13. How should headache pain be managed acutely?

Evidence-based guidelines similar to those published for adults for the treatment of headache do not exist. Treatment recommendations are generally based on experience, tradition, or extrapolation of information from adult studies. Most headaches in children respond to hydration and over-the-counter analgesics such as acetaminophen or nonsteroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen. Treatment for significant headaches should be initiated early, as it is more likely to be effective during this time.

14. What lifestyle changes can be made to prevent headaches?

Adequate sleep, avoiding use of electronics in the bedroom at night, a regular bedtime schedule, routine exercise, and eating well-balanced, healthy meals (including breakfast) can help reduce the frequency of headaches. Adequate hydration is also essential in the prevention of headaches. Water should be emphasized over juices and soda, and caffeine should be limited to less than three servings per week. Identifying, avoiding, and managing triggers and stressors are also important.

15. What treatments are available for migraine headaches?

Agents that have been well studied in the pediatric literature include ibuprofen, acetaminophen, and selected triptans. Many children with migraines respond to over-the-counter analgesics and rest in a dark, quiet room. Triptans in the nasal and oral preparations have demonstrated safety and efficacy in the adolescent population. Antiemetics such as prochlorperazine, metoclopramide, and ondansetron are useful adjuncts in the treatment of migraine but have the potential for causing dystonic reactions. Such dystonic reactions are rare (especially with ondansetron) and can be minimized by the concurrent administration of diphenhydramine. Ergotamines administered either nasally or intravenously are another treatment option but need to be administered in small, gradually increasing amounts and have potential for nausea and rebound headaches. Patients with persistent migraine headaches despite initiation of treatment in the acute care setting may require hospital admission for continued therapy. Other agents such as cyproheptadine, tricyclic antidepressants, antiepileptics, and antihypertensives have been used as preventive medications for pediatric migraine. Start these only after consultation with a neurologist or headache specialist. Avoid narcotics or other combination agents that can cause rebound headache.

Lewis DW: Pediatric migraine. Neurol Clin 2009;27:481-501.

Lewis KS: Pediatric headache. Semin Pediatr Neurol 2010;17:224-229.

Patel A, Mittal S, Manchanda S, Puliyel JM: Ondansetron-induced dystonia, hypoglycemia, and seizures in a child. Ann Pharmacother 2011;45(1):e7.

16. What treatments are available for TTH?

The management of TTH must be multidisciplinary and viewed from a biopsychosocial perspective. Treat infrequent episodic TTH with over-the-counter analgesics. These headaches do not generally come to the attention of the emergency department. For patients with frequent episodic or chronic TTH, pharmacologic or nonpharmacologic therapies may be effective, though few studies regarding medication efficacy can be found in the literature. Amitriptyline may be effective for prophylaxis, and valproic acid was shown to be better than placebo for chronic TTHs in adolescents and adults, but the effect was greater for chronic migraine. Nonpharmacologic therapies include biofeedback, relaxation techniques, and cognitive-behavioral therapy. A headache diary can also be useful to identify inciting factors that may be overcome, especially poor sleep hygiene.

Parisi P, Papetti L, Spalice A, et al: Tension-type headache in pediatric age. Acta Paediatr 2011;100:491-495.

Yurekli VA, Akhan G, Kutluhan S, et al: The effect of sodium valproate on chronic daily headache and its subgroups. J Headache Pain 2008;9:37-41.

17. How do I evaluate a child with a persistent headache several days after a head injury?

A thorough history of the injury is the first step in the evaluation of a patient with posttraumatic headache. The history should include mechanism of injury, level of consciousness, presence of amnesia, other neurologic symptoms, duration of all symptoms, level of activity since the injury, previous evaluation including imaging results, and previous history of migraine in the patient or family. Accompanying symptoms such as sleep disturbance, behavioral changes, dizziness, and decreased concentration may be collectively known as postconcussion syndrome. The next step is a complete head-to-toe physical examination including a thorough neurologic evaluation. Routine neuroimaging is not indicated, but should be considered in children with focal neurologic signs, persistent vomiting, decreased mental status, or seizures.

Wilson M-C, Krolczyk SJ: Pediatric post-traumatic headache. Curr Pain Headache Rep 2006;10:387-390.

18. Describe the management of posttraumatic headaches.

There are no controlled trials describing the treatment of posttraumatic headache in children. A study published in 2013 showed that greater than 90% of patients with posttraumatic headaches experience resolution of symptoms within 3 months. NSAIDs may be effective agents for the treatment of migraine and TTHs and serve as a good starting point for the treatment of posttraumatic headaches. Use caution to prevent analgesia overuse headaches. Limit medication use to no more than 2 or 3 days each week. Triptans may also be effective in the treatment of headaches that do not respond to anti-inflammatory therapy. A recent

case report showed improvement in severe intractable posttraumatic headaches with a tapered short course of low-dose corticosteroids. Agents used for migraine prophylaxis, such as amitriptyline, propranolol, topiramate, and valproic acid, may also be options, although they have not been studied for this indication. Opiates are generally not effective. Chronic posttraumatic headaches are best managed with a multidisciplinary approach that includes medications, physical therapy, biofeedback, and psychological and behavioral interventions. Strongly consider referral to a headache specialist.

Bramley H, Melinosky C, Silvis M, Ross S: Pediatric posttraumatic headache: Two cases using steroids as abortive therapy. Pediatr Emerg Care 2012;28:1081-1084.

Kuczynski A, Crawford S, Bodell L, et al: Characteristics of post-traumatic headaches in children following mild traumatic brain injury and their response to treatment: A prospective cohort. Dev Med Child Neurol 2013;55:636-641.

Lew HL, Lin P-H, Fuh J-L, et al: Characteristics and treatment of headache after traumatic brain injury: A focused review. Am J Phys Med Rehabil 2006;85:619-627.

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CHAPTER 16

HEMATURIA AND DYSURIA

Sandra H. Schwab and Elizabeth R. Alpern

1. What is the definition of hematuria?

Hematuria is the presence of red blood cells (RBCs) in the urine. Hematuria can be categorized as gross or microscopic. Gross hematuria is defined as pink to brown discoloration of the urine with confirmation of RBCs by microscopy. Microscopic hematuria is normal-appearing urine that, when centrifuged, has more than three RBCs per high-power field (HPF) on microscopy.

2. What else, besides RBCs, can cause urine to appear red or brown?

Reddish or brownish discoloration of the urine from some compound other than blood is common. Myoglobinuria and porphyrinuria present with red or tea-colored urine. Many dyes, drugs, pigments, and metabolites also discolor the urine. Common substances that turn urine red include red food dye, beets, blackberries, bile pigments, rifampin, phenazopyridine (Pyridium), ibuprofen, salicylates, and deferoxamine. Infants may have precipitation of urate crystals that make their urine appear red or orange in the diaper.

Liebelt EA: Hematuria. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 310-314.

Pan CG, Avner ED: Conditions particularly associated with hematuria. In Kleigman RM (ed): Nelson Textbook of Pediatrics, 19th ed. Philadelphia, WB Saunders Elsevier, 2011, pp 1778-1781.

Yap HK, Lau PYW: Hematuria and proteinuria. In Geary DF, Schaefer F (eds): Comprehensive Pediatric Nephrology. Philadelphia, Mosby Elsevier, 2008, pp 179-193.

3. What can cause a "false-positive" urine dipstick?

You may encounter a urine dipstick positive for blood, but upon microscopic examination no RBCs are seen. The dipstick is sensitive to any heme compound and will be positive for intact RBCs as well as hemoglobin from hemolyzed RBCs and myoglobin from rhabdomyolysis. Dehydration, strenuous exercise, and oxidizing compounds, such as bleach or microbial peroxidase, can turn the dipstick spuriously positive. Delayed reading of the dipstick may also produce a false-positive result.

Simerville JA, Maxted WC, Pahira JJ, et al: Urinalysis: A comprehensive review. Am Fam Physician 2005;71(6):153.

4. Is there a way to determine glomerular versus nonglomerular blood in the urine?

Yes. Determination of the origin of blood is important in directing further evaluation and testing. If the RBCs come from glomeruli, the acidic nature of the urine may change hemoglobin to hematin, causing the urine to appear brownish, tea-colored, or cola-colored. Blood from the lower collecting system frequently appears pink or red. Microscopic examination of the urine will give more specific information, including identifying the presence of urine casts or RBC dysmorphology if blood is from the glomeruli (Table 16-1).

Meyers KEC: Evaluation of hematuria in children. Urol Clin North Am 2004;31:559-573.

5. What is the most likely diagnosis of a child presenting to the emergency department (ED) with hematuria?

The causes of gross hematuria and microscopic hematuria are very different. In general, the majority of children who visit an ED for hematuria are diagnosed with urinary tract infections (UTIs) or cystitis. Common causes of gross hematuria include glomerulonephropathies, trauma (with or without underlying anatomic abnormality), nephrolithiasis, hypercalciuria, bleeding disorders, and nutcracker syndrome (left renal vein entrapment). (See Question 6.)

Most children with asymptomatic microscopic hematuria have a benign process that is self-limited.

Even though parents or patients may assume that blood in the diaper or toilet is from urine, it may actually be from several other sources: vaginal bleeding (menses, foreign body, infection, trauma), rectal bleeding (fissure, hemorrhoid, trauma), or urethral prolapse.

Table 16-1. Glomerular Versus Nonglomerular Blood in the Urine

GLOMERULAR	NONGLOMERULAR
Brown or tea-colored urine	Bright red or pink urine
RBC casts	Blood clots
Dysmorphic RBCs	Normal RBC morphologic appearance
Proteinuria	Blood at initiation or termination of urination

RBC, red blood cell.

6. What is nutcracker syndrome?

Nutcracker syndrome refers to compression of the left renal vein between the aorta and the superior mesenteric artery before the renal vein joins the inferior vena cava. Children usually present with unilateral flank pain and intermittent gross or microscopic hematuria. Some experts believe this is an under-recognized cause of hematuria, and diagnosis is increasing with improved diagnostic testing such as ultrasound and magnetic resonance imaging (MRI).

Jodorkovsky R, Milman E: A child with recurrent gross hematuria caused by the nutcracker syndrome: Lessons learned. Clin Pediatr 2012;51(3):291-293.

Waseem W, Upadhyay R, Prosper G: The nutcracker syndrome: An underrecognized cause of hematuria. Eur J Pediatr 2012;171(8):1269-1271.

7. What is the differential diagnosis of hematuria in a child?

The differential diagnosis of hematuria in a child is in Table 16-2.

8. Why are children at more risk than adults for renal injury after blunt trauma?

In children, the kidneys are situated lower, are less protected by the ribs and abdominal wall, and are proportionally larger and more mobile than in adults. Children usually have less protective perirenal fat and musculature. All of these factors make them more susceptible to renal injury.

Raz O, Haifler M, Copel L, et al: Use of adult criteria for slice imaging may limit unnecessary radiation exposure in children presenting with hematuria and blunt abdominal trauma. Urology 2010;77(1):187-190.

9. How would you evaluate a child with blunt abdominal trauma for renal injury?

Children in shock or who are unstable following abdominal trauma require immediate evaluation for significant renal injury. A computed tomography (CT) scan with intravenous (IV) contrast agent is the standard imaging study of choice.

Children with focal examination findings or significant mechanisms of injury (rapid deceleration, direct flank trauma, falls from heights) should also be evaluated further, regardless of the presence of hematuria. Using urinalysis as a screening test for significant injury is helpful, but it should be interpreted with caution. Most experts agree that children with macrohematuria (>50 RBC/HPF on microscopy) or gross hematuria should undergo a CT scan. Many children with microscopic hematuria also undergo a CT scan, but these patients may instead be able to be monitored with reliable follow-up in order to reduce exposure to radiation.

Raz O, Haifler M, Copel L, et al: Use of adult criteria for slice imaging may limit unnecessary radiation exposure in children presenting with hematuria and blunt abdominal trauma. Urology 2010;77(1):187-190.

10. Why is it so important to measure the blood pressure of a child with hematuria?

Hypertension is often found in patients with hematuria, and this combination may indicate a renal emergency. Consider glomerulonephritis, obstructive uropathy, polycystic kidney disease, hemolytic uremic syndrome, systemic lupus erythematosus, and Wilms' tumor. Between 12% and 25% of patients with Wilms' tumor have hematuria at the time of presentation. About 25% have hypertension. A large abdominal mass is likely to be present with Wilms' tumor but may have been undetected by clinicians who saw the child recently.

11. What signs and symptoms associated with hematuria require urgent/emergent evaluation?

Children with hematuria should be evaluated with a full history, family history, and physical examination, which will allow a directed workup of the cause. In general, gross hematuria

Table 16-2. Differential Diagnosis of Hematuria in Children

GLOMERULAR	NONGLOMERULAR
Benign familial hematuria	Chemical cystitis
Cystic renal disease	Coagulopathy or hemoglobinopathy
Glomerulonephritis	Exercise
Henoch-Schönlein purpura	Hypercalciuria
IgA nephropathy	Malignancy
Membranoproliferative GN	Leukemia
Membranous nephropathy	Nephroblastoma
Polyarteritis nodosa	Neuroblastoma
Poststreptococcal GN	Rhabdomyosarcoma
Rapidly progressive GN	Wilms' tumor
Systemic lupus erythematosus	Medications
Wegener granulomatosis	Nutcracker syndrome
Goodpasture's disease	Structural anomalies
Hemolytic uremic syndrome	Trauma
Hereditary nephritis (Alport syndrome)	Urinary tract infection
Interstitial nephritis	Urolithiasis
Renal vein thrombosis	Vascular malformation

GN, glomerulonephritis.
Adapted from Yap HK, Lau PYW: Hematuria and proteinuria. In Geary DF, Schaefer F (eds): Comprehensive Pediatric Nephrology. Philadelphia, Mosby Elsevier, 2008, p 180.

requires emergent evaluation for significant renal trauma, tumor, congenital abnormality, or bleeding disorder. Hematuria accompanied by proteinuria or systemic symptoms such as edema, oliguria, hypertension, or headache may portend glomerular renal disease that requires emergent evaluation. Additional signs and symptoms such as fever, dysuria, flank pain, abdominal pain, rash, recent sore throat, or respiratory illness are important factors that will help lead to a diagnosis. Asymptomatic isolated microscopic hematuria is found at one time or another in 1% to 4% of children and, in most cases, is benign. This is a diagnosis of exclusion, and referral for outpatient evaluation is indicated.

Liebelt EA, Hematuria. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 310-314.
Meyers KEC: Evaluation of hematuria in children. Urol Clin North Am 2004;31:559-573.
Pan CG, Avner ED: Conditions particularly associated with hematuria. In Kleigman RM (ed): Nelson Textbook of Pediatrics, 19th ed. Philadelphia, WB Saunders Elsevier, 2011, pp 1778-1781.
Yap HK, Lau PYW: Hematuria and proteinuria. In Geary DF, Schaefer F (eds): Comprehensive Pediatric Nephrology. Philadelphia, Mosby Elsevier, 2008, pp 179-193.

12. When is a renal and bladder ultrasound indicated in the workup of hematuria?

Ultrasound is a noninvasive test without exposure to radiation, so it should be considered early in the evaluation of gross hematuria or persistent microscopic hematuria. Children who present with gross hematuria in the absence of proteinuria or RBC casts should have an ultrasound to evaluate for malignancy or cystic renal disease. Ultrasound is helpful to rule out anatomic abnormalities, obstruction, and nutcracker syndrome.

13. Which laboratory tests are helpful in the evaluation of hematuria?

All patients with the sign or finding of hematuria should have a urine dipstick and microscopic urinalysis. Depending on the history and physical examination, further blood tests may be

indicated. Hematuria with fever, flank pain, urgency, and dysuria usually indicates a UTI and should be evaluated with a urine Gram stain and culture. Complete blood count, blood urea nitrogen, and serum creatinine may be helpful, except in cases of isolated microscopic hematuria or obvious UTI. If the child has sustained or suspected trauma, liver function tests, amylase, and lipase are also recommended. Prothrombin time and partial thromboplastin time will help diagnose any bleeding disorder. If the clinical picture indicates nephritis, then electrolytes, complement levels (C3 and C4), antistreptolysin O or streptozyme test, antinuclear antibody titer, hepatitis screen, and erythrocyte sedimentation rate may be helpful.

A patient with a positive dipstick without evidence of RBCs on urine microscopic examination should be evaluated with plasma creatine kinase and urinary myoglobin concentration for rhabdomyolysis, and a bilirubin level for signs of hemolysis.

14. Describe the causes of nephrolithiasis in children.

Nephrolithiasis is an increasing problem in children in the United States. Stone formation results from environmental and hereditary factors. Hypercalciuria is the most common cause of pediatric urinary stones and has many causes. Genetic syndromes, such as familial idiopathic hypercalciuria, as well as other diseases such as Bartter syndrome and distal renal tubular acidosis, lead to increased urinary calcium excretion and stone formation. Iatrogenic causes include treatment with loop diuretics and prednisone.

Other causes of stone formation include infection with urease-producing organisms (struvite or "staghorn" calculi); cystinuria; hyperoxaluria; medications such as protease inhibitors; and high-protein, low-carbohydrate diets (ketogenic diet).

Gillespie RS, Stapleton FB: Nephrolithiasis in children. Pediatr Rev 2004;25:131-138.

15. How do you diagnose urinary stones in children?

Evaluation and diagnosis should begin with a complete history and physical examination, including a history of urinary infections, current medications, and a detailed family history. The majority of children present with flank or abdominal pain. A urinalysis and urine culture should be obtained. Microscopic hematuria is present in more than 90% of children with urinary stones. Infection is commonly seen with stones, so evidence of UTI does not exclude stones.

A nonenhanced helical CT scan is the most sensitive imaging method of diagnosis. Very small stones throughout the entire urinary tract can be identified. Renal ultrasound is an appropriate alternative in many cases, especially when exposure to radiation is a concern. Ultrasound can detect radiolucent stones and urinary obstruction but is limited in identifying small stones (<5 mm). Most stones in children are composed of radiopaque substances and may be seen on plain abdominal radiographs, but the small size of most stones makes this test less sensitive.

Gillespie RS, Stapleton FB: Nephrolithiasis in children. Pediatr Rev 2004;25:131-138.
Hulton SA: Evaluation of urinary tract calculi in children. Arch Dis Child 2001;84:320-323.

Key Points: Hematuria

1. UTIs are the most common cause of hematuria in children.
2. Distinguishing between glomerular and nonglomerular hematuria can help narrow the differential diagnosis and lead to a diagnosis.
3. Any child with gross hematuria requires emergent evaluation.
4. Any child with hematuria should have a urine dipstick, microscopic urinalysis, and blood pressure measurement.
5. Asymptomatic microscopic hematuria without proteinuria is usually benign and self-limited.

16. What are the most common causes of dysuria in children?

Dysuria (painful urination) in usually caused by irritation of the bladder or urethra. Common causes of this symptom include infectious cystitis (viral or bacterial), urethritis (infectious, chemical, or traumatic), vaginitis, and balanitis. Children may report or exhibit perineal discomfort and parents may interpret this discomfort as dysuria in the case of pruritus from pinworms (*Enterobius vermicularis*) or in cases of sexual abuse.

Corboy JB: Pain—Dysuria. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 450-454.
Kellogg ND, Parra JM, Menard S: Children with anogenital symptoms and signs referred for sexual abuse evaluations. Arch Pediatr Adolesc Med 1998;152:634-641.

17. Which systemic diseases may present with dysuria?

Urethritis, producing dysuria, is associated with several serious systemic diseases, such as Stevens-Johnson syndrome, Reiter syndrome, and Behçet's syndrome.

Corboy JB: Pain—Dysuria. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 450-454.

18. Name some common causes of urethritis in children.

Infectious urethritis in adolescents is most commonly due to *Neisseria gonorrhoeae* and *Chlamydia trachomatis*. Bubble baths, detergents, fabric softeners, and perfumed soaps are often the cause for cases of chemical urethritis in younger children.

Corboy JB: Pain—Dysuria. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 450-454.

19. A child reports dysuria but is afebrile, with no genital lesions, and has a negative urinalysis for pyuria. What are the most probable diagnoses?

Dysuria in this child may be due to viral cystitis, chemical irritation of the urethra, pinworms, fused labia minora, idiopathic hypercalciuria, urinary tract stone, or bacterial UTI (a minority of children will present with a negative urinalysis but positive urine cultures).

20. What parasitic infection should be considered in a traveler or recent immigrant presenting with hematuria or dysuria?

Urinary schistosomiasis, caused by *Schistosoma haematobium*, is a common cause of hematuria and dysuria worldwide. This worm is found mostly in sub-Saharan Africa and enters through the skin after contact with fresh water that harbors the larval form. The worms eventually migrate to the bladder mucosa, where eggs are produced and secreted in the urine. Diagnosis can be made by examination of the urine for eggs. Praziquantel is the mainstay of treatment and is curative in most cases.

Ross A, Bartley PB, Sleigh AC, et al: Schistosomiasis. N Engl J Med 2002;346:1212-1220.

Key Points: Dysuria

1. The most common causes of dysuria in children are UTIs and urethritis.
2. Chemical urethritis is a common cause of dysuria in young children.
3. Consider diagnoses such as pinworms, systemic illness, or sexual abuse in children presenting with dysuria.

HYPERTENSION

Deirdre Fearon and Susan J. Duffy

CHAPTER 17

1. What is considered high blood pressure or hypertension in a child?

There's hypertension, and then there's *hyper*tension. The distinction is between *stage 1* (95th percentile or above) and *stage 2* (99th percentile or ≥ 99th percentile +5 mm Hg) hypertension. These classifications by age and sex were established by the Task Force on Blood Pressure Control in Children, updated in 2004. In addition to these classifications, the task force also developed standardized definitions for hypertensive urgency and hypertensive emergency. *Hypertensive urgency* refers to a severely elevated blood pressure without evidence of end-organ damage. A *hypertensive emergency* occurs when a child's blood pressure is severely elevated and the child shows evidence of end-organ damage. Although relatively uncommon in children as compared to adults, hypertensive urgencies and emergencies are known to cause significant health problems and even death.

Constantine E: Hypertension. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 316-323.

National High Blood Pressure Education Program Working Group on Hypertension Control in Children and Adolescents: The fourth report on the diagnosis, evaluation, and treatment of high blood pressure in children and adolescents. Pediatrics 2004;114:555-576.

Key Points: Classification of Hypertension Based on Age, Height, and Sex

1. Stage 1 hypertension is at 95th percentile or above.
2. Stage 2 hypertension is at 99th percentile or ≥ 99th percentile +5 mm Hg.
3. Hypertensive urgency is an acute severe blood pressure elevation *without* evidence of end-organ damage.
4. Hypertensive emergency is an acute severe blood pressure elevation *with* evidence of end-organ damage.

2. If a high blood pressure is found incidentally by the triage nurse, what two questions should you ask before you get too worried?

- **"What size cuff did you use?"** Inappropriate cuff size can give spuriously high or low blood pressure readings. They will be falsely elevated if the cuff is too small, and low if the cuff is too big. The width of the cuff bladder should be about 40% of the circumference of the arm measured at the midpoint between the shoulder and the elbow (technically, between the acromion and the olecranon). The cuff should encircle 80% to 100% of the circumference of the upper arm and be about two thirds of its length.
- **"Will you please repeat it?"** Nonpathologic elevations in blood pressure can be caused by white coats, pain, recent activity, heat, and agitation. Ideally, a child's blood pressure should be measured after a few minutes of inactivity, as he or she sits calmly in a parent's lap.

3. Which patients need evaluation and treatment in the emergency department (ED), and which patients can follow up with their primary care physician?

- **Workup and treatment:** Clearly, patients with hypertensive emergencies should be treated the ER with the initial focus on airway, breathing, and circulation (ABCs) and rapidly establishing intravenous (IV) access.
- **Workup only:** For asymptomatic patients with *stage 2* elevated blood pressure readings, perform a thorough history and physical examination and some screening laboratory tests. If there are no abnormalities in this workup, the patient can be discharged with close follow-up.
- **Discharge without workup:** Patients being seen for another problem who were incidentally found to have *stage 1* hypertension and are asymptomatic can be discharged to the care of their primary care physician. Ideally, the doctor should record several readings in a series of visits before confirming the diagnosis of hypertension (Fig. 17-1).

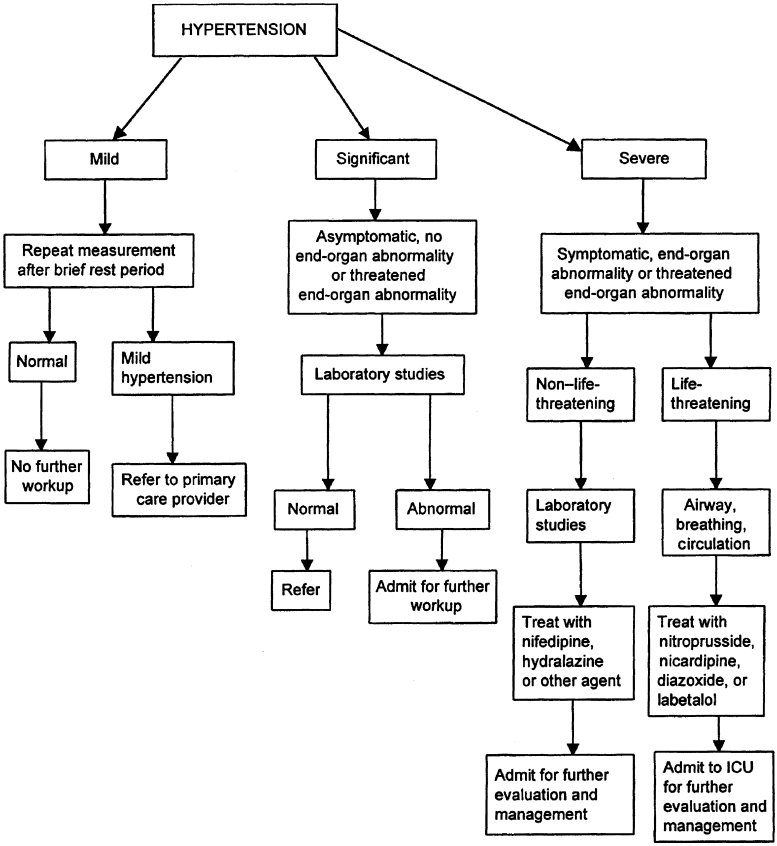


Figure 17-1. Approach to the initial emergency department triage and stabilization of the hypertensive child. *(From Linakis JG: Hypertension. In Fleisher GR, Ludwig S [eds]: Textbook of Pediatric Emergency Medicine, 5th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2006, used with permission.)*

Key Points: Approach to the Hypertensive Child

1. Work up, treat, and admit patients with hypertensive emergencies.
2. Work up and discharge asymptomatic patients with *stage 2* hypertension and normal screening laboratory test results.
3. Discharge with follow-up patients with incidentally found *stage 1* hypertension.

4. Name the causes of pathologic hypertension. Discuss likely causes in babies, small children, and older children.

In general, as the age of a child increases, the likelihood of finding a secondary cause for hypertension decreases. A newborn infant with hypertension is most likely to have either a congenital renal anomaly or a vascular problem (e.g., a renal artery or venous thrombosis or stenosis or coarctation of the aorta). Small children may present with these vascular or congenital causes but become more likely to have renal parenchymal disease such as pyelonephritis, glomerulonephritis, or reflux nephropathy. Older children and teenagers are

more likely to have essential hypertension (including obesity), though they may still present with parenchymal disease. The mnemonic HYPERTENSION may help you recall some of the major causes of high blood pressure:

- **H:** Hyperthyroidism (and other autoimmune diseases)
- **Y:** Why? Cause unknown—primary hypertension
- **P:** Pheochromocytoma
- **E:** Eats too much—obesity (or other unhealthy habits—alcohol, tobacco)
- **R:** Renal parenchymal disease
- **T:** Thrombosis (renal artery, particularly if umbilical catheter was used as neonate)
- **E:** Endocrine disorder (*congenital adrenal hyperplasia*, primary aldosteronism, hyperparathyroidism)
- **N:** Neurologic disorder (increased intracranial pressure [ICP], Guillain-Barré syndrome, neurofibromatosis)
- **S:** Stenosis (renal artery stenosis or coarctation of the aorta, supravalvular aortic stenosis with Williams syndrome)
- **I:** Ingestion (cocaine, sympathomimetics, birth control pills, steroids, decongestants, sudden withdrawal, or chemotherapy)
- **O:** Obstetric cause (eclampsia)
- **N:** Neuroblastoma

Constantine E: Hypertension. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 316-323.

5. How might a child present with hypertension?

Often hypertension is silent and picked up on routine physical examination. If a child does present with symptoms, they may be as vague as irritability and headache or as significant as seizure and coma. Other possible symptoms include visual disturbances, personality changes, dizziness, nausea and vomiting, weight loss, polyuria and polydipsia, and facial nerve palsy.

6. What historical questions are important to ask the parent of a hypertensive child?

- Does the child have a history of recurrent urinary tract infections? Unexplained fevers? Hematuria? Frequency? Dysuria? Any recent illness? Sore throats? Chest pain? Shortness of breath?
- Did the child have an umbilical artery catheter as a neonate?
- Does he or she have intermittent sweating, flushing, and palpitations?
- Has the child been growing well?
- Has the child suffered any recent head trauma?
- Is there a family history of hypertension, renal disease, or deafness?
- Has the child ingested anything? Decongestants? Birth control pills? Steroids? Cocaine?

7. What physical examination findings are important with a hypertensive child?

Carefully examine the child for evidence of end-organ damage from hypertension. Look for clinical features that may suggest the possible causes of hypertension:

- **General:** dysmorphism (e.g., elfin facies of Williams syndrome), cushingoid features, over/underweight
- **Head, ears, eyes, nose, throat:** evidence of head trauma, decreased visual acuity, papilledema, retinal infarcts, abnormal pupillary reflex
- **Neck:** webbed neck, thyroid enlargement
- **Lungs:** crackles (evidence of ventricular failure), increased work of breathing
- **Heart:** displaced point of maximal impulse (suggestive of left ventricular hypertrophy), murmurs
- **Abdomen:** bruits, hepatomegaly, abdominal masses
- **Renal:** flank pain
- **Back:** signs of spina bifida
- **Extremities:** decreased femoral pulses, discrepant four extremity blood pressures, edema
- **Skin:** café-au-lait spots or skinfold freckling (neurofibromatosis), xanthomas (hyperlipidemia), hirsutism, purpuric rash on lower extremities
- **Neurologic:** headache, seizures, altered mental status, encephalopathy, cranial nerve palsies, sensorimotor asymmetry, hyperreflexia

8. What laboratory tests should you obtain in the initial workup of a patient with hypertension?

Much information can be obtained with relatively few laboratory tests. Usually a set of electrolytes with a blood urea nitrogen and creatinine, a complete blood count, and a urine dipstick test are enough to get started in the asymptomatic patient. In obese children, a lipid profile may be helpful, as would a urine culture in all girls and those boys with known renal disease. In patients who are symptomatic, electrocardiography and chest radiography may help you determine the degree of end-organ damage. If ultrasonography is readily available in the Emergency Department, renal ultrasonography can be particularly helpful in the diagnosis of a renal cause of hypertension in a baby.

Key Points: Laboratory Workup for a Hypertensive Child

1. Urine dipstick test (and culture if indicated)
2. Electrolytes, blood urea nitrogen, creatinine, with or without lipid profile
3. Electrocardiography and chest radiograph if patient is symptomatic
4. Renal ultrasonography if available

9. If a child has a dangerously high blood pressure, should it be lowered to normal as quickly as possible?

Well, sort of. It is important to normalize blood pressure to below the 95th percentile to prevent further end-organ damage, but doing so too quickly can be harmful to those same organs, specifically the brain. Cerebral autoregulation maintains a relatively constant cerebral blood flow with variations in peripheral pressures. When peripheral blood pressure increases, cerebral vessels constrict. When peripheral pressure drops, those vessels dilate. There are points, however, at extremes of blood pressure, at which this system is exhausted. When the cerebral vessels are maximally constricted or dilated, the brain can no longer autoregulate. It is particularly important when treating a hypertensive emergency to bear in mind that under hypertensive conditions, the low point at which autoregulation ceases to function is *raised* (i.e., maximal cerebral vasodilatation occurs at a higher blood pressure). Lowering the blood pressure below this point can lead to cerebral ischemia. For this reason, an elevated blood pressure should be lowered to normal as slowly as is safely possible, ideally over days rather than minutes. When treating hypertensive emergencies, the mean arterial pressure (MAP) should be lowered no more than 25% of the initial value in the first hour, and a gradual reduction should be obtained over the next 24 to 48 hours.

National High Blood Pressure Education Program Working Group on Hypertension Control in Children and Adolescents: The fourth report on the diagnosis, evaluation, and treatment of high blood pressure in children and adolescents. Pediatrics 2004;114:555-576.

Chandar J, Zilleruelo G: Hypertensive crisis in children, *Pediatr Nephrol* 27:741, 2012.

Hari P, Sinha A: Hypertensive emergencies in children, *Indian J Pediatr* 78(5):569-575, 2011.

Salgado D, Silva E, Vincent JL: Control of hypertension in the critically ill: a pathophysiological approach, *Ann Intensive Care* 3:17, 2013.

10. Which medications would you use to treat hypertension in the ED?

Treat patients with hypertensive emergencies in the ED. The medicines you choose will depend on the patient's current medications, the suspected cause of the hypertension, your comfort with particular medicines, and whether the child's life is in danger. For hypertensive urgencies, nifedipine and hydralazine are safe, effective choices. They begin to work about 10 minutes after administration and last for several hours. Enalapril, clonidine, minoxidil, and furosemide are other reasonable choices, particularly for targeted causes of hypertension (Table 17-1). For immediate results in hypertensive emergencies, a nitroprusside infusion will give dose-related effects that will cease within minutes of stopping the infusion. Nicardipine is an effective calcium channel blocker that acts quickly to reduce peripheral vascular resistance. Another alternative is labetalol, which has rapid effects on both α- and β-adrenergic receptors; is a bit more difficult to titrate; and is contraindicated in patients with asthma, heart block, heart failure, and pheochromocytoma. Esmolol is a beta blocker that can be easily titrated but is contraindicated in amphetamine-like ingestions. Alternatives for specific clinical indications include fenoldopam, phentolamine, and others (see Table 17-1).

Table 17-1. Medications for Treatment of Emergent and Urgent Hypertension in Children

MEDICATION	TYPE	ROUTE	ONSET/ DURATION	MECHANISM OF ACTION	COMMON INDICATIONS	CONTRAINDICATION/ CONCERNS
Hypertensive Emergencies						
Nicardipine	Calcium channel blocker	IV (titrate to effect)	Onset 1-10 min/ duration 2-4 hours	Arterial vasodilator promotes cerebral and coronary dilation, decreases SVR	Acute severe HTN; perioperative HTN, stroke or intracranial hemorrhage related HTN, acute renal failure; sympathetic crisis	Hypovolemia, may cause reflex tachycardia
Labetalol	Alpha and beta blocker	IV (titrate to effect)	Onset 2-10 min/ duration 2-6 hours	Decreases SVR	Acute severe HTN; stroke or intracranial hemorrhage related HTN	Asthma, CHF
Sodium nitroprusside	Vasodilator	IV (titrate to effect)	Onset <1-2 min/ duration <10 min	Direct venous and arterial vasodilator	Acute severe HTN; CHF pulmonary edema	Intracranial hypertension, may cause cyanide toxicity
Esmolol	Beta blocker	IV (titrate to effect)	Onset 1-2 min/ duration 10-30 min	Reduction in CO (through contractility and HR)	Acute aortic dissection; perioperative HTN (primarily OR/ICU)	Asthma, CHF cocaine or amphetamine toxicity bradycardia
Hydralazine	Vasodilator	IV/IM (bolus dosing)	Onset 10-20 min IV, 20-30 min IM/duration 2-6 hours	Direct arterial vasodilator	No IV access, IM bolus dosing, preeclampsia	Heart disease, may overshoot desired BP due to bolus dosing
Fenoldopam	Dopamine D1 receptor agonist	IV	Onset 5-20 min/ duration 30-60 min	Increases renal blood flow/natriuresis and urine output	Acute renal failure; sympathetic crisis; perioperative HTN	Anaphylaxis in patients with sulfite sensitivity, glaucoma

Continued on following page

Table 17-1. Medications for Treatment of Emergent and Urgent Hypertension in Children (*Continued*)

MEDICATION	TYPE	ROUTE	ONSET/ DURATION	MECHANISM OF ACTION	COMMON INDICATIONS	CONTRAINDICATION/ CONCERNS
Phentolamine	α-Adrenergic blocker	IV	Onset 5-10 min/ duration 30-60 min	Antagonism of circulating epinephrine and norepinephrine, inotropic and chronotropic effects on heart	Pheochromocytoma, cocaine, pseudoephedrine toxicity	Myocardial infarction or coronary artery disease
Hypertensive Urgency						
Clonidine	Central α_2-agonist	PO	Onset 15-30 min/ duration 6-8 hours	Reduces cerebral sympathetic output	Hemodialysis patients; HTN from pain, anxiety, drug withdrawal	May cause sedation
Nifedipine	Calcium channel blocker	PO/SL	Onset 10-30 min/ duration 4-8 hours	Coronary vasodilatation, reduces peripheral vascular resistance	When IV therapy is delayed	Cardiogenic shock, myocardial infarction
Furosemide	Rapid-acting diuretic	IV/PO		Diuretic, inhibits NaCl and water reabsorption	HTN with fluid overload in addition to antihypertensives	

BP, blood pressure; CHF, congestive heart failure; CO, cardiac output; HR, heart rate; HTN, hypertension; ICH, intracranial hemorrhage; ICU, intensive care unit; IM, intramuscular; IV, intravenous; NaCl, sodium chloride; OR, operating room; PO, per os (taken orally); SL, sublingual; SVR, systemic vascular resistance.

Constantine E: Hypertension. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 316-323.
Flynn JT, Tullus K: Severe hypertension in children and adolescents: Pathophysiology and treatment. Pediatr Nephrol 2009;24(6):1101-1112.
Meyers K, Falkner B: Hypertension in children: An approach to management of complex hypertension in pediatric patients. Curr Hypertens Rep 2009;11:315-322.
National High Blood Pressure Education Working Group on Children and Adolescents: The Fourth Report on the Diagnosis, Evaluation and Treatment of High Blood Pressure in Children and Adolescents. Bethesda, MD, National Heart, Lung, and Blood Institute, U.S. Department of Health and Human Services, 2005, NIH Publication No. 05:5267.
Singh D, Akinbola O, Yosypiv I, El-Dahr S: Emergency management of hypertension in children. Int J Nephrol 2012;2012:420247. Epub Apr 19, 2012.

Key Points: Medical Treatment of Hypertension

1. Focus treatment on the specific cause of the hypertension.
2. Non-life-threatening: Use nifedipine or hydralazine or other condition-appropriate medication (see Table 17-1).
3. Life-threatening: Use nicardipine, nitroprusside, or similar IV titratable agents (see Table 17-1).

11. Name some other classes of medications that might be useful in the treatment of a hypertensive emergency.

Depending on the type of end-organ damage, treatment of complications associated with severely elevated blood pressures may be necessary. For example, lorazepam for seizures, diuretics for heart failure, and appropriate intubation medications should be available when treating a child with a hypertensive emergency.

12. An adolescent boy is brought to the ED at 5:00 AM after a night out dancing. He is euphoric and diaphoretic and has a dry mouth. He is tachycardic and hypertensive. You consider giving him a beta blocker, but think better of it. Why?

The patient has probably ingested an amphetamine-related compound, such as 3,4-methylenedioxymethamphetamine (MDMA, or "ecstasy"). The cardiovascular effects of phenylethylamines are due to both α- and β-adrenergic stimulation. A beta blocker would leave α-adrenergic effects unopposed and result in vasospasm and paradoxical hypertension. Instead, consider using nitroprusside or nifedipine.

Albertson TE, Van Hoozen BE, Allen RP: Amphetamines. In Haddad LM, Shannon MW, Winchester JF (eds): Clinical Management of Poisoning and Drug Overdose, 3rd ed. Philadelphia, WB Saunders, 1998, pp 560-568.

13. A school-aged child is in your ED being evaluated for an upper respiratory tract infection. You notice he is hypertensive on repeated measurements. He is an otherwise healthy child. When asked, he admits to some leg pain after playing soccer. He has a systolic ejection murmur. His radial pulses are bounding, but you have trouble feeling his dorsalis pedis pulses. What might be the cause of his hypertension?

Coarctation of the aorta can present after infancy, with hypertension as the presenting sign. Claudication symptoms and a heart murmur may or may not be present. It is important to feel for differential upper- and lower-extremity pulses in hypertensive patients of all ages, and to check four extremity blood pressures if there is any concern about coarctation of the aorta. In normal patients, lower-extremity blood pressures are higher than upper-extremity pressures by 10 to 20 mm Hg. In patients with coarctation of the aorta, the blood pressure in the legs is lower than that in the arms.

14. A 6-year-old boy complains of abdominal pain and is found to be hypertensive. When you get him undressed, you notice multiple purpuric lesions on his buttocks and lower extremities. What is the most likely diagnosis?

Henoch-Schönlein purpura. Although hypertension is unusual in this condition, it is still important to measure the child's blood pressure in all cases. Hypertension may justify admission to the hospital.

15. A teenager presents with "dark-colored urine" and is found to be hypertensive. What is the most likely diagnosis?

Poststreptococcal glomerulonephritis. Gross hematuria is present in about 30% to 50% of patients with this condition. The urine looks smoky, and is tea- or cola-colored. Hypertension is present in 50% to 90% of patients with this condition and varies from mild to severe. It is primarily caused by fluid retention. Hypertensive encephalopathy is an uncommon but serious complication. These patients require emergent intervention. Generalized edema is present in about two thirds of patients with acute glomerulonephritis due to sodium and water retention. In severe cases, fluid overload leads to respiratory distress due to pulmonary edema. A chest radiograph may show cardiomegaly and signs of heart failure.

JAUNDICE

James M. Callahan

CHAPTER 18

1. What is jaundice?

Jaundice, or icterus, is a yellow or green-yellow discoloration of the skin, mucous membranes, sclerae, and body fluids caused by increased levels of circulating bilirubin. Jaundice is usually noticeable at serum bilirubin levels of 5 mg/dL.

2. Where does bilirubin come from?

The major source of bilirubin is the breakdown of heme pigment released from senescent erythrocytes. Bilirubin is usually cleared from the circulation by the liver and, once conjugated, is excreted in bile. Hemolysis that exceeds the liver's capacity to conjugate bilirubin or processes that impair the excretion of bile cause increased levels of bilirubin.

Schwartz HP, Haerman BE, Ruddy RM: Hyperbilirubinemia: Current guidelines and emerging therapies. Pediatr Emerg Care 2011;27:884-889.

3. Is jaundice always pathologic?

Up to two thirds of newborns develop visible jaundice at some point in the first few weeks of life. Most of these infants have "physiologic" or "breast milk" jaundice and not a pathologic process. Potentially harmful causes must be excluded before deciding that these benign conditions are present. In children over 3 months of age, jaundice is almost always associated with a pathologic process.

4. How do you begin the evaluation of a patient with jaundice?

The first step in evaluation, whether the patient is a newborn or an older child, is to determine whether the jaundice is due to conjugated or unconjugated hyperbilirubinemia by obtaining total and direct (or conjugated) bilirubin levels. *Conjugated* hyperbilirubinemia is defined as jaundice in which the direct bilirubin level is higher than 2 mg/dL or accounts for more than 20% of the total bilirubin. *Unconjugated* hyperbilirubinemia (high indirect bilirubin level) is usually due to hemolytic processes or defects in conjugation. Conjugated hyperbilirubinemia is pathologic at any age and is usually associated with cholestatic processes due to hepatic disease or anatomic obstruction to bile flow. Further workup and treatment are guided by the determination of the type of hyperbilirubinemia. The approach also varies depending on the age of the patient (newborn versus older infant/child).

Brumbaugh D, Mack C: Conjugated hyperbilirubinemia in children. Pediatr Rev 2012;33:291-302.

5. What are the causes of unconjugated hyperbilirubinemia in a newborn?

- Placental dysfunction
- Diabetes in the mother
- Swallowing of maternal blood
- Cephalohematoma or extensive bruising
- Sepsis
- Upper gastrointestinal obstruction (pyloric stenosis, duodenal web, or atresia)
- Red blood cell defects
- ABO, Rh, and minor blood group incompatibility
- Crigler-Najjar syndrome (defect in bilirubin conjugation)
- Lucey-Driscoll syndrome (familial benign unconjugated hyperbilirubinemia)
- Breast milk jaundice
- Physiologic jaundice

6. What are acute bilirubin encephalopathy (ABE) and kernicterus?

At high levels, unconjugated hyperbilirubin may cross a compromised blood-brain barrier and cause lethargy, hypotonia, poor feeding, fever, seizures, and signs of increased

intracranial pressure in the first days and weeks following birth. The American Academy of Pediatrics now recommends that the term *acute bilirubin encephalopathy* be used to describe this constellation of signs and symptoms. Untreated, unrecognized, and severe cases of ABE in which treatment was instituted but not effective may lead to mental retardation, cerebral palsy, and sensorineural hearing loss. These chronic and irreversible manifestations of bilirubin toxicity are termed kernicterus.

American Academy of Pediatrics Subcommittee on Hyperbilirubinemia: Management of hyperbilirubinemia in the newborn infant 35 or more weeks of gestation. Pediatrics 2004; 114:297-316.
Schwartz HP, Haerman BE, Ruddy RM: Hyperbilirubinemia: Current guidelines and emerging therapies. Pediatr Emerg Care 2011;27:884-889.

7. Which children are at risk for ABE and kernicterus?

Neonates with jaundice associated with a coexisting serious condition are most likely to develop kernicterus. Children with sepsis, hypoxia or hypercarbia, brisk hemolysis (e.g., that seen with Rh incompatibility), hypoglycemia, or prematurity are most at risk. In healthy, full-term infants, kernicterus is almost never seen at unconjugated bilirubin levels lower than 25 mg/dL. Premature infants and those with intercurrent illnesses may experience kernicterus at lower levels. Kernicterus, though rare, still occurs, even in otherwise healthy children born at or near term.

American Academy of Pediatrics Subcommittee on Hyperbilirubinemia: Management of hyperbilirubinemia in the newborn infant 35 or more weeks of gestation. Pediatrics 2004; 114:297-316.
Ip S, Chung M, Kulig J, et al: An evidence-based review of important issues concerning neonatal hyperbilirubinemia. Pediatrics 2004;114:e130-e153.

8. What are some indicators that jaundice is *not* physiologic and therefore should prompt further investigation in newborns?

- Jaundice in first day of life
- Bilirubin level that increases more than 5 mg/dL per day
- Conjugated bilirubin level higher than 1.5 mg/dL or higher than 10% of total bilirubin
- Jaundice beyond the first week of life
- Jaundice with hepatosplenomegaly and anemia
- Jaundice in infants who are ill-appearing

9. What other laboratory tests are indicated in neonates with unconjugated hyperbilirubinemia?

In most children, no other laboratory tests are needed. In addition to children meeting the preceding criteria, consider pathologic causes of jaundice in all children who appear ill or dehydrated. In children who are at risk of hemolysis based on the maternal blood type, obtain a complete blood count (CBC) with differential and examination of the smear, a reticulocyte count, and direct Coombs test.

Anemia or reticulocytosis suggests blood loss or ongoing hemolysis. If the Coombs test result is positive, isoimmunization has probably occurred (because of Rh, ABO, or minor blood group incompatibility). Determine maternal and neonatal blood types (this may be known at the hospital of delivery). Hemolysis in the absence of isoimmunization suggests a red blood cell defect in this age group, and a glucose-6-phosphate dehydrogenase (G6PD) assay as well as fragility testing would be useful.

Ill-appearing children and children with fever or hypothermia may have an underlying infection. Obtain a CBC in these children as well. The white blood cell count and differential may indicate an infectious process. Unconjugated hyperbilirubinemia in the absence of hemolysis may be seen with infections (although conjugated hyperbilirubinemia is more commonly seen), and a urinalysis, Gram stain, and culture may be helpful.

Garcia FJ, Nager AL: Jaundice as an early diagnostic sign of urinary tract infection in infancy. Pediatrics 2002;109:846-851.
Wolff M, Schinasi DA, Lavelle J, et al: Management of neonates with hyperbilirubinemia: Improving timeliness of care using a clinical pathway. Pediatrics 2012;130:e1688-e1694.

10. What is breast milk jaundice?

Breast-fed infants are about three times as likely as formula-fed babies to have high bilirubin levels in the first week of life. In the first few days of life, this is probably due to decreased fluid and caloric intake (until their mother's milk is fully in) and decreased passage of

meconium stools. Later in the first week of life, it has been proposed that lipase and nonesterified long-chain fatty acids in breast milk inhibit hepatic excretion of bilirubin, and the β-glucuronidase in breast milk increases enterohepatic circulation of bilirubin. Children with breast milk jaundice often have the onset of jaundice (or continued increases in levels of unconjugated bilirubin) between the fourth and tenth days of life.

Schwartz HP, Haerman BE, Ruddy RM: Hyperbilirubinemia: Current guidelines and emerging therapies. Pediatr Emerg Care 2011;27:884-889.

11. What is physiologic jaundice? What causes it?

Most infants (approximately two thirds) develop visible jaundice at some point in the first week of life. The vast majority of these children have physiologic jaundice. Many factors contribute to this "normal" hyperbilirubinemia, including a large red blood cell (RBC) mass, decreased survival of RBCs, decreased hepatic uptake of bilirubin, decreased conjugation of bilirubin in the liver, and increased enterohepatic recirculation of bilirubin. Physiologic jaundice is a diagnosis of exclusion, and bilirubin levels usually peak by the third day of life.

12. When is treatment indicated in neonates with unconjugated hyperbilirubinemia?

The American Academy of Pediatrics Subcommittee on Hyperbilirubinemia published a Clinical Practice Guideline for the management of hyperbilirubinemia in newborns who are at least 35 weeks gestational age. Nomograms based on the age of the child and a risk stratification based on gestational age and other risk factors are provided to guide the use of both phototherapy and exchange transfusions. In general, treat healthy, full-term infants with phototherapy when their bilirubin level is higher than 15 mg/dL in the first 2 days of life, or higher than 18 mg/dL after that time. Some infants are lethargic, and the mother may report that the infant is not nursing well. Additional feedings with formula may be helpful, and in some infants supportive measures with intravenous (IV) saline may be needed. If despite phototherapy the bilirubin level increases to higher than 20 mg/dL, or if at any time the bilirubin level is higher than 25 mg/dL, consider an exchange transfusion. For full-term infants who are ill (those with hemolysis, sepsis, hypoglycemia, acidosis, or hypoxia) and preterm infants, begin phototherapy and institute exchange transfusion at lower serum bilirubin levels. Temporary interruption of breastfeeding may lead to a rapid and sustained decrease in bilirubin levels in children with breast milk jaundice.

American Academy of Pediatrics Subcommittee on Hyperbilirubinemia: Management of hyperbilirubinemia in the newborn infant 35 or more weeks of gestation. Pediatrics 2004; 114:297-316.

Key Points: Treatment of Unconjugated Hyperbilirubinemia in Neonates

1. Always obtain fractionated bilirubin levels (total and direct or conjugated bilirubin) in neonates who present to the emergency department (ED) with jaundice.
2. Most children will not need other testing.
3. Treat healthy, full-term infants with a serum bilirubin level higher than 15 mg/dL in the first 2 days of life or higher than 18 mg/dL after 2 days of age with phototherapy.
4. Treat preterm infants and infants who are ill (e.g., sepsis or other infections or hemolytic disease) at lower levels of bilirubin.
5. Provide infants with good supportive care.
6. Consider exchange transfusion for infants who do not respond to phototherapy or whose bilirubin level is higher than 25 mg/dL (lower in preterm and sick infants).

13. Is treatment for unconjugated hyperbilirubinemia an emergency?

Yes, when the level is very high. An outside laboratory or a visiting home nurse often does a bilirubin test, and the abnormal result prompts the emergency department (ED) visit. Thus, the report may be several hours old by the time the infant presents to the ED. The bilirubin level can sometimes rise quickly to a dangerous level. Avoid unnecessary delays in evaluation and management. When the unconjugated or indirect bilirubin approaches a dangerous level, make arrangements for prompt phototherapy. This is sometimes difficult to do in an ED setting, and some believe jaundiced infants are better managed as direct admissions to the hospital, where phototherapy can begin quickly. However, one group recently showed that institution of a

clinical pathway for the treatment of neonates with jaundice in their ED led to marked improvements in time to phototherapy and a decreased ED length of stay.

Maisels MJ: Jaundice in a newborn—How to head off an urgent situation. Contemp Pediatr 2005;22:41-54.
Wolff M, Schinasi DA, Lavelle J, et al: Management of neonates with hyperbilirubinemia: Improving timeliness of care using a clinical pathway. Pediatrics 2012;130:e1688-e1694.

14. List the causes of conjugated hyperbilirubinemia in neonates.

- Sepsis
- TORCH infections (toxoplasmosis, other agents, rubella, cytomegalovirus, and herpes simplex)
- Idiopathic neonatal hepatitis
- Alagille syndrome (arteriohepatic dysplasia)
- α_1-Antitrypsin deficiency
- Inborn errors of metabolism (e.g., galactosemia, tyrosinosis)
- Urinary tract infections
- Hypopituitarism
- Postshock or postasphyxia biliary sludging
- Cholestasis associated with total parenteral nutrition
- Cystic fibrosis
- Biliary atresia
- Anatomic obstruction (e.g., choledochal cyst)

15. Which laboratory tests are helpful in determining the cause of conjugated hyperbilirubinemia in neonates?

Obtain a CBC with differential, urinalysis, Gram stain, blood culture, and urine cultures to look for signs of sepsis or urinary tract infection. A urinalysis with increased reducing substances other than glucose suggests galactosemia, especially in a patient with hypoglycemia. Viral and toxoplasmosis titers may help establish the diagnosis of a TORCH infection. Any vesicular lesions of the skin or mucous membranes can be tested with immunofluorescence assays and cultured for herpes simplex virus. Hepatic aminotransferase levels that are markedly elevated suggest hepatitis. Sweat chloride testing (to rule out cystic fibrosis), α_1-antitrypsin phenotyping, and repeated thyroid hormone testing may be needed in some infants. Albumin and clotting studies will reflect the synthetic capabilities of the liver. Closely monitor serum glucose because infants with hepatic disease may be at risk for hypoglycemia.

Key Points: Approach to Neonatal Jaundice

1. Determine whether the jaundice is due to conjugated or unconjugated hyperbilirubinemia.
2. Conjugated hyperbilirubinemia is always due to a pathologic process in this age group and requires further diagnostic workup (including imaging studies to rule out biliary obstruction) and hospital admission.
3. Most children with unconjugated hyperbilirubinemia will have physiologic or breast milk jaundice, but the history, physical examination, and diagnostic workup must be thorough to exclude pathologic causes.

16. What imaging studies are helpful?

Abdominal ultrasonography can evaluate the extrahepatic biliary tract, looking for choledochal cysts as well as other signs of obstruction. A radionuclide scan (DISIDA [diisopropyl iminodiacetic acid] scan) that demonstrates excretion of radionuclide into the bowel excludes biliary atresia. Absence of excretion means that biliary atresia remains a possibility and further workup (hepatic biopsy) is warranted. Early diagnosis of biliary atresia is imperative to preserve hepatic function. The preceding studies are generally obtained on the inpatient unit rather than in the ED.

17. What treatment is required for neonates with conjugated hyperbilirubinemia?

Because conjugated hyperbilirubinemia always indicates a pathologic process, admit all infants with conjugated hyperbilirubinemia and consult with a gastroenterologist. Although diagnostic testing is in progress, supportive therapy may be required, including IV fluids and dextrose; antibiotics or antivirals if indicated (e.g., acyclovir for neonatal herpes infection); vitamin K; and clotting factor replacement. Specific therapy is based on the outcome of the

diagnostic evaluation and does not usually occur in the ED. Phototherapy is not helpful unless there is also a markedly elevated indirect bilirubin level.

Brumbaugh D, Mack C: Conjugated hyperbilirubinemia in children. Pediatr Rev 2012; 33:291-302.

18. In older infants and children, can anything else be mistaken for jaundice?

Yes. Children who eat large amounts of yellow, orange, or red vegetables can develop a yellow discoloration of their skin. Carotene-containing vegetables can produce carotodermia, and red vegetables (e.g., tomatoes) can produce lycopenemia. Unlike jaundice, this discoloration does not affect the sclerae, and the child with carotodermia will be thriving and well-appearing.

19. What are the causes of unconjugated hyperbilirubinemia in older infants and children?

Unconjugated hyperbilirubinemia after the neonatal period is due to either hemolysis or decreases in the hepatic conjugation of bilirubin. Hemolysis is seen in children with hemoglobinopathies (e.g., sickle cell disease), RBC defects (e.g., G6PD deficiency after exposure to an oxidant stress or children with hereditary spherocytosis or elliptocytosis), autoimmune hemolytic anemia, or Wilson's disease. Unconjugated hyperbilirubinemia in the absence of hemolysis is seen in Gilbert disease or Crigler-Najjar syndrome.

20. What is the diagnostic approach to children with unconjugated hyperbilirubinemia?

Obtain a CBC with examination of the smear, reticulocyte count, and direct Coombs test. In the absence of hemolysis, there is probably a defect in hepatic uptake of albumin-bound bilirubin, as seen with Gilbert disease and Crigler-Najjar syndrome. Seek a gastroenterology consultation.

If hemolysis is present, findings of sickle cells, spherocytes, or elliptocytes on the CBC smear may be diagnostic. Hemoglobin electrophoresis or osmotic fragility tests may confirm these diagnoses. If the Coombs test result is positive, the patient is probably experiencing autoimmune-mediated hemolysis. Hemolysis without suggestive RBC morphologic appearance and a negative Coombs test result may be seen in patients with G6PD deficiency (usually after exposure to salicylates, sulfonamides, naphthalene, etc.) or Wilson's disease (usually accompanied by psychiatric problems and physical or laboratory signs of hepatic disease).

21. List the causes of conjugated hyperbilirubinemia in older infants and children.

- Viral infections (hepatitis A, B, and C; cytomegalovirus; Epstein-Barr virus)
- Bacterial infections (sepsis, pneumonia, hepatic abscess)
- Toxins (*Amanita* mushrooms, carbon tetrachloride and solvents, drugs)
- Total parenteral nutrition
- Biliary tract disease (cholelithiasis, cholecystitis, choledochal cyst, cholangitis)
- Inflammatory disease (autoimmune chronic active hepatitis, primary sclerosing cholangitis)
- Genetic diseases (Wilson's disease, α_1-antitrypsin deficiency, cystic fibrosis)
- Hemochromatosis

22. What medications can cause cholestasis leading to conjugated hyperbilirubinemia?

Anticonvulsants (phenobarbital, phenytoin, carbamazepine, and valproic acid); antibiotics (estolate preparations of erythromycin, tetracycline, sulfonamides, isoniazid, rifampin, ketoconazole, and griseofulvin); corticosteroids; oral contraceptives; acetaminophen; salicylates; chlorpromazine; cimetidine; and immunosuppressants (cyclosporine, azathioprine, and methotrexate) have all been associated with cholestasis.

23. What is the diagnostic approach to *older* children with conjugated hyperbilirubinemia?

Consider biliary tract disease in patients with a predisposition to hemolysis (e.g., sickle cell disease) and in patients with severe right-upper-quadrant pain and vomiting. Cholelithiasis and other entities causing biliary tract obstruction are more likely in these patients, although there seems to be an increasing incidence of biliary tract disease in children, even in the absence of underlying hemolytic disease. Always obtain abdominal ultrasonography.

If the onset of jaundice is acute, consider toxic and infectious causes. Hepatic aminotransferase levels are usually increased in these patients. Seek history of medication

use or toxic exposures. A prodrome of nonspecific symptoms and fever is more often seen with infections. Obtain viral serologic tests.

A less acute onset and jaundice associated with signs of chronic, systemic disease should prompt investigations of possible genetic or autoimmune causes (e.g., Pi typing to rule out α_1-antitrypsin deficiency or a sweat test to exclude cystic fibrosis).

Brumbaugh D, Mack C: Conjugated hyperbilirubinemia in children. Pediatr Rev 2012; 33:291-302.
Clemente MG, Schwarz K: Hepatitis: General principles. Pediatr Rev 2011;32:333-340.

24. When does a child with jaundice require admission to the hospital?

Admit children to the hospital for dehydration, hypoglycemia, signs or laboratory findings of active biliary tract disease, severe bacterial infections, ongoing hemolysis, or signs of systemic disease or a change in mental status. Also admit any patient with indications of hepatic failure and monitor them closely. When the underlying diagnosis is uncertain, admission for continued diagnostic investigations is often warranted.

Children without signs of hepatic failure who are able to maintain oral hydration and have laboratory evidence of intact hepatic synthetic function (normal albumin, normal prothrombin time) often can be safely discharged. Many of these patients will have viral hepatitis. If discharged, obtain serologic tests and ensure close follow-up.

Key Points: Hepatic Failure in Patients with Jaundice

1. In all patients with jaundice, be sure that there are no signs of hepatic failure.
2. Abnormal albumin, prothrombin time, and ammonia levels indicate hepatic failure and warrant admission, close observation, and supportive care, including treatment with vitamin K (fresh frozen plasma if there is active hemorrhage).

25. What are the complications of severe hepatic disease (i.e., hepatic failure)?

Abnormal bleeding and a decreased level of consciousness are the most worrisome complications of severe hepatic disease. Decreased hepatic synthetic function leads to decreased levels of coagulation proteins and prolonged bleeding. The prothrombin time is elevated. Spontaneous hemorrhages, including intracerebral hemorrhages, may occur. Increased plasma ammonia levels and cerebral edema are associated with hepatic encephalopathy.

Devictor D, Tissieres P, Afanetti M, Debray D: Acute liver failure in children. Clin Res Hepatol Gastroenterol 2011;35:430-437.

26. What treatment is required for an older infant or child with jaundice?

The main goal is to determine the cause of the patient's jaundice so that specific therapies can be started. While the diagnostic evaluation is proceeding, supportive treatment may involve the administration of IV fluids, vitamin K, or clotting factors as necessary; lactulose and neomycin for the treatment of hepatic encephalopathy; and possible intubation and ventilatory support in patients with a decreased level of consciousness.

LIMP

Susanne Kost

CHAPTER 19

1. What is the most common cause of a nontraumatic acute limp in children?

Transient synovitis, formerly known as toxic synovitis, is the most common nontraumatic cause of limp in children, affecting up to 3% of the pediatric population. Transient synovitis is believed to be a postinfectious reactive arthritis, occurring most commonly in the hip, but occasionally in the knee or ankle. It is usually preceded by a viral respiratory or gastrointestinal illness.

2. Describe a classic case of transient synovitis.

The classic patient with transient synovitis is a white boy (male-female ratio, 2:1) between 3 and 10 years old (incidence peaks at age 6) with a 1- to 2-day history of limp, reporting unilateral pain in the hip, thigh, or knee. His past medical history will include viral symptoms within the past 1 to 2 weeks in over half the cases. He will have little or no fever and will appear well (unlike septic arthritis). His examination will be remarkable for a painful hip on passive range of motion, and he will prefer to hold the hip abducted and externally rotated. Transient synovitis typically causes a lesser degree of pain and limitation of motion than septic arthritis.

3. If you had to choose one clinical feature and one laboratory test to distinguish transient synovitis from septic arthritis, what would be most helpful?

Ability to bear weight and C-reactive protein (CRP). White blood cell (WBC) counts are normal (<15,000 cells/mL) in the majority of patients with both conditions. Erythrocyte sedimentation rate (ESR) is elevated (<20 mm/hour) in most patients with septic arthritis and about half of patients with transient synovitis. Fluid in the joint space on plain radiographs and ultrasound is common in both conditions. A review of over 300 children with hip pain and effusion found that patients with both inability to bear weight and a CRP higher than 2.0 mg/dL had a 74% probability of septic arthritis, and those patients with neither feature had less than 1% probability. Elevated CRP was the best independent predictor of septic arthritis, with an odds ratio of 82.

Singhal R, Perry DC, Khan FN, et al: The use of CRP within a clinical prediction algorithm for the differentiation of septic arthritis and transient synovitis of the hip in children. J Bone Joint Surg Br 2011;93(11):1556-1561.

4. Describe the common presentations of the following hip diseases of children: developmental dysplasia of the hip, Legg-Calvé-Perthes disease, and slipped capital femoral epiphysis (SCFE).

The common presentations of hip disorders of children can be seen in Figure 19-1A and B and Table 19-1.

5. What is the likelihood of the previously mentioned hip diseases affecting both hips?

Developmental dysplasia of the hip is bilateral in about 20% of cases, Legg-Calvé-Perthes disease in about 20%, and SCFE in 25% to 80%. SCFE is more likely to affect both hips in patients who are younger at presentation (<12 years of age), or who have underlying contributing factors such as endocrine or genetic disorders.

6. Which underlying conditions are associated with SCFE?

SCFE has been associated most commonly with obesity. Associated endocrinopathies include hypothyroidism and, more rarely, acromegaly, excess growth hormone, hypopituitarism, and hypogonadism. Hypothyroidism is especially important to consider if the SCFE occurs in a patient under the age of 10. SCFE is also associated with trisomy 21.

7. Explain the importance of modified Klein's line in diagnosing SCFE.

Klein's line is a line drawn along the superior border of the femoral neck on an anteroposterior (AP) radiograph of the hip. This line would normally pass through a portion of the femoral

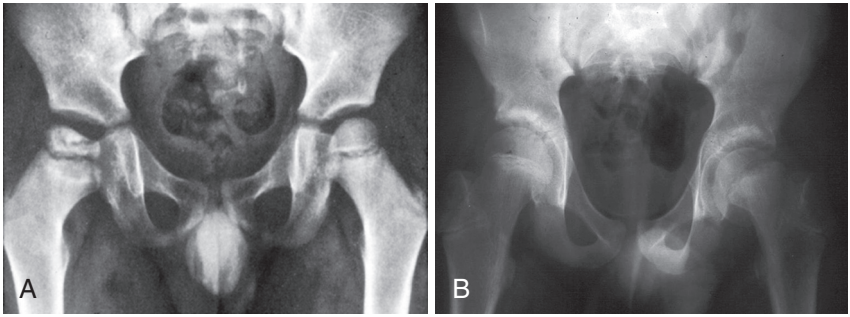


Figure 19-1. **A,** Aseptic necrosis of the femoral head (right hip) in Legg-Calvé-Perthes disease. **B,** Slipped capital femoral epiphysis, more prominent on the left hip ("ice cream slipping off the cone").

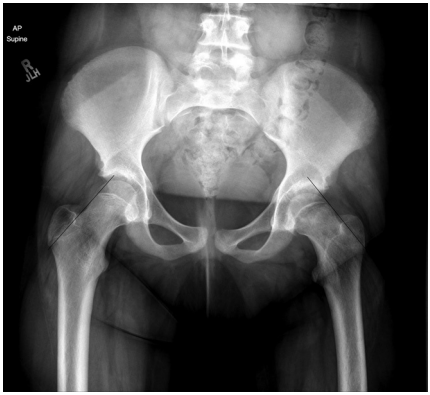
Table 19-1. Common Presentations of Hip Disorders in Children

VARIABLE	DDH	LCP	SCFE
Typical patient	Newborn-toddler; more common in breech positioning; female-male ratio, 9:1	Early school age; male-female ratio, 5:1	Early adolescence; obese; more common in African Americans; male-female ratio, 3:2
Pathologic features	Spectrum of acetabular dysplasia, hip subluxation, and hip dislocation; physiologic and mechanical factors	Avascular necrosis of the femoral head; cause unknown; associated with delayed bone age	Weakness of the femoral head physis; cause unknown (endocrinologic?)
Clinical presentation	Positive result on Barlow test in a newborn; painless limp with hip contracture in a toddler; "waddling" gait if bilateral	Hip or knee pain; insidious limp, with limitation of flexion and internal hip rotation	Can be acute (sudden severe hip or knee pain) or chronic (gradually worsening, mildly antalgic, externally rotated limp)
Best imaging test	Ultrasonography of the hips	MRI of the hips (early); plain radiography later	Plain radiographs (anteroposterior and frog-leg lateral)

DDH, developmental dysplasia of the hip; LCP, Legg-Calvé-Perthes disease; MRI, magnetic resonance imaging; SCFE, slipped capital femoral epiphysis.

Adapted from Brancati DS, Jewell J, Omori M: Evaluation of the child with a limp. Pediatr Emerg Med Rep 2004;9:25-36.

Figure 19-2. Klein's line.



head (Fig. 19-2). If not, SCFE is diagnosed. A proposal has recently been published to modify Klein's line such that we measure the width of the epiphysis lateral to Klein's line, improving sensitivity from 40% to 79% (defining a difference of 2 mm between hips as indicative of a slip). This modification more than doubled the ability to detect SCFE in a retrospective review of 55 patients (from 39% to 87%); however, 100% of the cases were identified on frog-leg lateral radiographs; thus, both AP and frog-leg lateral views should be obtained when SCFE is suspected.

Green DW, Mogekwu N, Scher DM, et al: A modification of Klein's line to improve sensitivity of the A-P radiograph in slipped capital femoral epiphysis. J Pediatr Orthop 2009;29(5):449-453.

Pinkowsky GJ, Hennrikus WL: Klein line on the anteroposterior radiograph is not a sensitive diagnostic radiologic test for slipped capital femoral epiphysis. J Pediatr 2013;162(4):804-807.

8. What is the best test for distinguishing Legg-Calvé-Perthes disease (avascular necrosis of the hip) from transient synovitis?

Tincture of time. The typical duration of transient synovitis is 1 to 2 weeks; Legg-Calvé-Perthes disease is chronic, with a typical course of 18 to 24 months. Radiographs of the hips may be helpful later in the course of the disease. Changes are best seen in the frog-leg lateral position and begin with a small, dense epiphysis and widening of the medial joint space due to cartilage hypertrophy. Later, a subchondral fracture (crescent sign) or fragmentation may be seen. As the disease progresses, reossification and healing result in deformity of the proximal femur.

9. Name a quick and easy screening test for leg-length discrepancy.

The Galeazzi test. This test, typically used to assess hip dislocation in infants, can also be used in toddlers and young children to assess for leg-length inequality. The examination is done with the patient supine and the hips and knees flexed. The test is positive if the height of the knees is asymmetrical. It can also help to determine whether the leg-length inequality is primarily from the femur or the tibia and in assessing leg length in someone with knee or hip flexion contractures.

10. A 4-year-old girl comes to the emergency department (ED) at 3:00 AM for vague leg pain, worse now but present intermittently for 3 weeks. No fever, rash, or trauma is reported. Her primary care provider blames growing pains, or maybe early juvenile idiopathic arthritis (JIA). What test should you perform?

You should order a complete blood count (CBC). This clinical scenario is very concerning for the possibility of acute lymphoblastic leukemia (ALL). A review of 277 children referred to a rheumatology clinic for nonspecific limb pain found the combination of low WBC count, mild thrombocytopenia, and nighttime limb pain had a high likelihood of ALL. Seventy-five percent of the 71 patients found to have ALL did not have blast cells on peripheral smear.

A similar study found that limb pain and thrombocytopenia were significant independent variables for differentiating ALL from JIA, with odds ratios of 553 for limb pain and 754 for thrombocytopenia.

Jones OY, Spencer CH, Bowyer SL, et al: A multicenter case-control study on predictive factors distinguishing childhood leukemia from juvenile rheumatoid arthritis. Pediatrics 2006;117(5):e840-844.
Tamashiro MS, Aikawa NE, Campos LMA, et al: Discrimination of ALL from systemic-onset JIA at disease onset. Clinics (Sao Paulo) 2011;66(10):1665-1669.

11. What is osteochondritis dissecans? What part of the body is most commonly involved?

In osteochondritis dissecans, a portion of articular cartilage and underlying subchondral bone gradually separates from the surrounding tissue. The cause is unknown. The knee, specifically the lateral aspect of the medial femoral condyle or the posterior aspect of the lateral femoral condyle, is the most commonly involved site. The clinical picture is one of intermittent pain that worsens with activity; clicking or locking may also be described. Radiographs may show a delineated, dense fragment of subchondral bone separated from the rest of the affected bone by a radiolucent crescent line. Add the tunnel view to the AP/lateral films if this disease is suspected. The talus, elbow, and rarely the patella can also be affected.

12. A well-appearing toddler is refusing to bear weight. Parents deny trauma and your examination is nonfocal. Is your x-ray study made from the hips down or from the knees down?

Tibial films may be sufficient as an initial screen. Results of a retrospective case-control study looking at 261 patients aged 9 months to 4 years presenting with this clinical picture found an unsuspected fracture in 40 patients—39 of the 40 fractures were located in the tibia and 1 in a metatarsal bone. No patient in the "hips down" group had a pelvic or femur fracture, Legg-Calvé-Perthes disease, or developmental dysplasia of the hip. Tibial films spared radiation to the pelvis and saved $500 per patient compared to total lower extremity films. One patient in the "hips down" group was later found to have diskitis with epidural abscess (initial films negative), emphasizing the need for good follow-up care if no cause is found with initial evaluation.

Baron CM, Seekins J, Hernanz-Schulman M, et al: Utility of total lower extremity radiography investigation of nonweight bearing in the young child. Pediatrics 2008;121(4):e817-e820.

13. A 2-year-old child refuses to walk after jumping off a toy box. He is tender over the distal portion of his left tibia. Plain radiographs of the lower leg are negative. What is the most likely explanation for his pain?

The most likely reason for his pain is a nondisplaced spiral (single helix) fracture of the tibia, also known as a toddler's fracture. This type of fracture may be seen in children from the age of walking up to 5 or 6 years. Although radiographically subtle, the fracture is usually seen on the AP view, rarely on the lateral. Additional views, particularly the internal oblique, may demonstrate the fracture if it is not apparent on the AP view. Even with normal radiographs, if clinical suspicion is high, presumptive treatment with follow-up films in 10 to 14 days is indicated. In the absence of trauma, if occult osteomyelitis is included in the differential diagnosis, bone scintigraphy or magnetic resonance imaging (MRI) can distinguish between the two entities.

14. An oblique view of the tibia in the previously described case reveals a subtle spiral fracture. The resident wants to consult child protective services given the spiral fracture with minimal trauma. What do you recommend?

Unlike spiral fractures of other long bones, even with a history of minor or unobserved trauma, toddler's fractures in an ambulatory child generally do not indicate abuse, and a report is not warranted. Conversely, transverse or metaphyseal fractures of the tibia are concerning for the possibility of inflicted trauma.

15. A 14-year-old boy presents with a limp after twisting his ankle. In performing a complete ankle examination, why should you palpate the lateral aspect of the foot?

The peroneus brevis tendon inserts at the proximal end of the fifth metatarsal, and an ankle inversion may result in an avulsion fracture of this bone. An avulsion fracture is sometimes

mistaken for a less common Jones fracture, located at the metadiaphyseal junction (about 2 cm from the proximal tip of the fifth metatarsal), which has a predominantly horizontal course. The Jones fracture is believed to occur as a result of significant adduction force to the forefoot, and it has a high risk of malunion. Further complicating the interpretation of radiographs in this area is the presence of a normal apophysis in children and young teens. Pain and tenderness over the lateral foot is important in distinguishing normal from abnormal.

16. A young adolescent child presents with a chronically painful flat foot. Radiographs in the past have not been helpful. What condition would you suspect, and what imaging study would be most useful in detecting it?

Tarsal coalition. A coalition is a congenital fibrous band between the talus and calcaneus bones, or occasionally between the calcaneus and the navicular bone. Coronal computed tomography (CT) images obtained through the posterior foot will show narrowing of the subtalar joint, sclerosis, or complete absence of joint space. "Beaking" of the talus on plain films is a clue to this condition.

17. A teenager with upper leg pain and a limp for 2 weeks is found to have a lesion in the proximal femur on plain radiograph. What are the most common benign and malignant bone tumors, and how do you tell the difference?

Adolescence is the peak age of incidence for many types of bone tumor, both benign and malignant. Benign tumors are more common, and their prevalence is unknown as they are frequently asymptomatic and discovered incidentally. Common benign tumors are bone cysts (unicameral and aneurysmal), osteochondromas, and osteoid osteomas. Cysts and chondromas don't typically hurt (unless they cause a pathologic fracture), although osteoid osteomas can be quite painful. The most common malignant pediatric bone tumor is osteosarcoma, followed by Ewing's sarcoma. Ewing's sarcoma has been found to be a primitive neuroectodermal tumor (PNET).

Clues to radiologic distinction between benign and malignant bone tumors are outlined in Table 19-2.

18. A 4-year-old girl presents with limping secondary to knee pain, fever, and a rash. Match the following rashes to the underlying disease.

1. Purpuric rash primarily on the lower extremities	A. Lyme disease
2. Salmon-pink evanescent diffuse maculopapular rash	B. Erythema multiforme
3. Multiple large oval papules, some with central clearing	C. Henoch-Schönlein purpura
4. Symmetrical papulovesicular rash with target lesions	D. JIA

Table 19-2. Radiologic Distinction Between Benign and Malignant Bone Tumors

BENIGN FEATURES	MALIGNANT FEATURES	EXAMPLES
Well-defined or sclerotic border	Poor definition	Osteoid osteoma (benign, small, sclerotic, pain relieved by aspirin or NSAID)
Small, or multiple lesions	Large	Langerhans cell histiocytosis (multiple small lytic lesions)
Lack of cortical destruction	Cortex spiculated or extensive periosteal reaction	Osteosarcoma (spiculated "sunburst" cortical pattern); Ewing's sarcoma (layered "onion skin" periosteal reaction)
Confined to bone	Extension into soft tissue	Benign bone cysts (metaphyseal, may expand bone but don't invade soft tissue)

NSAID, nonsteroidal anti-inflammatory drug.

Answers:
1. C
2. D
3. A
4. B

19. Match the following potential causes of limp with the location of pain.

1. Sever's disease	A. Patella
2. Sinding-Larsen-Johansson syndrome	B. Calcaneus
3. Osgood-Schlatter disease	C. Proximal tibia
4. Kohler's disease	D. Metatarsal
5. Freiberg's disease	E. Navicular

These osteochondroses are a group of lesions that occur in the lower extremity in growing bones, with a clinical picture of localized pain and limp. They are presumed to be related to repetitive stress. Radiographic changes include irregularity, increase in density, and decrease in size of the affected bone.

Answers:
1. B. Sever's disease: insertion of the Achilles tendon on the calcaneus
2. A. Sinding-Larsen-Johansson syndrome: the attachment of the patellar tendon to the inferior patellar pole
3. C. Osgood-Schlatter disease: insertion of the patellar tendon on the proximal tibial tuberosity
4. E. Kohler's disease: the tarsal navicular
5. D. Freiberg's disease: usually the second metatarsal head, less often in the third and fourth

Key Points: Evaluation of a Child with a Limp

1. SCFE is an orthopedic emergency (and may present with referred knee or thigh pain).
2. Limp may be the acute presentation of a more chronic problem, abdominal or back problems, or even systemic illness.
3. Normal laboratory studies do not exclude the possibility of septic arthritis or osteomyelitis.

NECK MASSES

Magdy W. Attia

CHAPTER 20

1. What are the important components of the medical history in a child with a neck mass?

The history should include the following:

- Onset
- Duration of symptoms
- Rapidity of growth with changes in size or position
- Associated pain or tenderness
- History of recent infection or trauma
- Generalized symptoms, such as fever, weight loss, poor appetite, night sweats, or fatigue
- History of exposure to communicable diseases
- Travel history

2. What are the important components of the physical examination of a child with a neck mass?

Observe the general appearance of the child. Screen rapidly for true emergencies. *Inspect* the patient in the neutral position to discern subtle swellings. *Palpate* the neck from behind with the patient seated or with the child on the mother's lap. Examine all masses to determine location, size, multiplicity, consistency, mobility, color, and temperature. Note other characteristics, such as tenderness, compressibility, and mobility with swallowing and with tongue protrusion. Pay special attention to associated systems, such as the chest, abdomen, and lymphatic system.

Davenport M: ABCs of general surgery in children. Lumps and swellings of the head and neck. BMJ 1996;312:368-371.

3. Which true emergencies are associated with neck masses?

True emergencies involving a neck mass include respiratory distress, vascular compromise, and cervical spinal cord compression.

4. Describe an initial focused evaluation of a child with a neck mass.

Address the airway, breathing, and circulation (ABCs) appropriately. Immediately note the child's level of consciousness and work of breathing. Obtain a focused and pertinent history. Evaluate for stridor, hoarseness, dysphagia, and drooling. Perform a neurologic examination.

5. List the etiologic classifications of neck lesions and masses.

The etiologic classifications of neck lesions and masses are listed in Table 20-1.

6. List the anatomic classifications of a neck mass, with the most common masses in each category.

The anatomic classifications of a neck mass, with the most common masses in each category, are listed in Table 20-2.

7. How do you conduct a workup for a child with a neck mass?

Generally, a thorough history and physical examination lead to a provisional diagnosis and initiation of either therapy or watchful waiting. The lack of rapid resolution or the presence of worrisome clinical findings should trigger the workup. The choice of study depends on the type of mass (solid versus cystic, benign versus malignant) and the location and extent of the lesion. Initially, ultrasonography is a good screening study; if further anatomic details are needed, contrast-enhanced computed tomography (CT) or magnetic resonance imaging (MRI) is then indicated. If signs of malignancy are present (i.e., a painless, firm, slow-growing mass in the upper third of the neck, or lymph nodes fixed to underlying tissue) or the lesion does not resolve, then surgical referral and biopsy are warranted. Consider a complete blood count, erythrocyte sedimentation rate (ESR), chest radiography, and purified protein derivative test in the early workup.

Table 20-1. Etiologic Classifications of Neck Lesions and Masses

CONGENITAL	INFECTIOUS	NEOPLASTIC	TRAUMATIC
Hemangiomas	Lymphadenitis	Lymphomas	Subcutaneous emphysema
Cystic hygromas	Soft tissue abscess	Thyroid neoplasms	Hematoma
Preauricular pits, sinuses, and cysts	Infected cysts (i.e., thyroglossal duct, branchial cleft, dermoid)	Teratoma	Cervical spine trauma
Thyroglossal duct cysts		Rhabdomyosarcoma	
		Neuroblastoma	
Sternocleidomastoid mass			
Cervical ribs			
Branchial cleft abnormalities			

Table 20-2. The Anatomic Classifications of a Neck Mass with the Most Common Masses in Each Category

MIDLINE	LATERAL
Thyroglossal duct cyst	Lymphadenopathy
Lymphadenopathy	Cystic hygroma
Dermoid cyst	Branchial cleft cyst
Epidermoid cyst	Sternocleidomastoid mass
Parotitis	Sialadenitis
Ectopic thyroid tissue	Thyroid gland tumors

Gross E, Sichel JY: Congenital neck lesions. Surg Clin North Am 2006;86:383-392.
Turkington JR, Paterson A, Sweeney LE, Thornbury GD: Neck masses in children. Br J Radiol 2005;78:75-85.

8. What are cystic hygromas? When do they usually appear?

Cystic lymphatic malformations are believed to be formed from congenital failure of the lymphatic primordial buds to establish drainage into the venous system. This leads to the accumulation of lymphatic fluid and cyst formation, and a large neck mass. They are discrete, soft, mobile, and nontender masses that are most commonly found in the posterior triangle of the neck. They may be transilluminated and usually grow in proportion to the child.

Cystic hygromas usually appear by the end of the second year of life (80-90% of cases) during the period of active lymphatic growth; they appear less commonly at birth (10-20% of cases).

9. What are the most common locations of cystic hygromas?

They develop in close proximity to large veins and lymphatic ducts:

- Lateral neck (75%)
- Axilla (20%)
- Mediastinum (5%)
- Retroperitoneum (5%)

- Pelvis (5%)
- Groin (5%)

10. What is the most serious complication of cystic hygroma? What are the treatments for this condition?

Respiratory compromise, which is seen when a large mass extrinsically compresses the airway. The treatment is usually surgical excision of the lesion; however, sclerotherapy, repeated aspiration, incision and drainage, radiation therapy, and intralesional injections are other treatment modalities that have been recommended.

Koch BL: Cystic malformations of the neck in children. Pediatr Radiol 2005;35:463-477.

11. What is a thymic cyst? What are the clinical features and treatment?

Thymic cysts result from faulty implantation of thymic tissue in the neck during embryologic descent into the chest. They are present in a midline position, but they can be present anywhere between the angle of the mandible and the midline of the neck. Thymic cysts are managed with surgical excision.

Emerick K, Lin D: Differential diagnosis of a neck mass. UpToDate, 2013. Available from http://www.uptodate.com. Last accessed 3/13/2013.

12. What is the most common head and neck congenital lesion identified in infancy?

Hemangioma. These lesions are most noticeable after the first month of life. Females are affected three times more commonly than males.

13. How do hemangiomas appear on physical examination?

These masses are soft, mobile, and nontender and have a bluish or fiery red hue. Regressing hemangiomas are gray. The most common locations of hemangiomas are on the head:

- Face: lips, nose, eyelids, and ears
- Scalp

14. List some of the complications of hemangiomas.

- Hemorrhage
- Ulceration
- Infection
- Necrosis
- Thrombocytopenia and coagulopathies, due to platelet trapping; Kasabach-Merritt syndrome
- Airway compromise—the child may have stridor
- Congestive heart failure
- Visual impairment with some periorbital hemangiomas

15. What is the origin of branchial cleft cysts?

These cysts develop from malformations of the branchial arches (first through fourth). Most of them arise from the second branchial cleft.

16. How does a child with a branchial cleft sinus or cyst present?

Generally, the child presents with a single painless cystic lesion deep to the anterior upper one third of the sternocleidomastoid. The lesion is movable, smooth, tense, and nontranslucent and occasionally has clear mucoid drainage from a small opening along the anterior border of the sternocleidomastoid muscle. Sometimes the tract is palpable.

17. What is the most common complication of branchial cysts?

Infection is the most common complication, and this usually responds well to antibiotics and warm soaks. The definitive treatment is complete excision of the cyst and tract.

Key Points: Branchial Cleft Cysts

1. Usually a single painless cystic lesion
2. May become infected, and thus red and tender
3. Found on the lateral neck, at the anterior upper third of sternocleidomastoid muscle
4. Treat infection with antibiotics and surgical excision

18. Compare dermoid and epidermoid cysts.
Dermoid and epidermoid cysts are compared in Table 20-3.

19. How is radiologic imaging helpful in management of a dermoid cyst?
Either MRI or CT may be used to evaluate a dermoid cyst prior to complete excision, because dermoid cysts occasionally have intracranial extension.

20. What is a thyroglossal duct cyst?
This common congenital neck mass is due to a ductal ectodermal remnant that never obliterated. Remember, a thyroglossal duct cyst has no external opening, because the tract does not reach the skin surface.

21. How does a thyroglossal duct cyst present clinically?
This cyst is commonly noted in a child 2 to 10 years old as a midline cystic structure, immediately adjacent to the hyoid bone, at or about the level of the thyroid cartilage. Some may be sublingual (3%) or suprasternal (7%). A thyroglossal duct cyst is a soft, smooth, nontender (unless infected) mass that moves when the child swallows or with tongue protrusion.

22. Describe the workup of a child with a thyroglossal duct cyst.
The diagnosis is often made on clinical grounds alone. Ultrasonography, CT, or MRI may be indicated in unusual clinical presentations. CT provides better visualization of lesions adjacent to the hyoid bone. Radioisotope scanning is used for detecting ectopic thyroid tissue within thyroglossal ducts. Postoperative thyroid function tests may be indicated to determine whether thyroid replacement therapy is needed.

Key Points: Thyroglossal Duct Cysts

1. Midline swelling, usually near the thyroid cartilage
2. Moves when the child swallows or protrudes the tongue
3. Diagnosis is made clinically

23. Is cervical lymphadenopathy common in childhood?
Yes, cervical lymphadenopathy is common during the middle years of childhood, but it is seldom pathologic. Children usually present with an indolent, nontender, dominant node in the jugular chain that persists over several months. Systemic symptoms are rare. Although laboratory studies are not usually indicated, most children with cervical lymphadenopathy have a normal blood count and normal chest radiograph, and if a node is biopsied, it shows reactive hyperplasia.

Table 20-3. Comparison between Dermoid and Epidermoid Cysts

FEATURES	DERMOID CYST	EPIDERMOID CYST
Origins	Congenital: inclusion of embryonic epidermis within embryonal fusion planes	Traumatic or inflammatory: follicular infundibulum of the hair shaft
Contents	Lined by epithelium and contains sebaceous glands, hair follicles, connective tissues, and papillae	Lined by epithelium, does not contain cutaneous appendages but rather keratinized cellular debris
Location	Head and neck: lateral to the supraorbital palpebral ridge, midline face, especially nasal bridge or neck	Head and neck: lateral to the supraorbital palpebral ridge, midline face, especially nasal bridge or neck
Treatment	Surgical excision	Surgical excision

24. Is a supraclavicular lymph node more concerning?

Yes. Supraclavicular adenopathy in any age group should be considered pathologic until proved otherwise. Swelling of these nodes may be the first sign of occult thoracic or abdominal disease, such as malignancy (lymphoma). Initiate a workup including a chest radiograph and consult with a surgeon promptly.

25. What is the most common cause of acute cervical lymph node enlargement?

Infection is most often the cause:

- **Viral**: Severe viral pharyngitis can cause cervical lymph nodes to enlarge acutely, usually bilaterally. Infections of the upper respiratory tract can lead to lymph node enlargement. Agents most commonly implicated are Epstein-Barr virus and adenovirus. Also consider HIV (human immunodeficiency virus) infection.
- **Bacterial**: Acute suppurative cervical lymphadenitis secondary to bacterial infection is another cause for cervical lymph node enlargement. Common bacterial pathogens include *Staphylococcus aureus*, group A β-hemolytic streptococci, *Streptococcus pneumoniae* (rarely), anaerobes from mouth flora, and *Bartonella henselae* (cat scratch disease).

26. How does a child with acute suppurative cervical lymphadenitis present?

Acute suppurative cervical lymphadenitis often presents after an upper respiratory tract infection, pharyngitis, or tonsillitis, with rapid, unilateral enlargement of multiple adjacent lymph nodes. Marked tenderness, warmth, and erythema are often seen. Fever, irritability, and toxic appearance may be seen in the younger child. Spontaneous purulent drainage may occur if the infection is untreated. Occasionally, the infected cervical lymph nodes herald a retropharyngeal abscess. Consider this entity in the differential diagnosis of cervical adenitis.

27. How is acute suppurative cervical lymphadenitis best treated?

The initial treatment consists of oral antibiotics and warm soaks to the neck. If the child is young and toxic, admission is warranted for intravenous (IV) antibiotic therapy and close observation. Direct the initial antimicrobial therapy to the common organisms listed. Suspect MRSA (methicillin-resistant *S. aureus*) if initial therapy is ineffective. When fluctuance is identified, drainage by surgical incision or ultrasonography-guided needle aspiration is recommended for both therapeutic and diagnostic purposes.

28. What is the differential diagnosis of subacute or chronic cervical lymphadenitis?

This condition refers to a child with a large, minimally tender, mildly inflamed, and nonfluctuant cervical lymph node that appears slowly over several weeks with no associated prodrome or systemic illness. The differential diagnosis includes the following:

- Reactive response to a nonspecific infection (bacterial or viral)
- Cat scratch disease
- Infectious mononucleosis
- *Mycobacterium* tuberculosis: typical or atypical
- Toxoplasmosis
- Cytomegalovirus infection
- HIV infection
- Sarcoidosis
- Histoplasmosis
- Actinomycosis
- Malignancy

29. What is the workup for a child with subacute or chronic cervical lymphadenitis?

- Perform a complete history and physical examination.
- Note the child's overall general appearance.
- Assess the number, size, and location of lymph nodes.
- Palpate for any organomegaly.
- Consider laboratory studies, including a complete blood count with differential, ESR, chest radiograph, and purified protein derivative test.
- Begin antibiotics to cover staphylococcus and streptococcus.
- Arrange for the patient to be followed closely by his or her primary care physician.

30. When should a child with chronic lymphadenopathy be referred for biopsy?

Referral is warranted if the condition does not respond to treatment or a lymph node persists or enlarges despite adequate antibiotic therapy (of a few weeks' duration).

31. Describe the clinical picture of cat scratch disease.

Cat scratch disease is a common infectious disease in all age groups. The child presents with a tender node unilaterally. Kittens, in particular, are the reservoir for the pathogen. Children with cat scratch disease may give a history of a scratch by a kitten or only exposure to a kitten. Although most children do not recall the scratch, a papule at the primary inoculation "scratch site" and regional lymphadenopathy usually develop in 1 to 2 weeks. A positive result on a cat scratch disease antigen skin test (not recommended) or demonstration of the microorganism (*B. henselae*) by silver stain from an aspirated node is confirmatory. If cat scratch disease is suspected, the best initial test is the *B. henselae* IgM enzyme immunoassay. If the test result is negative, consider a polymerase chain reaction (PCR) for *Bartonella* by using the biopsy tissue.

32. How should I test for cat scratch disease?

The diagnosis can be easily established clinically in most cases. There are a number of confirmatory tests:

1. **Serologic test**: This type of test can be helpful; however, there are considerable limitations that are due to both suboptimal sensitivity and false-positive rates. Indirect fluorescence assay titers of IgG higher than 1:256 (rises 10-14 days after infection) and positive IgM (rises early and briefly) tests strongly suggest either active or recent cat scratch disease. The serologic diagnosis of acute infection can be elusive because of the timing of the rise of these titers.
2. **Blood and tissue culture**: Growing *B. henselae* requires specific laboratory conditions for optimal growth, rendering it impractical. Histopathologic examination of the initial entry site and the involved gland can be supportive of the diagnosis in the presence of other criteria. This testing is not routinely performed.
3. **PCR**: Though highly specific, the sensitivity of this test remains a significant limitation.
4. **Skin testing**: This test is no longer used.

Vermeulen MJ, Diederen BM, Verbakel H, Peeters MF: Low sensitivity of Bartonella henselae PCR in serum samples of patients with cat-scratch disease lymphadenitis. J Med Microbiol 2008;57 (Pt 8):1049-1050.

33. What is the treatment for cat scratch disease?

Cat scratch disease is a self-limited condition. Resolution is expected in 6 to 8 weeks. Initial treatment may consist of warm compresses and analgesics. Reserve antibiotic treatment for those with systemic symptoms or a severe local reaction. However, many physicians start antibiotics, such as trimethoprim-sulfamethoxazole or azithromycin, upon clinical suspicion.

Key Points: Cat Scratch Disease

1. Most patients do not recall a cat scratch.
2. Kittens are usually the reservoir.
3. Lymphadenopathy develops 1 to 2 weeks after the scratch.
4. *B. henselae* IgM enzyme immunoassay is the best test during the acute phase.
5. Treat with observation or trimethoprim-sulfamethoxazole or azithromycin.

34. What other conditions should be considered in the differential diagnosis of a unilateral neck mass?

A unilateral neck mass consisting of a nontender 1.5-cm or larger cervical lymph node associated with fever raises the possibility of mucocutaneous lymph node syndrome or Kawasaki disease. This disease is associated with severe vasculitis of medium-size blood vessels with predilection for the coronary arteries.

The acronym FRAME is easy to remember, and it lists the typical findings: *f*ever for 1 to 2 weeks with 39° C or higher; *r*ash of various forms and desquamation of the skin of the hands,

feet, and occasionally perianal area; *a*denopathy (as described earlier); *m*ucositis involving the bulbar conjunctivae, oral mucosa, lips, and external urethral meatus; *e*dema and redness of the hands and feet. Other findings include irritability and fatigue.

Early diagnosis is suspected clinically and confirmed by elevated sedimentation rate and platelet counts. Findings include mild normocytic anemia, sterile pyuria, and abnormal lipid profile. An emergent echocardiogram for the early detection of coronary aneurysms is a must. When the diagnosis is suspected, initiate therapy to avoid myocardial ischemia or infarction.

Baer AZ, Rubin LG, Shapiro CA, et al: Prevalence of coronary artery lesions on the initial echocardiogram in Kawasaki syndrome. Arch Pediatr Adolesc Med 2006;160(7):686-690.

35. Describe the most common neoplasm of the neck based on age.

Approximately 5% of all neoplastic lesions occur in the head and neck region.

- In preschool-age children: Neuroblastoma, non-Hodgkin's lymphoma, rhabdomyosarcoma, and Hodgkin's lymphoma are most common.
- In school-age children: Lymphoma, either non-Hodgkin's or Hodgkin's, thyroid carcinoma, and rhabdomyosarcoma are more likely.
- In adolescents: Hodgkin's lymphoma predominates.

36. When should a neoplastic lesion in the neck be suspected? What evaluation is necessary?

Suspect a neoplastic lesion when a child presents with a painless, firm, fixed cervical mass. Other presenting signs and symptoms are unilateral ptosis, nasal obstruction, and otorrhea. A detailed history and physical examination, complete blood count with differential, renal and liver profiles, chest radiograph, or CT/MRI are sometimes obtained in the emergency department. Urgent referral to a surgeon or oncologist is recommended for further studies, such as a bone marrow examination.

Acknowledgment

The author wishes to thank Dr. Eileen Quintana for her contributions to this chapter in the previous edition.

CHAPTER 21

PEDIATRIC RASHES

Ronald I. Paul

1. Name five bioterrorism agents that may have skin manifestations.

Five bioterrorism agents that may have skin manifestations are listed in Table 21-1.

http://dermatology.about.com/cs/bioterrorism/

O'Brien KK, Higdon ML, Halverson JJ: Recognition and management of bioterrorism infections. Am Fam Physician 2003;67:1927-1934.

2. What are four skin findings associated with syphilis?

Four skin findings associated with syphilis can be found in Table 21-2.

Sexually transmitted disease. Syphilis pictures. Available at http://herpes-coldsores.com/std/syphilis_pictures.htm.

3. How does perianal streptococcal dermatitis manifest?

Perianal streptococcal infection usually occurs in children between 6 months and 10 years of age. It presents as sharply circumscribed superficial perianal erythema. In some patients, the rash is bright red with a wet surface, and in others it is dry and pink. Fever is generally absent. Delays in culturing, diagnosing, and initiating therapy are common.

Block SL: Perianal dermatitis: Much more than just a diaper rash. Pediatr Ann 2013; 42(1):12-14.

Herbst RA: Perineal streptococcal dermatitis/disease: Recognition and management. Am J Clin Dermatol 2003;4:555-560.

4. An atopic child with chronic eczema suddenly develops a painful vesicular eruption in previous areas of eczema. What is the most likely diagnosis?

Eczema herpeticum, caused by the herpes simplex virus, is a vesicular eruption concentrated in areas of eczematous skin. Children with eczema herpeticum often become seriously ill with high fever because the disease can be complicated by secondary bacterial infections and viremia, leading to multiple organ involvement with meningitis and encephalitis. Treatment with intravenous (IV) acyclovir is necessary for children with extensive or rapidly progressing involvement (Fig. 21-1).

Wollenberg A, Wetzel S, Burgdorf HC, et al: Viral infections in atopic dermatitis: Pathogenic aspects and clinical management. J Allergy Clin Immunol 2003;112:667-674.

5. What are the clinical features of measles?

Measles, caused by an RNA virus in the paramyxovirus family, has almost been eradicated in the United States. Epidemics were seen most recently in the early 1990s, and sporadic cases still occur among travelers and immigrants. It is an acute disease characterized by fever, cough, coryza, conjunctivitis, and an erythematous maculopapular rash that begins on the forehead and behind the ears, spreading to the face, neck, torso, and extremities. Koplik spots, bright red punctae with central white flecks on the buccal mucosa near the second molars, are seen early in the disease course and are pathognomonic for measles.

American Academy of Pediatrics: Measles. In Pickering LK, Baker CJ, Kimberlin DW, et al (eds): Red Book: 2012 Report of the Committee on Infection Diseases, 29th ed. Elk Grove Village, IL, American Academy of Pediatrics, 2012, pp 489-499.

6. How can one differentiate between erythema multiforme (EM), Stevens-Johnson syndrome (SJS), and toxic epidermal necrolysis (TEN)?

EM is now thought to be a separate process from SJS and TEN. It is an erythematous maculopapular rash, characterized by target lesions that may coalesce and develop annular or serpiginous borders. The course is brief and EM usually resolves within 2 weeks, although recurrences are common.

Table 21-1. Bioterrorism Agents That May Have Skin Manifestations

AGENT	SKIN FINDINGS
Smallpox	Maculopapular rash on face, forearms, and mucous membranes that becomes vesicular/pustular within 48 hours
Anthrax	Painless pruritic papule on skin that develops into a painless ulcerated black eschar within a few days
Tularemia	Painful maculopapular lesion that ulcerates; associated with painful inflamed regional lymph nodes
Plague	Acutely swollen lymph nodes called buboes
Viral hemorrhagic fever	Maculopapular rash on trunk followed by mucosal bleeding

Table 21-2. Skin Findings Associated with Syphilis

FINDING	DESCRIPTION
Chancre	Painless ulcer of skin and mucous membranes at site of inoculation
Rash	Maculopapular rash of secondary syphilis frequently involving palms and soles
Condylomata lata	Cauliflower-appearing warts on penis, labia, or rectum
Gumma	Painless pink to dusky red nodules of various sizes that may necrose or ulcerate

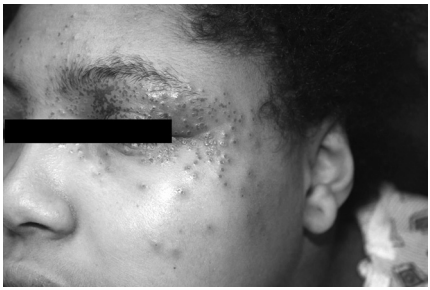


Figure 21-1. Adolescent with diffuse eczema herpeticum on face.

SJS and TEN are now thought to represent a spectrum of the same disease. They are severe drug eruptions, and TEN has a mortality rate of around 30%. SJS is characterized by febrile erosive stomatitis, ocular involvement, and a diffuse rash of discrete dark red macules, sometimes with a necrotic center. TEN involves extensive loss of epidermis due to necrosis that leaves the skin surface looking scalded.

Bachot N, Roujeau JC: Differential diagnosis of severe cutaneous drug eruptions. Am J Clin Dermatol 2003;4:561-572.

Weston WL, Lane AJ, Morelli JG: Drug eruptions. In Weston WL, Lane AJ, Morelli JG: Color Textbook of Pediatric Dermatology, 4th ed. St. Louis, Mosby, 2007, pp 365-380.

7. How does the rash of Rocky Mountain spotted fever change over time?

The rash is initially erythematous and macular but later becomes more papular and, frequently, petechial. The rash first appears on the wrists and ankles, spreading centrally within hours to involve the proximal extremities and trunk. The palms and soles are usually involved.

8. A child helps his mother cut limes before going outside to play and then returns with inflamed hands that quickly develop hyperpigmentation. What is the name of the rash and the causative agent?

This condition is a type of toxic photoreaction called phytophotodermatitis. It is caused when the skin comes into contact with psoralen, a photosensitizing agent, and then immediately with sunlight. Psoralen can be found in some perfumes, plants, grasses, fruits, and vegetables. Limes, celery, and parsley are examples of foods that contain psoralen.

Bergeson PS, Weiss JC: Picture of the month. Phytophotodermatitis. Arch Pediatr Adolesc Med 2000;154:201-202.

9. What are the typical features of pityriasis rosea?

Seen primarily in adolescents, pityriasis rosea begins in 80% of patients with a large, oval, solitary lesion known as a *herald patch* somewhere on the trunk or upper thighs. This is followed by an eruption of smaller, oval, and slightly raised papules that are pink to brown and have peripheral scales. The lesions are described as having a Christmas tree pattern on the back and involve mostly the truncal areas (Fig. 21-2), usually sparing the face, scalp, and distal extremities. Eruption is prolonged and can last 4 to 8 weeks.

Pityriasis rosea pictures. Available at http://hardinmd.lib.uiowa.edu/cdc/pityriasis.html.

10. List key features that help differentiate the purpuric rash of Henoch-Schönlein purpura from more serious infectious purpuric rashes, such as purpura fulminans.

See Table 21-3 for features that help differentiate Henoch-Schönlein purpura from purpura fulminans.

11. What are five skin manifestations that may be seen in Kawasaki disease?

1. Dry, cracked erythematous lips
2. Erythematous polymorphic truncal rash that may be scarlatiniform or morbilliform
3. Red, swollen hands and feet
4. Peeling around nails and fingers
5. Desquamating perineal rash

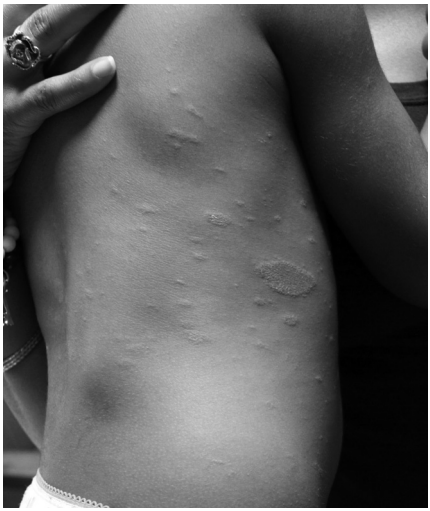


Figure 21-2. Pityriasis rosea with typical small oval lesions and larger herald patch.

Table 21-3. Henoch-Schönlein Purpura Versus Purpura Fulminans

HENOCH-SCHÖNLEIN PURPURA	PURPURA FULMINANS
Distribution usually limited to extremities, appearing most commonly on lower legs, buttocks, and occasionally upper extremities. In infants, facial involvement may be seen.	Distribution of purpura is widespread.
Associated features include arthralgias, abdominal pain, and hematuria.	Associated features include lethargy, hypoventilation, and shock.
Children appear well except for painful joints and abdominal pain.	Children appear ill, with varying degrees of toxicity.
Platelet count and results of other coagulation tests are normal.	Thrombocytopenia is present, and coagulation test results are abnormal.

Eleftheriou D, Levin M, Shingadia D, et al: Management of Kawasaki disease. Arch Dis Child 2014;99(1):74-83.

12. What are some skin findings that may be mistaken for child abuse?

See Table 21-4 for skin findings that may be mistaken for child abuse.

Mudd SS, Findlay JS: The cutaneous manifestations and common mimickers of physical child abuse. J Pediatr Health Care 2004;18:123-129.

13. How does scabies manifest in infants?

Scabies in infants and young toddlers may be more eczematous and less typical than in adults. In addition to the characteristic burrows on the hands and feet, young infants may present with diffuse dermatitis on the trunk, face, neck, and scalp. The axilla, diaper area, palms, and soles are frequent sites of distribution. Because infants are not able to scratch the intensely pruritic lesions, they may become irritable and sleep poorly.

Gunning K, Pippitt K, Kiraly B, et al: Pediculosis and scabies: Treatment update. Am Fam Physician 2012;15:535-554.

14. What are the cutaneous manifestations of disseminated gonococcemia?

The skin lesions associated with disseminated gonococcemia are often located on the extremities and may overlie the involved joints. Small macules appear initially and progress to papules. These tender lesions may develop a small vesicle and then a gray umbilicated center. A diagnosis can be established with a Gram stain or culture of the skin lesion.

15. Is poison ivy contagious?

No. A common misconception among families and school officials is that the rash of poison ivy is contagious. This belief occurs because of the vesicles and weepy bullae that develop in some individuals. Once the skin, clothes, and fingernails are cleaned of the sap from this treacherous plant, the rash will not spread beyond the areas already exposed. Contributing to this

Table 21-4. Skin Findings That May Be Mistaken for Child Abuse

FINDING	DESCRIPTION
Lichens sclerosis	Indurated and shiny atrophic plaques found in vulvar and perianal areas
Mongolian spots	Hyperpigmented areas commonly seen over sacrum
Coining	Asian folk remedy of rubbing coin or spoon on back and trunk to rid body of "bad winds"
Accidental ecchymoses	Normal childhood bruises found over bony prominences, such as shins, knees, forearms, elbows, foreheads, and chins

misconception is the fact that areas with minimal contamination can develop 5 to 10 days after the heavily contaminated areas appear. The bottom line is the child should go back to school!

16. Should topical agents be used to treat poison ivy?

Unfortunately, good studies have shown that topical hydrocortisone and topical antihistamine creams have no more effect on poison ivy than do bland, soothing lotions, such as calamine. To be effective, use steroid creams and ointments that are at least of moderate potency. Supportive care includes baths, cool compresses, and calamine lotion for mild cases. Systemic oral steroids almost always bring the acute dermatitis under control and are appropriate for moderate to severe cases. Oral antihistamines may also be helpful.

Ianelli V: Poison ivy pictures. Available at http://pediatrics.about.com/od/poisonivy/ig/Poison-Ivy-Pictures/Poison-Ivy-Rash.–PF.htm.

Weston WL, Lane AJ, Morelli JG: Dermatitis. In Weston WL, Lane AJ, Morelli JG: Color Textbook of Pediatric Dermatology, 4th ed. St. Louis, Mosby, 2007, pp 51-55.

17. What is a pyogenic granuloma?

A pyogenic granuloma is a rapidly growing vascular proliferation that develops at the site of an obvious or unnoticed trauma (Fig. 21-3). Despite its name, this lesion is not infectious. Patients usually present to the emergency department with spontaneous bleeding, or after local minor trauma. Acute bleeding can be controlled with prolonged pressure or silver nitrate sticks. Ultimately, treatment consists of electrodessication and curettage.

18. Transient pustular melanosis and erythema toxicum neonatorum are two newborn rashes; how do they differ?

Transient pustular melanosis is a benign newborn rash consisting of superficial vesiculopustular lesions that are present at birth. The lesions rupture easily with the first bath, leaving a collarette of fine, white scales and brown, hyperpigmented macules. Lesions fade within several weeks to months and are asymptomatic. Erythema toxicum neonatorum is a benign, self-limited eruption that usually appears during the first 3 to 4 days of life and sometimes as late as day 10. The lesions begin as blotchy erythema that develops into pale yellow or white papules or pustules. Individual lesions last an average of 2 days, and cytologic examination, if needed to differentiate from other rashes, reveals clusters of eosinophils and neutrophils and an absence of bacteria.

O'Connor NR, McLaughlin MR, Ham P: Newborn skin: Part 1. Common rashes. Am Fam Physician. 2008;77:47-52.

19. What is the appropriate treatment for a herpetic whitlow?

A herpetic whitlow is a localized herpes simplex infection consisting of a single or multiple vesicular lesions on the distal fingers or toes. The first episode may accompany herpetic gingivostomatitis. Treatment consists of local care for mild cases, and those with severe lesions may benefit from oral acyclovir. Avoid incision and drainage, which can prolong recovery and worsen the condition.

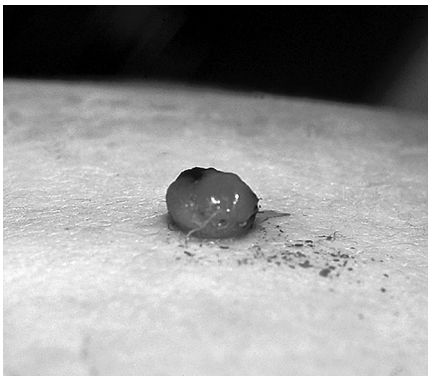


Figure 21-3. Pyogenic granuloma. *(From Morelli JG: Vascular neoplasms. In Fitzpatrick JE, Morelli JG [eds]: Dermatology Secrets in Color, 3rd ed. Philadelphia, Mosby, 2007, Fig. 42-7, p 351.)*

Herpetic whitlow. Painful grouped red-blue vesicles on the middle finger of a child. Available at http://www.medicinenet.com/image-collection/herpetic_whitlow_picture/picture.htm.
Wu IB, Schwartz RA: Herpetic whitlow. Cutis 2007;79:193-196.

20. What condition is associated with recurrent pustules on the feet of infants?

Infantile acropustulosis consists of 7- to 10-day episodes of pruritic pustules and papulovesicles on the hands and feet of infants. The age of onset is usually between 2 and 10 months, and the condition resolves by 2 to 3 years of age. Treatment with mid- to high-potency topical corticosteroids is effective.

Dromy R, Raz A, Metzker A: Infantile acropustulosis. Pediatr Dermatol 1991;8:284-287.
Weston WL, Lane AJ, Morelli JG: Skin diseases in newborns. In Weston WL, Lane AJ, Morelli JG: Color Textbook of Pediatric Dermatology, 4th ed. St. Louis, Mosby, 2007, pp 381-411.

21. A diaper rash consisting of diffuse papular, scaly, and fissuring eruptions does not respond to anti-inflammatory or antifungal agents. What is a potential serious cause?

This rash may be one of the skin manifestations of histiocytosis X, which is a disorder characterized by proliferation of Langerhans histiocytes in the skin and other organ systems. Consider a biopsy to confirm the diagnosis or absence of histiocytosis X when a difficult-to-treat diaper rash does not improve with standard therapy.

Histiocytosis X. Available at http://www.dermis.net/dermisroot/en/12377/image.htm.

22. What is Nikolsky's sign?

Nikolsky's sign is a vulnerability of the skin such that apparently normal epidermis can be rubbed off with slight trauma. It may be seen in epidermal blistering diseases, such as scalded skin syndrome and pemphigus vulgaris.

23. How long do patients with erythema infectiosum (fifth disease) need isolation?

This infection is caused by parvovirus B19. For most patients with the typical presentation of a "slapped cheek" and a lacelike rash on the arms and legs, no isolation is needed at the time of diagnosis. By the time the rash becomes clinically obvious, these patients are unlikely to be infectious. The rash usually resolves within 3 to 5 days of onset.

American Academy of Pediatrics: Parvovirus B19. In Pickering LK, Baker CJ, Kimberlin DW, et al (eds): Red Book: 2012 Report of the Committee on Infectious Diseases, 29th ed. Elk Grove Village, IL, American Academy of Pediatrics, 2012, pp 539-541.

24. What are the similarities and differences between granuloma annulare and tinea corporis?

Both are characterized by circular plaques consisting of a ring of papules around a depressed center. Granuloma annulare is a ring of *nonscaling* skin-colored or red papules that are most commonly seen on the dorsal surface of the hands and feet. They are asymptomatic and resolve spontaneously after a few months to a year. Tinea corporis is a common, superficial fungal infection consisting of lesions with *scaly* inflammatory borders that appear anywhere on the body. There may be multiple lesions.

25. What causes roseola? What are the usual features?

Roseola infantum (exanthem subitum) is an acute febrile illness that primarily affects young children between the ages of 6 and 36 months. Most cases are now thought to be caused by human herpesvirus 6 and human herpesvirus 7. After 3 or more days of high fever, the patient abruptly defervesces and an erythematous, morbilliform rash with discrete rose-pink macules appears. The rash begins first on the trunk and then spreads rapidly to the extremities, neck, and face. By this time, the rash is bothering the parents more than the patient.

American Academy of Pediatrics: Human herpes virus 6 (including roseola) and 7. In Pickering LK, Baker CJ, Kimberlin DW, et al (eds): Red Book: 2012 Report of the Committee on Infectious Diseases, 29th ed. Elk Grove Village, IL, American Academy of Pediatrics, 2012, pp 539-541.

26. What are the usual findings in Gianotti-Crosti syndrome?

This self-limiting condition is characterized by symmetrical erythematous papules that are primarily found on the extremities but often involve the cheeks and buttocks. Also known as

papular acrodermatitis, the rash may last 2 to 8 weeks, and may become recurrent. Although first identified in Europe in children with hepatitis B, it is seen in the United States primarily in association with other viruses, including Epstein-Barr virus, cytomegalovirus, and coxsackievirus A16.

Weston WL, Lane AJ, Morelli JG: Viral infections. In Weston WL, Lane AJ, Morelli JG: Color Textbook of Pediatric Dermatology, 4th ed. St. Louis, Mosby, 2007, pp 113-147.

27. A toddler presents with several small, round, erythematous, and inflamed macules in a straight column down the middle of his chest and abdomen. What is the most likely cause?

This is likely to be a contact dermatitis related to allergy to nickel. Infants commonly present with skin lesions corresponding to the location of snaps on their pajamas or other garments. Avoidance of the offending object, by using an undershirt or nonsnap clothes, and application of a mild topical steroid will solve the problem.

Mayo Foundation for Medical Education and Research: Nickel allergy. Available at http://www.mayoclinic.com/health/medical/IM00384.

28. Which organisms cause bullous and nonbullous impetigo?

- Bullous impetigo involves vesicles and is usually caused by toxin-producing strains of *Staphylococcus aureus*.
- Nonbullous impetigo consists of small vesicles or pustules that rupture and then develop honey-colored, crusting lesions. It is mostly caused by group A streptococcus.

29. What are the dermal manifestations of zinc deficiency?

Dietary deficiency or inadequate absorption of zinc leads to acrodermatitis enteropathica. It is characterized by erythema, crusting, and fissuring of the perioral skin and cheeks. The diaper area may develop a diffusely erythematous rash with a sharply marginated border on the abdomen. Psoriasiform lesions may develop around the anus and on the buttocks and feet. Treatment with dietary zinc supplementation provides dramatic resolution of all dermal and systemic symptoms.

Chumpitazi CE, Tran JQ: Images in emergency medicine. Child with diarrhea and rash. Acrodermatitis enteropathica. Ann Emerg Med 2013;62(4):303,318.

Perafan-Riveros C, Franca LF, Alves AC, et al: Acrodermatitis enteropathica: Case report and review of the literature. Pediatr Dermatol 2002;19:426-431.

Ravanfar P, Wallace JS, Pace NC: Diaper dermatitis: A review and update. Curr Opin Pediatr 2012;24(4):472-479.

30. Compare and contrast smallpox with chickenpox.

Smallpox and chickenpox are compared in Table 21-5.

Koenig KL, Boatright C: Derm and doom: The common rashes of chemical and biological terrorism. Critic Decis Emerg Med 2003;17:1-7.

Table 21-5. Smallpox Versus Chickenpox

CHARACTERISTIC	SMALLPOX	CHICKENPOX
History	Febrile with systemic symptoms for several days prior to rash	Mild fever with minimal symptoms for 1-2 days prior to rash
Severity	Very ill from start	Not severely ill unless complications develop
Lesions	Hard circumscribed pustules	Vesicles on an erythematous base
Distribution	Face and distal extremities, involving palms and soles	Face and trunk, with no involvement of palms or soles
Lesion development	Slow, with all lesions at same stage of development	Rapid, with lesions at different stages

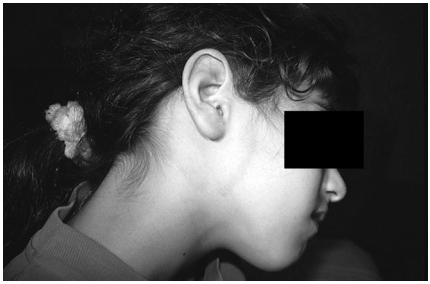


Figure 21-4. Patient with erythema migrans rash from Lyme disease.

31. Describe and name the skin finding associated with Lyme disease.

Erythema chronicum migrans (ECM) develops 4 to 20 days after a tick bite in 60% to 70% of patients who have contracted Lyme disease (Fig. 21-4). The first sign may be a red papule at the site of the tick bite. An annular ring with a flat border slowly grows while clearing develops in the center. Some patients develop multiple secondary annular rings days after the primary lesion appears.

Erythema migrans. Available at http://www.picsearch.com/Erythema-migrans-pictures.html.

Weston WL, Lane AJ, Morelli JG: Bacterial infections (pyodermas) and spirochetal infections of the skin. In Weston WL, Lane AJ, Morelli JG: Color Textbook of Pediatric Dermatology, 4th ed. St. Louis, Mosby, 2007, pp 61-80.

32. What is the typical skin finding following a bite from the brown recluse spider?

Loxosceles recluse spiders are often found in old buildings and produce a number of toxins in their venom. A painful hemorrhagic blister may initially appear. Over several days a dry gangrenous eschar will develop.

Swanson DL, Vetter RS: Bites of brown recluse spiders and suspected necrotic arachnidism. N Engl J Med 2005;352:700-707.

Vetter R: Identifying and misidentifying the brown recluse spider. Dermatol Online J 1999;5(2):7. Available at http://dermatology.cdlib.org/DOJvol5num2/special/recluse.html.

33. What does the term *morbilliform* mean?

Morbilliform literally means "measles-like." Eruptions that are generalized, discrete red to pink macules are referred to as being morbilliform. Morbilliform rashes are seen in association with several viral infections, including rubella, enterovirus, adenovirus, roseola, parvovirus, and Epstein-Barr virus.

34. A 7-year-old child presents with four café-au-lait spots that are 2 to 4 mm in diameter. Does this child have neurofibromatosis?

Probably not. The diagnostic criteria for neurofibromatosis type 1 include six or more café-au-lait spots greater than 5 mm in diameter in prepubertal children and greater than 15 mm in older children.

Listernick R, Charrow J: Neurofibromatosis-1 in childhood. Adv Dermatol 2004; 20:75-115.

Nunley KS, Gao F, Albers AC, et al: Predictive value of café au lait macules at initial consultation in the diagnosis of neurofibromatosis type 1. Arch Dermatol 2009;145(8):883.

35. What common skin features are present in children with scarlet fever?

- Flushed face with perioral pallor
- Diffuse, blanching, erythematous rash that has a sandpaper consistency, with accentuation in the axillae and groin
- Pastia's lines in the flexural surface of the elbows.
- Desquamation as the acute phase of illness resolves

36. How can you distinguish irritant contact diaper rash from candidal diaper dermatitis?

- Generic diaper rash is caused by contact of the skin with urine or feces in the moist, closed environment created by a diaper. Red papules or patches appear on the prominent surfaces of the diapered areas, especially in areas of overlapping skinfolds and skin directly adjacent to the plastic parts of the diaper or elastic.
- Candidal diaper dermatitis comes from the gut flora *Candida albicans*. Features of this infectious rash consist of perianal erythema and maceration spreading to produce moist, bright red, confluent plaques in the diaper area, especially in the intertriginous folds. Satellite lesions are common.

37. How long does acne neonatorum last?

Lesions from acne neonatorum are primarily on the face, upper chest, and back in a distribution similar to that seen in adolescents. They usually start around 3 weeks of age and last for 4 months. Most infants have resolution of the rash by 6 months of age.

Weston WL, Lane AJ, Morelli JG: Acne. In Weston WL, Lane AJ, Morelli JG: Color Textbook of Pediatric Dermatology, 4th ed. St. Louis, Mosby, 2007, pp 25-38.

38. Name five cutaneous manifestations of lupus erythematosus.

1. Erythematous maculopapular eruption on cheeks and nose in a butterfly distribution
2. Discoid lesions—chronic persistent skin changes that progress to scarring and pigmentary changes
3. Transient annular papulosquamous lesions in sun-exposed areas of skin
4. Erythema on the palms and pulps of the fingers and diffuse erythematous scaly macules on the dorsum of the fingers
5. Pallor and cyanosis of the digits when exposed to cold (Raynaud's phenomenon)

Weston WL, Lane AJ, Morelli JG: Papulosquamous disorders. In Weston WL, Lane AJ, Morelli JG: Color Textbook of Pediatric Dermatology, 4th ed. St. Louis, Mosby, 2007, pp 149-180.

39. What are the two main types of epidermolysis bullosa (EB)?

Epidermolysis bullosa is used to describe a group of inherited conditions that result in blisters after mild trauma (Fig. 21-5). There are two main categories:

- Nonscarring types (EB simplex and junctional type EB)
- Scarring or dystrophic forms of EB

Epidermolysis bullosa photos. Available at http://www.dermnet.com/images/Epidermolysis-Bullosa.
Weston WL, Lane AJ, Morelli JG: Genodermatoses I. In Weston WL, Lane AJ, Morelli JG: Color Textbook of Pediatric Dermatology, 4th ed. St. Louis, Mosby, 2007, pp 343-364.

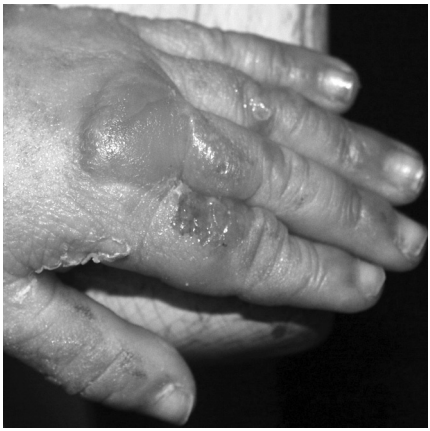


Figure 21-5. Blistering rash on a child with epidermolysis bullosa following minor hand trauma.

Key Points: Bioterrorism Agents with Dermatologic Findings

1. Smallpox
2. Anthrax
3. Tularemia
4. Plague
5. Hemorrhagic viral fevers

Key Points: Signs and Symptoms of Scabies in Infancy

1. Infant is often irritable.
2. Lesions may appear more eczematous than in older children.
3. Characteristic burrows are seen on the hands and feet.
4. Axillae, groin, palms, and soles are often involved.
5. Dermatitis may be present on trunk, face, neck, and scalp.

CHAPTER 22

RED EYE

Kathy Palmer

1. What are the associated features of a "red eye" that may be dangerous?

- Severe ocular pain
- Photophobia
- Persistent blurred vision or decreased visual acuity
- Proptosis
- Irregular corneal light reflection
- Worsening signs after 3 days of pharmacologic treatment
- Corneal epithelial defect or opacity
- Pupil unreactive to direct light
- Ciliary flush
- Reduced ocular movements
- Compromised host: neonate, immunosuppressed patient, and contact lens wearer
- Severe headache with nausea

The serious conditions that cause a red eye will cause 360-degree involvement of the bulbar conjunctiva, often in a ciliary flush pattern, but will spare the tarsal conjunctiva.

Jacobs DS: Conjunctivitis. UpToDate, 2013. Available from http://www.uptodate.com.

2. What should be included in the differential diagnosis for a red eye in a pediatric patient?

- Abnormalities of the lids or lashes
- Conjunctivitis (allergic, bacterial, viral, or chemical)
- Periorbital (preseptal) or orbital cellulitis
- Corneal abrasion (trauma)
- Chemical burn
- Subconjunctival hemorrhage
- Contact lens–related problems
- Ocular inflammation from systemic disease (uveitis, episcleritis)
- Neoplasms
- Foreign body

Levin AV: Eye—Red. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 240-244.

3. What systemic conditions are associated with red eyes?

Collagen vascular disorders, juvenile rheumatoid arthritis, infectious diseases (varicella, measles, mumps, otitis media), Kawasaki disease, inflammatory bowel disease, cystic fibrosis, Stevens-Johnson syndrome.

Levin AV: Eye—Red. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 240-244.

4. What are the clinical characteristics of bacterial conjunctivitis, and how can this be treated?

Bacterial conjunctivitis is characterized by mucopurulent discharge that involves one eye and usually spreads to the second eye, associated with injection of the bulbar conjunctiva. *Haemophilus influenzae*, *Streptococcus pneumoniae*, and staphylococci are the most common pathogens. Treat this condition with topical antibiotic drops every 4 to 6 hours or ointment three times daily for younger children. The combination of trimethoprim sulfate and polymyxin B sulfate (Polytrim) is a good choice because it provides broad-spectrum coverage and is well tolerated (minimal stinging with application).

5. What is hyperacute bacterial conjunctivitis?

This is profuse purulent discharge, marked chemosis and lid swelling, and tender preauricular adenopathy. It is caused by *Neisseria* species (*Neisseria gonorrhoeae*). The organism usually spreads from the genitalia to the hands and then to the eyes; concurrent urethritis is usually present. This condition requires admission to the hospital for systemic antibiotics and emergent ophthalmology consultation, as it can cause keratitis and perforation. A Gram stain of the eye discharge may be helpful in making the diagnosis.

6. Describe the distinguishing features of stye, chalazion, and hordeolum and their treatment.

A hordeolum can be a stye or a chalazion. An external hordeolum or stye is an acute infection of Zeis's gland. An internal hordeolum or chalazion is caused by obstruction of the mebomian gland. A stye is usually caused by *Staphylococcus aureus* or *S. epidermidis*. It is treated with warm compresses and topical antibiotics. If the infection is centered at the base of an eyelash follicle, plucking the lash may promote healing. A chalazion results in sterile inflammation. Pain is mild or absent. Predisposing factors for a chalazion include seborrhea, acne, or blepharitis. This condition is treated with warm compresses and topical antibiotics if there are signs of infection.

Sethuraman U: The red eye: Evaluation and management. Clin Pediatr 2009;48:588-600.

7. What clinical symptoms help to distinguish allergic versus viral conjunctivitis?

Itching is the cardinal symptom of allergic conjunctivitis, distinguishing it from viral conjunctivitis, which is associated with complaints of grittiness, burning, or irritation. Patients with allergic conjunctivitis often have a history of atopy, seasonal allergy, or specific allergy.

8. What are the different forms of allergic conjunctivitis?

- Acute: Sudden onset once in contact with a known allergen (i.e., cat dander); this usually resolves within 24 hours after removal of the allergen
- Seasonal: Milder, more persistent symptoms during a particular pollen season
- Perennial: Related to year-round environmental allergens (usually indoor)

Dana R: Allergic conjunctivitis. UpToDate, 2013. Available from http://www.uptodate.com.

9. What is the treatment for allergic conjunctivitis?

It is best to avoid rubbing the eyes and minimize exposure to the allergen. Antihistamine/vasoconstrictor eye drops are helpful for acute symptoms. Seasonal or perennial forms of allergic conjunctivitis are usually treated with a combination antihistamine/mast cell stabilizer eye drop (Ketotifen is available over the counter), as well as oral antihistamines.

Dana R: Allergic conjunctivitis. UpToDate, 2013. Available from http://www.uptodate.com.

10. What is the most common cause of conjunctivitis in the newborn period?

Red, watery eyes in an infant who is only a few hours old is almost always due to a chemical conjunctivitis secondary to the use of topical prophylaxis (1% silver nitrate, 1% tetracycline ointment, or 0.5% erythromycin ointment) for prevention of ophthalmia neonatorum. True neonatal conjunctivitis rarely presents prior to 48 hours of age. The infectious organisms associated with neonatal conjunctivitis include *N. gonorrhoeae* (onset, 2-4 days of age), staphylococci or streptococci (onset, 4-7 days of age), *Chlamydia* (onset, 4-10 days of age), *Haemophilus* (onset, 5-10 days of age), and herpes simplex virus type II (onset, 6 days to 2 weeks).

Wright KW: Pediatric "pink eye." In Wright KW: Pediatric Ophthalmology for Pediatricians. Baltimore, Williams & Wilkins, 1999, pp 165-193.

11. Which groups of patients are most often diagnosed with gonococcal conjunctivitis?

Neonates and sexually active adolescents.

12. When are systemic antibiotics indicated in neonatal conjunctivitis?

Systemic antibiotics are used in chlamydial and gonococcal disease to control the risk of systemic involvement.

13. What are the classic signs of orbital cellulitis?

Orbital cellulitis is an infection of the tissues posterior to the orbital septum. Erythema and edema of the eyelids, decreased eye movement, proptosis, decreased vision, and papilledema are the classic signs of this infection.

14. Do all patients with orbital cellulitis require hospital admission?

Yes. Orbital cellulitis is a potentially vision-threatening, or life-threatening, condition.

15. What are the potential complications of orbital cellulitis?

Vision loss, meningitis, cavernous sinus thrombosis, brain abscess, and death.

16. How is orbital cellulitis treated?

Treat orbital cellulitis with intravenous (IV) antibiotics. Many cases can be managed medically, but others require surgical drainage. Obtain computed tomography to look for evidence of abscess formation and sinusitis, which is most often the inciting process. Urgent ophthalmologic consultation is indicated once the diagnosis of orbital cellulitis is considered. If operative drainage is necessary, an otolaryngologist often consults in conjunction with the ophthalmologist to drain the sinuses.

Greenberg MF: The red eye in childhood. Pediatr Clin North Am 2003;50:105-124.

17. What is periorbital cellulitis?

Periorbital cellulitis (also called preseptal cellulitis) is an infection of the tissues of the eyelids that does *not* involve the posterior orbit. Periorbital cellulitis is associated with predominantly unilateral swelling, erythema, and tenderness of the eyelids but normal visual acuity and eye motility. Computed tomography is often useful to distinguish between periorbital and orbital cellulitis, especially when severe eyelid edema precludes an adequate eye examination to ensure normal eye motility.

Levin AV: Ophthalmic emergencies. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 1595-1602.

18. What problems can lead to the development of periorbital cellulitis?

Eyelid trauma, impetigo, dacrocystitis (infection of the tear sac), and infections of the oil glands or hair follicles on the lid margins.

Greenberg MF: The red eye in childhood. Pediatr Clin North Am 2003;50:105-124.

19. Do all patients with periorbital cellulitis require hospitalization?

No. Children beyond infancy who are otherwise healthy and have no signs of systemic infection may be treated for periorbital cellulitis with intramuscular or oral antibiotics as outpatients. They require close follow-up for at least 24 to 48 hours. Admit patients to the hospital for IV therapy if they demonstrate no improvement or worsening disease.

Levin AV: Ophthalmic emergencies. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 1595-1602.

20. Which conditions can mimic periorbital (preseptal) cellulitis?

- Insect bites
- Contact dermatitis (poison ivy)
- Allergic reactions (usually bilateral swelling)
- Sinusitis
- Severe conjunctivitis (often due to adenovirus)

21. How should you treat a red eye in a contact lens wearer?

A red eye in a contact lens wearer may signify a sight-threatening condition (infection or a breakdown of corneal epithelium). Remove the contact lens and refer the patient for urgent ophthalmologic examination (within 12 hours).

Levin AV: Eye—Red. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 240-244.

22. Which corneal abrasions require ophthalmologic consultation?

1. Abrasions with a branching pattern; especially if not improving after 24 to 48 hours of therapy
2. Abrasions in a contact lens wearer
3. Larger epithelial defect at 24 hours or a drop in vision of more than 1 to 2 lines
4. Anyone with persistent discharge or unwillingness to keep the eye open the day after injury; these findings suggest a retained foreign body, poor healing, superinfection, or infectious keratitis

Jacobs DS: Corneal abrasions and corneal foreign bodies. UpToDate, 2013. Available from http://www.uptodate.com.

23. How do you treat a corneal abrasion?

- **Topical antibiotics**: Ointment is preferable to drops if tolerated; apply three to four times a day for 3 to 5 days.
- **Patching**: Because of lack of proven benefit, patching is unnecessary except if the abrasion is very large (occupying more than half of the corneal surface; in this case it is helpful for pain control). Do not patch if there is a retained foreign body; never leave a patch on for more than 24 hours.
- **Pain control**:
  - Mechanical (patching)
  - Cycloplegia (cyclopentolate 0.5% or 1% is the shortest acting)
  - Systemic pain medications
  - Topical analgesia with nonsteroidal anti-inflammatory drug (NSAID) ophthalmic solutions (diclofenac or ketorolac); do not use topical anesthetics (may cause delayed wound healing)
- Never treat corneal abrasions with a topical steroid!

24. Why do cycloplegic drops provide pain relief in corneal abrasions?

They inhibit the miotic response to light that causes pain and photophobia.

25. What is ciliary flush and why is this finding concerning on the physical examination of a red eye?

Ciliary flush, or injection, is a ring of red or violet spreading out from the cornea. It is most marked at the limbus (the area where the cornea transitions to the sclera). This finding is associated with more concerning causes of a red eye such as infectious keratitis, iritis, or acute angle closure glaucoma.

26. What are the characteristics of herpes simplex keratoconjunctivitis?

Ocular involvement with herpes simplex virus is characterized by an episode of primary ocular herpes (skin eruption with multiple vesicles) that lasts for 2 to 3 weeks, followed later by recurrent corneal disease. The recurrences are almost always *unilateral* and associated with *preauricular adenopathy*. A *dendritic pattern* of corneal defects is a classic sign of recurrent herpes simplex keratitis. Viral replication causes punctate, dendritic, or geographic epithelial defects. Treatment is with topical antiviral agents, usually Viroptic 1% every 2 hours while awake. Corticosteroids are contraindicated because they diminish the body's immune response. The cumulative effect of repeated infections can lead to corneal clouding and vision loss.

Wright KW: Pediatric "pink eye." In Wright KW: Pediatric Ophthalmology for Pediatricians. Baltimore, Williams & Wilkins, 1999, pp 165-193.

Key Points: Red Eye

1. A red eye in a contact lens wearer is an emergent problem requiring consultation with an ophthalmologist.
2. Orbital cellulitis is a potentially vision-threatening, or even life-threatening, condition that requires hospital admission and emergent consultation with an ophthalmologist.
3. Any patient who complains of severe eye pain with a foreign body sensation preventing opening of the eye generally can be presumed to have a corneal epithelial defect.
4. All patients with conjunctivitis have a red eye; however, not all red eyes are caused by conjunctivitis.
5. The serious conditions that cause a red eye will cause 360-degree involvement of the bulbar conjunctiva, often in a ciliary flush pattern, but will spare the tarsal conjunctiva.
6. Corneal abrasions are usually associated with a small pupil due to reactive miosis.
7. If eye pain is relieved by a drop of topical anesthetic, the patient likely has a surface problem (foreign body or corneal abrasion).

CHAPTER 23

SCROTAL PAIN

Joel A. Fein

1. Do infants and toddlers get testicular torsion?

Neonates most frequently have *extravaginal* torsion (twisting above the tunica vaginalis), which usually occurs in utero and is more often but not invariably associated with a nonviable testicle despite early discovery. *Intravaginal* testicular torsion (twisting of the spermatic cord within the tunica vaginalis) is most common in the second decade of life and is rare before the age of 10 years. Nevertheless, boys of any age with acute scrotal pain and swelling require prompt attention, radiologic evaluation, and possible surgical evaluation.

Al-Salem AH: Intrauterine testicular torsion: A surgical emergency. J Pediatr Surg 2007;42(11):1887-1891.

Sorensen MD, Galansky SH, Striegl AM, et al: Perinatal extravaginal torsion of the testis in the first month of life is a salvageable event. Urology 2003;62(1):132-134.

2. What are some of the important factors in the patient's history that suggest testicular torsion?

Boys with testicular torsion will frequently report relatively sudden onset of unilateral scrotal pain and often nausea and vomiting. They may report that they had this pain on prior occasions, but that information does not argue against acute testicular torsion. Fever and painful voiding are uncommon. A history of trauma does not preclude and can even predispose the patient to torsion. It is important to recognize that the child with scrotal disease often reports lower abdominal or groin pain. Always perform a genital examination on the male patient reporting abdominal pain. Conversely, it is important to examine the abdomen of all patients with scrotal pain to evaluate peritoneal inflammation, intestinal obstruction, and abdominal masses.

Beni-Israel T, Goldman M, Bar Chaim S, Kozer E: Clinical predictors for testicular torsion as seen in the pediatric ED. Am J Emerg Med 2010;28(7):786-789.

Boettcher M, Bergholz R, Krebs TF, et al: Clinical predictors of testicular torsion in children. Urology 2012;79(3):670-674.

Liang T, Metcalfe P, Sevcik W, Noga M: Retrospective review of diagnosis and treatment in children presenting to the pediatric department with acute scrotum. AJR Am J Roentgenol 2013;200(5): W444-W449.

Seng YJ, Moissinac K: Trauma induced testicular torsion: A reminder for the unwary. Emerg Med J 2000;17:381-382.

3. What is the "bell clapper deformity"?

Normally, the testicle is fixed to the posterior wall of the scrotum. Although it is almost impossible to detect this on physical examination, the majority of patients with testicular torsion have a congenital condition called the *bell clapper deformity*, whereby the tunica vaginalis completely envelops the testicle. This allows the testicle and spermatic cord to twist in relation to the tunica vaginalis, compressing the vessels and nerves within. Although the deformity is bilateral, symptoms usually occur unilaterally. The left testicle is more commonly affected because the left spermatic cord is usually longer than the right. With this deformity, some children present with intermittent symptoms, and in these cases, do not discount the diagnosis of testicular torsion.

Hayn MH, Herz DB, Bellinger MF, Schneck FX: Intermittent torsion of the spermatic cord portends an increased risk of acute testicular infarction. J Urol 2008;180(4 Suppl):1729-1732.

4. What is manual detorsion?

This is a temporizing procedure to manage testicular torsion. Manual detorsion in the emergency setting can allow the testicle to remain viable until emergency surgery can be

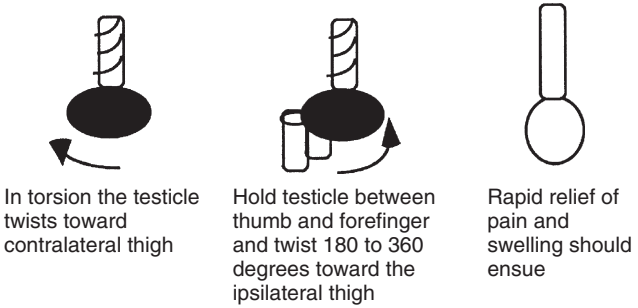


Figure 23-1. Technique for manual detorsion of the testicle.

performed. Although the testicle can twist in either direction, it more commonly twists medially, toward the contralateral thigh. In the manual detorsion procedure, one holds the affected testicle between the thumb and forefinger and untwists 360 degrees toward the ipsilateral thigh—like opening the pages of a book (Fig. 23-1). If relief is noted, the testicle should be rotated another 360 degrees or more, because the usual twist is 720 degrees. The direction can be reversed if more pain or swelling occurs after the initial maneuver. The trick is not to be shy or reserved while performing this procedure; it will hurt while it is being done, but if done correctly it will greatly relieve symptoms almost immediately. Sedation and analgesia may be warranted to facilitate the detorsion procedure. When this procedure is successful, bedside ultrasound may reveal increased blood flow to the testicle, but surgical correction (bilateral orchiopexy) is still necessary after this maneuver. It is important to get urology involved promptly.

Bomann JS, Moore C: Bedside ultrasound of a painful testicle: Before and after manual detorsion by an emergency physician. Acad Emerg Med 2009;16(4):366.

Sessions AE, Rabinowitz R, Hulbert WC, et al: Testicular torsion: Direction, degree, duration and disinformation. J Urol 2003;169:663-665.

5. How long will a torsed testicle remain viable after the onset of pain?

Timing is crucial in the diagnosis and management of testicular torsion. In general, the prognosis is excellent if the testis is detorsed within 3 hours of symptom onset. Almost 100% of testes detorsed within 3 hours of onset of symptoms will be viable. About 75% of testes detorsed after 8 hours, 10% to 20% of testes detorsed after 12 hours, and 0% of testes detorsed after 24 hours will be viable.

6. What is the "blue dot sign"?

This refers to a blue discoloration on the upper outer pole of the scrotum, associated with torsion of the appendix testis. It signifies the appearance of a hemorrhagic appendix testis visible through the scrotal wall.

7. What is torsion of the appendix testis?

There are many appendages to the testis that are not functional but can certainly cause problems for younger children. The testicular appendix that is located on the superior pole of the testis is the most common appendage to twist on its pedicle and compromise vascular supply. This causes pain and swelling of the scrotum, albeit less so than with testicular torsion. This occurs most often in school-aged children and early adolescents, and rarely in those over the age of 20 years. Nausea and vomiting are rare, and on physical examination the cremasteric reflex should be brisk unless swelling is severe. The diagnosis is more easily made if tenderness is located on the anterior or lateral pole of the testicle or there is a "blue dot sign." If the diagnosis can be secured, torsion of the appendix testis is treated with oral analgesics and bed rest. The appendix will likely autoamputate, with no known sequelae.

Kadish HA, Bolte RG: A retrospective review of pediatric patients with epididymitis, testicular torsion, and torsion of testicular appendages. Pediatrics 1998;102:73-76.

Key Points: Differentiating Testicular Torsion and Torsion of Appendix Testis

Testicular Torsion

1. It is most commonly seen in mid- to late adolescence.
2. Pain is sudden in onset, located in the entire testicle.
3. Cremasteric reflex is absent.

Torsion of Appendix Testis

1. It is most commonly seen in children and early adolescents.
2. Pain can be more gradual, located in a specific area of the testicle.
3. Cremasteric reflex is brisk.

8. What is the difference between epididymitis and orchitis, other than one being easier to spell?

Epididymitis is most often caused by infection. If the inflammation spreads to the testicles, the resulting epididymo-orchitis can be difficult to differentiate from primary orchitis. There are, however, some distinguishing features that can guide diagnosis. Fever, nausea, and vomiting are uncommon in patients with epididymitis. Conversely, orchitis is almost always due to a viral infection and accompanies the other signs and symptoms of the specific virus, such as mumps. A history of urinary tract infections, penile discharge, or urologic abnormalities indicates the need for a workup for infectious causes of scrotal swelling. Epididymitis causes tenderness posteriorly and superiorly within the scrotum, whereas orchitis usually causes diffuse and often bilateral tenderness. Epididymitis can be due to *Chlamydia trachomatis* or *Neisseria gonorrhoeae*; however, prepubertal patients may be infected with gram-negative bacteria, such as *Pseudomonas* spp., *Escherichia coli*, or enterococci. The urinalysis is positive for white blood cells in very few pediatric patients with epididymitis and even less frequently in patients with orchitis, and antibiotic therapy is often reserved for those who have a positive urinalysis, culture, or fever.

Graumann LA, Dietz HG, Stehr M: Urinalysis in children with epididymitis. Eur J Pediatr Surg 2010;20(4):247-249.

Santillanes G, Gausche-Hill M, Lewis RJ: Are antibiotics necessary for pediatric epididymitis? Pediatr Emerg Care 2011;27(3):174-178.

Yang C Jr, Song B, Liu X, et al: Acute scrotum in children: An 18-year retrospective study. Pediatr Emerg Care 2011;27(4):270-274.

9. What are some reasons why emergency physicians fail to diagnose testicular torsion?

- It may not be suspected/considered in an infant.
- The chief complaint may be abdominal pain rather than scrotal pain.
- History of trauma may confuse the diagnosis.
- Physical examination findings can be inconsistent.
- Examination of the genitalia is sometimes omitted.
- A diagnostic study such as an ultrasound is trusted rather than physical examination findings.
- A urologist is not consulted even though the history or examination is worrisome.

10. Can you rely on physical examination or ultrasound to diagnose testicular torsion?

Unfortunately, this is not always the case. Physical examination findings vary, and a high index of suspicion is needed in all cases of scrotal pain. A high-riding testicle is associated with torsion, and the presence of a cremasteric reflex is usually reassuring that torsion is not present. Most patients with testicular torsion do not have a cremasteric reflex on the affected side. However, this reflex remains present in a significant number of patients (8-20%) with torsion, and it alone cannot be relied upon to make the diagnosis or rule out torsion.

Some of these cases are just too close to call clinically. Color Doppler ultrasound of the scrotum is generally very helpful in determining the presence of testicular torsion. It is considered the best test to assess perfusion and exclude testicular torsion, if it cannot be excluded clinically. Arterial blood flow is absent or diminished in the case of testicular torsion. But false-negative results are possible. No diagnostic study is completely reliable, and in some cases when the examination is worrisome, urologic consultation and surgical exploration of the scrotum are still warranted regardless of what the study shows.

11. Are there any rare entities causing scrotal pain or swelling that would be helpful to mention on rounds?

Some of the childhood vasculitides can cause scrotal swelling, including Henoch-Schönlein purpura (HSP), Kawasaki disease, and familial Mediterranean fever (FMF). Scrotal involvement occurs in more than 10% of patients with HSP. Genital involvement usually follows the skin, joint, and intestinal symptoms by several days and lasts up to 1 week. Surgical management is rarely necessary, but testicular torsion has occasionally been noted to occur during the course of SHP.

Some children with Kawasaki disease may report mild to moderate scrotal pain. The perineal rash involves the scrotal walls rather than the testicle and usually accompanies the acute phase of the illness. Some patients with Kawasaki disease may have swelling of the epididymis as well.

In contrast to children with Kawasaki disease and HSP, children with FMF may report scrotal pain even before the final diagnosis is recognized. FMF is a genetic disease that affects Sephardic Jews, Turks, Armenians, and Middle Eastern Arabs. The illness is characterized by acute attacks of serositis, as well as the possibility of renal amyloidosis. Although rare in this disease, granulocytic infiltration can cause inflammation of the spermatic cord or the epididymis. Children report the gradual onset of unilateral, red, painful scrotal swelling. They have fever, leukocytosis, and an elevated sedimentation rate.

Eshel G, Vinograd I, Barr J, Zemer D: Acute scrotal pain complicating familial Mediterranean fever in children. Br J Surg 1994;81:894-896.

Hara Y, Tajiri T, Matsuura K, Hasegawa A: Acute scrotum caused by Henoch-Schönlein purpura. Int J Urol 2004;11:578-580.

12. How can I differentiate among the variety of painless scrotal masses?

The most common painless scrotal masses are inguinal hernia, hydrocele, varicocele, spermatocele, and testicular tumor. Table 23-1 describes the features of each of these entities.

Skoog SJ: Benign and malignant pediatric scrotal masses. Pediatr Clin North Am 1997;44:1229-1250.

13. Someday, somehow, every male will be kicked in the testicles. When do I worry about the patient who has this chief complaint?

Traumatic injuries to the testicle include, in order of increasing severity, contusion, hematoma, rupture, and dislocation. Physical examination should focus on the severity of swelling, the presence of both testicles inside the scrotal sac, and the relative size of each testicle. In general, the patient who has suffered recent scrotal trauma and who continues to report pain while in the emergency department setting deserves further evaluation. Similarly, patients with massive swelling of one or both sides of the scrotum require urgent urologic consultation. In general, these patients will require ultrasonography or surgical exploration to rule out testicle-threatening lesions. Ultrasonographic findings of testicular rupture may be subtle, and normal findings do not rule out this diagnosis. Patients in whom the pain resolves quickly and the physical examination is normal may be referred for follow-up but need not undergo radiologic or surgical evaluation in the acute setting.

Finally, it is important to remember that testicular torsion may follow minor trauma to the region, and this diagnosis must be considered for any patient with testicular pain regardless of the preceding history.

Micallef M, Ahmad I, Ramesh N, et al: Ultrasound features of blunt testicular injury. Injury 2001;32:23-26.

Key Points: Most Common Causes of Scrotal Pain in Children

Older Children and Adolescents

1. Testicular torsion
2. Epididymitis
3. Orchitis
4. Scrotal trauma

Infants and Young Children

1. Torsion of the appendix testis
2. Incarcerated hernia
3. Vasculitis (Kawasaki disease, Henoch-Schönlein purpura)

Table 23-1. Features of Painless Scrotal Masses

FEATURE	HISTORY	PHYSICAL EXAMINATION	MANAGEMENT
Inguinal hernia	Increases with Valsalva maneuver; painful only when incarcerated or strangulated	Mass in groin or scrotum; palpable at internal inguinal ring; bowel feels "sausage-shaped"	Assess for obstruction; Trendelenburg position, ice packs, analgesia, muscle relaxation
Varicocele (dilated veins of pampiniform plexus due to incompetent valves located along spermatic cord)	15% prevalence in adolescence; left > right; if found on the right, consider tumor, situs inversus, or renal vein thrombosis	Decreases when supine; "wormy" vessels around the cord	Surgical ligation of internal spermatic vein only if causing testicular atrophy
Spermatocele (sperm-containing cysts of the epididymis or efferent duct system)	Postpubertal males; painless	Nontender, small, cystic nodule located posterior or superior to testicle	Surgical intervention only if painful or unusually large
Neoplasm	15-35 years old; incidence increased if undescended, worse if intra-abdominal; painless unless bleeding into tumor	Hard mass, smooth or irregular, adherent to testicle; gynecomastia or other paraneoplastic phenomena from β-human chorionic gonadotropin or estrogen	Orchiectomy and investigation of preaortic lymph node involvement

SORE THROAT

Magdy W. Attia and Yamini Durani

CHAPTER 24

1. How do you define acute pharyngitis, tonsillitis, and pharyngotonsillitis?

Acute pharyngitis is an infection of the tonsils or the pharynx. *Pharyngitis, tonsillitis*, and *pharyngotonsillitis* are interchangeable terms often used to describe the clinical diagnosis of sore throat regardless of the cause.

2. What is the incidence of pharyngitis in children?

The exact incidence of pharyngitis is not known. It is, however, the second most common diagnosis in children 1 to 15 years of age in the ambulatory setting.

3. What is the most common cause for pharyngitis?

Viral agents account for most cases of pharyngitis seen in children as well as adults (70-80%). These viruses include the following:

- Rhinoviruses
- Epstein-Barr virus
- Parainfluenza viruses
- Adenovirus
- Influenza A and B viruses
- Herpes simplex virus types 1 and 2
- Enteroviruses
- Cytomegalovirus
- Human immunodeficiency virus (HIV)

4. How can I distinguish throat infections caused by these viruses?

The signs and symptoms often overlap between different causes of throat infection. Some helpful diagnostic clues are as follows:

- When pharyngitis is associated with conjunctivitis, the diagnosis of pharyngoconjunctival infection secondary to *adenovirus* is highly likely.
- The presence of ulcerative lesions on an erythematous base on the posterior palate is associated with *coxsackievirus type* A, a condition also known as herpangina. Hand, foot, and mouth disease is another variant of coxsackie pharyngitis that is characterized by the presence of small vesiculopustular lesions or shallow ulcers that are seen on the soft palate, palms, and soles.
- The association of significant cervical or generalized lymphadenopathy and hepatosplenomegaly is highly suspicious for infectious mononucleosis due to *Epstein-Barr virus* or, less commonly, *cytomegalovirus*.

5. Which bacterial pharyngitis is the most common?

Streptococcus pyogenes, also known as group A β-hemolytic streptococcus (GABHS), is the most common bacterium causing pharyngitis in children. It is implicated in as many as 20% to 30% of all cases of pharyngitis in children. Other bacterial causes of sore throats (group C β-hemolytic streptococci, group G β-hemolytic streptococci, *Mycoplasma pneumoniae, Neisseria gonorrhoeae*) are much less common. *Neisseria gonorrhoeae should be considered in sexually active patients with symptoms of pharyngitis*.

6. Why is it important for clinicians to diagnose and treat GABHS pharyngitis?

Antibiotic treatment of GABHS pharyngitis prevents rheumatic fever and suppurative complications. In addition, early treatment of GABHS pharyngitis shortens the course of the disease and reduces transmission to contacts.

7. What are the suppurative complications of GABHS?

These complications include peritonsillar cellulitis or abscess (quinsy), retropharyngeal abscess, cervical adenitis, otitis media, and sinusitis.

8. What are the symptoms of a retropharyngeal abscess?

A retropharyngeal abscess is an infection in the deep tissues of the neck that may be a complication of a preceding pharyngitis or upper respiratory tract infection. It is generally more common in children under 5 years of age due to the more prominent lymph nodes in the retropharyngeal space in younger children. Typical symptoms include sore throat, fever, neck pain, neck stiffness or torticollis, dysphagia, odynophagia, and drooling, and as it progresses it may lead to airway obstruction, causing symptoms such as tachypnea and stridor.

9. What are the symptoms of peritonsillar abscess?

A peritonsillar abscess is another possible suppurative complication of pharyngitis that generally occurs in older children and adolescents. Patients often have a severe sore throat that is worse or more inflamed on one side. Fever, dysphagia, a muffled sounding voice, and drooling are other common symptoms. On physical examination, patients have visible tonsillar inflammation that is unilaterally more enlarged, often with a midline shift of the uvula to the contralateral side.

10. What are other important complications of GABHS infections? Does treatment of GABHS prevent these complications?

Poststreptococcal glomerulonephritis (GN) is one of the complications of GABHS infection. Treating GABHS infection does not prevent poststreptococcal GN. Poststreptococcal GN can occur following non-GABHS infection, particularly with groups C and D. Toxin-mediated diseases, such as scarlet fever and toxic shock syndrome, are also important complications of GABHS infection.

11. Can rheumatic fever occur after a skin GABHS infection or after non-GABHS pharyngitis?

No. It is only known to occur after GABHS pharyngitis.

Key Points: Treatment of GABHS Pharyngitis (Strep Throat)

1. Shortens the course of the disease
2. Prevents suppurative complications
3. Prevents rheumatic fever
4. Reduces transmission to contacts
5. Does not prevent poststreptococcal GN

12. What are the clinical features of GABHS pharyngitis?

- Sudden onset of fever and sore throat in a school-aged child. Fever is often low grade or absent.
- Scarlatiniform rash—an erythematous, fine, sandpaper-like exanthem that generally appears in axillary and inguinal folds before it is generalized. It is pathognomonic for GABHS infection.
- Headache, vague abdominal pain, nausea, vomiting, and halitosis may be present.
- The pharynx is erythematous, and the tonsils are enlarged. Tonsillar exudate, palatine petechiae, and nasal excoriation may be noticed. Nasal excoriation is rare but more common in the younger child.
- Significant submandibular lymphadenopathy is usually present.
- Absence of cough or coryza is characteristic.

Attia MW, Zaoutis T, Klein JD, Meier FA: Performance of a predictive model for streptococcal pharyngitis in children. Arch Pediatr Adolesc Med 2001;155:687-691.

13. True or false: GABHS pharyngitis is usually obvious and easy to diagnose clinically.

False. Initially, signs and symptoms are often absent because of the child's early presentation to clinicians. GABHS and viral pharyngitis have many overlapping clinical features and are often difficult to distinguish from one another.

14. Are some of the signs and symptoms more reliable than others in diagnosing strep throat?

Yes. Scarlatiniform rash, large and tender submandibular lymph nodes, and the absence of coryza are more reliable than fever and tonsillar exudates in children. The latter two features are more common and reliable in adults.

Del Mar CB: A clinical prediction model did well in diagnosing pediatric group A beta-hemolytic streptococcal pharyngitis. ACP J Club 2002;136:37.

15. What laboratory tests are available to diagnose GABHS infections? What are their advantages and disadvantages?

Throat culture is considered the gold standard for diagnosing GABHS pharyngitis, though it has limitations. Throat culture has a relatively high incidence of false-negative results (10-20%). Also, a positive throat culture for GABHS without serologic evidence does not distinguish between carrier state and an acute infection. Finally, cultures may take 24 to 48 hours of incubation to detect GABHS, and this time may delay diagnosis and care.

Alternative tests include serologic testing (antistreptolysin O [ASO], anti-DNase) and rapid antigen tests. Serologic testing is impractical in the acute evaluation of suspected GABHS pharyngitis. Rapid antigen tests by a reference laboratory method are highly specific, but the sensitivity varies (60-90%) depending on the practice. Hence, the clinical guidelines recommend the use of rapid antigen tests in conjunction with a back-up throat culture if the rapid test is negative. Positive rapid antigen tests do not require a throat culture, because it is a highly specific test. Indiscriminate testing (i.e., in patients with a clear picture of upper respiratory infection) worsens both the sensitivity and specificity of rapid antigen tests as well as throat cultures.

Shulman ST, Bisno AL, Clegg HW, et al: Clinical practice guideline for the diagnosis and management of group A streptococcal pharyngitis: 2012 update by the Infectious Diseases Society of America. Clin Infect Dis 2012;55:1279.

16. What is the carriage rate of GABHS in healthy children?

Of healthy children, 10% to 20% have a positive throat culture for GABHS while they are asymptomatic (i.e., carriers).

17. What is the antibiotic of choice for treating GABHS sore throat?

Penicillin is the mainstay of treatment for GABHS pharyngitis. Resistance to penicillin has never been documented. It is the least expensive and has a proven efficacy. Oral penicillin V or a penicillin derivative such as amoxicillin is most often used (because liquid penicillin has an unpleasant taste). The recommended dose is 40 mg/kg/day for 10 days. Although the symptoms improve rapidly, it is crucial to complete the entire 10-day period of therapy. If compliance is in question, intramuscular benzathine penicillin G (0.6-1.2 million units) is the alternative. Cephalosporins, clindamycin, clarithromycin, or azithromycin are alternate antibiotic choices for penicillin-allergic patients.

Shulman ST, Bisno AL, Clegg HW, et al: Clinical practice guideline for the diagnosis and management of group A streptococcal pharyngitis: 2012 update by the Infectious Diseases Society of America. Clin Infect Dis 2012;55:1279.

18. Do GABHS carriers need to be treated with antibiotics?

Carriers do not need to be treated. Up to 20% of school-aged children may be carriers of GABHS. They are at low risk for disease transmission or development of suppurative and nonsuppurative complications.

Bisno AL, Gerber MA, Gwaltney JM, et al: Practice guidelines for the diagnosis and management of group A streptococcal pharyngitis. Clin Infect Dis 2002;35:113-125.

Gerber MA: Diagnosis and treatment of pharyngitis in children. Pediatr Clin North Am 2005; 52:729-747.

19. Should sore throat secondary to non-GABHS infections be treated?

There is not enough evidence in the literature to answer this question, and there are currently no clear recommendations on the necessity of treating these infections. In one report, children infected with group G β-hemolytic streptococci seem to have improved more quickly with oral penicillin. Group C infection is usually seen in older adolescents and young adults and is generally a milder illness than GABHS pharyngitis.

Key Points: GABHS Pharyngitis

1. This type accounts for 20% to 30% of pharyngitis in children.
2. Sore throat, red pharynx, tender submandibular lymph nodes, and absence of cough and coryza are characteristic in the presentation. Scarlatiniform rash, though rare, is almost always pathognomonic.
3. Indiscriminate testing is discouraged.
4. Penicillin and amoxicillin are still the drugs of choice for treatment.

20. What is the current recommendation for tonsillectomy in patients with recurrent pharyngitis?

Tonsillectomy should be considered in patients with three or more episodes of pharyngitis per year for 3 consecutive years, five episodes per year in 2 consecutive years, or seven episodes in 1 year despite adequate medical therapy.

Paradise JL, Bluestone CD, Colborn DK, et al: Tonsillectomy and adenotonsillectomy for recurrent throat infection in moderately affected children. Pediatrics 2002;110:7-11.

21. True or false: Tonsillectomy affects the incidence and the course of pharyngitis.

False. The presence or absence of the tonsils has no bearing on the overall incidence or the course of the disease. However, after tonsillectomy, the frequency of streptococcal pharyngitis diminishes for 1 to 2 years, after which the incidence returns to that of the general population.

22. Should steroids be used routinely in patients with pharyngitis?

There are not sufficient data to support the routine use of steroids in patients with pharyngitis, but it may be helpful in some patients with severe symptoms of pain who are also on antibiotics. Steroids may decrease the time to onset of pain relief. There is not a defined dose, preparation, or duration for steroid use, but some clinicians use a single dose of dexamethasone (Decadron). Consider other modalities of analgesia such as nonsteroidal anti-inflammatory drugs and acetaminophen.

Attia MW: ACP Journal Club. Review: Adding corticosteroids to antibiotics improves pain relief in patients with sore throat. Ann Intern Med 2013;158(6):JC11. (Comment on corticosteroids as standalone or add-on treatment for sore throat [Cochrane Database Syst Rev 2012].)

Hayward G, Thompson MJ, Perera R, et al: Corticosteroids as standalone or add-on treatment for sore throat. Cochrane Database Syst Rev 2012;(10):CD008268.

23. How soon can children with GABHS pharyngitis return to school or child care?

The American Academy of Pediatrics and recent studies recommend that children receive a full 24 hours of antibiotics before returning to school or child care.

American Academy of Pediatrics: Group A streptococcal infections. In Pickering L (ed): 2006 Redbook. Report of the Committee on Infectious Diseases, 27th ed. Elk Grove Village, IL, American Academy of Pediatrics, 2006, pp 610-620.

24. How can recurrent episodes of rheumatic fever be prevented?

If GABHS pharyngitis develops in a patient who has already had rheumatic fever in the past, he or she is at high risk for recurrent rheumatic fever. GABHS pharyngitis should therefore be prevented in any patient who has previously had rheumatic fever by administering continuous antibiotic prophylaxis. In most cases, intramuscular benzathine penicillin G every 4 weeks is preferred.

25. Can GABHS pharyngitis occur in children younger than 3 years of age?

Yes. Although it is a more prevalent disease in school-aged children, it is a common myth that GABHS pharyngitis does not occur in younger children. Studies have reported disease rates between 5% and 25% in this age group. Rheumatic fever is very rare in these young children.

STIFF NECK

Marla Friedman Cotzen and Nicholas Tsarouhas

CHAPTER 25

1. **What is the pathophysiology of meningismus, or stiff neck?**
   Flexion of the neck stretches the inflamed nerve roots and meninges of the cervical region. Autoprotective muscle spasm manifests as neck stiffness, or meningismus.

2. **Is meningismus usually seen in neonates with meningitis?**
   No. Neonates rarely manifest meningismus or nuchal rigidity. The most common symptoms of meningitis in neonates are lethargy, irritability, poor feeding, dyspnea, and apnea. Fever is uncommon in neonates; hypothermia is a more likely manifestation of sepsis/meningitis.
   Heath PT, Nik Yusoff NK, Baker CJ: Neonatal meningitis. Arch Dis Child Fetal Neonatal Ed 2003;88(3): F173-F178.

3. **At what age is meningismus or nuchal rigidity reliable in evaluation of children for meningitis?**
   Meningismus is usually seen after 18 to 24 months. Importantly, it is a late finding in meningitis in younger infants.

4. **What are the Kernig and Brudzinski signs?**
   These signs are physical examination maneuvers to evaluate for the presence of meningeal inflammation. The Kernig sign is positive when extension of the patient's hip and knee causes pain and flexion of the neck. The Brudzinski sign refers to involuntary flexion of the knees with flexion of the patient's neck.

5. **What are the indications for lumbar puncture (LP) in a child with a stiff neck?**
   Consider performing an LP to rule out meningitis in any febrile child with a stiff neck and no other obvious source of infection. This is especially important in children who are not well appearing. Lethargy and irritability should always raise concern for meningitis.
   Ostenbrink R, Moons KGM, Theunissen CCW, et al: Signs of meningeal irritation at the emergency department: How often bacterial meningitis? Pediatr Emerg Care 2001;17:161-164.

6. **What are the contraindications to doing an LP?**
   LP may be dangerous in an unstable, critically ill child. "Curling" the young patient into position for the LP may limit ventilation and lead to respiratory arrest in an already compromised infant. If the child is unstable, draw a blood culture and administer antibiotics. Defer the LP until the child is more stable. Other contraindications to LP include increased intracranial pressure, lumbosacral cutaneous infection, and coagulopathy.
   Cronan K, Wiley J: Lumbar puncture. In Henretig FM, King C (eds): Textbook of Pediatric Emergency Procedures, 2nd ed. Philadelphia, Lippincott Williams & Wilkins, 2008, p 507.

7. **What are some possible complications of LP?**
   Although complications are rare, they do occur. Examples include hematomas, persistent cerebrospinal fluid (CSF) leaks, radiculopathies, nerve injuries, epidermoid cysts, post-LP headache, and infection. Headaches after LP in children are uncommon, because a small needle is used for the procedure and minimal CSF leak occurs. Life-threatening and extremely rare complications include herniation, respiratory arrest, and cardiac arrest.
   Cronan K, Wiley J: Lumbar puncture. In Henretig FM, King C (eds): Textbook of Pediatric Emergency Procedures, 2nd ed. Philadelphia, Lippincott Williams & Wilkins, 2008, p 507.

8. **What other spinal infections may present with a stiff neck?**
   Osteomyelitis, epidural abscess, and diskitis may occur in the cervical region. Focal spine tenderness in the presence of fever should raise suspicion for these infections. Conventional radiography is sometimes helpful, but radionuclide bone scanning and magnetic resonance imaging are more diagnostic.

9. Which serious deep neck infection can present with stiff neck?

Retropharyngeal abscess. Retropharyngeal infections (cellulitis, adenitis, abscess) can develop in the potential space between the anterior border of the cervical vertebrae and the posterior wall of the esophagus. These infections generally occur in infants and toddlers and are rarely seen in children older than 5 years. They are usually caused by group A streptococcus, *Staphylococcus aureus*, or anaerobes. These ill-appearing children may present with fever, respiratory distress, drooling, difficulty swallowing, stridor, or meningismus. Although the neck is "stiff" with retropharyngeal abscess, the patient can often flex the neck (unlike a child with meningitis). However, he is unwilling to move the neck laterally to the side of the adenitis or abscess.

Obtain a lateral neck radiograph to investigate for widening of the upper cervical prevertebral tissues. Computed tomography is confirmatory. Management includes intravenous (IV) antibiotics and, sometimes, surgical drainage.

Craig FW, Schunk JE: Retropharyngeal abscess in children: Clinical presentation, utility of imaging, and current management. Pediatrics 2003;111(6):1394-1398.

10. Define torticollis.

Torticollis, which is derived from the Latin *tortus*, meaning "twisted," and *collum*, meaning "neck," is a characteristic tilting of the head to one side secondary to some underlying disorder. The child with torticollis may or may not have pain and usually holds the head tilted to one side with the chin rotated in the opposite direction. There is unilateral neck muscle contraction. This condition can be due to a variety of causes, including minor trauma.

Tomczak KK, Rosman NP: Torticollis. J Child Neurol 2013;28(3):365-378.

11. What are the most common causes of torticollis in well-appearing, *afebrile* children?

Minor irritation, muscle spasm, and awkward sleep malposition are quite common in young children. The onset is usually sudden, often occurring after waking from sleep. These children have no history of fever or trauma and should have a completely normal neurologic examination. Supportive care and analgesics/anti-inflammatory agents are usually the only therapies indicated.

12. What is a common cause of stiff neck in a well-appearing, *febrile* child?

Cervical adenitis, which presents with an enlarged tender lymph node, may be associated with stiff neck. Group A streptococcus and *S. aureus* are the most likely organisms responsible, but *Bartonella henselae* (cat scratch disease) and mycobacterial disease should also be considered. Pharyngitis/tonsillitis and upper respiratory tract infections may also be associated with stiff neck.

13. Name a common neurologic condition that may be associated with torticollis.

Pseudotumor cerebri. Although most patients with pseudotumor cerebri present with headache and vomiting, in some cases, a stiff neck or head tilt may be the presenting symptom. Consequently, the optic disks should be examined carefully in children/adolescents presenting with unexplained stiff neck. Symptoms resolve quickly with removal of CSF by LP.

Straussberg R, Harel L, Amir J: Pseudotumor cerebri manifesting as stiff neck and torticollis. Pediatr Neurol 2002;26(3):225-227.

14. What common pulmonary condition may be associated with torticollis?

Upper lobe pneumonia, which causes referred pain to the neck. Tachypnea is the most reliable physical examination sign in patients with pneumonia. Of course, fever and cough are also usually seen.

15. Which gastroenterologic condition may be associated with torticollis?

Sandifer syndrome, which is characterized by intermittent torticollis, opisthotonus, and irritability, is caused by severe gastroesophageal reflux with esophagitis in infants. Many infants with this condition exhibit failure to thrive.

16. What is the most common oncologic cause of torticollis in children?

Posterior fossa tumors. In addition to head tilt, torticollis, or stiff neck, children with these tumors may present with headache, early-morning vomiting, clumsiness, ataxia, strabismus, visual changes, or papilledema.

Extremera VC, Alvarez-Coca J, Rodriguez GA, et al: Torticollis is a usual symptom in posterior fossa tumors. Eur J Pediatr 2008;167:249.

17. What is the most common form of torticollis in infancy?

Congenital muscular torticollis (also called sternocleidomastoid [SCM] tumor of infancy or pseudotumor of infancy) usually presents in the first 2 weeks of life as a unilateral, hard, immobile, and fusiform swelling in the inferior aspect of the SCM muscle. The infant's head is tilted toward the affected side. The cause is controversial. The most common explanation implicates birth trauma with resultant hematoma formation followed by muscle contracture. Another theory postulates that intrauterine abnormal fetal position causes unilateral shortening of the SCM muscle. Passive stretching of the involved muscle is usually curative; recalcitrant cases may require surgical release (<5% of all cases). Intramuscular injections with botulinum toxin have also been employed.

Celayir AC: Congenital muscular torticollis: Early and intensive treatment is critical. A prospective study. Pediatr Int 2000;42:504-507.

Ta JH, Krishnan M: Management of congenital muscular torticollis in a child: A case report and review. Int J Pediatr Otorhinolaryngol 2012;76(11):1543-1546.

18. Define paroxysmal infantile torticollis.

This is a self-limited condition characterized by intermittent episodes of torticollis. It may last for minutes to hours and may recur for weeks to months. Onset is usually in the first few months of life. It usually remits by 2 to 3 years of age. These episodes are associated with pallor, ataxia, nystagmus, migraines, vomiting, agitation, and lethargy. The cause of the syndrome remains unknown and, at present, there is no effective therapy.

Rosman NP, Douglass LM, Sharif UM, et al: The neurology of benign paroxysmal torticollis of infancy: Report of 10 new cases and review of the literature. J Child Neurol 2009;24(2):155-160.

19. Which congenital syndrome is identified by the triad of short neck (brevicollis), limited neck motion, and low occipital hair line?

Klippel-Feil syndrome. This is a skeletal malformation characterized by the fusion of a variable number of cervical vertebrae. It may also be associated with other bony anomalies and significant scoliosis. These children may have anomalies of multiple organ systems as well. The cause is unknown.

Driscoll DJ, Rigamonti D, Gailloud P: Klippel-Feil syndrome. In National Organization for Rare Disorders: NORD Guide to Rare Disorders. Philadelphia, Lippincott Williams & Wilkins, 2003.

20. What is Sprengel's deformity?

This is a congenital failure of the scapula to descend to its correct position. In its most severe form, the scapula is connected by bone to the cervical spine and limits neck motion. The treatment of choice is surgery and physical therapy.

Guebert GM, Rowe LJ, Yochum TR, et al: Congenital anomalies and normal skeletal variants: Sprengel's deformity. In Yochum TR, Rowe LJ: Essentials of Skeletal Radiology, 3rd ed. Baltimore, Lippincott Williams & Wilkins, 2005.

21. What are the first priorities in managing a trauma victim with a stiff neck?

While maintaining in-line stabilization of the cervical spine, ensure adequacy of the airway. The physical examination should then focus on the presence of neurologic deficits (weakness, paresthesias, bowel or bladder dysfunction). If neurologic deficits are present, ensure complete immobilization and obtain emergent neurosurgical consultation.

Tzimenatos L, Vance C, Kuppermann N: Neck stiffness. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 392-401.

22. Which initial radiographs should be obtained in the trauma victim with neck pain?

The routine initial views include a lateral cervical spine (the most important view), an open-mouth odontoid view, and an anteroposterior cervical spine view.

Woodward GA: Neck trauma. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 1376-1421.

23. If the initial radiographs are inconclusive, which other images may be helpful?

Flexion/extension views of the cervical spine may be useful in the patient who reports pain yet has no abnormality noted on the initial films. If plain radiographs are still inconclusive,

computed tomography of the cervical spine may be indicated. If focal neurologic findings are present, spinal magnetic resonance imaging should be strongly considered.

Woodward GA: Neck trauma. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 1376-1421.

24. Are traumatic cervical spine injuries more common in the upper or lower cervical spine in young children?

Traumatic cervical spine injuries are more common in the upper cervical spine in younger children, due to the higher fulcrum of the cervical spine, as well as the relative weakness of the neck muscles, as compared with adolescents and adults.

Tzimenatos L, Vance C, Kuppermann N: Neck stiffness. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 392-401.

25. What is the most common anatomic abnormality identified in cases of traumatic torticollis?

Atlantoaxial rotary subluxation. The ligamentous laxity of the pediatric cervical spine predisposes children to this condition, which is often seen after only a minor injury, such as a fall from a low height. These patients report stiff neck with pain but have no neurologic deficits. Plain films of the cervical spine may reveal asymmetry of the odontoid relative to the atlas. Computed tomography is usually confirmatory. In most cases, soft collar, rest, and anti-inflammatory agents or analgesics are curative. Severe cases, however, may require traction or surgery.

Kim HJ: Cervical spine anomalies in children and adolescents. Curr Opin Pediatr 2013;25(1):72-77.

26. Is atlantoaxial subluxation always a result of trauma?

No, atlantoaxial subluxation may result from ligamentous laxity following an infection or inflammatory process. It may be associated with rheumatoid arthritis, systemic lupus erythematosus, or tonsillitis/pharyngitis.

Also, think of this condition if a child comes to the emergency department (ED) with a stiff neck following an otolaryngology procedure (e.g., tonsillectomy, adenoidectomy), in which the neck is maneuvered during surgery. This is called Grisel's syndrome. Children with Down and Marfan syndromes are particularly susceptible to this subluxation, secondary to the laxity of the transverse ligament of the atlas.

Chipparini L, Zorzi G, De Simone T, et al: Persistent fixed torticollis due to atlanto-axial rotatory fixation. Neuropediatrics 2005;36:45-49.

27. Are there other injuries to consider in a child with a stiff neck after a minor fall?

Some children with a clavicle fracture may hold their neck to one side and limit movement because of pain and SCM muscle spasm. Always remember to examine/palpate the clavicles of a child who has sustained a fall.

28. What is the name given to drug-induced torticollis?

Dystonic reaction. This is characterized by muscle spasm and abnormal postures, and it most commonly affects the eyes, face, neck, and throat. Patients with dystonic reactions may present with nuchal rigidity, opisthotonus, trismus, oculogyric crisis, or cogwheel rigidity. These patients are awake and often very distraught over their condition. The torticollis is caused by increased cholinergic activity and change in dopaminergic activity in the basal ganglia, which are responsible for muscle tone.

29. Which drugs most commonly cause dystonic reactions in children?

Metoclopramide (Reglan) and phenothiazines such as prochlorperazine (Compazine). These reactions are dose-related and usually begin within 2 to 5 days of initiating therapy.

30. How are dystonic reactions treated?

IV diphenhydramine (Benadryl) at a dose of 1 mg/kg usually terminates the reaction. Continue oral diphenhydramine for several days after the patient is discharged from the emergency department. Benztropine mesylate (Cogentin) is an alternate therapy. Of course, discontinue the offending drug.

31. What arachnid envenomation may be associated with torticollis?

The bite of *Lactrodectus mactans*, the dreaded black widow spider, is a neurotoxic envenomation that causes muscle pain and sometimes nuchal rigidity. There is little to no local reaction at the site of the bite. Black widow spider bites are the leading cause of death from spider bites in the United States.

32. Summarize the extensive differential diagnosis of stiff neck in children.

The extensive differential diagnosis of stiff neck in children is summarized in Table 25-1.

Key Points: Evaluating Stiff Neck in Children and Infants

1. Don't count on meningismus as a symptom of meningitis in neonates and young infants; instead focus on the nonspecific signs and symptoms, which include lethargy, irritability, poor feeding, dyspnea, and apnea.
2. An LP may be dangerous in an unstable, critically ill child; if the child is very sick, draw a blood culture, administer antibiotics, and defer the LP until the child is more stable.
3. Consider the following common infectious causes of stiff neck:
   - **Meningitis**: This is characterized by ill appearance, irritability, lethargy; fever; pain with flexion.
   - **Tonsillitis/pharyngitis**: Tonsils are red and inflamed, but not always exudative; consider peritonsillar abscess if there is posterior, superior, soft palatal bulge, often with uvular deviation.
   - **Retropharyngeal abscess**: The febrile, ill-appearing child may be drooling and will be unwilling to move the neck laterally to the side of the abscess.
   - **Cervical adenitis**: Single enlarged, tender cervical node is seen; fever is common but not universal.
   - **Viral myositis/myalgia**: Cervical muscles are diffusely tender to palpation; other viral symptoms may be present.
   - **Upper respiratory tract infection**: Appearance is similar to that for viral myositis, with prominent upper respiratory tract symptoms.
4. The most common cause of torticollis in an afebrile, well-appearing child with no history of trauma is minor irritation, muscle spasm, or awkward sleep malposition.

Table 25-1. Causes of Stiff Neck in Children

Infectious
Meningitis,* brain/epidural abscess, encephalitis, septic arthritis/osteomyelitis, epiglottitis, diskitis, retropharyngeal abscess, viral myositis/myalgia,* tonsillitis/pharyngitis,* upper respiratory tract infection,* cervical adenitis,* pneumonia (upper lobe), otitis media, mastoiditis
Traumatic
Muscle contusion/spasm,* subarachnoid hemorrhage, atlantoaxial rotary subluxation, epidural hematoma, congenital muscular torticollis,* spinal cord injury, cervical spine fracture, clavicle fracture*
Congenital
Benign paroxysmal torticollis, skeletal malformation (Klipple-Feil syndrome, congenital muscular torticollis,* Sprengel's deformity), atlantoaxial instability (Down syndrome), Arnold-Chiari malformation
Toxic
Dystonic reaction,* black widow spider bite
Oncologic
Posterior fossa tumors, lymphoma
Miscellaneous
Migraine, spasmus nutans, Sandifer syndrome, syringomyelia, collagen vascular disease (juvenile rheumatoid arthritis, pseudotumor cerebri, ankylosing spondylitis), psychogenic causes

*Denotes most common causes of stiff neck.

CHAPTER 26

STRIDOR

Susanne Kost

1. List the four primary diagnostic considerations in a febrile child with acute stridor.

- Croup
- Epiglottitis
- Retropharyngeal abscess
- Bacterial tracheitis

Table 26-1 illustrates the clinical criteria helpful in distinguishing among the four.

2. What is the most common cause of acute stridor in young children?

Laryngotracheobronchitis (croup) accounts for more than 90% of all emergency department (ED) visits for stridor in the pediatric population. The key features of croup include barky cough, stridor, and hoarseness, usually preceded by symptoms of a mild upper respiratory tract infection. Croup is most common in older infants and toddlers, though it also occurs in school-aged children.

3. A mother reports her infant has "always had noisy breathing." What is the most common cause of chronic stridor?

Laryngomalacia is the most common cause of chronic stridor in infants. It is generally benign and resolves spontaneously as the child grows. The exact cause is unknown, though it is usually related to one or more supraglottic abnormalities, including a long epiglottis that prolapses posteriorly, bulky arytenoids that prolapse anteriorly, or shortened aryepiglottic folds.

4. What is the best test for diagnosing laryngomalacia?

Clinical clues to the diagnosis of laryngomalacia include stridor that improves with prone positioning and worsens with crying. Airway fluoroscopy is suggestive of the diagnosis, but the gold standard for diagnosis is direct laryngoscopy in an awake, upright, spontaneously breathing infant. Direct laryngoscopy also rules out other, less common causes of congenital stridor, including vocal cord paralysis and laryngeal webs and cysts.

5. What is meant by the term *spasmodic croup*?

Spasmodic croup, also known as laryngismus stridulus, is a variant of croup that lacks the typical viral prodrome (low-grade fever, runny nose). Symptoms start suddenly, often in the middle of the night, and resolve quickly. The patient is often improved when he arrives in the ED. Symptoms may recur for several nights in a row. The cause of spasmodic croup is unclear. Because the diagnosis can be made only upon resolution of the symptoms, the distinction between viral and spasmodic croup during the initial presentation lacks clinical relevance.

6. You decide to treat a croupy child with nebulized epinephrine. Is it better to use racemic epinephrine rather than L-epinephrine?

No, the two forms of epinephrine are equally effective and the rate of side effects is not significantly different. The recommended dose is 0.05 mL/kg (maximal dose: 0.5 mL) of racemic epinephrine 2.25% or 0.5 mL/kg (maximal dose: 5 mL) of L-epinephrine 1:1,000 via nebulizer. L-epinephrine may be more readily available in clinical settings along with other resuscitation supplies.

Bjornson C, Russell KF, Vandermeer B, et al: Nebulized epinephrine for croup in children. Cochrane Database Syst Rev 2011;(2):CD006619.

Zoorob R, Sidani M, Murray J: Croup: An overview. Am Fam Physician 2011;83(9):1067-1073.

Table 26-1. Clinical Criteria Helpful in Distinguishing Among the Four Primary Diagnoses in a Febrile Child with Acute Stridor

CRITERIA	CROUP	EPIGLOTTITIS	RETROPHARYNGEAL ABSCESS	BACTERIAL TRACHEITIS
Anatomy	Subglottic	Supraglottic	Retropharyngeal nodes	Trachea
Cause	Viral: parainfluenza	Bacterial*	Bacterial: oral flora	Bacterial: *Staphylococcus aureus*
Age range	6 mo-3 y	Any, sporadic	6 mo-4 y	Any, sporadic
Onset	1-3 d	Hours (prodrome—days)	1-3 d	3-5 d
Toxicity	Mild-moderate	Marked	Marked	Marked
Drooling	No	Yes	Yes	No
Hoarseness	Yes	No	No	No
Cough	Barky	No	No	Yes, painful
White blood cell count	Normal	Elevated	Elevated	Elevated
Radiograph	"Steeple" sign (anteroposterior)	"Thumb" sign (lateral)	Widened soft tissues†	"Shaggy" trachea

*Hib (Haemophilus influenzae type b) now accounts for less than 25% of cases; other causes include staphylococci, streptococci, and, in immunosuppressed patients, Candida species and herpes simplex virus.
†Expiratory film may also show significant widening; the best test is computed tomography of the neck.

7. An adolescent female cross-country runner presents to the ED for the third time in a month for "croup" worsened by running. What is the likely cause of her stridor? What is the treatment?

This teen athlete likely suffers from paradoxical vocal fold movement (PVFM), a condition in which the vocal cords adduct abnormally on inspiration, expiration, or both. It has been associated with stress, exercise, gastroesophageal reflux, and irritant exposures. One case series found a mean age of 13 years, a female-to-male ratio of 3:1, and successful treatment in the majority of the cases with speech therapy.

Maturo S, Hill C, Bunting G, et al: Pediatric paradoxical vocal fold motion: Presentation and natural history. Pediatrics 2011;128(6):e1443-e1449.

8. What is the characteristic radiographic finding in a patient with croup? In epiglottitis?

The classic radiographic finding in croup is the "steeple sign," a narrowing of the laryngotracheal air column just below the vocal cords on an anteroposterior view (Fig. 26-1). Another finding includes "ballooning" (distention) of the hypopharynx during inspiration, seen on a lateral view. The steeple sign lacks sensitivity and specificity. Radiographs are not indicated for most children with suspected croup. Radiographs are reserved for evaluation of children with stridor when other causes are considered or atypical cases. The classic radiographic finding in epiglottitis is the "thumb sign," referring to the lateral view of the swollen epiglottis resembling a lateral view of one's thumb (Fig. 26-2). The thumb sign is also subjective, and radiographs alone should not be used to diagnose epiglottitis. If clinical suspicion is high, imaging should be deferred in favor of direct visualization of the airway under controlled circumstances.

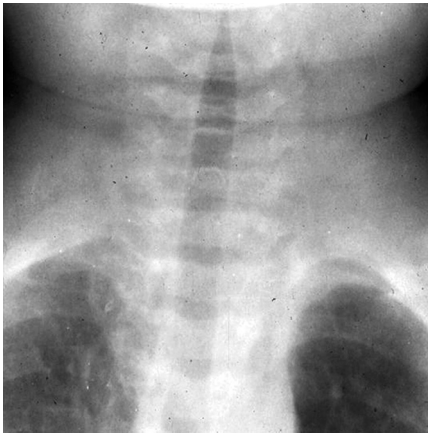


Figure 26-1. Radiographic view of the steeple sign.

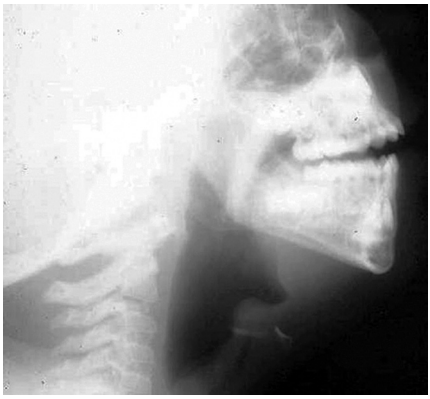


Figure 26-2. Radiographic view of the thumb sign.

9. Which bacterial infection of the upper airway most closely clinically mimics croup: epiglottitis, bacterial tracheitis, or retropharyngeal abscess?

The clinical presentation of bacterial tracheitis is nearly indistinguishable from that of severe croup. In fact, croup caused by parainfluenza and influenza type A may lead to bacterial superinfection in some cases. Bacterial tracheitis presents with symptoms of stridor, fever, and toxicity generally worsening over a 3- to 7-day period. Racemic epinephrine and steroids are not effective, and many patients will require intubation and surgical débridement of the membranous tracheal exudates. The most common organism causing bacterial tracheitis is *Staphylococcus aureus*, though *Moraxella catarrhalis* infection is also prevalent and potentially more severe.

Miranda AD, Valdez TA, Pereira KD: Bacterial tracheitis: A varied entity. Pediatr Emerg Care 2011;27 (10):950-953.

10. Why are retropharyngeal abscesses uncommon in children over the age of 5 years?

A retropharyngeal abscess generally occurs in infants and toddlers, typically under the age of 4 years. The abscess is caused by seeding of the retropharyngeal nodes with bacteria, usually oral pathogens. After the age of 4, the retropharyngeal nodes atrophy.

11. What is stertor? How does it differ from stridor?

Stertor is the noise most aptly described as snoring. It refers to the low-pitched vibratory noise made when airflow is obstructed in the nose and soft tissues of the pharynx. Stridor is higher pitched and refers to the noise of turbulent airflow through the larynx and trachea. Thus, a snoring patient is stertorous, and a "croupy" patient is stridulous (not stridorous!).

12. A sedated patient develops acute stridor followed by sudden complete airway obstruction. What steps do you take to relieve suspected laryngospasm?

Stop the procedure and remove the offending stimulus. Make sure the upper airway is clear and in good position. Apply continuous positive airway pressure, and apply firm digital pressure to the laryngospasm notch (Fig. 26-3). If laryngospasm persists, deepen sedation with propofol or paralyze with succinylcholine. Consider tracheal intubation if the patient remains heavily sedated or if bag-mask ventilation is difficult.

Al-alami A, Zestos M, Baraka A: Pediatric laryngospasm: Prevention and treatment. Curr Opin Anesthesiol 2009;22:388-395.

13. What is the significance of stridor that is loudest in the expiratory phase?

Expiratory stridor suggests tracheal disease. The differential diagnosis includes complete tracheal rings, primary tracheomalacia (faulty tracheal development), and secondary tracheomalacia (associated with external compression). External compression may be caused by vascular abnormalities or mediastinal masses, such as thymic cysts, cystic hygroma, thyroid hyperplasia, or mediastinal tumor.

Key Points: Clinical Clues to the Etiology of Stridor

1. Hoarseness in the presence of stridor indicates vocal cord inflammation and is reassuring for the lack of supraglottic disease, such as epiglottitis or retropharyngeal abscess.
2. Conversely, drooling with stridor is concerning for supraglottic obstruction.
3. Inspiratory stridor suggests an extrathoracic lesion (e.g., laryngeal, nasal, pharyngeal).
4. Expiratory stridor implies an intrathoracic lesion (e.g., tracheal, bronchial).
5. Biphasic stridor may represent subglottic or glottic disease.

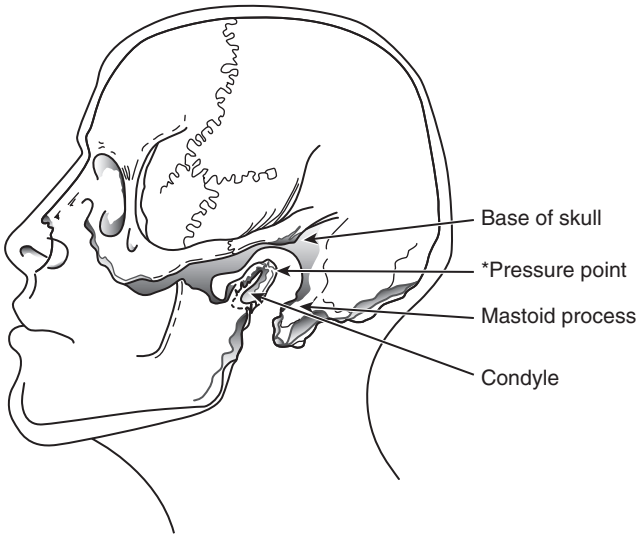


Figure 26-3. Laryngospasm notch. **Pressure point. (Adapted from Larson PC Jr: Laryngospasm—The best treatment [correspondence]. Anesthesiology 1998;89(5):1293-1294.)*

14. What are the three most common vascular anomalies associated with tracheal compression?

- Double aortic arch (the most common anomaly, where two arches encircle the trachea and esophagus)
- Pulmonary sling (aberrant left pulmonary artery arising from the right pulmonary artery, passing between the trachea and esophagus)
- Aberrant innominate artery (arising from aortic arch or left carotid, causing pressure on the anterior tracheal wall)

Kussman BD, Geva T, McGowan FX: Cardiovascular causes of airway compression. Paediatr Anaesth 2004;14:60-74.

15. A toddler born to an adolescent mother develops hoarseness over several months and presents with an acute exacerbation of stridor. What important pathologic condition must be considered?

Recurrent respiratory papillomatosis, caused by certain strains of the human papillomavirus (HPV), may cause significant airway obstruction preceded by an indolent history of hoarseness, chronic or intermittent coughing spells, or poor feeding. HPV infection is very common in sexually active females, with adolescent prevalence rates ranging from 20% to 40%, and about 1% of infants born to mothers with vaginal condyloma will develop recurrent respiratory papillomatosis. Treatment includes both surgical (laser) and medical (antiviral) modalities.

Wiatrak BJ: Overview of recurrent respiratory papillomatosis. Curr Opin Otolaryngol Head Neck Surg 2003;11:433-441.

16. An asymptomatic 2-year-old coughed and choked while playing with small plastic blocks. Parents are resistant to bronchoscopy. What is the best diagnostic test?

Computed tomography (CT). Three-dimensional postprocessing of spiral CT images allows virtual views of the airway similar to those seen with endoscopy. CT has been shown to be accurate and may be useful in both showing the exact location of a foreign body prior to bronchoscopy and ruling out a foreign body in patients with a low level of suspicion.

Bhat KV, Hegde JS, Nagalotimath US, Patil GC: Evaluation of computed tomography virtual bronchoscopy in pediatric tracheobronchial foreign body aspiration. J Laryngol Otol 2010;124(8):875-879.

17. Name five causes of stridor that are not directly related to the anatomy of the upper airway.

- **Allergic:** Anaphylaxis or hereditary angioedema
- **Cardiac:** Vascular rings; surgical injury to recurrent laryngeal nerve
- **Gastrointestinal:** Extraesophageal reflux
- **Neurologic:** Arnold-Chiari malformation with brainstem compression
- **Psychiatric:** Paradoxical vocal cord adduction

18. What electrolyte abnormality has been associated with stridor?

Hypocalcemia may be associated with laryngospasm and stridor, in addition to tetany and the characteristic Chvostek's and Trousseau's signs.

19. Why is it important to examine the skin of a baby who presents with stridor?

Examine the skin and look for a hemangioma. A baby with a cutaneous hemangioma located in a cervicofacial, mandibular, or "beard" distribution (including the preauricular skin, mandible, lower lip, chin, or anterior neck) has a risk of an airway hemangioma (Fig. 26-4). A baby with an airway hemangioma may develop progressive hoarseness or stridor, most likely between the ages of 6 and 12 weeks, when hemangioma proliferation is most rapid. Symptoms can progress from initial hoarseness or stridor to respiratory failure. Airway involvement can be confirmed using endoscopic visualization. Airway hemangiomas can also develop in children who do not have cutaneous hemangiomas.

Orlow SJ, Isakoff MS, Blei F: Increased risk of symptomatic hemangiomas of the airway in association with cutaneous hemangiomas in a "beard" distribution. J Pediatr 1997;131(4):643.

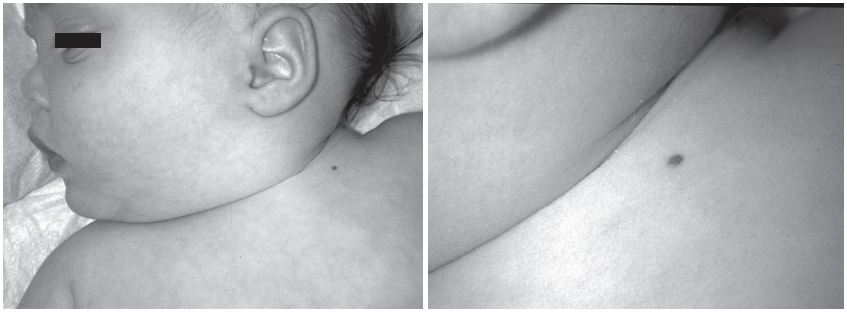


Figure 26-4. Hemangioma of the neck. *(Photograph courtesy of J. Loiselle.)*

Key Points: Methods to Diagnose Laryngomalacia

1. Clinical diagnosis
2. Airway fluoroscopy
3. Direct laryngoscopy

CHAPTER 27

SYNCOPE

James M. Callahan

1. What is syncope?

Syncope is a sudden, transient loss of consciousness and muscle tone due to a reversible impairment in cerebral perfusion or substrate delivery (oxygen or glucose). Unconsciousness usually lasts no longer than 1 to 2 minutes. Patients or their families may say they fainted, passed out, or blacked out.

2. How common is syncope in the pediatric age group?

Syncope is common. The overall incidence is 0.5% to 1.0% in children and adolescents. In younger children, there is no difference in incidence by gender. Syncope is more common in adolescents, with 15% to 50% of all adolescents experiencing at least one episode of syncope by adulthood. In adolesence, twice as many females as males have syncope. In various series, it has been found to account for about 0.1% to 3% of visits to pediatric emergency departments (EDs).

Fischer JWJ, Cho CS: Pediatric syncope: Cases from the emergency department. Emerg Med Clin North Am 2010;28:501-516.

Massin MM, Bourguignont A, Coremans C, et al: Syncope in pediatric patients presenting to an emergency department. J Pediatr 2004;145:223-228.

Pratt JL, Fleisher GR: Syncope in children and adolescents. Pediatr Emerg Care 1989;5:80-82.

Weitlin W, Ganzeboom KS, Saul JP: Reflex syncope in children and adoloescents. Heart 2004;90:1094-1100.

3. Is syncope in children serious?

Most syncope in children is due to benign causes. One third to as many as 80% of cases are *simple neurocardiogenic or vasodepressor (vasovagal) episodes*. In the adult population, only 5% of episodes are vasodepressor events, and one quarter are due to a cardiac cause. Even so, syncope may be due to a life-threatening condition in children and adolescents. Up to 25% of pediatric and adolescent patients with sudden death have had at least one prior episode of syncope.

Friedman KG, Alexander ME: Chest pain and syncope in children: A practical approach to the diagnosis of cardiac disease. J Pediatr 2013;163:896-901.

Massin MM, Bourguignont A, Coremans C, et al: Syncope in pediatric patients presenting to an emergency department. J Pediatr 2004;145:223-228.

Pratt JL, Fleisher GR: Syncope in children and adolescents. Pediatr Emerg Care 1989;5:80-82.

4. What are the common causes of syncope in children and adolescents?

More than one half of patients have experienced a simple vasodepressor episode. Other disorders of autonomic control, including orthostatic hypotension (20% of cases in one series), breath-holding spells, situational syncope (e.g., tussive syncope, micturition syncope), and hyperventilation are also common. Hysterical faints (pseudosyncope) are common in adolescents.

5. Name the potentially life-threatening causes of syncope in children and adolescents.

- Cardiac arrhythmias
- Structural heart disease
- Seizures
- Subarachnoid hemorrhage
- Carbon monoxide poisoning
- Effects of medications and ingestions

Key Points: Potentially Fatal Causes of Syncope in Children and Adolescents

1. Cardiac arrhythmias (including causes of long QT intervals and Brugada syndrome)
2. Structural heart disease (including hypertrophic cardiomyopathy)
3. Seizures
4. Subarachnoid hemorrhage
5. Carbon monoxide poisoning
6. Effects of medications and ingestions

6. How is the cause of a syncopal event usually determined?

The history and physical examination are enough to suggest the most likely cause of syncope in the vast majority of patients. Even so, remember that tachyarrhythmias often present in much the same way as vasodepressor syncope. Obtain an electrocardiogram (ECG) for all patients presenting with syncope.

Fischer JWJ, Cho CS: Pediatric syncope: Cases from the emergency department. Emerg Med Clin North Am 2010;28:501-516.

Friedman KG, Alexander ME: Chest pain and syncope in children: A practical approach to the diagnosis of cardiac disease. J Pediatr 2013;163:896-901.

Steinberg LA, Knilans TK: Syncope in children: Diagnostic tests have a high cost and low yield. J Pediatr 2005;146:355-358.

7. What historical factors help to separate benign from serious causes of syncope?

Patients with vasodepressor syncope usually experience a prodrome or may faint after a precipitating event (e.g., pain, fright, startle). They can usually describe dizziness or lightheadedness, nausea, warmth, and a visual gray-out, often with tunnel vision, before fainting. They often have time to lower themselves slowly or brace themselves against something. Associated injuries such as lacerations or large hematomas are rare. Vasodepressor syncope usually occurs in a standing or sitting position. Syncope with associated injuries, that occurs with exertion or when the patient is supine, in infancy, and that is recurrent is more likely due to a serious underlying disorder.

Friedman KG, Alexander ME: Chest pain and syncope in children: A practical approach to the diagnosis of cardiac disease. J Pediatr 2013;163:896-901.

Moodley M: Clinical approach to syncope in children. Semin Pediatr Neurol 2013;20:12-17.

Key Points: Warning Signs of Potentially Serious Syncope

1. History of heart murmur or congenital heart disease, a new murmur on examination, or a murmur that is heard as a patient moves from squatting to standing; other abnormal findings on cardiac examination
2. Family history of arrhythmias, sudden death, cardiomyopathy, sensorineural hearing loss
3. Exertional syncope
4. Syncope without a prodrome consistent with vasodepressor syncope
5. Use of medications associated with arrhythmias
6. Attacks associated with hyperpnea or cyanosis
7. Syncope that leads to other injuries (lacerations, large hematomas)

8. What is the goal of the evaluation of syncope in the ED?

It has been said: "Syncope and death are the same—except that in one you wake up." The goal of the ED evaluation is to identify the rare pediatric patient with a serious underlying disorder, while realizing that the majority of patients have probably suffered a vasodepressor episode (a diagnosis of exclusion). Extensive workups are rarely required, usually nondiagnostic, and often expensive.

Steinberg LA, Knilans TK: Syncope in children: Diagnostic tests have a high cost and low yield. J Pediatr 2005;146:355-358.

9. Are there diagnostic tests that *every* patient with syncope should undergo in the ED?

A thorough history (including family history and social history) and physical examination are the most important parts of the ED evaluation. Perform orthostatic blood pressures and

complete cardiac and neurologic examinations on every patient. Screen all patients with an ECG, looking for signs of hypertrophy or abnormal conduction times (e.g., long QT syndrome) or ST changes (e.g., Brugada syndrome). Other diagnostic testing is guided by the results of the history, physical examination, and ECG. If there are diagnostic questions after this initial workup or when syncope is recurrent, referrals for specialized testing (echocardiography, tilt table testing, Holter or event-recorder monitoring, or stress testing) may be made.

Key Points: Initial Workup for All Pediatric Patients with Syncope

1. Complete history, including history of event and medication use as well as family history of syncope, sudden death, cardiac disease, and sensorineural hearing loss
2. Thorough physical examination, including orthostatic vital signs, complete cardiac examination, and complete neurologic examination
3. ECG

10. How are episodes of vasodepressor syncope and seizures different?

The description of the syncopal event and surrounding circumstances may help distinguish between the two. With vasodepressor syncope, the patient is usually unconscious for only a matter of seconds, and incontinence is rare. Once awake, there may be some mild fatigue, but a true postictal period with decreased responsiveness and marked confusion is absent. Tonic-clonic movements usually occur near the end of a syncopal episode and last for only a few seconds, but they often are more persistent and last for at least a few minutes in patients with seizures. If the nature of the event is uncertain, an electroencephalogram (EEG) may be helpful.

11. What is the proposed pathophysiology of vasodepressor syncope?

It is thought that a prolonged upright position leads to venous pooling in the lower extremities, causing a decreased left ventricular volume. In response to this and possibly a precipitating event, there is a catecholamine surge causing increased contractility and a strong contraction against a relatively empty ventricle. Cardiac vagal fibers in the ventricular wall are activated and stimulate the medulla oblongata, causing a sympathetic withdrawal with or without vagal stimulation (Bezold-Jarisch reflex). These events produce bradycardia and a profound loss in systemic vascular resistance, decreased cerebral perfusion, and syncope. The term *vasodepressor* syncope is more accurate than *vasovagal* syncope, as it has been shown that vagal activity may contribute but is not necessary for these events to occur.

Batra AS, Hohn AR: Palpitations, syncope and sudden cardiac death in children: Who's at risk? Pediatr Rev 2003;24:269-275.

12. What are breath-holding spells?

Breath-holding spells are common in infants and toddlers, probably related to developmental differences in autonomic control. Two types are described. In *cyanotic* breath-holding spells, some provocation produces crying. The child develops a sustained expiration and becomes silent. There is deepening cyanosis, a loss of muscle tone, and, often, opisthotonus. There may be a brief period of tonic-clonic movement at the event's conclusion. The child then makes an inspiratory gasp, normal respirations resume, and the child slowly awakens.

With *pallid* breath-holding spells, there is usually the abrupt onset of pallor and loss of consciousness after one to two cries. Opisthotonus is followed by relaxation and gradual awakening. In one series, 17% of children with pallid breath-holding spells went on to have vasodepressor syncope in later life.

Lombroso CT, Lerman P: Breathholding spells (cyanotic and pallid infantile syncope), Pediatrics 39:563–581, 1967.

13. How common are breath-holding spells, and at what ages are they seen?

Breath-holding spells associated with unconsciousness are seen in up to 5% of all children. Onset of spells is usually in the first year of life and almost always by age 2. These are benign events that resolve spontaneously, and the spells cease occurring in 50% of children by age 4, 90% by age 6, and more than 99% by age 8.

14. What types of syncope are associated with orthostatic changes?

Orthostatic hypotension may be due to dehydration or anemia. *Micturition syncope*, *syncope with defecation*, and *syncope occurring during menses* are all related to orthostatic changes. A variety of

medications (prescribed; accidentally or intentionally ingested) may cause orthostasis, including antihypertensives, antidepressants, phenothiazines, sedatives, and diuretics. Tussive syncope (seen in patients with pertussis and severe asthma) results when coughing, respiratory spasm, and Valsalva maneuver cause increased intrapleural pressure, decreased venous return, and decreased left ventricular filling.

15. What do POTS (and pans?) have to do with syncope?

Postural orthostatic tachycardia syndrome, or POTS, has been recognized increasingly in pediatric and adolescent patients. Orthostatic symptoms are common, including dizziness, lightheadedness, presyncope, weakness, nausea, visual changes, and occasionally syncope. The diagnosis of POTS is given when there are chronic complaints of orthostatic intolerance (>3 months) and an increased heart rate when standing. In adults, an increase of 30 beats per minute or more, or a heart rate of 120 beats per minute or higher within 10 minutes of standing, is considered increased for this diagnosis. In children and adolescents, an increase of 35 to 40 beats per minute or higher has been used. POTS may be associated with symptoms of chronic fatigue, fibromyalgia, and other somatic complaints. Iron deficiency and low iron stores have been associated with POTS. A recent study reported an association with vitamin B_{12} deficiency. A variety of therapies may be useful in various individuals, including improved hydration, medications, and improved physical conditioning.

Pans are not related to syncope in children!

Jarjour IT: Postural tachycardia syndromes in children. Semin Pediatr Neurol 2013;20:18-26.
Oner T, Guven B, Tavli V, et al: Postural orthostatic tachycardia syndrome (POTS) and vitamin B_{12} deficiency in adolescents. Pediatrics 2014;133:e138-e142.
Stewart JM: Common syndromes of orthostatic intolerance. Pediatrics 2013;131:968-980.

16. What arrythmias are associated with syncope in children and adolescents?

Ventricular tachycardia, although rare, is a potentially life-threatening cause of syncope. Supraventricular tachycardia may cause presyncopal symptoms, but rarely true syncope. Congenital complete heart block may not cause symptoms until later childhood or adolescence. Complete heart block may also be acquired (e.g., in patients with untreated Lyme disease). Patients with structural heart disease and those who have had previous surgery to repair congenital heart disease are at increased risk of arrhythmias. Paroxysmal episodes of ventricular tachycardia may occur in the setting of a long QT interval.

17. How is the diagnosis of long QT syndrome made?

Long QT syndrome is diagnosed by finding a long QT interval on the patient's ECG. The QT interval must be corrected (QTc) for the patient's heart rate using Bazett's formula:

$$QTc = (QT/\sqrt{RR'})$$

The QT interval should be measured from the beginning of the QRS complex to the end of the T wave in lead II, V_5, or V_6. The measured RR′ time should be the RR interval that immediately precedes the measured QT interval. The QTc should be less than 0.45 second in children younger than 16 years of age.

Friedman MJ, Mull CC, Sharieff GQ, Tsarouhas NT: Prolonged QT syndrome in children: an uncommon but potentially fatal entity, J Emerg Med 24:173–179, 2003.

18. What are the familial forms of long QT syndrome?

Long QT syndrome is associated with sensorineural hearing loss and autosomal recessive inheritance in the Jervell and Lange-Nielsen syndrome, and with autosomal dominant inheritance and normal hearing in the Romano-Ward syndrome. Ask about family history of recurrent syncope, tachyarrhythmias, seizures, sudden death, and hearing loss.

19. If a diagnosis of long QT syndrome is made, what should be done?

Patients with long QT syndrome have a mortality rate of up to 70% if not treated. Immediate cardiology consultation is required. Beta blockers are the usual medical treatment for this disease. Disposition often includes admission for further monitoring. Advise family members to have ECGs to investigate for the familial forms of this syndrome. A long QT interval may be acquired in the setting of electrolyte abnormalities (e.g., hypocalcemia) and may be due to certain medications.

20. What medications and other drugs may be associated with tachyarrhythmias?

- Nonsedating antihistamines (terfenadine and astemizole)*
- Cisapride*
- Antiemetics (e.g., ondansetron)
- Tricyclic antidepressants
- Antipsychotics (e.g., haloperidol)
- Cocaine (including crack)
- Carbamazepine (usually in overdose only)
- Amphetamines
- Inhalants (especially Freon)

21. Are there other causes of syncope that are cardiac in nature?

Structural heart disease may cause syncope or sudden death. Hypertrophic cardiomyopathy (hypertrophic obstructive cardiomyopathy [HOCM] or idiopathic hypertrophic subaortic stenosis) is associated with a thickened left ventricular wall, especially along the septum in the subaortic outflow tract. With exertion and increased contractility, outflow tract obstruction and syncope occur. *Hypertrophic cardiomyopathy* is the most common autopsy-proven cause of death in young athletes.

An aberrant coronary artery that courses between the aorta and pulmonary artery also can be associated with exertional syncope, due to ischemia resulting in arrhythmias. Other rare causes that result in ventricular outflow obstruction include valvar aortic stenosis and primary pulmonary hypertension. Also, consider atrial myxoma, a very rare cardiac tumor that may cause recurrent syncope in infants and toddlers. Finally, dilated cardiomyopathies and myocarditis due to a variety of infectious agents can be associated with arrhythmias or pump failure.

22. Other than seizures, are there other central nervous system (CNS) events that may precipitate syncope?

Syncope may be a presenting symptom of *atypical migraines*. Often there is a history of an aura and headaches. Nausea and vomiting are common. Pain is frequently unilateral and may be throbbing in nature. Basilar artery migraines may affect equilibrium and the patient's vision. A family history of migraines can help make the diagnosis.

Spontaneous subarachnoid hemorrhage is rare in children but may present with severe thunderclap headache (worst of the patient's life) and syncope.

23. What metabolic derangements can cause syncope?

Hypoglycemia can cause syncope. This is rare in children after infancy, except those on insulin. A decreased level of consciousness due to hypoglycemia usually does not resolve spontaneously. Fasting, which leads to increased counterregulatory hormones including catecholamines, may play a role in vasodepressor syncope. Carbon monoxide poisoning, anemia, dehydration, and pregnancy may also be associated with syncope.

24. How does a patient with pseudosyncope (hysterical faints) present?

Hysterical faints are usually seen in adolescents. They typically occur in front of an audience, with an absence of physical findings. There is often eye fluttering behind half-closed eye lids. Self-protective behaviors are preserved (e.g., patients will not allow their own hand to fall and hit their face). Social history often reveals marked stress at home, in school, or in other social situations.

25. What is the treatment for the most common causes of syncope (vasodepressor and orthostatic)?

The most effective treatment for vasodepressor and orthostatic causes of syncope is to ensure adequate fluid intake. Older children and adolescents should drink at least 64 ounces of noncaffeinated fluids daily. The patient should drink enough that the urine remains pale and clear. Small amounts of salty foods may also help to maintain intravascular volume. Counsel patients to lie down when they have prodromal symptoms to prevent episodes of syncope. Refer patients who continue to have episodes of syncope for tilt table testing and possible pharmacologic therapy.

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*Especially when taken with macrolide antibiotics, metronidazole, or ketoconazole; of note, these medications are no longer available in the United States.

26. Which patients with syncope require referral to a cardiologist or neurologist?

Refer patients to a cardiologist if they have syncope with exertion; syncope associated with chest pain, arrhythmias, or palpitations; or syncope that is recurrent or not responding to usual therapies. Also refer patients with syncope who have an abnormal cardiac history, physical examination, or ECG or those who have a family history of sudden death or who have atypical episodes of syncope. Patients with focal neurologic findings, other neurologic abnormalities, or a history that is consistent with the presentation of a seizure should be seen by a neurologist as soon as possible.

Delgado CA: Syncope. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 589-595.

27. What findings in patients with syncope require admission?

Admit patients to the hospital if they have cardiovascular disease or an abnormal cardiac examination, serious ECG abnormalities (e.g., long QTc interval or complete heart blocks), syncope with chest pain, or cyanotic spells, apnea, or focal neurologic findings. Also admit patients for observation, further diagnostic testing, and treatment if they have a toxic ingestion or orthostatic hypotension that doesn't respond to intravenous fluids.

Delgado CA: Syncope. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 589-595.

CHAPTER 28

VAGINAL BLEEDING/DISCHARGE

Jane M. Lavelle

1. What is the average age at menarche?

The average age at menarche is 12.7 years, with a normal range of 11 to 14 years. Typically, it occurs approximately 2 years after thelarche and 1 year after peak height velocity.

2. Describe the normal menstrual cycle.

One menstrual cycle is the time between the onset of one menses to the onset of another. Normal cycle length varies (21-45 days in teens), and menses lasts from 2 to 8 days and results in an average blood loss of 30 to 40 mL. Clinically, the menstrual cycle is usually defined by the ovarian cycle, which includes the follicular, ovulatory, and luteal phases.

During the follicular phase (7-22 days), low levels of estradiol and progesterone result in elevated gonadotropin-releasing hormone levels and, thus, rises in both follicle-stimulating hormone (FSH) and luteinizing hormone (LH). FSH stimulates the maturation of one follicle, and LH stimulates the theca cells to produce androgens, which are converted to estrogens that stimulate proliferation of the endothelium. As estradiol levels rise, FSH levels begin to fall. During the ovulatory phase, a preovulatory estradiol surge causes an LH surge, resulting in release of the ovum. During the luteal phase, the corpus luteum produces large amounts of progesterone and estrogen, resulting in development of the secretory endometrium. If fertilization does not occur, involution of the corpus luteum occurs, and there is loss of estrogen and progesterone. Sloughing of the endometrium follows, and increased levels of FSH lead to a new cycle.

Adams Hillard PJ, Deitch HR: Menstrual disorders in the college age female. Pediatr Clin North Am 2005;52:179-198.

Gordon CM, Neinstein LS: Normal menstrual physiology. In Neinstein LS (ed): Adolescent Health Care: A Practical Guide, 4th ed. Baltimore, Wolters Kluwer/Williams & Wilkins, 2002, pp 947-952.

3. What is dysfunctional uterine bleeding (DUB)?

DUB is excessive menstrual bleeding occurring during the menses or outside the normal intervals in the absence of underlying structural abnormalities. An orderly sequence of hormonal and endometrial events is responsible for the regular and limited bleeding that occurs in adult women. In adolescents, the most common cause of DUB results from anovulatory menstrual cycles due to "immaturity" of the hypothalamic-pituitary-ovary axis, with lack of normal negative feedback. During anovulatory cycles, estrogen levels are increased without increasing FSH responsible for the subsequent fall in the estrogen level. A lack of progesterone normally produced by the corpus luteum, which stabilizes the endometrium, results in sporadic growth and sloughing of the endometrium. In adolescents, it may be more useful to think of this as anovulatory uterine bleeding, because this term reflects the most common cause in adolescents.

Adams Hillard PJ, Deitch HR: Menstrual disorders in the college age female. Pediatr Clin North Am 2005;52:179-198.

Mitan LA, Slap GB: Dysfunctional uterine bleeding. In Neinstein LS (ed): Adolescent Health Care: A Practical Guide, 4th ed. Baltimore, Wolters Kluwer/Williams & Wilkins, 2002, pp 966-972.

4. List the causes of abnormal vaginal bleeding in the adolescent female

Although anovulation is the most common cause of dysfunctional vaginal bleeding in the adolescent, it remains a diagnosis of exclusion. The diseases listed in Table 28-1 must be considered when excessive vaginal bleeding is present.

Table 28-1. Causes of Abnormal Vaginal Bleeding in the Adolescent Female

Life-Threatening
Ectopic pregnancy, vaginal/cervical laceration

Common
Anovulation, sexually transmitted infections, pregnancy/complications of pregnancy, hormonal contraception

Complete Differential Diagnosis by Category

Pregnancy-Related	Systemic Disease	Genital Tract
Pregnancy, ectopic pregnancy, threatened abortion, spontaneous abortion, hydatidiform mole	Coagulation abnormalities, von Willebrand disease, idiopathic thrombocytopenic purpura, renal failure, liver failure, systemic lupus erythematosus, malignancies	Sexually transmitted diseases, trauma, tumor, foreign body, malignancy, endometriosis, myoma, polyp
Endocrine	**Drugs**	
Anovulation, polycystic ovary syndrome, hypothyroidism/ hyperthyroidism, Cushing's disease, Addison's disease, premature ovarian failure, ovarian tumor	Hormonal contraceptives, anticonvulsants, anticoagulants, chemotherapeutic agents	

5. How should a clinician evaluate a patient to determine the source of vaginal bleeding?

After a careful physical examination, include a pelvic examination to evaluate the source of the bleeding and any disease. Screens for sexually transmitted diseases (STDs), a pregnancy test, and a serum hemoglobin are useful. In the young teenager who is not sexually active and has mild symptoms, a pelvic examination may be deferred. As always, follow-up is an important part of patient care.

Adams Hillard PJ, Deitch HR: Menstrual disorders in the college age female. Pediatr Clin North Am 2005;52:179-198.

Mitan LA, Slap GB: Dysfunctional uterine bleeding. In Neinstein LS (ed): Adolescent Health Care: A Practical Guide, 4th ed. Baltimore, Wolters Kluwer/Williams & Wilkins, 2002, pp 966-972.

6. What are the recommended therapies for DUB?

Patients with DUB present with a wide spectrum of severity of illness. Therapy is aimed at stopping the bleeding by converting the endometrium to the secretory state so that sloughing can occur under controlled conditions, correcting the anemia, restoring normal cyclic bleeding, and preventing recurrence and long-term sequelae of anovulation.

A combination of estrogen and progesterone is needed in patients with active bleeding. Any pill combining 35 or 50 μg of ethinyl estradiol or mestranol and a progestin can be used. Progestin only may be used in patients who are not actively bleeding (Table 28-2).

In patients with severe bleeding, attention to the ABCs (airway, breathing, and circulation) is necessary, with intravenous (IV) access and fluid/blood resuscitation. All patients with active bleeding, low hemoglobin, and change in vital signs require admission for treatment. Include coagulation studies in the evaluation. Bleeding usually stops after 24 hours of treatment. Combination pills with a higher dose of estrogen (50 μg of ethinyl estradiol) are the first-line therapy. IV estrogen is reserved for use in unstable patients; pulmonary embolism is associated with this therapy. In patients for whom estrogen is contraindicated,

Table 28-2. Therapies for Dysfunctional Uterine Bleeding

SEVERITY	HEMOGLOBIN LEVEL (G/DL)	THERAPY
Mild	>12	Menstrual calendar Iron therapy Follow-up 3-6 months
Moderate	10-12, not bleeding	Low-dose OCP Iron therapy Follow-up 3-6 months
	<10, not bleeding	Low-dose OCP or progestin only Iron therapy Follow-up 3-6 months
	<10, bleeding	High-dose OCP 1 pill four times daily for 4 days 1 pill three times daily for 3 days 1 pill twice daily for 2 weeks Follow-up 3-6 months
Severe	<7, hemodynamic symptoms	High-dose OCP IV conjugated estrogen (unstable patients) Coagulation workup, blood transfusion prn Iron therapy Follow-up 3-6 months

IV, intravenous; OCP, oral contraceptive pill (combination of estrogen, progesterone, and suggested minimum of 30 μg ethinyl estradiol); prn, as needed. Antiemetics are usually needed when a higher dose of estrogen is given.

progesterone regimens can be tried. If this fails, other therapies include aminocaproic acid, desmopressin, and surgical curettage.

Mitan LA, Slap GB: Dysfunctional uterine bleeding. In Neinstein LS (ed): Adolescent Health Care: A Practical Guide, 4th ed. Baltimore, Wolters Kluwer/Williams & Wilkins, 2002, pp 966-972.
Slap GB: Menstrual disorders in adolescence. Best Prac Res Clin Obstet Gynecol 2003;17:75-92.
Strickland JL, Wall JW: Abnormal uterine bleeding in adolescents. Obstet Gynecol Clin North Am 2003;30:321-335.

7. What is primary dysmenorrhea?

Primary dysmenorrhea is painful menses without associated pelvic disease that typically appears 6 to 24 months following menarche. Dysmenorrhea typically occurs with ovulatory cycles, which become more frequent as the hypothalamic-pituitary-ovarian axis matures. Two years after menarche, 20% to 50% of teens have ovulatory cycles. Occasionally, dysmenorrhea occurs with anovulatory cycles. Typical symptoms include crampy lower abdominal pain beginning a few days before or at the start of the menstrual cycle and lasting for 1 to 3 days. Associated symptoms include fatigue, back pain, headache, nausea, vomiting, and diarrhea.

8. What causes dysmenorrhea?

Dysmenorrhea is caused by prostaglandins E_2 and $F_{2\alpha}$. These are produced in higher concentrations in ovulatory cycles during the secretory phase. Locally, prostaglandins cause myometrial contraction; however, when they enter the systemic circulation they can cause fatigue, headache, dizziness, nausea, vomiting, diarrhea, and back pain. Prostaglandin $F_{2\alpha}$ causes uterine contractions, vasoconstriction, and ischemia. Prostaglandin E_2 causes platelet disaggregation and vasodilation.

9. How common is dysmenorrhea?

It occurs very commonly during adolescence. Half to three quarters of teens experience dysmenorrhea that affects their daily activities. Fifteen percent of teens describe severe symptoms that incapacitate them for 1 to 3 days during each menstrual cycle. However, only 15% of teens seek medical care for menstrual pain.

Banikarim C, Middleman AB: Primary dysmenorrhea in adolescents. UpToDate, 2013. Available at www.uptodate.com.
Braverman PK, Neinstein LS: Dysmenorrhea and premenstrual syndrome. In Neinstein LS (ed): Adolescent Health Care: A Practical Guide, 4th ed. Baltimore, Wolters Kluwer/Williams & Wilkins, 2002, pp 952-963.

10. What is the approach to a patient with primary dysmenorrhea?

Teens who present 6 to 12 months after menarche, who are younger than 20 years of age, and who describe pain with menses can be treated and followed for resolution of symptoms. A pelvic examination is not necessary in young teens who are not sexually active. Pelvic examination, laboratory testing, and imaging are reserved for patients who have atypical symptoms; have signs, symptoms, or risk factors for other diseases; or do not respond to therapy.

Banikarim C, Middleman AB: Primary dysmenorrhea in adolescents. UpToDate, 2013. Available at www.uptodate.com.
Braverman PK, Neinstein LS: Dysmenorrhea and premenstrual syndrome. In Neinstein LS (ed): Adolescent Health Care: A Practical Guide, 4th ed. Baltimore, Wolters Kluwer/Williams & Wilkins, 2002, pp 952-963.

11. What is the treatment for primary dysmenorrhea?

Nonsteroidal anti-inflammatory drugs (NSAIDs) are the first-line therapy for dysmenorrhea; a majority (70-80%) of patients experience relief with their use. Ibuprofen, naproxen, and naproxen sodium have all been used successfully. Begin the medication at the onset of the premenstrual symptoms or at the onset of menses and continue it for 1 to 3 days as needed to control symptoms. Use the chosen NSAID for four cycles until treatment failure is considered. A second NSAID can be tried at that time. Mefenamic acid competes with prostaglandin-binding sites, antagonizes existing prostaglandin, and inhibits prostaglandin synthesis and thus may be more effective than other NSAIDs.

If the patient continues with symptoms despite NSAID use, a 3- to 6-month course of combination oral contraceptive therapy may alleviate dysmenorrhea. Ovulation is suppressed, as are prostaglandin production and menstrual flow.

Follow patients closely. If they do not respond to the described therapies, a reevaluation seeking secondary causes is indicated.

Banikarim C, Middleman AB: Primary dysmenorrhea in adolescents. UpToDate, 2013. Available at www.uptodate.com.
Braverman PK, Neinstein LS: Dysmenorrhea and premenstrual syndrome. In Neinstein LS (ed): Adolescent Health Care: A Practical Guide, 4th ed. Baltimore, Wolters Kluwer/Williams & Wilkins, pp 952-963.

12. What are the causes of secondary dysmenorrhea?

The causes of secondary dysmenorrhea are listed in Table 28-3.

Key Points: Vaginal Bleeding

1. Anovulation due to immaturity of the hypothalamic-pituitary-ovarian axis is the most common cause of DUB in the adolescent patient.
2. Dysmenorrhea is common in adolescents and begins 6 to 24 months after menarche, when ovulatory cycles occur with more frequency. It is associated with significant morbidity.

Table 28-3. Causes of Secondary Dysmenorrhea

GYNECOLOGIC DISORDERS	NONGYNECOLOGIC DISORDERS
Endometriosis, pelvic inflammatory disease, pelvic adhesions, ovarian cysts, mass, polyps, fibroids, congenital obstructive müllerian malformations (i.e., septate or biconate uterus), vaginal septum	Inflammatory bowel disease, irritable bowel syndrome, ureteropelvic junction obstruction, renal stone, cystitis, psychogenic disorder

13. When should a pregnancy test be included in a workup?

Unfortunately, teenage pregnancy continues to be a common occurrence; thus, always include pregnancy in the differential diagnosis of many chief complaints, and have a low threshold for performing a pregnancy test. Fortunately, these tests have become very sensitive and specific and are quick and relatively inexpensive.

The most commonly used tests rely on enzyme-linked immunosorbent assay (ELISA) for the detection of the β-human chorionic gonadotropin (β-hCG) subunit. The level of β-hCG secreted by the trophoblast doubles every other day during the first 6 weeks of pregnancy. By the time it reaches 1000 to 2000 mIU/mL, a gestational sac can be seen via vaginal ultrasound (US). Urine ELISA detects β-hCG levels of 30 to 50 mIU/mL and is typically positive within 7 days of implantation. Thus, the result of this test is positive at the time of the missed menses. Practically speaking, if the teen is concerned about pregnancy and the urine test result is negative, she should return the following week for a repeat test.

Neinstein LS, Farmer M: Teenage pregnancy. In Neinstein LS (ed): Adolescent Health Care: A Practical Guide, 4th ed. Baltimore, Wolters Kluwer/Williams & Wilkins, 2002, pp 809-833.

14. What is the differential diagnosis of vaginitis in young women?

Vaginal discharge, foul odor, itching and irritation of the vulva, spotting, and dyspareunia characterize vaginitis. The most common causes include infections due to *Trichomonas vaginalis*, *Candida albicans*, and *Gardnerella vaginalis*. Vaginal discharge can also occur with cervicitis due to *Neisseria gonorrhoeae* or *Chlamydia trachomatis*. Noninfectious causes include foreign body (tampon), trauma, allergies, chemical irritants, and poor hygiene. Cause of vaginitis is determined by physical examination, the vaginal pH, the whiff test, saline and potassium hydroxide (KOH) microscopy, and polymerase chain reaction (PCR) testing for *N. gonorrhoeae* and *Chlamydia*.

Centers for Disease Control and Prevention: Sexually Transmitted Diseases Treatment Guidelines 2010. MMWR Morb Mortal Wkly Rep 2010;59:RR-12.

15. How is the diagnosis of cervicitis made?

Most young women with cervicitis are asymptomatic. Some, however, may present with abnormal vaginal discharge or bleeding, dysuria, frequency, dyspareunia, or bleeding with intercourse. On pelvic examination, the cervix appears inflamed, and mucopurulent discharge is visible. Often, increased friability leads to bleeding. Identifiable causes of cervicitis include *C. trachomatis*, *N. gonorrhoeae*, *T. vaginalis*, *C. albicans*, and herpes simplex virus (HSV). Thus, evaluation should include a wet mount, a KOH preparation, and diagnostic tests for *C. trachomatis* and *N. gonorrhoeae*. However, a significant number of patients do not have laboratory evidence of *C. trachomatis* and *N. gonorrhoeae* infection.

Comkornuecha K: Gonococcal infections. Pediatr Rev 2013;34(5):228-234.

16. What is the treatment and follow-up for cervicitis?

Treatment at the time of the examination depends on the level of clinical suspicion, as well as reliability of follow-up of the patient. Currently, unless cervicitis is known to be caused by one of the organisms listed here, provide therapy to cover both *C. trachomatis* and *N. gonorrhoeae*. At this time, for quinolone-resistant *N. gonorrhoeae*, ceftriaxone is the recommended therapy; oral cephalosporin therapy is no longer recommended. Practitioners should review the prevalence of *N. gonorrheoae* and resistance patterns in their geographic area and refer to the Centers for Disease Control (CDC) Sexually Transmitted Disease Treatment Guidelines.

The recommended treatment regimens are effective; thus, "test of cure" or retesting is not routinely recommended. Patients with persistent symptoms require reevaluation. Patients who test positive for *Neisseria gonorrhoeae* or *Chlamydia* should be retested at 3 to 6 months, as risk for reinfection is high. All sexual partners within the preceding 2 months should be evaluated and treated.

Centers for Disease Control and Prevention: Sexually Transmitted Diseases Treatment Guidelines 2010. MMWR Morb Mortal Wkly Rep 2010;59:RR-12.

17. What is pelvic inflammatory disease (PID)?

PID is a polymicrobial infection of the upper genital tract in postpubertal women that is caused by a sexually transmitted infection (STI). This disease presents with a broad spectrum of clinical manifestations, making an accurate diagnosis challenging. Further, many teens have

mild or subtle symptoms. Clinical diagnosis is imprecise; in symptomatic patients salpingitis can be demonstrated by laparoscopy in 65% to 95% of cases. There is no single historical finding, physical examination finding, or laboratory test that is sensitive and specific for the diagnosis. Many cases of PID are missed. Common symptoms include crampy lower abdominal pain; abnormal vaginal discharge/bleeding; anorexia/vomiting; fever; diarrhea; dysuria; and dyspareunia. Important parts of the differential diagnosis include ectopic pregnancy, ovarian torsion, appendicitis, threatened abortion, and endometriosis. Teens are at high risk for this disease; include PID in the differential diagnosis of abdominal pain and have a low threshold for treatment due to significant morbidity associated with missed diagnosis.

18. How is the diagnosis of PID made?

The diagnosis is considered in sexually active teens with abdominal or pelvic pain who have one or more of the following minimum criteria: (1) uterine tenderness, (2) cervical motion tenderness, or (3) adnexal tenderness.

If these signs are noted, treat the patient empirically unless another diagnosis is present. Additional criteria include oral temperature higher than 38.3° C, abnormal cervical or vaginal mucopurulent discharge, presence of white blood cells on saline microscopy of vaginal secretions, elevated erythrocyte sedimentation rate, and elevated C-reactive protein level, and laboratory evidence of *C. trachomatis* or *N. gonorrhoeae* infection can be used along with the minimum criteria to increase specificity.

Centers for Disease Control and Prevention: Sexually Transmitted Diseases Treatment Guidelines 2010. MMWR Morb Mortal Wkly Rep 2010;59:RR-12.

Pletcher JR, Slap GB: Pelvic inflammatory disease. In Neinstein LS (ed): Adolescent Health Care: A Practical Guide, 4th ed. Baltimore, Wolters Kluwer/Williams & Wilkins, 2002, pp 1161-1170.

19. What is the treatment for PID? When is hospitalization necessary?

Regimens for the treatment of PID have been developed to cover the polymicrobial nature of the disease. Thus, therapy must be effective against *N. gonorrhoeae* and *C. trachomatis* as well as anaerobes, *Streptococcus* spp., gram-negative enterics, and *Mycoplasma* spp. Outpatient treatment is effective and results in similar outcomes as compared to parenteral therapy. Consider hospitalization in the following instances: pregnancy, unclear diagnosis, vomiting, peritoneal signs, the young teenager (age < 15 years), tubo-ovarian abscess present or suspected, failed outpatient treatment, or patient's inability to follow the outpatient regimen. Instruct patients to take their medications, rest, and avoid intercourse. Testing and treatment of all sexual partners within the preceding 2 months is indicated.

The CDC 2010 STD Guidelines recommend several regimens for both outpatient and inpatient treatment for PID. The most common outpatient regimen in patients with no history of drug allergy includes intramuscular (IM) or IV ceftriaxone plus a 10-day course of doxycycline, with or without a 14-day course of metronidazole. Metronidazole enhances anaerobic coverage and also treats bacterial vaginosis (BV), which is often present. Reevaluate patients 2 to 3 days following initiation of therapy. If there is not substantial clinical improvement at this time, inpatient therapy and additional diagnostic evaluation is recommended. Offer counseling about sexual health and STI screening.

Centers for Disease Control and Prevention: Sexually Transmitted Diseases Treatment Guidelines 2010. MMWR Morb Mortal Wkly Rep 2010;59:RR-12.

Pletcher JR, Slap GB: Pelvic inflammatory disease. In Neinstein LS (ed): Adolescent Health Care: A Practical Guide, 4th ed. Baltimore, Wolters Kluwer/Williams & Wilkins, 2002, pp 1161-1170.

Key Points: Sexually Transmitted Infections

1. Adolescents between the ages of 15 and 19 years have the highest rate of STIs.
2. Consider STIs when evaluating teens with lower abdominal pain, vaginal discharge/discomfort, dysuria, or abnormal menstrual bleeding.
3. STIs are rarely found in prepubertal children who are victims of sexual abuse.
4. Routine vaginal cultures for STIs are not indicated in prepubertal children who are victims of sexual abuse.

20. How can urine be helpful in diagnosisng STDs?

The development of Food and Drug Administration (FDA)–approved nucleic acid amplification tests (NAATs) for *N. gonorrhoeae* and *C. trachomatis* has revolutionized the ability to screen at-risk populations as well as to identify infection in symptomatic patients. The sensitivity of these tests ranges from 85% to 100%. The specificity is 99%. Because these tests can be done by using urine, NAAT offers the most noninvasive method for screening and diagnosing STIs. These tests can also be used on cervical, urethral, or vaginal swab specimens and can be incorporated into the annual evaluation of adolescents. NAAT has not been cleared by the FDA for use with oral or rectal swabs; however, preliminary data reveal improved sensitivity as compared to culture from these sites. In many circumstances, a careful sexual history and physical examination, review of symptoms, along with the NAATs, can replace routine pelvic examinations. In the teen with suspected PID, the pelvic examination can be limited to the bimanual examination.

Centers for Disease Control and Prevention: Recommendations for the laboratory-based detection of Chlamydia trachomatis and Neisseria gonorrhoeae—2014. MMWR Recomm Rep 2014;63:1-19.

Pickering LK (ed): Red Book: 2012 Report of the Committee on Infectious Diseases, 29th ed. Elk Grove Village, IL, American Academy of Pediatrics, 2012, pp 336-344.

21. What other diagnostic tests should be considered when evaluating an adolescent with a suspected STI?

The following laboratory tests may be helpful in the evaluation of postpubertal females with vaginal discharge/discomfort or suspected PID:

- BV: Gram stain for clue cells, whiff test, vaginal pH
- *Trichomonas vaginalis:* Wet prep (sensitivity, 60-70%), antigen detection (sensitivity, 79-99%)
- Candidiasis (based on clinical symptoms): 10% KOH or Gram stain on vaginal swab for pseudohyphae, culture
- HSV-2 (based on clinical symptoms, examination): Viral culture or PCR taken from unroofed blister
- Urinalysis and urine culture
- Gram stain on blind vaginal swab for white blood cell detection
- Urine β-hCG (affects therapy choices)
- Hepatitis B serologic test (based on clinical symptoms, immunization history, and examination)
- Syphilis serologic test (based on clinical symptoms, examination)
- Human immunodeficiency virus (HIV): Testing in the emergency department (ED) setting is not optimal; refer patient to anonymous testing site, adolescent clinic
- Suspected PID: Complete blood count, C-reactive protein, erythrocyte sedimentation rate

Centers for Disease Control and Prevention: Sexually Transmitted Diseases Treatment Guidelines 2010. MMWR Morb Mortal Wkly Rep 2010;59:RR-12.

Pletcher JR, Slap GB: Pelvic inflammatory disease. In Neinstein LS (ed): Adolescent Health Care: A Practical Guide, 4th ed. Baltimore, Wolters Kluwer/Williams & Wilkins, 2002, pp 1161-1170.

22. What is Fitz-Hugh-Curtis syndrome?

Fitz-Hugh-Curtis syndrome, or perihepatitis, results from salpingitis in 5% to 20% of patients. Inflammatory exudate travels up the paracolic gutter of the abdominal cavity and settles around the liver capsule. The hallmark of this disease is acute right-upper-quadrant pain and tenderness. The patient may also have splinting, anorexia, vomiting, or fever. Other important diagnoses to consider include pneumonia, pulmonary embolus, hepatitis, and gallbladder disease. It is not unusual for the patient to have silent pelvic infection. Liver function test results should be minimally elevated. The treatment regimens for PID are appropriate for these patients.

Pletcher JR, Slap GB: Pelvic inflammatory disease. In Neinstein LS (ed): Adolescent Health Care: A Practical Guide, 4th ed. Baltimore, Wolters Kluwer/Williams & Wilkins, 2002, pp 1161-1170.

23. List the causes of vaginal discharge in the prepubertal girl.

The complaint of vaginal discharge in young girls presenting to the ED is not uncommon. Remember that a very small number of these children are victims of sexual abuse, and that in most a variety of infectious and noninfectious causes are possible. The most common cause is "nonspecific vaginitis," or vaginitis without an identifiable cause. This is attributed to poor hygiene, tight-fitting clothes, soaps, creams, and bubble baths. Pathogens include group

A β-hemolytic streptococcus, *Haemophilus influenzae, Staphylococcus aureus, Moraxella catarrhalis, Streptococcus pneumoniae, Neisseria meningitidis, Shigella* spp., *Yersinia enterolitica*, respiratory pathogens (*S. pneumoniae* and *Streptococcus pyogenes*), and enteric organisms (*Shigella* spp.). STDs can also be associated with vaginitis. The most common cause of bloody vaginal discharge is a foreign body. Other causes to consider include pinworms, trauma, urethral prolapse, atopic dermatitis, lichen sclerosis, scabies, polyps or tumors, and systemic diseases.

Emans SJ: Vulvovaginal problems in the prepubertal child. In Emans SJ, Laufer MR, Goldstein DP (eds): Pediatric and Adolescent Gynecology, 5th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2005, pp 83-119.

Giardino A, Christian C: Vaginal foreign body removal. In King C, Henretig FM, (eds): The Textbook of Pediatric Emergency Procedures, 2nd ed. Philadelphia, Lippincott Williams & Wilkins, 2008, pp 871-874.

24. What treatment is recommended for nonspecific vaginal discharge in a prepubertal girl?

After other causes have been considered, therapy includes daily sitz baths in warm water, avoiding perfumed soaps and bubble baths, and good toilet hygiene. A hair dryer set on a cool temperature can be used to dry the genital area after the bath to help promote healing. Creams with zinc oxide, such as Triple Paste, may be used to protect areas of skin breakdown. Avoid tight-fitting clothes and pajamas.

25. When should you consider vaginal cultures in the prepubertal girl?

Consider a culture/PCR testing on prepubertal patients with vaginal pain or itching, abnormal discharge, genital ulcers or warts, odor, or urinary tract symptoms. The rate of STIs in prepubertal children as a result of sexual abuse is only 1% to 3%. The decision for STI screening in victims of suspected assault/abuse is made based on the history and physical examination findings. Testing is indicated if any of the following are present: (1) vaginal discharge, (2) genital/rectal lesions, (3) genital, oral, or anal trauma, (4) evidence of ejaculation, (5) history suggesting oral, genital, or anal penetration, (6) STI confirmed in another sibling or child in the same home, (7) confirmed STI in the perpetrator, or (8) assault by stranger.

PCR for *N. gonorrhoeae* and *C. trachomatis* can now be used for testing in prepubertal girls on "dirty" urine or specimens obtained using a Dacron swab placed in an appropriate transport medium. A rapid trichomonas antigen test can be used to detect trichomonas, and cultures or PCR can be sent for HSV. Obtain serum for HIV and rapid plasma reagin (RPR) testing, and hepatitis B if not fully immunized.

Centers for Disease Control and Prevention: Sexually Transmitted Diseases Treatment Guidelines 2010. MMWR Morb Mortal Wkly Rep 2010;59:RR-12.

Emans SJ: Vulvovaginal problems in the prepubertal child. In Emans SJ, Laufer MR, Goldstein DP (eds): Pediatric and Adolescent Gynecology, 5th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2005, pp 83-119.

26. What are the symptoms and signs of ectopic pregnancy?

Consider this diagnosis in any postpubertal female with abdominal pain. Classically, patients present with abdominal pain, with or without vaginal bleeding in the face of either missed menses or irregular vaginal bleeding. Other symptoms of pregnancy may also be present. Importantly, remember that 50% of patients are asymptomatic. The differential diagnosis of lower abdominal pain includes PID, spontaneous/threatened abortion, ovarian torsion, ovarian or corpus luteal cyst, endometriosis, appendicitis, and urinary tract infection (UTI). Diagnosis is made by US (usually transvaginal) to determine the location of the gestational sac and a quantitative β-hCG test. A negative urine pregnancy test result rules out the presence of an ectopic pregnancy.

Goyal M, Mollen C, Lavelle J: Adolescent emergencies. In Fleisher GR, Ludwig S, (eds): The Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters-Kluwer/Lippincott Williams & Wilkins, 2010, pp 1634-1649.

27. What is the initial approach to a patient with suspected ectopic pregnancy?

Attention to the ABCs is important in these patients because they are at risk for severe hemorrhage. Immediate gynecologic consultation is mandatory. Follow stable patients with positive results on a β-hCG and a closed cervical os with serial quantitative β-hCG determination and transvaginal US. Several protocols for the care of these patients exist in the literature. Patients with suspected ectopic pregnancy should be evaluated by a gynecologist.

Sowter MC, Farquhar CM: Ectopic pregnancy: An update. Curr Opin Obstet Gynecol 2004;16:289-293.

Goyal M, Mollen C, Lavelle J: Adolescent emergencies. In Fleisher GR, Ludwig S, (eds): The Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters-Kluwer/Lippincott Williams & Wilkins, 2010, pp 1634-1649.

28. What is BV?

BV is the most common cause of vaginitis. This condition results from replacement of the normal hydrogen peroxide–producing *Lactobacillus* spp. with overgrowth of facultative anaerobes, including *Gardnerella vaginalis*, *Prevotella* spp., *Mobiluncus* spp., and *Mycoplasma hominis*. Epidemiologic studies strongly support sexual transmission of this infection, as the condition is more common in women who have had multiple partners and rare in women who are not sexually active. BV is associated with an increased risk for premature labor, premature rupture of membranes, and postpartum or postprocedure endometritis or PID, as well as STI and HIV acquisition. It may also play a role in the development of precancerous cervical lesions. The majority (50-75%) of women with BV are asymptomatic. Symptomatic patients complain of thin, gray vaginal discharge or "fishy"odor. Presence of burning, itching, inflammation, dyspareunia, or dysuria suggests another cause. The clinical diagnosis is made when patients have three of the four following Amsel criteria: (1) presence of a homogeneous, gray-white discharge; (2) vaginal pH higher than 4.5; (3) clue cells present on Gram stain of vaginal fluid (epithelial cells studded with many small bacteria, producing a fuzzy border; offers a "clue" to the diagnosis); and (4) a malodorous, fishy smell before or after exposure to 10% KOH. A Gram stain using Nugent Criteria is the gold standard for diagnosis but is rarely used as it requires time, resources, and expertise. A PAP (Papanicolaou) smear is unreliable, and a culture has no role in making the diagnosis.

29. What is the treatment for BV?

Treat all symptomatic women. Treatment options for nonpregnant patients include metronidazole, 500 mg twice daily for 7 days; tinidazole for 2 days; clindamycin cream 2%, one full applicator once a day at bedtime for 7 days; metronidazole gel 0.75%, one full applicator twice daily for 5 days; or clindamycin, 300 mg twice daily for 7 days. The recurrence rate 1 month after therapy is 30%; accordingly, instruct patients to return if symptoms reappear. Patients with multiple recurrences can be treated with a 4- to 6-month regimen using metronidazole gel twice weekly. The role of probiotics remains under investigation.

The American Academy of Pediatrics: Bacterial vaginosis. In Pickering LK (ed): Red Book 2012 Report of the Committee on Infectious Diseases, 29th ed. Elk Grove Village, IL, American Academy of Pediatrics, 2012, pp 247-249.

30. What are genital ulcers? What causes them?

Genital ulcers are lesions characterized by disruption of the epithelium/mucosa with associated inflammation. Infections associated with ulcers include HSV, syphilis, chancroid (*Haemophilus ducreyi*), and *Lymphogranuloma venereum* (*C. trachomatis* serovars L1-3). In the United States, HSV is by far the most common cause, followed by syphilis and then, rarely, chancroid. HSV causes multiple small vesicles on an erythematous base that are often very painful. Open vesicles appear as shallow ulcers. Syphilis usually causes a single, large, indurated ulcer with a smooth border that is usually painless. Chancroid causes multiple sharply circumscribed, deep ulcers with ragged undermined edges that are painful. Clinical diagnosis is challenging; laboratory testing is useful.

Noninfectious causes of genital ulcers include inflammatory bowel disease, Behçet's disease, fixed drug eruptions, trauma, and neoplasms.

Centers for Disease Control and Prevention: Sexually Transmitted Diseases Treatment Guidelines 2010. Available at http://www.cdc.gov/std/treatment/2010/default.htm.

31. How are genital ulcers caused by HSV treated?

In addition to analgesia and hygiene, antiviral therapy reduces the length of symptoms and recurrence rate. Treat all patients with their first episode of HSV. The Red Book recommends the dose and length of therapy using acyclovir, famcyclovir, and valacyclovir. For patients with recurrent infection, initiate antivirals within 1 day of the onset of lesions for maximal benefit. Provide a prescription with instructions to begin treatment when symptoms and lesions appear. Daily suppression reduces the frequency of recurrence by 70% to 80% and is indicated

in patients with more than six recurrences in 1 year. The risk of drug-resistant HSV infection while on suppressive antiviral therapy is rare. Patients who are immunocompromised, or who have severe or disseminated infection, such as hepatitis, pneumonia, encephalitis, or meningitis, require systemic acyclovir therapy.

Centers for Disease Control and Prevention: Sexually Transmitted Diseases Treatment Guidelines 2010. Available at http://www.cdc.gov/std/treatment/2010/default.htm.

The American Academy of Pediatrics: Bacterial vaginosis. In Pickering LK (ed): Red Book 2012 Report of the Committee on Infectious Diseases, 29th ed. Elk Grove Village, IL, American Academy of Pediatrics, 2012, pp 247-249.

CHAPTER 29

VOMITING

Martha S. Wright

1. What is the pathophysiology of vomiting?

True vomiting is the forceful elimination of gastrointestinal contents through the mouth or nose. Vomiting is caused by coordinated diaphragmatic and abdominal contractions in conjunction with pyloric constriction and gastroesophageal relaxation. This motor activity occurs in response to stimulation of the medullary "vomiting center" by impulses from a variety of anatomic locations. Sources of these impulses include the pelvic and abdominal viscera, the heart, the peritoneum, the labyrinth, and the "chemoreceptor trigger zone," an area on the floor of the fourth ventricle that is sensitive to circulating drugs, toxins, and metabolic derangements.

2. What is the clinical difference between vomiting and "spitting up"?

It is important to differentiate vomiting from "spitting up" because the causes of spitting up are rarely serious but vomiting may indicate a potentially life-threatening condition. Spitting up is characterized by effortless regurgitation of stomach or esophageal contents and in most infants and children is due to gastroesophageal reflux or overfeeding. Vomiting, on the other hand, is forceful, may be accompanied by retching, and is frequently associated with autonomic symptoms, such as salivation, pallor, sweating, tachycardia, and mydriasis.

Key Points: Forceful Vomiting

1. Effortless regurgitation is usually caused by non–life-threatening conditions.
2. Forceful vomiting may be associated with more serious conditions, such as gastrointestinal obstruction, metabolic disease, or toxic ingestions.

3. What is the differential diagnosis of vomiting in the pediatric patient?

Vomiting may be caused by abnormalities in a variety of organ systems. When preschool-aged patients report "vomicking," they help us to remember the wide-ranging differential diagnosis with the following mnemonic:

- **V**: Vestibular: labyrinthine disorders, otitis media
- **O**: Obstruction: malrotation, volvulus, adhesions, intussusception, obstipation, pyloric stenosis, incarcerated hernia, intestinal atresias, annular pancreas, duodenal hematoma
- **M**: Metabolic: diabetic ketoacidosis, inborn errors of metabolism (e.g., urea cycle defects, carbohydrate or amino acid metabolic defects), congenital adrenal hyperplasia (CAH), Reye's syndrome
- **I**: Infection/inflammation: gastrointestinal (appendicitis, hepatitis, pancreatitis, cholecystitis, gastroenteritis, gastritis, necrotizing enterocolitis) or extragastrointestinal (upper respiratory tract infections, sinusitis, pharyngitis, pneumonia, sepsis, cystitis, asthma)
- **C**: Central nervous system disease: increased intracranial pressure (brain tumor, intracranial hematoma, cerebral edema), hydrocephalus, meningitis, pseudotumor cerebri, concussion, migraine, ventriculoperitoneal shunt malfunction
- **K**: Kidney disease: acute kidney injury, chronic kidney disease, pyelonephritis, renal calculi, renal tubular acidosis, obstructive uropathy
- **I**: Intentional: eating disorders, rumination
- **N**: Nasty drugs/poisons: chemotherapeutics, ipecac, iron, salicylates, organophosphates, theophylline, alcohols, lead and other heavy metals, poisonous mushrooms
- **G**: Other GI/GU/GYN (gastrointestinal, genitourinary, gynecologic) causes
  - Gastrointestinal: gastroesophageal reflux, formula intolerance, peptic ulcer disease, cyclic vomiting syndrome
  - Genitourinary: testicular torsion, epididymitis
  - Gynecologic: dysmenorrhea, ovarian torsion, pregnancy, pelvic inflammatory disease)

Parashette KR, Croffie J: Vomiting. Pediatr Rev 2013;34:307-321.

4. The differential diagnosis for vomiting depends on the age of the pediatric patient. What are the life-threatening causes of vomiting in the different pediatric age groups?

The life-threatening causes of vomiting in the different pediatric age groups can be found in Table 29-1.

Kamboj M: Clinical approach to the diagnoses of inborn errors of metabolism. Pediatr Clin North Am 2008;55:1113-1127.

Stevens M, Henretig FM: Vomiting. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 682-683.

5. What are the most common causes of vomiting in the different pediatric age groups?

The most common causes of vomiting in the different pediatric age groups are in Table 29-2.

Parashette KR, Croffie J: Vomiting. Pediatr Rev 2013;34:307-321.

6. What should be the first steps in evaluating the infant or child with vomiting?

In the stable child who does not require resuscitation, evaluation of the infant or child who is vomiting should begin with a careful history and physical examination. There is little value in screening laboratory or radiologic tests in most infants and children who have uncomplicated gastroenteritis. The information gathered will help to identify any acute needs the child may have, quantify the degree of dehydration caused by the vomiting, and allow the clinician to focus the differential diagnosis.

7. What information should be obtained in the history of a child who is vomiting?

Useful historical information includes the following:

- Appearance of the vomitus
- Duration, frequency, and forcefulness of vomiting
- Presence of other gastrointestinal symptoms (e.g., abdominal pain, diarrhea, constipation)
- Presence of other nongastrointestinal symptoms (e.g., headache, neck stiffness, fever, polydipsia/polyphagia/polyuria, dysuria, respiratory symptoms, vaginal discharge, menstrual abnormality, vertigo)

8. What clinical clues can be obtained from the appearance of the vomitus?

When obtaining a history from a patient with vomiting, details about the appearance of the vomitus can help pinpoint the location of the problem (Table 29-3).

Orenstein SR, Peters JM: Vomiting and regurgitation. In Kliegman RM, Greenbaum LA, Lye PS (eds): Practical Strategies in Pediatric Diagnosis and Therapy, 2nd ed. Philadelphia, WB Saunders, 2004, pp 291-321.

Sadow KB, Atabaki SM, Johns CM, et al: Bilious emesis in the pediatric emergency department: Etiology and outcome. Clin Pediatr 2002;41:475-479.

Key Points: Bilious Emesis

1. All infants and children with bilious emesis should be presumed to have a bowel obstruction until proved otherwise.
2. Only 10% to 38% of infants and children evaluated in the emergency department (ED) with yellow-green emesis are found to have a surgical emergency.

9. What information should be obtained during the physical examination of a child with vomiting?

The physical examination should initially focus on adequacy of perfusion and cardiovascular stability. Then, assess the patient's degree of dehydration. Several clinical decision rules (CDRs) have been developed that can be used to determine the degree of dehydration from physical examination findings. One of the easiest to apply demonstrates that the presence of two of four findings (dry mucous membranes, absent tears, abnormal lethargy or restlessness, and capillary refill time longer than 2 seconds) is associated with at least 5% dehydration; the presence of three or more is associated with 5% dehydration or greater. Next, comprehensively examine the abdomen as well as the respiratory, cardiac, and neurologic systems.

Table 29-1. Life-Threatening Causes of Vomiting by Age

AGE	CAUSE
Neonate	GI obstruction Congenital intestinal obstruction Atresias Malrotation with volvulus Renal Obstructive uropathy Uremia Trauma Shaken baby syndrome with subdural hematoma Abdominal trauma Metabolic Inborn metabolic errors Congenital adrenal hyperplasia Infectious Sepsis Meningitis Severe gastroenteritis Necrotizing enterocolitis Neurologic Hydrocephalus
Older infant/toddler	GI obstruction Pyloric stenosis Intussusception Incarcerated hernia Malrotation with volvulus Renal Uremia Trauma Shaken baby syndrome with subdural hematoma Abdominal trauma Infectious Sepsis Meningitis Severe gastroenteritis Neurologic Hydrocephalus Mass lesion Toxic ingestions
Older child/adolescent	GI obstruction Malrotation with volvulus Small bowel obstruction Renal Uremia Infectious Meningitis Metabolic Diabetic ketoacidosis Reye's syndrome Neurologic Intracranial mass lesions (e.g., tumor, hematoma) Toxic ingestions Inflammatory Appendicitis

GI, gastrointestinal.

Table 29-2. Common Causes of Vomiting by Age

AGE	CAUSES
Neonates	GI GE reflux Congenital GI obstruction (intestinal atresias, malrotation) Milk-protein allergy Infectious Sepsis/meningitis
Older infant/toddler	GI GE reflux Gastroenteritis Milk-protein allergy Incarcerated hernia Pyloric stenosis Intussusception Infectious Otitis media Urinary tract infection Toxic ingestion
Older child/adolescent	GI Gastroenteritis Appendicitis Infectious Urinary tract infection Metabolic Diabetic ketoacidosis Toxic ingestion Other Eating disorder

GE, gastroesophageal; GI, gastrointestinal.

Table 29-3. Clues from Appearance of Vomitus

APPEARANCE	SOURCE/CAUSE
Undigested food	Esophageal lesion or reflux
Digested food, milk curds	Stomach, proximal to pylorus
Yellow-green, bilious	Obstruction distal to ampulla of Vater or retrograde peristalsis during retching causing gastroduodenal reflux
Feculent	Distal obstruction, colonic stasis
Blood	Lesion proximal to ligament of Treitz
Bright red blood	Esophagus or stomach above the cardia, minimal contact of blood with gastric secretions
Brown, "coffee grounds"	Gastric bleeding or swallowed blood mixed with gastric secretions
Mucous	Upper respiratory tract, gastric mucous hypersecretion

Gorelick MH, Shaw KN, Murphy KO: Validity and reliability of clinical signs in the diagnosis of dehydration in children. Pediatrics 1997;99:e6.

10. What laboratory tests are indicated in the child with vomiting?

Laboratory testing in the child with vomiting should be guided by the history and physical examination. In children with significant dehydration or those whose initial assessments suggest causes other than uncomplicated gastroenteritis, carefully selected laboratory tests can provide useful clues or confirm diagnoses (Table 29-4).

Colletti JE, Brown KM, Sharieff GQ, et al; ACEP Pediatric Emergency Medicine Committee: The management of children with gastroenteritis and dehydration in the emergency department. J Emerg Med 2010;5:686-698.

Orenstein SR, Peters JM: Vomiting and regurgitation. In Kliegman RM, Greenbaum LA, Lye PS (eds): Practical Strategies in Pediatric Diagnosis and Therapy, 2nd ed. Philadelphia, WB Saunders, 2004, pp 291-321.

11. When are radiographic tests indicated in the pediatric patient with vomiting?

Radiographic tests may help differentiate causes of vomiting that require surgical intervention from those that do not. Plain radiographs of the abdomen are an appropriate initial

Table 29-4. Laboratory Testing in Pediatric Patients with Vomiting

TEST	DIAGNOSTIC UTILITY
Serum electrolytes	Sodium Elevated in hypernatremic dehydration Decreased in hyponatremic dehydration, adrenal insufficiency Potassium Elevated in renal failure, adrenal insufficiency Decreased in pyloric stenosis Chloride Decreased in pyloric stenosis, bulimia Bicarbonate Elevated in significant or chronic vomiting (e.g., bulimia), pyloric stenosis Decreased in inborn metabolic errors, renal tubular acidosis, other causes of metabolic acidosis (sepsis, uremia, toxic ingestions, shock, acute gastroenteritis with dehydration) Glucose Elevated in diabetic ketoacidosis Decreased in inborn metabolic errors, starvation, toxic ingestion
Serum blood urea nitrogen/creatinine	Elevated in dehydration, renal failure
White blood cell count	Elevated in serious bacterial infection
Urinalysis	Glucose with or without ketones: present in diabetes, diabetic ketoacidosis Specific gravity: elevated in dehydration Ketones: elevated in starvation, dehydration, inborn metabolic error Red blood cells: renal calculi, nephritis, UTI White blood cells: UTI
Urine pregnancy test	Pregnancy
Amylase, lipase	Elevated in pancreatitis
Aminotransferases	Elevated in hepatitis

UTI, urinary tract infection.

study in this clinical situation. A plain abdominal film together with an upright film (or cross-table lateral view in the nonambulatory patient) may demonstrate distended bowel loops or air-fluid levels consistent with obstruction. A plain radiograph may also demonstrate abnormal calcifications, such as renal or biliary stones or fecaliths. Free air may be observed on the upright/cross-table film in the case of hollow viscus perforation. Basilar infiltrates caused by lower-lobe pneumonias may be noted serendipitously on an abdominal film, although chest radiography would be the better diagnostic test for pulmonary disease.

12. Which radiographic tests are most useful when further evaluating specific causes of vomiting that may require surgical intervention?

The most useful radiographic tests for evaluating the child with vomiting are listed in Table 29-5.

Pepper VK, Stanfill AB, Pearl RH: Diagnosis and management of pediatric appendicitis, intussusception, and Meckel diverticulum. Surg Clin North Am 2012;3:505-526.

13. What treatment is indicated for the infant or child with vomiting?

Treatment of the infant or child with vomiting is focused first on treating dehydration or maintaining adequate hydration and then on treating the specific cause of the vomiting, when indicated. Dehydration can be treated effectively by either rapid intravenous (IV) rehydration using isotonic crystalloid solution or by appropriately supervised oral rehydration with a suitable rehydration solution. Because most vomiting in children is self-limited or resolves when the underlying cause is treated, antiemetics are not always needed. In children who require ED rehydration for mild to moderate dehydration secondary to an acute gastrointestinal illness associated with vomiting, randomized controlled trials have demonstrated reductions in emesis and the need for hospitalization or IV rehydration after a single dose of the 5-HT_3 (5-hydroxytryptamine) receptor antagonist ondansetron.

Guarino A, Ashkenazi S, Gendrel D, Lo Vecchio A, Shamir R, Szajewska H: European Society for Pediatric Gastroenterology, Hepatology, and Nutrition/European Society for Pediatric Infectious Diseases Evidence-Based Guidelines for the Management of Acute Gastroenteritis in Children in Europe: Update 2014. J Pediatr Gastroenterol Nutr 2014;59:132-152.

Fedorowicz Z, Jagannath VA, Carter B: Antiemetics for reducing vomiting related to acute gastroenteritis in children and adolescents. Cochrane Database Syst Rev 2011;(9):CD005506.

14. A 6-week-old infant presents with vomiting. What are the important historical findings that will help in distinguishing pyloric stenosis from gastroesophageal reflux?

The typical history of an infant with pyloric stenosis is that of nonbilious vomiting beginning around 2 weeks of age that worsens in force and volume over the next several weeks. As

Table 29-5. Radiographic Studies for Evaluating the Child with Vomiting

CLINICAL CONCERN	RADIOGRAPHIC STUDY OF CHOICE
Appendicitis	Abdominal ultrasonography or abdominal CT with or without rectal contrast
Intussusception	Abdominal ultrasonography, contrast enema
Malrotation, intestinal atresias	Upper GI series
Pyloric stenosis	Abdominal ultrasonography or upper GI series
Rental calculi	Abdominal CT without contrast
Ovarian or uterine disease	Pelvic ultrasonography
Pancreatic disease	Abdominal CT with IV and oral contrast
Duodenal hematoma/other intestinal disease	Abdominal CT with IV and oral contrast
Abdominal mass	Abdominal CT with IV and oral contrast

CT, computed tomography; GI, gastrointestinal; IV, intravenous.

the degree of obstruction increases, the vomiting becomes projectile and typically occurs during or soon after feeding. Most commonly seen in first-born infants, pyloric stenosis affects males five times more often than females. By contrast, gastroesophageal reflux typically presents soon after birth and is characterized by effortless, nonprogressive spitting up, frequently occurring with burping or within 30 to 60 minutes after feeding. In most cases, the infant with gastroesophageal reflux will thrive despite the parent's impression that "he's been vomiting everything since birth."

Taylor ND, Cass DT, Holland AJ: Infantile hypertrophic pyloric stenosis: Has anything changed? J Paediatr Child Health 2013;49:33-37.

15. What are the important physical examination and laboratory findings that will help distinguish pyloric stenosis from gastroesophageal reflux?

If the vomiting has progressed significantly, the physical examination in an infant with pyloric stenosis may reveal a fussy, hungry infant who sucks vigorously unless weakened by dehydration. Peristaltic waves may be visible on inspection of the abdomen, and an olive-shaped mass (the hypertrophied pylorus) may be palpable in the subxiphoid region. The classic electrolyte abnormalities noted in pyloric stenosis are *hypochloremia, hypokalemia,* and *metabolic alkalosis*. Increasingly, however, infants are presenting for medical attention earlier in their course, so laboratory and physical findings are likely to be normal. This makes the differentiation of pyloric stenosis from other causes of infantile vomiting challenging and requires the physician to maintain a heightened index of suspicion for this condition.

Tutay GJ, Capraro G, Spirko B, et al: Electrolyte profile of pediatric patients with hypertrophic pyloric stenosis. Pediatr Emerg Care 2013;294:465-468.

Vandenplas Y, Rudolph CD, Di Lorenzo C, et al: North American Society for Pediatric Gastroenterology, Hepatology, and Nutrition: European Society for Pediatric Gastroenterology, Hepatology, and Nutrition. Pediatric gastroesophageal reflux clinical practice guidelines: Joint recommendations of the North American Society for Pediatric Gastroenterology, Hepatology, and Nutrition (NASPGHAN) and the European Society for Pediatric Gastroenterology, Hepatology, and Nutrition (ESPGHAN). J Pediatr Gastroenterol Nutr 2009;49:498-547.

16. Why are babies with pyloric stenosis described as "hungry vomiters"?

Despite projectile vomiting, infants with pyloric stenosis are often demanding to eat soon after emesis. Rarely, they become lethargic, dehydrated, and emaciated if the diagnosis is not made after a significant amount of time.

Key Points: Pyloric Stenosis

1. A high index of suspicion for pyloric stenosis is necessary in young infants who present with vomiting because the characteristic physical examination and laboratory findings may not be present early in the course.
2. Infants with pyloric stenosis always have nonbilious vomiting.

17. A 12-month-old infant presents with vomiting and intermittent abdominal pain. How will you differentiate gastroenteritis from intussusception in this patient?

The classic patient with intussusception is between 3 months and 2 years of age and presents with the triad of episodic cramping abdominal pain, vomiting, and bloody ("currant jelly") stools. The pain typically lasts 5 to 10 minutes and is associated with crouching or drawing legs up, after which the infant may appear well or be lethargic. Many young infants with intussusception are lethargic even after vomiting only once or twice.

A tender mass may be palpated in the right upper quadrant, and in 75% of patients the stool will be positive for occult blood or grossly bloody. Early intussusception may be confused with gastroenteritis, although the latter is most often characterized initially by vomiting that progresses to diarrhea, fever, and nonspecific abdominal pain or cramping pain with defecation.

Applegate KE: Intussusception in children: Evidence-based diagnosis and treatment. Pediatr Radiol 2009;39 (Suppl 2):S140-S143.

Key Points: Intussusception

1. The classic triad of intermittent crampy pain, vomiting, and currant jelly stool is seen in fewer than 25% of children with intussusception.
2. Intussusception should be considered in any infant or toddler with unexplained lethargy.

18. A 2-week-old baby presents with significant vomiting and hypotension. Blood studies show hyponatremia, hyperkalemia, and a severe metabolic acidosis. What condition is most likely?

This infant likely has CAH due to 21-hydroxylase deficiency. The electrolyte picture is classic, and ambiguous genitalia may be another clue with such infants.

19. An 8-year-old boy presents with headache and vomiting. How will you distinguish vomiting caused by an intracranial mass lesion from that associated with migraine headaches?

After headache, vomiting is the second most commonly noted symptom in children with intracranial mass lesions. However, vomiting is not likely to be the only abnormality. In more than 90% of children with vomiting from an intracranial mass, other neurologic or ocular abnormalities will be present. Vomiting that accompanies brain tumors is commonly seen in the mornings, is usually effortless (although it may become projectile), and is not particularly associated with meals or abdominal pain. The vomiting may persist intermittently for weeks. Vomiting associated with migraine headaches is typically associated with infrequent, severe, diffuse headaches that resolve with sleep or when the cephalgia is treated.

Squires RH: Intracranial tumors. Vomiting as a presenting sign. A gastroenterologist's perspective. Clin Pediatr 1989;28:351-354.

Wilne SH, Ferris RC, Nathwani A, Kennedy CR: The presenting features of brain tumours: A review of 200 cases. Arch Dis Child 2006;91:502-506.

ANAPHYLAXIS

Linda D. Arnold

1. What is anaphylaxis?

Anaphylaxis is an acute, systemic allergic reaction that can be mild, life threatening, or fatal. Signs and symptoms develop rapidly as mast cells and basophils release potent biologically active mediators and clotting and complement cascades are recruited. Multiple organs may be affected, producing an acute constellation of signs and symptoms involving the skin, mucosa, respiratory and gastrointestinal (GI) tracts, and cardiovascular system. There is no universally accepted definition of anaphylaxis, and proposed clinical criteria are confusing even for medical professionals—especially when making treatment decisions during an acute reaction.

2. How common is anaphylaxis?

The overall prevalence of anaphylaxis is rising and may be as high as 2%, with food allergies as the leading cause. In the United States, 8% of children have food allergies and 1% to 2% are allergic to peanuts, tree nuts, or both. Serious systemic reactions to hymenoptera stings occur in up to 1% of children and 3% of adults.

Ellis AK, Day JH: Clinical reactivity to insect stings. Curr Opin Allergy Clin Immunol 2005;5:349-354.

Gupta RS, Springston EE, Warrier MR, et al: The prevalence, severity, and distribution of childhood food allergy in the United States. Pediatrics 2011;128(1):e9-e17.

3. What makes anaphylaxis so dangerous?

Anaphylaxis usually occurs outside the hospital setting, with case fatality rates approaching 1%. In fatal reactions, the median time to respiratory or cardiac arrest is 5 minutes for reactions to medications or contrast material, 15 minutes for insect venom, and 30 minutes for food. These are sobering statistics, given the huge percentage of patients with severe allergies who seldom carry their epinephrine autoinjectors. Fatal reactions are frequently the first reactions for sting, medication, and contrast agent deaths, and anaphylaxis occurs with initial exposure to a food 25% of the time. The majority of allergic reactions in children take place in school or day care. Most schools lack full-time nurses, do not allow children to carry their own autoinjectable epinephrine, and often fail to adhere to written emergency management plans.

Young MC, Munoz-Furlong A, Sicherer SH: Management of food allergies in schools: A perspective for allergists. J Allergy Clin Immunol 2009;124(2):175-182.

4. What are common causes of anaphylaxis?

Anaphylaxis in young children is mostly triggered by ingestion of cow milk, egg, or peanuts, though tree nuts, seafood, soy, wheat, and sesame can also cause severe reactions. The majority of reactions in teens and adults are due to peanuts, tree nuts, or seafood. In patients with known food allergies, anaphylaxis is often triggered by unknown ingredients or cross-contamination of foods believed to be safe. Peanuts and tree nuts are responsible for 90% of all food anaphylactic events, the majority of which occur in the setting of commercial catering. Among antibiotics, penicillin is the most common cause of anaphylaxis; contrary to common belief, cross-reactivity with cephalosporins is low. Aspirin and nonsteroidal anti-inflammatory drugs (NSAIDs) are the second most common cause of drug-induced anaphylaxis. Insect stings and allergen immunotherapy are also important causes of anaphylaxis.

5. Who is at the greatest risk for severe anaphylactic reactions?

Most deaths from anaphylaxis due to food occur in adolescents and young adults, who are more apt to take risks and less likely to carry their epinephrine autoinjectors. Patients with a history of previous anaphylaxis or poorly controlled asthma are at greater risk. Reactions to peanuts, tree nuts, fish, and shellfish are the most severe, with peanuts and tree nuts responsible for 94% of

food-related deaths. Failure or delays in administration of epinephrine are associated with near-fatal and fatal reactions. Many severe reactions to foods and insect venom occur in children who have not required previous urgent medical intervention and thus lack access to epinephrine. Although large local reactions to insect venom are not predictive of anaphylaxis, 10% of children with generalized urticaria following a sting will develop severe reactions in the future.

Key Points: Risk Factors for Severe Anaphylaxis

1. Adolescent or young adult
2. History of previous severe reaction
3. Peanuts, tree nuts, fish, or shellfish as inciting agent
4. Asthma, especially if poorly controlled
5. Failure to administer epinephrine promptly
6. Taking beta blockers

6. Which medications/interventions can trigger anaphylaxis in children?

- Neuromuscular blockers (succinylcholine, vecuronium, atracurium) account for 60% of episodes of anaphylaxis related to medical treatment. Airway obstruction and cardiovascular collapse may be mistaken for the effects of sedatives and anesthetic agents in this setting.
- Latex, antibiotics, induction agents (etomidate, propofol, thiopental), and narcotics (fentanyl, meperidine, morphine) are also important causes.
- Colloids, opioids, radiocontrast media, and blood products are implicated less than 10% of the time.
- All rabies vaccines, except Imovax, contain egg protein. Children with a history of systemic reaction to egg protein should undergo an allergy evaluation and vaccine testing prior to rabies vaccine administration.

Centers for Disease Control and Prevention: Human rabies prevention—United States. Morb Mortal Wkly Rep MMWR 2008;57:1-26,28.

7. What are the clinical manifestations of anaphylaxis?

Anaphylaxis is characterized by the abrupt onset of symptoms minutes to hours after an ingestion or exposure (Table 30-1). The timing, sequence, and severity of symptoms vary.

- The oral cavity and throat are affected first; a tingling or pruritic sensation of the oral mucosa may develop, often followed by edema of the lips, uvula, larynx, and epiglottis.
- GI signs are common in children and include nausea, vomiting, and colicky pain.
- Skin manifestations include flushing, pruritus, and localized or diffuse urticaria.
- Respiratory symptoms, such as stridor or wheezing, may develop in more severe reactions.
- Cardiovascular effects include tachycardia and hypotension, which may cause dizziness and altered mental status.

8. How can I recognize severe reactions?

Onset of symptoms is generally rapid, with early signs of respiratory insufficiency and hemodynamic compromise. Cutaneous findings may be absent. Severe bronchospasm and

Table 30-1. Clinical Signs and Symptoms of Anaphylaxis

Oropharyngeal: Metallic taste, pruritus, edema of lips, tongue, palate, or uvula
Upper airway: Congestion, rhinorrhea, pruritus, sneezing, throat tightness, hoarseness, dysphagia
Dermatologic: Erythema, pruritus, urticaria, angioedema, morbilliform rash
Gastrointestinal: Nausea, colicky abdominal pain, vomiting, diarrhea
Respiratory: Cough, shortness of breath, dyspnea, chest tightness, stridor, wheezing
Cardiovascular: Faintness, tachycardia, syncope, chest pain, hypotension
Neurologic: Headache, mental status changes
General: Anxiety, sense of impending doom

upper airway swelling can both lead to asphyxia. Increased vascular permeability results in rapid shifts of intravascular fluid into the extravascular space. Concomitant vasodilation leads to a mixed distributive/hypovolemic shock, which may be refractory to aggressive fluid resuscitation and pressor support, particularly in patients taking beta blockers. Neurologic changes are ominous, signaling severe hypotension. Myocardial ischemia, conduction defects, T-wave abnormalities, and atrial and ventricular arrhythmias may result from direct mediator effects on the myocardium. Most deaths are caused by respiratory arrest or cardiovascular collapse.

9. Which specific questions help confirm the diagnosis?

Confirm the diagnosis by a careful history that focuses on the nature and timing of known exposures, onset and description of symptoms, and response to medications. Ask about cutaneous, mucosal, and GI manifestations and signs of upper or lower airway obstruction and hypotension. When the cause of anaphylaxis is unknown, ask about all foods and medications ingested in the hours preceding the reaction. Prepared or packaged foods safely consumed in the past may have undergone changes in ingredients or been subject to cross-contamination. Questions about preceding exercise (exercise-induced anaphylaxis) and sexual activity (latex or seminal plasma allergies; transfer of food or drug allergens by partner) may also yield important clues.

10. What conditions can mimic anaphylaxis?

- **Vasovagal/neurocardiogenic syncope:** Bradycardia and pallor, but no hives or bronchospasm
- **Scombroid poisoning:** Urticaria, nausea, headache, dizziness within a half hour of eating spoiled fish
- **Physical urticaria:** Skin manifestations only (e.g., cold urticaria, cholinergic urticaria)
- **Severe asthma exacerbations:** Bronchospasm and stridor, but no skin findings or GI symptoms
- **Angioedema (hereditary form):** Difficult to distinguish from early anaphylaxis
- **Panic disorder or vocal cord dysfunction (VCD)**: Can present with functional stridor, but none of the other symptoms
- **Oral allergy syndrome (OAS):** Reaction limited to lips and oropharynx

11. What is OAS?

OAS is a localized IgE-mediated response that follows ingestion of certain fruits and vegetables, mostly in patients allergic to pollens. Although physical symptoms are limited to itching or tingling of the lips, tongue, roof of mouth, and throat, with or without angioedema, children with a history of anaphylaxis may also exhibit significant anxiety. A careful history and examination helps distinguish between OAS and early signs of a systemic reaction.

12. How should anaphylaxis be managed?

See Table 30-2. The extent and severity of symptoms should be rapidly and frequently assessed, with a focus on airway, oxygenation, cardiac output, circulation, and tissue perfusion.

Table 30-2. Acute Management of Anaphylaxis

Rapid and frequent assessment of ABCs
Patient placed in supine position, with legs elevated
Supplemental oxygen and airway management
Epinephrine IM or IV, as indicated
IV fluids for hypotension
Albuterol for bronchospasm persisting after IM epinephrine
H_1 and H_2 antagonists considered
IV or PO steroids considered
Monitored for a minimum of 4-6 hours

ABCs, airway, breathing, and circulation; IM, intramuscular; IV, intravenous; PO, per os (taken orally).

Administer epinephrine promptly and repeat as necessary. Place the patient supine, with legs elevated, to improve venous return and help maintain adequate blood pressure. When hypotension is present, aggressively support the circulation with intravenous (IV) crystalloids. Provide oxygen and albuterol for wheezing that persists after epinephrine. Antihistamines and steroids have no effect on symptom progression and should *never* be given in lieu of epinephrine or delay its administration.

13. What are the most common errors in the management of anaphylaxis?

Anaphylaxis continues to be underrecognized and undertreated in both prehospital and emergency department (ED) settings. Failure to administer epinephrine, delayed administration, and errors in dosing and route of administration are common among patients, caregivers, school health and EMS (emergency medical service) personnel, and ED providers. Hesitancy or failure to administer epinephrine by medical personnel, or delays until signs and symptoms are severe, not only sends the wrong message to patients and caregivers but is associated with increased morbidity and higher fatality rates.

14. When should epinephrine be given?

When it comes to anaphylaxis, teach patients and practitioners to err on the side of injecting epinephrine. As the saying goes: If you're thinking about it: Do it! Serious adverse effects from epinephrine are rare in children receiving appropriate doses, and the transient pallor and palpitations many experience "pale" in comparison to the symptoms of anaphylaxis itself and the potential consequences of delays in treatment.

Epinephrine is clearly indicated for allergic reactions with signs of respiratory distress or cardiovascular instability, or when hypotension develops following exposure to a known allergen. Epinephrine is also recommended for children exposed to allergens that previously caused anaphylaxis, even when initial symptoms are mild. Although generalized urticaria alone is not life threatening, it should prompt epinephrine administration in children at risk for severe reactions, including those with a history of anaphylaxis, asthma, or severe atopy, or when the inciting agent is peanuts, tree nuts, shellfish, milk, or insect venom. A good rule of thumb in all other situations is to give intramuscular (IM) epinephrine for acute systemic reactions involving two or more organ systems or any of the following severe symptoms: significant swelling of the lips, tongue, or uvula; throat tightness or trouble breathing or swallowing; persistent cough or respiratory compromise (dyspnea, bronchospasm, stridor, hypoxemia); persistent vomiting; cardiac effects (pallor, tachycardia, dizziness, syncope); or anxiety, confusion, or sense of impending doom.

15. How does epinephrine work in anaphylactic reactions?

Epinephrine is a direct-acting sympathomimetic with complex effects on many organs. Vasoconstriction, the primary α-adrenergic effect, reverses peripheral vasodilation and increases peripheral vascular resistance. Its β-adrenergic effects include downregulation of further release of inflammatory mediators, bronchodilation via smooth muscle relaxation, and increased heart rate and contractility via direct effects on the myocardium. Clinically, these effects decrease mucosal edema and cutaneous signs of angioedema and urticaria. More importantly, they lead to improvements in cardiovascular function by increasing blood pressure and enhancing coronary blood flow.

Key Points: Epinephrine

1. Most important medication in the treatment of anaphylaxis
2. Prevents further mediator release
3. Decreases upper airway edema
4. Reverses bronchospasm by relaxing bronchial smooth muscle
5. Increases blood pressure via peripheral vasoconstriction
6. Increases heart rate and contractility
7. Delays in administration associated with higher fatality rates

16. How should epinephrine be administered?

Administer epinephrine by IM injection at a dose of 0.01 mg/kg body weight, given as 0.01 mL/kg of the 1:1000 solution (maximum dose, 0.5 mg = 0.5 mL). Repeat doses every 5 minutes,

as needed. The anterolateral thigh is the preferred injection site because of superior vascularity; plasma levels peak in 8 minutes, on average. Do not give epinephrine subcutaneously, as slow absorption by this route leads to extreme delays in peak plasma concentration. Longer needles may thus be necessary for obese children. IV epinephrine may be required for refractory anaphylaxis, but dilution and dosing errors are common. Infused epinephrine should be diluted, administered slowly, and titrated to effect in order to minimize the risk of adverse cardiac effects.

17. When should bronchodilators be used in anaphylaxis?

Use oxygen and intermittent or continuous aerosolized β-adrenergic agents (e.g., albuterol) when wheezing fails to resolve after epinephrine administration.

18. Are H_1 and H_2 blockers effective in treating anaphylaxis?

Even when H_1 antihistamines are given in combination with H_2 blockers, such as ranitidine, they are considered to be second-line agents and should never be given in place of epinephrine. Though histamine is an important mediator in anaphylaxis, levels peak early and transiently. Antihistamines may provide symptomatic relief in limited cutaneous reactions, but they do not reduce the release of mediators, halt the progression of symptoms, or treat airway edema or hypotension.

19. Do steroids have a role in treatment?

Steroids have no role in the acute management of anaphylaxis but may help to modulate late-phase responses. Despite the lack of supporting evidence, many experts recommend giving prednisone (1-2 mg/kg orally) for mild to moderate reactions and methylprednisolone (2 mg/kg via IV route) for severe allergic reactions, and oral steroids are often prescribed for 48 to 72 hours. Table 30-3 summarizes the drugs used to treat anaphylaxis.

20. What are biphasic anaphylactic reactions?

Classically, anaphylaxis is monophasic, with complete resolution within 2 hours of treatment. Biphasic reactions occur in 6% to 20% of patients, whose symptoms recur 2 to 72 hours after initial resolution, without a new exposure. Management of biphasic reactions may be complicated by a poor response to epinephrine and by severe bronchospasm refractory to β-agonists. Prompt administration of epinephrine during the initial episode is associated with decreased occurrence of biphasic reactions, but steroids have not been shown to have any effect.

21. How long should patients be monitored in the ED?

Observe children presenting with systemic allergic reactions for at least 4 to 6 hours for progression or recurrence of symptoms. Those who have had a mild reaction, responded quickly

Table 30-3. Medications Used to Treat Anaphylaxis

MEDICATION	DOSAGE
Epinephrine	0.01 mg/kg (1:1000) IM in anterolateral thigh (max 0.5 mg); repeat q 5 min as needed; 0.01-1.0 μg/kg/min (1:10,000) IV infusion for refractory hypotension
Albuterol	Intermittent or continuous nebulized solution for bronchospasm
Diphenhydramine	1 mg/kg PO, IM, or IV (max 75 mg)
Ranitidine	1-2 mg/kg PO or IV (max 75 mg)
Prednisone	1-2 mg/kg PO (max 75 mg)
Methylprednisolone	1-2 mg/kg IV (max 125 mg)
Glucagon	20-30 μg/kg or 5-15 μg/min IV infusion for refractory hypotension
Dopamine	2-20 μg/kg/min IV infusion for refractory hypotension
Atropine	Titrate to effect for bradycardia, asystole, or PEA

IM, intramuscular; IV, intravenous; PEA, pulseless electrical activity; PO, per os (taken orally); q, every.

Figure 30-1. Two frequently used autoinjectors and their relative size. *Directions for use of epinephrine autoinjectors*: EpiPen /EpiPen Jr. (1) Unscrew yellow or green cap and remove autoinjector from case. (2) Form a fist around the unit, with orange tip pointing downward. (3) Pull off the blue safety cap with other hand. (4) Firmly jab unit, at a 90-degree angle, into anterolateral thigh. (5) After a click is heard, hold firmly in place for 10 seconds. (6) Remove unit and massage area for 10 seconds. (7) Call 911 and seek immediate medical attention. The Auvi-Q autoinjector automatically activates step-by-step voice instructions for administration.

to therapy, and can be closely monitored at home can then be discharged with detailed instructions about signs, symptoms, and treatment of a biphasic reaction. Admit to the hospital, or an extended-stay unit, those children with severe reactions or a history of severe reactions, those who live far from a medical facility or cannot return promptly if symptoms recur, and those who are unable to fill a prescription for autoinjectable epinephrine prior to discharge.

22. Who should be discharged with epinephrine autoinjectors?

Prescribe autoinjectable epinephrine (Fig. 30-1) for all children meeting criteria for anaphylaxis, for those with generalized acute urticaria after an insect sting, and for systemic reactions in patients with a history of wheezing, a personal or family history of a severe reaction, or an allergy to peanuts, tree nuts, fish, shellfish, or milk. Teach caregivers when and how to administer IM epinephrine prior to discharge, using a trainer device. Epipen autoinjectors are most commonly prescribed, though the recently launched Auvi-Q autoinjectors are much smaller and provide step-by-step voice instructions for administration. The threshold for epinephrine administration should be lower if nonmedical people are with the child; if the reaction is from peanuts, nuts, or seafood; or when the reaction occurs in a location that is more than 15 minutes from a medical facility. After epinephrine administration, emergency transport to a hospital is imperative.

Sicherer SH, Simons ER, Section on Allergy and Immunology: Self-injectable epinephrine for first-aid management of anaphylaxis. Pediatrics 2007;119:638-646.

23. How is autoinjectable epinephrine dosed?

Autoinjectable epinephrine comes in just two unit doses. The 0.15-mg dose is ideal for a 15-kg child and is generally prescribed for children weighing between 10 and 25 kg. The 0.3-mg units are intended for children who weigh 30 kg or more. For those in between, assess the dosage based on the assessed risk of a severe reaction, taking history, specific inciting agents, and comorbid conditions into account. For children under 10 kg, there are two imperfect options. The 0.15 mg autoinjector will provide a fixed dose of epinephrine that exceeds 0.01 mg/kg. Parents may also be taught to draw up and administer a weight-based dose of epinephrine,

though this method is highly prone to errors. Anticipatory guidance about allergen avoidance is particularly important in this group.

24. What other discharge instructions should be given?

All patients must be discharged with a friend or family member who has been instructed to watch for signs and symptoms of a biphasic reaction. Include a written emergency action plan, to be accessible at all times, that details symptoms of anaphylaxis, medications prescribed, and indications for their use. Emphasize the importance of having autoinjectors available at all times, protecting them from direct sunlight or extreme temperatures, and promptly replacing expired medication. Provide education on allergen avoidance, particularly with respect to hidden ingredients in commercially prepared or catered foods and cross-contamination of food during factory processing and preparation in restaurants. Educational resources are very helpful in this regard. Make a referral to an allergist or strongly recommend this to the family.

Boyce JA, Assa'ad A, Burks AW, et al: Guidelines for the diagnosis and management of food allergy in the United States: Report of the NIAID-sponsored expert panel. J Allergy Clin Immunol 2010;126:S1-S58.

Key Points: Discharge Instructions Following Anaphylaxis

1. Instruct a family member to watch for signs of a biphasic reaction.
2. Outline a straightforward management plan for future reactions, including names, doses, and indications for medications (www.foodallergy.org/document.doc?id=234).
3. Provide education and links to resources on allergen avoidance.
4. Prescribe autoinjectable epinephrine for patients at risk of a severe reaction; demonstrate its usage with a trainer device; stress importance of carrying it at all times.
5. Make sure other caretakers (grandparents and childcare providers) have autoinjectable epinephrine when they are caring for the child.
6. Recommend timely follow-up with primary care provider (PCP); consider referral to an allergist.

Websites

Food Allergy Research & Education (FARE). www.foodallergy.org.
The National Agricultural Library: Allergies. www.nal.usda.gov/fnichttp://fnic.nal.usda.gov.

CHAPTER 31

CARDIAC EMERGENCIES

Alexandra A. Taylor and Susan M. Kelly

1. How do the pediatric advanced life support (PALS) guidelines differ from previous recommendations regarding the initial assessment of an unresponsive child?

The 2010 guidelines advise:

- Take no more than 10 seconds to feel for a pulse.
- If no pulse is felt within 10 seconds, start chest compressions. The new mnemonic is C-A-B (circulation, airway, breathing) rather than A-B-C.
- When assessing the airway, use a head-tilt, chin-lift maneuver. Observe for respiratory effort; "*look, listen, feel*" is no longer recommended as part of the initial assessment.

Hafeez W, Ronca L, Maldonado T: Pediatric advanced life support for the emergency physician: Review of 2010 guideline changes. Clin Pediatr Emerg Med 2011;12(4):255-265.

2. What are the revised PALS guidelines for airway management in an unresponsive child?

- A cuffed endotracheal tube (ETT) can be used for children of all ages.
- Cricoid pressure is not recommended for endotracheal intubation.
- ETT placement should be confirmed with capnography.
- The respiratory rate for intubated children is 8 to 10 breaths per minute, regardless of age.

Hafeez W, Ronca L, Maldonado T: Pediatric advanced life support for the emergency physician: Review of 2010 guideline changes. Clin Pediatr Emerg Med 2011;12(4):255-265.

3. List the underlying causes of pulseless electrical activity (6H's and 4T's):

- Hypoxemia
- Hypovolemia
- Hydrogen ion (acidosis)
- Hypoglycemia
- Hypothermia
- Hypokalemia/hyperkalemia
- Tension pneumothorax
- Tamponade (pericardial)
- Thrombosis (pulmonary/coronary)
- Toxin

Hafeez W, Ronca L, Maldonado T: Pediatric advanced life support for the emergency physician: Review of 2010 guideline changes. Clin Pediatr Emerg Med 2011;12(4):255-265.

Kattwinkel J, Perlman JM, Aziz K, et al: Part 15: Neonatal resuscitation: 2010 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. Circulation 2010;122: S909-S919.

4. How is bradycardia defined?

Based on the PALS and Neonatal Resuscitation Program guidelines, bradycardia is defined as a heart rate of less than 60 beats per minute.

Hafeez W, Ronca L, Maldonado T: Pediatric advanced life support for the emergency physician: Review of 2010 guideline changes. Clin Pediatr Emerg Med 2011;12(4):255-265.

Kattwinkel J, Perlman JM, Aziz K, et al: Part 15: Neonatal resuscitation: 2010 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. Circulation 2010;122: S909-S919.

5. What are the indications for emergency or urgent intervention in a patient with bradycardia?

PALS differentiates symptomatic from asymptomatic bradycardia. Symptomatic bradycardia requires emergent intervention; symptomatic bradycardia is defined as a heart rate of less than

60 beats per minute along with one of the following: poor pulses, inadequate perfusion (hypotension, delayed capillary refill), or abnormal respirations.

Hafeez W, Ronca L, Maldonado T: Pediatric advanced life support for the emergency physician: Review of 2010 guideline changes. Clin Pediatr Emerg Med 2011;12(4):255-265.

6. What are the initial steps in treating a patient with symptomatic bradycardia?

- Initiate chest compressions immediately and support the airway.
- Administer 0.01 mg/kg 1:10,000 epinephrine via intravenous/intraosseous (IV/IO) route (0.1 mL/kg of 1:10,000 solution). Repeat epinephrine every 3 to 5 minutes.
- If increased vagal tone or primary atrioventricular (AV) block is suspected as the cause of bradycardia, administer atropine 0.02 mg/kg (minimum dose 0.1 mg; maximum dose 0.5 mg for a child and 1 mg for an adolescent).
- If complete heart block is discovered on electrocardiogram (ECG) or if there is a history of congenital or acquired heart disease, consider emergent pacing.

Hafeez W, Ronca L, Maldonado T: Pediatric advanced life support for the emergency physician: Review of 2010 guideline changes. Clin Pediatr Emerg Med 2011;12(4):255-265.

7. What is a normal QTc? What are the symptoms associated with long QT syndrome?

In children, the maximum normal corrected QT interval (QTc) is 440 ms. Long QT syndrome is associated with syncope, seizure, or cardiac arrest preceded by emotion or exercise.

Moss AJ, Long QT syndrome. JAMA 2003;289:2041.

8. List the ECG findings associated with long QT syndrome.

- Intermittent T-wave inversions (T-wave alternans)
- Torsades de pointes
- AV block
- Premature ventricular contractions

Moss AJ, Long QT syndrome. JAMA 2003;289:2041.

9. What history in a patient presenting with sudden and unexpected syncope would make you consider congenital long QT syndrome?

- An episode of syncope precipitated by sudden stress or startle
- History of sudden unexplained death in an immediate family member under the age of 30
- Family history of long QT syndrome or familial deafness

Moss AJ, Long QT syndrome. JAMA 2003;289:2041.

10. What is the first-line treatment for patients with known congenital long QT syndrome?

First-line treatment is a beta blocker, such as propranolol (2-4 mg/kg/day). Treatment should be instituted regardless of the presence or absence of symptoms.

Moss AJ, Long QT syndrome. JAMA 2003;289:2041.

11. List several reasons for an abnormally long QTc interval that is not due to congenital long QT syndrome.

- Electrolyte disturbances such as hypomagnesemia, hypocalcemia, or hypokalemia
- Central nervous system insult (increased intracranial pressure, hypoxia)
- Anorexia nervosa or liquid protein diets
- Many drug ingestions

Key Points: Long QT Syndrome

1. Normal QTc is less than 440 ms.
2. ECG changes include intermittent T-wave inversions, torsades de pointes, low heart rate.
3. First-line treatment is the use of beta blockers.

12. What is the most common arrhythmia in childhood?

Supraventricular tachycardia (SVT).

Sacchetti A, Moyer V, Baricella R, et al: Primary cardiac arrhythmias in children. Pediatr Emerg Care 1999;15(2):95-98.

13. What is the usual heart rate for infants and children with SVT?

- Infant: heart rate faster than 220 beats per minute
- Child: heart rate faster than 180 beats per minute

Hafeez W, Ronca L, Maldonado T: Pediatric advanced life support for the emergency physician: Review of 2010 guideline changes. Clin Pediatr Emerg Med 2011;12(4):255-265.

14. What is the most likely presentation for children with SVT?

Older children may report chest pain, palpitations, or dizziness. Infants may present with tachypnea, irritability or lethargy, poor perfusion, poor feeding, and other signs of congestive heart failure.

15. What is the treatment of SVT?

- SVT with hemodynamic compromise: Treat with cardioversion, 0.5 to 1 J/kg.
- SVT without hemodynamic compromise:
  - Establish IV access.
  - Administer supplemental oxygen.
  - Attempt vagal stimulation: For infants and children, apply ice to the face. Older children may be coached to perform a Valsalva maneuver.
  - If vagal maneuvers are ineffective, administer IV adenosine 0.1 mg/kg (maximum dose 6 mg). Use a three-way stopcock, immediately flushing with 5 to 10 mL normal saline (NS) after pushing adenosine rapidly. If the initial dose is ineffective, administer a second dose (0.2 mg/kg adenosine, maximum dose 12 mg).

Hafeez W, Ronca L, Maldonado T: Pediatric advanced life support for the emergency physician: Review of 2010 guideline changes. Clin Pediatr Emerg Med 2011;12(4):255-265.

16. What is the definition of wide complex tachycardia?

QRS complex longer than 0.09 second.

Hafeez W, Ronca L, Maldonado T: Pediatric advanced life support for the emergency physician: Review of 2010 guideline changes. Clin Pediatr Emerg Med 2011;12(4):255-265.

17. Give the initial rhythm differential diagnosis for patients presenting with sustained wide complex tachycardia.

Consider ventricular tachycardia (VT) first in a patient with wide complex tachycardia. Other causes include SVT with bundle branch block or SVT with aberrant conduction.

18. Describe the initial assessment and treatment of a patient presenting with sustained wide complex tachycardia.

- Pulse with wide complex tachycardia and hemodynamic instability: Perform synchronized cardioversion with 0.5 to 2 J/kg.
- Pulse with wide complex tachycardia *without* hemodynamic instability:
  - Establish IV access.
  - Provide supplemental O_2.
  - Administer amiodarone 5 mg/kg (maximum dose 150 mg) over 20 to 60 minutes. Alternatively, administer lidocaine 1 mg/kg (maximum dose 100 mg) as a rapid bolus.
- Pulseless patient with wide complex tachycardia on the monitor:
  - Initiate cardiopulmonary resuscitation (CPR).
  - Defibrillate at 2 J/kg (maximum 200 J); if no response, defibrillate at 4 J/kg.
  - Administer epinephrine (1:10,000 solution) 0.01 mg/kg IV/IO route every 3 to 5 minutes.
  - Consider using amiodarone or lidocaine.
  - Consider torsades de pointes; administer magnesium 25 to 50 mg/kg (maximum 2 g).

Hafeez W, Ronca L, Maldonado T: Pediatric advanced life support for the emergency physician: Review of 2010 guideline changes. Clin Pediatr Emerg Med 2011;12(4):255-265.

19. Define heart failure for pediatric patients.

Heart failure occurs in children when excessive preload, excessive afterload, arrhythmias, or decreased contractility results in inadequate cardiac output to meet the metabolic demands of the body.

Madriago E, Silberbach M: Heart failure in infants and children. Pediatr Rev 2010;13(1):4-12.

20. What are the signs and symptoms of heart failure in children?

Children may have tachycardia, tachypnea, and dyspnea. Infants may have poor feeding, easy fatigability, and failure to thrive. Older children may be sleepy and have poor exercise tolerance and anorexia. On physical examination, grunting, S_3/S_4 gallop, hepatomegaly, crackles/rales, wheezing peripheral edema, and jugular venous distention may be present.

Madriago E, Silberbach M: Heart failure in infants and children. Pediatr Rev 2010;13(1):4-12.

21. What are the most likely causes of heart failure in neonates presenting to the emergency department (ED)?

Structural congenital heart disease is the most likely cause of heart failure in the neonate. In the first week of life, transposition of the great vessels or total anomalous pulmonary venous return is the most likely cause. Between weeks 1 and 4, critical aortic stenosis or pulmonary stenosis or preductal coarctation of the aorta is the most likely cause.

Madriago E, Silberbach M: Heart failure in infants and children. Pediatr Rev 2010;13(1):4-12.

22. What are the treatment options for pediatric patients with heart failure?

The initial goals of management are to recognize and treat underlying causes, including cardiac structural abnormalities. Additional medical management is aimed at reducing preload (diuretics), reducing afterload (ACEIs [angiotensin-converting enzyme inhibitors], ARBs [angiotensin receptor blockers], milrinone, nitrates), sympathetic inhibition (beta blockers), and inotropy (digoxin). Patients failing medical management will ultimately require heart transplant.

Madriago E, Silberbach M: Heart failure in infants and children. Pediatr Rev 2010;13(1):4-12.

Key Points: Heart Failure

1. Heart failure is defined as cardiac output that does not meet the metabolic demands of the body.
2. Infants may present with poor feeding, failure to thrive, and grunting.
3. Children may present with tachycardia, tachypnea, and dyspnea.
4. Structural heart disease is the most likely cause of heart failure in neonates.

23. List the ECG findings of patients who have received overdoses of the following medications.

- **Beta blockers:** Bradycardia, increased PR interval
- **Calcium channel blockers:** Bradycardia, prolonged AV node conduction or block
- **Tricyclic antidepressants:** Tachycardia, prolonged QRS complex, decreased AV conduction time, VT, ventricular fibrillation, torsades de pointes
- **Phenothiazines:** QTc prolongation, torsades de pointes
- **Type 1A antiarrhythmic medications (quinidine, procainamide, disopyramide):** Prolongation of QT interval, torsades de pointes, VT
- **Amiodarone:** AV block, sinus node dysfunction (marked bradycardia), torsades de pointes

Clancy C: Electrocardiographic evaluation of the poisoned or overdosed patient. In Goldfrank LR, Flomenbaum NE, Lewin NA, et al (eds): Goldfrank's Toxicologic Emergencies, 6th ed. Stamford, CT, Appleton & Lange, 2002, pp 105-128.

24. Which chemotherapeutic agents cause cardiotoxicity?

- Anthracyclines (doxorubicin, daunorubicin) cause left ventricular dysfunction, which may lead to overt heart failure.
- Bevacizumab causes hypertension and endothelial damage.
- Imatinib causes QTc prolongation.

Colombo A, Meroni CA, Cipolla CM, Cardinale D: Managing cardiotoxicity of chemotherapy. Curr Treat Options Cardiovasc Med 2013;15(4):410-424.

25. Which ECG changes are associated with hyperkalemia?

Peaked T waves, widened QRS, increased PR interval, flattened P waves, ventricular fibrillation, and asystole.

Green D, Green HD, New DI, Kalra PA: The clinical significance of hyperkalaemia-associated repolarization abnormalities in end-stage renal disease. Nephrol Dial Transpl 2013;28(1):99-105.

26. What are some cardiac causes of chest pain in children?

Cardiac causes of childhood chest pain are rare. Consider hypertrophic cardiomyopathy, drugs of abuse (such as cocaine), myocarditis, pericarditis, and arrhythmia.

Selbst S, Palermo R, Durani Y, Giordano K: Adolescent chest pain: Is it the heart? Clin Pediatr Emerg Med 2011;12(4):289-300.

27. Which ECG changes are associated with hypertrophic cardiomyopathy?

- Left ventricular hypertrophy (increased R-wave voltage in leads V_5-V_6)
- Left atrial hypertrophy (widened, notched P waves)
- Atrial fibrillation
- SVT

Selbst S, Palermo R, Durani Y, Giordano K: Adolescent chest pain: Is it the heart? Clin Pediatr Emerg Med 2011;12(4):289-300.

28. What risk factors are associated with sudden death in patients with hypertrophic cardiomyopathy?

Nonsustained VT on a Holter monitor, unexplained syncope, extreme left ventricular hypertrophy on echocardiogram, inadequate rise in blood pressure during exercise, family history of sudden death in a first-degree relative, and left ventricular outflow tract obstruction seen on echocardiogram.

Selbst S, Palermo R, Durani Y, Giordano K: Adolescent chest pain: Is it the heart? Clin Pediatr Emerg Med 2011;12(4):289-300.

29. How should chest pain from suspected cocaine ingestion be evaluated? What is the treatment?

- Evaluation: Urine drug screen (detects cocaine for 24-48 hours after use), ECG (may see findings consistent with acute coronary ischemia or arrhythmias)
- Treatment: Nitroglycerin (may help decrease coronary artery vasospasm), aspirin (or clopidogrel), and benzodiazepines (to blunt the central stimulation effects of cocaine)

Selbst S, Palermo R, Durani Y, Giordano K: Adolescent chest pain: Is it the heart? Clin Pediatr Emerg Med 2011;12(4):289-300.

30. How does a child with myocarditis present?

The classical presentation of myocarditis includes a viral prodrome (fever, upper respiratory infection symptoms), followed by chest pain or symptoms of heart failure.

Selbst S, Palermo R, Durani Y, Giordano K: Adolescent chest pain: Is it the heart? Clin Pediatr Emerg Med 2011;12(4):289-300.

31. What are the ECG changes of myocarditis?

ECG changes may include sinus tachycardia, low-voltage QRS complexes, abnormal T-wave inversion, heart block, arrhythmias, bundle branch blocks, infarct patterns, and ST-segment changes.

Selbst S, Palermo R, Durani Y, Giordano K: Adolescent chest pain: Is it the heart? Clin Pediatr Emerg Med 2011;12(4):289-300.

32. What ECG changes are associated with pericarditis?

There is a progression of ECG abnormalities in pericarditis: diffuse ST-segment elevation (leads I, II, III, aVL, aVF, V_2-V_6), PR depression (with normalized ST segments), T-wave flattening/inversion, low-voltage QRS (due to evolving pericardial effusion).

Selbst S, Palermo R, Durani Y, Giordano K: Adolescent chest pain: Is it the heart? Clin Pediatr Emerg Med 2011;12(4):289-300.

Key Points: Pediatric Chest Pain

1. Most chest pain in pediatric patients is not cardiac in origin.
2. ECG changes associated with hypertrophic cardiomyopathy include left ventricular hypertrophy.
3. Maintain a high suspicion of ingestion (such as cocaine) as the cause of chest pain in adolescents.
4. Focus on possible viral prodrome symptoms in a child with suspected myocarditis.

33. What is the differential diagnosis of congenital cyanotic heart disease?

- Ductal-dependent lesions: Tetralogy of Fallot, tricuspid atresia, pulmonary stenosis, pulmonary atresia
- Ductal-dependent lesions: Truncus arteriosis, transposition of the great vessels, total anomalous pulmonary venous return, hypoplastic left-sided heart syndrome

34. What is the initial management of a distressed newborn presenting with suspected congenital cyanotic heart disease?

- Secure the airway and ventilate.
- Maintain SpO_2 75% to 85%.
- Obtain IV access and administer prostaglandin E_1 at 0.05 to 0.1 μg/kg/minute.
- Obtain a chest radiograph.
- Consult cardiology.

35. Describe the typical cardiac examination of a patient with tetralogy of Fallot.

Patients with unrepaired tetralogy of Fallot often have a single, loud S_2. In addition, a harsh systolic ejection murmur will be heard, which is due to the obstructed pulmonary outflow tract. Though the constellation of abnormalities in tetralogy of Fallot includes a ventricular septal defect (VSD), flow across the VSD is generally not turbulent, and thus not audible.

Bailliard F, Anderson RH: Tetralogy of Fallot. Orphanet J Rare Dis 2009;4:2.

36. What is a hypercyanotic episode, or "tet spell"?

Hypercyanotic spells occur in patients with uncorrected tetralogy of Fallot. The rapid desaturation is related to acute and partial/complete obstruction of the subpulmonary outflow tract (thus rapidly decreasing pulmonary blood flow). After cyanosis develops, metabolic acidosis ensues, which results in increased pulmonary vascular resistance and decreased systemic vascular resistance; this may lead rapidly to myocardial ischemia, lethargy, and death. Spells may be triggered by agitation or decreased hydration.

Bailliard F, Anderson RH: Tetralogy of Fallot. Orphanet J Rare Dis 2009;4:2.

37. How should a "tet spell" be managed?

Manage "tet spells" at home by placing a child in a knee-chest position, which increases systemic vascular resistance and promotes systemic venous return to the heart.

In the ED, obtain IV access and administer IV fluids to improve right ventricular preload. Give supplemental oxygen to decrease peripheral pulmonary vasoconstriction. Give intravenous morphine (0.1 mg/kg/dose) to decrease the release of catecholamines, which, in turn, results in decreased heart rate (increasing filling time) and relaxation of the infundibular spasm. If the "tet spell" persists despite IV fluids, oxygen, and morphine, paralyze the child and intubate the child's trachea. Then administer phenylephrine (to increase systemic vascular resistance).

Bailliard F, Anderson RH: Tetralogy of Fallot. Orphanet J Rare Dis 2009;4:2.

38. List some of the complications of repaired hypoplastic left-sided heart syndrome (Fontan operation) that may be encountered in the ED.

- Supraventricular arrhythmias: Atrial flutter, AV reentry tachycardia, atrial ectopic tachycardia, pleural effusions
- Thromboembolic complications: Cerebrovascular accident, inferior or superior vena cava syndrome, pulmonary embolus
- Protein-losing enteropathy
- Pulmonary arteriovenous fistulas

Tsai W, Klein B: The postoperative cardiac patient. Clin Pediatr Emerg Med 2005;6:216-221.

Key Points: Congenital Cyanotic Heart Disease

1. In an infant presenting with symptoms of heart failure, consider structural cardiac anomalies.
2. Have a low threshold for starting prostaglandin in a cyanotic neonate.
3. Treat "tet spells" in the ED with IV fluids, supplemental oxygen, and intravenous morphine (after placing the child in a "knee-chest position").

39. When should echocardiography be performed in a patient with suspected Kawasaki disease?

Obtain an echocardiogram as soon as Kawasaki disease is suspected. It may help to confirm the disease; if the disease is certain, an echocardiogram allows a baseline evaluation of cardiac function and determines the extent of coronary artery aneurysms.

Newburger J, Takahashi M, Gerber M, et al: Diagnosis, treatment, and long-term management of Kawasaki disease: A statement for health professionals from the Committee on Rheumatic Fever, Endocarditis, and Kawasaki Disease, Council on Cardiovascular Disease in the Young, American Heart Association. Circulation 2004;110:2747.

40. What are the cardiac complications of Kawasaki disease?

- Coronary artery aneurysm (major complication)
- Decreased myocardial contractility
- Pericardial effusion
- Mild valvular disease
- Coronary arteritis

41. Which cardiac conditions cause sudden death in young athletes?

- Hypertrophic cardiomyopathy
- Coronary artery anomalies
- Myocarditis
- Aortic rupture (Marfan syndrome)
- Commotio cordis
- Arrhythmogenic right ventricular hypertrophy

Maron B, Shirani J, Poliac LC, et al: Sudden death in young competitive athletes. Clinical, demographic, and pathological profiles. JAMA 1996;276:199-204.

42. Which is the most common cause of sudden cardiac death in the pediatric population?

Hypertrophic cardiomyopathy is the most common cause of sudden cardiac death in children and adolescents, accounting for about half of the 500 annual deaths. There currently are no guidelines for screening of athletes, although the ECG will be abnormal in 75% to 95% of cases.

Maron BJ, Doerer JJ, Haas TS, et al. Sudden death in young competitive athletes. Analysis of 1866 deaths in the US 1980-2006. Circulation 2009, 119:1085.

43. What is Brugada syndrome? What are the classic ECG abnormalities?

Brugada syndrome is a genetic disorder associated with sudden cardiac death. Diagnosis depends on the combination of Brugada pattern on ECG plus clinical criteria. ECG typically shows "pseudo-RBBB" (right bundle branch block) and ST-segment elevation in leads V_1 to V_3. There are three types of Brugada syndrome: The first type is associated with ST-segment changes described as an upward convexity of the ST segment, leading to an inverted T wave. Brugada types 2 and 3 are associated with ST-segment elevation leading to a single or biphasic, upright T wave. Clinical signs of Brugada syndrome include family history of sudden cardiac death, syncope, a history of ventricular fibrillation, agonal nocturnal respirations, and self-terminating polymorphic VT.

Wilde A, Antzelevitch C, Borggrefe M, et al: Proposed diagnostic criteria for the Brugada syndrome: Consensus report. Circulation 2002;106(19):2514-2519.

44. What is the mechanism of sudden cardiac death in Brugada syndrome? Can this be treated?

Sudden cardiac death in Brugada syndrome is caused by ventricular fibrillation. The only proven effective treatment for Brugada syndrome is an implanted cardioverter-defibrillator.

Acknowledgment

The authors wish to thank Dr. Sarah Alander and Dr. James Hulse III for their contributions to this chapter in the previous edition.

CHAPTER 32

CENTRAL NERVOUS SYSTEM EMERGENCIES

Nanette C. Dudley

1. Describe the physical findings in Bell's palsy.

Bell's palsy refers to peripheral facial nerve weakness on one side of the face, including inability to wrinkle the forehead on the affected side, inability to close the affected eye, and flattening of the affected nasolabial fold. Bell's palsy may also involve hyperacusis in the affected ear (noises sound excessively loud) because the site of injury is thought to be in the facial canal and may involve other branches of C7—including those to the stapedius muscle, which dampens sound waves.

2. Does treatment with corticosteroids aid in the recovery of facial nerve function in Bell's palsy?

Yes. Corticosteroid treatment increases the likelihood of complete recovery.

Berg T, Bylund N, Marsk E, et al: The effect of prednisolone on sequelae in Bell's palsy. Arch Otolaryngol Head Neck Surg 2012;138(5):445-449.

3. What is Todd's paralysis?

Todd's paralysis refers to paresis or paralysis of one or more areas of the body after a seizure. This condition is transient, usually disappearing within 24 hours following a seizure.

4. A child is having an acute seizure. You know that intravenous (IV) access will not be possible quickly. What are your options?

- Rectal diazepam
- Intranasal midazolam
- Buccal midazolam
- Intraosseous access (any medication listed in Table 32-1 can be given through an intraosseous line)

Wiznitzer M: Buccal midazolam for seizures. Lancet 2005;366:182-183.

5. List the drugs commonly used to stop seizures acutely in the emergency department (ED).

Drugs commonly used to stop seizures in the ED are listed in Table 32-1.

6. Describe the characteristics of absence (petit mal) seizures.

Absence seizures usually develop in school-age children before puberty. The seizures are characteristically of abrupt onset with a brief loss of consciousness. Most are less than 30 seconds in duration. Children may appear to be staring or may have eye blinking or rhythmic nodding. The child does not fall, although he or she may drop things. These seizures are often easily precipitated by having the child hyperventilate.

7. What are infantile spasms?

Infantile spasms are characteristic seizures usually presenting between 3 and 7 months of age. The seizures are usually of sudden onset and generalized, with bilateral and symmetric contraction of the muscles in the neck, trunk, and extremities. The initial contraction typically lasts less than 2 seconds and is then followed by a sustained contraction of 2 to 10 seconds. Patients with infantile spasms have a poor developmental prognosis, and 85% have developmental slowing to various degrees.

Glaze DG: Management and prognosis of infantile spasms. UpToDate, 2013. Available at www.uptodate.com.

Table 32-1. Drugs Commonly Used to Stop Seizures in the Emergency Department

DRUG	DOSE	ROUTE	MAXIMUM DOSE
Lorazepam	0.05-0.1 mg/kg	IV	4 mg
Diazepam	0.05-0.3 mg/kg	IV	10 mg
	0.5 mg/kg	PR	
Midazolam	0.05-0.3 mg/kg	IV/IM	10 mg
	0.2 mg/kg	IN	
Phenytoin	15-20 mg/kg	IV, IM	1000 mg
Fosphenytoin	15-20 PE/kg	IV	1000 mg
Phenobarbital	15-20 mg/kg	IV, IM	1000 mg
Levetiracetam	20-40 mg/kg	IV	2500 mg
Valproic acid	20-40 mg/kg	IV	

The latter drugs have a longer onset of action (10-20 min) and are a better second- or third-line therapy. IM, intramuscular; IN, intranasal; IV, intravenous; PE, phenytoin sodium equivalents; PR, per rectum.
In part, from Gorelick MH, Blackwell CD: Neurologic emergencies. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1011-1032, and from Abend NS, Gutierrez-Colina AM, Dlugos DJ: Medical treatment of pediatric status epilepticus. Semin Pediatr Neurol 2010;17:169-175.

8. A 16-year-old boy presents after an early-morning generalized tonic clonic seizure. On history, he describes occasional, brief jerking movements in the morning that make teeth brushing and hair combing difficult. These jerking movements resolve and do not recur later in the day. What does this symptom pattern suggest?

Juvenile myoclonic epilepsy most commonly presents between 12 and 18 years of age with brief, bilateral flexor jerking movements of the arms. The seizures can be precipitated by sleep deprivation, alcohol ingestion, and awakening from sleep. Patients may have generalized tonic clonic seizures but are normal neurologically.

Yu KT: Approach to the child with seizures. In Osborn LM, Dewitt TG, First LR, Zenel JA (eds): Pediatrics. St. Louis, Elsevier Mosby, 2005, pp 752-756.

9. How are seizures distinguished from breath-holding spells?

Involuntary muscular contractions occur in both, although they are more prominent with seizures. In breath-holding spells, these contractions are due to transient cerebral hypoperfusion. With breath-holding spells, a precipitating event causes the child to cry or be angry or frustrated, and thus hold his or her breath or perform the Valsalva maneuver. Seizures usually do not have an obvious precipitating event. Breath-holding spells are usually brief and may feature a short period of sleepiness afterward. Seizures may be brief, but suspicion for a seizure disorder occurs when the episode lasts longer than 1 minute or if the postictal period is prolonged. Finally, breath-holding spells usually occur in children 6 months to 4 years of age; episodes outside these limits are more suspicious for seizures.

10. What are the indications for urgent computed tomography (CT) after a seizure?

- Postictal focal neurologic deficits that do not resolve quickly
- Signs of increased intracranial pressure
- More than a transient change in level of consciousness
- Posttraumatic seizures (not impact seizures)

Magnetic resonance imaging (MRI) is the preferred study in all cases but is usually not immediately available in the emergency setting. Patient stability may also be a factor, as CT is typically faster and often does not require sedation.

Hirtz D, Ashwal S, Berg A, et al: Practice parameter: Evaluating a first nonfebrile seizure in children. Neurology 2000;55:616-623.

11. What are the indications for nonurgent MRI after a seizure?

- Focal features of the seizure
- Cognitive or motor impairment of unknown cause
- Changes in seizure character, neurologic examination, or electroencephalogram
- Age younger than 1 year

MRI testing for the preceding indications avoids unnecessary radiation exposure from CT.

Hirtz D, Ashwal S, Berg A, et al: Practice parameter: Evaluating a first nonfebrile seizure in children. Neurology 2000;55:616-623.

12. What is the incidence of febrile seizures? When do they occur?

About 2% to 5% of all children have a febrile seizure; most occur in children between the ages of 6 months and 5 years.

American Academy of Pediatrics: Febrile seizures. Available at www.aap.org/patiented/febrileseizures.htm.

13. What makes a febrile seizure "simple" or "complex"?

Both occur when a child has fever. Usually there is high fever of abrupt onset.

A simple febrile seizure has all of the following characteristics:

- The seizure is brief (<15 minutes).
- The seizure is generalized.
- The child appears well shortly after the seizure stops.

A complex febrile seizure has at least one of the following characteristics:

- The seizure is prolonged (>15 minutes).
- The seizure is focal.
- The child has two or more seizures in 1 day.

14. What is the recurrence risk for a febrile seizure?

Approximately 30%. This number varies by age at onset of first febrile seizure.

15. Is the risk of recurrence increased if the initial febrile seizure is complex?

No. Children at greater risk of recurrence may have one or more of the following features:

- Young age at onset
- History of febrile seizures in a first-degree relative
- Lack of hyperpyrexia in the ED
- Brief duration between the onset of fever and the initial seizure

Berg AT, Shinnar S, Darefsky AS, et al: Predictors of recurrent febrile seizures. A prospective cohort study. Arch Pediatr Adolesc Med 1997;151:371-378.

16. Is the risk of epilepsy increased in a child with a simple febrile seizure?

No. The risk of developing epilepsy in a child after a simple febrile seizure is the same as that of a child without febrile seizures. However, children with multiple simple febrile seizures, those less than 12 months old at the time of the first simple febrile seizure, and those with a family history of epilepsy have a higher risk of developing epilepsy.

American Academy of Pediatrics, Steering Committee on Quality Improvement and Management, Subcommittee on Febrile Seizures: Febrile seizures: Clinical practice guideline for the long-term management of the child with simple febrile seizures. Pediatrics 2008;121:1281-1286.

17. What tests should be performed on children after a simple febrile seizure?

There is no need for brain imaging studies or electroencephalography after a simple febrile seizure. Perform laboratory testing or radiography as appropriate for the diagnosis and management of the cause of the fever.

American Academy of Pediatrics, Steering Committee on Quality Improvement and Management, Subcommittee on Febrile Seizures: Clinical practice guideline: Febrile seizures: Guideline for the neurodiagnostic evaluation of the child with a simple febrile seizure. Pediatrics 2011;127:389-394.

18. When should you consider a lumbar puncture in a child with a febrile seizure?

- History concerning for meningitis
- Cranky, irritable child who is difficult to console

- Meningeal signs or bulging fontanel
- Infants 6 to 12 months of age who are deficient in *Haemophilus influenzae* or *Streptococcus pneumoniae* immunizations or if immunization status is undetermined
- Pretreatment with antibiotics

American Academy of Pediatrics, Steering Committee on Quality Improvement and Management, Subcommittee on Febrile Seizures. Clinical practice guideline: Febrile seizures: Guideline for the neurodiagnostic evaluation of the child with a simple febrile seizure. Pediatrics 2011;127: 389-394.

Key Points: Seizures

1. Many common seizure types have characteristic presentations that can be identified with the initial history and physical examination.
2. Immediate testing or imaging is not always indicated after a seizure.
3. Workup after a febrile seizure should be directed to the cause of the fever.
4. When IV access is unavailable, absorption of benzodiazepines by the mucous membranes of the nose, buccal mucosa, or rectum may terminate the seizure.

19. In a child with suspected meningitis, what are some contraindications to performing an immediate lumbar puncture?

- Focal neurologic findings on examination
- Evidence of spinal cord trauma
- Infection in the tissues near the puncture site
- Focal seizures
- Coma
- Papilledema
- Severe coagulation defects (not corrected)
- Cardiopulmonary instability

In these cases, antibiotic therapy may be initiated presumptively and lumbar puncture delayed.

Cronan K, Wiley J: Lumbar puncture. In Henretig F, King C (eds): The Textbook of Pediatric Emergency Medicine Procedures. Philadelphia, Lippincott Williams & Wilkins, 2008, p 507.

20. What is an impact seizure?

An impact seizure is a brief, generalized seizure occurring immediately at the time of an injury. The seizure activity is thought to be due to traumatic depolarization of neurons on impact. These children do not have a higher incidence of epilepsy.

21. How is a hyponatremic seizure managed?

Hyponatremic seizures are often managed with hypertonic saline to transiently raise plasma sodium levels by 5 to 10 mEq to stop the seizure. The dose is 2 to 4 mL/kg body weight of 3% sodium chloride (0.5 mEq/mL), given rapidly and intravenously.

22. What are some differentiating features of Guillain-Barré syndrome and transverse myelopathy?

The differentiating features of Guillain-Barré syndrome and transverse myelopathy are shown in Table 32-2.

23. What does the term "transverse" in transverse myelitis refer to?

The term "transverse" refers to the bandlike sensory level at which dysfunction occurs, leading to the common presenting symptoms.

Wolf VL, Lupo PJ, Lotze TE: Pediatric acute transverse myelitis overview and differential diagnosis. J Child Neurol 2012;27(11):1426-1436.

24. What is the Miller Fisher variant of Guillain-Barré syndrome?

Patients with this form of Guillain-Barré syndrome also have ophthalmoplegia, areflexia, and ataxia.

Table 32-2. Guillain-Barré Syndrome Versus Transverse Myelopathy

SYMPTOM	GUILLAIN-BARRÉ SYNDROME	TRANSVERSE MYELOPATHY
Early pain/paresthesias	+	+
Progressive symmetrical weakness	Arms and legs	Legs
Bilateral facial weakness	+	–
Areflexia	+	+
Autonomic dysfunction	+	+
Sensory level*	–	+
Abnormal rectal tone	–	+

*A level below which the patient has absence of sensation.
From Gorelick MH, Blackwell CD: Neurologic emergencies. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott William & Wilkins, 2010, pp 1011-1032.

25. What simple grooming measure should be performed on any patient with acute onset of ascending paralysis?

Combing the hair. Tick paralysis can present very similarly to Guillain-Barré syndrome, although the rate of progression is faster and tick paralysis is accompanied by ataxia (not present in Guillain-Barré syndrome). Removal of the tick can bring about rapid improvement, and the tick is often found serendipitously.

Felz MW, Smith CD, Swift TR: A six-year-old girl with tick paralysis. N Engl J Med 2000;342:90-94.

26. A 2-month-old baby with a history of hypotonia presents in acute respiratory distress. He has abdominal respirations and appears to be tiring out. In preparing for intubation, what skeletal muscle relaxant is relatively contraindicated?

Succinylcholine. In children with undiagnosed skeletal myopathy, concern is raised about the use of succinylcholine and the potential for ventricular dysrhythmias and cardiac arrest from hyperkalemia.

27. What is the initial symptom seen in infant botulism?

Constipation is often the first symptom in babies with infant botulism. Constipation may occur for days to weeks before lethargy, feeding difficulties, diminished reflexes, weakness, hypotonia, and diminished gag reflex appear.

28. How is the diagnosis of infant botulism made?

The diagnosis is largely clinical and relies on the elimination of other causes of lethargy and weakness. *Clostridium botulinum* toxin may be identified in the stool, but this test is not available in the ED. Brief, small, abundant motor action potentials are characteristic on electromyogram.

American Academy of Pediatrics: Clostridial infections. In Pickering LK (ed): 2012 Red Book: Report of the Committee on Infectious Diseases, 29th ed. Elk Grove Village, IL, American Academy of Pediatrics, 2012, pp 281-284.

29. What is the currently recommended treatment for infant botulism?

Human-derived botulism immune globulin decreases the duration of hospitalization, mechanical ventilation, and tube or parenteral feeding.

Arnon SS, Schechter R, Maslanka SE, et al: Human botulism immune globulin for the treatment of infant botulism. N Engl J Med 2006;354(5):462-471.

30. How would you differentiate a migraine headache from that due to idiopathic intracranial hypertension (previously called pseudotumor cerebri)?

Both conditions present with headache, although headache due to idiopathic intracranial hypertension may be worse in the morning, and migraine symptoms are often relieved by sleep. Nausea, vomiting, and visual problems may also occur with both, and the neurologic

examination may be normal. However, papilledema is seen in most cases of idiopathic intracranial hypertension and is not present in migraine. Perform CT or MRI to rule out other causes of elevated intracranial pressure. If imaging does not demonstrate a mass lesion, perform a lumbar puncture to measure opening pressure, which will be elevated in idiopathic intracranial hypertension.

31. Describe the worst complication of idiopathic intracranial hypertension.
Elevated optic nerve pressure can lead to optic nerve ischemia and blindness.

32. Describe the weakness in myasthenia gravis.
Bilateral ptosis and ophthalmoplegia are the most common manifestations of myasthenia gravis. The weakness is variable, and the specific muscles affected may vary from examination to examination. Those with generalized weakness may report a history of worsening throughout the day or with continued activity. In the ED, easy fatigability of muscle strength may be demonstrated.

Gorelick MH, Blackwell CD: Neurologic emergencies. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1011-1032.

33. Which electrolyte abnormality is responsible for periodic paralysis?
Periodic paralysis can be due to hypokalemia or hyperkalemia. Paralytic episodes may occur after strenuous exercise. Persistent weakness is possible.

34. Acute cerebellar ataxia is most commonly attributed to which viral illness?
Varicella. Acute cerebellar ataxia typically occurs within 10 days of a viral illness and is felt to be a parainfectious or postinfectious phenomenon. The onset of ataxia is acute, and nystagmus and slurred speech can occur, but most children are otherwise normal. Recovery typically takes a few weeks, and residual neurologic deficits are possible.

35. What is "milkmaid's hand"?
Milkmaid's hand is found in patients with chorea. The child cannot maintain a continuous grasp when asked to squeeze the examiner's fingers, but instead performs intermittent squeezing as if he or she were "milking" the finger. These low-amplitude jerking movements are characteristic of chorea.

36. What is the most common cause of acquired chorea in children?
Sydenham's chorea, which is a manifestation of rheumatic fever.

37. In uncal (unilateral transtentorial) herniation, is pupillary dilation present on the same side as the increased intracranial pressure or the opposite side?
The pupil dilates on the *same side* where the temporal lobe causes the uncus to bulge into the tentorial notch.

38. Why does the pupil dilate in uncal herniation?
Direct pressure on the oculomotor nerve (cranial nerve III) causes the ipsilateral pupil to dilate.

Key Points: Intracranial Pressure

1. A careful cranial nerve examination may be the clue to a mass lesion in the brain.
2. The presence of papilledema should prompt an imaging study of the brain.
3. A dilated unilateral pupil after head injury is concerning for herniation.
4. Headache from sinusitis may not be focused directly over the sinuses.

39. How do brain abscesses occur?
- From direct extension from chronic infection in the sinuses, ears, or dental structures
- Following acute infections such as meningitis
- Hematogenously from endocarditis or congenital heart disease (particularly with right-to-left shunting)
- Following penetrating brain injury or neurosurgery

40. What are the neurologic complications of sinusitis?

- Orbital cellulitis
- Ophthalmoplegia
- Cavernous sinus thrombosis
- Intracranial empyema or abscess
- Meningitis

41. Why is the diagnosis of sphenoid sinusitis often difficult?

The sphenoid sinuses are located posterior to the ethmoid sinuses, and the usual physical examination techniques, such as percussion and transillumination, are not useful. Headache is usually severe but can mimic migraine and is often located on top of the head, behind the eyes, or at the back of the neck. Painful paresthesias may occur in the trigeminal nerve distribution. Eighty percent of pediatric patients with intracranial complications of sinusitis have sphenoid involvement.

Saitoh A, Beall B, Nizet V: Fulminant bacterial meningitis complicating sphenoid sinusitis. Pediatr Emerg Care 2003;19:415-417.

42. Where are pediatric brain tumors most commonly located?

Posterior fossa tumors account for 50% of brain tumors in children of all ages and are the most common location for brain tumors in children ages 1 to 8 years. This explains why gait disturbances are common in children with brain tumors.

43. Brain tumors in children commonly present with which ocular nerve palsy?

The sixth cranial nerve (abducens) is commonly affected in children with posterior fossa brain tumors. In some, the inability to perform lateral rectus motion may be the sole manifestation of the tumor. The sixth nerve lies close to the cerebellum, fourth ventricle, and pons, which may explain its involvement in childhood brain tumors.

Rheingold SR, Lange BJ: Oncologic emergencies. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 5th ed. Philadelphia, Lippincott Williams & Wilkins, 2006, pp 1239-1274.

44. What is acute demyelinating encephalomyelitis (ADEM)?

ADEM is an inflammatory demyelinating disease that presents in children and young adults. It is an uncommon illness usually occurring in the winter and spring. In many cases it follows an upper respiratory tract infection. Children present with multifocal neurologic signs. Symptoms last for 2 to 4 weeks.

Alper G: Acute disseminated encephalomyelitis. J Child Neurol 2012;27(11):1408-1425.

45. What is the differential diagnosis of ADEM?

- Multiple sclerosis
- Optic neuritis
- Transverse myelitis
- Devic's syndrome

46. The symptoms of headache, confusion or altered mental status, seizures, and visual disturbance classically describe what syndrome?

Posterior reversible encephalopathy syndrome (PRES). Most (but not all) patients with PRES are hypertensive.

Endo A, Fuchigami T, Maki H, et al: Posterior reversible encephalopathy syndrome in childhood; Report of 4 cases and review of the literature. Pediatr Emerg Care 2012;28(2): 153-157.

ENDOCRINE DISORDERS

Ramsey C. Tate and Robert E. Sapien

CHAPTER 33

1. Why are children susceptible to hypoglycemia?

Children are susceptible because:

- Glycogen stores are small.
- Glucose utilization is high because of increased metabolic demands.
- Fewer gluconeogenesis precursors are generated because of comparatively less fat and muscle mass.
- A child's developing brain needs a constant source of substrate.

Clarke W, Jones T, Rewers A, et al: Assessment and management of hypoglycemia in children and adolescents with diabetes. Pediatr Diabetes 2009;10:134-145.

2. What glucose level is considered hypoglycemia? How do you correct it?

The level at which to treat is controversial, but most clinicians treat at a level of less than 60 mg/dL. Infants are generally treated at a lower threshold level of 40 mg/dL. Treat infants with $D_{10}W$ (10% dextrose in water), 0.25 mg/kg (2.5 mL/kg) body weight; children 1 year of age and older can also be treated with 0.25 mg/kg (2.5 mL/kg) of $D_{10}W$ or 1 mL/kg of $D_{25}W$ (25% dextrose in water).

Clarke W, Jones T, Rewers A, et al: Assessment and management of hypoglycemia in children and adolescents with diabetes. Pediatr Diabetes 2009;10:134-145.

3. At what point should diagnostic tests for hypoglycemia be obtained? Which tests?

A bedside glucose test is essential because a delay in treatment can be neurologically devastating. A diagnostic workup is indicated for infants and children in whom the hypoglycemia has no explanation, such as oral hypoglycemic ingestion, sepsis, poor intake, or anorexia in a diabetic child who received insulin. Obtain the diagnostic tests immediately when the hypoglycemic child presents to the emergency department (ED). The tests include plasma glucose, electrolytes, lactate, pyruvate, ketohydroxybutyrate, acetoacetate, insulin, glucagon, cortisol, growth hormone, free fatty acids, alanine, glycerol, and urinalysis, including reducing substances.

Key Points: Hypoglycemia

1. Rapid identification is essential.
2. Children are susceptible because of small glycogen stores and high glucose utilization.
3. Treat hypoglycemia as follows: for infants (glucose level $<$ 40 mg/dL), $D_{10}W$ 0.25 mg/kg (2.5 mL/kg); for children 1 year of age or older (glucose level $<$ 60 mg/dL), $D_{10}W$ 0.25 mg/kg (2.5 mL/kg) or $D_{25}W$ (1 mL/kg).

4. What is the most common cause of hyperthyroidism in children?

Graves' disease is the most common cause. Children at risk include females (female-male ratio is 5:1), those with familial autoimmune diseases (e.g., diabetes, adrenal insufficiency), and those from families with a history of thyroid disease.

Léger J, Carel JC: Hyperthyroidism in childhood: Causes, when and how to treat. J Clin Res Pediatr Endocrinol 2013;5(Suppl 1):50-56.

5. Presenting symptoms of hyperthyroidism are commonly attributed to which other body systems or disorders?

Symptoms from hyperthyroidism are commonly confused with other conditions. For example, cardiac disorders (palpitations, sinus tachycardia) and psychological problems (school difficulties, labile emotions, hyperactivity, new-onset psychiatric symptoms) mimic hyperthyroidism.

McKeown NJ, Tews MC, Gossain VV, Shah SM: Hyperthyroidism. Emerg Med Clin North Am 2005;23 (3):669-685.

6. What are the symptoms of thyroid storm? What is the treatment?

The symptoms of thyroid storm may be nonspecific (fever, sweating, tachycardia, hypertension, agitation, confusion, nausea, vomiting), making the diagnosis difficult unless there is a clear history of hyperthyroidism. The three systems most affected include the cardiovascular, gastrointestinal, and central nervous systems. Supportive care is the first step in management (oxygen, intravenous [IV] fluids [dextrose-containing fluids preferred to address high metabolic demand], acetaminophen for fever, cooling measures). Follow this with an IV beta blocker for the cardiac effects and routine hyperthyroid treatments (methimazole, propylthiouracil, iodine) in high doses.

McKeown NJ, Tews MC, Gossain VV, Shah SM: Hyperthyroidism. Emerg Med Clin North Am 2005;23 (3):669-685.

Thyroid storm. MedlinePlus Medical Encyclopedia. Available at http://www.nlm.nih.gov/medlineplus/ency/article/000400.htm. Accessed July 14, 2013.

7. If congenital hypothyroidism is missed in the neonatal period, when will signs and symptoms present?

Patients will present with symptoms at 6 to 12 weeks of age. The first signs and symptoms are constipation, a large posterior fontanel, poor feeding, hypotonia, jaundice, and hypothermia.

Abduljabbar MA, Afifi AM: Congenital hypothyroidism. J Pediatr Endocrinol Metab 2012;25(1-2):13-29.

8. Congenital hypothyroidism is familiar to pediatricians, but can newborns be hyperthyroid?

Indeed they can. In fact, substantial mortality risk is associated with neonatal thyrotoxicosis. Neonatal thyrotoxicosis presents in the first 2 weeks of life in infants born to hyperthyroid mothers. Symptoms are nonspecific and can mimic neonatal sepsis, including irritability, sweating, weight loss, poor feeding, vomiting, and diarrhea. Signs are typical of hyperthyroidism, such as exophthalmos, goiter, hyperthermia, tachycardia, jaundice, hepatomegaly, and even cardiac failure.

Léger J, Carel JC: Hyperthyroidism in childhood: Causes, when and how to treat. J Clin Res Pediatr Endocrinol 2013;5(Suppl 1):50-56.

9. How do the adrenal glands respond to stress?

The adrenal glands respond to stress by secreting increased amounts of glucocorticoids and mineralocorticoids. Physiologic stresses include surgery, trauma, and infection. Both children with adrenal insufficiency and children taking long-term exogenous glucocorticoid therapy require stress dose replacement of glucocorticoids and mineralocorticoids because their adrenal glands cannot respond to such stressors.

Shulman DI, Palmert MR, Kemp SF, Lawson Wilkins Drug and Therapeutics Committee: Adrenal insufficiency: Still a cause of morbidity and death in childhood. Pediatrics 2007;119(2):e484-e494.

10. What signs and symptoms are likely in children with acute adrenal insufficiency?

Signs and symptoms are due to failure of the adrenals to respond to stress, and they are fairly nonspecific. They include dehydration with hypovolemic shock, profound hypoglycemia, hyponatremia, hyperkalemia, hypotension, abdominal pain, and altered mental status. Acute adrenal insufficiency is often triggered by an infection or other physiologic stressor.

Shulman DI, Palmert MR, Kemp SF, Lawson Wilkins Drug and Therapeutics Committee: Adrenal insufficiency: Still a cause of morbidity and death in childhood. Pediatrics 2007;119(2):e484-e494.

11. What is the emergency treatment for acute adrenal insufficiency?

Initial resuscitation includes the following:

- Monitoring of vital signs, cardiac rhythm, perfusion, glucose, and electrolytes
- Fluid resuscitation with 0.9% saline for shock and dehydration
- Correction of hypoglycemia
- Hydrocortisone 50 to 75 mg/m^2 IV or intramuscular (IM)
- Correction of hyperkalemia (sodium polystyrene sulfonate, glucose/insulin, calcium gluconate, sodium bicarbonate)

Initial laboratory tests include random cortisol levels, electrolytes, glucose, adrenocorticotropic hormone (ACTH) levels, plasma renin activity, aldosterone level, and urine electrolytes.

Shulman DI, Palmert MR, Kemp SF, Lawson Wilkins Drug and Therapeutics Committee: Adrenal insufficiency: Still a cause of morbidity and death in childhood. Pediatrics 2007;119(2):e484-e494.

12. What is the most common form of congenital adrenal hyperplasia (CAH) presenting in infancy? What is the incidence of this type?

The most common form of CAH during infancy is 21-hydroxylase deficiency. It accounts for 95% of all cases. Clinical salt wasting develops in 30% to 70% of patients. The incidence of this type of CAH is 1 in 10,000 to 1 in 20,000 live births.

13. When does a child with salt-wasting CAH generally present to the ED?

The infant generally presents between 2 and 5 weeks of life. Symptoms are usually nonspecific and can be similar to neonatal sepsis, consisting of poor feeding, lethargy, irritability, vomiting, and poor weight gain. Perform a general examination, including a thorough evaluation of the genitals, as these patients often have ambiguous genitalia. The mortality rate for salt-wasting CAH that is not diagnosed by newborn screening is 4% to 10%.

14. In the ED, what are the most urgent laboratory studies to perform if CAH is suspected?

Check serum electrolytes and glucose. In salt-wasting CAH, hyperkalemia and hyponatremia are typically seen; the glucose level can be normal or low. Metabolic acidosis is usually present.

15. How is this type of CAH treated in the ED?

If dehydration is present, begin volume expansion with 0.9% saline promptly. Give hydrocortisone immediately as a 25 mg IV bolus. Treat hypoglycemia with 0.25 g/kg of IV dextrose (2.5 mL/kg of $D_{10}W$). Infants with CAH tolerate hyperkalemia better than do other infants and children, but hyperkalemia needs to be addressed. Volume expansion is the primary treatment. Do not use dextrose and insulin for acute management of hyperkalemia due to CAH, because treatment may worsen hypoglycemia.

Agus MSD: Endocrine emergencies. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 758-782.

Speiser PW, Azziz R, Baskin LS, et al: Congenital adrenal hyperplasia due to steroid 21-hydroxylase deficiency: An endocrine society clinical practice guideline. J Clin Endocrinol Metab 2010;95 (9):4133-4160.

16. What is the most common cause of amenorrhea in adolescents?

Pregnancy! Other causes include oral contraceptive use, pituitary infarction, uterine synechiae in postpartum adolescents, outflow obstruction (imperforate hymen, vaginal or uterine agenesis), and hypoestrogenic states (anorexia, athletics).

Deligeoroglou E, Athanasopoulos N, Tsimaris P, et al: Evaluation and management of adolescent amenorrhea. Ann N Y Acad Sci 2010;1205(1):23-32.

17. What endocrine conditions are associated with dysfunctional uterine bleeding?

Dysfunctional uterine bleeding is associated with immaturity of the hypothalamic-pituitary axis, polycystic ovary syndrome, prolactinomas, thyroid dysfunction, von Willebrand disease, diabetes, adrenal hyperplasia or tumors, and other chronic illnesses.

LaCour DE, Long DN, Perlman SE: Dysfunctional uterine bleeding in adolescent females associated with endocrine causes and medical conditions. J Pediatr Adolesc Gynecol 2010;23(2):62-70.

18. What questions or clinical findings may help you consider type 1 diabetes in your differential diagnosis?

Consider type 1 diabetes in children who are clinically dehydrated yet continue with urine output. Information regarding the number of wet diapers per day, nocturnal incontinence, or leaving the classroom often to urinate may help you hone in on the diagnosis. Consider children with weight loss and a history of vague abdominal pain as possibly having type 1 diabetes.

Gan MJ, Albanese-O'Neill A, Haller MJ: Type 1 diabetes: Current concepts in epidemiology, pathophysiology, clinical care, and research. Curr Probl Pediatr Adolesc Health Care 2012;42 (10):269-291.

19. What are acute complications of type 1 diabetes?

Diabetic ketoacidosis (DKA) and hypoglycemia are common, recurrent, serious complications. They are related to poor blood glucose control, socioeconomic factors, psychiatric problems, duration of the disease, and the child's age.

Rewers A, Chase HP, Mackenzie T, et al: Predictors of acute complications in children with type 1 diabetes. JAMA 2002;287(19):2511-2518.

20. What usually leads to DKA?

Insulin underutilization is the usual factor leading to DKA in a child known to have diabetes. As DKA starts, the child develops anorexia and nausea and begins to vomit. Parents or the patient may stop administering insulin even though the child is hyperglycemic, hence worsening the condition. Acute infections, psychologically stressful situations, medical noncompliance, and inflammatory processes can also lead to DKA. Signs and symptoms include dehydration, abdominal pain, tachypnea, nausea, vomiting, listlessness, coma, fruity odor, ketonuria(emia), hyperglycemia(uria), measured hyponatremia, and intravascular hyperkalemia (despite total body depletion). DKA may be the initial presentation of new-onset diabetes.

Gan MJ, Albanese-O'Neill A, Haller MJ: Type 1 diabetes: Current concepts in epidemiology, pathophysiology, clinical care, and research. Curr Probl Pediatr Adolesc Health Care 2012;42 (10):269-291.

21. Is there more than one level of DKA severity?

Yes. DKA is defined as hyperglycemia, venous pH less than 7.3, and bicarbonate less than 15 mmol/L with glycosuria, ketonuria, and ketonemia. DKA can be mild, moderate, or severe. Mild DKA is a pH of 7.21 to 7.3 and bicarbonate level 11 to 15 mmol/L. Children with mild DKA have normal mental status and respiratory status. They are good candidates for outpatient therapy as long as they can keep themselves hydrated orally. Moderate DKA is pH 7.11 to 7.2 and bicarbonate 6 to 10 mmol/L. Severe DKA is pH less than 7.1 and bicarbonate less than 5 mmol/L or mental status changes. The degree of DKA is *not* dependent on the extent of hyperglycemia. The glucose level is always elevated with DKA, but there is a common misconception that the glucose has to be *severely* elevated for DKA to exist.

Gan MJ, Albanese-O'Neill A, Haller MJ: Type 1 diabetes: Current concepts in epidemiology, pathophysiology, clinical care, and research. Curr Probl Pediatr Adolesc Health Care 2012;42 (10):269-291.

Wolfsdorf J, Craig ME, Daneman D, et al: Diabetic ketoacidosis in children and adolescents with diabetes. Pediatr Diabetes 2009;10(Suppl 12):118-133.

22. Of what do children and adolescents with type 1 diabetes die?

The mortality rate for DKA is 0.5%, and the most common reason that children with type 1 diabetes in DKA die is cerebral edema. Cerebral edema occurs in 1% of cases of DKA but has a 60% to 80% mortality rate. Risk factors for cerebral edema are high initial blood urea nitrogen level, lower partial pressure of arterial carbon dioxide, more severe acidosis (lower bicarbonate and pH), administration of bicarbonate or bolus IV insulin, and inadequate increase in serum sodium concentration. Children younger than age 3 and children with their first presentation of DKA are also at increased risk of cerebral edema.

Gan MJ, Albanese-O'Neill A, Haller MJ: Type 1 diabetes: Current concepts in epidemiology, pathophysiology, clinical care, and research. Curr Probl Pediatr Adolesc Health Care 2012;42 (10):269-291.

23. What is the standard treatment for DKA?

Although protocols for DKA management vary, keeping to a standard treatment protocol helps ensure consistent care. Standard DKA management requires fluid replacement, metabolic correction, and electrolyte correction.

- **Fluid replacement:** Fluid replacement with 0.9% saline at 10 to 20 mL/kg in the first hour is recommended. The remaining fluid deficit is replaced over the subsequent 24 to 48 hours in addition to maintenance fluids. In moderate DKA, assume 5% to 7% dehydration (50-70 mL/kg), and in severe DKA assume 7% to 10% (70-100 mL/kg).

- **Metabolic correction:** After the first hour, administer insulin as an IV drip at 0.1 U/kg/hour for moderate and severe DKA. Do *not* administer bolus insulin. Glucose should decrease no faster than 50 to 100 mg/dL/hour. When the serum glucose level is 300 g/dL or below, change fluids to 0.45% saline with 5% dextrose, but do not discontinue the insulin. If glucose is falling too rapidly, increase the amount of dextrose in fluids to a maximum of 12.5%.
- **Electrolyte correction:** Careful monitoring of potassium, sodium, and phosphate is also indicated. There is total-body potassium depletion, which is initially masked, and extracellular potassium levels are generally normal to high. As the acidosis corrects, potassium moves intracellularly and the serum potassium level begins to fall, more accurately reflecting the total-body depletion. Most patients should have 40 mmol/L of potassium added to fluids at the time that insulin is started. If patients are hyperkalemic, add potassium to fluids only after the patient is urinating.

Gan MJ, Albanese-O'Neill A, Haller MJ: Type 1 diabetes: Current concepts in epidemiology, pathophysiology, clinical care, and research. Curr Probl Pediatr Adolesc Health Care 2012;42 (10):269-291.
Wolfsdorf J, Craig ME, Daneman D, et al: Diabetic ketoacidosis in children and adolescents with diabetes. Pediatr Diabetes 2009;10(Suppl 12):118-133.

24. Which signs and symptoms should make you consider the diagnosis of type 2 diabetes mellitus?

Obesity (over 85% of patients are overweight); acanthosis nigricans (seen in over 85% of patients), a sign of insulin resistance; nonketotic hyperglycemia; glycosuria; weight loss (less common than with type 1 diabetes); polyuria and polydipsia (less common than with type 1 diabetes); rarely DKA; and ketonuria in 25% of patients. Type 2 diabetes mellitus is becoming more prevalent among children between 10 and 21 years old.

Copeland KC, Silverstein J, Moore KR, et al: Management of newly diagnosed type 2 diabetes mellitus (T2DM) in children and adolescents. Pediatrics 2013;131(2):364-382.
Nesmith JD: Type 2 diabetes mellitus in children and adolescents. Pediatr Rev 2001;22(5):147-152.

25. What is the probable abnormality in type 2 diabetes mellitus?

The probable abnormality is insulin resistance. This develops over a long period of time and results in a hyperinsulinemic condition. It can lead to dysfunctional uterine bleeding, hypertension, polycystic ovary disease, and acanthosis nigricans. The islets of Langerhans eventually become unable to maintain this level of insulin production, which leads to a relative insulin deficiency and hence hyperglycemia.

Copeland KC, Silverstein J, Moore KR, et al: Management of newly diagnosed type 2 diabetes mellitus (T2DM) in children and adolescents. Pediatrics 2013;131(2):364-382.
Nesmith JD: Type 2 diabetes mellitus in children and adolescents. Pediatr Rev 2001;22(5):147-152.

Key Points: Diabetes Mellitus

1. Children in DKA need skillful and judicious correction of electrolyte, metabolic, and glucose abnormalities as well as careful clinical monitoring and reassessment.
2. For children in DKA, try to reduce the glucose level at a rate no faster than 50 to 100 mg/dL/hour. Cerebral edema is more dangerous than hyperglycemia.
3. Do not discontinue insulin when treating DKA. Add glucose when the serum level is less than 300 mg/dL to avoid hypoglycemia.

26. True or false: DKA is the only acute metabolic abnormality that occurs in children with type 2 diabetes mellitus.

False. There are reported deaths from hyperglycemic hyperosmolar states. A hyperglycemic hyperosmolar state is described as hyperglycemia higher than 600 mg/dL, serum osmoles higher than 330 mOsm/kg, small or no ketonemia (ketonuria), and serum carbon dioxide level higher than 15 mmol/L. Just as in DKA, cerebral edema is a grave complication that can occur from the hyperglycemic, hyperosmolar state of type 2 diabetes.

Copeland KC, Silverstein J, Moore KR, et al: Management of newly diagnosed type 2 diabetes mellitus (T2DM) in children and adolescents. Pediatrics 2013;131(2):364-382.

Gan MJ, Albanese-O'Neill A, Haller MJ: Type 1 diabetes: Current concepts in epidemiology, pathophysiology, clinical care, and research. Curr Probl Pediatr Adolesc Health Care 2012;42 (10):269-291.

27. What are the key differences between diabetes insipidus (DI) and syndrome of inappropriate secretion of antidiuretic hormone (SIADH)?

Both DI and SIADH involve disorders of water homeostasis. In DI, free water is not reabsorbed. Dilute urine produced by an otherwise clinically dehydrated child, polydipsia, polyuria, fever, irritability, poor feeding and poor weight gain, constipation, and hypernatremia are presenting signs and symptoms. In contrast, children with SIADH are unable to excrete free water. Clinical symptoms may be absent except in rapidly progressive cases of hyponatremia, which may present as confusion, weakness, or seizures. Key laboratory studies in determining water homeostasis include serum electrolytes, serum osmoles, and urine osmoles.

Ranadive SA, Rosenthal SM: Pediatric disorders of water balance. Endocrinol Metab Clin North Am 2009;38(4):663-672.

Saborio P, Tipton GA, Chan JC: Diabetes insipidus. Pediatr Rev 2000;21(4):122-129.

28. Can children develop metabolic syndrome?

The prevalence of childhood obesity is skyrocketing among our youth and stresses all body systems. Obesity leads to the development of insulin resistance and other endocrine abnormalities that were previously reported only in adults, such as metabolic syndrome. Metabolic syndrome in children is defined as the presence of at least three of the following: obesity, dyslipidemia, hypertension, and impaired glucose tolerance. Other implications of childhood obesity include advanced physical maturation, type 2 diabetes, and polycystic ovary disease.

D'Adamo E, Santoro N, Caprio S: Metabolic syndrome in pediatrics: Old concepts revised, new concepts discussed. Pediatr Clin North Am 2011;58(5):1241-1255.

29. What are the most common symptoms of pheochromocytoma?

Common symptoms are headache, palpitations, and excessive sweating. The headache is usually pounding and severe, and palpitations may be associated with tachycardia. Other symptoms include tremor, fatigue, chest or abdominal pain, and flushing.

FLUIDS AND ELECTROLYTES

Ronald F. Marchese and Kathy N. Shaw

CHAPTER 34

1. How do you estimate the extent of dehydration in children?

Serial weights on the same scale are best but are rarely available in the emergency department (ED) setting. Although not as accurate, a physical examination is used to estimate the degree of fluid loss (Table 34-1).

2. What are the most reliable clinical examination findings that help you predict how dry a child has become?

An abnormal capillary refill time is probably the most reliable sign for predicting dehydration in children. However, a combination of examination signs is better than any individual sign. The presence of two or more of the following four clinical measures seems most reliable in predicting at least 5% dehydration:

- "Ill" general appearance
- Lack of tears
- Capillary refill time at the fingertip longer than 2 seconds
- Dry mucous membranes

Gorelick MH, Shaw KN, Murphy KO: Validity and reliability of clinical signs in the diagnosis of dehydration in children. Pediatrics 1997;99:e6.

Steiner MJ, DeWalt DA, Byerley JS: Is this child dehydrated? JAMA 2004;291:2746-2754.

3. How does the extent of dehydration translate into fluid lost?

For each kilogram of weight loss, 1 L of fluid was lost. Using weight obtained in the ED and your estimate of the percentage of dehydration, you can calculate the "well" or rehydrated weight. The difference between the estimated well weight and current weight is converted into liters or kilograms. For example, a 9-kg ill-appearing baby presents with dry mucous membranes, crying with no tears, and a capillary refill time of longer than 2 seconds. She is estimated to have lost 10% of her body weight (severe dehydration).

To calculate the well weight, divide the current weight by 1 minus the dehydration percentage:

$$\begin{aligned}\text{Calculated well weight} &= \text{current weight}/(1-\text{dehydration percentage})\\ &= 9\,\text{kg}/(1-10\%)\\ &= 9\,\text{kg}/0.9\\ &= 10\,\text{kg}\end{aligned}$$

$$\text{Weight lost} = 10\,\text{kg} - 9\,\text{kg} = 1\,\text{kg} = 1\,\text{L}$$

4. How much fluid and what type do you use for a "bolus" to begin intravenous (IV) hydration?

The first objective in treating a child with dehydration is to restore intravascular volume and treat shock. Use isotonic fluid, such as normal saline or Ringer's lactate. Give boluses of 20 mL/kg and reassess the child. The goal is to restore blood pressure, reduce heart rate, restore perfusion to the tissues (return of capillary refill < 2 seconds), improve mental status or general appearance, and produce urine. If initial fluid boluses greater than 60 mL/kg are needed, reconsider the diagnosis and management plan.

5. Should 5% dextrose in water (D_5W) be used as a fluid bolus for dehydration?

No, D_5W should not be used to treat dehydration. In general, only isotonic crystalloid fluid should be used for treatment of dehydration in pediatrics. There is some evidence that dextrose

Table 34-1. Clinical Findings of Dehydration

SIGNS AND SYMPTOMS	*Degree of Impairment*		
	None or Mild (0-5%)	Moderate (5-10%)	Severe (>10%)
General condition, infants	Thirsty; alert; restless	Lethargic or drowsy	Limp; cold, cyanotic extremities; may be comatose
General condition, older children	Thirsty; alert; restless	Alert; postural dizziness	Apprehensive; cold, cyanotic extremities; muscle cramps
Quality of radial pulse	Normal	Thready or weak	Feeble or impalpable
Quality of respiration	Normal	Deep	Deep and rapid
Skin elasticity	Pinch retracts immediately	Pinch retracts slowly	Pinch retracts very slowly (>2 sec)
Eyes	Normal	Sunken	Very sunken
Tears	Present	Absent	Absent
Mucous membranes	Moist	Dry	Very dry
Urine output (by report of parent)	Normal	Reduced	None passed in many hours

Adapted from World Health Organization: The Treatment of Diarrhea: A Manual for Physicians and Other Senior Health Workers, 3rd ed. Washington, DC, Division of Diarrheal and Acute Respiratory Disease Control, World Health Organization. 1995.

containing normal saline boluses decreases the level of serum ketones compared to normal saline boluses without dextrose.

Levy JA, Bachur RG, Monuteux MC, et al: Intravenous dextrose for children with gastroenteritis and dehydration: A double-blind randomized controlled trial. Ann Emerg Med 2013;61:281-288.

6. Are there any children who should not receive a rapid fluid bolus?

Children with diabetic ketoacidosis or hypernatremic dehydration (serum sodium level > 150 mEq/dL) who are in a hyperosmolar state require slower and more cautious fluid resuscitation. Clearly, uncompensated shock (hypotension) must be treated rapidly, but use 10-mL/kg boluses of normal saline and reassess the child after each bolus. Also administer fluids more cautiously to children in cardiogenic shock (heart failure).

7. What stock should be used after the initial IV bolus or if the child is not dehydrated?

Traditionally, it has been recommended to use 5% dextrose in one-half (0.5) normal saline (D_5 0.5NS), with 20 mEq/L potassium chloride added if the child has urinated. For infants less than 10 kg, use D_5 0.25NS, with 10 mEq/L potassium chloride added if the child has urinated. Recently, it has been shown that hypotonic maintenance fluids are associated with the risk of hyponatremia. Therefore, some recommend giving isotonic fluid (IV normal saline) for maintenance fluids to hospitalized children. Children with burns, pyloric stenosis with hypochloremia, diabetic ketoacidosis, or critical illness are usually kept on a normal saline infusion in the ED.

Foster BA, Tom D, Hill V: Hypotonic versus isotonic fluids in hospitalized children: A systemic review and meta-analysis. J Pediatr 2014;165:163-169.

Wang J, Xu E, Xiao Y: Isotonic versus hypotonic maintenance IV fluids in hospitalized children: A meta-analysis. Pediatr 2014;133:105-113.

8. At what rate should fluids run on a child who has orders to receive nothing by mouth? How is this maintenance rate adjusted for the dehydrated child?

All children who are unable to drink should receive maintenance fluids, and if they are dehydrated, the rate is higher to replace some of the remaining fluid deficit. Calculate all rates by using the child's well or rehydrated weight. Increase rates above maintenance if the child is febrile or has increased insensible or gastrointestinal losses.

$$\begin{aligned}\text{Maintenance rate} = \ & 4\,\text{mL/kg/hour for the first 10 kg} \\ & + 2\,\text{mL/kg/hour for the second 10 kg} \\ & + 1\,\text{mL/kg/hour for each kg over 20}\end{aligned}$$

Example: Maintenance rate for a 16-kg child: 40 mL/hour (first 10 kg × 4 mL/hour) + 12 mL/hour (next 6 kg × 2 mL/hour) = 52 mL/hour (if the child is febrile, add 10% more, or 55-60 mL/hour). To determine the rate for the dehydrated child, half of the total fluid deficit (minus the fluid boluses already given) is added to the maintenance rate for the first 8 hours. The other half of the deficit is added to the maintenance rate over the next 16 hours (hopefully outside the ED!). For children with hypertonic dehydration, the remaining fluid deficit after initial boluses is replaced evenly over the next 48 hours.

Example: A 9-kg dehydrated baby was given 400 mL of NS (40 mL/kg) as an initial fluid bolus. The fluid deficit was 1000 mL. Half of the deficit is 500 mL. Because 400 mL was already given, 100 mL of the deficit should be added to the maintenance rate over the next 8 hours. Thus, 100 mL/8 hours = 12.5 mL/hour should be added to the maintenance rate of 40 mL/hour (based on 10-kg "well" weight), or 52 mL/hour of D_5 0.25NS should be ordered.

Shaw KN, Spandorfer P: Dehydration. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 206-211.

9. Will use of hypotonic maintenance solutions cause dangerous hyponatremia?

Most children admitted with fever and volume depletion require free water. However, some children are at risk for secretion of antidiuretic hormone (ADH), which may cause retention of free water and hyponatremia. Conditions that have been associated with this state include bronchiolitis, meningitis, and pneumonia, as well as perioperative and postoperative states. These children may require fluid restriction. In children with electrolyte abnormalities or prolonged IV fluid administration, serial electrolytes and urine output are monitored and fluid rates adjusted on the basis of the child's hydration status, urine output, and presence or absence of increased ADH. Keeping children on isotonic parenteral maintenance solution may cause hypernatremia and may not replace insensible water loss, whereas hypotonic fluids may cause acute hyponatremia and encephalopathy. Therefore, individualize the IV fluid rate and composition on a case-by-case basis.

Hatherill M: Rubbing salt in the wound. Arch Dis Child 2004;89:414-418.
Moritz ML, Ayus JC: Prevention of hospital-acquired hyponatremia: Do we have the answers? Pediatrics 2011;128:980-983.

10. Which children may be treated with oral rehydration?

Strongly consider oral rehydration in all children with mild or moderate dehydration who do not have uncompensated shock, severe vomiting, high stool output of more than 20 mL/kg/hour, or poor adherence. This is very effective in treating dehydration and has a low failure rate.

Centers for Disease Control and Prevention: Managing acute gastroenteritis in children. MMWR Morb Mortal Wkly Rep 2003;52:1-16.

11. Then why aren't more children treated orally rather than with IV fluids?

Oral rehydration therapy (ORT) takes time, one-to-one care, and patience. Many parents bring the child to the ED because they want a "quick fix." Nevertheless, recent studies have shown that ORT is as effective as IV rehydration and may require less time and have a lower complication rate.

Hartling L, Bellemare S, Wiebe N, et al: Oral versus intravenous rehydration for treating dehydration due to gastroenteritis in children. Cochrane Database Syst Rev 2006;(3):CD004390.
Spandorfer PR, Alessandrini EA, Joffe MD, et al: Oral versus intravenous rehydration of moderately dehydrated children: A randomized, controlled trial. Pediatrics 2005;115:295-301.

Key Points: Dehydration

1. The first objective in treating a child with severe dehydration is to restore intravascular volume and treat shock, regardless of cause.
2. In children with electrolyte abnormalities or prolonged IV fluid administration, serial electrolytes and urine output are monitored and fluid rates adjusted on the basis of the child's hydration status, urine output, and presence or absence of increased ADH.
3. Compared with IV therapy, ORT is effective in treating dehydration in most children, has fewer complications, and may be faster.

12. How do you calculate the amount of fluid to give for oral rehydration?

Calculate the fluid deficit based on the estimate of the degree of dehydration, and give *the entire deficit by mouth over 4 hours*. Ask the parent to offer a small amount of fluid every 5 to 10 minutes. For mild to moderate dehydration, give 1 mL/kg of an oral electrolyte solution every 5 minutes. For moderate to severe dehydration, give 1 to 2 mL/kg by mouth every 5 minutes.

Example: A 9-kg moderately dehydrated baby with an estimated "well" weight of 10 kg has a fluid deficit of 1000 mL. We ask the mother to insert via syringe 20 mL (2 mL/kg) into the baby's mouth every 5 minutes for the next 4 hours (1000 mL/4 hours = 250 mL/hour; 250 mL/60 minutes = ~4 mL/minute).

13. What type of fluid should be used for oral rehydration?

Use an oral rehydration solution (ORS). These solutions have the correct proportion of dextrose and salt to allow for maximal absorption of electrolytes. Although solutions with higher concentrations of sodium (75-90 mEq/dL) are recommended initially (Rehydralyte [manufactured by Ross], ORS packets [Jaianas]), solutions with a lower sodium content (40-60 mEq/dL) may be used and are more readily available (Pedialyte [Ross], Infalyte [Mead Johnson]).

14. Why can't I mix half juice and half rehydration solution to make it taste better?

Juice and soda have high sugar contents, and a 50% mixture raises the sugar content above the amount that allows for maximum sodium/glucose transport across the cell membrane in the gastrointestinal tract. Use flavored solutions from the manufacturer or add only a small amount of juice (1:8 solution) to the rehydration solution.

15. When should oral rehydration not be used?

- Severe dehydration
- Inability to tolerate oral fluids because of vomiting
- Altered mental status with risk of aspiration
- Ileus
- Short gut or other conditions with carbohydrate malabsorption

Batra B, Stanton B: Oral rehydration therapy. UpToDate, 2006. Available at www.uptodate.com.

16. Are there alternatives to ORT and IV therapy?

There is some evidence to suggest that recombinant human hyaluronidase-facilitated subcutaneous rehydration may be a reasonable alternative therapy in mild to moderately dehydrated children, especially with failed ORT or with difficult IV access.

Spandorfer PR, Mace SE, Okada PJ, et al: A randomized clinical trial of recombinant human hyaluronidase-facilitated subcutaneous versus intravenous rehydration in mild to moderately dehydrated children in the emergency department. Clin Ther 2012;34(11):2232-2245.

17. When should ondansetron be used?

An antiemetic such as ondansetron, a serotonin 5-HT_3 selective receptor antagonist, may be useful for children with moderate or severe dehydration with severe nausea and persistent vomiting. Ondansetron can be administered orally, as an oral disintegrating tablet, or intravenously to reduce vomiting and improve a child's ability to maintain oral hydration. Oral ondansetron has been shown to improve the success rate of ORT in children with gastroenteritis.

Freedman SB, Adler M, Seshadri R, et al: Oral ondansetron for gastroenteritis in a pediatric emergency department. N Engl J Med 2006;354(16):1698-1705.

18. What concerns should I have about a child with hypertonic dehydration?

The deficit in children with hypertonic dehydration may be underestimated. They appear less dry because their intravascular space is maintained longer. Additionally, rapid rehydration can cause cerebral edema, hemorrhage, or thrombosis. Hyperglycemia or hypocalcemia may also occur.

19. How do I treat hyponatremia?

Treat the patient, not the laboratory value. Treat children with seizures, severe lethargy, hypoventilation, coma, or shock immediately. For water-intoxicated children or those with the syndrome of inappropriate ADH secretion—in whom sodium, not fluid, is needed—give 2 to 4 mL/kg of 3% saline to stop seizures, followed by 6 to 12 mL/kg of 3% saline over the next 2 to 4 hours. For children with dehydration and low serum sodium, a fluid bolus of 20 to 40 mL/kg NS quickly corrects symptomatic hyponatremia.

20. How do I treat hypoglycemia?

A 0.5-g/kg bolus is adequate to treat most cases of hypoglycemia. This can be given as 5 mL/kg of D_{10} (dextrose in water) or 2 mL/kg of D_{25}. D_{50} is not used in children.

21. How and when do I treat metabolic acidosis?

Give sodium bicarbonate to children with a pH of less than 7.1 who are critically ill or are unable to compensate. Most children with dehydration whose serum bicarbonate level is more than 8 correct their metabolic acidosis with fluid resuscitation and do not require bicarbonate. Bicarbonate should be given only for metabolic acidosis, not respiratory acidosis. Children with respiratory insufficiency should not receive bicarbonate. Bicarbonate does not cross the blood-brain barrier; however, its byproduct, carbon dioxide, does cross this barrier. Increases in carbon dioxide may cause cerebral acidosis, and therefore bicarbonate should be given slowly. Usually 1 to 2 mEq/kg is given over a minimum of 30 to 60 minutes. Administration of bicarbonate is the only treatment factor found to be associated with development of cerebral edema in patients with diabetic ketoacidosis.

Glaser N, Barnett P, McCaslin I, et al, for the Pediatric Emergency Medicine Collaborative Research Committee of the American Academy of Pediatrics: Risk factors for cerebral edema in children with diabetic ketoacidosis. N Engl J Med 2001;344:264-269.

22. How do I treat hypercalcemia?

Give furosemide, 1 to 2 mg/kg, and address the cause.

23. How do I treat hyperkalemia?

The type and speed of treatment depend on the potassium levels and electrocardiogram changes. Potassium may be forced out of the cell by acidosis. Treatment of the acidosis causes potassium to return to the cell and out of the serum (Table 34-2).

Key Points: Acidosis/Hyperkalemia

1. Beware of bicarbonate—especially in the treatment of diabetic ketoacidosis—because it has been associated with cerebral edema.
2. Treat hyperkalemia on the basis of the electrocardiogram, clinical picture, and cause, not just the serum value.

24. How do I treat hypocalcemia?

First, check the ionized calcium and confirm the diagnosis. Calcium gluconate, 10% solution, can be given via peripheral IV route and is preferred. For patients with cardiac disturbance, treat with 50 to 100 mg/kg calcium gluconate per dose infused over 3 to 5 minutes. For patients with tetany, treat with 100 to 200 mg/kg calcium gluconate per dose infused over 5 to 10 minutes.

Rodig, NM: Renal and electrolyte emergencies. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 1099-1126.

Table 34-2. Treatment of Hyperkalemia

POTASSIUM LEVEL (MEQ/DL)	ECG FINDING	TREATMENT
<7.0	Peaked T waves only, or normal	Remove potassium source, treat acidosis with Kayexalate (1 g/kg orally or rectally) every 4-6 hr
7.0	Widespread ECG changes without arrhythmia	Glucose (0.5 g/kg or 5 mL/kg of D_{10} over 30-60 min) *and* insulin (0.1 U/kg over 30-60 min) *plus* bicarbonate (2 mEq/kg over 30-60 min)
8.0	Arrhythmia	10% calcium gluconate (0.5 mL/kg over 2-5 min with ECG monitoring; discontinue if heart rate <100 beats/min) *plus* glucose *and* insulin, bicarbonate as earlier

ECG, electrocardiogram.

GASTROINTESTINAL EMERGENCIES

Susan Fuchs

CHAPTER 35

1. What is the most common cause of vomiting in older children?

Acute gastroenteritis. Although usually associated with diarrhea, vomiting can occur alone in the early stages of gastroenteritis. The most common infectious viral cause is rotavirus. *Salmonella, Shigella, Yersinia, Campylobacter* spp., and *Escherichia coli* can also cause bacterial gastroenteritis with vomiting.

Stevens MW, Henretig FM: Vomiting. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 617-625.

2. What are other medical causes of vomiting in children?

Metabolic disorders, such as galactosemia, fructose intolerance, and amino acid or organic acid defects (phenylketonuria, urea cycle defects) usually present in infancy. Along with vomiting, symptoms such as lethargy, seizures, or coma may occur. Diabetes mellitus can cause vomiting because of ketoacidosis or slowed gastric motility (gastroparesis may occur after 10 years of diabetes mellitus). Other causes include milk/soy protein allergies, pancreatitis, urinary tract infection, neurologic disorders associated with increased intracranial pressure (tumor, hydrocephalus), migraines, pregnancy, and psychological causes (rumination, bulimia).

Stevens MW, Henretig FM: Vomiting. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 617-625.

3. Where are you most likely to find an ingested coin that has not passed?

In the esophagus. The cricopharyngeus/thoracic inlet (proximal third) is the most common place, with 60% to 70% lodged here. The level of the aortic arch (middle third) accounts for 10% to 20%, and the gastroesophageal junction/sphincter (lower third) accounts for the other 20%. Other areas of the gastrointestinal (GI) tract where the coin may be found are the stomach, pylorus, ligament of Treitz, and ileocecal valve. Objects larger than 2 cm in diameter or longer than 5 cm are more likely to get "caught" at the pylorus.

Kay M, Wyllie R: Foreign bodies and bezoars. In Liacouras CA, Piccoli DA (eds): Pediatric Gastroenterology—The Requisites in Pediatrics. Philadelphia, Mosby/Elsevier, 2008, pp 64-73.

4. What is the best way to remove an esophageal coin?

This is a highly controversial topic. When the foreign body has been lodged for less than 24 hours, no respiratory symptoms are present, and the child has no prior esophageal disease or surgery, 80% to 90% will pass spontaneously. About 10% to 20% require endoscopic removal, and less than 1% require surgery. There are several methods of coin removal in infants and children with no respiratory symptoms and no history of esophageal disease. Some advocate the use of Foley catheter extraction under fluoroscopic guidance for coins that have been lodged less than 24 hours. Others use esophageal bougienage for coins lodged in the distal esophagus less than 24 hours. Laryngoscopy with Magill forceps removal, esophagoscopy, or flexible or rigid endoscopy under general anesthesia are also options. In the presence of respiratory symptoms, a history of esophageal disease, esophageal edema, or focal narrowing of the adjacent trachea (seen on the lateral neck radiograph), the best way to remove the foreign body is under direct vision or flexible endoscopy. The choice of method is often based upon the expertise and availability of the specialists (radiology, otolaryngology, surgery, or gastroenterology) at a particular hospital.

Fun fact: A dime is 18 mm, a nickel 21 mm, and a quarter 24 mm in diameter.

Gilger MA, Jain AK, McOmber ME: Foreign bodies of the esophagus and gastrointestinal tract in children. UpToDate, 2013. Available at www.uptodate.com.

Kay M, Wyllie R: Foreign bodies and bezoars. In Liacouras CA, Piccoli DA (eds): Pediatric Gastroenterology—The Requisites in Pediatrics. Philadelphia, Mosby/Elsevier, 2008, pp 64-73.
Sinclair K, Hill ID: Button and cylindrical batteries. UpToDate, 2013. Available at www.uptodate.com.

5. What is the "big deal" about button batteries?

Although they are small (watch or hearing aid batteries), there is a risk of leakage of battery contents, electrical discharge, and pressure necrosis. If the battery remains in the esophagus, there is fear of tissue damage, with resultant erosions, tracheoesophageal fistulas, and strictures. Batteries larger than 12 mm are most likely to become lodged in the esophagus.

6. How should an ingestion of a button battery be managed?

When a child swallows a button battery, obtain a radiograph to determine the battery's location. On the lateral radiograph, batteries have a step-off (coins do not, but two coins could look like a battery). On anteroposterior (AP) radiographs, batteries have a double-ring or halo effect (coins do not). Button batteries in the esophagus should be immediately removed under direct visualization to assess for any damage to the esophageal mucosa. Pressure necrosis can result when the battery places pressure on surrounding tissue, leading to inflammation and irritation, which can lead to tissue necrosis. Lithium batteries tend to be larger ($\geq$ 20 mm) and have the most complications.

If the battery is in the stomach, which is acidic, the seal of the battery may erode and potentially release alkali solutions that can cause liquefaction necrosis of the stomach mucosa. If there are no symptoms of GI injury (pain, hematemesis, hematochezia, obstruction), the patient can be observed at home after initial evaluation. Obtain follow-up radiographs to look for passage in 4 days if the battery diameter is 15 mm or larger and the child is less than 4 years old, or in 1 to 2 weeks otherwise, if the battery has not been seen in the stool.

Fun fact: Many batteries are made of mercuric oxide. There is little risk of mercury poisoning, however, because mercuric oxide is poorly absorbed from the GI tract.

Fun fact: Battery identification can provide the chemical content, diameter, and height of the battery. The battery code uses letters followed by three or four numbers. The letters identify the chemical content (e.g., S, silver oxide; G, copper oxide; LR or AG, alkaline; CR, lithium/manganese oxide; BR, lithium carbon monofluoride). The three-number codes give the diameter (mm) in the first number and the height in tenths of millimeters in the last two. If there are four numbers, the first two are the diameter, then the last two are the height. For example, S712 is a silver oxide battery that is 7 mm in diameter and 1.2 mm high.

Sinclair K, Hill ID: Button and cylindrical battery ingestion. UpToDate, 2013. Available at www.uptodate.com.

7. How should a caustic ingestion be managed?

First, ensure that there is a patent airway. Next, determine what was ingested. This may require calling the poison control center (1-800-222-1222) or going to a computer-based system that determines treatment, signs/symptoms, and toxicity in the emergency department. The child may have symptoms such as drooling, mouth pain, pain on swallowing, chest pain, or cough. Examine the mouth for mucosal and oral burns. Alkali ingestions (lye, drain cleaners) are more damaging to the oropharynx and esophagus than are acid burns because they cause deeper injury. In addition, with an alkali ingestion, esophageal burns can occur without an oral burn. The amount of caustic agent ingested does not always correlate with the degree of burns or the symptoms. Induced vomiting is contraindicated in these children because of the risk of injury and aspiration. After a thorough examination, if any burns are seen, or if the child ingested alkali, the child should undergo endoscopy under general anesthesia. If there are no burns and no symptoms, and if a weak or very dilute formulation of an acid was ingested, observation is indicated.

Erickson TB: Toxicology: Ingestions and smoke inhalation. In Fuchs S, Yamamoto L (eds): APLS: The Pediatric Emergency Medicine Resource, 5th ed. Burlington, MA, Jones & Bartlett Learning, 2012. Available at APLSonline.com.

8. If a child swallowed a magnet, should I be worried?

Yes. There is a high rate of complications from magnet ingestions. Although the Consumer Product Safety Commission (CPSC) raised the recommended age for magnetic

children's toys to 6 years in 2006, the emergence of "rare earth" magnets in adult toys, cordless tools, and other household products has led to more cases of magnet ingestion. These magnets are made of neodymium, iron, and boron and are 5 to 10 times stronger than traditional magnets. Although the CPSC issued a ban on the sale of these magnets for children's use in 2009, a study using CPSC and the National Electronic Injury Surveillance System (NEISS) data demonstrated an increasing number of magnet ingestions between 2002 and 2011.

The main concern is that loops of bowel can be trapped between two magnets, or on a magnet and another ingested metal object, leading to intestinal obstruction, perforation, fistula formation, and even death. Even if a magnet is removed promptly (within 8 hours), ulcerations can occur. Obtain abdominal and chest radiographs in two views (AP[chest]/flat plate[abdomen] and lateral) to determine the number and position of the magnets. A concern is that radiographs do not reliably distinguish between one and two magnets.

Abbas MI, Oliva-Hemker M, Choi J, et al: Magnet ingestions in children presenting to US emergency departments, 2002-2011. J Pediatr Gastroenterol Nutr 2013;57:18-22.

Gilger MA, Jain AK, McOmber ME: Foreign bodies of the esophagus and gastrointestinal tract in children. UpToDate, 2013. Available at www.uptodate.com.

Hussain SZ, Bousvaros A, Gilger M, et al: Management of ingested magnets in children. J Pediatr Gastroenterol Nutr 2012;55:239-242.

Midgett J, Inkster S, Rauschschwalbe R, et al: Consumer Product Safety Commission: Gastrointestinal injuries from magnet ingestion in children—United States, 2003-2006. MMWR Morbid Mortal Wkly Rep 2006;55(48):1296-1300.

Otjen JP, Rohrmann CA, Iyer RS: Imaging pediatric magnet ingestion with surgical pathological correlation. Pediatri Radiol 2013;43:851-859.

9. Do all magnets need to be removed?

If there are multiple magnets, all within the esophagus or stomach, the magnets should be removed, especially if it has been less than 12 hours since ingestion. Endoscopic removal can be performed by a pediatric gastroenterologist or a pediatric specialist. There is concern that after this time, the risk of complications is increased, so consult a pediatric surgeon as well. If the magnets are beyond the stomach, consult a pediatric gastroenterologist and pediatric surgeon. If the patient is symptomatic (vomiting, abdominal pain or cramps, abdominal distention), consult a pediatric surgeon to remove the magnets. If asymptomatic, removal by enteroscopy or colonoscopy is an option. Another option is serial radiographs every 4 to 6 hours, as long as there are no signs of bowel obstruction or perforation on radiograph, until passage is confirmed.

In 2012, the North American Society of Pediatric Gastroenterology, Hepatology, and Nutrition (NASPGHAN) developed a rare earth magnet ingestion algorithm. For a single magnet, consider endoscopic removal. Following the patient with serial radiographs to confirm passage, as an outpatient, is another option. Keep clothes with metallic buttons, belts with buckles, and any other magnets or metal objects away from the child so they will not be ingested.

Gilger MA, Jain AK, McOmber ME: Foreign bodies of the esophagus and gastrointestinal tract in children. UpToDate, 2013. Available at www.uptodate.com.

Hussain SZ, Bousvaros A, Gilger M, et al: Management of ingested magnets in children. J Pediatr Gastroenterol Nutr 2012;55:239-242.

10. Distinguish among hematemesis, hematochezia, and melena.

- Hematemesis is the vomiting of bright red or denatured blood ("coffee-ground" appearance). The source of the blood is proximal to the ligament of Treitz.
- Hematochezia is bright red blood or maroon-colored blood or stools per rectum and implies a lower GI source (distal to the ligament of Treitz).
- Melena is the rectal passage of black tarry stools (black from the bacterial breakdown of blood); the source is proximal to the ileocecal valve.

Kamath BM, Mamula P: Gastrointestinal bleeding. In Liacouras CA, Piccoli DA (eds): Pediatric Gastroenterology—The Requisites in Pediatrics. Philadelphia, Mosby/Elsevier, 2008, pp 87-97.

Ramsook C, Endom EE: Diagnostic approach to lower gastrointestinal bleeding in children. UpToDate, 2013. Available at www.uptodate.com.

Villa X: Approach to gastrointestinal bleeding in children. UpToDate, 2013. Available at www.uptodate.com.

11. Distinguish between upper GI and lower GI bleeding in terms of site of bleeding.

- Upper GI bleeding occurs when the site of bleeding is proximal to the ligament of Treitz or the second portion of the duodenum.
- Lower GI bleeding implies a site distal to the ligament of Treitz, or those structures supplied by mesenteric vessels.

Kamath BM, Mamula P: Gastrointestinal bleeding. In Liacouras CA, Piccoli DA (eds): Pediatric Gastroenterology—The Requisites in Pediatrics. Philadelphia, Mosby/Elsevier, 2008, pp 87-97.
Ramsook C, Endom EE: Diagnostic approach to lower gastrointestinal bleeding in children. UpToDate, 2013. Available at www.uptodate.com.
Villa X: Approach to gastrointestinal bleeding in children. UpToDate, 2013. Available at www.uptodate.com.

12. What are some of the common tests used to determine the presence of blood? What causes them to be falsely positive or falsely negative?

The Hematest is a qualitative stool test that uses orthotolidine (a leukodye). False-positive results occur when the patient has ingested red meats, iron preparations, or plant peroxidases (found in horseradish, turnips, tomatoes, and fresh red cherries). False-negative results occur in the presence of ascorbic acid (vitamin C). The Hemoccult test is a qualitative stool test that uses guaiac (also a leukodye). False-positive results occur with red meats, broccoli, grapes, cauliflower, cantaloupe, turnips, iron preparations, plant peroxidases, and cimetidine. False-negative results occur with hard stool, penicillamine, antacids, and ascorbic acid. The HemoQuant test is the only quantitative test (2 mg hemoglobin per gram of stool) and uses a fluorescent antibody to porphyrin. It is falsely positive with red meat. Gastroccult tests check gastric fluids for blood.

Cadranel S, Scaillon M: Approach to gastrointestinal bleeding. In Guandalini S (ed): Textbook of Pediatric Gastroenterology and Nutrition. London, Taylor & Francis, 2004, pp 639-654.
Kamath BM, Mamula P: Gastrointestinal bleeding. In Liacouras CA, Piccoli DA (eds): Pediatric Gastroenterology—The Requisites in Pediatrics. Philadelphia, Mosby/Elsevier, 2008, pp 87-97.
Ramsook C, Endom EE: Diagnostic approach to lower gastrointestinal bleeding in children. UpToDate, 2013. Available at www.uptodate.com.

13. What ingested foods can cause red stools?

Fruit punch, Kool-Aid beverage (especially cherry or strawberry), red beets, tomatoes, and gelatin. Note that commercial dyes can also be found in breakfast cereals. *Serratia marcescens* in stool can cause red coloring in diapers.

Cadranel S, Scaillon M: Approach to gastrointestinal bleeding. In Guandalini S (ed): Textbook of Pediatric Gastroenterology and Nutrition. London, Taylor & Francis, 2004, pp 639-654.
Kamath BM, Mamula P: Gastrointestinal bleeding. In Liacouras CA, Piccoli DA (eds): Pediatric Gastroenterology—The Requisites in Pediatrics. Philadelphia, Mosby/Elsevier, 2008, pp 87-97.
Ramsook C, Endom EE: Diagnostic approach to lower gastrointestinal bleeding in children. UpToDate, 2013. Available at www.uptodate.com.

14. What foods or items can cause black stools?

Bismuth (Pepto-Bismol), iron preparations, charcoal, licorice, beets, spinach, blueberries, grape juice, dark chocolate, and swallowed blood.

Cadranel S, Scaillon M: Approach to gastrointestinal bleeding. In Guandalini S (ed): Textbook of Pediatric Gastroenterology and Nutrition. London, Taylor & Francis, 2004, pp 639-654.

15. What is the APT-Downey test? How is it used?

This test is for fetal red blood cells and is used when an infant spits up blood. If the infant is breast-feeding, it is often unclear if the blood is from the baby's GI tract or maternal milk. Adult hemoglobin is denatured to alkaline globin hematin in an alkaline solution (sodium hydroxide), resulting in a yellow-brown color. Fetal hemoglobin resists the effect of alkali and stays pink. However, if the blood you use for the test is coffee-ground color (not red), it will be read falsely as adult blood.

Cadranel S, Scaillon M: Approach to gastrointestinal bleeding. In Guandalini S (ed): Textbook of Pediatric Gastroenterology and Nutrition. London, Taylor & Francis, 2004, pp 639-654.
Villa X: Approach to gastrointestinal bleeding in children. UpToDate, 2013. Available at www.uptodate.com.

16. What are some common causes of upper GI bleeding in infants and children?

- **Neonates:** Swallowed maternal blood, esophagitis, coagulopathy, sepsis, gastritis (stress ulcer)
- **Infants (age 1-12 months):** Gastritis, esophagitis, Mallory-Weiss tear, duplication
- **Children (age 1-12 years):** Epistaxis, esophagitis, gastritis, ulcers, Mallory-Weiss tear, esophageal varices, toxic ingestion
- **Adolescents:** Ulcers, esophagitis, varices, gastritis, Mallory-Weiss tear, toxic ingestion

Kamath BM, Mamula P: Gastrointestinal bleeding. In Liacouras CA, Piccoli DA (eds): Pediatric Gastroenterology—The Requisites in Pediatrics. Philadelphia, Mosby/Elsevier, 2008, pp 87-97.
Stevens MW, Henretig FM: Vomiting. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 617-625.
Villa X: Approach to gastrointestinal bleeding in children. UpToDate, 2013. Available at www.uptodate.com.

17. What are some common causes of lower GI bleeding in infants and children?

- **Neonates:** Swallowed maternal blood, anorectal lesions, milk- or soy-induced colitis, necrotizing enterocolitis, midgut volvulus, Hirschsprung's disease
- **Infants (age 1-12 months):** Anal fissure, midgut volvulus, intussusception, Meckel's diverticulum, infectious diarrhea, milk- or soy-induced colitis
- **Children (age 1-12 years):** Anal fissures, polyps, Meckel's diverticulum, intussusception, infectious diarrhea, inflammatory bowel disease, duplications, hemangiomas, Henoch-Schönlein (HSP) purpura, hemolytic uremic syndrome (HUS)
- **Adolescents:** Inflammatory bowel disease (ulcerative colitis, Crohn's disease), polyps, hemorrhoids, anal fissure, infectious diarrhea

Kamath BM, Mamula P: Gastrointestinal bleeding. In Liacouras CA, Piccoli DA (eds): Pediatric Gastroenterology—The Requisites in Pediatrics. Philadelphia, Mosby/Elsevier, 2008, pp 87-97.
Ramsook C, Endom EE: Diagnostic approach to lower gastrointestinal bleeding in children. UpToDate, 2013. Available at www.uptodate.com.
Stevens MW, Henretig FM: Vomiting. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 617-625.

18. Distinguish between malrotation and volvulus.

Malrotation is the incomplete or reverse rotation of the embryonic midgut about the superior mesenteric artery. The small bowel then hangs on a mesenteric stalk. Volvulus occurs when the midgut twists around the stalk, resulting in small bowel obstruction and vascular compromise due to compression of the mesenteric vessels. Midgut volvulus occurs in 70% of infants with malrotation.

Okada PJ, Hicks BA: Nontraumatic surgical emergencies. In Fuchs S, Yamamoto L (eds): APLS: The Pediatric Emergency Medicine Resource, 5th ed. Burlington, MA, Jones & Bartlett Learning, 2012, pp 298-359.

19. What are the clinical features of malrotation with volvulus?

An infant with malrotation and volvulus is usually less than 1 month of age. Symptoms include bilious vomiting, irritability, and abdominal pain. When volvulus is present, there are bloody or heme-positive stools. The infant can rapidly progress to a shock-like state. The physical examination reveals abdominal distention (from dilated loops of bowel), lethargy, pallor, and possibly hypotension. Therapy includes fluid resuscitation, nasogastric tube, broad-spectrum antibiotics, and emergent surgery (Ladd's procedure).

Brandt ML: Intestinal malrotation. UpToDate, 2013. Available at www.uptodate.com.
Okada PJ, Hicks BA: Nontraumatic surgical emergencies. In Fuchs S, Yamamoto L (eds): APLS: The Pediatric Emergency Medicine Resource, 5th ed. Burlington, MA, Jones & Bartlett Learning, 2012, pp 298-359.

20. What is the radiographic finding in an infant with malrotation and volvulus?

Abdominal radiographs may be normal (50-60% of patients with malrotation and volvulus) or show duodenal obstruction ("double bubble"). The upper GI study shows a displaced duodenal-jejunal junction (both to the right of the spine), with the jejunum on the right side of the abdomen. There may be a corkscrew appearance of contrast material. A barium enema may show a displaced cecum, usually in the right upper quadrant, or an abnormally oriented superior mesenteric artery.

An ultrasound may be useful to diagnose malrotation, but a normal ultrasound does not exclude malrotation. Findings may include an abnormal position of the superior mesenteric vein, a dilated duodenum, the third part of the duodenum not in the normal position, or the "whirlpool sign" of volvulus (due to vessels twisting).

Brandt ML: Intestinal malrotation. UpToDate, 2013. Available at www.uptodate.com.
Okada PJ, Hicks BA: Nontraumatic surgical emergencies. In Fuchs S, Yamamoto L (eds): APLS: The Pediatric Emergency Medicine Resource, 5th ed. Burlington, MA, Jones & Bartlett Learning, 2012, pp 298-359.

Key Points: Etiology of Vomiting

1. The most common cause of vomiting in older children is acute gastroenteritis.
2. Vomiting can occur from extra-GI problems, such as infections (meningitis, urinary tract infections), metabolic disorders (inborn errors of metabolism, diabetic ketoacidosis), drugs/toxins (iron, lead), and pregnancy.
3. An infant under 1 month of age with bilious vomiting and abdominal pain and distention has malrotation (with or without volvulus) until proved otherwise.

21. Is a "currant jelly" stool classic for intussusception?

The "currant jelly" stool is classic but not common. Up to 75% of children with intussusception never have visible blood in the stool (although the stool is guaiac-positive). A currant jelly stool is a late finding, because it implies that bowel necrosis has occurred. Intussusception generally occurs in children under 2 years of age, with a peak age range of 5 to 9 months. Classic symptoms occur in 10% of cases and include the sudden onset of severe, intermittent, crampy abdominal pain, with crying and drawing up of legs in episodes every 15 minutes. This is followed by vomiting and the passage of a "currant jelly" stool. There is also a "neurologic presentation," which consists of lethargy followed by brief periods of irritability.

22. Which studies help to diagnose intussusception?

Abdominal radiographs may show a soft tissue mass, a nascence of cecal gas and stool, a target sign (two radiolucent circles over the right kidney), a meniscus or crescent sign (a soft tissue density projecting into the gas of the large bowel), a paucity of bowel gas, or a bowel obstruction. Ultrasound is now the preferred method of diagnosis in many hospitals. The findings include a "bull's-eye" or "coiled spring" appearance, which is the enveloping of the bowel. Color Doppler is useful to see if there is lack of blood flow within the intussusceptum, which indicates ischemia. Definitive diagnosis and treatment in more than 75% of cases is made by barium or air contrast enema (hydrostatic reduction) under fluoroscopic guidance. The most common location for the intussusception is ileocolic. A "lead point" is usually not present in younger children but is somewhat common in older children (e.g., Meckel's diverticulum, duplication, vasculitis due to Henoch-Schönlein purpura).

Kitagawa S, Miqdady M: Intussusception in children. UpToDate, 2013. Available at www.uptodate.com.
Okada PJ, Hicks BA: Nontraumatic surgical emergencies. In Fuchs S, Yamamoto L (eds): APLS: The Pediatric Emergency Medicine Resource, 5th ed. Burlington, MA, Jones & Bartlett Learning, 2012, pp 298-359.

23. What is a Meckel's diverticulum?

Meckel's diverticulum is a remnant of the vitellointestinal or omphalomesenteric duct that may result in painless rectal bleeding. It is present in 2% of the population, is 2 inches long (or less), and is found within 2 feet of the ileocecal valve. It contains ectopic gastric mucosa in 60% to 90% of patients and in almost all of those who have painless rectal

bleeding. (The gastric mucosa can result in an ulcer, which bleeds.) Diagnosis is by a Meckel's scan (technetium-99 m pertechnetate scan). Gastric mucosa concentrates the technetium 99. An intestinal duplication may also contain gastric mucosa and can cause GI bleeding or symptoms of obstruction or intussusception. A duplication occurs most commonly in the ileocecal region but also is found in the distal esophagus, stomach, and duodenum.

Javid P, Pauli EM: Meckel's diverticulum. UpToDate, 2013. Available at www.uptodate.com.

24. A child who has had surgical correction for Hirschsprung's disease presents with fever, abdominal distention, and diarrhea. What is your concern?

This child probably has Hirschsprung's enterocolitis. Enterocolitis is the most common cause of death in children with Hirschsprung's disease. Other complications include persistent obstructive symptoms and fecal incontinence. This patient requires intravenous (IV) fluids, nasogastric drainage, broad-spectrum antibiotics, and decompression of the rectum or colon by rectal stimulation or irrigation.

Staiano A, Quaglietta L, Auricchio R: Hirschsprung's disease and intestinal neuronal dysplasia. In Guandalini S (ed): Textbook of Pediatric Gastroenterology and Nutrition. London, Taylor & Francis, 2004, pp 259-268.

25. True or false: Pain is worse after meals in younger children with ulcers.

False. In most young children with ulcers, pain is not related to meals. In some older children and adolescents, pain may be exacerbated by acidic foods or spicy meals, as in adults. Symptoms in neonates include vomiting; infants may feed poorly or vomit; toddlers may have poorly localized abdominal pain or vomiting; adolescents may have epigastric pain. All age groups can have melena or upper GI bleeding.

Stevens MW, Henretig FM: Vomiting. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 617-625.

26. What is the relationship between *Helicobacter pylori* and gastritis/peptic ulcers in children?

H. pylori is a gram-negative, spiral-shaped bacterium that is the most common cause of peptic ulcers (both duodenal and gastric ulcers).

Gold B: *Helicobacter pylori* infection. In Liacouras CA, Piccoli DA (eds): Pediatric Gastroenterology—The Requisites in Pediatrics. Philadelphia, Mosby/Elsevier, 2008, pp 98-113.

27. What is the best way to determine the presence of *H. pylori*?

Diagnosis can be made by invasive testing as well as noninvasive testing. Invasive testing by biopsy is the gold standard. The tissue can be tested for urease activity, can undergo histologic evaluation, and can be sent for bacterial culture. Noninvasive tests have been gaining in popularity as their sensitivity and specificity have improved. The ^{13}C-urea breath test is based upon the hydrolysis of urea to CO_2 and ammonia by *H. pylori* and can be used for diagnosis in children of all ages. Serologic testing to detect IgG antibodies has been done but is limited by variable specificity. Laboratory-based and rapid stool antigen assays have been used to detect the presence of *H. pylori* in the stool of infected patients and have been used for diagnosis as well as eradication of *H. pylori*.

Crowe SE: Indications and diagnostic tests for *Helicobacter pylori* infection. UpToDate, 2013. Available at www.uptodate.com.

Gold B: *Helicobacter pylori* infection. In Liacouras CA, Piccoli DA (eds): Pediatric Gastroenterology—The Requisites in Pediatrics. Philadelphia, Mosby/Elsevier, 2008, pp 98-113.

28. What is the therapy for *H. pylori* infection in children?

Eradication therapy is recommended for children with a definitive peptic ulcer and *H. pylori* on histologic examination. Triple therapy is recommended and includes the following first-line therapies in a twice-daily fashion for 14 days:

- Amoxicillin, 50 mg/kg/day (up to 1 g twice daily)
- Clarithromycin, 15 mg/kg/day (up to 500 mg twice daily)
- Proton-pump inhibitor (e.g., omeprazole), 1 mg/kg/day (up to 20 mg twice daily)

One can substitute metronidazole, 20 mg/kg/day (up to 500 mg twice daily), for clarithromycin, or use clarithromycin, metronidazole, and omeprazole.

Crowe SE: Indications and diagnostic tests for *Helicobacter pylori* infection. UpToDate, 2013. Available at www.uptodate.com.

DeGiacomo C: *Helicobacter pylori* gastritis and peptic ulcer disease. In Guandalini S (ed): Textbook of Pediatric Gastroenterology and Nutrition. London, Taylor & Francis, 2004, pp 73-93.

Gold B: *Helicobacter pylori* infection. In Liacouras CA, Piccoli DA (eds): Pediatric Gastroenterology—The Requisites in Pediatrics. Philadelphia, Mosby/Elsevier, 2008, pp 98-113.

Key Points: Gastritis/Peptic Ulcers and *H. pylori*

1. *H. pylori* is associated with gastric and duodenal ulcers in children.
2. Endoscopy with biopsy is the preferred method for diagnosis. The urea breath test can be used as a noninvasive test in children.
3. *H. pylori* is an infrequent cause of recurrent abdominal pain in children.
4. Treatment is indicated in symptomatic patients with proven *H. pylori* infection.

29. Describe some of the anatomic and histologic differences between ulcerative colitis and Crohn's disease.

- **Ulcerative colitis:** Inflammation of the mucosa and submucosa is limited to the colon and rectum, with continuous involvement in these regions. There are various degrees of ulceration, hemorrhage, edema, and pseudopolyps.
- **Crohn's disease:** Disease can involve any portion of the alimentary tract, including the upper GI tract (30-40% of cases), small bowel (90%), and terminal ileum (50-70%). There is transmural inflammation, with discrete lesions ("skip lesions"). Because of the full-thickness involvement, there can be focal ulcerations, fistulas, strictures, adhesions, and a cobblestone appearance.

Jacobstein D, Baldassano R: Inflammatory bowel disease. In Liacouras CA, Piccoli DA (eds): Pediatric Gastroenterology—The Requisites in Pediatrics. Philadelphia, Mosby/Elsevier, 2008, pp 131-141.

Peppercorn M: Definition, epidemiology, and risk factors in inflammatory bowel disease. UpToDate, 2013. Available at www.uptodate.com.

30. What are some of the extraintestinal features of ulcerative colitis and Crohn's disease?

Because both can present with bloody diarrhea, abdominal pain, weight loss, and fever, extraintestinal manifestations may help determine which is more likely prior to endoscopy.

- **Ulcerative colitis:** Growth failure, arthropathy/arthritis, pyoderma gangrenosum
- **Crohn's disease:** Growth failure, delayed puberty, perianal disease, stomatitis, erythema nodosum, arthritis, clubbing, uveitis, nephrolithiasis

Jacobstein D, Baldassano R: Inflammatory bowel disease. In Liacouras CA, Piccoli DA (eds): Pediatric Gastroenterology—The Requisites in Pediatrics. Philadelphia, Mosby/Elsevier, 2008, pp 131-141.

Peppercorn M: Definition, epidemiology, and risk factors in inflammatory bowel disease. UpToDate, 2013. Available at www.uptodate.com.

31. What are some of the food allergy disorders of infancy and their treatment?

Food protein–induced proctitis/proctocolitis is found in infants between 2 and 8 weeks of age who are breast-fed or fed cow's milk or soy-based formulas. The infant presents with blood-tinged mucus or stool, may be fussy or irritable, and may have increased stool frequency, but not diarrhea. The problem resolves when the mother stops eating the presumed food antigen or the formula is changed.

Food protein–induced enteropathy presents in infancy with diarrhea and poor weight gain. Vomiting and malabsorption also occur. Milk sensitivity is the usual culprit, but soy, egg, wheat, and other food can cause this. Biopsy of the small intestine shows villous atrophy with cellular infiltration. These symptoms also improve when the agent is removed, but these reactions often resolve by 1 year of age.

Food protein–induced enterocolitis syndrome (FPIES) occurs in the first few months of life and involves allergy mainly to milk protein and soy. The child has protracted vomiting and diarrhea and may present in shock.

In these cases, substitute whole cow's milk with casein hydrolysate formulas (Nutramigen, Pregestimil, Alimentum) or amino acid–based formulas (Elecare, Neocate, Puramino). Even whey hydrolysate or soy protein formulas are not appropriate, as some children have allergies to these as well. An amino acid–based formula (Neocate, Vivonex) can also be used.

Lake AM: Food protein-induced proctitis/colitis and enteropathy of infancy. UpToDate, 2013. Available at www.uptodate.com.

32. What is eosinophilic esophagitis?

Eosinophilic esophagitis is a chronic immune/antigen-mediated disease of the esophagus characterized by eosinophil-predominant inflammation that results in esophageal dysfunction. There may be a link between environmental (food allergies) and genetic factors. The clinical symptoms in children include vomiting, abdominal pain, feeding dysfunction, dysphagia, and food impaction.

Bonis PA, Furata GT: Pathogenesis, clinical manifestations, and diagnosis of eosinophilic esophagitis. UpToDate, 2013. Available at www.uptodate.com.

33. What is celiac disease?

This is a specific food protein–induced enteropathy due to gluten, a protein found in grains. The specific proteins are gliadin in wheat, secalines in rye, and hordeins in barley. Some patients may also have problems with oats. It is an HLA-associated condition (HLA [human leukocyte antigen] DQ2/DQ8). Presenting symptoms include weight loss/failure to thrive, chronic diarrhea, steatorrhea, and abdominal distention. Diagnosis is by small intestinal biopsy and symptom resolution with a gluten-free diet. Treatment is avoidance of these grains.

Hill ID: Clinical manifestations and diagnosis of celiac disease in children. UpToDate, 2013. Available at www.uptodate.com.

34. What are some of the malignant abdominal masses in children?

Wilms' tumor, neuroblastoma, hepatoblastoma, hepatocarcinoma, pelvic sarcoma, lymphoma, teratomas, and ovarian tumors.

Billett AL, Kesselheim JL: Oncologic emergencies. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1033-1066.

35. What is the classic triad of findings associated with HUS?

Acute hemolytic anemia, thrombocytopenia, and renal injury (manifested by oliguria, abnormal urinalysis, and increasing blood urea nitrogen [BUN] and creatinine levels). Although it is usually associated with bloody diarrhea, there are actually two phenotypes of HUS. One is Shiga toxin–associated HUS, which is associated with diarrhea (D+HUS, or typical HUS); the other is non–Shiga toxin–associated HUS, which does not present with diarrhea (D−HUS, or atypical HUS). The Shiga toxin is elaborated by *E. coli* O157:H7 or *Shigella dysenteriae* type 1.

Niaudet P: Clinical manifestations and diagnosis of Shiga toxin associated (typical) hemolytic uremic syndrome. UpToDate, 2013. Available at www.uptodate.com.

Rodig NM: Renal and electrolyte abnormalities. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1099-1126.

36. A child has diarrhea as a result of *E. coli* O157:H7. Should he receive antibiotics?

No, because there is an increased risk of that child developing HUS if antibiotics are given during the diarrheal phase of the illness.

Niaudet P: Clinical manifestations and diagnosis of Shiga toxin associated (typical) hemolytic uremic syndrome. UpToDate, 2013. Available at www.uptodate.com.

37. Which laboratory tests should you order for a child you think has pancreatitis?

Serum amylase levels become elevated within 6 to 12 hours of symptoms of acute pancreatitis, peak at 12 to 72 hours, and often return to normal in 3 to 5 days. Because amylase is produced in

the salivary glands and ovaries, an isolated serum amylase level does not necessarily reflect pancreatic origin. Serum lipase increases 4 to 8 hours after the onset of symptoms, peaks at 24 hours, and remains elevated for 8 to 14 days. A serum lipase level three times normal has better diagnostic accuracy for pancreatitis than serum amylase.

Pietzak MM: Acute and chronic pancreatitis. In Guandalini S (ed): Textbook of Pediatric Gastroenterology and Nutrition. London, Taylor & Francis, 2004, pp 303-318.

Vege SS: Clinical manifestations and diagnosis of acute pancreatitis. UpToDate, 2013. Available at www.uptodate.com.

38. What causes pancreatitis in children?

Acute pancreatitis in children is due to one of several causes:

- Anatomic/structural abnormalities (choledochal cysts, biliary stone, tumors)
- Drugs and toxins (acetaminophen overdose, antibiotics, anticonvulsants, antihypertensives, anti-inflammatory drugs, neoplastic agents, ethanol overdose)
- Infections (*E. coli*, *Ascaris lumbricoides*, varicella, mumps, coxsackievirus, influenza B virus, human immunodeficiency virus [HIV])
- Trauma (disruption of pancreatic ducts, compression injury)
- Familial/hereditary disease (cystic fibrosis)
- Metabolic disorder (hyperlipidemia, hyperparathyroidism, malnutrition)
- Idiopathic

Pietzak MM: Acute and chronic pancreatitis. In Guandalini S (ed): Textbook of Pediatric Gastroenterology and Nutrition. London, Taylor & Francis, 2004, pp 303-318.

39. What is cyclic vomiting syndrome?

This syndrome of vomiting in a cyclic pattern is severe, recurring, and stereotypical. There is a high intensity of vomiting (6-12 times an hour) that occurs 1 to 2 times a month and lasts approximately 24 hours. The child is well between episodes. Vomiting is often precipitated by psychological stress, infection, exhaustion, a menstrual period, or certain foods (cheese, chocolate, caffeine, monosodium glutamate), and there is usually a strong family history of migraine headaches. Treatment includes IV fluids with 10% dextrose in 0.45 normal saline (D_{10}.45NS) at 1.5 times maintenance, ondansetron, and lorazepam (or chlorpromazine or diphenhydramine).

Dulude E, Desielts DJ, Boles RG: Cyclic vomiting syndrome. UpToDate, 2013. Available at www.uptodate.com.

Sunku B, Li BUK: Cyclic vomiting syndrome. In Guandalini S (ed): Textbook of Pediatric Gastroenterology and Nutrition. London, Taylor & Francis, 2004, pp 289-302.

40. What are the criteria necessary to make the diagnosis of cyclic vomiting syndrome?

- At least five attacks in any interval or a minimum of three episodes of vomiting within a 6-month period
- Acute attacks of intense nausea and vomiting lasting 1 hour to 10 days, occurring at least 1 week apart
- Vomiting that occurs during attacks at least four times an hour for at least 1 hour
- Stereotypical pattern and symptoms for each patient
- No symptoms between episodes of vomiting
- No identifiable organic cause of the vomiting

Dulude E, Desielts DJ, Boles RG: Cyclic vomiting syndrome. UpToDate, 2013. Available at www.uptodate.com.

41. What is the typical history of the GI symptoms associated with Schönlein-Henoch purpura (IgA vasculitis)?

GI symptoms normally include nausea, vomiting, and abdominal pain and can occasionally result in intussusception, GI hemorrhage, and, rarely, bowel necrosis. The GI symptoms usually develop within 8 days of the rash. However, it has been noted that GI symptoms, including abdominal pain, precede the rash in about 15% to 35% of cases, making the diagnosis of this condition more challenging.

Dedeoglu F, Kim S, Sundel R: Clinical manifestations and diagnosis of Henoch-Schönlein purpura (IgA vasculitis). UpToDate, 2013. Available at www.uptodate.com.

42. What is the suggested imaging study in suspected intussusception associated with Henoch-Schönlein purpura?

Abdominal ultrasonography is recommended as opposed to air or barium contrast enema (typically ordered when intussusception is suspected in patients without Henoch-Schönlein purpura). Contrast enemas cannot make the diagnosis of ileoileal intussusception, which is typically seen in patients with Schönlein-Henoch purpura–related intussusception.

Dedeoglu F, Kim S, Sundel R: Clinical manifestations and diagnosis of Henoch-Schönlein purpura (IgA vasculitis). UpToDate, 2013. Available at www.uptodate.com.

CHAPTER 36

GYNECOLOGIC EMERGENCIES

Kate M. Cronan

1. What are the best ways to perform a genital examination in a prepubertal girl?

Examine the external genitalia with the child lying supine in a "frog-leg" position, either on the examining table or on the parent's lap. Use gentle lateral traction on the labia majora to obtain an adequate view of the introitus. To visualize the vaginal vault, examine the child in the "knee-chest" position. Ask the child to get up on her hands and knees ("like you are going to crawl") and to rest her head on her folded arms. The labia and buttocks can be separated while the child relaxes her abdominal muscles. An otoscope *without* speculum can be used as a light source. If discharge or bleeding is noted, return the child to the supine position to take samples/cultures. If there is vaginal bleeding from an injury, it may be necessary to perform a speculum examination under sedation.

Paradise J: Vaginal bleeding. In Fleisher GR, Ludwig S (eds): The Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 606-612.

2. A mother brings her 2-year-old daughter to the emergency department (ED) because she has noticed that her "vagina seems to be closing up." What could this be caused by?

Labial adhesions are a benign condition occurring in 3% to 7% of girls between the ages of 3 months and 5 years (Fig. 36-1). The exact cause is not known, but it could be related to repeated bouts of vulvovaginitis. The medial epithelial surfaces of the labia minora gradually adhere to one another, and the fusion proceeds anteriorly. Physical examination using labial traction reveals a flat area of adherent tissue, with a characteristic vertical raphe obscuring the introitus. A small opening remains through which urine may pass. Therapy consists of twice-daily applications of estrogen cream at the point of midline fusion. Continue treatment until the lesions resolve. Once the adhesions have separated, apply zinc oxide or petroleum jelly for an additional 2 weeks to prevent readherence. Medical therapy may rarely fail when the adhesions are thick (3-4 mm in width) with no evidence of a thin raphe.

Do not confuse labial adhesions with congenital abnormalities of the genitalia, such as ambiguous genitalia or imperforate hymen.

Jacobs A, Alderman E: Gynecologic examination of the prepubertal girl. Pediatr Rev 2014;35:97-105.

3. A healthy 14-year-old girl presents with symptoms of urgency, frequency, and dysuria, as well as intermittent lower abdominal pain. Her urinalysis is normal. Although her sexual development has been normal, she has never menstruated. What might be the problem?

A thorough physical examination, including a genital examination, will likely reveal the source of her distress. This patient probably has a congenital obstruction of the vagina, either an imperforate hymen or vaginal atresia. If this condition is not noticed during infancy, a young adolescent girl can present as described here, with large quantities of menstrual blood accumulated behind the hymen. The collection of blood is termed *hematocolpos*. Examination of the abdomen may reveal a mass, which might initially be confused with a tumor or even pregnancy. Examination of the genitalia shows a bulging, membranous covering over the introitus, often appearing bluish from the blood behind. Treatment is surgical.

4. How does hydrocolpos differ from hematocolpos?

Vaginal obstruction during infancy leads to vaginal distention that is due to a buildup of mucus. This is called *hydrocolpos* or *mucocolpos*. Infants present with an abdominal mass, trouble with urinating, and a bulging membrane at the introitus. When the amount of mucus secretion is large, the uterus also becomes distended, and *hydrometrocolpos* results. If vaginal obstruction is not

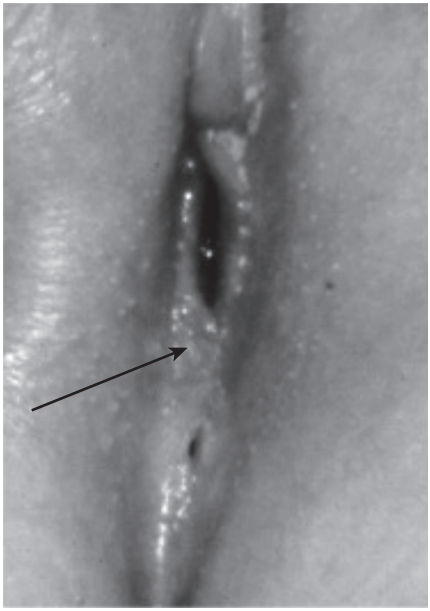

Figure 36-1. Labial adhesions. *(Modified from Stukus KS, Zuckerbraun NS: Review of the prepubertal gynecologic examination: Techniques and anatomic variation. Clin Pediatr Emerg Med 2009;10(9):3-9. Photograph courtesy of Dr. Janet Squires, Children's Hospital of Pittsburgh.)*

recognized until menarche, menstrual blood accumulates and distends the vagina, producing *hematocolpos*. In late puberty the child may present with amenorrhea. Eventually urinary symptoms may develop as the collection of blood increases. A lower abdominal mass is often palpable.

5. How does perineal trauma in young girls typically occur?

Most perineal trauma in young girls results from a "straddle injury," wherein the girl falls on a narrow object (e.g., bicycle crossbar, jungle gym, chair arm, fence). The most common injuries are vulvar hematomas and superficial lacerations. Most of these may be treated conservatively with sitz baths several times a day. Ensuring in the ED that the patient can void is crucial.

6. How should urinary retention after a straddle injury be managed?

In most cases urinary retention is mild and brief, and it occurs because of the discomfort of the urine passing over the injured perineum. Girls may be more comfortable urinating while sitting in a tub of warm water.

Garcia CT, Thompson VT: Genitourinary trauma. In Fleisher GR, Ludwig S (eds): The Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1316-1327.

7. What concerns must you have regarding more complicated perineal trauma?

With a very forceful straddle injury, deep lacerations may occur in the vagina and may extend into the rectum or urethra. Some hematomas may expand rapidly and become massive, requiring evacuation. These injuries must be explored thoroughly, either with effective sedation or under general anesthesia in the operating room. Consult colleagues from pediatric gynecology or pediatric urology as appropriate for surgical repair. Concerns are similar for penetrating trauma to the perineum by a foreign object.

8. What are the causes of vaginal bleeding before menarche?

In the prepubertal child, "vaginal bleeding" may originate in the vagina, the vulva, or both. Causes of apparent vaginal bleeding can be divided into nonhormonal and hormonal:

- **Nonhormonal** causes include trauma, tumor, urethral prolapse, infectious vaginitis, intravaginal foreign body, and genital warts.
- **Hormonal** causes include neonatal bleeding, exogenous estrogen, and precocious puberty.

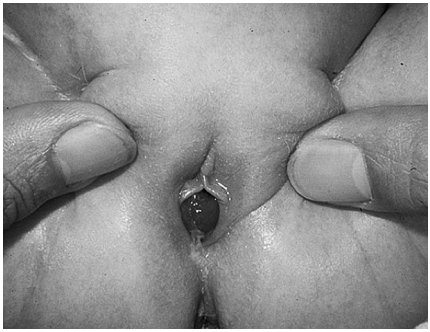

Figure 36-2. Urethral prolapse.

9. A 3-year-old African-American girl presents to the ED with vaginal bleeding and a donut-shaped mass of purplish tissue protruding from her vagina. Her mother is concerned that she might have been abused. What's your diagnosis?

Although sexual assault must always be in the differential diagnosis of genital trauma, the soft donut-shaped mass in this child is most likely *not* protruding from the vagina but rather is a urethral prolapse (Fig. 36-2). It is the most common cause of *apparent* vaginal bleeding in childhood, with the bleeding resulting from ischemia of the protruding urethral mucosa. For reasons that remain obscure, 95% of cases reported in the literature are in African-American girls. If the segment of prolapsed urethra is not necrotic, warm compresses or sitz baths in combination with 2 weeks of topical estrogen may be effective. Dark red necrotic mucosa requires surgical reduction of the prolapse within several days.

10. A mother brings in her 6-year-old daughter with a concern that the girl seems to be urinating more frequently than usual and says that it hurts when she urinates. You are sure she has a urinary tract infection, and you confidently send a sample to the laboratory. The urinalysis is normal. What could be causing her dysuria and frequency?

Symptoms of vulvovaginitis can include urinary frequency and dysuria. The most frequent reason for vulvovaginitis is simple irritation, as the result of either poor hygiene or cleansing products, such as bubble bath, shampoo, powders, soaps, and some feminine hygiene products. Infectious causes include *Shigella* spp., group A streptococci, *Neisseria gonorrhoeae*, and *Candida* spp. Pinworm infections, although rectal in origin, may lead to such intense itching and vigorous scratching that the patient presents with perineal excoriations and bleeding.

Treatment is frequent sitz baths. If the vulva is irritated, the patient may be more comfortable voiding while sitting in a tub of warm water. If symptoms of nonspecific vulvovaginitis persist for 2 to 3 weeks, consider a 10-day course of oral antibiotics (e.g., amoxicillin). When a specific microbial cause has been identified in a prepubertal child (such as *Neisseria* [gonorrhea], *Chlamydia*, coliform bacteria, group A streptococci, *Candida albicans*, *Trichomonas* spp., or *Gardnerella* spp.), give appropriate antimicrobial treatment and alert child protective services.

11. What is the most common cause of foul-smelling vaginal discharge in young girls?

Foul-smelling discharge most commonly signals a retained foreign body in the vagina. The most common foreign body is toilet paper, which may be contaminated with feces. To assess for a foreign body, first place the patient in the knee-chest position to examine the vaginal area. Attach a 60-mL syringe filled with warmed saline to an 8 F feeding tube. Place the patient in the supine position and separate the labia majora. Viscous lidocaine may be applied prior to the insertion of the catheter. Then place the tube past the hymenal orifice and irrigate with saline.

Giardino A, Christian C: Vaginal foreign body removal. In King C, Henretig FM, (eds): The Textbook of Pediatric Emergency Procedures, 2nd ed. Philadelphia, Lippincott Williams & Wilkins, 2008, pp 871-874.

12. What causes vaginal bleeding after menarche?

Vaginal bleeding in the adolescent patient can result from hormonal contraception, endometritis, dysfunctional uterine bleeding, bleeding diathesis, or complications of pregnancy (spontaneous abortion, ectopic pregnancy, placenta previa, placental abruption).

13. What is the first test to perform on *any* adolescent patient with abnormal vaginal bleeding?

Qualitative urine pregnancy test. An ectopic pregnancy can rapidly progress into a life-threatening emergency. In many instances, the urine sample can be obtained even before the physical examination is begun.

14. A 13-year-old female patient arrives in the ED with abnormal vaginal bleeding. Her menarche was around age 12. Her menstrual periods generally occur 24 to 32 days apart, but it has been only 18 days since the start of her last period. Should you be concerned?

Probably not. During the first 2 years following menarche, it is not uncommon to have irregularities in both duration of menses and number of days between cycles. Note that 95% of adolescents' periods last 2 to 8 days. Ten or more days of bleeding should be considered abnormal. Most cycles start 21 days or more from the first day of the last period. Although occasional intervals of less than 21 days are normal for young teens, several short cycles in a row are abnormal. Although any amount of bleeding may be more than young teenagers would like, it is uncommon for adolescents to soak more than 6 to 8 pads or tampons per day.

15. What are the most common organisms causing sexually transmitted disease (STD) in the adolescent population?

Chlamydia trachomatis and *N. gonorrhoeae* infections are the most commonly reported STDs among adolescent patients. In fact, reported rates of chlamydial and gonorrheal infections are highest among females age 15 to 19 years. Human papillomavirus is probably the most common viral STD among teens, with herpes simplex virus type 2 also frequent. Neither are reportable diseases, so exact prevalence rates are not known. HIV (human immunodeficiency virus) infection is increasing among adolescents, but rates of both primary and secondary syphilis have steadily declined in all age groups since peaking in 1990.

Workowski KA, Berman S; Centers for Disease Control and Prevention (CDC): Sexually transmitted diseases treatment guidelines, 2010. MMWR Recomm Rep 2010;59:1-110.

16. True or false? Bacterial vaginosis occurs only in women who are sexually active.

False. Bacterial vaginosis is the most prevalent reason for vaginal discharge in sexually active females. Although many cases of bacterial vaginosis occur in sexually active women, those who have not been sexually active can also have bacterial vaginosis. It is associated with having a new sexual partner, an increasing number of sexual partners, and lack of condom use. Also, douching can result in bacterial vaginosis.

17. What are the criteria for the diagnosis of bacterial vaginosis?

Three out of four of the following are required:

- Thin vaginal discharge that is gray or white and coats the vaginal walls
- A fishy odor of the discharge
- Presence of "clue cells" (squamous vagina epithelial cells that are coated with bacteria) on microscopic examination
- Vaginal pH greater than 4.5

Pickering LK (ed): Red Book: 2012 Report of the Committee on Infectious Diseases, 29th ed. Elk Grove Village, IL, American Academy of Pediatrics, 2012, pp 247-249.

18. Pelvic inflammatory disease (PID) can be caused by a number of different microorganisms. What are they?

C. trachomatis, *N. gonorrhoeae*, or both can be identified in nearly 50% of cases of PID, with almost all first episodes of PID in adolescents attributable to these organisms. Evidence from laparoscopically obtained cultures demonstrates that upper tract infection can be polymicrobial, involving both aerobic and anaerobic organisms. Isolates have included genital mycoplasmas, such as *Mycoplasma hominis* and *Ureaplasma urealyticum*, aerobic and anaerobic streptococci, *Gardnerella vaginalis*, *Haemophilus influenzae*, and enteric organisms such as *Escherichia coli* and *Bacteroides* spp.

19. What distinguishes PID from uncomplicated cervicitis?

Cervicitis is an inflammation of the cervix characterized by a mucopurulent vaginal discharge; cervical inflammation with friability, edema, and ectopy; and presence of white blood cells in the cervical os. Symptoms include vaginal discharge, dysuria, dyspareunia, and postcoital bleeding. Unlike with PID, systemic signs of infection are *not* characteristic of cervicitis, nor is cervical motion tenderness or adnexal tenderness evident on examination. Cervicitis can be caused by a variety of organisms, including *C. trachomatis*, *N. gonorrhoeae*, *Trichomonas vaginalis*, *C. albicans*, and herpes simplex virus. Cervicitis can also be noninfectious, resulting from a foreign body or chemical irritation. In contrast to PID, in which there should be a low threshold for starting empiric antibiotics, treatment for uncomplicated cervicitis can be deferred until a specific microbial cause is identified.

20. How is PID diagnosed?

Acute PID is often difficult to diagnose because of the nonspecific nature of symptoms and the broad differential diagnosis of abdominal pain in an adolescent female. Diagnosis is made through a combination of historical and clinical findings. Laboratory and radiologic studies may be helpful. In evaluating an adolescent female with abdominal pain, interview the patient and examine her in private (without the parent in the room). Take a thorough gynecologic and sexual history, including a menstrual history, number of sexual partners, contraceptive practices, and history of previous sexually transmitted infections (STIs). Focus the physical examination on the many possible causes of abdominal pain, including pregnancy, complications of prior abdominal surgery, and pneumonia. Perform a careful examination of the abdomen and a thorough pelvic examination (both speculum and bimanual examination, checking for cervical motion tenderness, adnexal masses, and/or tenderness). Take a culture or DNA probe samples for *Chlamydia* and *Neisseria*.

21. What tests are very sensitive and specific in diagnosing genital gonorrhea?

Nucleic acid amplification tests (NAATs) are very sensitive and specific if used on female vaginal or endocervical swab or male urethral swab and male/female urine specimens. Using NAAT on urine specimens increases the likelihood of clinicians testing for STI and allows easier follow-up in young patients. Both gonorrhea and chlamydial infection can be detected through this method.

Centers for Disease Control and Prevention: Recommendations for the laboratory-based detection of *Chlamydia trachomatis* and *Neisseria gonorrhoeae*—2014. MMWR Recomm Rep 2014;63:1-19.

Pickering LK (ed): Red Book: 2012 Report of the Committee on Infectious Diseases, 29th ed. Elk Grove Village, IL, American Academy of Pediatrics, 2012, pp 336-344.

22. When should you treat a teenager for PID?

In the ED, have a low threshold for initiating empiric antibiotic treatment for any adolescent female patient with a history or physical examination suggestive of PID. Only minimal laboratory evaluation (testing for gonorrhea and trachomatis) need be performed. This approach minimizes the chance of missing PID in a patient presenting with only mild symptoms. If the patient presents with a severe clinical picture or is pregnant, undertake more extensive evaluation (laboratory and ultrasonography) to rule out other diagnoses. If no other definitive diagnosis is reached (e.g., appendicitis, urinary tract infection, pneumonia), start treatment.

23. What is the recommended treatment for gonorrhea?

For treatment of uncomplicated gonorrhea, the Centers for Disease Control (CDC) recommend one intramuscular (IM) dose of ceftriaxone 250 mg PLUS one dose of azithromycin 1 g OR doxycycline 100 mg twice a day for 7 days.

Centers for Disease Control and Prevention (CDC): Update to CDC's sexually transmitted diseases treatment guidelines, 2010: Oral cephalosporins no longer a recommended treatment for gonococcal infections. MMWR Morb Mortal Wkly Rep 2012;61(31):590-594.

24. What are the indicators for treating sexual partners?

Identification and treatment of sexual partners are key in limiting the spread of STI. Evaluate any sexual partners within the past 3 months and test for gonorrhea and chlamydial infection, whether or not they are symptomatic. In general, empiric treatment is appropriate for all sexual contacts of a patient with PID.

25. What is expedited partner therapy (EPT)?

EPT means that the clinician provides the patient's partner(s) with a prescription for treatment of infection without performing an evaluation or examination. For example, treatment will consist of cefixime and azithromycin for gonorrhea. This practice is not permitted in all states, and therefore, clinicians need to check the legal status of EPT. It is recommended that patients abstain from sexual contact until the therapy is completed and symptoms have resolved.

Comkornuecha K: Gonococcal infections. Pediatr Rev 2013;34(5):228-234.

26. Do all adolescents with PID need to be hospitalized for treatment?

No. The CDC recommends hospitalization when "surgical emergencies" such as appendicitis cannot be excluded; the patient is immunodeficient; the patient is pregnant; the patient does not respond clinically to oral antimicrobial therapy; the patient is unable to follow or tolerate an outpatient oral regimen; the patient has severe illness, nausea and vomiting, or high fever; or the patient has a tubo-ovarian abscess.

There is no evidence that adolescents benefit from hospitalization for treatment of PID. Nonetheless, many practitioners believe that hospitalization is appropriate for adolescents, especially when adherence to treatment is in question. Hospitalization also affords an opportunity for education regarding "safe sex" practices.

Workowski KA, Berman S; Centers for Disease Control and Prevention (CDC): Sexually transmitted diseases treatment guidelines, 2010. MMWR Recomm Rep 2010;59:1-110.

Key Points: Indications for Admission of Patients with PID

1. Pregnancy
2. Surgical emergency that cannot be ruled out
3. No response to oral antimicrobial agents
4. Nonadherent patient
5. Evidence of severe illness
6. Tubo-ovarian abscess
7. Immunodeficiency

27. What are the long-term complications of PID?

Long-term complications may occur in as many as 25% of females who have had PID. These include tubo-ovarian abscesses, Fitz-Hugh-Curtis syndrome, ectopic pregnancy (6-fold to 10-fold increased risk), infertility as a result of scarring (with the risk of infertility increasing with the number of episodes of PID), dyspareunia, and chronic and recurrent pelvic pain.

28. Why are adolescent patients at such high risk for STI?

Adolescents have some of the highest rates of STI. Many factors put adolescents at increased risk, including a high frequency of risky sexual behaviors and basic physiologic differences. Teenagers who are beginning to experiment with sexual behaviors often have multiple sexual partners and change partners frequently, with each new partner constituting a potential exposure to STI. Adolescents generally use contraceptives inconsistently, particularly barrier methods such as condoms, which prevent the passage of infectious organisms. Human papillomavirus infects actively dividing cells, and both *C. trachomatis* and *N. gonorrhoeae* adhere more easily to columnar epithelial cells. Finally, adolescents lack immunity to STIs.

29. What are the most typical presenting features of ovarian torsion, and which patients are most likely to have this condition?

- Stabbing abdominal pain
- Nausea and vomiting
- Pain radiating to the back or groin
- Sudden onset of sharp pain

A history of recent vigorous activity may be the precipitating event. Ovarian torsion is most frequently seen in postmenarchal females because it occurs more often when an ovarian cyst is present. However, ovarian torsion can occur in premenarchal females with normal ovaries.

Growdon WB, Laufer MR: Ovarian torsion. Uptodate, 2006. Available at www.uptodate.com.

30. How is the clinical diagnosis of ovarian torsion made?

In a female patient with symptoms of severe lower abdominal pain and nausea or vomiting, the differential diagnosis includes ectopic pregnancy, appendicitis, ovarian torsion, PID, tubo-ovarian abscess, endometriosis, and hemorrhagic cyst. Two-dimensional ultrasonography with Doppler and three-dimensional ultrasonography are the most effective modalities used to diagnose ovarian torsion. These studies demonstrate limited or absent blood flow in the ovary, which is suggestive of torsion. Open laparotomy provides definitive diagnosis.

31. A 15-year-old female presents to the ED with left lower abdominal pain. She has had some nausea but no vomiting or fever. On examination there is mild tenderness in the left lower quadrant. Ultrasonography of the pelvis is negative. What is the likely diagnosis?

Menarchal females who are ovulatory may have midcycle pain suggesting *mittelschmerz*, which means "middle pain." Mittelschmerz is attributable to follicular enlargement prior to ovulation. It can also be caused by bleeding associated with follicular rupture. The pain is usually mild and unilateral. It lasts from a few hours to several days.

32. What are the risk factors for ectopic pregnancy?

Risk factors include tubal abnormalities, history of assisted reproduction, use of an intrauterine device, prior upper gynecologic tract infection, and use of progestin-only contraceptives.

Goyal M, Mollen C, Lavelle J: Adolescent emergencies. In Fleisher GR, Ludwig S (eds): The Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1634-1649.

33. What is the clinical profile of a ruptured ectopic pregnancy, and what is the most crucial therapy?

Patients with ruptured ectopic pregnancy generally have a history of intermittent pelvic pain in association with abnormal vaginal bleeding. Shock (compensated or uncompensated) may be evident on physical examination. Abdominal examination reveals tenderness, and an adnexal mass may or may not be palpated after rupture. Surgical assessment must be immediate. Imperative components of treatment are ongoing cardiovascular monitoring, fluid resuscitation, and transfusion of packed red blood cells as needed.

Goyal M, Mollen C, Lavelle J: Adolescent emergencies. In Fleisher GR, Ludwig S (eds): The Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1634-1649.

34. What is the most common presenting sign of early pregnancy in adolescents?

A missed or abnormal period is the most common sign of pregnancy in teens. However, the menstrual history is often unreliable in adolescents because of anovulatory cycles. Therefore, other symptoms that should raise a red flag include fatigue, weight gain, nausea, morning sickness, and nonspecific gastrointestinal or genitourinary symptoms. Less common symptoms consist of vaginal bleeding or discharge, hyperemesis, and headache.

35. What is Plan B emergency contraception?

Plan B provides a method to prevent pregnancy after unprotected sex. Specifically packaged products that contain a total dose of 1.5 mg of levonorgestrel (progestin only) for emergency contraception are available as Plan B. The two pills can be taken together with no risk for increased side effects. Plan B needs to be taken within 120 hours of unprotected sex. But it is more effective the sooner it is taken. It may prevent or delay ovulation, or it may interfere with fertilization of an egg. It is not an abortion pill and will not work if a person is already pregnant. In the United States it is available for those over 17 without a prescription.

36. Why is Plan B called "Plan B"?

It is a backup method of contraception and shouldn't be considered as a routine form of birth control.

Upadhya K: Contraception for adolescents. Pediatr Rev 2013;34(9):384-394.

37. What is the leading cause of teenage short-term recurring school absenteeism in the United States?

Primary dysmenorrhea. The prevalence of dysmenorrhea is highest in adolescent females, and severe dysmenorrhea occurs in approximately 15% of female adolescents. The cause of

dysmenorrhea is thought to be secretion of prostaglandins in the menstrual fluid, leading to uterine contractions and subsequent pain. Vasopressin may also be involved. It increases uterine contractility, leading to vasoconstriction. Nonsteroidal anti-inflammatory drugs remain the most effective initial therapy for dysmenorrhea. They inhibit prostaglandin synthesis and are thought to decrease menstrual flow volume.

French L: Dysmenorrhea. Am Fam Physician 2005;71:285-291.

Key Points: Plan B Emergency Contraception

1. Progestin-only levonorgestrel
2. Can be taken up to 120 hours after unprotected sex (most effective closer to the time of unprotected sex)
3. Not a routine form of birth control
4. Will not affect a pregnancy
5. Adverse effects include nausea and abdominal pain

CHAPTER 37

HEMATOLOGIC AND ONCOLOGIC EMERGENCIES

Paul Ishimine and Jenny Kim

HEMATOLOGIC EMERGENCIES

1. How should one approach the evaluation of a child presenting with anemia to the emergency department (ED)?

Anemia is caused by increased red blood cell (RBC) destruction, decreased RBC or hemoglobin production, or blood loss. After patient stabilization, the following features can help determine the underlying cause:

- Historical features: Rapidity of onset, hemorrhage, diet, history of easy bruising or bleeding, previous episode of anemia, intercurrent illness, family history of blood disorders
- Physical examination findings: Jaundice, splenomegaly, enlarged lymph nodes, bruising/bleeding, occult blood on rectal examination
- Laboratory studies: Complete blood count (CBC) with manual differential, mean corpuscular volume (MCV), peripheral smear, reticulocyte count, direct Coombs' test

Singh SN: Pallor. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 483-490.

2. What is the underlying pathophysiology of sickle cell disease (SCD)?

SCD is caused by an inherited hemoglobinopathy in which a single mutation causes an amino acid substitution in the β-globin chain of hemoglobin. This results in distorted, elongated, sickle-shaped RBCs that are prone to aggregation and vascular injury, resulting in vaso-occlusion, hemolysis, and resultant complications, such as stroke, acute chest syndrome, splenic sequestration, and pain from ischemia. There are many genotypes of SCD, including SCD-SS, -SC, -$S\beta^+$ thalassemia, and -$S\beta^0$ thalassemia. Patients with SCD-SS and SCD-$S\beta^0$ thalassemia usually have a more severe clinical course.

Rees DC, Williams TN, Gladwin MT: Sickle-cell disease. Lancet 2010;376(9757):2018-2031.

3. What are the life-threatening complications of SCD?

- Sepsis
- Stroke
- Splenic sequestration crisis
- Acute chest syndrome
- Aplastic crisis

National Institutes of Health/National Heart, Lung, and Blood Institute Division of Blood Diseases and Resources: The Management of Sickle Cell Disease, 4th ed. Bethesda, MD, National Institutes of Health, 2002, NIH publication No. 02-2117.

4. Why is a patient with SCD immunocompromised?

Splenic microinfarctions due to sickling of RBCs cause altered splenic function. As a result, patients with SCD are prone to infection with encapsulated organisms, such as *Streptococcus pneumoniae*.

5. What causes painful crisis in patients with SCD?

Vaso-occlusive crises are due to localized sickling and vascular occlusion and can occur in any organ, but most frequently affect bone and viscera. Most older children report long bone, back, and abdominal pain. Precipitating events may include infection, dehydration, fever, and exposure to cold.

6. How should painful crisis be treated?

Assess the severity of pain and associated organ systems. Intravenous (IV) hydration, usually at 1.5 times the maintenance rate, and nonsteroidal anti-inflammatory drug (NSAID) and narcotic administration are frequently required. Administration of oxygen is recommended if the patient's oxygen saturation is below his or her baseline. It is important to inquire about pain medications given at home. If these were ineffective, administration of an appropriate dose of a more potent analgesic medication is warranted. If the patient is opioid-tolerant, doses higher than usual are often needed to control pain.

7. What is dactylitis?

Dactylitis, or "hand-foot syndrome," results from vaso-occlusion of the nutrient arteries that supply the metacarpal and metatarsal bones, causing bone marrow infarction in patients with SCD. This form of vaso-occlusive crisis is most common in young infants (mostly under age 2 years) and results in pain and swelling of the hands and feet, irritability, and refusal to walk. It is usually bilateral, distinguishing it from cellulitis.

8. What are worrisome causes of headaches in patients with SCD?

Headaches are common in children with SCD. Although most headaches in children with SCD are idiopathic, children with SCD are at higher risk than the general pediatric population for ischemic stroke, intracranial hemorrhage, and cerebral venous thrombosis. In one retrospective study, the prevalence of these events was 6.9% in SCD patients presenting acutely with headaches.

Hines PC, McKnight TP, Seto W, Kwiatkowski JL: Central nervous system events in children with sickle cell disease presenting acutely with headache. J Pediatr 2011;159(3):472-478.

9. Define acute chest syndrome and its causes and treatment.

Acute chest syndrome is a serious complication of SCD and can be life-threatening. The syndrome is defined as a new pulmonary infiltrate and chest pain, hypoxia, fever, tachypnea, wheezing, or cough. The cause is uncertain. Both infectious (e.g., chlamydia, mycoplasma, viruses, bacteria) and noninfectious (e.g., fat embolism, pulmonary infarction) causes have been described. Admit all patients with suspected acute chest syndrome to the hospital. Treatment involves supportive care measures including analgesia, cautious hydration, oxygen, antibiotics, bronchodilators, and blood transfusions.

Yawn BP, Buchanan GR, Afenyi-Annan AN, et al. Management of sickle cell disease. Summary of the 2014 evidence-based report by expert panel members. JAMA 2014;312:1033-1048.

10. What is splenic sequestration crisis?

Splenic sequestration crisis is a life-threatening complication of SCD that occurs when a large portion of a patient's blood volume becomes acutely trapped in the spleen. This crisis leads to massive splenomegaly, acute anemia, and hypovolemic shock. Treatment is supportive and includes hospitalization, fluid resuscitation, and blood transfusion. Splenic sequestration is seen in young children, typically between 3 months and 5 years of age. It is important to quickly recognize severe splenomegaly in an ill-appearing young sickle cell patient, as hypovolemic shock due to massive splenic sequestration can be mistaken for septic shock, and delays in blood transfusion can result in cardiovascular collapse in these patients.

Key Points: Life-Threatening Complications of Sickle Cell Disease

1. Sepsis
2. Stroke
3. Splenic sequestration crisis
4. Acute chest syndrome
5. Aplastic crisis

11. Which virus frequently leads to aplastic crisis in children with SCD?

Parvovirus B19 causes brief suppression of erythropoiesis, which is not tolerated in patients with SCD because they have shortened RBC survival and rely on brisk erythropoiesis to maintain their baseline hemoglobin levels. Patients may present with fatigue, shortness of

breath, and severe anemia with reticulocytopenia. Treatment is supportive, and RBC transfusions are often necessary.

12. Are aplastic crises due to parvovirus B19 seen only in patients with SCD?

No. Parvovirus B19 can also cause aplastic crises in patients with other chronic hemolytic diseases, such as thalassemia or hereditary spherocytosis. These patients have shortened RBC survival and frequently cannot tolerate even a brief suppression of erythropoiesis.

13. How should patients with SCD and fever be treated in the ED?

Because these children are at high risk for serious bacterial infections, febrile children with SCD need rapid treatment with parenteral antibiotics after appropriate cultures are obtained. The greatest risk of serious bacterial infection is between the ages of 6 months and 3 years, when protective antibodies are not adequate and splenic function is greatly diminished. After administration of IV antibiotics (typically ceftriaxone because of its longer duration of action and good coverage of *S. pneumoniae*), the traditional approach has been to admit these patients to the hospital. However, some hematologists advocate cautious outpatient management of selected well-appearing febrile patients.

14. What are considered to be low-risk criteria for outpatient management of febrile patients with SCD?

- Well appearance
- No focal physical examination abnormalities
- Temperature lower than 40° C
- White blood cell count greater than 5000 cells/mm^3 but less than 30,000 cells/mm^3
- No pulmonary infiltrates
- Baseline hemoglobin, white blood cell, and platelet counts

Wilimas JA, Flynn PM, Harris S, et al: A randomized study of outpatient treatment with ceftriaxone for selected febrile children with sickle cell disease. N Engl J Med 1993;329:472-476.

15. If a patient with SCD meets the preceding criteria in Question 14, what is the recommended treatment plan?

These patients should receive an initial dose of parenteral ceftriaxone in the ED. Close follow-up must be arranged if febrile patients with SCD are discharged, including a scheduled revisit within the next 24 hours, at which point the child may receive a second dose of ceftriaxone.

Wilimas JA, Flynn PM, Harris S, et al: A randomized study of outpatient treatment with ceftriaxone for selected febrile children with sickle cell disease. N Engl J Med 1993;329:472-476.

16. An ill-appearing 6-year-old girl presents with weakness for 2 days. On examination she has jaundice and pale conjunctivae but no bruising or petechiae. Her hemoglobin level is 4 g/dL, but her white blood cell and platelet counts are normal. Her reticulocyte count is 20%. She has unconjugated hyperbilirubinemia, and she has dark-brown urine (which is dipstick-positive for blood but has no RBCs on microscopic examination). This presentation suggests what broad category of anemia?

Hemolytic anemia is suggested by her unconjugated hyperbilirubinemia and her hemoglobinuria, which result from RBC destruction. The elevated reticulocyte count implies an appropriate compensatory response by this patient's bone marrow. Hemolytic disease may be seen in children with RBC membrane defects (e.g., hereditary spherocytosis) or enzyme defects (e.g., glucose-6-phosphate dehydrogenase deficiency). Autoimmune hemolysis occurs when antibodies directed against RBCs cause hemolysis. Nonimmune acquired hemolysis may be caused by drugs, infections, or chemicals that cause direct RBC injury. Patients with hemoglobinopathies, such as SCD and thalassemias, can have periods of increased hemolysis and worsening anemia. Finally, mechanical causes of RBC fragmentation, such as hemolytic uremic syndrome, may also cause hemolysis.

17. What are the typical laboratory findings in iron deficiency anemia?

- Low hemoglobin and hematocrit
- Low mean corpuscular volume and RBC count

- High RBC distribution width
- Low serum iron and ferritin
- Increased transferrin
- Increased free erythrocyte protoporphyrin
- Microcytosis, hypochromia, poikilocytosis, and anisocytosis on peripheral smear
- Elevated platelet count

18. Why is excessive cow milk consumption associated with iron deficiency anemia in young children?

Overconsumption of cow milk decreases the toddler's appetite for other foods, and cow milk does not contain adequate iron for nutrition. There may also be a concomitant cow milk enteropathy causing microscopic intestinal blood loss. Milk and dairy products also interfere with intestinal iron absorption.

19. Describe appetite abnormalities associated with iron deficiency anemia.

Pica is associated with iron deficiency anemia and refers to an appetite for unusual substances not regarded as food, such as clay, paper, or dirt. Pagophagia, or pica for ice, is thought to be a specific finding in iron deficiency anemia.

Osman YM, Wali YA, Osman OM: Craving for ice and iron-deficiency anemia: A case series from Oman. Pediatr Hematol Oncol 2005;22:12.

20. What is transient erythroblastopenia of childhood (TEC)? How does it present?

TEC is an idiopathic disorder of acquired RBC aplasia characterized by a gradual onset of pallor. The median age at the time of diagnosis is 23 months, and there is often a history of a preceding viral illness. Except for pallor, the patient's physical examination is otherwise normal, with the absence of bruising, fever, lymphadenopathy, and hepatosplenomegaly. The CBC in a patient with TEC shows an isolated normochromic normocytic anemia with reticulocytopenia. TEC can be confused with leukemia, and bone marrow biopsy may be needed to exclude leukemia in a patient suspected of having TEC.

21. What are some causes of methemoglobinemia?

Methemoglobinemia may occur after exposure to oxidizing agents, such as certain drugs (e.g., benzocaine, sulfonamide antibiotics), well water that contains nitrites, mothballs, and aniline dyes. Acute gastroenteritis can lead to methemoglobinemia in infants. The heme iron is converted from a ferrous to a ferric state, resulting in hemoglobin with impaired oxygen binding, and manifests as cyanosis. Congenital methemoglobinemia is a rare cause of cyanosis in the newborn.

22. How do patients with idiopathic (or immune) thrombocytopenic purpura (ITP) typically present?

ITP most commonly presents in children ages 1 to 4 years with the acute onset of bruising and petechiae. There is often a history of a preceding viral illness. Children with ITP generally do not appear ill, and frank bleeding is surprisingly less common than would be expected in patients with thrombocytopenia.

23. What is the most serious complication of ITP?

The most serious concern in young children with ITP is intracranial hemorrhage. The risk of intracranial hemorrhage is highest in the first week after diagnosis and when the platelet count is less than 20,000 cells/mm^3. Intracranial hemorrhage is seen more frequently in patients who present with mucosal bleeding.

24. How is ITP treated?

The treatment of ITP is controversial. Options include close observation only, IV γ-globulin (which is thought to block the uptake of antibody-coated platelets by the spleen), and corticosteroids. Treatment with antibodies directed against the D-antigen of RBCs is effective only in Rh-positive patients and is falling out of favor because of a concern that this treatment is associated with fatal intravascular hemolysis. Rituximab, high-dose steroids, or splenectomy are reserved for patients who are unresponsive to initial treatments. Some hematologists feel that many patients with ITP do not need treatment given the generally benign bleeding and significant cost and side effects of treatment, because ITP usually spontaneously remits within 6 months.

Neunert C, Lim W, Crowther M, et al; American Society of Hematology: The American Society of Hematology 2011 evidence-based practice guideline for immune thrombocytopenia. Blood 2011;117 (16):4190-4207.

25. Name the three most common bleeding disorders and the deficient factors associated with each.

The most common inherited bleeding disorders are as follows:

- Hemophilia A (factor VIII deficiency)
- Hemophilia B (factor IX deficiency)
- Von Willebrand disease (von Willebrand factor)

26. What therapies can be used to treat patients with hemophilia?

The mainstay of treatment for patients with hemophilia is factor replacement therapy. Although fresh frozen plasma, cryoprecipitate, and prothrombin complex concentrates contain some replacement factor, monoclonal antibody-purified or recombinant factor VIII and factor IX are used most commonly to treat hemophilia A and hemophilia B, respectively. Patients with mild hemophilia A and von Willebrand disease can be treated with desmopressin. Plasma-derived factor VIII concentrates also contain von Willebrand factor and can be used to treat more severe bleeding in patients with von Willebrand disease. Recombinant factor VIII products do not contain von Willebrand factor, so they should not be used for patients with von Willebrand disease.

Singleton T, Kruse-Jarres R, Leissinger C: Emergency department care for patients with hemophilia and von Willebrand disease. J Emerg Med 2010;39(2):158-165.

27. What are the typical findings of an acute hemolytic transfusion reaction?

- Fever and chills
- Apprehension
- Chest tightness
- Abdominal or flank pain
- Hypotension

ONCOLOGIC EMERGENCIES

28. What are the most common childhood malignancies?

- Leukemia is the most common childhood malignancy, and acute lymphoblastic leukemia (ALL) is the most common type of childhood leukemia.
- The most common solid organ tumors are brain tumors, and most of these tumors are infratentorial. Medulloblastomas and cerebellar astrocytomas are the most common central nervous system tumors in children.

Howlader N, Noone AM, Krapcho M, et al (eds): SEER Cancer Statistics Review, 1975-2010, National Cancer Institute. Bethesda, MD. Available at http://seer.cancer.gov/csr/1975_2010/, based on November 2012 SEER data submission, posted to the SEER website April 2013. Accessed on July 29, 2013.

29. Why are children with malignancies at risk for sepsis?

Several factors contribute to the risk of severe infections and sepsis in pediatric oncology patients:

- Most importantly, there can be replacement of the bone marrow by malignant cells and direct suppression of granulocyte production by chemotherapeutic agents, resulting in neutropenia.
- Other mechanisms of defense against infection, including mechanical barriers (e.g., intact mucous membranes and skin), cell-mediated and humoral immunity, and splenic function, are frequently impaired in patients with cancer.
- Indwelling central venous catheters, ventriculoperitoneal shunts, and other implanted devices may become sites of infection.

30. How should febrile children with cancer be managed?

Approach febrile children with cancer carefully. The most important component is the patient's overall appearance. Any ill-appearing child needs broad-spectrum antibiotic coverage and admission to the hospital. If a febrile child is severely neutropenic (absolute

neutrophil count < 500 cells/mm^3) or if his or her absolute neutrophil count is lower than 1000 cells/mm^3 and expected to drop further, administer broad-spectrum antibiotic therapy after blood and other appropriate cultures are obtained. Admit these patients to the hospital. Well-appearing febrile patients who are not neutropenic may be treated more selectively. Discuss the treatment and disposition of all febrile children with cancer with the child's oncologist.

Billett AL, Kesselheim JC: Oncologic emergencies. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1033-1066.

31. An ill-appearing 6-year-old boy with acute myelogenous leukemia presents with fever and right-lower-quadrant abdominal pain. In addition to the usual causes of fever and right-lower-quadrant pain, what other entity needs to be considered in a neutropenic patient?

Typhlitis, a necrotizing enterocolitis, can be seen in neutropenic patients. The cause is multifactorial and includes infection and mucosal injury from chemotherapy or radiation therapy and most commonly involves the cecum. Typhlitis usually presents with fever, abdominal pain, and bloody diarrhea. The diagnosis is usually made by computed tomography (CT), and treatment includes gut rest, antibiotic therapy, and, rarely, surgery. The mortality rate with typhlitis is very high.

32. A teenager with a diffuse headache is found to have a white blood cell count of 200,000 cells/mm^3. What treatment should be initiated in the ED while awaiting confirmatory studies?

This child has hyperleukocytosis, which is highly suggestive of leukemia. This degree of white blood cell elevation leads to increased blood viscosity, which, in turn, predisposes a patient to thrombosis, leading to neurologic, pulmonary, and hemorrhagic symptoms. These patients are also at high risk for tumor lysis syndrome (TLS). Treatment includes hydration and alkalinization; more refractory cases are treated with cytoreduction (either via chemotherapy or by leukapheresis). Give platelet transfusions if the platelet count is lower than 20,000 cells/mm^3 to reduce the risk of central nervous system hemorrhage. Give RBC transfusions judiciously because they can increase blood viscosity.

33. What is the differential diagnosis of an anterior mediastinal mass in a child?

The causes of anterior mediastinal mass are listed in Table 37-1.

34. What are some of the risks associated with an anterior mediastinal mass?

Anterior mediastinal masses are associated with superior mediastinal syndrome in 12% of patients at the time of presentation. This syndrome is defined as compression of the structures of the superior mediastinum, including the superior vena cava (SVC) and trachea. Anterior mediastinal masses also can cause SVC syndrome, which refers to upper body venous congestion from either extrinsic compression of the SVC or a thrombus within the SVC. These conditions can lead to signs of airway compromise (e.g., stridor, cough, dyspnea,

Table 37-1. Causes of Anterior Mediastinal Mass

1. The "Terrible T's": T-cell lymphoma/leukemia Thymoma Thyroid carcinoma Teratoma/germ cell tumors
2. Hodgkin's disease
3. Non-Hodgkin's lymphoma
4. Neuroblastoma
5. Cystic hygroma
6. Congenital diaphragmatic hernia

hemoptysis, orthopnea), facial edema, cardiovascular instability due to impaired venous return, and central nervous system findings (e.g., headache, mental status changes).

35. How should a patient with an anterior mediastinal mass be studied?

Very carefully! After the initial identification of anterior mediastinal mass on a chest radiograph, perform further evaluation in conjunction with a multidisciplinary team. Because these patients are at significant risk for airway compromise, obtaining a CT scan may be unsafe. Alternative means of diagnosis may be required (e.g., peripheral blood smear and flow cytometry, bone marrow aspiration, lymph node biopsy, or thoracentesis).

Perger L, Lee EY, Shamberger RC: Management of children and adolescents with a critical airway due to compression by an anterior mediastinal mass. J Pediatr Surg 2008;43(11):1990-1997.

Key Points: Metabolic Disturbances Seen in Oncology Patients

1. Hyperkalemia: Seen with TLS
2. Hyperphosphatemia: Seen with TLS
3. Hyperuricemia: Seen with TLS
4. Hypercalcemia: Seen in patients being treated with cis-retinoic acid or those with significant bony metastases
5. Hypernatremia: Seen in patients with diabetes insipidus, which can occur frequently in patients with central nervous system tumors or Langerhans cell histiocytosis
6. Hyperglycemia: Seen in patients on steroids or asparaginase

36. What are the pitfalls of managing the ABCs (airway, breathing, and circulation) of a patient with an anterior mediastinal mass?

Endotracheal intubation may be difficult because of tracheal or bronchial compression. Cardiac output may be compromised by positive-pressure ventilation. Be particularly wary of the patient who has worsening respiratory difficulty when placed in the recumbent position, and avoid sedating patients and positioning patients to lie flat because doing so may result in cardiorespiratory arrest.

37. A 4-year-old girl with lymphoma presents with shortness of breath. What are some possible causes of her symptoms?

Causes of shortness of breath in patients with cancer can be seen in Table 37-2.

38. Describe some of the symptoms and signs of neuroblastoma.

Symptoms:

- Abdominal pain
- Bone pain

Table 37-2. Causes of Shortness of Breath in Patients with Cancer

Superior vena cava (SVC) syndrome: Compression or thrombosis of the SVC can cause dyspnea and may result in facial plethora, jugular venous distention, and headache
Superior mediastinal syndrome: Compression of the trachea by tumor; often used interchangeably with SVC syndrome
Pleural effusions
Pericardial effusion
Cardiomyopathy: Commonly associated with anthracycline chemotherapeutics
Arrhythmias: Electrolyte disturbances
Pneumonia
Pulmonary embolism
Anemia

- Back pain
- Constipation
- Fever
- Weight loss

Signs:

- Abdominal mass
- Proptosis
- Horner's syndrome
- Raccoon eyes
- Hypertension

39. An 8-year-old girl who has reported back pain intermittently for several months presents to the ED with a sudden inability to move her right leg. What does this presentation suggest, and what is the treatment?

Spinal cord compression can present with back pain (typically worse when lying down), leg paresis or paralysis, and bowel and bladder incontinence or retention. Examination may reveal back tenderness, leg weakness, decreased rectal tone, and hyperreflexia or hyporeflexia. Plain radiography may reveal vertebral abnormalities, but the diagnostic study of choice is magnetic resonance imaging. Initiate treatment immediately to minimize permanent neurologic sequelae; such treatment may include a combination of chemotherapy, radiation therapy, and surgical decompression.

40. What is Tumor Lysis Syndrome (TLS)?

TLS is a metabolic syndrome of hyperkalemia, hyperphosphatemia, hyperuricemia, hypocalcemia, and acute kidney injury resulting from the release of intracellular contents from dying cells. TLS is seen most commonly in cancers with large tumor burdens and rapid cellular turnover, such as lymphomas and leukemias. Although TLS usually occurs soon after initiation of chemotherapy, patients can present at the time of initial diagnosis with TLS, especially those who present with Burkitt's lymphoma. TLS can cause numerous complications, such as arrhythmias and renal failure.

Howard SC, Jones DP, Pui CH: The tumor lysis syndrome. N Engl J Med 2011;364(19):1844-1854.

41. How is TLS treated?

Treatment of TLS consists of protection of renal function. The mainstay of treatment is IV hydration. Administer allopurinol or rasburicase to reduce uric acid, and start aluminum hydroxide or calcium carbonate for treatment of hyperphosphatemia. Hyperkalemia can be treated with a potassium-binding resin, calcium gluconate, sodium bicarbonate, or insulin with glucose, depending on the patient's clinical status. Urinary alkalinization is controversial. Hemodialysis may be used for uncontrolled TLS and severe kidney injury.

42. A 5-year-old girl with ALL presents with fever and hypotension. Antibiotics and IV fluid resuscitation are started immediately, but she has persistent hypotension. In addition to fluids and vasopressors, what other therapies should be considered?

Consider adrenal suppression in patients with persistent hypotension who recently (in the past year) have been treated with steroids. Protocols for ALL usually use frequent and sometimes prolonged courses of corticosteroids. These patients may need stress-dose hydrocortisone. If worsening hypotension is associated with flushing of the central line or infusion of IV antibiotics through the central line, be suspicious of a central line infection.

Gordijn MS, Gemke RJ, van Dalen EC, et al: Hypothalamic-pituitary-adrenal (HPA) axis suppression after treatment with glucocorticoid therapy for childhood acute lymphoblastic leukaemia. Cochrane Database Syst Rev 2012;5:CD008727.

CHAPTER 38

INFECTIOUS DISEASE EMERGENCIES

Robert P. Olympia, Kaynan Doctor, Robert Wilkinson, Sanjeev Swami, and Stephen Eppes

1. A 10-day-old, full-term male infant presents to the emergency department (ED) with a 1-day history of being fussy but consolable, slightly decreased oral intake, and a temperature of 38.5° C rectally. What is the risk for serious bacterial illness?

A published study demonstrated that the incidence in infants with fever who are younger than 28 days old is approximately 13%. Among 372 such infants, the cause of fever was determined to be a nonspecific viral syndrome (65%), serious bacterial infection (SBI) (12%), aseptic meningitis (8.1%), otitis media (5.1%), bronchiolitis (5.1%), nonbacterial gastroenteritis (2.7%), or viral stomatitis (1.0%).

Kadish H, Loveridge B, Tobey J, et al: Applying outpatient protocols in febrile infants 1-28 days of age: Can the threshold be lowered? Clin Pediatr 2000;39:81-88.

Teague JA, Harper MG, Bachur R, et al: Epidemiology of febrile infants 14-28 days of age. Pediatr Res 2003;53:214A.

2. Which bacterial agents are of most concern in an infant less than 28 days old presenting to the ED with a fever?

Group B streptococci (GBS), gram-negative enteric organisms (*Escherichia coli*, *Enterococcus* spp.), *Listeria monocytogenes*, and, less commonly, *Streptococcus pneumoniae*, *Haemophilus influenzae*, *Staphylococcus aureus*, *Neisseria meningitidis*, and *Salmonella* spp.

3. A 26-day-old infant presents to the ED with a 1-day history of fever up to 38° C rectally. What should your initial management include?

Initial management of a newborn infant younger than 28 days old presenting to the ED with a temperature of 38.0° C is as follows:

- Perform a complete history (including prenatal history) and physical examination of the newborn.
- Obtain laboratory studies, including a complete blood count (CBC) with differential and blood culture; urine obtained by catheterization or suprapubic aspiration for urinalysis (UA), Gram stain, and culture; cerebrospinal fluid (CSF) for protein/glucose, cell count, Gram stain, and bacterial/viral cultures; chest radiograph if there are signs of respiratory distress (tachypnea, cyanosis, wheezing, retractions, grunting, nasal flaring, rales, rhonchi, or decreased breath sounds); and stool for heme testing and culture if bloody or watery stool is noted.
- Admit patient to the hospital and administer parenteral antibiotics (ampicillin *plus* cefotaxime or gentamicin).
- Consider intravenous (IV) acyclovir if neonatal herpes is suggested by history or physical examination.

4. Can an infant younger than 28 days of age with fever be classified as low risk for an SBI?

Several screening tools originally applied to infants older than 28 days (Boston and Philadelphia protocols for the outpatient management of infants with fever) have been applied to infants younger than 28 days old presenting with fever to predict which of these newborns are less likely to have a positive bacterial culture on initial evaluation. Based on several studies, for infants less than 1 month old, risk stratification criteria are unreliable, and

therefore, these infants should have a full sepsis evaluation and be hospitalized for empiric antibiotic treatment pending culture results.

Baker MD, Bell LM: Unpredictability of serious bacterial illness in febrile infants from birth to 1 month of age. Arch Pediatr Adolesc Med 1999;153:508-511.

Kadish HA, Loveridge B, Tobey J, et al: Applying outpatient protocols to febrile infants 1-28 days of age: Can the threshold be lowered? Clin Pediatr 2000;39:81-88.

5. A 6-week-old male infant presents to the ED with a temperature of 38.4° C. The infant appears nontoxic and is without an obvious source for the fever. What is your workup?

There is variability in the recommended workup of this infant. Three common strategies for the management of infants in this age group include the Rochester criteria (less than 60 days), the Philadelphia criteria (29-60 days), and the Boston criteria (28-89 days). These studies have tested screening tools to identify low-risk infants having SBIs. Workup and laboratory values constituting low risk vary between the three sets of criteria, as does the sensitivity and negative predictive values. Thus, base the decision to manage fever in an infant as an outpatient on not only these criteria but also the experience of the practitioner, the reliability of follow-up, and the observational skills of the infant's guardians.

A CBC, blood culture, UA, and urine culture is recommended. Consider performing a lumbar puncture (according to the Philadelphia criteria).

Baker MD, Avner JR: The febrile infant: What's new? Clin Pediatr Emerg Med 2008;9:213-220.

6. What is the cause of a temperature of 38.0° C in infants age 1 to 3 months who present to the ED?

A published study of 422 such infants determined the following sources:

- Viral syndrome, 54.0%
- Nonbacterial gastroenteritis, 16.4%
- Aseptic meningitis, 11.8%
- Serious bacterial illness, 10.2% (growth of pathogen in cultures of blood, spinal fluid, urine, stool)
- Bronchiolitis, 4.7%
- Pneumonia, 1.9%
- Otitis media, 0.5%
- Varicella infection, 0.2%
- Conjunctivitis, 0.2%

Baker MD, Bell LM, Avner JR: The efficacy of routine outpatient management without antibiotics of fever in selected infants. Pediatrics 1999;103:627-630.

7. A 2-month-old presents with a temperature of 40.5° C and otherwise appears nontoxic. Do infants with hyperpyrexia have a higher risk of having an SBI?

A retrospective study of infants younger than 3 months of age demonstrated that the prevalence of SBI among febrile infants with temperatures higher than 40° C was 38%, compared with 8.8% of those with temperatures lower than 40° C.

Stanley R, Pagon Z, Bachur R: Hyperpyrexia among infants younger than 3 months. Pediatr Emerg Care 2005;21:291-294.

8. What is the most common cause of sepsis in newborns?

Early-onset (birth to 7 days) GBS infections, which may be secondary to maternal obstetric complications, prematurity, or lack of prophylactic antibiotics prior to delivery. Late-onset GBS infection (7 days to 3 months) is uncommonly associated with these factors (Table 38-1).

9. A 54-day-old infant presents to the ED with a fever of 38.6° C 1 day after receiving his 2-month immunizations. He otherwise appears well. What should your workup be?

One study investigated the prevalence of SBI in febrile infants age 6 to 12 weeks without a source of infection who have received immunizations within the preceding 72 hours. Among febrile infants, the prevalence of SBI is lower in the initial 24 hours following immunizations (0.6% vs. 8.9%). The authors recommended only urine testing (no blood or CSF testing) in febrile infants who present within 24 hours of immunization. Manage infants

Table 38-1. Early-Onset Versus Late-Onset Group B Streptococcal Infections

TYPE OF GBS INFECTION	USUAL CLINICAL PRESENTATIONS	COMMENTS
Early-onset GBS infection	Septicemia (25-40%) Meningitis (5-15%) Respiratory illness (35-55%)	5-20% mortality rate
Late-onset GBS infection	Meningitis (30-40%) Bacteremia without focus (40-50%) Osteomyelitis/septic arthritis (5-10%)	2-6% mortality rate

GBS, group B streptococcus.

who present with fever more than 24 hours after immunization similarly to infants without recent immunization.

Wolff M, Bachur R: Serious bacterial infection in recently immunized young febrile infants. Acad Emerg Med 2009;16:1284-1289.

10. Has the introduction of the heptavalent pneumococcal conjugate vaccine (PCV7) affected the incidence of occult bacteremia?

A retrospective cohort study of febrile infants age 2 to 36 months conducted from 2001 to 2003 demonstrated that 3 of 329 blood cultures yielded a pathogenic bacterium (0.91% [95% confidence interval, 0-2.4%]); all were *S. pneumoniae*. One patient had a nonvaccine serotype, one was unimmunized, and the third was infected with an unknown serotype.

Stoll ML, Rubin LG: Incidence of occult bacteremia among highly febrile young children in the era of the pneumococcal conjugate vaccine. Arch Pediatr Adolesc Med 2004;158:671-675.

11. How helpful is C-reactive protein (CRP) in detecting SBI in febrile children? How about other biomarkers for common infections?

In a prospective cohort study of febrile children (>39° C) age 1 to 36 months with a clinically undetectable source of fever, only CRP remained a predictor of SBI after multivariate logistic regression analysis compared with white blood cell (WBC) count and absolute neutrophil count. Receiver-operating characteristic analysis demonstrated CRP to be superior to WBC count and absolute neutrophil count. A CRP cutoff point of 7 mg/dL maximized both sensitivity and specificity.

Several novel disease-specific biomarkers have been studied in the detection of common infectious disease in the ED, such as acute respiratory infections (serum [25-(OH)D3] for bronchiolitis, serum [respiratory syncytial virus (RSV) f-protein] for RSV, and exhaled nitric oxide, nasal lavage nitric oxide, and interleukin 6 for influenza-like disease), acute central nervous system (CNS) infections (CNS nitric oxide and apolipoprotein E for bacterial meningitis), and sepsis/septic shock (plasma/urine nitric oxide).

Aranke M, Mian AI: A biomarker-based approach to infectious disease in the pediatric emergency department. Clin Pediatr Emerg Med 2013;14:95-101.

Pulliam PN, Attia MW, Cronan KM: C-reactive protein in febrile children 1 to 36 months of age with clinically undetectable serious bacterial infection. Pediatrics 2001;108:1275-1279.

12. A 6-week-old infant presents to the ED in January with a fever of 38.6° C and cough and is noted to have wheezing bilaterally on physical examination. Rapid RSV is positive. Should this affect your fever workup?

A multicenter study investigating infants younger than 60 days with fever determined that the incidence of SBI was less in infants with (+) RSV compared to the overall incidence (7% vs. 11.4%). In infants with RSV, 5.4% also had a urinary tract infection (UTI), 1.1% also had bacteremia, and 0% also had meningitis. Therefore, in infants less than 60 days of age with fever and (+) RSV, a lumbar puncture may not be necessary.

Levine DA, Platt SL, Dayan PS, et al: Risk of serious bacterial infection in young febrile infants with respiratory syncytial virus infections. Pediatrics 2004;113(6):1728-1734.

13. Are febrile infants with influenza at lower risk for an SBI?

A retrospective cross-sectional study of infants age 0 to 36 months presenting to the ED with fever demonstrated that febrile infants with influenza A had a lower prevalence of bacteremia (0.6% vs. 4.2%), UTIs (1.8% vs. 9.9%), consolidated pneumonia (25.4% vs. 41.9%), meningitis (0% vs. 2.2%), or any SBI (9.8% vs. 28.2%) compared with infants without influenza A.

Smitherman HF, Caviness AC, Macias CG: Retrospective review of serious bacterial infections in infants who are 0 to 36 months of age and have influenza A infection. Pediatrics 2005;115:710-718.

14. Distinguish between the presentations of preseptal and orbital cellulitis in children.

Preseptal versus orbital cellulitis in children is covered in Table 38-2. Preseptal cellulitis is usually due to minor trauma and *S. aureus* is the likely organism responsible.

Almost 75% of the cases of orbital cellulitis result from an extension of a sinus infection (most often the ethmoid sinus). The usual infectious agents are similar bacteria to those seen in bacterial sinusitis and include *S. pneumoniae* and *H. influenzae*.

15. What is the initial approach to a patient presenting with suspected orbital cellulitis?

- History: Ear, nose, throat, or sinus infection, tooth pain or recent dental disease, or a history of immunocompromise
- Examination: Afferent pupillary defect, pain with extraocular movements, proptosis, decreased color vision perception. Also check for mental status change or neck stiffness.
- Investigations:
  - WBC count (often reveals leukocytosis with predominance of bands)
  - Blood cultures obtained before antibiotics
  - Orbital computed tomography (CT) with contrast with thin and coronal cuts, including frontal lobes and paranasal sinuses
  - Lumbar puncture if meningeal signs are present (only after CT of the head shows no signs of increased intracranial pressure)
  - Admission to the hospital for IV antibiotics (IV ampicillin-sulbactam or piperacillin-tazobactam). Other antibiotic choices include trimethoprim-sulfamethoxazole, vancomycin, or clindamycin, especially in areas where methicillin-resistant *S. aureus* (MRSA) is prevalent.
  - Consultation with ophthalmology, otolaryngology, infectious disease, or neurosurgery, as necessary

Gerstenblith AT, Rabinowitz MP: The Wills Eye Manual: Office and Emergency Room Diagnosis and Treatment of Eye Disease, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2012, pp 159-162.

16. What are the indications for hospital admission of infants and children who present with preseptal cellulitis?

- Age younger than 1 year
- Moderate to severe preseptal cellulitis
- Inability to rule out orbital cellulitis
- Incomplete vaccination against *H. influenzae* type b (Hib)
- Toxic appearance or signs of meningitis
- Anorexia or inability to tolerate oral medications/antibiotics
- Presence of subcutaneous abscess
- Failure of outpatient management

Zaoutis LB, Chiang VW: Comprehensive Pediatric Hospital Medicine. Philadelphia, Mosby, 2007, pp 355-357.

17. For children discharged from the ED with the diagnosis of preseptal cellulitis, which antibiotics are best?

Amoxicillin-clavulanate 25 to 40 mg/kg/day, cefpodoxime 10 mg/kg/day, and cefdinir 14 mg/kg/day can all be given in two divided doses. In penicillin-allergic patients, give trimethoprim-sulfamethoxazole (8 to 12 mg/kg/day trimethoprim with 40 to 60 mg/kg/day sulfamethoxazole) in two divided doses. If MRSA is suspected, trimethoprim-sulfamethoxazole or clindamycin is an appropriate choice. Give all antibiotics orally for 10 days.

Gerstenblith AT, Rabinowitz MP: The Wills Eye Manual: Office and Emergency Room Diagnosis and Treatment of Eye Disease, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2012, pp 146-149.

Table 38-2. Preseptal Versus Orbital Cellulitis in Children

FEATURE	PRESEPTAL CELLULITIS	ORBITAL CELLULITIS
Location	Infection of the eyelids and periorbital soft tissues anterior to the orbital septum	Infectious process posterior to the orbital septum involving the tissues within the orbit (eye, fat, muscles, optic nerve)
Cause	Direct inoculation following trauma Direct spread from adjacent structures (skin)	Secondary to bacterial spread from ethmoid sinuses Extension of bacterial infection from sinuses (75%) Direct inoculation from penetrating trauma or surgery Spread from adjacent structure (skin)
Clinical presentation		
Fever/malaise	+/−	Usually +
Orbital/eye pain	+/−	+
Conjunctival hyperemia or swelling	+	+
Upper-/lower-eyelid edema or erythema	+	+
Signs of external trauma (insect bite, etc.)	+	+/−
Fluctuance	+/−	+/−
Photophobia	−	+/−
Proptosis*	−	+
Orbital pain	−	+
Pain on eye movement	−	+
Normal movement of eye*	+	−
Visual loss or abnormal pupillary reactivity*	−	+ (if severe)
Signs of cavernous sinus thrombosis, meningitis, or intracranial abscess formation	−	+ (if severe)

+ indicates present, − indicates absent.
*The three most important features.

18. What is ophthalmia neonatorum?

Ophthalmia neonatorum, also known as *neonatal conjunctivitis*, is defined as conjunctival inflammation during the first month of life. Infants typically present with eyelid edema, chemosis, conjunctival hyperemia, and purulent or mucopurulent ocular discharge. Maternal history may reveal exposure to sexually transmitted diseases (STDs) or previous genital infections. Birth history may reveal premature rupture of membranes in infants born via

cesarean section or vaginal discharge with a vaginal delivery. Infectious causes for ophthalmia neonatorum include *Chlamydia trachomatis*, *Neisseria gonorrhoeae*, and, less frequently, *S. aureus*, *S. pneumoniae*, *Haemophilus* species, enterococci, and viral agents (herpes simplex virus [HSV], adenovirus, coxsackievirus, cytomegalovirus, and echovirus).

Time of onset and type of eye discharge may help with the diagnosis, but diagnosis can be made using a Gram stain, culture on chocolate or Thayer-Martin agar (*N. gonorrhoeae*), chlamydial culture, direct immunofluorescent monoclonal antibody testing for chlamydia, and HSV immunochemical testing. Order this on every infant with these symptoms.

19. Distinguish between conjunctivitis caused by *C. trachomatis* and that caused by *N. gonorrhoeae*.

See Table 38-3 for *C. trachomatis* versus *N. gonorrhoeae* conjunctivitis.

20. A 5-year-old girl presents to the ED with "burning and itchy" eyes. She describes a sensation of "chalk in her eyes," with some blurry vision. On physical examination, she has bilateral conjunctival hyperemia, chemosis, and ocular discharge and has preauricular lymph nodes bilaterally. What is the differential diagnosis?

The presence of preauricular lymph nodes and conjunctivitis is associated with conjunctivitis caused by *N. gonorrhoeae* and adenoviral keratoconjunctivitis. Adenoviral keratoconjunctivitis is often associated with fever, other upper respiratory symptoms, or vomiting and diarrhea.

21. What is the treatment for adenoviral keratoconjunctivitis?

Treatment includes cold compresses to the eyes and oral acetaminophen to help with ocular discomfort, gentle removal of conjunctival membranes with a cotton swab, and possibly topical corticosteroids for marked follicular conjunctivitis and pseudomembrane formation. Ophthalmologic consultation is strongly recommended before using steroids. Prevention of transmission includes frequent handwashing and keeping the child out of day care or school until complete resolution of the conjunctival hyperemia.

Table 38-3. *Chlamydia Trachomatis* Versus *Neisseria Gonorrhoeae* Conjunctivitis

FEATURES	CHLAMYDIA TRACHOMATIS	NEISSERIA GONORRHOEAE
Presentation	First 1-2 weeks of life	72-96 hours after birth
Distinctive clinical features	Initially mild swelling, hyperemia, tearing, mucopurulent discharge Unilateral or bilateral	Initially mild conjunctival hyperemia but can progress to severe chemosis with purulent conjunctival discharge and marked eyelid edema
Potential complications	Self-limited Rarely conjunctival or corneal-scarring Potential development of upper and lower respiratory tract infections	Potential corneal ulceration and perforation Septicemia, meningitis, or arthritis
Diagnosis	ELISA, EIA, direct antibody tests, PCR, DNA-hybridization probe	Gram-negative diplococci seen on Gram stain
Treatment	Oral erythromycin elixir 50 mg/kg/d for 2 weeks with erythromycin ointment four times a day	Parenteral ceftriaxone or cefotaxime; also treat for concurrent suspected chlamydial infection

EIA, enzyme immunoassay; ELISA, enzyme-linked immunosorbent assay; PCR, polymerase chain reaction.
Gerstenblith AT, Rabinowitz MP: The Wills Eye Manual: Office and Emergency Room Diagnosis and Treatment of Eye Disease, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2012, pp 193-194.

22. What organisms are associated with deep neck infections (peritonsillar abscess, retropharyngeal abscess, and lateral pharyngeal abscess) in children?

Aerobic (*Streptococcus pyogenes*, *S. aureus*, and *H. influenzae*) and anaerobic (*Prevotella*, *Fusobacterium*, *and Peptostreptococcus* spp.) organisms are typically associated with deep neck infections in children. Almost 66% of deep neck abscesses contain β-lactamase–producing organisms.

23. Distinguish the features of deep neck infections in children.

See Table 38-4 for the features of deep neck infections in children.

24. Which IV antibiotics are appropriate for retropharyngeal abscess?

Treatment with a penicillin alone is likely to be unsuccessful. Although there is no consensus, the following are appropriate choices: a second- or third-generation cephalosporin combined with clindamycin or a combination of a second- or third-generation cephalosporin and metronidazole or amoxicillin-clavulanate or ampicillin-sulbactam, or piperacillin-tazobactam. A combination including clindamycin or vancomycin should be considered in cases of suspected MRSA.

25. Do all children with retropharyngeal abscess require surgical treatment?

Recent evidence suggests that the majority of patients with retropharyngeal abscess can be treated successfully with high-dose IV antibiotics. Consider surgical incision and drainage in cases that do not respond to antibiotic therapy or in cases of persistent large abscess and airway compromise.

Table 38-4. Features of Deep Neck Infections in Children

DEEP NECK INFECTION	AGE ASSOCIATED	HISTORICAL/ CLINICAL FEATURES	MANAGEMENT
Peritonsillar abscess	Adolescents and young adults	Difficulty swallowing or speaking, throat pain radiating to the ear, foul breath, swelling of one tonsil with lateral displacement of the uvula, trismus, drooling	Hospitalization, possible surgical drainage, intravenous (IV) antibiotics, tonsillectomy
Retropharyngeal abscess	<5 years	High fever, difficulty swallowing, drooling, dysphagia, dyspnea, hyperextension of the neck, unilateral posterior pharyngeal fullness, torticollis	Hospitalization, lateral neck radiograph (retropharyngeal space > half diameter of adjacent vertebral body), possible surgical drainage, IV antibiotics
Lateral pharyngeal abscess	Older children, adolescents, and young adults	Ill-appearing, high fever, odynophagia, dysphagia, dyspnea; may involve anterior compartment (swelling of parotid region, trismus) or posterior compartment (minimal pain or trismus)	Hospitalization, possible surgical drainage, IV antibiotics

Craig FW, Shunk JE: Retropharyngeal abscesses in children: Clinical presentation, utility of imaging, and current management. Pediatrics 2003;111:1394-1398.
Wong DKC, Brown C, Mills N, et al: To drain or not to drain—Management of pediatric deep neck abscesses: A case-control study. Int J Pediatr Otorhinolaryngol 2012;76(12):1810-1813.

26. A 3-year-old boy with a history of mild to moderate eczema presents to the ED with ear pain and drainage. On physical examination, his tympanic membrane appears normal, although swelling of the ear canal makes it difficult to view the entire tympanic membrane. There is pain on movement of the tragus. There is no lateral displacement of the ear and no signs of mastoiditis. What is the likely diagnosis?

The clinical findings are consistent with *otitis externa*. Otitis externa is associated with *Pseudomonas* spp. in the majority of cases along with *Escherichia*, *Proteus*, *Enterobacter*, and *Klebsiella* spp. Staphylococcus and streptococcus are etiologic agents when otitis externa occurs as an extension of a focal infection. Additionally, 5% of cases can arise due to fungal infections, primarily *Aspergillus* and *Candida*. There is an increased incidence of otitis externa in children with eczema and other dermatologic disorders, as well as in children with a history of swimming, extensive cleaning of the ear, hearing aid use, immunocompromised state, and histiocytosis X.

Long SS: Principles and Practice of Pediatric Infectious Disease, 4th ed. St. Louis, Elsevier, 2012, pp 220-222.

27. How should otitis externa be managed?

Management of otitis externa includes the following: In addition to adequate analgesia, a 7-day course of topical antibiotic drops such as ofloxacin, ciprofloxacin combined with either hydrocortisone or dexamethasone, or polymyxin-neomycin-hydrocortisone (Neosporin) is the first-line treatment. If there is severe canal edema, administration using a wick may be useful. Daily lavage and avoidance of submersion of the ear until canal edema has resolved is also recommended. Culturing of the exudate is indicated only in severe, refractory cases or when a fungal cause is suspected.

Long SS: Principles and Practice of Pediatric Infectious Disease, 4th ed. St. Louis, Elsevier, 2012, pp 220-222.

28. What is the best treatment for a young child with a runny nose, sore throat, cough, and fever?

The "common cold" or rhinosinusitis is caused by viruses, primarily rhinoviruses and coronaviruses. Rhinovirus has been associated with wheezing in the asthmatic child. Coronavirus causes most of the colds in young children. Do not use antibiotics to treat the common cold because of their lack of efficacy and, more importantly, to prevent the further emergence of resistant bacteria. Over-the-counter cold remedies, such as antihistamines and decongestants, have not shown significant benefit, and in younger children they may have significant side effects such as somnolence or hyperactivity. Nasal saline drops can be safely used for nasal congestion. Avoid aggressive suctioning. Additionally, vapor rub has been clinically shown to help with symptomatic relief and sleep disturbance associated with upper respiratory infections.

Paul IM, Beiler JS, King TS, et al: Vapor rub, petrolatum, and no treatment for children with nocturnal cough and cold symptoms. Pediatrics 2010;126(6):1092-1099.

29. List the three major causes of exudative pharyngitis in children.

Exudate refers to white or gray debris on the tonsils or pharynx. Causes include:

- Group A β-hemolytic streptococcus (GABHS)
- Adenoviruses
- Epstein-Barr virus

In developing countries, include *Corynebacterium diphtheriae* in this list. In sexually active adolescents or abused children, consider *N. gonorrhoeae*. More recently, *Arcanobacterium haemolyticum* has been shown to cause an exudative pharyngitis, especially in adolescents.

30. A 7-year-old has a fever, sore throat, tender anterior cervical lymph nodes, and lack of significant upper respiratory tract symptoms. Are these clinical features suggestive of group A streptococcus (GAS) pharyngitis?

Unfortunately, there are no clinical findings that are specific or sensitive for streptococcal pharyngitis. However, the following features are commonly associated with GAS throat infection:

- Age 5 to 15 years old
- Fever
- Headache
- Nausea, vomiting, abdominal pain
- Tonsillopharyngeal inflammation
- Patchy tonsillopharyngeal exudates
- Palatal petechiae
- Anterior cervical lymphadenopathy
- Presentation during winter or early spring
- History of streptococcus pharyngitis exposure
- Scarlatiniform rash

Features that are less commonly associated with GAS pharyngitis and more associated with a viral cause include the following:

- Conjunctivitis
- Coryza
- Cough
- Diarrhea
- Hoarse voice
- Ulcerative stomatitis
- Viral exanthem

It is very uncommon for children younger than 3 years old to develop these symptoms as a complication of GAS pharyngitis. They are also very unlikely to develop rheumatic fever at this age.

Shulman ST, Bisno AL, Clegg HW, et al: Clinical practice guideline for the diagnosis and management of group A streptococcal pharyngitis: 2012 update by the Infectious Diseases Society of America. Clin Infect Dis 2012;55(10):e86-e102.

31. What is the drug of choice for the treatment of streptococcal pharyngitis?

Amoxicillin (50 mg/kg/day) or penicillin for a full 10 days to prevent rheumatic fever is the oral drug of choice for non–penicillin-allergic patients. A first-generation cephalosporin (if there has been no anaphylactic penicillin reaction) can be used in the penicillin-allergic patient. Alternatively 10-day courses of clindamycin or clarithromycin is appropriate. A single dose of intramuscular (IM) penicillin G benzathine (600,000 U if <27 kg or 1,200,000 U if >27 kg) may be indicated in specific cases in which nonadherence is a concern.

Shulman ST, Bisno AL, Clegg HW, et al: Clinical practice guideline for the diagnosis and management of group A streptococcal pharyngitis: 2012 update by the Infectious Diseases Society of America. Clin Infect Dis 2012;55(10):e86-e102.

32. How is otitis media diagnosed clinically?

Per the new American Academy of Pediatrics (AAP) guidelines, acute otitis media is diagnosed by visualizing a moderately to severely bulging tympanic membrane or new onset of otorrhea not due to chronic otitis externa. Alternatively, acute otitis media may be diagnosed by visualizing a mildly bulging tympanic membrane with a recent (<48 hour) history of ear pain; tugging, holding, or rubbing; and visualization of an intensely erythematous tympanic membrane.

Lieberthal AS, Carroll AE, Chonmaitree T, et al: The diagnosis and management of acute otitis media. Pediatrics 2013;131(3):e964-e999.

33. Which pathogens are implicated in acute otitis media?

- *S. pneumoniae* (35%)
- *H. influenzae*, nontypeable (25%)
- *Moraxella catarrhalis*
- **Viruses:** Adenoviruses, coxsackievirus, measles virus, parainfluenza virus, rhinoviruses, RSV
- **Others:** Anaerobes; *Chlamydia*, *Mycoplasma*, and *Staphylococcus* spp.; *Mycobacterium tuberculosis*

Lieberthal AS, Carroll AE, Chonmaitree T, et al: The diagnosis and management of acute otitis media. Pediatrics 2013;131(3):e964-e999.
Pelton SI: Otitis media: Re-evaluation of diagnosis and treatment in the era of antimicrobial resistance, pneumococcal conjugate vaccine, and evolving morbidity. Pediatr Clin North Am 2005;52:711-728.

34. What is the drug of choice for treatment of acute otitis media?

High-dose amoxicillin (90 mg/kg) given in a divided dose twice daily for 10 days is the first-line treatment for otitis media as long as there is no penicillin allergy, there is no concurrent purulent conjunctivitis, and amoxicillin has not been prescribed within the last 30 days. Recurrent acute otitis media may require the use of other antibiotics to address the problem of β-lactamase–producing bacteria or highly resistant *S. pneumoniae*.

Lieberthal AS, Carroll AE, Chonmaitree T, et al: The diagnosis and management of acute otitis media. Pediatrics 2013;131(3):e964-e999.

35. What is the mechanism of pneumococcal resistance?

Pneumococcal resistance is mediated by alterations in the penicillin-binding proteins. Resistance of *H. influenzae* and *M. catarrhalis* is mediated by a β-lactamase. This difference has important therapeutic implications, because β-lactamase–stable agents, such as amoxicillin-clavulanate, are more effective against *H. influenzae* and *M. catarrhalis* but do not provide any advantage over amoxicillin in treating penicillin-resistant pneumococci.

36. Which antibiotics are considered second-line agents for the treatment of acute otitis media?

If there is purulent conjunctivitis, recent amoxicillin administration, or a history of otitis media refractory to penicillin, second-line treatment in the form of an antibiotic with additional β-lactamase coverage such as amoxicillin-clavulanate (90 mg/kg amoxicillin) is recommended.

Alternative therapies for otitis media include cefdinir, cefuroxime, or cefpodoxime. Reserve IM ceftriaxone for children who are vomiting and cannot tolerate oral medication, or for those with possible concomitant occult bacteremia.

Lieberthal AS, Carroll AE, Chonmaitree T, et al: The diagnosis and management of acute otitis media. Pediatrics 2013;131(3):e964-e999.

37. You decide to intubate the trachea of a young patient who presents with severe respiratory distress and stridor. On endotracheal intubation, purulent tracheal secretions are seen. What is the likely diagnosis?

Bacterial tracheitis, also called pseudomembranous tracheitis. This infection requires immediate attention and treatment because it is potentially life threatening. Copious amounts of secretions can obstruct the trachea or a major bronchus. This condition may resemble croup, which may be a predisposing risk factor. Because of inspiratory and expiratory wheezing, excessive drooling, and tripod posture, foreign body aspiration and epiglottitis may also be part of the differential diagnosis. The most common organisms recovered from infected patients are *S. aureus*, *H. influenzae*, *S. pyogenes*, *S. pneumoniae*, and *M. catarrhalis*. Base antimicrobial selection on these pathogens. A third-generation cephalosporin combined with a penicillinase-resistant penicillin administered intravenously is a reasonable choice. Vancomycin is appropriate if MRSA is suspected.

38. What are the common causes of stomatitis? How can they be distinguished?

Inflammation of the oral mucous membrane (stomatitis) is a common finding in children, usually presenting with ulcers or vesicles ("canker sores"). The differential diagnosis is based on the location of the lesions and clinical picture:

- **Buccal stomatitis** may be due to infectious agents, Behçet's syndrome, or trauma.
- **Gingivitis** may be due to HSV or enteroviruses (coxsackievirus: hand-foot-mouth disease). Herpes (HSV) usually causes lesions on the lips and anterior part of the mouth. Coxsackievirus usually involves the posterior pharynx.
- **Gingivostomatitis** may be due to an infectious agent (HSV, *Candida albicans*) or Stevens-Johnson syndrome.
- **Glossitis** may be due to GAS or HSV.

Recurrent lesions may be due to abnormalities in the immune system, such as in cyclic neutropenia or chemotherapy-induced neutropenia and chronic granulomatous disease.

In association with recurrent fever, stomatitis may be due to PFAPA (**p**eriodic **f**ever, **a**denitis, **p**haryngitis, and **a**phthous stomatitis).

Fisher RG, Boyce TG: Moffet's Pediatric Infectious Diseases: A Problem-Oriented Approach, 4th ed. Philadelphia, Lippincott Williams & Wilkins, 2005, pp 62-67.

39. What are the clinical manifestations and management of a child with suspected acute mastoiditis?

Mastoiditis is a severe complication of otitis media and occurs when infection spreads from the middle ear space into the mastoid portion of the temporal bone. It commonly manifests as fever, postauricular tenderness, and erythema, along with retroauricular swelling resulting in a downward and outward deviation of the auricle. Significant external otitis may also be present. CT may be helpful when intracranial complications of mastoiditis are suspected. Admit children with mastoiditis to the hospital and treat with IV antibiotics and myringotomy with tympanostomy tube placement. Meningitis and brain abscess are potential complications of mastoiditis. Perform a lumbar puncture after the CT scan if the patient presents with meningeal signs or altered mental status, headaches, or a neurologic deficit.

40. What are the typical organisms causing sinusitis?

The typical organisms associated with acute sinusitis (according to correlations with otitis media) include the following:

- *S. pneumoniae* (30%)
- Nontypeable *H. influenzae* (30%)
- *M. catarrhalis* (10%)

S. aureus is rarely isolated unless the sinusitis is associated with a dental infection. However, it is common when sinusitis is complicated by orbital or CNS involvement.

41. What clinical symptoms suggest that a child has bacterial sinusitis?

The most recent AAP guidelines suggest that a presumptive diagnosis of bacterial sinusitis should be made in a child/adolescent between the ages of 1 and 18 years presenting with an upper respiratory infection and any of the following additional features:

- Persistent illness, i.e., nasal discharge (of any quality) or daytime cough or both lasting more than 10 days without improvement
- Worsening course, i.e., worsening or new onset of nasal discharge, daytime cough, or fever after initial improvement
- Severe onset, i.e., concurrent fever (temperature $\geq 39°$ C/102.2° F) and purulent nasal discharge for at least 3 consecutive days

Wald ER, Applegate KE, Bordley C, et al: Clinical practice guideline for the diagnosis and management of acute bacterial sinusitis in children aged 1 to 18 years. Pediatrics 2013;132(1):e262-e280.

42. What is the differential diagnosis of acute bacterial sinusitis?

- Mucopurulent rhinitis
- Allergic rhinitis
- Pharyngitis
- Nasal foreign body
- Adenoiditis
- Poor dental hygiene

43. When is CT useful in the diagnosis of sinusitis?

Do not use CT (Fig. 38-1) to differentiate between bacterial sinusitis and viral upper respiratory infection. However, it is helpful in children with complications of acute bacterial sinus infection such as orbital or CNS involvement or those with very persistent or recurrent infections who do not respond to medical management.

Wald ER, Applegate KE, Bordley C, et al: Clinical practice guideline for the diagnosis and management of acute bacterial sinusitis in children aged 1 to 18 years. Pediatrics 2013;132(1):e262-e280.

44. Describe the treatment of acute bacterial sinusitis.

- For uncomplicated acute bacterial sinusitis of mild severity, use amoxicillin (45-90 mg/kg/day given twice daily).
- For moderate to severe acute bacterial sinusitis, or suspected sinusitis in children less than 2 years old, in patients who recently received antibiotics for sinusitis, or in patients

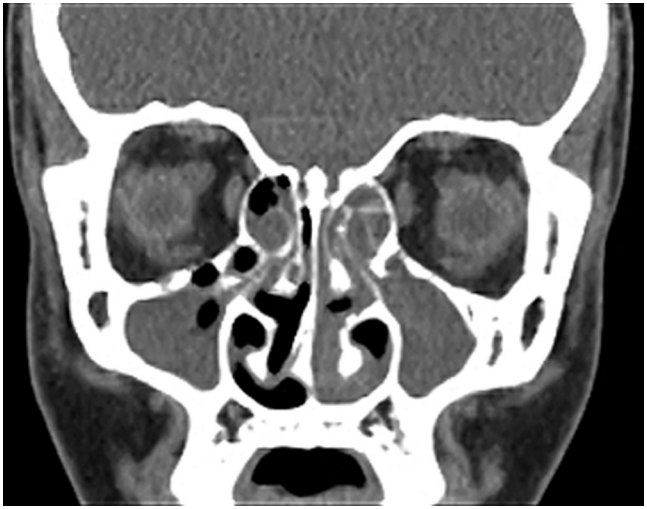

Figure 38-1. Computed tomographic scan of sinusitis.

attending day care, amoxicillin-clavulanate (80-90 mg/kg/day in two divided doses) is recommended.

- Treat children unable to tolerate oral antibiotics with one dose of IV/IM ceftriaxone, followed by oral antibiotics if clinical improvement is observed within 24 hours.
- In children who have a penicillin allergy but a history of a nonanaphylactic reaction, use cefdinir, cefuroxime, or cefpodoxime to treat acute bacterial sinusitis safely.

Wald ER, Applegate KE, Bordley C, et al: Clinical practice guideline for the diagnosis and management of acute bacterial sinusitis in children aged 1 to 18 years. Pediatrics 2013;132(1):e262-e280.

45. What is Pott's puffy tumor?

Pott's puffy tumor (Fig. 38-2) was first described by Sir Percivall Pott in 1760 and appears as a soft, fluctuant, painful forehead or scalp swelling usually associated with frontal sinusitis. Patients tend to be febrile and appear toxic. It is usually seen in children after 8 years of age when the frontal sinuses begin to develop. It represents osteomyelitis of the frontal bone with

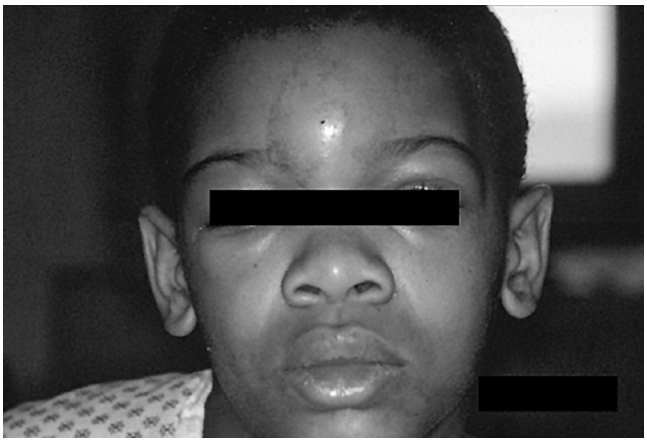

Figure 38-2. Pott's puffy tumor.

subsequent subperiosteal elevation. CT is essential for diagnosis and to evaluate other possible areas of spread. Successful treatment usually involves both antibiotics and surgical drainage.

46. Which infectious organisms are associated with the three primary cardiac infections?

See Table 38-5 for infectious organisms associated with the three primary cardiac infections.

47. What are some of the signs and symptoms of myocarditis in infants and children?

Children presenting to the ED with myocarditis may initially report a nonspecific, flulike illness or gastroenteritis. Within 1 to 4 weeks, fever, malaise, dyspnea, tachypnea, chest pain, pallor, or cool, poorly perfused distal extremities may develop. Infants may present with feeding difficulties, listlessness, cardiac signs, or respiratory distress. The cardiac examination may reveal sinus tachycardia at rest, muffled and distant heart sounds, lateral displacement of the point of maximal impulse, and a gallop rhythm suggestive of congestive heart failure. Other findings on physical examination associated with heart failure include jugular venous distention, weak pulses, cyanosis, poor perfusion, and hepatosplenomegaly.

48. What diagnostic test results in the ED support the suspicion of myocarditis?

- Chest radiography can demonstrate cardiomegaly, interstitial pulmonary edema, or an engorged pulmonary venous pattern.
- Electrocardiography may demonstrate sinus tachycardia, mild to moderate PR interval prolongation, generalized low-voltage QRS complexes, ST-segment elevation or depression, decreased precordial voltages, high-grade atrioventricular block, and complex ventricular or supraventricular arrhythmias.
- Echocardiography typically demonstrates global cardiac chamber enlargement with poorly contracting ventricles or atrioventricular valve regurgitation. Pericardial effusion may also be present.
- WBC count, erythrocyte sedimentation rate (ESR), and creatine kinase (MB) fraction may be abnormal but are nonspecific.

49. What is the acute management of myocarditis in infants and children?

- Admission to the hospital (intensive care unit for infants and children with heart failure or arrhythmias) for serial echocardiography, electrocardiography, and monitoring
- Diuretics or inotropic agents for heart failure

Table 38-5. Infectious Organisms Associated with the Three Primary Cardiac Infections

INFECTION	BACTERIAL	VIRAL	OTHERS
Myocarditis	*Borrelia burgdorferi* *Neisseria meningitidis* *Staphylococcus aureus* *Salmonella*	Enteroviruses Adenovirus Influenza Cytomegalovirus Epstein-Barr virus	*Trypanosoma cruzi* *Toxoplasma gondii*, *Cryptococcus* *Candida*
Endocarditis	*Streptococcus viridans* *Streptococcus pneumoniae* Enterococci *S. aureus* Coagulase-negative staphylococci Gram-negative bacilli		Fungi Culture negative (HACEK)
Pericarditis	*S. aureus* *N. meningitidis* *S. pneumoniae*	Enteroviruses	*Mycobacterium tuberculosis* *Mycoplasma pneumoniae*

- Specific antibiotics if a bacterial cause is considered
- Debated: use of corticosteroid and immunosuppressive agent

50. What are the common symptoms, signs, and laboratory findings in infants and children with infective endocarditis?

See Table 38-6 for symptoms, signs, and laboratory findings in infective endocarditis.

51. Differentiate among Osler nodes, Janeway lesions, and Roth spots.

- **Osler nodes** are painful, red, nodular lesions seen most frequently on the pulp areas of the distal digits.
- **Janeway lesions** are small, erythematous, nontender areas typically on the palms and soles.
- **Roth spots** are retinal hemorrhages with central clearing.

Although commonly seen in adults, these findings are rare in infants and children with infective endocarditis. Other embolic phenomena associated with endocarditis include splinter hemorrhages and conjunctival hemorrhages.

52. How do you make the clinical diagnosis of infective endocarditis?

Two major criteria or one major plus three minor or five minor criteria according to the modified Duke criteria are required for *definitive clinical diagnosis* of infective endocarditis described *in brief* here:

- **Major criteria:** (1) positive blood culture for infective endocarditis (ideally from two initial blood cultures) and (2) evidence of endocardial involvement by echocardiogram
- **Minor criteria:** (1) predisposing cardiac condition or history of IV drug use, (2) fever higher than 38° C, (3) vascular manifestations (septic pulmonary infarcts, mycotic aneurysm, intracranial or conjunctival hemorrhages, Janeway lesions, major arterial emboli), (4) immunologic manifestations (glomerulonephritis, positive rheumatoid factor, Osler nodes, Roth spots), and (5) microbiologic evidence that is not included in the major criteria

Li JS, Sexton DJ, Mick N, et al: Proposed modifications to the Duke criteria for the diagnosis of infective endocarditis. Clin Infect Dis 2000;30:633-638.

53. What antibiotics are recommended for the presumptive diagnosis of infective endocarditis?

Although viridans group streptococci are the most common etiologic agents, it is not essential and may even be harmful to immediately administer antibiotics without definitive knowledge of the causative organism. However, in emergency cases an aminoglycoside such as gentamicin combined with a penicillinase-resistant penicillin (e.g., oxacillin) would be appropriate. Alternative therapy includes ampicillin with gentamicin, with the addition of vancomycin in MRSA-prevalent areas.

Table 38-6. Symptoms, Signs, and Laboratory Findings in Infective Endocarditis

SYMPTOMS	SIGNS	LABORATORY FINDINGS
Fever	Fever	Positive blood culture (75-100%)
Malaise	Petechiae	Elevated erythrocyte sedimentation rate (75-100%)
Anorexia/weight loss	Splenomegaly	Anemia (75-90%)
Arthralgias	New or changed murmur	
Less frequent		
Gastrointestinal symptoms	Embolic phenomenon	Hematuria (25-50%)
Neurologic deficits	Heart failure	Positive rheumatoid factor (25-50%)
Chest pain	Meningismus	Low complement level (5-40%)

54. What are the clinical manifestations and diagnostic findings associated with pericarditis?

Clinical manifestations:

- Substernal chest pain that is worse with inspiration and relieved by sitting upright and forward; pain may radiate to the scapular ridge
- Irritability
- Grunting expiratory sounds
- Fever
- Exercise intolerance
- Muffled heart sounds
- Pericardial friction rub best heard in second to fourth intercostal spaces, midclavicular line, and left sternal border during deep inspiration with the patient kneeling or in the knee-chest position (a pathognomonic finding)
- If tamponade: tachycardia, peripheral vasoconstriction, decreased arterial pulse pressure, or pulsus paradoxus

Diagnostic findings:

- WBC count, CRP, ESR, and troponin may be elevated
- Pericardial fluid analysis suggestive of infection
- Increased size of cardiac shadow in the absence of pulmonary congestion on chest radiography ("water bottle heart")
- Electrocardiography:
  - Stage I: diffuse ST-segment elevation and PR interval depression
  - Stage II: normalization of ST and PR segments
  - Stage III: widespread T-wave inversions
  - Stage IV: normalization of T waves
- Echocardiography: presence of pericardial fluid
- Microbiologic evaluation of the pericardial fluid by pericardiocentesis
- Viral cultures, serologic tests, and molecular genetic techniques

Blanco CC, Parekh JB: Pericarditis. Pediatr Rev 2010;31(2):83-84.

55. What is the antimicrobial agent of choice for the presumptive diagnosis of bacterial pericarditis?

Until definitive pericardial cultures return, treatment should include coverage for gram-positive and gram-negative bacteria. Vancomycin and cefotaxime/ceftriaxone are appropriate first-line agents, especially in highly resistant areas.

56. How common are UTIs in infants and children?

Roughly 14% of pediatric ED visits annually are due to UTIs. Girls have an overall higher incidence of childhood UTI at 8% than do boys at 1% to 2%. In infants younger than 3 months of age who present to the ED with fever but without a source of infection, the prevalence is about 7% to 9%, regardless of sex; this decreases to about 2% for both males (>3 months of age) and females (>12 months of age). The prevalence of UTI among whites is 8.0% as compared to African Americans at 4.7%.

Shaikh NM, Morone NEM, Bost JEP, Farrell MHB: Prevalence of urinary tract infection in childhood: A meta-analysis. Pediatr Infect Dis J 2008;27(4)302-308.

57. What are the signs and symptoms of UTIs in infants and children?

See Table 38-7. Approximately 50% of *adolescents* who present to the ED with dysuria, increased frequency, and urgency on urination have a UTI. Only 10% of *children* who present with these symptoms have a UTI; their symptoms may instead be due to irritation from bubble bath, vaginitis, pinworms, or sexual abuse.

58. What is the definition of pyuria?

Pyuria is the presence of 5 WBCs/high-power field (uncentrifuged) or 10 WBCs/mm^3 (centrifuged specimens). Without other findings it is not adequate to diagnose a UTI and is not a substitute for a urine culture. It is also seen with chemical irritation, fever, viral infection, appendicitis, glomerulonephritis, Kawasaki disease, and renal tuberculosis.

Table 38-7. Signs and Symptoms of Urinary Tract Infections According to Age

NEWBORNS	INFANTS AND TODDLERS	SCHOOL-AGE CHILDREN
Fever	Fever	Fever
Hypothermia	Failure to thrive	Vomiting
Vomiting	Vomiting	Diarrhea
Failure to thrive	Diarrhea	Strong-smelling urine
Sepsis	Strong-smelling urine	Abdominal pain
Jaundice	Irritability	Dysuria
Irritability		Frequency
		Urgency
		Enuresis

59. What screening tests are most useful to rule out a UTI?

Several rapid screening tests are used frequently in the ED to evaluate for a UTI in an infant or child presenting with a fever without a source. They include a dipstick biochemical analysis of urine for nitrites or leukocyte esterase and a microscopic analysis of urine for WBCs or bacteria. A meta-analysis examining screening tests for UTIs concluded that both a Gram stain on an uncentrifuged urine specimen and dipstick analysis of an uncentrifuged urine specimen for nitrite and leukocyte esterase performed similarly, and these tests were superior to microscopic analysis for pyuria.

Gorelick MH: Screening tests for urinary tract infection in children: A meta-analysis. Pediatrics 1999;104:e54.

60. Which infants age 2 to 24 months with a fever should be evaluated for a UTI?

In females, features increasing the likelihood of UTI include a temperature of 39.0° C, fever for 2 or more days, white race, age younger than 1 year, and absence of another potential source of fever. In males, clinical features predictive of a UTI include age younger than 6 months, nonblack race, being uncircumcised, and absence of another potential source for the fever. Consider screening tests and urine cultures in all infants and children with these risk factors.

61. Can a urine specimen collected with a bag be used to diagnose a UTI in an infant younger than 2 years old?

No. The false-positive rate of a positive urine culture obtained by the bag method is approximately 85%, although a negative result from bagged urine rules out a UTI. If a UTI is considered in an infant younger than 2 years of age, obtain urine by transurethral catheterization of the urinary bladder (sensitivity of 95% and specificity of 99%) or suprapubic aspiration. In older, toilet-trained children, the midstream clean-catch method is preferred (contamination rates of 0-29%).

62. What treatment options are available for infants and children with simple cystitis?

Consider outpatient therapy in infants who are well hydrated and nontoxic appearing, those who can tolerate oral medication, those without underlying urologic abnormalities, and those with reliable parents and social situations. When empiric therapy is chosen, base the antibiotic choice on patterns of antibiotic sensitivity in your community. A 3- to 7-day course of oral antibiotics is recommended. Cefixime or cefdinir is the first-line choice. Antibiotics such as amoxicillin and sulfamethoxazole/trimethoprim are not first-line agents in the treatment of most pediatric UTIs.

Gaspari RJ, Dickson E, Karlowsky J, Doern G: Antibiotic resistance trends in paediatric uropathogens. Int J Antimicrob Agents 2005;26(4):267-271.

63. Which young children with UTIs need to be admitted to the hospital?

It is recommended that all children less than 3 months of age be admitted and started on IV antibiotic therapy. Furthermore, AAP recommends that older infants through adolescents

who are dehydrated or ill-appearing or patients with chronic disease such as sickle cell or diabetes mellitus should also be hospitalized and started on IV antibiotic therapy. Also, consider hospital admission if the patient is vomiting and cannot tolerate oral medications.

64. What is pelvic inflammatory disease (PID), and what are the most likely etiologic agents?

The diagnostic criteria for PID based on the Centers for Disease Control guidelines include a sexually active female patient at risk for sexually transmitted infections experiencing pelvic or lower abdominal pain, if no cause for the illness other than PID can be identified, and if one or more of the following minimum criteria are present on pelvic examination: cervical motion tenderness, uterine tenderness, or adenexal tenderness. The etiologic agents causing PID primarily include *N. gonorrhoeae* and *C. trachomatis*, although PID has been also associated with anaerobes, *Gardnerella vaginalis*, *H. influenza*, enteric gram-negative rods, *Streptococcus agalactiae*, cytomegalovirus, *Mycoplasma hominis*, *Ureaplasma urealyticum*, and *Mycoplasma genitalium*.

Workowski KA, Berman S; Centers for Disease Control and Prevention: Sexually transmitted diseases treatment guidelines, 2010. MMWR Recomm Rep 2010;59:1-110.

65. What is the cause of vaginitis in postpubertal girls? What characteristics distinguish the infections?

Patients with vaginitis typically present with a history of dysuria, vaginal discharge, burning, or vulvar lesions. Physical examination reveals vulvar irritation and vaginal discharge. The infectious causes in postpubertal girls include bacterial vaginosis (40-50%), vulvovaginal candidiasis (20-25%), and *Trichomonas* vaginitis (15-20%) (Table 38-8).

Table 38-8. Features of Vaginitis in Postpubertal Girls

FEATURE	BACTERIAL VAGINOSIS	VULVOVAGINAL CANDIDIASIS	TRICHOMONAS
Clinical presentation	Gray-white homogeneous vaginal discharge with fishy odor without signs of erythema or infection	Intense burning, pruritus, erythema of vulva with milky white discharge, cottage-cheese–appearing, dysuria and dyspareunia	Vaginal pruritus, malodorous green-gray or cream-colored discharge that is bubbly or frothy, cervix with strawberry appearance
Diagnosis	Clue cells on wet prep, pH > 4.5, positive result on whiff test and routine culture	Wet prep with potassium hydroxide revealing budding yeast with pseudohyphae, vaginal culture, pH normal	Culture, wet prep revealing organism, polymerase chain reaction on urine sample
Treatment	Metronidazole × 7 days Clindamycin vaginal cream × 7 days Metronidazole gel × 5 days	Monistat (cream or suppository) Gyne-Lotrimin (cream or vaginal tablet) Nystatin vaginal tablet × 14 days Fluconazole orally × 1 dose	Metronidazole orally × 1 dose or for 7 days

Table 38-9. Differential Diagnosis for Genital Ulcers

DISEASE	CLINICAL PRESENTATION	DIAGNOSIS	TREATMENT
Herpes genitalis	Single or multiple vesicles on the genitalia that rupture to form shallow ulcers; very painful and resolve without scarring	Tzanck smear: multinucleated giant cells, HSV viral culture, DFA, or PCR	No cure; symptomatic treatment (acyclovir, valacyclovir, famciclovir)
Syphilis	Chancre at site of the incubation (painless papule eroding to an indurated ulcer) with lymphadenopathy; heals in 4-6 weeks	Dark-field examination of chancre, serologic tests (VDRL, RPR)	Benzathine penicillin IM × 1 dose (doxycycline or tetracycline × 14 days if penicillin allergic)
Lymphogranuloma venereum	Lesion (papule, ulcer, herpetiform, or urethritis/cervicitis), painful regional unilateral inguinal or femoral adenopathy with constitutional symptoms or systemic complications	Clinical presentation, culture of *Chlamydia* of node, or complement fixation	Doxycycline, tetracycline, or erythromycin for 21 days
Chancroid	Usually single, superficial, painful ulcer surrounded by an erythematous halo; bleeds easily with purulent exudate; unilateral adenopathy	Clinical appearance, smears from ulcer or aspiration from infected lymph nodes, or culture positive for *Haemophilus ducreyi*	Azithromycin orally × 1 dose *or* IM ceftriaxone × 1 dose

DFA, direct fluorescent antibody; HSV, herpes simplex virus; IM, intramuscular; PCR, polymerase chain reaction; RPR, rapid plasmin reagin; VDRL, Venereal Disease Research Laboratory.

66. What is the differential diagnosis for genital ulcers? How do you distinguish between each disease process?

See Table 38-9 for the differential diagnosis for genital ulcers.

67. What organisms most commonly cause cellulitis?

Cellulitis, which is an infection of the dermis and subcutaneous fat, is caused most often by *S. aureus* and β-hemolytic streptococci in children.

68. Are cultures helpful in diagnosing cellulitis?

In a published study of 224 pediatric patients, 96% had a blood culture done initially in the ED. Only two of the cultures were positive. Furthermore, wound cultures were positive in only 26% of children with cellulitis.

Gouin S, Chevalier I, Gauthier M, Lamarre V: Prospective evaluation of the management of moderate to severe cellulitis with parenteral antibiotics at a paediatric day treatment centre. J Paediatr Child Health 2008;44:214-218.

69. When is parenteral treatment recommended for cellulitis?

Criteria for parenteral antibiotics include signs of systemic toxicity, rapidly progressing involvement, lymphangitis, involvement of the face or neck, and absence of improvement

following 48 hours of outpatient management. A penicillinase-resistant penicillin or a first-generation cephalosporin may be used for empiric outpatient therapy. Consider clindamycin or trimethoprim-sulfamethoxazole for suspected MRSA infection.

70. A 9-year-old child presents to the ED with a chief complaint of right foot pain. Four days ago he stepped on a nail that went through his sneaker into his sole. He presents with no history of fever, but with erythema and warmth of the sole of his foot and point tenderness at the proximal end of his fifth metatarsal. What diagnosis do you suspect?

This scenario is classic for a *Pseudomonas aeruginosa* infection (most likely osteochondritis) in a child who has a history of stepping on a nail through a sweaty sneaker. Management of this child includes hospital admission for IV antibiotics following a workup for osteomyelitis (i.e., magnetic resonance imaging or nuclear bone scanning, CBC with differential, ESR/CRP, blood culture). Consult orthopedic surgery for probable surgical débridement.

71. What is the differential diagnosis of perianal dermatitis/cellulitis?

Streptococcal infections (GBS in infants, GABHS in older children), inflammatory bowel disease, sexual abuse, pinworms, diaper dermatitis, psoriasis, and seborrheic dermatitis.

72. Match the superficial bacterial skin infection with its classic presentation and treatment.

Skin infection:

A. Impetigo
B. Ecthyma
C. Erysipelas
D. Paronychia
E. Folliculitis
F. Furuncles/carbuncles

Presentation and treatment:

1. Infection may begin following minor trauma or an insect bite; initially vesiculopapular with surrounding erythema and later developing a thick, adherent crust that, when removed, reveals a punched-out, painful ulcerative lesion. Treatment includes cleansing and topical and systemic antibiotics covering streptococci and *S. aureus*. This infection is typically seen in immunocompromised patients.
2. Infection results from local injury to the nail fold and is seen in children who suck their fingers or bite their nails; the lateral nail fold becomes warm, erythematous, edematous, and painful. Treatment includes warm compresses and, for deep infections, incision and drainage and antibiotics covering mixed oral flora.
3. Infection originates from either isolated nodular subcutaneous abscesses or multiple abscesses separated by connective tissue septae clinically presenting as painful red papules or boils in a nontoxic-appearing child. Treatment includes local care, incision and drainage, and systemic antibiotics for larger lesions.
4. This superficial infection of the skin is caused by either *S. aureus* or GABHS and appears as mildly painful lesions with an erythematous base and honey-crusted exudates in a nontoxic child; absence of constitutional symptoms and presence of regional adenopathy are noted. Treatment includes topical mupirocin or systemic antibiotics (widespread lesions, lesions near the mouth, evidence of deeper infection, constitutional symptoms).
5. A clearly demarcated, raised, and advancing red border extends from the site of inoculation with lymphangitic streaks extending from the involved area; skin is shiny and warm to touch; and presence of systemic signs and high fever are noted. Treatment consists of IV antibiotics until patient is afebrile and lesion begins to regress, then oral antibiotics.
6. Small, red pustules are seen at the site of hair follicles. Treatment includes local care and topical antibiotics.

Answers: 1, B; 2, D; 3, F; 4, A; 5, C; 6, E.

73. A 4-year-old presents with a boggy, purulent, eczematoid mass measuring 5 cm × 5 cm on his right temporal scalp area. He also has alopecia and a right-sided, posterior chain cervical adenopathy. What is the likely diagnosis?

This presentation is consistent with a kerion, which is a cell-mediated response to tinea capitis often confused with a bacterial skin infection. Therapy includes a minimum of 4 weeks of oral antifungal therapy (griseofulvin or ketoconazole). Systemic antibiotics and topical agents are not indicated.

74. What findings may be associated with an invasive skin or soft tissue infection?

- Necrosis of skin or soft tissue
- Crepitance on physical examination
- Nonadherence of skin and subcutaneous tissue to underlying fascia on exploration
- Abnormal skin color other than erythema (such as bronzed, cyanotic, violaceous)
- Severe systemic toxicity, anxiety, or confusion
- Pain on palpation out of proportion to physical findings
- Tachycardia out of proportion to fever (suggestive of clostridial infection)
- Hypocalcemia (calcium deposition in necrotic subcutaneous fat)
- Gas present on radiograph
- Failure to respond to medical management
- Bullae with thin, brown discharge with "sweet but foul" odor (clostridial infection) or "dishwater" fluid (anaerobic fluid)

75. What characteristics of lymph nodes distinguish infectious from noninfectious causes?

Characteristics of lymph nodes that distinguish infectious from noninfectious causes can be seen in Table 38-10.

76. Lymphadenopathy isolated to a particular region may indicate a specific infection. List some of these associations.

Those associations are listed in Table 38-11.

77. How does a child with cat-scratch disease typically present?

Typically there is a history of a scratch by a kitten on an extremity or the face, followed by the appearance (3-10 days after the inoculation) of a macule, papule, or vesicle, which may last several days to weeks. Single lymph nodes appear proximal to the site around 1 to 2 weeks after the scratch in approximately 50% of children, with several lymph nodes in the same region in 20%. The nodes are most often tender and erythematous. They can also be suppurative in 10% to 15% of cases. Other clinical manifestations of cat-scratch disease

Table 38-10. Characteristics of Lymph Nodes That Distinguish Infectious from Noninfectious Causes

VIRAL INFECTION	BACTERIAL INFECTION	MALIGNANCY
Soft	Soft to firm	Firm
Nontender	Tender	Nontender
Mobile	Fixed	Mobile
Nonerythematous	Erythematous	Nonerythematous
Small; usually < 2 cm	Large; usually > 2 cm	Increase in size over time
Discrete in a specific region or generalized	Unilateral	Matted with adjacent nodes

Table 38-11. Regional Lymphadenopathy and Associated Infectious Etiology

REGION	*Infectious Etiology*	
	Common	Less Common
Occipital	Impetigo Tinea capitis Seborrhea	Toxoplasmosis Rubella
Preauricular	Pediculosis Chlamydial conjunctivitis Adenoviral conjunctivitis	Tularemia Herpes simplex Parinaud's syndrome
Cervical	Viral upper respiratory tract infection Bacterial infection of head/neck Primary bacterial adenitis Epstein-Barr virus/cytomegalovirus Cat-scratch disease Atypical mycobacterium *Mycobacterium tuberculosis*	Kawasaki disease Toxoplasmosis Anaerobic infection Tularemia Histoplasmosis Leptospirosis Brucellosis
Axillary	Local pyogenic infection Cat-scratch disease	Toxoplasmosis
Epitrochlear	Local infection Chronically inflamed hand	Secondary syphilis Tularemia
Inguinal	Lower-extremity infection Genital herpes Primary syphilis	Chancroid Lymphogranuloma venereum
Iliac	Lower-extremity infection Abdominal infection Urinary tract infection	
Popliteal	Severe local pyogenic infection	

include fever, malaise/fatigue, headaches, splenomegaly, anorexia/emesis/weight loss, sore throat, exanthems, conjunctivitis, and parotid swelling. Lymphadenopathy associated with cat-scratch disease is self-limited, lasting 1 to 4 months.

Spach DH, Kaplan SL: Cat scratch disease. UpToDate, 2013. Available from www.uptodate.com.

78. What are *atypical* clinical manifestations seen in cat-scratch disease (5-20% of all presentations)?

- Parinaud oculoglandular syndrome (conjunctival granuloma and preauricular adenopathy)
- Thrombocytopenic purpura
- Osteitis resembling bacterial osteomyelitis
- CNS manifestations (encephalopathy, encephalitis, radiculitis, polyneuritis, myelitis, neuroretinitis)
- In immunocompromised children, bacteremia, bacillary angiomatosis, and bacillary peliosis

79. What is appropriate treatment for a nontoxic-appearing child who presents with lymphadenitis?

For a child with a nonfluctuant node consistent with an acute bacterial infection or a staphylococcal or streptococcal infection, a β-lactamase–resistant penicillin (e.g., dicloxacillin) or a cephalosporin (e.g., cephalexin) for 2 weeks is appropriate treatment. For a penicillin-allergic child, clindamycin or erythromycin may be used. In those patients who are toxic-appearing or unresponsive to oral antibiotics, or in young infants, IV antibiotics (e.g., nafcillin, cefazolin, or clindamycin) are effective. Percutaneous aspiration or incision and drainage may be necessary for fluctuant nodes.

80. Distinguish among bacteremia, systemic inflammation response syndrome (SIRS), sepsis, severe sepsis, and septic shock.

- **Bacteremia:** The presence of bacteria in the blood
- **SIRS:** A clinical syndrome in response to a variety of insults (e.g., infection, trauma, acute respiratory distress syndrome, neoplasms, pancreatitis, burns) manifested by at least two of the following conditions:
  - Temperature higher than 38.0° C or lower than 36.0° C
  - Tachycardia (heart rate >160 beats per minute in infants, >150 beats per minute in children)
  - Tachypnea (respiratory rate >60 breaths per minute in infants, >50 breaths per minute in children)
  - WBC count more than 12,000 cells/mm^3 or more than 10% immature forms
- **Sepsis:** The systemic response to infections manifested by two or more of the criteria for SIRS
- **Severe sepsis:** Sepsis associated with hypotension (systolic blood pressure < 65 mm Hg in infants, <75 mm Hg in children, and < 90 mm Hg in adolescents, or reduction > 40 mm Hg from baseline), hypoperfusion (lactic acidosis, oliguria, hypoxemia, or acute change in mental status), or organ dysfunction
- **Septic shock:** Sepsis with hypotension, despite adequate fluid resuscitation, with the presence of perfusion abnormalities, which may include lactic acidosis, oliguria, or acute change in mental status

81. What are the common signs/symptoms and laboratory values associated with septic shock in infants and children?

Infants:

- Signs/symptoms
  - Hyperthermia or hypothermia
  - Tachycardia
  - Tachypnea
- Laboratory results
  - Lactic acidosis
  - Leukocytosis or leukopenia
  - Increased bands, myelocytes, promyelocytes

Children:

- Signs/symptoms
  - Hypotension
  - Delayed capillary refill
  - Weak peripheral pulses
  - Cool extremities
  - Irritability
  - Lethargy
  - Confusion
  - Oliguria
  - Petechiae or purpura
- Laboratory results
  - High or low serum glucose level
  - Hypocalcemia
  - Hypoalbuminemia
  - Positive blood, urine, CSF cultures
  - Abnormal coagulation factors/disseminated intravascular coagulation
  - Thrombocytopenia
  - Abnormal renal function

82. What are the antibiotic choices for empirical therapy in infants and children presenting in septic shock?

See Table 38-12 for the antibiotic choices for empirical therapy for septic shock according to age.

Table 38-12. Empirical Therapy for Septic Shock According to Age

AGE	BACTERIAL ETIOLOGY	ANTIBIOTIC CHOICE FOR EMPIRICAL THERAPY
Neonate	GBS	Ampicillin plus aminoglycoside or cefotaxime; if nosocomial, then add vancomycin
	Gram-negative bacilli	Cefotaxime or ceftriaxone plus vancomycin (if you suspect gram-positive infection)
Child	*Streptococcus pneumoniae*, *Neisseria meningitidis*, *Staphylococcus aureus*, GAS	If nosocomial, vancomycin plus antibiotic against gram-negative bacteria (ceftazidime, cefepime)
	Invasive GAS (e.g., post varicella)	Aminoglycoside, carbapenem, or extended spectrum penicillin with β-lactamase–inhibitor penicillin and clindamycin

GAS, group A streptococci; GBS, group B streptococci.

83. What is the differential diagnosis of fever and petechiae?

Children and infants who present to the ED with fever and petechiae require immediate attention because there are life-threatening causes that may progress rapidly to death. The differential diagnosis of common infectious causes includes both treatable and nontreatable organisms:

Treatable organisms:

- *N. meningitidis* (meningococcemia)
- *N. gonorrhoeae* (gonococcemia)
- *P. aeruginosa*
- *S. pyogenes*
- *Rickettsia prowazekii* (epidemic typhus)
- *Rickettsia rickettsii* (Rocky Mountain spotted fever [RMSF])
- *S. aureus* (endocarditis)

Nontreatable organisms:

- Adenovirus
- Rubeola (atypical measles)
- Enterovirus
- Epstein-Barr virus

84. When is fever associated with a petechial rash less concerning?

A child with fever and petechiae is less likely to have a serious bacterial illness if he or she appears well and has one isolated petechial lesion, petechiae only above the nipple line, a mechanical cause of the petechiae (coughing, emesis, screaming or crying, blood pressure cuff or tourniquet), and normal WBC count and platelet count. In most cases these children can be safely discharged from the ED.

Mandl KD: Incidence of bacteremia in infants and children with fever and petechiae. J Pediatr 1997;131:398-404.

85. What factors predict poor prognosis in infants and children with meningococcemia?

- Absence of CSF pleocytosis
- Rapidly evolving hemorrhagic skin lesions
- Shock
- Hyperpyrexia
- Leukopenia
- Thrombocytopenia
- Low plasma levels of fibrinogen

- Disseminated intravascular coagulation
- Metabolic acidosis
- Rapid clinical deterioration
- Low serum CRP level
- Low absolute neutrophil count
- Low serum potassium level

86. What is acute arthritis and dermatitis syndrome?

Also known as disseminated gonococcal infection, acute arthritis and dermatitis syndrome occurs in approximately 1% to 3% of persons with untreated mucosal gonorrhea. Clinical findings occur in two stages: a bacteremic stage and a joint-localized stage with suppurative arthritis. Often a clear delineation of these stages is difficult to make. The bacteremic stage consists of high fever and rigors as well as dermatitis (tender necrotic pustule with an erythematous base located distally on an upper extremity). The joint-localized stage consists of migratory polyarthralgias or tenosynovitis affecting smaller joints and arthritis (pyogenic monoarticular or polyarticular with effusion, especially of knee, ankle, or wrist).

Rice PA: Gonococcal arthritis (disseminated gonococcal infection). Infect Dis Clin North Am 2005; 19(4):853-861.

87. List clinical criteria that define toxic shock syndrome.

The diagnosis is made by the presence of three major criteria and at least three minor criteria, with negative blood, throat, and CSF cultures (except for *S. aureus*) and no rise in titers to RMSF, leptospirosis, and rubeola antigens.

Major criteria:

- Temperature higher than 38.9° C
- Hypotension (shock or orthostatic)
- Macular erythroderma—late desquamation within 1 to 2 weeks of the illness (especially of palms and soles)

Minor criteria:

- Mucous membrane inflammation (conjunctival or pharyngeal)
- Gastrointestinal (vomiting, diarrhea)
- Musculoskeletal (myalgia or elevation of creatine phosphokinase)
- Central nervous system (alteration of consciousness)
- Hepatic (elevated bilirubin, aminotransferase levels)
- Renal (elevated blood urea nitrogen level, >5 WBCs/high-power field on UA)
- Decreased platelet count ($<100{,}000/mm^3$)

88. Distinguish between staphylococcal and streptococcal toxic shock syndrome.

The differences between staphylococcal and streptococcal toxic shock syndrome are listed in Table 38-13.

89. Which two infectious agents are most commonly associated with erythema multiforme minor and major (Stevens-Johnson syndrome), respectively?

Erythema multiforme minor is most commonly associated with HSV, and Stevens-Johnson syndrome is most commonly associated with *Mycoplasma pneumoniae*.

90. What is staphylococcal scalded skin syndrome?

Staphylococcal scalded skin syndrome is a dermatosis caused by an epidermolytic toxin produced by certain strains of staphylococci. It generally affects children younger than 5 years old. White children are more prone and classically present with fever, irritability, and tender erythematous skin. The skin begins to exfoliate, with crusting and exudation around the mouth, eyes, and paranasal areas, within 2 to 3 days of the initial rash. The *Nikolsky sign* (wrinkling of the upper layer of the epidermis and removal of the layer by light stroking, like the peeling of wet tissue paper) may be present. A skin biopsy from the blister base can distinguish staphylococcal scalded skin syndrome from toxic epidermal necrolysis. The mortality rate is less than 5%. Hospitalization is often required for skin care, fluid management, and IV antibiotics.

Ladhani S, Joannou CL, Lochrie DP, et al: Clinical, microbial, and biochemical aspects of the exfoliative toxins causing staphylococcal scalded-skin syndrome. Clin Microbiol Rev 1999;12(2):224-242.

Table 38-13. Staphylococcal Versus Streptococcal Toxic Shock Syndrome

FEATURE	STAPHYLOCOCCAL TOXIC SHOCK SYNDROME	STREPTOCOCCAL TOXIC SHOCK SYNDROME
General presentation	Acute onset of severe symptoms (vomiting/diarrhea)	Gradual onset of mild symptoms (malaise/myalgia)
Fever	High; abrupt onset	Gradual onset (if fever is present)
Rash	Erythroderma	Scarlatina
Shock	Responds to aggressive intravascular volume expansion	Unpredictable response to intravascular volume expansion
Source of infection	Menstrual related, sinusitis, surgical wound	Cellulitis, necrotizing myositis, fasciitis, pneumonitis
Response to antibiotics	Beneficial for treatment of acute infection and recurrence; β-lactamase–resistant penicillins or cephalosporins	More difficult in treating acute infection; clindamycin superior to β-lactam agents
Complications	Infrequent coagulopathies, complicated hospitalizations, gangrene	Common coagulopathies, complicated hospitalizations, gangrene
Mortality rate	10%	30%-50%

91. What are the typical cutaneous findings associated with scarlet fever?

These findings appear following a 2- to 4-day incubation period and accompany the abrupt onset of fever, headache, vomiting, malaise, abdominal pain, and sore throat:

- Erythematous oral mucous membranes with scattered palatal petechiae
- White or red strawberry tongue
- Circumoral pallor
- Erythematous, punctate rash with sandpaper-like texture; first appears (12 to 48 hours) on upper trunk, and then becomes more generalized (and more intense in skin folds of axillae, antecubital, popliteal, and inguinal regions and sites of pressure such as buttocks and small of back). The rash is rapidly evolving, which can distinguish it from viral rashes.
- Pastia's lines (transverse areas of hyperpigmentation with petechial character in antecubital fossa, axilla, and inguinal regions)
- Scaly exfoliation on the hands, palms, knees, feet, and perineum within 4 to 5 days of beginning of sandpaper rash. This can potentially last 3 to 6 weeks.

Festekjian A, Pierson SB, Zlotkin D: Index of suspicion. Pediatr Rev 2006;27(5):189-194.

92. A 10-month-old ill-appearing infant has a history of multiple episodes of vomiting, lethargy, and rapidly spreading rash consistent with petechiae and purpura. The infant is resuscitated immediately (oxygen applied by face mask; IV access obtained; blood, urine, and lumbar puncture performed and laboratory tests ordered; antibiotics given). What antibiotic prophylaxis is needed for the ED staff?

The scenario is most consistent with meningococcemia. Take droplet precautions, which include the use of a respiratory mask if within 3 feet of the child. Chemoprophylaxis is strongly recommended if mouth-to-mouth resuscitation is provided or there is unprotected contact during endotracheal intubation. Options for chemoprophylaxis include oral rifampin for 2 to 4 days, IM ceftriaxone for one dose, or oral ciprofloxacin for one dose (the latter is not recommended for use in children <18 years old).

93. An 18-year-old teen is escorted to the ED from prison with a chief complaint of "coughing up blood." He has had a chronic cough for approximately 6 months and has lost 10 pounds over the last year. What should the ED staff do to protect themselves from possibly being exposed to this disease?

The scenario is most consistent with tuberculosis. Take airborne precautions, which include a private room with negative air-pressure ventilation and properly fitted or sealing respiratory masks to be worn by all health care providers in contact with the patient.

94. A 16-year-old boy with a medical history of mental retardation and cerebral palsy who lives in a long-term care facility has extensive infected decubitus ulcers on his buttocks and lower extremities. What should the ED staff do to protect themselves from being exposed to this disease?

The scenario is most consistent with MRSA. Use contact precautions, including a private room, gloves at all times, and handwashing with an antimicrobial agent after glove removal, and wear a gown at all times.

95. What is PANDAS?

This condition has been described as **p**ediatric **a**utoimmune **n**europsychiatric **d**isorders **a**ssociated with **s**treptococcal infections. An association between GAS and PANDAS has not been proved.

American Academy of Pediatrics: Group A streptococcal infections. In Pickering, LK (ed): Red Book: 2006 Report of the Committee on Infectious Diseases, 27th ed. Elk Grove Village, IL, American Academy of Pediatrics, 2006, p 611.

96. What are the most common etiologic agents of bacterial meningitis?

- **0 to 4 weeks:** GBS, *E. coli*, *Klebsiella pneumoniae*, *Salmonella* spp., other gram-negative bacilli, *L. monocytogenes*, enterococci
- **4 weeks to 3 months:** GBS, *S. pneumoniae*, *N. meningitidis*, *E. coli*, *H. influenzae*, *L. monocytogenes*
- **3 months to 18 years:** *S. pneumoniae*, *N. meningitidis*, *H. influenzae*

Mann K, Jackson A: Meningitis. Pediatr Rev 2008;29:417-429.

97. You are evaluating a 12-month-old infant for possible meningitis. He comes from a community that does not believe in immunization for religious reasons. Given the history, which of the three possible pathogens should you be most concerned about?

Hib. The epidemiology and microbiology of childhood meningitis have changed dramatically since the introduction of the Hib conjugate vaccines. Prior to the vaccine, Hib caused the majority of cases of bacterial meningitis in children. Peak incidence of meningitis was between 6 and 18 months. It is no longer a significant pathogen in countries where Hib vaccination is used because of the vaccine's ability to decrease pharyngeal colonization. Since 1988, when the Hib vaccine was introduced, we now see fewer than 1/100,000 cases of meningitis caused by Hib in children younger than 5 years old. Hib disease is currently seen in the United States in closed communities that do not believe in immunizations, or in very young infants who have not completed their vaccination series.

98. Which antibiotics should be used to treat neonatal meningitis (age 0-3 months)?

Ampicillin and cefotaxime. Ampicillin and gentamicin are commonly used for the treatment of neonatal sepsis, but if the CSF findings are consistent with meningitis, consider a third-generation cephalosporin in addition to ampicillin because of its superior CNS penetration. Consider acyclovir therapy for HSV, especially in those febrile neonates with a rash, seizure, maternal history, or ill appearance.

99. Why is ampicillin used to treat meningitis in the 0- to 3-month-old infant?

It is used to provide adequate antimicrobial coverage for *L. monocytogenes*. Frequently, an aminoglycoside, such as gentamicin, is added to improve the effectiveness of the ampicillin. Penicillin-allergic patients may be treated with trimethoprim-sulfamethoxazole.

100. What empirical antibiotic therapy should be initiated for the treatment of bacterial meningitis outside the neonatal period (>3 months)?

Administer vancomycin and ceftriaxone to all infants with definite or presumed bacterial meningitis. Vancomycin is recommended for all CNS infections that may involve

pneumococcus as a pathogen because of pneumococcal resistance to penicillin and other β-lactam antibiotics. For the child with hypersensitivity to β-lactam antibiotics, consider the combination of rifampin plus vancomycin. Vancomycin should not be given alone.

American Academy of Pediatrics: Pneumococcal infections. In Pickering LK (ed): Red Book: 2006 Report of the Committee on Infectious Diseases, 27th ed. Elk Grove Village, IL, American Academy of Pediatrics, 2006, pp 529-530.

101. Should corticosteroids be administered as adjunctive therapy in the treatment of bacterial meningitis?

Corticosteroids have been shown to reduce hearing loss and neurologic sequelae in children with Hib meningitis. The effectiveness of corticosteroids in disease caused by *S. pneumoniae* and *N. meningitidis* is less clear, although retrospective studies have shown significant reductions in hearing loss among those treated with dexamethasone with antibiotics versus antibiotics alone. There is evidence that dexamethasone reduces the incidence of neurologic complications in adults with *S. pneumoniae* meningitis. Current recommendations suggest that dexamethasone be considered for children with pneumococcal meningitis. If used, give dexamethasone before or at the time of antibiotic administration.

van de Beek D, de Gans J, McIntyre P, Prasad K: Corticosteroids in acute bacterial meningitis. Cochrane Database Syst Rev 2007;CD004405.

102. What is aseptic meningitis?

This term has become synonymous with viral meningitis but in fact describes meningitis caused by viruses, fungi, or certain bacteria that cannot be seen on a Gram stain. It also applies to meningitis caused by drugs (nonsteroidal anti-inflammatory drugs), systemic illness, neoplasms, or parameningeal conditions. This term does not imply an aseptic course of illness or an aseptic-appearing child. A more accurate term would be *Gram stain–negative meningitis*.

103. How can viral meningitis be distinguished from bacterial meningitis?

It is often difficult to distinguish these two clinical entities. Both can present with fever, headache, stiff neck, vomiting, and photophobia. Young infants may show nonspecific signs such as irritability, poor oral intake, and somnolence. The typical CSF findings can sometimes be used to distinguish viral from bacterial meningitis, although considerable overlap can exist (Table 38-14). More recent studies have demonstrated the utility of the Bacterial Meningitis Score (predictors of bacterial meningitis being a positive CSF Gram stain, CSF protein 80 mg/dL or greater, CSF neutrophils 1000 cells/uL or greater, peripheral absolute neutrophil count 10,000 cells/uL or greater, seizure before or at the time of presentation), enteroviral polymerase chain reaction (PCR), and serum procalcitonin in differentiating viral and bacterial meningitis.

Kowalski RH, Jaffe DM: Bacterial meningitis post-PCV7. Pediatr Emer Care 2013;29:758-769.
Nigrovic L, Kuppermann N, Macias C, et al: Clinical prediction rule for identifying children with cerebrospinal fluid pleocytosis at very low risk of bacterial meningitis. JAMA 2007;297:52-60.

Table 38-14. Typical Cerebrospinal Fluid Findings in Viral and Bacterial Meningitis

CSF VARIABLE	BACTERIAL	VIRAL
WBC count (cells/mm^3)	>1000	<500
Neutrophils (%)	>50	<50
Glucose level (mg/dL)	<20	>30
Protein level (mg/dL)	>100	50-100

CSF, cerebrospinal fluid; WBC, white blood cell.

104. On a hot summer day, a 5-year-old boy presents with fever, headache, and photophobia. You suspect meningitis and perform a lumbar puncture. The CSF evaluation reveals a WBC count of 400 cells/mL with a polymorphonuclear cell predominance, normal glucose, normal protein, and negative Gram stain. What is the most likely diagnosis?

Enteroviral meningitis is the most likely diagnosis. Nonpolio enteroviruses are the leading cause of viral meningitis, accounting for 80% to 90% of all cases in which an etiologic agent is identified. This group includes group A and B coxsackieviruses, echoviruses, and enteroviruses (types 68-71 and 73). They are called *summer viruses* because resulting infections occur during the warmer months. The CSF examination in this patient is typical of enterovirus infection. The predominance of neutrophils is often seen *early* in the course of the disease. The presence of other identifiable enteroviral syndromes in the community, such as herpangina and hand-foot-mouth disease, can be useful in the diagnosis because outbreaks tend to be epidemic and seasonal. The availability of reliable enteroviral PCR in CSF makes it easier to distinguish these two entities.

Pasquinelli L: Enterovirus infections. Pediatr Rev 2006;27(2):e14-e15.

105. A 2-week-old infant presents with fever and focal seizures. What therapy should be instituted immediately?

Start ampicillin, cefotaxime, and acyclovir as soon as possible. This child's presentation, especially the seizures, is consistent with HSV encephalitis. Typically, fever, lethargy, and seizures occur at 2 to 3 weeks of age. Skin lesions are present in about 50% of patients, and in most will appear late in the illness if at all. CSF examination usually reveals fewer than 100 WBCs but may have an increased number of red blood cells. CSF cultures are usually negative for HSV. HSV DNA in the CSF can best be detected using PCR. Neonatal infections are serious, with high mortality and morbidity rates even with prompt treatment. About one third of neonatal HSV infections involve the CNS, and although most patients survive, there is a high incidence of neurologic impairment. This patient needs empirical therapy for both bacterial and HSV infection.

Shah S, Aronson P, Mohamed Z, et al: Delayed acyclovir therapy and death among neonates with herpes simplex virus infection. Pediatrics 2011;128:1153-1160.

106. A 10-year-old boy has a fever and is suddenly acting strangely, with combative behavior and garbled speech. What is the differential diagnosis for this presentation?

Fever in combination with a change in mental status suggests a diagnosis of encephalitis. Increased intracranial pressure without signs of meningitis can also be seen. Infectious causes of encephalitis are predominately viral in origin. Epidemic acute encephalomyelitis is usually due to arboviruses (*ar*thropod *bo*rne viruses) such as West Nile and LaCrosse viruses. Sporadic acute encephalitis can be due to many viruses such as enteroviruses, HSV, varicella, cytomegalovirus, and Epstein-Barr virus. Bacterial causes are less common, but in a child with exposure to cats (especially kittens), consider infection with *Bartonella henselae* (cat-scratch disease). Other infectious diseases that cause encephalitis include RMSF, Lyme disease, and *M. pneumoniae* infection. Acute disseminated encephalomyelitis (ADEM), also known as a postinfectious encephalomyelitis, is a rare monophasic inflammatory disorder of the CNS that can also present in this manner. ADEM has a propensity to occur after minor upper respiratory tract infections. CSF, by definition, reveals no bacterial or viral cause. The existence of bilateral optic neuritis and transverse myelitis, however, is consistent with the diagnosis of ADEM.

Falchek SJ: Encephalitis in the pediatric population. Pediatr Rev 2012;33:122-133.

107. A 12-year-old boy with a 2-week history of sinusitis presents with persistent fever, headache, and altered mental status. What serious complication of sinusitis should be considered?

Brain abscess is an uncommon but serious complication of frontal sinusitis. It is seen in older children and teenagers because of the late development of the frontal sinuses. Maintain a high index of suspicion when patients with sinusitis present with persistent symptoms and neurologic findings. Other potential complications include epidural abscess or meningitis.

Hicks CW, Weber JG, Reid JR, et al: Identifying and managing intracranial complications of sinusitis in children: A retrospective series. Pediatr Infect Dis J 2011;30:222-226.

Table 38-15. Distinguishing Between Infectious Causes of Back Pain

VARIABLE	DISKITIS OSTEOMYELITIS	VERTEBRAL	SPINAL EPIDURAL ABSCESS	SACROILIAC JOINT INFECTION
Age (y)	<5	<8	>8	Any
Symptoms	Gradual onset	Gradual onset	Severe	Gradual onset
	Increased with activity	Constant, dull	Constant	Sciatica
			Radiation to legs	Buttock pain
Fever	Low	High	High	High
Examination	Locally tender Lumbar lordosis Spinal mobility Refusal to walk	Locally tender Spinal mobility	Locally tender Spinal mobility Deep tendon reflexes Strength	

108. An 8-year-old boy has fever and back pain. What is your approach to diagnosis and management?

Back pain is an unusual reason for children to be seen in the ED. Frequently, back pain in children indicates significant disease. The differential diagnosis includes infectious, neoplastic, and rheumatologic disorders. The major infections producing back pain in children include diskitis, vertebral osteomyelitis, spinal epidural abscess, and sacroiliac joint infection (Table 38-15).

109. What is the appropriate treatment for a child who presents with erythema migrans?

Erythema migrans is the characteristic rash of Lyme disease, seen in approximately 70% of patients. It begins as a red papule or macule that rapidly expands to become a round or oval lesion at least 5 cm in diameter, with an erythematous periphery and central clearing. The treatment of choice is doxycycline for children 8 years old or older. Amoxicillin is recommended for younger children (because of possible tetracycline toxicity). Treatment for 2 to 3 weeks is recommended. Cefuroxime axetil is an approved alternate therapy.

110. What is the recommended treatment for Lyme disease in children?

See Table 38-16 for the recommended treatment for Lyme disease in children.

111. A child presents with bilateral facial palsy. What disease entities would you consider?

In areas that are endemic for Lyme disease in the United States (Northeast, upper Midwest, and Pacific Northwest), always suspect Lyme disease. It is one of the few diseases that can produce bilateral facial palsy. Other causes of facial palsy, usually unilateral, include mastoiditis, herpesvirus infection, and tumors. Guillain-Barré syndrome can cause bilateral facial palsy, usually associated with generalized weakness.

112. A 16-year-old otherwise healthy girl presents with a syncopal episode. Electrocardiography reveals complete heart block. What is the differential diagnosis?

- Lyme carditis
- Repaired congenital heart disease
- Enteroviral myocarditis
- Digoxin toxicity

In Lyme disease–endemic areas, always consider Lyme disease when children present with rhythm disturbances, as a fatal outcome may occur and because antibiotic treatment is usually curative.

Table 38-16. Treatment of Lyme Disease

DRUG	PEDIATRIC DOSING
Amoxicillin	50 mg/kg/day in three divided doses (max 1500 mg/day)
Doxycycline	4 mg/kg/day in two divided doses (max 200 mg/day) (see text regarding doxycycline use in children)
Cefuroxime axetil	30 mg/kg/day in two divided doses (max 1000 mg/day)
Ceftriaxone ([intravenous]IV)	50-75 mg/kg/day once daily (max 2000 mg/day)
Recommended Therapy Based on Clinical Manifestation	
Erythema migrans	Oral regimen, 14-21 days
Meningitis	Ceftriaxone, 10-28 days
Cranial nerve palsy	Oral regimen, 14-21 days (see text regarding possible need for lumbar puncture)
Cardiac disease	Oral regimen or ceftriaxone, 14-21 days (see text for specifics)
Arthritis	Oral regimen, 28 days
Late neurologic disease	Ceftriaxone, 14-28 days

Adapted from Wormser GP, Dattwyler RJ, Shapiro ED, et al: The clinical assessment, treatment, and prevention of Lyme disease, human granulocytic anaplasmosis, and babesiosis: Clinical practice guidelines by the Infectious Diseases Society of America. Clin Infect Dis 2006;43:1089-1134.

113. What is the time course of clinical manifestations of Lyme disease?

The time course of clinical manifestations of Lyme disease can be seen in Table 38-17.

Key Points: Possible Presentations of Lyme Disease in Children

1. Rash
2. Arthritis
3. Heart block
4. Meningeal signs
5. Facial palsies

Table 38-17. Time Course of Clinical Manifestations of Lyme Disease

DISEASE STAGE	TIMING AFTER TICK BITE	TYPICAL CLINICAL MANIFESTATIONS
Early localized	3-30 days	Erythema migrans (single), variable constitutional symptoms (headache, fever, myalgia, arthralgia, fatigue)
Early disseminated	3-12 wk	Erythema migrans (single or multiple), worse constitutional symptoms, cranial neuritis, meningitis, carditis, ocular disease
Late	>2 mo	Arthritis

114. Describe the distribution of the rash in RMSF.

RMSF is a systemic disease caused by *R. rickettsii*. The rash usually occurs between the third and fifth days of the febrile illness (temperature, 38.5° C) as a blanching maculopapular rash on the ankles and wrists. It spreads centripetally to the trunk but usually spares the face. In most patients, the rash involves the palms and soles and eventually becomes petechial or purpuric.

Other clinical features of RMSF include headache, myalgia, conjunctival hyperemia, and photophobia. Approximately 20% of cases may have an atypical rash or no rash at all.

American Academy of Pediatrics: Rocky Mountain spotted fever. In Pickering LK (ed): Red Book: 2012 Report of the Committee on Infectious Diseases, 29th ed. Elk Grove Village, IL, American Academy of Pediatrics, 2012, pp 623-625.

115. Which electrolyte abnormality is commonly associated with RMSF?

Hyponatremia (sodium level < 130 mEq/L) is seen in about 25% of affected patients. Other laboratory abnormalities commonly include thrombocytopenia, anemia, and a variable WBC count with a left shift.

116. In which Rocky Mountain state is RMSF most commonly seen?

Trick question. "Rocky Mountain" is a misnomer. Even though Howard Ricketts (both genus and species are named for him) performed his seminal work in the Bitterroot Valley of Montana a century ago, most modern cases of RMSF occur in the South Atlantic, South Central, and the Southeastern United States.

117. What laboratory abnormalities are found in ehrlichiosis?

- Leukopenia
- Hepatitis (elevated aminotransferase levels)
- CSF pleocytosis (with predominance of lymphocytes)
- Anemia
- Thrombocytopenia
- Hyponatremia

118. When a child is found to have bloody diarrhea, which infectious agents are most likely responsible?

The most common agents are *Salmonella*, *Shigella*, *Campylobacter* spp., shiga toxin producing *E. coli* (*E. coli* O157:H7), and *Yersinia* spp. Consider other infectious agents, including *Vibrio parahaemolyticus*, *Aeromonas* spp., and *Entamoeba histolytica* (acute amebiasis), based on a patient's potential for exposure.

119. Which bacterial stool pathogens require antimicrobial treatment?

- ***Shigella:*** Most infections are self-limiting; however, antibiotics can reduce the duration of symptoms. Antibiotics are therefore recommended for severe disease with dehydration or in immunocompromised individuals.
- ***Salmonella:*** Treatment does not shorten the duration of the symptoms and may actually prolong the carrier state. However, bacteremia and invasive disease are common in infants younger than 3 months of age, in patients with sickle cell disease, and in other immunocompromised individuals. Therefore, treatment is indicated in those populations.
- ***E. coli* O157:H7:** No treatment has shown a proven benefit. Although there are conflicting data regarding an increased risk for hemolytic-uremic syndrome when antibiotics are used to treat *E. coli* 0157:H7, most experts recommend against this practice.

120. A 2-year-old presents with vomiting, profuse diarrhea, and new-onset seizures. What could be responsible for this clinical picture?

Shigella infections are associated with CNS symptoms, including seizures and toxic encephalopathy (Ekiri syndrome). The seizures are self-limited. Severe diarrhea, regardless of the cause, however, may cause electrolyte abnormalities that result in seizures.

American Academy of Pediatrics: *Shigella* infections. In Pickering LK (ed): Red Book: 2012 Report of the Committee on Infectious Diseases, 29th ed. Elk Grove Village, IL, American Academy of Pediatrics, 2012, pp 645-647.

121. Should antimotility agents be used to manage gastroenteritis in children?
In general, no. The medications have limited benefit and have a high rate of side effects. They are actually contraindicated in dysenteric illness, as they impede excretion of infectious agents and toxins. Nonnarcotic antimotility agents may be used in afebrile patients with nonbloody diarrhea.

122. Which organisms cause food-borne illnesses? How can you distinguish them?
The onset, symptoms, and cause of food-borne illnesses are listed in Table 38-18.

123. A 3-month-old infant presents with a 3-day history of constipation, progressively poor feeding, and lethargy. On physical examination you notice a quiet, inactive child who is otherwise alert with good perfusion. The neurologic examination reveals a weak cry, poor suck, and hypotonia. What is the likely diagnosis?
Infant botulism. Infants with botulism may appear to be septic but usually are afebrile and have stable vital signs. Botulism occurs after ingestion of airborne spores from soil or dust. The ingested spores then germinate and produce toxin in the intestine of the infant. Peak incidence is in infants 6 weeks to 6 months of age. Breastfeeding is a significant risk factor. The ingestion of honey or corn syrup was often implicated in the past. A history of soil disruption, which occurs near construction sites, has been a common epidemiologic finding in reported cases. Hospitalize babies with suspected infant botulism and observe for progressive weakness

Table 38-18. Onset, Symptoms, and Etiology of Food-Borne Illnesses

TIME OF ONSET	MAIN SYMPTOMS	ORGANISM OR TOXIN
Upper Gastrointestinal Tract		
1-6 hours	Nausea, vomiting, usually afebrile	*Staphylococcus aureus*
1-6 hours	Nausea, vomiting, afebrile	*Bacillus cereus* (emetic form)
Lower Gastrointestinal Tract		
8-16 hours	Diarrhea, afebrile	*B. cereus* (diarrheal form)
6-24 hours	Foul-smelling diarrhea, cramps, afebrile	*Clostridium perfringens*
16-48 hours	Abdominal cramps, diarrhea, fever	*Vibrio cholerae*, Norwalk virus, *Escherichia coli* O157:H7, *Cryptosporidium* spp.
16-72 hours	Bloody diarrhea, fever, abdominal cramps	*Salmonella*, *Shigella*, and *Campylobacter* spp., *E. coli*
16-72 hours	Bloody diarrhea, fever, pseudoappendicitis, pharyngitis	*Yersinia enterocolitica*
1-6 weeks	Mucoid diarrhea (fatty stools), abdominal pain, weight loss	*Giardia lamblia*
Neurologic Infection		
12-36 hours	Vertigo, diplopia, areflexia, weakness, difficulty breathing and swallowing, constipation	*Clostridium botulinum*
Generalized Infection		
14 days	All of the preceding symptoms, plus vomiting, rose spots, constipation, abdominal pain, fever, chills, malaise, swollen lymph nodes	*Salmonella typhi*

Adapted from American Academy of Pediatrics: Appendix IX Clinical syndromes associated with food borne diseases. In Pickering LK (ed): Red Book: 2012 Report of the Committee on Infectious Diseases, 28th ed. Elk Grove Village, IL, American Academy of Pediatrics, 2012, pp 861-863.

and respiratory failure. Botulism immune globulin IV is an antitoxin that, when given early, has been found to decrease the duration of illness and hospital stay.

American Academy of Pediatrics: Clostridial infections. In Pickering LK (ed): Red Book: 2012 Report of the Committee on Infectious Diseases, 29th ed. Elk Grove Village, IL, American Academy of Pediatrics, 2012, pp 281-284.

124. Why is the diagnosis of *Clostridium difficile* colitis tricky in infants?

Almost 50% of infants can have asymptomatic colonization, so toxin production is present without symptoms. Immature enterocytes with a decreased amount of toxin-binding capacity and maternal antibodies have been postulated as mechanisms for disease protection in infants. Disease is rarely seen in infants younger than 3 months of age. Most experts discourage testing for *C. difficile* toxin in infants younger than 1 year who have diarrhea.

125. A 5-year-old who resides in a group home is transported to the ED because he looks "yellow." What are the diagnostic considerations?

Hepatitis A virus (HAV) is highly contagious and spreads among children with poor hygiene or those in close quarters. Outbreaks at child care facilities usually represent an extension of a community outbreak. HAV infection is abrupt in onset and self-limiting, consisting of fever, anorexia, nausea, and headache. Jaundice is uncommon in young children compared with older children and adults, in whom jaundice can occur in 70% of cases. Liver failure in HAV is not common. The differential diagnosis includes hepatitis B or C (obtain a good history for associated risk factors: maternal history, blood transfusions, sexual contact) and Epstein-Barr virus infection.

Key Points: Evaluation of Bloody Diarrhea

1. Send stool cultures for *Salmonella*, *Shigella*, *Yersinia*, and *Campylobacter* spp. and *E. coli* testing.
2. Initiate antimicrobial treatment for *Salmonella* and *Shigella* infections if the patient is clinically unstable, severely dehydrated, immunocompromised, or younger than 3 months of age.
3. Consider other noninfectious causes (e.g., inflammatory bowel disease).

126. Which animals should be considered at high risk for transmission of rabies? How is rabies managed?

Bats, foxes, and raccoons should be considered rabid. In the United States between 2000 and 2009, 77% of human deaths from rabies were associated with bats. Cats and dogs may also carry rabies. Rabies in small rodents (squirrels, hamsters, rats, and mice) is uncommon. If bitten or significantly exposed, patients should receive postexposure prophylaxis, including both passive and active therapy. Rabies immune globulin can be used for passive immunization. An exposed individual will need four total doses of active immunization with an approved rabies vaccine (administered on days 0, 3, 7, and 14 after the exposure). Domestic animals, such as dogs, can be observed for 10 days after a bite for observation, because canine rabies in the United States is rare.

American Academy of Pediatrics: Rabies. In Pickering LK (ed): Red Book: 2012 Report of the Committee on Infectious Diseases, 29th ed. Elk Grove Village, IL, American Academy of Pediatrics, 2012, pp 600-607.

127. Should a child who has been in proximity to bats (e.g., in the same room during sleep), but without known physical contact, receive prophylaxis?

Yes. Cases of rabies have been reported in the absence of known contact with a rabid animal. Insignificant physical contact or airborne transmission of the virus may be responsible.

American Academy of Pediatrics: Rabies. In Pickering LK (ed): Red Book: 2012 Report of the Committee on Infectious Diseases, 29th ed. Elk Grove Village, IL, American Academy of Pediatrics, 2012, pp 600-607.

128. True or false: Cat bites have a greater chance of becoming infected than dog bites.

True. Wounds from cat bites are more than twice as likely to become infected, in part because of the deep puncture wounds that result. The rate of infection from cat bites can be more than 50% in some cases. *Pasteurella multocida* is the most common organism seen in cat bite infections; *Pasteurella canis* is seen in 25% of dog bites.

129. When should antibiotics be prescribed for animal bites?

Antibiotic use is recommended for all deep puncture wounds (e.g., cat bites), moderate and severe wounds (with edema or crush injury), bites involving certain areas of the body (face, hand/foot, genitals), and bites in immunocompromised or asplenic patients. The recommended oral agent is amoxicillin-clavulanate; IV ampicillin-sulbactam is recommended for more severe infections. These antibiotics cover all β-hemolytic streptococci, many *S. aureus* and anaerobes, and *P. multocida*. For penicillin-allergic patients, either a third-generation cephalosporin *or* trimethoprim-sulfamethoxazole in combination with clindamycin is recommended.

130. An afebrile 2-month-old presents with tachypnea and cough. What are the causes of pneumonia in this age group?

Respiratory viruses account for up to 90% of pneumonias in the 1- to 3-month-old age group. Pneumonitis without fever in this age group may represent an infection with *C. trachomatis* or *Bordetella pertussis*. *C. trachomatis* is perinatally acquired and peaks at 4 to 11 weeks of age. Infants usually present with nasal discharge and a staccato cough and have a history of conjunctivitis (50%). The chest radiograph reveals interstitial infiltrates and hyperinflation. *B. pertussis* usually presents with upper respiratory symptoms, paroxysms of cough, often followed by vomiting, and sometimes apnea in the young infant.

131. Describe the acute management of bronchiolitis due to RSV.

- Measurement of oxygen saturation
- Supportive treatment (mist treatments, suction, oxygen if needed)
- Hydration (IV fluid if dehydrated)
- Trial of a bronchodilator (albuterol nebulized treatment), although various studies have demonstrated variable response (some children may respond if they have a component of reactive airways disease)

Most studies do not support routine use of either bronchodilators or glucocorticoids (or a combination) in RSV bronchiolitis. Inhaled ribavirin may be lifesaving in severely immunocompromised infants and children but is not recommended for routine use.

132. What is the treatment of choice for presumed *C. trachomatis* infection (either conjunctivitis or pneumonia)?

Oral erythromycin for 14 days. Chlamydial conjunctivitis alone is treated systemically to prevent the pneumonitis. Exercise caution with use of macrolides in infants younger than 6 weeks of age because they have been associated with hypertrophic pyloric stenosis. Azithromycin is probably effective based on limited data.

American Academy of Pediatrics: Chlamydial infections. In Pickering LK (ed): Red Book: 2012 Report of the Committee on Infectious Diseases, 29th ed. Elk Grove Village, IL, American Academy of Pediatrics, 2012, pp 276-281.

133. Describe the characteristics of pertussis in infancy.

Most severe pertussis cases occur in infants younger than 6 months of age. This population does not always have the classic "whoop" with cough. Apnea can be a common presenting symptom. Fever is absent or low grade. Adolescents and adults in the household are a major reservoir for transmission of pertussis because of their waning vaccine-induced immunity. Consider pertussis in any infant with cough for more than 10 to 14 days. Complications in infants with pertussis include pneumonia, seizures, encephalopathy, and death.

134. What is the drug of choice for the treatment of pertussis?

Oral erythromycin for 14 days has long been the standard treatment. If started early in the illness, antibiotic therapy may be associated with amelioration of clinical symptoms. After the paroxysmal cough is established, however, antibiotics have no effect on the illness and are used more for prevention of spread to others. Oral azithromycin for 5 days has also been shown to be effective and is associated with better adherence. The AAP considers azithromycin the drug of choice in infants younger than 1 month because of the association of pyloric stenosis with erythromycin in this age group.

135. What are the most common types of pneumonia in a 2-year-old?

Respiratory viruses are the most common causes of pneumonia in toddlers. Viral causes include the following:

- RSV
- Influenza viruses
- Parainfluenza viruses
- Adenovirus
- Human metapneumovirus
- Rhinovirus

Viral pneumonia is usually gradual in onset and preceded by upper respiratory tract symptoms; examination reveals diffuse auscultatory findings. Most of these children appear ill but not toxic.

136. When should you consider admitting an older child with pneumonia?

- Hypoxia
- Toxic appearance
- Complicated pneumonia, e.g., empyema
- Failed outpatient treatment
- Respiratory distress, e.g., tachypnea, retractions, nasal flaring, and grunting
- Questionable adherence
- Inability to maintain oral intake
- Underlying condition predisposing to severe pneumonia, e.g., cardiopulmonary disease, immunocompromised state
- Suspected infection with virulent pathogen, e.g., MRSA

Bradley JS, Byington CL, Shah SS, et al: The management of community-acquired pneumonia in infants and children older than 3 months of age: Clinical practice guidelines by the Pediatric Infectious Diseases Society and the Infectious Diseases Society of America. Clin Infect Dis 2011;53: e25-e76.

137. What clinical clues suggest typical bacterial pneumonia?

Bacterial pneumonia is often characterized by abrupt onset of high fever, respiratory distress (tachypnea, shortness of breath, hypoxia), and sometimes vomiting. Physical examination may reveal rales or decreased breath sounds. Chest radiograph may show lobar or segmental dense infiltrates associated with pleural effusion. A WBC count more than 15,000 cells/mm^3 or elevated CRP level may indicate a bacterial process.

138. What are the typical bacterial causes of pneumonia in children outside the neonatal period?

S. pneumoniae accounts for most bacterial pneumonias in children. Other pyogenic causes include GAS and *S. aureus*. All these pathogens may cause severe, necrotizing pneumonia, often with pneumatoceles and pleural empyema.

139. A 10-year-old presents with nonproductive cough, sore throat, low-grade fever, and malaise. On physical examination, the patient appears well, has minimal respiratory distress, and has bilateral lower lung field crackles. What is the most frequent cause of pneumonia in this age group?

This is a classic presentation of a child with atypical pneumonia caused by *M. pneumoniae* or *C. pneumoniae*. *M. pneumoniae* is a common cause of pneumonia in older children and young adults. It accounts for about 40% of community-acquired pneumonias (CAPs) in certain populations. Bilateral diffuse infiltrates are the most common radiographic findings, which are frequently out of proportion to clinical findings. Extrapulmonary manifestations, including a maculopapular rash (10%), CNS disease, hemolytic anemia, arthritis, and hepatitis, are occasionally seen in atypical pneumonia syndromes caused by *M. pneumoniae*.

Key Points: Common Etiologic Agents of Pneumonia in Children

1. **Neonate:** GBS, *E. coli*, and other gram-negative bacilli, *L. monocytogenes*, *U. urealyticum* (premature)
2. **Infant (<3 months):** RSV, human metapneumovirus, influenza virus, parainfluenza virus, adenovirus, *C. trachomatis*
3. **Infant to toddler (<5 years):** Respiratory viruses, *S. pneumoniae*, *M. pneumoniae*
4. **School age to adolescence:** *M. pneumoniae*, *C. pneumoniae*, *S. pneumoniae*, respiratory viruses

140. What test can be used to assist in the diagnosis *of M. pneumoniae* infection?

Cold agglutinins are IgM antibodies that agglutinate human red blood cells and are found in approximately 50% of patients with acute mycoplasmal infection, and in some viral infections as well (adenovirus, Epstein-Barr virus, and measles virus). Although available, cold agglutinins have a poor specificity profile. Serologic testing is not very useful in the ED management of these patients. PCR has become more widely available and can have a rapid turnaround time, but the reported sensitivity and specificity of the various tests vary widely.

141. Which antimicrobial agents can be used for outpatient treatment of CAP?

The treatment of CAP is based on the most likely etiologic agents. When typical bacterial pneumonia is suspected, begin treatment with adequate coverage against penicillin-resistant *S. pneumoniae*. Among available oral antibiotics, amoxicillin at a high dose (80-100 mg/kg) provides the best coverage against resistant *S. pneumoniae*. Alternatives include oral second- or third-generation cephalosporins or levofloxacin. In the older child, include treatment with coverage against atypical organisms such as *M. pneumoniae* and *C. pneumoniae* with azithromycin.

Bradley JS, Byington CL, Shah SS, et al: The management of community-acquired pneumonia in infants and children older than 3 months of age: Clinical practice guidelines by the Pediatric Infectious Diseases Society and the Infectious Diseases Society of America. Clin Infect Dis 2011;53:e25-e76.

Key Points: Community-Acquired Pneumonia

1. In younger children, consider viruses and coverage for *S. pneumoniae*.
2. In older school-aged children, consider treatment to cover *M. pneumoniae*.

142. What are the most common sites for septic arthritis?

The lower extremity joints, such as the hip, knee, and ankle (less common), are common sites for septic arthritis. Primary pyogenic arthritis is uncommon in the post–*H. influenzae* era. Always suspect associated osteomyelitis when the joint "looks septic."

143. A 2-year-old presents with a limp. A radiograph of the suspected limb is negative. Does a negative radiograph eliminate the possibility of osteomyelitis?

No. In the first 2 weeks of osteomyelitis, plain radiographs are not sensitive for the detection of osteomyelitis (bony changes may not be evident yet). The gold standard for diagnosis is magnetic resonance imaging; a radionuclide bone scan is another option.

Frank G, Mahoney HM, Eppes SC: Musculoskeletal infections in children. Pediatr Clin North Am 2005;52:1083-1106.

144. What is the classically described position in which the leg is held by a child with septic arthritis of the hip?

The leg is flexed, externally rotated, and abducted.

145. What are the most common bones affected in osteomyelitis?

The long bones of the leg—femur and tibia—are most often affected. In neonates and infants, osteomyelitis may affect any bone, including the humerus, hips, mandible, or calcaneus.

Table 38-19. Differential Diagnosis of Septic Arthritis Based on White Blood Cell Count and Neutrophils

DIAGNOSIS	WHITE BLOOD CELLS (CELLS/MM^3)	NEUTROPHILS (%)
Normal	<200	10-20
Traumatic effusion	<2000	10-30
Rheumatologic	10,000-50,000	50-80
Septic arthritis	>50,000	≥80
Lyme disease	10,000-25,000	>50

146. Aspiration of the hip joint in a 2-year-old febrile child with a limp reveals a WBC count of 60,000 cells/mm^3 with a neutrophil predominance. What diagnosis is likely?

Septic arthritis is most likely. Table 38-19 can aid in diagnosis based on the WBC count and differential but demonstrates considerable overlap. Epidemiology, presence of fever, and detailed history may help establish a diagnosis.

Frank G, Mahoney HM, Eppes SC: Musculoskeletal infections in children. Pediatr Clin North Am 2005;52:1083-1106.

147. What are the differences between the clinicial presentations of Lyme arthritis and septic arthritis?

Lyme arthritis usually affects the knee. The affected joint has a moderate-size to large effusion, the joint may be warm but not hot to touch, the patient often can bear weight on the affected limb, and there is some active range of motion of the joint. A septic joint usually has a small to moderate-size effusion and is red and hot to touch. The patient is non–weight bearing if the hip or knee is affected, and there is little or no active or passive range of motion of the joint.

148. Match the disease with the appropriate management plan.

Disease:

A. Osteomyelitis
B. Septic arthritis of hip
C. Toxic synovitis

Management plan:

1. No antibiotics needed. Anti-inflammatory medications may be needed.
2. Admission to hospital. No IV antibiotics until patient is evaluated by orthopedic surgery and cultures of bone have been obtained.
3. Emergency, requiring surgical drainage and IV antibiotics.

Answers: 1, C; 2, A; 3, B.

149. What are the most common organisms found in bone and joint infections?

The most common organisms found in bone and joint infections are listed in Table 38-20.

150. What is pyomyositis?

Pyomyositis is an infection of skeletal muscle that may be associated with antecedent trauma. The cause is often *S. aureus* or GABHS in immunocompetent children, but gram-negative organisms may also be involved in infections seen in immunocompromised children. Fever, myalgias, muscle tenderness to palpation, or swelling of the involved muscle may be seen. Magnetic resonance imaging is extremely helpful in making the diagnosis. Treatment includes surgical drainage if abscesses are involved and IV antibiotics.

151. What is the classic description of roseola (primary human herpesvirus 6 infection)?

High fever (temperature > 39.5° C) for 3 to 7 days, nonspecific signs (e.g., irritability, decreased feeding, or toxic appearance), and a diffuse maculopapular rash soon after defervescence (about days 4-6 of illness) lasting hours to days. This classic presentation is seen in only 20% of patients. Human herpesvirus 6 infection accounts for approximately 20% of acute febrile illness visits for children 6 to 8 months of age. Seizures may occur in about 10% of patients during the febrile period. A bulging anterior fontanel occurs occasionally.

Table 38-20. Organisms Most Commonly Found in Bone and Joint Infections*

AGE	SEPTIC ARTHRITIS	OSTEOMYELITIS
Neonate	*Staphylococcus aureus*	*S. aureus*
	Group B streptococci	Group B streptococci
	Gram-negative bacilli	Gram-negative bacilli
Toddler	*S. aureus*	*S. aureus*
	Group A streptococci	Group A streptococci
	Streptococcus pneumoniae	*Kingella kingae*
	K. kingae	
School-age	*S. aureus*	*S. aureus*
	Group A streptococci	Group A streptococci

*In sexually active adolescents, also consider *Neisseria gonorrhoeae*. *Salmonella* is a common cause of osteomyelitis in children with sickle cell disease.

Adapted from Frank G, Mahoney HM, Eppes SC: Musculoskeletal infections in children. Pediatr Clin North Am 2005;52:1083-1106; and Gutierrez K: Bone and joint infections in children. Pediatr Clin North Am 2005;52:779-794.

152. A medical student reports to you that a 3-year-old febrile child appears to have been slapped on both cheeks by someone. What infectious disease are you considering?

Erythema infectiosum is the most common manifestation of parvovirus B19 infection. This infection is associated with a rash, characterized by intense erythema of the cheeks (slapped-cheek appearance) with circumoral pallor. On the trunk and extremities, the rash is maculopapular with a lacelike or reticular appearance.

153. List three other clinical presentations of parvovirus B19 infections.

- Polyarthritis syndrome—usually symmetrical and involves the small joints of the hands and feet, may also involve the knees
- Papular purpuric gloves-and-socks syndrome—rash on the hands and feet, often accompanied by edema, erythema, and fever
- Transient aplastic crisis, especially in patients with an underlying hemoglobinopathy

154. A 15-year-old with fever, exudative pharyngitis, lymphadenopathy, and splenomegaly presents to the ED. You suspect infectious mononucleosis. What laboratory tests should be obtained to aid in diagnosis?

Obtain a CBC and heterophil antibody (IgM) test. The WBC count can vary, but sometimes anemia, leukopenia, and thrombocytopenia can be seen. An increase in the number of atypical lymphocytes (10%) is a characteristic finding. The heterophil antibody test (Monospot) is specific for Epstein-Barr virus. In adolescents, the heterophil response occurs more reliably after the first week of illness. Its sensitivity is poor in young children (<4 years of age). The younger child is more likely to be asymptomatic or have a nonspecific infection.

155. What should you recommend to a patient with infectious mononucleosis upon discharge from the ED?

Tell the patient to avoid strenuous and contact sports until examined by a physician and until the spleen is no longer palpable to prevent traumatic rupture of the spleen, which can be life-threatening.

156. Should patients with infectious mononucleosis be treated with corticosteroids?

Steroids are indicated for significant tonsillar inflammation with impending airway obstruction, hemophagocytic syndrome, massive splenomegaly, hemolytic anemia, and myocarditis.

American Academy of Pediatrics: Epstein-Barr virus infections. In: Pickering LK (ed): Red Book: 2012 Report of the Committee on Infectious Diseases, 29th ed. Elk Grove Village, IL, American Academy of Pediatrics, 2012, pp 318-321.

157. Match the disease with the clinical description.

Disease:

A. Varicella (chickenpox)
B. Measles
C. Rubella

Clinical description:

1. Fever, cough, coryza, and conjunctivitis. Confluent maculopapular rash beginning on upper part of body; can involve palms and soles.
2. Fever; coalescent, pink, maculopapular rash that begins on face and extends downward; tender postauricular, suboccipital, and posterior cervical lymph nodes. Prodromal symptoms of cough, malaise, and conjunctivitis are uncommon.
3. Prodrome of fever, papules, vesicles, and umbilicated and scabbed lesions. Hallmark is lesions present in different stages at the same time. Lesions develop in crops. Initial lesion typically appears in scalp, and lesions can involve oral mucosa.

Answers: 1, B; 2, C; 3, A.

158. How long is the child with chickenpox contagious?

The period of communicability begins 1 to 2 days prior to the outbreak of lesions and lasts until all lesions are crusted over.

159. A child presents with chickenpox, high fever, and toxic appearance. What should you suspect?

Examine the child closely for evidence of secondary bacterial infection with *S. aureus* and *S. pyogenes* (group A). Secondary bacterial infection is the most common cause of morbidity in children with varicella infection. Clinical features include high fever, toxic appearance, and a varicella lesion that has become erythematous, warm, and painful. Superinfection with GAS can progress to soft tissue necrosis, necrotizing fasciitis, and sepsis.

160. A 1-year-old infant has a fever and rash 10 days after the annual visit to the pediatrician. What important history should be obtained regarding the recent visit to the pediatrician?

Immunization history. Determine if the measles, mumps, and rubella vaccine was administered. Fever with body temperature as high as 39° C develops in 5% to 15% of children 7 to 12 days after vaccination. The fever typically lasts 1 to 2 days, and approximately 5% of patients develop a rash. Varicella vaccination may also be given at 1 year of age. Within 1 month of vaccination, 5% to 10% of children develop a mild maculopapular or varicelliform rash. The rash is limited to only a few lesions that can be at the injection site or elsewhere.

161. A 4-year-old boy presents with a chief complaint of body temperature of 40° C for 7 days. On physical examination, you notice bilateral conjunctivitis, a strawberry red tongue, anterior cervical lymphadenopathy, and diffuse erythema of the perineal area. What is your diagnosis?

Kawasaki disease. This multisystem vasculitis has a peak incidence in childhood; 80% of cases occur in children younger than 4 years old.

162. What are the diagnostic criteria for Kawasaki disease?

"CRASH" and burn:

1. Fever for at least 5 days (burn)
2. Presence of four of the following:
   - **C**onjunctivitis: bilateral, bulbar, nonpurulent conjunctival injection
   - **R**ash: diffuse polymorphous rash
   - **A**denopathy: cervical lymphadenopathy larger than 1.5 cm, usually unilateral
   - **S**trawberry tongue: mucous membrane changes (cracked or erythematous lips, strawberry tongue, oral or pharyngeal erythema)
   - **H**ands and feet: swelling and erythema of hands and feet

Newburger JW, Takahashi M, Gerber MA, et al: Diagnosis, treatment and long-term management of Kawasaki disease: A statement for health professionals from the Committee on Rheumatic Fever, Endocarditis, and Kawasaki Disease, Council on Cardiovascular Disease in the Young, American Heart Association. Pediatrics 2004;114(6):1708-1733.

163. What is the differential diagnosis of Kawasaki disease?

- Measles
- Scarlet fever
- Toxic shock syndrome
- Juvenile rheumatoid arthritis
- Viral syndromes (adenovirus, Epstein-Barr virus, enteroviruses)
- RMSF
- Leptospirosis
- Mercury poisoning
- Drug reactions

Dedeoglu F, Sundei RP: Vasculitis in children. Pediatr Clin North Am 2005;52:547-575.

164. What laboratory findings support the diagnosis of Kawasaki disease?

- WBC count increased with left shift (>20,000 cells/mm^3 in 50% of patients, >30,000 cells/mm^3 in 15% of patients)
- Increased ESR or CRP
- Platelets increased during second week of illness
- Sterile pyuria (on a clean-catch UA)
- Elevation of serum transaminase levels
- Mild anemia
- Hypoalbuminemia
- Low phosphorus level

165. What is the management of a patient with suspected Kawasaki disease?

Management includes admission to the hospital for treatment with high-dose aspirin and IV immunoglobulin within 10 days of fever onset. All patients should have echocardiography performed as part of the initial hospital evaluation and then a repeat test 6 to 8 weeks after onset. Treatment within the first 10 days of the illness has been shown to substantially decrease the risk of coronary artery disease (a known complication of Kawasaki disease), from 20% to less than 5%.

Newburger JW, Takahashi M, Gerber MA, et al:: Diagnosis, treatment and long-term management of Kawasaki disease: A statement for health professionals from the Committee on Rheumatic Fever, Endocarditis, and Kawasaki Disease, Council on Cardiovascular Disease in the Young, American Heart Association. Pediatrics 2004;114(6):1708-1733.

166. What are some agents of bioterrorism?

Agents are broken down into categories A, B, and C. Category A agents provide the greatest risk. They can be easily transmitted from person to person and tend to have the highest mortality rates. Category B agents have lower mortality rates and are not as easily spread as category A agents. Category C agents can be engineered to be biologic weapons of mass destruction with the potential for high morbidity and high mortality rates.

- **Category A agents:** anthrax, smallpox, plague, tularemia, hemorrhagic fever viruses, botulinum toxin
- **Category B agents:** *Coxiella burnetii* (Q fever), *Brucella* species, *Burkholderia mallei*, alphaviruses (Venezuelan, eastern, and western equine encephalomyelitis), *Salmonella* species, *Shigella dysenteriae*, *E. coli* O157:H7, *Vibrio cholerae*, *Cryptosporidium parvum*
- **Category C agents:** hantavirus, tick-borne hemorrhagic fever viruses, yellow fever, and *M. tuberculosis* (multidrug-resistant)

Shannon M: Management of infectious agents of bioterrorism. Clin Pediatr Emerg Med 2004;5:63-71.

167. Match the bioterrorism disease with the organism and symptoms.

Disease:

A. Anthrax
B. Tularemia
C. Plague
D. Smallpox

Organism and symptoms:

1. Gram-negative rod resulting in pneumonia-like illness with fever, cough, dyspnea, large lymph node (bubo), and hemoptysis 2 to 4 days after exposure. Chest radiograph reveals bilateral infiltrates or lobar consolidation.
2. Spore-forming gram-positive bacillus resulting in a flulike illness: fever, chills, malaise, nonproductive cough, absence of rhinorrhea. Chest radiograph reveals widened mediastinum.
3. Gram-negative coccobacillus resulting in possible skin ulceration, pharyngitis, conjunctival injection, lymphadenitis, fever, and pneumonia. Chest radiograph reveals hilar adenopathy.
4. Member of the Poxviridae family resulting in low-grade fever and vesicular centrifugal rash 14 days after exposure. Lesions are umbilicated and in the same stage of development.

Answers: A, 2; B, 3; C,1; D, 4.

Shannon M: Management of infectious agents of bioterrorism. Clin Pediatr Emerg Med 2004;5:63-71.

168. What antimicrobials would you use to treat community-acquired MRSA?

When considering community-acquired MRSA as a source of infection, do not use antibiotics such as β-lactams, oxacillin, or cefazolin. Consider clindamycin and trimethoprim-sulfamethoxazole. However, in parts of the United States where community-acquired MRSA with resistance to clindamycin is greater than 10%, use alternative therapies such as vancomycin and linezolid.

Kaplan SL: Evaluation and management of suspected methicillin-resistant *Staphylococcus aureus* skin and soft tissue infections in children. UpToDate, 2014. Available from www.uptodate.com.

169. What is SARS and how can it be diagnosed?

SARS (severe acute respiratory syndrome) is a severe lower respiratory tract infection caused by a coronavirus also called SARS-associated coronavirus (SARS-CoV). It was first reported in Asia in February 2003 and caused a global outbreak that year. Symptoms were often nonspecific, including fever, dyspnea, and extreme hypoxia in adults. Children had a less severe course and outcome. No cases of SARS have been reported since 2004.

Centers for Disease Control and Prevention: Severe acute respiratory syndrome (SARS). Available from www.cdc.gov/sars.

Williams JV: The clinical presentation and outcomes comes of children infected with newly identified respiratory tract viruses. Infect Dis Clin North Am 2005;19:569-584.

170. What is MERS?

MERS (Middle East respiratory syndrome) is caused by a coronavirus and was first reported in Saudi Arabia in 2012. The mortality rate is about 50%. It causes a severe lower respiratory tract infection. To date, all cases have been linked with countries in or near the Arabian Peninsula. Human-to-human transmission has been documented, but sustained transmission throughout a community has not yet occurred.

Centers for Disease Control and Prevention: Middle East respiratory syndrome (MERS). Available from www.cdc.gov/coronavirus/mers/.

171. What do pigs and birds have in common regarding the flu?

The influenza A (H5N1) virus occurs mainly in birds. During late 2003 to 2004, outbreaks occurred among poultry in Asia. In 1997, the first case of spread from bird to human was seen in Hong Kong, with more cases seen with the outbreaks during 2003 and 2004. It is also possible for strains that have passed through an intermediate host, most often the pig, to infect a human. H5N1 virus spread from person to person is rare.

H7N9 is another avian influenza virus. It was first reported in humans in 2013. All reported cases to date have occurred in China, with an approximately 33% mortality rate. Most cases have been linked to contact with ill poultry, but some human-to-human spread has been documented.

Centers for Disease Control and Prevention: Information on avian influenza. Available from www.cdc.gov/flu/avianflu.

Williams JV: The clinical presentation and outcomes of children infected with newly identified respiratory tract viruses. Infect Dis Clin North Am 2005;19:569-584.

172. What are the three most common causes of fever in a returning traveler? Name two other emerging travel-related infections.

Malaria, typhoid fever, and tuberculosis are the classic causes of fever in a returning traveler. Dengue fever and chikungunya fever are mosquito-borne viral infections that have been increasing in incidence over the past few years.

Centers for Disease Control and Prevention: Travelers' health. Available from www.cdc.gov/travel.

173. A 4-year-old boy presents with 2 weeks of fever and abdominal pain beginning 1 week after returning from a summer spent in India visiting family. What is the most likely diagnosis, and what tests should you order in the ED?

Typhoid fever (caused by *Salmonella typhi* or *Salmonella paratyphi*) is most likely. The organism can be isolated from blood cultures (about 60% sensitivity). It is less commonly isolated from stool cultures, unlike strains of *Salmonella* that cause gastroenteritis. A CBC and comprehensive metabolic panel can also aid in making the diagnosis.

174. What are the distinguishing features of malaria?

Malaria is characterized by paroxysms of fever, chills, and rigors occurring at certain time intervals depending on the species. Infection with *Plasmodium falciparum* carries high morbidity and mortality rates (secondary to cerebral malaria, in addition to renal and respiratory failure).

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CHAPTER 39

POISONINGS

James F. Wiley II and Kevin C. Osterhoudt

1. How many poisonings occur in the United States each year? How many exposures involve children younger than 6 years?

Over 2 million calls are made to regional poison control centers annually. Of these, 50% to 60% involve toxic exposures in children younger than 6 years of age. Estimates suggest that 4 million people are poisoned in the United States each year. Not all poisonings are reported to poison control centers.

Bronstein AC, Spyker DA, Cantilena LR, et al: 2011 annual report of the American Association of Poison Control Centers' National Poison Data System. Clin Toxicol 2012;50:911-1164.

2. How often can poisonings be managed at home with the assistance of a regional poison control center?

Of all poison calls to a regional center, 80% to 90% are typically handled safely via phone advice and home observation.

Bronstein AC, Spyker DA, Cantilena LR, et al: 2011 annual report of the American Association of Poison Control Centers' National Poison Data System. Clin Toxicol 2012;50:911-1164.

3. When was the first poison control center established and why?

Dr. Edward Press in Chicago established the first center in 1953 in response to an American Academy of Pediatrics study published in 1952, which found that 50% of all childhood injuries were due to potentially poisonous ingestions.

Press E, Mellins RB: A poisoning control program. Am J Public Health 1954;44:1515-1525.

4. What is the Poison Prevention Packaging Act?

Passed in 1970, this act mandated safety packaging for pharmaceuticals. It followed other important landmark legislation, including the Federal Hazardous Substances Labeling Act of 1960, which promoted product labeling, and the Child Protection Act of 1966, which required appropriate labeling of pesticides and other previously unlabeled hazardous substances.

5. What was the impact of the Poison Prevention Packaging Act?

Frequency of poisoning and death due to poisoning in children decreased significantly in 1969, 1 year prior to the passage of the act. This effect was due to voluntary initiation of safety packaging (childproof caps) by manufacturers in anticipation of the law.

Arena J: The pediatrician's role in the poison control movement and poison prevention. Am J Dis Child 1983;137:870-873.

6. What are the most common poisons to which young children are exposed?

In 2011, the top 10 exposures in young children, from most frequent to least frequent, were cosmetics, analgesics, household cleaning substances, foreign bodies (e.g., coins, button batteries), topical preparations, vitamins, antihistamines, pesticides, antimicrobials, and cough and cold preparations.

Bronstein AC, Spyker DA, Cantilena LR, et al: 2011 annual report of the American Association of Poison Control Centers' National Poison Data System. Clin Toxicol 2012;50:911-1164.

7. Who is more likely to ingest a poison: a 2-year-old boy or 2-year-old girl?

The boy is more likely to ingest a poison.

8. Who is more likely to ingest a poison: a 15-year-old boy or 15-year-old girl?

The girl is more likely to ingest a poison and usually does so intentionally.

9. What is the typical setting for a pediatric poisoning incident?

The child is usually younger than 6 years of age and is at home. The poison is typically readily available to the child. Prescription medicines that children ingest frequently belong to a grandparent or older nonparent relative. In most cases, only one poison is ingested. Social history often discloses a recent stress in the household, such as a recent move, new baby, marital discord, visiting relatives, or distracting event (holiday, wedding).

Henretig FM: Special considerations in the poisoned pediatric patient. Emerg Med Clin North Am 1994;12:549-567.

10. What is the most common cause of death due to poisoning?

Unintentional opioid drug overdose is the leading cause of death due to poisoning in the United States, and poisoning has overtaken motor vehicle collisions as the leading cause of death due to injury overall. Increased prescribing of oxycodone (Oxycontin) and methadone is highly correlated with the marked increase in opioid deaths over the past 10 years, while deaths due to heroin overdose have remained relatively flat. For example, in 2007, deaths due to unintentional ingestion of prescription opioid medications accounted for almost 12,000 deaths versus 2000 deaths caused by heroin overdose.

Okie S: A flood of opioids, a rising tide of deaths. N Engl J Med 2010;363:1981-1985.

11. Which medicinal drugs are potentially life-threatening or fatal to a 10-kg toddler following ingestion of a single dose?

Benzocaine, buprenorphine, calcium channel blockers, camphor, chlorpromazine, chloroquine, clonidine, clozapine, cyclic antidepressants, diphenoxylate/atropine (Lomotil), disopyramide, flecainide, hydroxychloroquine, methadone (and other opioids), methyl salicylate (oil of wintergreen), quinidine, quinine, propranolol, and sulfonylurea oral hypoglycemics.

Braitberg G, Oakley E: Small dose . . . big poison. Aust Fam Physician 2010;39:826-833.

Key Points: Poisoning Epidemiology

1. More than half of all poisoning exposures are reported among children younger than age 6 years.
2. Natural exploratory behaviors of young children put them at risk for poisoning.
3. Drug abuse, experimental risk taking, and depression put adolescents at risk for poisoning.
4. Unintentional overdose and intentional abuse of prescription opioids are the leading causes of accidental death in the United States.

12. What are the most important actions that provide the best chance for recovery after poisoning?

Most poisoned patients will do well with provision of timely supportive care that ensures careful assessment and prompt management of airway, breathing, circulation, and neurologic disability.

13. Which three antidotal drugs should be immediately considered in the resuscitation of the comatose child?

Two are often not thought of as drugs per se, but they are essential substrates for brain function: oxygen and glucose. Administration of the opioid antagonist naloxone may also be warranted.

14. What is a "toxidrome"?

The word *toxidrome* can be thought of as a combination of the words "toxic" and "syndrome." Toxidromes are groupings of physical signs that may help to suggest the drug class responsible for a poisoning. Note these signs in the examination of every potentially poisoned patient: mental status, vital signs, pupil size and reactivity, skin color and moisture, and bowel sounds.

Osterhoudt KC: No sympathy for a boy with obtundation. Pediatr Emerg Care 2004;20:403-406.

15. Describe the most common toxidromes.

The most common toxidromes can be found in Table 39-1.

Table 39-1. Common Toxidromes

VARIABLE	SYMPATHOMIMETICS	ANTICHOLINERGICS	ORGANOPHOSPHATES	OPIATES/ CLONIDINE	BARBITURATES/ SEDATIVE-HYPNOTICS	SALICYLATES
Mental status	A, D, P, S	C, D, P, S	C, D, F, S	C	C	C, S
Heart rate	↑	↑	↓ (↑)	↑	—	— (↑)
Blood pressure	↑	↑	— (↑)	↓	↓	—
Temperature	↑	↑	—	↓	↓	↑
Respirations	—	—	↑	↓	↓	↑
Pupil size	↑ (reactive)	↑ (sluggish)	↓	↓↓	—	—
Bowel sounds	—	↓	↑	↓	—	—
Skin	Sweaty	Flushed/dry	Sweaty	—	Bullae (IV use)	Sweaty

A, agitation; C, somnolence/coma; D, delirium; F, fasciculations; IV, intravenous; P, psychosis; S, seizure.

16. How can the anticholinergic toxidrome be differentiated from the sympathomimetic toxidrome?

Anticholinergic agents and sympathomimetic agents may both produce altered mental status, tachycardia, hypertension, hyperthermia, and mydriatic pupils, so distinguishing between these drugs is not easy. Look to the skin, the bowels, and the eyes for help. Anticholinergic poisoning is likely to produce flushed skin that is surprisingly dry to the touch, diminished or absent bowel sounds, and less reactive pupils. Many people remember the characteristics of the anticholinergic toxidrome with the mnemonic "mad as a hatter, blind as a bat (pupils do not accommodate well), red as a beet, dry as a bone, and hot as Hades."

17. What would be the significance of a bitter almond smell to your morning coffee?

Consider the possibility of cyanide in the coffee.

18. What other toxicants are associated with characteristic odors?

- Acetone, isopropyl alcohol, and phenol have the odor of acetone.
- Cyanide has the smell of almonds.
- Arsenic, thallium, organophosphates, and selenious acid have the odor of garlic.
- Chloral hydrate and paraldehyde have the scent of pears.
- Hydrogen sulfide has the smell of rotten eggs.
- Methyl salicylate has the aroma of wintergreen.

19. What is the gastrointestinal decontamination technique of choice for most poisonings?

Administration of activated charcoal is the primary method of gastrointestinal decontamination for ingested poisons.

Osterhoudt KC, Durbin D, Alpern ER, et al: Activated charcoal administration in a pediatric emergency department. Pediatr Emerg Care 2004;20:493-498.

20. What is activated charcoal?

Most would recognize charcoal as the byproduct of the pyrolysis of wood or other organic materials. "Activation" of charcoal refers to a process of further oxidizing the charcoal, either chemically or at high temperatures, to increase its surface area and adsorptive capacity.

21. When is activated charcoal administration recommended?

Evidence suggests that patients who have ingested a substance known to bind well to activated charcoal and who have an intact airway or have undergone endotracheal intubation should receive activated charcoal if it can be administered within 60 minutes of ingestion (when drug is likely to remain in the stomach). Children who would not otherwise require endotracheal intubation should not be intubated solely for the purpose of receiving activated charcoal. The potential for benefit from activated charcoal administration after 60 minutes cannot be excluded, but volunteer studies indicate that the benefit is likely to be low.

Chyka PA, Seger D, Krenzolok EP, Vale JA: Position paper: Single-dose activated charcoal. Clin Toxicol 2005;43:61-87.

22. List common drugs and toxicants that are poorly adsorbed by activated charcoal.

- Ineffective: Alcohols, iron, lithium
- Poorly effective: Hydrocarbons, metals

23. What is whole-bowel irrigation?

Whole-bowel irrigation uses nonabsorbable polyethylene glycol solution (well known as a surgical bowel preparation) to wash intestinal contents through before absorption can take place. Typical administration is 500 mL/hour in a toddler and up to 2 L/hour in an adolescent, continued until the rectal effluent is clear and clinical signs of continued drug absorption have subsided.

24. List the possible indications for whole-bowel irrigation.

Although not yet proven to improve patient outcomes, use of whole-bowel irrigation has been advocated for the following situations:

- Body packers/stuffers
- Ingestion of metals, lithium
- Ingestion of sustained-release preparations
- Ingestion of pharmaceutical patches
- Massive overdoses
- Concretions of pills

25. What is a body packer or a body stuffer?

Body packers are smugglers who attempt to evade customs officials by swallowing packages (typically tied condoms) of drugs. Body stuffers are drug sellers or users who hurriedly ingest illicit substances to hide evidence from authorities. Because of the planning involved, packers are less likely to become toxic than stuffers; however, packers typically ingest more dangerous quantities of the drugs.

Traub SJ, Hoffman RS, Nelson LS: Body packing: The internal concealment of illicit drugs. N Engl J Med 2003;349:2519-2526.

26. How often do poisoned patients require special therapy, such as antidote administration, elimination enhancement, or extracorporeal elimination?

Antidotes and elimination enhancement, such as urinary alkalinization or multiple-dose activated charcoal, are only performed in about 1% of all reported poisonings. Extracorporeal elimination (hemodialysis or hemoperfusion) is needed in less than 0.1%.

Bronstein AC, Spyker DA, Cantilena LR, et al: 2011 annual report of the American Association of Poison Control Centers' National Poison Data System. Clin Toxicol 2012;50:911-1164.

27. What are the characteristics of the ideal toxic agent that would be amenable to multiple-dose activated charcoal?

The ideal toxicant has a small volume of distribution (<1 L/kg), has low protein binding, binds to activated charcoal, and either has an enterohepatic circulation of an active metabolite in the bowel or undergoes enteroenteric dialysis with evidence of excretion of agent from the microvillous blood circulation into the gut, where it is bound by the charcoal. Multiple-dose activated charcoal may also be of benefit after ingestion of sustained-release compounds or after a toxic gastric concretion has formed.

28. Multiple doses of activated charcoal are believed to enhance clearance of what poison(s)?

Multiple-dose activated charcoal may be helpful to treat life-threatening poisoning caused by carbamazepine, dapsone, phenobarbital, quinine, and theophylline. Elimination of amitriptyline, dextropropoxyphene, digoxin, disopyramide, nadolol, phenylbutazone, phenytoin, piroxicam, and sotalol increases with multiple-dose activated charcoal; however, improved outcomes for ingestion of these poisons with respect to morbidity and mortality rates have not been shown in controlled trials.

American Academy of Clinical Toxicology, European Association of Poisons Centers and Clinical Toxicologists: Position statement and practice guideline on the use of multi-dose activated charcoal in the treatment of acute poisoning. Clin Toxicol 1999;37:731-751.

29. Are lipid emulsions helpful in some poisoned patients?

Lipid emulsions (e.g., intralipids) are intravenous (IV) preparations of fats primarily used to provide total parenteral nutrition. Small animal trials and human case reports indicate potential benefit of lipid emulsions in patients with serious overdoses of bupivacaine, verapamil, beta blockers, some tricyclic antidepressants, and chlorpromazine. The clinician should consult a medical toxicologist or regional poison control center to determine if lipid emulsion administration is appropriate.

Jamaty C, Bailey B, Larocque A, et al: Lipid emulsions in the treatment of acute poisoning: A systematic review of human and animal studies. Clin Toxicol 2010;48:1.

30. What is extracorporeal removal?

Extracorporeal removal refers to elimination of a poison from the blood after removing the blood or a portion of the blood from the body. The forms of extracorporeal elimination include hemodialysis, charcoal hemoperfusion, arteriovenous hemofiltration, venovenous hemofiltration, exchange transfusion, and plasmapheresis. Of these, hemodialysis is used most commonly.

31. What factors predict adequate removal of a poison by hemodialysis?

The substance should have the following characteristics: small volume of distribution, little protein binding, water solubility, low molecular weight, low endogenous clearance, and single-compartment kinetics.

32. For which toxic agents is hemodialysis most commonly considered?

- Ethylene glycol
- Lithium
- Methanol
- Salicylate
- Theophylline

Dargan PI, Jones AL: Acute poisoning: Understanding 90% of cases in a nutshell. Postgrad Med J 2005;81:204-216.

33. When should hemodialysis be used?

The decision to perform hemodialysis is based on physical findings as well as drug levels. Always repeat an elevated level and check units of measurement before instituting hemodialysis.

34. What is the rationale behind urinary alkalinization?

Urinary alkalinization refers to the administration of sodium bicarbonate or sodium acetate to raise the urine pH to 8.0. This procedure converts renally excreted toxicants, which are weak acids, to their ionized form within the proximal renal tubules, and thereby prevents reabsorption in the distal tubules (ion trapping). This technique is most useful for enhancing the excretion of salicylates and phenobarbital (but not other barbiturates). Urinary alkalinization may also be useful to protect the kidney during rhabdomyolysis.

Proudfoot AT, Krenzelok EP, Vale JA: Position paper on urine alkalinization. J Toxicol Clin Toxicol 2004;42:1-26.

35. What are the pitfalls of urinary alkalinization?

Urinary alkalinization is inhibited by the presence of hypokalemia. Urinary alkalinization has been associated with pulmonary and cerebral edema, and fluid administration must be monitored carefully. Electrolyte and acid-base disturbances can result from sodium bicarbonate administration and merit frequent monitoring.

Proudfoot AT, Krenzelok EP, Vale JA: Position paper on urine alkalinization. J Toxicol Clin Toxicol 2004;42:1-26.

36. List some drugs that may lead to a delayed expression of clinical toxicity.

- Acetaminophen
- Monoamine oxidase inhibitors
- Oral hypoglycemic agents
- Sustained-release drug formulations
- Thyroid hormones
- Warfarin

Key Points: Initial Poisoning Management

1. Toxidrome analysis is more clinically useful than toxicology urine analysis.
2. Prompt supportive care of airway, breathing, circulation, and neurologic disability is key to good outcomes after pediatric poisonings.
3. Oral activated charcoal administration is the primary means of gastrointestinal decontamination for children who present for care within 60 minutes of ingestion and have ingested a potentially toxic substance that binds to charcoal.

37. Describe the pathophysiology of acetaminophen poisoning.

Acetaminophen is the drug most commonly administered to children. Toxicity may occur after acute overdose of 150 to 200 mg/kg or after repeated supratherapeutic ingestions. Overdose of acetaminophen saturates typical hepatic metabolism pathways and expends glutathione, resulting in the production of an intermediate metabolite, *N*-acetyl-p-benzoquinone imine via cytochrome oxidase (P-450) metabolism. This metabolite binds to hepatocytes and causes centrilobular liver necrosis.

38. What are the three stages of acetaminophen overdose poisoning?

- **Stage I** (30 minutes to 24 hours after ingestion): Often asymptomatic; occasionally nausea, vomiting, diaphoresis, and pallor

- **Stage II** (24-48 hours after ingestion): Nausea, vomiting, right-upper-quadrant abdominal pain, elevation of hepatic aminotransferase levels
- **Stage III** (72-96 hours after ingestion): Fulminant hepatic failure with jaundice, thrombocytopenia, prolonged prothrombin time, and hepatic encephalopathy. Renal failure and cardiomyopathy may occur. If the patient survives, complete resolution of liver abnormalities is possible.

39. How is acetaminophen intoxication diagnosed?

The potential for acetaminophen poisoning must be thoughtfully considered, because most patients are initially asymptomatic. Measure the serum acetaminophen level in all patients with intentional drug overdose. Frequently, acetaminophen is overlooked as a coingestant with cough/cold preparations and combination analgesics such as Percocet (oxycodone and acetaminophen). An acetaminophen level at 4 to 24 hours after ingestion can predict the potential for toxicity and determine the need for antidote administration based on the Rumack-Matthew nomogram.

40. What is the antidote for acetaminophen overdose? How does it work?

N-acetylcysteine (NAC) replenishes, and substitutes for, depleted glutathione stores in the liver and detoxifies the toxic metabolite. NAC also seems to alleviate existing hepatotoxicity through antioxidant and procirculatory properties.

41. When is NAC given after acute acetaminophen overdose?

All patients with acute overdose whose acetaminophen levels fall within the potential toxicity area on the Rumack-Matthew nomogram merit administration of NAC. This therapy is most effective when initiated within 8 hours of ingestion, though it may provide benefit even if given later.

42. How is NAC administered?

Historically, NAC had been given orally with a loading dose of 140 mg/kg, followed by 17 doses of 70 mg/kg given every 4 hours. However, IV NAC infusion over 21 hours provides the same protection from toxicity in a shorter period of time if initiated within 16 hours of ingestion and avoids the problem of vomiting that is frequently encountered with oral administration. The IV route is also preferred in a pregnant patient to ensure adequate NAC delivery to the fetus. In addition, IV administration is preferrable in patients who present with signs of liver failure because it can augment the potential antioxidant effect and lower mortality even more than 24 hours after overdose.

Rumack BH, Bateman DN: Acetaminophen and acetylcysteine dose and duration: Past, present and future. Clin Toxicol 2012;50:91-98.

43. Does IV NAC have any particular risks?

The IV administration of NAC may cause a life-threatening anaphylactoid reaction, which most commonly occurs during the first, higher-dose infusion. Patients with asthma may be at highest risk.

44. What is a patient-tailored NAC regimen?

Rather than a 2½-day duration of oral NAC therapy or a 21-hour duration of IV NAC therapy, many toxicologists now recommend a duration of therapy tailored to each patient based upon serum acetaminophen concentration, time of presentation, and clinical condition.

Dart RC, Rumack BH: Patient-tailored acetylcysteine administration. Ann Emerg Med 2007;50:280-281.

45. Describe the treatment for acute salicylate poisoning.

Activated charcoal may be considered early after overdose. Restore intravascular volume. Urinary alkalinization is appropriate for patients with symptoms and a peak salicylate level of more than 35 mg/dL (350 mg/L). Mild alkalemia will reduce salicylate access to the brain. Patients with unremitting metabolic acidosis, pulmonary edema, severe renal impairment, coma or seizures, liver impairment, and salicylate level greater than 70 mg/dL meet criteria to receive hemodialysis.

Dargan P, Wallace CI, Jones AL: An evidence based flowchart to guide the management of acute salicylate (aspirin) overdose. Emerg Med J 2002;19:206-209.

46. What hazard is associated with endotracheal intubation of salicylate-poisoned patients?

Salicylate-poisoned patients typically have profound respiratory alkalosis. This process is abruptly reversed by paralytic and sedative medications, and resulting acidemia may increase salicylate entry to the brain and cause seizures.

47. How does iron produce toxicity?

Iron acts as a gastrointestinal mucosal irritant and as an inhibitor of oxidative phosphorylation in the mitochondria. The body has no mode of excretion of excess iron.

48. How much iron is toxic?

Expected toxicity can be estimated by the amount of elemental iron ingested (Table 39-2).

49. How much elemental iron is present in commonly available preparations?

- 65 mg (20% of tablet strength) is present in ferrous sulfate, 325 mg.
- 40 mg (12% of tablet strength) is present in ferrous gluconate, 325 mg.
- 105 mg (32% of tablet strength) is present in ferrous fumarate, 325 mg.
- 44 mg/5 mL is present in Feosol elixir (ferrous sulfate).
- 4 to 18 mg is present in children's chewable vitamins.
- 10 mg/mL is present in infant liquid vitamins.

50. How has the epidemiology of childhood iron poisoning changed in recent years?

From 1988 to 1998, iron poisoning was the leading cause of pediatric overdose death from pharmaceutical products, but this pattern abated with changes in iron formulations and packaging.

Pou-yen T, Rangan C: Iron poisoning: A literature-based review of epidemiology, diagnosis, and management. Pediatr Emerg Care 2011;27:978-985.

Tenenbein M: Unit-dose packaging of iron supplements and reduction of iron poisoning in young children. Arch Pediatr Adolesc Med 2005;159:557-560.

51. What are the clinical manifestations of iron poisoning?

Gastrointestinal irritation leads to vomiting, diarrhea, abdominal pain, melena, and hematemesis. Coma and shock are the hallmarks of severe poisoning. Multiorgan system failure may ensue. Patients recovering from severe iron poisoning may develop gastric scarring with pyloric obstruction 4 to 6 weeks later.

52. What laboratory studies are helpful in the management of iron poisoning?

An abdominal radiograph can confirm iron ingestion and give an impression of the amount of unabsorbed iron. However, a negative radiograph does not rule out iron ingestion, particularly more than 2 hours after ingestion, because dissolved or absorbed iron is *not* radiopaque. A serum iron level 4 to 6 hours after ingestion confirms and helps categorize iron poisoning:

- Serum iron level less than 350 μg/dL is minimal toxicity.
- Serum iron level 350 to 500 μg/dL is mild toxicity.
- Serum iron level greater than 500 μg/dL is serious toxicity.

The presence of metabolic acidosis may be the most telling indicator of cellular dysfunction from iron poisoning.

53. What is the antidote for iron poisoning?

Deferoxamine is an iron chelator. The dose is 5 to 15 mg/kg/hour, up to 6 g/day intravenously. Use of deferoxamine has been associated with pulmonary fibrosis and the acute respiratory

Table 39-2. Expected Toxicity of Elemental Iron

DOSE INGESTED (MG/KG ELEMENTAL IRON)	TOXICITY
20-60	Mild gastrointestinal symptoms
60-100	Moderate toxicity
100-200	Serious toxicity
>200	Possibly lethal

distress syndrome, children given deferoxamine infusion for longer than 24 hours. Stop the infusion for 6 hours per each 24-hour period.

Pou-yen T, Rangan C: Iron poisoning: A literature-based review of epidemiology, diagnosis, and management. Pediatr Emerg Care 2011;27:978-985.

Yatscoff RW, Wayne EA, Tenenbein M: An objective criterion for the cessation of deferoxamine therapy in the acutely iron poisoned patient. J Toxicol Clin Toxicol 1991;29:1-10.

54. List the major toxicities of cyclic antidepressants.

- Sodium channel blockade in the cardiac conduction system (dysrhythmia)
- Seizures
- α_1-Adrenergic blockade (hypotension)
- Inhibition of norepinephrine reuptake
- Anticholinergic toxicity

55. What electrocardiographic findings correlate with potential toxicity from cyclic antidepressants?

- QRS complex longer than 0.1 second
- Corrected QT interval prolonged for age
- R wave height greater than 3 mm in lead aVR

Boehnert MT, Lovejoy FH: Value of the QRS duration versus the serum drug level in predicting seizures and ventricular arrhythmias after an acute overdose of tricyclic antidepressants. N Engl J Med 1985;313:474-479.

Liebelt E, Ulrich A, Francis PD, Woolf A: Serial electrocardiogram changes in acute tricyclic antidepressant overdoses. Crit Care Med 1997;25:1721-1726.

Neimann JT, Bessen HA, Rothstein RJ, Laks MM: Electrocardiographic criteria for tricyclic antidepressant cardiotoxicity. Am J Cardiol 1986;57:1154-1159.

56. Why is sodium bicarbonate helpful for drug-induced ventricular dysrhythmias associated with a prolonged QRS interval?

Many agents that cause ventricular tachycardia in overdose block fast sodium channels, which are essential to proper myocardial conduction. Sodium bicarbonate competitively inhibits this blockade by increasing available serum sodium. In addition, the modest serum alkalinization appears to promote, in a synergistic fashion, improved conduction that clinically appears as narrowing of the QRS interval and cessation of ventricular tachycardia.

Liebelt E: Targeted management strategies for cardiovascular toxicity from tricyclic antidepressant overdose: The pivotal role for alkalinization and sodium loading. Pediatr Emerg Care 1998;14:293-298.

57. What are some special considerations for drug-induced bradydysrhythmias?

Bradydysrhythmias commonly follow ingestion of beta blockers, calcium channel blockers, and digitalis-containing compounds. These rhythm disturbances may not be amenable to standard therapy with atropine, epinephrine, and pacing.

58. Name antidotes that may be useful for specific causes of drug-induced bradydysrhythmias.

- Glucagon, lipid emulsions: used for beta blocker overdose
- Calcium, insulin/glucose, lipid emulsions: used for calcium channel blocker overdose
- Digoxin-specific Fab fragments: used for digoxin overdose

Jamaty C, Bailey B, Larocque A, et al: Lipid emulsions in the treatment of acute poisoning: A systematic review of human and animal studies. Clin Toxicol 2010;48:1.

Kenny J: Treating overdose with calcium channel blockers. BMJ 1994;308:992-993.

Weinstein RS: Recognition and management of poisoning with beta-adrenergic blocking agents. Ann Emerg Med 1984;13:1123-1131.

Woolf AD, Wenger T, Smith TW, et al: The use of digoxin-specific Fab fragments for severe digitalis intoxication in children. N Engl J Med 1992;326:1739-1744.

59. What is flumazenil?

Flumazenil is a specific benzodiazepine receptor antagonist that reverses benzodiazepine-induced central nervous system depression.

60. List problems associated with flumazenil administration after drug overdose.

- May precipitate benzodiazepine withdrawal
- May precipitate seizures and their complications
- Has short duration of action compared with duration of benzodiazepine toxicity

Perry HE, Shannon MW: Diagnosis and management of opioid- and benzodiazepine-induced comatose overdose in children. Curr Opin Pediatr 1996;8:243-247.

61. How are poison-induced seizures treated?

Benzodiazepines and barbiturates are the treatments of choice. Seizures may recur and be difficult to treat. The cardiovascular effects of IV phenytoin may complicate cyclic antidepressant overdose. Pyridoxine is a specific antidote for seizures caused by isoniazid overdose. Drug-induced status epilepticus requires advanced modalities, such as pentobarbital coma or general anesthesia, more frequently than does status epilepticus complicating other types of seizure disorders.

Wills B, Erickson T: Drug- and toxin-associated seizures. Med Clin North Am 2005;89:1297-1321.

Key Points: Pharmaceuticals

1. Dangerous acetaminophen poisoning may be asymptomatic initially but can be predicted with serum levels.
2. Prevention of acidemia, maintenance of intravascular volume, and urinary alkalinization are key goals in the treatment of moderate aspirin poisoning.
3. Prevention strategies have reduced the pediatric mortality rate from ingestion of iron products.
4. Sodium bicarbonate therapy may be beneficial for toxic cardiac syndromes manifested by wide QRS tachycardia.

62. What signs and symptoms are expected after isolated benzodiazepine overdose in children?

Benzodiazepines usually do not cause severe symptoms when ingested alone. They typically cause sedation, ataxia, and, in rare cases, respiratory depression. Ataxia without lethargy may occur in up to 30% of children after benzodiazepine ingestion. Apnea, deep coma, or cardiovascular instability suggests coingestion of another agent (e.g., ethanol, barbiturates, other sedative-hypnotics). Benzodiazepines have frequently been implicated in child abuse by poisoning. Typically, the situation involves an elderly caretaker sedating a normally active toddler.

Wiley C, Wiley JF: Pediatric benzodiazepine ingestion resulting in hospitalization. J Toxicol Clin Toxicol 1998;36:227-231.

63. How do opioids cause toxicity?

Opioids bind specific opioid receptors in the brain (μ, κ, σ, and δ), causing global depression of the central and autonomic nervous systems. Meperidine (Demerol) and propoxyphene with acetaminophen (Darvocet) also have active metabolites that can cause seizures and cardiac dysrhythmias.

64. What is the mnemonic for the opioid toxidrome?

FAME: flaccid coma, **a**pnea, **m**iosis, and **e**xtraocular paralysis.

65. Which opioids may require adjusted doses of naloxone to reverse toxicity?

Doses up to 10 mg of naloxone may be required to completely reverse the following opioids: methadone, LAAM (levo-α-acetyl-methadol), dextromethorphan, pentazocine, and fentanyl and its derivatives. Also, anyone habituated to opioids that primarily bind μ receptors and are shorter acting (e.g., heroin, morphine) may need only small doses of naloxone to reverse respiratory depression. A dose of 1 to 2 mg of naloxone may induce opioid withdrawal in these patients.

66. What common pediatric drug toxicity closely mimics opioid intoxication but does not reliably respond to naloxone?

The α_2-adrenergic agonist clonidine acts centrally to reduce sympathetic outflow, and its toxidrome may mimic opioid poisoning. Naloxone reverses coma and respiratory depression in

10% to 15% or less of severely poisoned patients and cannot be considered a consistent antidote for clonidine poisoning. A trial of naloxone is reasonable in severely poisoned children. Yohimbine has been proposed as a specific α_2 antagonist but is available only in oral form and may cause clonidine withdrawal in patients receiving clonidine therapeutically.

Osterhoudt KC: Clonidine and related imidazoline poisoning. UpToDate, 2013. Available at www.uptodate.com. Accessed Sept. 12, 2013.

67. Describe the typical findings after clonidine ingestion.

As little as 0.1 mg (1 tablet) of clonidine has been associated with miosis, coma, apnea, bradycardia, and hypotension. Transient hypertension may also occur. More rare findings include modest hypothermia and pallor.

68. Are there any other distinguishing features between clonidine poisoning and opioid poisoning?

Children with clonidine poisoning often have transient arousal and improvement in respiratory and hemodynamic instability with stimulation. Patients comatose from opioid poisoning are usually not arousable despite painful stimulation.

69. Hallucinations or psychosis may be prominent with which drugs of abuse?

- Anticholinergics (antihistamines, Jimsonweed)
- Dissociatives (phencyclidine, ketamine, dextromethorphan)
- Hallucinogens (lysergic acid diethylamide, psilocybin, mescaline)
- Sympathomimetics (amphetamines, cocaine, 3,4-methylenedioxy-N-methylamphetamine [MDMA–ecstasy], synthetic canthinones [bath salts], synthetic cannabinoids [Spice, K2])
- Withdrawal from ethanol or sedative-hypnotics

70. What are the best agents for sedating patients with drug-induced agitation?

Benzodiazepines are preferred. In addition to providing sedation, they also have anticonvulsant properties. Intramuscular or IV midazolam (0.05 to 0.1 mg/kg, maximum initial dose 5 mg) or intramuscular or IV lorazepam (0.1 mg/kg, maximum initial dose 2 to 4 mg, depending upon degree of agitation) is often used. If intramuscular sedation is required, midazolam may be preferred because of its more rapid onset of action when compared to intramuscular lorazepam. Repeated doses may be given every few minutes until adequate sedation is achieved. In some patients with severe agitation caused by sympathomimetics or hallucinogens, the total dose necessary to achieve adequate sedation may exceed 100 mg.

Greene SL, Kerr F, Braitberg G: Review article: Amphetamines and related drugs of abuse. Emerg Med Australas 2008;20:391-402.

71. Why should neuroleptics, such as haloperidol, be used cautiously in the treatment of drug-induced psychoses?

These agents may lower the seizure threshold of the overdose patient, may add to the cardiovascular toxicity of some drugs, and may limit the patient's ability to dissipate heat.

72. What adverse effects are associated with cocaine intoxication?

Cocaine abusers seek a pleasurable "rush" of euphoria and increased energy. However, intoxicating doses may produce agitation, tachycardia, hypertension, hyperthermia, mydriasis, and tremor. Altered judgment may lead to an increased incidence of accidents and interpersonal violence, and abusers may neglect societal obligations while engaging in high-risk behaviors. Seizures, intracranial hemorrhage, myocardial ischemia, rhabdomyolysis, pneumothorax, psychosis, and death may occur from cocaine abuse.

73. What is the treatment for the tachycardia, hypertension, and hyperpyrexia associated with cocaine overdose?

High doses of benzodiazepines are the most effective and safest pharmacotherapy for cocaine intoxication. Extreme hyperthermia may also warrant aggressive environmental cooling methods. Treat rhabdomyolysis with mechanical ventilation, paralysis, and bicarbonate infusion to reduce the chance of renal failure. Reserve sympatholytics for the most extreme cases. β-Adrenergic antagonist therapy may lead to detrimental unopposed α-adrenergic toxicity. Persistent hypertensive crisis may respond well to nitroprusside infusion or phentolamine.

Zimmerman JL: Cocaine intoxication. Crit Care Med 2012;28:517-526.

74. Name seven hyperthermic syndromes in toxicology.

1. Sympathomimetic poisoning
2. Anticholinergic poisoning
3. Neuroleptic malignant syndrome
4. Malignant hyperthermia
5. Serotonin syndrome
6. Uncoupling of oxidative phosphorylation (e.g., salicylate poisoning)
7. Acute withdrawal syndrome

75. Describe the serotonin syndrome.

The serotonin syndrome is characterized by autonomic hyperactivity, increased neuromuscular tone (especially prominent in the lower extremities), hyperreflexia, and central nervous system depression after exposure to a drug or drugs with proserotonergic properties.

Boyer EW, Shannon M: The serotonin syndrome. N Engl J Med 2005;352:1112-1120.

76. Name some of the methods by which inhalants are abused.

- *Sniffing* describes direct inhalation of vapors from an open container.
- *Huffing* implies inhaling from a cloth soaked with the volatile substance.
- *Bagging* involves holding a volatile-containing bag over the nose.

77. Describe the most widely proposed mechanism for "sudden sniffing death."

Inhaled hydrocarbons are believed to potentiate the cardiovascular effects of catecholamines. Ventricular dysrhythmia may occur, especially if an inhalant abuser becomes surprised (perhaps by parents or police) or agitated.

78. What modification to advanced cardiac life support protocol might be beneficial in the treatment of tachydysrhythmia after inhalant abuse?

Theoretical and anecdotal evidence suggests that β-adrenergic antagonists may help reverse these catecholamine-driven cardiac disturbances. Epinephrine may be relatively contraindicated in this scenario.

Albertson TE, Dawson A, de Latorre F, et al: TOX-ACLS: Toxicologic-oriented advanced cardiac life support. Ann Emerg Med 2001;37:S78-S90.

79. What is Spice or K2?

These are just two of the names used by dealers selling synthetic cannabinoids. These substances contain analogs of naturally occurring cannabinoids, including tetrahydrocannabinol (THC), cannabidiol (CBD), and cannabinol (CBN). Unlike naturally occurring cannabinoids, synthetic cannabinoids often are not detected by urine tests for drugs of abuse.

Vardakou I, Pistos C, Spiliopoulou CH: Spice drugs as a new trend: Mode of action, identification and legislation. Toxicol Lett 2010;197:157.

80. How does the toxicity of synthetic cannabinoids differ from that of marijuana?

Reports have described greater neurologic toxicity, including aggressive behavior, paranoia, dystonia, prolonged psychosis, and seizures. Synthetic cannabinoid use has also been associated with acute kidney injury and myocardial infarction.

Centers for Disease Control and Prevention (CDC): Acute kidney injury associated with synthetic cannabinoid use—Multiple states, 2012. MMWR Morb Mortal Wkly Rep 2013;62:93.

Cohen J, Morrison S, Greenberg J, Saidinejad M: Clinical presentation of intoxication due to synthetic cannabinoids. Pediatrics 2012;129:e1064.

Mir A, Obafemi A, Young A, Kane C: Myocardial infarction associated with use of the synthetic cannabinoid K2. Pediatrics 2011;128:e1622.

81. What are "bath salts"?

Bath salts are synthetic cathinones that are related to the naturally occurring substance khat. Bath salt intoxication has many features of amphetamine overdose but is especially notable for causing prolonged agitation, aggressive and violent behavior, hallucinations, paranoia, and seizures.

Prosser JM, Nelson LS: The toxicology of bath salts: A review of synthetic cathinones. J Med Toxicol 2012;8:33.

82. Why have "bath salts" and "plant food" become popular drugs of abuse?

Bath salts and plant food are drugs of abuse—the names are just clever marketing. The Federal Analog Act that restricts drug sale, possession, and use has a loophole that a chemical has to be "for human consumption." By calling new synthetic drugs of abuse by names such as "bath salts," the drug dealers try to limit their legal liability.

Osterhoudt KC, Cook MD: Clean but not sober: A 16-year-old with restlessness. Pediatr Emerg Care 2011;27:892-894.

Key Points: Drugs of Abuse

1. Naloxone dosing in patients overdosed on heroin requires careful consideration of habituation to avoid precipitation of withdrawal.
2. Benzodiazepines are the most appropriate initial treatment for agitation caused by sympathomimetic and hallucinogenic drugs of abuse.
3. Malignant hyperthermia from overdoses of cocaine, amphetamines, or serotonergic drugs requires rapid treatment, including cooling measures, benzodiazepine administration, and mechanical ventilation with paralysis and bicarbonate infusion in the setting of rhabdomyolysis.
4. A wide variety of synthetic drugs of abuse are created and marketed each year.

83. What is the differential diagnosis for an increased anion gap metabolic acidosis?

One can make mountains out of the mnemonic MUDPILES:

- **M:** Methanol, metformin
- **U:** Uremia
- **D:** Diabetic, alcoholic, or starvation ketoacidosis
- **P:** Paraldehyde (and other aldehydes)
- **I:** Iron, isoniazid, inborn errors of metabolism
- **L:** Lactic acidosis
- **E:** Ethylene glycol
- **S:** Salicylates

Louie JP, Peterson J: Pick your poison: Not a basic case. Pediatr Emerg Care 2006;22:461-463.

84. What is the differential diagnosis for lactic acidosis?

Lactic acidosis may be caused by sepsis, shock, seizures, anoxia, ischemia, or trauma. Toxic causes include poisoning from carbon monoxide (CO), cyanide, sodium azide, hydrogen sulfide, ibuprofen, adrenergic agents, isoniazid, and others.

85. How is the osmolar gap calculated?

$$\text{Osmolar gap} = \text{measured osmolality} - \text{calculated osmolarity}$$

$$\text{Calculated osmolarity} = 2\,([\text{sodium}]\ \text{mEq/L}) + ([\text{blood urea nitrogen}]\ \text{mg/dL})/2.8 + ([\text{glucose}]\ \text{mg/dL})/18$$

86. What is a "normal" osmolar gap?

Normal range is −7 to 10 mOsm.

87. List the potential causes of a large osmolar gap.

- Acetone
- Ethanol
- Glycols
- Isopropanol
- Magnesium
- Mannitol
- Methanol
- Renal failure
- Severe ketoacidemia
- Severe lactic acidemia

88. How do toxic alcohols produce an increased osmolar gap?

Once absorbed into the bloodstream, alcohols are osmotically active. This principle explains why people have to urinate so frequently at Happy Hour. It is also the principle behind administering mannitol to head-injured patients.

89. How can the osmolar gap be used to estimate the levels of toxic alcohols?

This calculation is based upon the molecular weight of the alcohols:

- **Methanol:** molecular weight = 32 mg/mmol; conversion factor = 3.2
- **Ethanol:** molecular weight = 46 mg/mmol; conversion factor = 4.6
- **Ethylene glycol:** molecular weight = 62 mg/mmol; conversion factor = 6.2
- Alcohol level (mg/dL) = (osmolar gap) × (conversion factor)

90. How does ethanol intoxication lead to life-threatening hypoglycemia in young children?

Ethanol is metabolized by the enzymes alcohol dehydrogenase and acetaldehyde dehydrogenase. This process involves the production of NADH (the reduced form of nicotinamide adenine dinucleotide [NAD]). An increased ratio of NADH to NAD inhibits gluconeogenesis and may lead to hypoglycemia in patients with insufficient stores of glycogen.

Walters D, Betensky M: An unresponsive 3-year-old girl with an unusual whine. Pediatr Emerg Care 2012;28:943-945.

91. How quickly does an inebriated 14-year-old metabolize an ethanol level of 150 mg/dL to zero?

Ethanol is metabolized at a constant rate according to zero order kinetics. The typical decrease is 15 mg/dL/hour in the noninduced (nonalcoholic) patient. Therefore, the level should be zero in 10 hours.

Gershman H, Steper J: Rate of clearance of ethanol from the blood of intoxicated patients in the emergency department. J Emerg Med 1991;9:307-311.

92. How do some toxic alcohols lead to metabolic acidosis?

Early after methanol or ethylene glycol ingestion, there will be increased serum osmolality but little acidosis. The body subsequently metabolizes methanol to formic acid, and ethylene glycol to glycolic and oxalic acids. The osmolar gap may drop, while the anion gap increases. Isopropyl alcohol is somewhat distinctive, as it is metabolized to acetone and typically does not lead to profound acidosis.

93. True or false: Ethylene glycol in the urine fluoresces when examined with a Wood's lamp.

False, but not entirely. The most common source of ethylene glycol exposure is automobile antifreeze. Some manufacturers put fluorescein in the antifreeze solution to allow detection of radiator leaks. Lack of fluorescent urine does not rule out ethylene glycol poisoning.

Casavant MJ, Shah MN, Battels R: Does fluorescent urine indicate antifreeze ingestion by children? Pediatrics 2001;107:113-114.

94. What is the physiologic basis for the treatment of methanol or ethylene glycol poisoning?

Formic acid is toxic to the retina, and glycolic and oxalic acids are toxic to the kidneys. The treatment goal is to prevent metabolism to these toxic metabolites. Alcohol dehydrogenase is the enzyme responsible for this biotransformation, and inhibition of this enzyme will be protective.

95. What agents can be used to inhibit alcohol dehydrogenase in the setting of methanol or ethylene glycol poisoning?

- Fomepizole
- Ethanol

Barceloux DG, Krenzelok EP, Olson K, et al: American Academy of Clinical Toxicology practice guidelines on the treatment of ethylene glycol poisoning. J Toxicol Clin Toxicol 1999;37:537-560.

96. Do any vitamins or nutritional supplements have a role in the treatment of methanol or ethylene glycol poisoning?

Folic acid may promote the metabolic degradation of formic acid. Pyridoxine and thiamine may speed transformation of glycolic acids to nontoxic metabolites.

97. When should an ingested disc or button battery be emergently removed?

Disc batteries (button batteries) may contain potentially toxic components, such as mercury or lithium, but are most dangerous as mucosal corrosives. Remove disc batteries lodged in the nares, ear canal, or esophagus immediately, within 2 to 4 hours if possible. Once in the stomach they will almost always pass uneventfully.

Litovitz T, Whitaker N, Clark L, et al: Emerging battery-ingestion hazard: Clinical implications. Pediatrics 2010;125:1168.

98. List some of the dangerous caustic agents frequently found in U.S. households.

Many cleaning products cause only mild irritation. Important exceptions include dishwashing detergent and laundry gel packets, which have caused serious caustic injuries. Hair-relaxing products typically have basic pH. Oven cleaners, toilet bowl cleaners, and drain products often contain hydrochloric acid or sodium hydroxide.

Fraser L, Wynne D, Clement WA, et al: Liquid detergent capsule ingestion in children: An increasing trend. Arch Dis Child 2012;97:1007.

Leape LL, Ashcraft KW, Scarpelli DG, Holder TM: Hazard to health—Liquid lye. N Engl J Med 1971;284:578.

99. What characteristics of a caustic agent are most predictive of injury?

- pH
- Concentration
- Volume ingested
- Viscosity of product
- Manner of exposure
- Duration of exposure

Therefore, thick hair-relaxer creams licked by an explorative child rarely lead to severe esophageal injury, but suicidal ingestions of liquid acids can be expected to lead to significant morbidity.

Friedman EM, Lovejoy FH Jr: The emergency management of caustic ingestions. Emerg Med Clin North Am 1984;2:77-86.

100. What are the complications of ingestion of caustic agents?

Acute complications:

- Upper airway obstruction
- Aspiration pneumonitis
- Gastrointestinal bleeding or perforation
- Systemic acidosis or disseminated intravascular coagulation
- Sepsis

Chronic complications:

- Esophageal stricture
- Impaired gastric function/pyloric obstruction

101. Which patients should be examined endoscopically after caustic ingestion?

- Ingestion involving a concentrated, strong acid or base
- Suicidal ingestions
- Large-volume ingestions
- Patients with vomiting or two or more signs or symptoms of injury

Note: The absence of oral burns is not a sensitive sign for excluding esophageal injury.

102. Give the advantages and disadvantages of using corticosteroids to palliate caustic esophageal burns.

Advantages:

- Anti-inflammatory
- May decrease tissue scarring and stricture

Disadvantages:
- May increase perforation risk
- May increase infection risk
- May mask signs of infection or perforation

103. When should steroids be used to palliate caustic esophageal burns?

Esophageal burns should be graded at endoscopy. First-degree burns do not lead to stricture formation, so steroids are not warranted. Third-degree burns will probably lead to stricture regardless of treatment, and the benefit is not likely worth the risk. Controversy surrounds the value of using steroids together with antibiotics to treat circumferential second-degree burns of the esophagus. Many clinicians believe that corticosteroids may modestly reduce stricture formation in this situation. However, the preponderance of evidence suggests no benefit for the use of corticosteroids for second-degree esophageal burns. Note that a less controversial indication for steroid therapy is the palliation of glottic edema and airway obstruction.

Fulton JA, Hoffman RS: Steroids in second degree caustic burns of the esophagus: A systematic pooled analysis of fifty years of human data: 1956-2006. Clin Toxicol 2007;45:402.

104. What are the indications for surgical exploration after caustic ingestion?

- Evidence of perforation
- Abdominal tenderness after acid ingestion
- Inability to evaluate injuries endoscopically
- Significant central nervous system depression
- Progressive metabolic acidosis
- Hypotension with tachycardia

105. What is the major long-term concern for patients who have recovered from caustic burns of the esophagus?

These patients are at a 1000-fold increased risk for esophageal carcinoma. The mean latency period is over 40 years.

Appelqvist SM: Lye corrosion carcinoma of the esophagus: A review of 63 cases. Cancer 1980;45:2655-2658.

106. Which characteristics of certain hydrocarbon products make them prone to aspiration?

- Low viscosity
- Low surface tension
- High volatility

107. Describe the time course of aspiration injury after hydrocarbon ingestion.

Children may cough, gag, or sputter at the time of ingestion. A gradual evolution of radiographic findings occurs, but most children who will develop pneumonitis will have visible abnormality by 6 hours. More than 98% of children who will develop clinical pneumonitis will do so by 24 hours.

Jolliff HA, Fletcher E, Roberts KJ, et al: Pediatric hydrocarbon-related injuries in the United States: 2000-2009. Pediatrics 2013;131:1139.

108. Do any hydrocarbons have serious systemic toxicity?

Central nervous system depression, seizures, hepatotoxicity, nephrotoxicity, and bone marrow toxicity are among the injuries that can occur after specific hydrocarbon ingestions. The hydrocarbons most noted for systemic toxicity can be remembered with this CHAMPion of mnemonics:
- **C:** Camphor
- **H:** Halogenated hydrocarbons (e.g., carbon tetrachloride, trichloroethane)
- **A:** Aromatic hydrocarbons (e.g., benzene, toluene)
- **M:** Metal-containing hydrocarbons
- **P:** Pesticide-containing hydrocarbons

109. List the clinical manifestations of organophosphate or carbamate insecticide poisoning.

These insecticides are examples of "cholinergic" poisoning.

1. **Muscarinic effects** (pre- and postganglionic parasympathomimetic)
   - **SLUDGE: s**alivation, **l**acrimation, **u**rination, **d**efecation, **g**astric cramping, **e**mesis
   - Bronchorrhea, bronchoconstriction
   - Bradycardia
   - Miotic pupils
2. **Nicotinic effect** (preganglionic sympathomimetic)
   - Tachycardia (often prominent early after exposure in children)
3. **Nicotinic effects** (neuromuscular end plate)
   - Muscle fasciculations
   - Muscle weakness
4. **Central nervous system effects**
   - Depressed consciousness
   - Seizures

110. What antidotes are used to treat organophosphate poisoning?

Atropine may be needed in large doses to reverse muscarinic toxicity. Pralidoxime regenerates active acetylcholinesterase, the enzymatic site of action of organophosphate insecticides.

111. What is the appropriate response to an exploratory anticoagulant rodenticide ingestion by a toddler?

Suicidal ingestions of anticoagulant rodenticides have led to catastrophic consequences. Single exploratory ingestions by curious children have not been reported to produce dangerous bleeding diatheses. Some poison centers recommend decontamination with activated charcoal at the time of the event and evaluation of a prothrombin time 2 to 3 days afterward. Other poison centers recommend no treatment other than expectant observation at home.

112. How should coagulopathy from the warfarin-like rodenticides be treated?

These rodenticides inhibit hepatic production of the vitamin K–dependent coagulation factors II, VII, IX, and X. Acute bleeding can be palliated with transfusion of fresh frozen plasma. Administration of vitamin K_1 will restore production of clotting factors with a lag time of approximately 6 hours. Prophylactic use of vitamin K_1 often complicates evaluation of the anticoagulant-poisoned patient. As the "superwarfarins" have a very long duration of effect, any patient treated with vitamin K_1 warrants monitoring for at least 5 days after the last dose.

113. Describe the typical symptoms of CO poisoning.

Typical symptoms are nonspecific and include malaise, nausea, lightheadedness, and headache, leading many children to be misdiagnosed with a viral illness. More severe poisoning may manifest as confusion, coma, syncope, seizure, or death. Survivors of acute intoxication are at risk of delayed neurologic sequelae, including cognitive deficits, personality changes, and movement disorders.

114. Which member of the family is most likely to suffer the most from equivalent CO exposure?

Infants and small children have higher oxygen consumption and a higher basal metabolic rate than adults. Therefore, they may be most susceptible to the effects of CO. This effect is the basic concept behind the "canary in a coal mine."

115. Describe the pathophysiology of toxicity from CO exposure.

1. CO displaces oxygen from the hemoglobin molecule, leading to decreased oxygen-carrying capacity. CO has an affinity for hemoglobin approximately 250 times that for O_2.
2. Allosteric inhibition of oxygen release to tissues occurs (displacement of the oxyhemoglobin dissociation curve to the left).
3. Cellular oxidative metabolism is reduced through inhibition of cytochrome oxidase enzyme systems.
4. Oxidative injury to the brain endothelium begins a cascade of leukocyte activation, lipid peroxidation, and impaired cerebral metabolism.

Martin JD, Osterhoudt KC, Thom SR: Recognition and management of carbon monoxide intoxication in children. Clin Pediatr Emerg Med 2000;1:244-250.

116. How long does it take the body to eliminate carboxyhemoglobin?

Carboxyhemoglobin elimination depends on inspired oxygen concentration.

- **Room air:** 4 to 6 hours
- **100% O_2:** 40 to 90 minutes
- **Hyperbaric O_2 (3 atm):** 15 to 30 minutes

117. When is hyperbaric oxygen therapy indicated for CO intoxication?

Administer oxygen in the highest concentration to all symptomatic patients until CO levels are below 5% and symptoms subside. Hyperbaric oxygen therapy may prevent oxidative cerebral vasculitis and may prevent delayed neurologic injury. Proposed criteria for considering hyperbaric oxygen include syncope, confusion, central nervous system depression, and very high carboxyhemoglobin levels.

Weaver LK: Carbon monoxide poisoning. N Engl J Med 2009;360:1217-1225.

118. Describe the characteristic brain abnormalities after significant CO poisoning on magnetic resonance imaging of the brain.

Approximately half of patients with neurologic dysfunction will have bilateral lucencies in the basal ganglia. They are most prominent in the globus pallidus, an area of high metabolic demand. Less frequently, subcortical white matter lesions may also be noted.

119. Cyanosis that is unresponsive to supplemental oxygen therapy in the face of a normal partial pressure of oxygen in arterial blood is characteristic of what physiologic abnormality?

Poor response to oxygen therapy makes a pulmonary disorder unlikely. Normal partial pressure of oxygen rules out intracardiac shunting. This finding is the characteristic clinical scenario of an abnormal hemoglobin, most commonly methemoglobinemia.

120. What is methemoglobin?

Hemoglobin is a heme (iron-containing) protein. Methemoglobin exists when the iron moiety is ferric (3^+) rather than ferrous (2^+). Methemoglobin is incapable of transporting oxygen.

121. Provide the differential diagnosis for methemoglobinemia.

Congenital:

- Hemoglobin M
- NADH (reduced form of nicotinamide adenine dinucleotide [NAD])–dependent methemoglobin reductase deficiency
- Cytochrome b5 reductase deficiency

Acquired:

- Transient, illness-associated methemoglobinemia of infancy
- Toxicant-induced

122. Describe the transient, illness-associated methemoglobinemia of infancy.

Young infants have been found to be cyanotic with methemoglobinemia in conjunction with a number of illnesses, including diarrheal dehydration, metabolic acidosis, urinary tract infection, and others. Babies are uniquely susceptible to oxidant stress. Consider this condition in the evaluation of septic-appearing infants.

123. What are some of the more common toxicants associated with the production of excessive methemoglobin?

Many chemicals and pharmaceuticals can produce methemoglobinemia in susceptible people. Among the most commonly noted exposures are benzocaine, dapsone, environmental (i.e., well water) nitrates, nitrites (i.e., amyl nitrite), and phenazopyridine.

124. What is the appropriate therapy for methemoglobinemia?

Provide supplemental oxygen. Eliminate or treat the oxidative stress responsible for methemoglobin formation. Antidotal therapy with methylene blue (1-2 mg/kg of a 1% solution) is indicated in the presence of tissue hypoxia. Methylene blue is unlikely to be effective, and may worsen illness, when administered to patients with severe forms of glucose-6-phosphate dehydrogenase deficiency.

Wright RO, Lewander WJ, Woolf AD: Methemoglobinemia: Etiology, pharmacology, and clinical management. Ann Emerg Med 1999;34:646-656.

Table 39-3. Description of Toxic Mushroom Syndromes

MUSHROOM CLASS	TARGET TOXICITY
Severe Toxicity; Vomiting Typically Begins > 6 Hours After Ingestion	
Gyromitra	Inhibits pyridoxine phosphokinase
Orellanine (*Cortinarius* spp.)	Nephrotoxic
Amanita + *Galerina* + *Lepiota*	Hepatotoxic (amatoxins inhibit RNA polymerase II)
Severe Toxicity; Vomiting Typically Begins < 6 Hours After Ingestion	
Amanita smithiana	Nephrotoxic
Mild Toxicity; Vomiting or Symptoms Typically Begin < 6 Hours After Ingestion	
Psilocybe	"Magic mushrooms" (serotonin)
Ibotenic acid (*Amanita muscaria*)	Ibotenic acid is glutamatergic; muscimol is GABA-like
Coprine ("inky cap")	Disulfiram-like
Emetogenic mushrooms (many types)	Gastric irritants
Muscarinic (*Clitocybe* + *Inocybe* spp.)	Cholinergic
Myotoxic (*Tricholoma* sp.)	Rhabdomyolysis

GABA, γ-aminobutyric acid.

125. What historical feature often serves to differentiate "dangerous" toxic mushrooms from those that are only gastrointestinal irritants?

The time course of vomiting. The delayed onset of vomiting (>6 hours) is an ominous symptom. See Table 39-3 for mushroom types and their toxicities.

126. What are the most common toxic plant poisonings?

Of the 20 most common plant exposures, the following toxic plants are most commonly encountered:

- The Arum family (*Dieffenbachia*, *Philodendron*), which may cause severe pharyngeal edema and possible airway obstruction through the elaboration of calcium oxalate crystals and proteolytic enzymes and direct mucosal irritation
- The *Taxus* species (yew), which cause gastrointestinal irritation, cardiac dysrhythmias, coma, and seizures if large amounts of leaves or seeds are ingested
- The *Solanum* family (Jerusalem cherry, black nightshade, climbing nightshade), which cause gastrointestinal effects, lethargy, or delirium
- *Rhododendron* and *Azalea* spp., which cause gastrointestinal effects, bradycardia, lethargy, and paresthesias through elaboration of grayanotoxins

127. What other plants have potential for severe toxicity? Describe the treatment.

Categories of toxic plants and their potential treatments are listed in Table 39-4.

128. What venomous snakes are indigenous to the United States?

The Crotalinae family includes rattlesnakes, copperheads, and water moccasins. The Elapidae family includes the coral snakes.

129. What are the clinical differences between bites from rattlesnakes and bites from coral snakes?

Rattlesnakes, copperheads, and water moccasins are long-fanged "pit vipers" whose bites produce significant local inflammation and may lead to shock, thrombocytopenia, and coagulopathy. The venom of coral snakes is less irritating locally and more typically produces neuromuscular paralysis. Of note, envenomation from the Mojave rattlesnake may more closely mimic that of coral snakes.

Table 39-4. Categories of Toxic Plants

SYMPTOM CLASS	PLANTS	POTENTIAL TREATMENT
Gastrointestinal irritants	Pokeweed Horse chestnut English ivy	Supportive care
Toxalbumin	Castor bean Rosary pea Autumn crocus (colchicine-containing)	Supportive care for multisystem organ failure
Digitalis-like toxin	Foxglove Oleander Lily of the valley	Digoxin Fab fragments
Other cardiac effects	Mistletoe Monkshood False hellebore Mountain laurel	Supportive care, standard treatment for dysrhythmias
Nicotinic effects	Wild tobacco Tobacco Poison hemlock	Atropine, supportive care for weakness, paralysis
Anticholinergic effects	Jimsonweed Angel's trumpet Matrimony vine Henbane Belladonna	Physostigmine for seizures, malignant hyperthermia Benzodiazepines for delirium
Seizures	Water hemlock	Anticonvulsants
Hallucinations	Morning glory Nutmeg Peyote	Sedation
Cyanogenic	Chokecherry Cherry (pit) Plum (pit) Peach (pit) Apple (seeds) Pear (seeds) Cassava Elderberry (leaves and shoots) Black locust	Cyanide antidote (rarely needed)

130. When should a bystander use his or her mouth to suck the venom out of a snakebite site?

Never! This practice is more likely to infect the wound than it is to reduce toxicity. The Sawyer extractor, a commercial suction device, is also of little value.

Bush S: Snakebite suction devices don't remove venom: They just suck. Ann Emerg Med 2004;43:187-188.

131. What are the most important principles of first aid for a North American snakebite victim?

Remove the victim from the snake and, if possible, wash the wound with soap and water. Keep the victim calm. Activate emergency medical systems to transport the victim to a hospital as soon as possible. Remove any potentially constricting apparel. Immobilize a bitten extremity near the level of the heart. Tourniquets are more likely to increase injury to an extremity and are not routinely advised. Ice is not advised.

132. What are the indications for administration of antivenom to a patient following crotaline envenomation?

Administer antivenom to most patients with progressive local injury, cardiovascular compromise, or coagulopathy.

Gold BS, Barish RA, Dart RC: North American snake evenomation: Diagnosis, treatment, and management. Emerg Med Clin North Am 2004;22:423-443.

133. What antivenom product is available for treatment of North American pit viper envenomation?

Crotalidae polyvalent immune Fab (ovine).

134. Compare the toxic manifestations of bites from black widow spiders (*Latrodectus*) to those of brown recluse spiders (*Loxosceles*).

The venom of black widow spiders leads to increased stimulation of the motor end plate. Restlessness, tachycardia, hypertension, muscular fasciculations, and muscle cramping are common signs and symptoms. In severe cases, black widow bites can mimic a surgical abdomen. In contrast, brown recluse spider venom is predominantly digestive in nature. Local inflammation may progress to tissue necrosis at the bite site. Systemic toxicity is possible but uncommon.

Key Points: Environmental Poisons and Venoms

1. Fomepizole is an antidote that inhibits alcohol dehydrogenase to provide protection in methanol and ethylene glycol poisoning.
2. After CO poisoning, hyperbaric oxygen is most beneficial in its ability to halt an inflammatory cascade in the brain.
3. The low surface tension and viscosity of hydrocarbons create great risk for pulmonary injury after ingestion.

Osterhoudt KC, Burns Ewald M, Shannon M, Henretig FM: Toxicologic emergencies. In Fleisher GR, Ludwig S, Henretig FM, editors: *Textbook of Pediatric Emergency Medicine*, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, pp 1171-1223, 2010.

Osterhoudt KC, Perrone J, De Roos F, et al., editors: *Toxicology Pearls*. Philadelphia, 2004, Elsevier Mosby.

PSYCHIATRIC EMERGENCIES

Ashlee Murray and Philip V. Scribano

CHAPTER 40

1. What constitutes a psychiatric emergency?

Most practically defined, a psychiatric emergency is a potentially preventable or treatable condition that threatens the following:

- The patient's own bodily integrity by suicide, self-mutilation, or drug ingestion
- Someone else's bodily integrity by assault or homicide
- The patient's own psychological and functional integrity (i.e., ability to perceive reality, feel appropriately, make judgments, remember)
- The psychological and functional integrity of the family unit

2. What is the epidemiology of psychiatric illness in children?

- The U.S. Surgeon General estimates that as many as 1 in 10 children and adolescents in the United States suffers a serious psychiatric disturbance per year, whereas the National Institute on Mental Health (NIMH) has found that up to 1 in 5 children either currently or at some point during their life has a serious debilitating mental health disorder.
- In addition, only an estimated 20% of children in the United States with some form of mental health problem severe enough to require treatment are actually identified as such and are receiving mental health services.
- Psychiatric conditions account for 3% to 4% of all pediatric emergency department (ED) visits, which is an increase from approximately 2% in the prior 10 years. Up to 31% to 54% of these encounters result in hospitalization.

Brauner CB, Stephens CB: Estimating the prevalence of early childhood serious emotional/behavioral disorders: Challenges and recommendations. Public Health Rep 2006;121(3):303-310.

Huffman LC, Wang NE, Saynina O, et al: Predictors of hospitalization after an emergency department visit for California youths with psychiatric disorders. Psychiatr Serv 2012;63(9):896-905.

Mahajan P, Alpern ER, Grupp-Phelan, et al: Epidemiology of psychiatric-related visits to emergency departments in a multicenter collaborative research pediatric network. Pediatr Emerg Care 2009;25 (11):715-720.

Merikangas KR, He JP, Burstein M, et al: Lifetime prevalence of mental disorders in U.S. adolescents: Results from the national comorbidity study—adolescent supplement (NCS-A). J Am Acad Child Adolesc Psychiatry 2010;49(10):980-989.

Merikangas KR, He JP, Burstein M, et al: Service utilization for lifetime mental disorders in U.S. adolescents: Results of the national comorbidity survey—adolescent supplement (NCS-A). J Am Acad Child Adolesc Psychiatry 2011;50(1):32-45.

3. What are the most common psychiatric emergencies in children?

Pediatric psychiatric visits to EDs in the United States reveal unspecified neurotic disorders (13%), depressive disorders (13%), and anxiety states (11%) as the three most common principal diagnoses, and behavioral disruptions, ingestions, suicide attempts, and violence as the most common chief complaints.

Santucci K, Sather J, Douglas M: Psychiatry-related visits to the pediatric emergency department: A growing epidemic? Pediatr Res 2000;47(4 Suppl 2):117A [abstract].

Sills MR, Bland SD: Summary statistics for pediatric psychiatric visits to US emergency departments, 1993-1999. Pediatrics 2002;110:e40.

4. What is the leading cause of death due to psychiatric emergencies?

Suicide is the leading cause of adolescent death due to psychiatric illness and is the third leading cause of death behind accidents and homicides overall in older adolescents.

Miniño AM: Mortality among teenagers aged 12-19 years: United States, 1999-2006. NCHS data brief, no 37. Hyattsville, MD, National Center for Health Statistics, 2010.

Key Points: Epidemiology of Psychiatric Emergencies

1. Up to 1 in 10 children and adolescents suffers from a serious emotional disturbance per year in the United States.
2. Psychiatric conditions account for under 4% of all ED visits, and up to 50% of these encounters result in hospitalization.
3. Suicide is the leading cause of death from a psychiatric cause and the third leading cause of death overall in older adolescents.

5. How should the initial approach be conducted when evaluating a child with a possible psychiatric emergency?

- A rapid and thoughtful approach is important.
- Perform an acute medical evaluation: assess ABCs (see Question 6), vital signs, and central nervous system (CNS) function, including pupillary response.
- Assess for possible toxic ingestion: Identify possible toxidromes and obtain history of all possible medications in the home or accessible to the child.
- Obtain history of potential traumatic injury.
- Evaluate past medical history that might indicate an organic cause of the acute psychiatric emergency.
- Consider laboratory evaluation: electrolytes, glucose, osmolality, blood gas, toxicologic screen; electrocardiography; and cranial computed tomography. Obtain a urine pregnancy test in all adolescent females.

6. What are the ABCs of the mental status examination in the ED?

A: Appearance/affect: dress/grooming; abnormal movements; eye contact; facial expression; affect (depressed, blunted, flat, anxious, constricted, hostile, euphoric)
B: Behavior: attitude (cooperative, manipulative, guarded, suspicious, angry, violent, withdrawn)
C: Cognition:

- Thought content—delusions, suicidal or homicidal intent; paranoia; somatic preoccupation; depression, obsessions, fears, phobias; belief of special powers; thought control; depersonalization; feelings of helplessness or hopelessness; guilt
- Thought process—rate, organization, goal directedness, tangential, flight of ideas
- Level of consciousness—orientation, attention, concentration, abstraction

7. List some of the medical considerations that could mimic acute psychosis.

- **Trauma:** Intracranial hemorrhage
- **Drug intoxication:** Ethanol, barbiturates, cocaine, opiates, amphetamines, hallucinogens, marijuana, phencyclidine, anticholinergic medications (antihistamines, tricyclics), heavy metals, corticosteroids, neuroleptic medications, and newer substances like bath salts, synthetic cannabinoids, energy drinks, and piperazine derivatives
- **CNS lesions/infections:** Tumor, hemorrhage, temporal lobe epilepsy, abscess/meningitis/encephalitis, human immunodeficiency virus (HIV)
- **Cerebral hypoxia:** Carbon monoxide poisoning, cardiopulmonary failure
- **Metabolic/endocrinologic:** Hypoglycemia, hypocalcemia, hyperthyroidism or hypothyroidism, adrenal insufficiency, uremia, liver failure, diabetes mellitus, porphyria, Wilson's disease
- **Collagen vascular diseases:** Systemic lupus erythematosus
- **Infectious diseases:** Malaria, typhoid fever, Epstein-Barr virus infection

8. How should the psychiatric evaluation be incorporated into the ED management of a child with a psychiatric emergency?

Once the child has been medically evaluated and reversible, organic causes of the mental disturbance have been ruled out, the psychiatric assessment may proceed. It is most important in this process to maintain confidentiality while also understanding the role of a patient's family and friends, as well as your state's laws regarding mandatory reporting. The appropriate

interview setting is crucial to effectively assess the child's and family's crisis. A private room with monitoring equipment that is out of the patient's reach and is away from the noisy distractions of the main ED setting is optimal. One-to-one supervision is necessary throughout the ED stay. An effort to establish an effective relationship with a specialized mental health team is crucial for engaging the child and family in discussing the issues as well as for developing trust. A predefined health care team should be established and available within a reasonable amount of time in order to minimize ED team changes as well as length of stay in the ED. Avoid confrontation and escalation of the crisis, and attempt to de-escalate the arousal of a potentially violent patient. Use psychiatric emergency services and social services for the risk assessment of the crisis event.

Dolan MA, Fein JA: Pediatric and adolescent mental health emergencies in the emergency medical services system. Pediatrics 2011;127(5):e1356-e1366.

9. What are the indications for the use of physical restraint or seclusion in the ED setting?

- To prevent imminent harm to the patient or other persons when other means of control (e.g., verbal restraint) are not effective or appropriate
- To prevent serious disruption of the treatment plan or significant damage to the physical environment
- To decrease the stimulation a patient receives (i.e., for those with PCP [phencyclidine] or ethanol intoxications)
- When the patient is feeling out of control and requests it

10. Describe the proper procedure in the use of physical restraint.

- Explain to the patient why physical restraint is necessary.
- Provide a physician's order specifying the specific type of restraint, the clinical indication, and an estimate of the duration for restraint. Review this order on an ongoing basis.
- Enlist at least five caretakers, one for each limb and one for the head. Avoid pressure on the patient's throat or chest, and keep hands away from the patient's mouth.
- Closely supervise (1:1) the patient in physical restraints; assess restraints at least every 30 minutes and document findings.
- Provide continuous vital-sign monitoring and frequent evaluation of limb neurovascular status, and assist with nutritional and bathroom needs while the patient is in restraints.
- Avoid placement of the restrained child in the prone position (this could interfere with ventilation).
- Remove restraints only with adequate staff present, and when the patient has regained control (either of his or her own volition or with the use of chemical restraint).

American Academy of Pediatrics, Committee on Pediatric Emergency Medicine: The use of physical restraint interventions for children and adolescents in the acute care setting. Pediatrics 1997;99(3):497-498.

Dolan MA, Fein JA: Pediatric and adolescent mental health emergencies in the emergency medical services system. Pediatrics 2011;127(5):e1356-e1366.

Joint Commission on Accreditation of Healthcare Organizations. Restraint and Seclusion, 2009. Available at http://www.jointcommission.org. Accessed on July 19, 2013.

Key Points: Proper Administration of Physical Restraints

1. Explain to the patient why physical restraint is necessary.
2. Have at least five caretakers to apply restraints.
3. Avoid pressure on the patient's throat or chest.
4. Avoid placement of the restrained child in a prone position.
5. Provide close supervision (1:1) while the patient is in physical restraints.
6. Assess restraints at least every 30 minutes and document assessment.
7. Remove restraints only with adequate staff present, and when the patient has regained control.

11. What are the drugs of choice to restrain a child?

Drugs of choice to restrain a child are listed in Table 40-1.

Table 40-1. Drugs of Choice to Restrain a Child

DRUG	DOSE	COMMENT
Benzodiazepines		
Diazepam (Valium)	0.1 mg/kg PO or IM/IV	Dose range, 2-10 mg
Lorazepam (Ativan)	0.05 mg/kg PO or IM/IV	Dose range, 1-2 mg with titrated dose every 30 min
Midazolam (Versed)	0.25-0.5 mg/kg PO (max 15 mg) 0.035 mg/kg IV (max 2 mg) 0.07-0.08 mg/kg IM (max 5 mg) 0.2-0.4 mg/kg IN (max 10 mg)	Dose range, 2-15 mg; has a significantly shorter time to onset of sedation and a more rapid arousal
Typical Antipsychotics		
High-potency*		
Haloperidol (Haldol)	0.05 mg/kg PO or IM/IV	Dose range, 2-5 mg
Droperidol (Inapsine)†	0.05 mg/kg IM/IV	Dose range, 2.5-5 mg (0.1-0.15 mg/kg max dose) (has shorter half-life [2-4 hr] than haloperidol)
Low-potency		
Chlorpromazine (Thorazine)	0.5-1 mg/kg PO or IM/IV	Usually 200 mg/day
Thioridazine (Mellaril)	0.5-3 mg/kg PO	May cause hypotension
Atypical Antipsychotics		
Risperidone	0.25-0.5 mg ODT	Lower dose for patients 15-29 kg Can use 0.5 mg for >30 kg
Olanzapine	5-10 mg ODT	Onset of action 15 min Lower dose for patients 30-60 kg Can use 10 mg for >60 kg
Ziprasidone	10 mg IM	For use in patients >60 kg
Antihistamines		
Diphenhydramine (Benadryl)	1-2 mg/kg PO or IM/IV	Usually 12.5-75 mg
Combination Therapy		
Haloperidol + lorazepam	Max 5 mg + 2 mg IM	Results in a more rapid onset of sedation with a similar adverse effect profile
Haloperidol + promethazine	Max 5 mg + 0.5-1 mg/kg /dose IM	Promethazine can be given PO/IM/IV/rectally

IM, intramuscularly; IN, intranasally; IV, intravenously; ODT, oral dissolving tablet; PO, per os (taken orally).

Note: For substance-induced psychosis due to hallucinogens (such as PCP), benzodiazepines given at 30- to 60-minute intervals appear to be more effective than the antipsychotics. For substance-induced psychosis due to stimulant intoxication (such as with cocaine or amphetamine), droperidol may be the optimal choice because of its shorter half-life.

*Children have a slightly higher risk of dystonia compared to adults. Some authors use concomitant antihistamine with neuroleptics to decrease the likelihood of dystonia. Although very rare, neuroleptic malignant syndrome can occur with these medications.

†Droperidol has come under increased scrutiny since the U.S. Food and Drug Administration issued a "black box" warning because of concerns of a weak association with QT prolongation and torsades de pointes. The extensive previous use and safety of this agent in the rapid sedation of severely agitated and violent patients leave a quandary regarding the use of this drug.

Data from the following sources: *Battaglia J, Moss S, Rush J, et al: Haloperidol, lorazepam, or both for psychotic agitation? A multicenter, prospective, double-blind, emergency department study. Am J Emerg Med 1997;15(4):335-340. Nobay F, Simon BC, Levitt MA, Dresden GM: A prospective, double-blind, randomized trial of midazolam versus haloperidol versus lorazepam in the chemical restraint of violent and severely agitated patients. Acad Emerg Med 2004;11(7):744-749. Shale JH, Shale CM, Mastin WD: A review of the safety and efficacy of droperidol for the rapid sedation of severely agitated and violent patients. J Clin Psychiatry 2003;64:500-505. Huf G, Coutinho ES, Adams CE: Haloperidol plus promethazine for agitated patients—A systematic review. Rev Bras Psiquiatr 2006;31(3):265-270.*

12. What is neuroleptic malignant syndrome (NMS)? How is it treated?

NMS is an acute, life-threatening reaction to dopamine receptor–blocking agents (typical and atypical antipsychotics) that presents with fever, axial muscular rigidity, autonomic instability/shock, and altered consciousness. An elevated creatine phosphokinase level and leukocytosis are common. Treatment is supportive with intravenous (IV) fluids, antipyretics, and discontinuation of the neuroleptic medication. Bromocriptine and dantrolene are specific medications to treat NMS. The differential diagnosis of hyperthermia includes malignant hyperthermia, anticholinergic poisoning, serotonin syndrome, and adverse reactions to sympathomimetics. The presence of rigidity and elevated creatine phosphokinase levels are distinguishing features of NMS that are rare in serotonin syndrome.

13. What is serotonin syndrome? How is it treated?

Given the high prevalence in use of selective serotonin reuptake inhibitors (SSRIs) as well as increased abuse of amphetamines such as ecstasy, serotonin syndrome has become a common toxicologic emergency. The following clinical features must be present: use of SSRI, agitation, stupor, myoclonus, hyperreflexia, diaphoresis, shivering, tremor, diarrhea, incoordination, and fever. Treatment is supportive, with discontinuation of the offending agent.

14. What is required for hospital admission of a minor with a psychiatric emergency?

For both involuntary and voluntary admissions, most states require parental consent for commitment of a child under the age of 14 years. In children older than age 12 to 16 years, many states require consent of the minor. A legal process is required if the child does not consent to voluntary commitment. Most states allow some period of emergency involuntary admission, but this period varies greatly from state to state. Commonly, involuntary commitment requires physician (and/or designee) documentation of the patient's danger to self or others and can be in effect for several days without a court order (depending on state statutes). In general, there are more inpatient psychiatric units that will accept patients under voluntary commitment than under involuntary commitment.

Fortunati FG, Zonana HV: Legal considerations in the child psychiatric emergency department. Child Adolesc Psychiatr Clin North Am 2003;12:745-761.

15. Identify risk factors for suicide attempts in children and adolescents.

- Male sex
- Age older than 16 years
- Previous suicide attempts
- Substance abuse
- Poor social support
- Mood disorder
- Recent psychosocial stressor
- Family history of mental illness
- History of physical or sexual abuse
- Homosexual orientation
- Drug or alcohol abuse
- Access to firearms or other lethal means

16. What are some medical conditions associated with depression?

- **Neurologic:** Hydrocephalus, migraine, myasthenia gravis, seizure, tumor
- **Endocrine:** Adrenal insufficiency, type 1 diabetes, hyperthyroidism or hypothyroidism, menses-related condition, postpartum status
- **Metabolic:** Hypercalcemia or hypocalcemia, hyponatremia, uremia, porphyria, Wilson's disease
- **Infectious/inflammatory:** HIV infection, influenza, Epstein-Barr virus infection, hepatitis, collagen vascular diseases

Any chronic illness—malignancy, cystic fibrosis, or congenital heart disease, for example—can exacerbate a patient's mood to cause depression.

Table 40-2. Medications Associated with Depression

CATEGORY	SPECIFIC MEDICATIONS
Neuropsychiatric	Neuroleptics, barbiturates, benzodiazepines, carbamazepine, phenytoin, stimulants
Antimicrobial	Ampicillin, griseofulvin, metronidazole, trimethoprim
Anti-inflammatory/ analgesic	Corticosteroids, opiates
Cardiovascular	Clonidine, propranolol
Miscellaneous	Chemotherapy, ethanol, caffeine, oral contraceptives

Adapted from Milner KK, Florence T, Glick RL: Mood and anxiety syndromes in emergency psychiatry. Psychiatr Clin North Am 1999;22:755-777.

Guerrero APS: General medical conditions in child and adolescent patients who present with psychiatric symptoms. Child Adolesc Psychiatr Clin North Am 2003;12:613-628.

17. Name some medications associated with depression?

See Table 40-2 for medications associated with depression.

18. What questions can a clinician ask to help identify patients with depression and suicide risk?

The Patient Health Questionnaire-9 Item has specifically been validated to detect depression among adolescents. A score of 5-9 indicates mild depression, 10-14 moderate depression, and greater than 15 moderately severe to severe depression. A score of 11 or more has a sensitivity of 89.5% and a specificity of 77.5% of detecting depression in adolescents (Table 40-3).

The abbreviated (four-item) Suicide Ideation Questionnaire (SIQ) has high sensitivity (98%) and negative predictive value (97%) and can be used as a screening tool to detect suicidality in children and adolescents. Each of the four items listed in Table 40-4 when positive were individually predictive of suicidality and, therefore, can be effectively used to quickly assess mental health concerns in the busy ED.

Horowitz LM, Wang PS, Koocher GP, et al: Detecting suicide risk in a pediatric emergency department: Development of a brief screening tool. Pediatrics 2001;107:1133-1137.

Richardson LP, McCauley E, Grossman DC, et al: Evaluation of the Patient Health Questionnaire-9 Item for detecting major depression among adolescents. Pediatrics 2010;126(6):1117-1123.

19. What is the role of the pediatric ED in identifying mental illness in children and adolescents?

- Many vulnerable children with undiagnosed psychiatric illnesses seek their care in the ED.
- The Joint Commission recommends a suicide risk assessment for any patient presenting with an emotional or behavioral disorder.
- The U.S. Preventive Services Task Force recommends screening all adolescents for depression if systems are in place to ensure accurate diagnosis and proper intervention.
- It is necessary to have efficient, culturally sensitive, developmentally appropriate, and preferably self-administered screening tools in place.

Dolan MA, Fein JA: Pediatric and adolescent mental health emergencies in the emergency medical services system. Pediatrics 2011;127(5):e1356-e1366.

Table 40-3. Patient Health Questionnaire-9 Item (PHQ-9)—Modified for Teens

Over the last 2 weeks, how often have you been bothered by any of the following problems?				
	Not at all	Several days	More than half the days	Nearly every day
Little interest or pleasure in doing things	0	1	2	3
Feeling down, depressed, or hopeless	0	1	2	3
Trouble falling or staying asleep, or sleeping too much	0	1	2	3
Poor appetite or overeating	0	1	2	3
Feeling bad about yourself—or that you are a failure or have let yourself or your family down	0	1	2	3
Trouble concentrating on things, such as reading the newspaper or watching television	0	1	2	3
Moving or speaking so slowly that other people could have noticed—or the opposite, being so fidgety or restless that you have been moving around a lot more than usual	0	1	2	3
Thoughts that you would be better off dead, or of hurting yourself	0	1	2	3
In the past year have you felt depressed or sad most days, even if you felt okay sometimes?			[ ] Yes	[ ] No
Has there been a time in the past month when you have had serious thoughts about ending your life?			[ ] Yes	[ ] No
Have you ever in your whole life tried to kill yourself or made a suicide attempt?			[ ] Yes	[ ] No
If you are experiencing any of the problems on this form, how difficult have these problems made it for you to do your work, take care of things at home, or get along with other people?	[ ] Not difficult at all	[ ] Somewhat difficult	[ ] Very difficult	[ ] Extremely difficult

Adapted from Guidelines for Adolescent Depression in Primary Care- GLAD-PC Tool Kit, Version 2. 2010, pp 71-73. Available at http://www.glad-pc.org/. Accessed July 18, 2013.

Table 40-4. Suicide Ideation Questionnaire (SIQ)

1. Are you here because you tried to hurt yourself?
2. In the past week, have you been having thoughts about killing yourself?
3. Have you ever tried to hurt yourself in the past other than this time?
4. Has something very stressful happened to you in the past few weeks?

Adapted from Horowitz LM, Wang PS, Koocher GP, et al: Detecting suicide risk in a pediatric emergency department: Development of a brief screening tool. Pediatrics 2001;107:1133–1137.

20. Describe the specific ED management issues in the evaluation of a child with depression or suicidal ideation.

First, assess the potential medical causes of the child's depression, with specific attention to evaluation of toxic ingestions. Maintain suicide precautions with 1:1 observation until the patient has been fully evaluated regarding this risk. Search the patient and his or her personal belongings for possible harmful objects and remove them.

21. List essential criteria for outpatient disposition of the suicidal child/adolescent.

- No requirement of inpatient medical care, including intoxication or delirium
- No prior history of suicide attempt, psychiatric disorder, or substance abuse
- No active suicide ideation
- Presence in home of supportive adult with good relationship with child/adolescent
- Adult agreement to safety plan with close observation of patient until scheduled outpatient follow-up appointment
- Adult agreement to remove or secure all lethal risks (firearms, medications, drugs, alcohol) in home
- Adult and patient provided with indications to return to ED if condition deteriorates
- Follow-up arranged for additional evaluation and treatment
- Both patient and adult agreement with plan and recommendations

Kennedy SP, Baraff LJ, Suddath RL, Asarnow JR: Emergency department management of suicidal adolescents. Ann Emerg Med 2004;43:452-460.

Key Points: Criteria for Outpatient Management of the Patient with Suicidal Ideation

1. No inpatient medical care required
2. No prior history of suicide attempt, psychiatric disorder, or substance abuse
3. No active suicidal ideation
4. Presence in home of supportive adult who agrees with safety plan and agrees to remove or secure all lethal risks
5. Follow-up information provided
6. Agreement of both patient and adult with plan

22. How often do children with aggression and violence present to the ED?

Twenty-five percent of adolescent emergency psychiatric presentations are due to aggression. Aggression or dangerous behavior is often the final common pathway from a variety of causes, such as psychosis, child maltreatment, mood disorders, and behavioral/cognitive deficiencies.

Sills MR, Bland SD: Summary statistics for pediatric psychiatric visits to US emergency departments, 1993-1999. Pediatrics 2002:110:e40.

23. In assessing the child with aggression, what key information is necessary to accurately evaluate the cause of this behavior?

- Onset of the symptoms and their severity
- Use of weapons
- Injury to self or others, including animals
- Indications of intent to harm and lethality
- Presence of other related symptoms
- Presence of precipitants versus impulsive acts
- Response to limit setting
- Destruction of property, including fire setting

24. What approach is suggested to address the child with aggression?

Clinicians must avoid responding to the patient's aggression in a punitive manner. A respectful, nonjudgmental, and reassuring attitude helps the patient gain control and can reduce the aggressive behavior.

25. Is it possible to provide oral medications to an agitated/aggressive child?

Yes. Most patients will cooperate with an oral dosing of a medication, despite the belief that they may be too agitated and uncooperative. Aggressive behavior frequently causes shame and embarrassment to the child. Therefore actions that reflect a respectful, nonjudgmental, and reassuring attitude, such as offering the patient the option of oral medication versus "a shot," can accomplish much in reducing the agitation and additionally avoid any potentially punitive response. Lastly, for some sedatives, oral administration can be as effective as intramuscular dosing to control agitation, and the onset of intramuscular dosing is not sufficiently more rapid to warrant its routine, first-line use.

Yildiz A, Sachs GS, Turgay A: Pharmacological management of agitation in emergency settings. Emerg Med J 2003;20:339-346.

26. What are the three diagnostic criteria for anorexia nervosa?

- Restriction of energy intake relative to requirements, leading to a significantly low body weight, which is defined as a weight that is less than minimally normal
- Fear of weight gain or persistent behavior that interferes with weight gain
- Severe body image disturbance in which body image is the predominant measure of self-worth with denial of the seriousness of the illness

American Psychiatric Association: Diagnostic and Statistical Manual of Mental Disorders, 5th ed. Arlington, VA, American Psychiatric Association, 2013. Available at dsm.psychiatryonline.org. Accessed June 19, 2013.

27. What are some of the medical complications of starvation or persistent purging seen in anorexia nervosa or bulimia?

- Osteopenia
- Cardiac impairment, bradycardia
- Cognitive changes
- Psychological functioning difficulties
- Nausea or bloating
- Amenorrhea
- Hypokalemia, hyponatremia, and metabolic alkalosis in patients who purge by vomiting, laxative abuse, or diuretic abuse
- Dental erosion and enlarged salivary glands in patients with bulimia
- Infertility

Miller KK, Grinspoon SK, Ciampa J, et al: Medical findings in outpatients with anorexia nervosa. Arch Intern Med 2005;165:561-566.

28. What is the initial medical evaluation for a patient with a suspected eating disorder?

- Complete blood count (CBC), electrolytes, blood urea nitrogen (BUN), creatinine, calcium (Ca), magnesium (Mg), phosphate (PO_4), glucose
- Uric acid, cholesterol, triglycerides, liver transaminases
- Thyroid function tests, urine pregnancy test, urinalysis
- Electrocardiogram (ECG), orthostatic vital signs

29. What is a conversion disorder?

Conversion disorder is a stress-related condition in which there is an alteration in physical functioning suggesting a physical disorder, but expressing psychological conflict. It is a diagnosis offered to explain signs and symptoms that do not correspond to a recognizable medical condition. Conversion disorders are not under voluntary control and cannot be explained by any physical disorder. The incidence of conversion disorders in pediatrics is unknown, but they are fairly common, and children often present to the ED with a conversion disorder. Some examples include psychogenic cough, pseudoseizures, syncope, and inability to walk or use an extremity when there is no organic cause. Conversion disorders are found in girls two to three times as often as boys. (Younger girls and boys, less than 10 years old, are affected equally.) Conversion disorders are unrelated to socioeconomic status but are often related to

parental discord, divorce, and sometimes sexual abuse of the child. With these conditions, there is often secondary gain for the child or parents.

30. How can you identify a child with a psychogenic cough (or habit cough)?

A child with a psychogenic cough often begins with an upper respiratory infection. The history usually reveals that many family members had similar symptoms, but they all improved except for the patient, whose cough worsened. A psychogenic cough is typically loud ("honking") and disruptive, and consequently, the child is often forced out of school. Frequently, the family has been to multiple doctors and tried multiple treatments with no response. Despite these symptoms, the child generally sleeps well and has a normal appetite. On physical examination, the child appears well, despite a very loud cough (often heard throughout the ED).

No chest abnormalities are found. The child is usually able to speak in full sentences and may briefly be able to stop coughing on command. Overprotective parents may speak for the child, who is old enough to answer for himself/herself. Despite absence from school and multiple visits to health care providers, 50% of patients have an attitude of "la belle indifference."

Haydour Q, Alahdab F, Farah M, et al: Management and diagnosis of psychogenic cough, habit cough and tic cough: A systematic review. Chest 2014;10:1518.

Velasco-Zuniga R, Benito-Pastor H, Del Villar-Guerra P, et al: Psychogenic cough: A diagnosis of exclusion. Pediatr Emerg Care 2012;28:1218.

31. How should you approach a child with psychogenic cough?

Although it is important to consider organic disorders (reactive airway disease [RAD], foreign body, tuberculosis [TB]), they are highly unlikely with the scenario described earlier. Interview the child and the family and ask about a possible stressful home situation. Assure parents that you are concerned and do not imply the child is malingering, or "faking it." (This disorder is a conversion disorder, related to stress, and the child is not malingering.) Order necessary tests, but avoid repeating the numerous studies that have already been obtained by other providers. When explaining the diagnosis to the family, be confident and convincing. Explain that the child's symptoms are related to stress, similar to a tension headache or abdominal pain. Discontinue unnecessary medications and try to end any secondary gain resulting from the symptoms. Encourage the parents to stop "doctor shopping" and consider psychiatric treatment or counseling.

32. What is a pseudoseizure? How can you distinguish a pseudoseizure from a true epileptic seizure?

A pseudoseizure is a conversion disorder in which the patient has seizure-like activity. These seizure-like movements are not rhythmic, unlike a true seizure. Occasionally the patient with a pseudoseizure may be moaning or even conversing and has purposeful movements and may reach up to grab the examiner. The pseudoseizure may be prolonged, compared to most true seizures, and may continue even if anticonvulsants are administered. Urinary incontinence is rare with pseudoseizures, and the patient is unlikely to suffer an injury during the event. Occasionally a pseudoseizure may be coaxed by examiners at the bedside. Furthermore, a patient with a pseudoseizure rarely has autonomic changes, which is very different from a true seizure. Despite a prolonged pseudoseizure, the postictal period may be very brief.

It is very important to recognize a pseudoseizure and to avoid treatment with anticonvulsants, which could have serious side effects. Consider admitting the child with a pseudoseizure to the hospital for prompt psychiatric evaluation.

Patel H, Dunn DW, Austin JK, et al: Psychogenic nonepileptic seizures (pseudoseizures). Pediatr Rev 2011;32:e66-e72.

Plioplys S, Laux LC: Pediatric psychogenic nonepileptic seizures in the emergency department: Recognition and interventions. Clin Pediatr Emerg Med 2008;9:101-105.

Reilly C, Menlove L, Fenton V, Das KB: Psychogenic nonepileptic seizures in children: A review. Epilepsia 2013;54:1715-1724.

Selbst SM, Clancy R: Pseudoseizures in the pediatric emergency department. Pediatr Emerg Care 1996;12:185-188.

RESPIRATORY EMERGENCIES

Marc H. Gorelick and Sabina B. Singh

CHAPTER 41

1. What is the best clinical indicator of airway obstruction in children with asthma?

There is no single best indicator of obstruction. It is generally true that in younger children, observation is often more revealing than auscultation; it is hard to listen for wheezes in a crying child. Signs of airway obstruction include the following:

- Evidence of work of breathing (retractions, use of accessory muscles, nasal flaring, abdominal breathing)
- Decreased breath sounds
- Tachypnea
- Prolongation of the expiratory phase
- Wheezing

Several validated scores combine multiple clinical indicators into an index to measure asthma severity and include the Pediatric Asthma Severity Score (PASS) and Preschool Respiratory Assessment Measure (PRAM).

Gouin S, Robidas I, Gravel J, et al: Prospective evaluation of two clinical scores for acute asthma in children 18 months to 7 years of age. Acad Emerg Med 2010;17:598-603.

2. Describe the role of measuring peak expiratory flow rate (PEFR) during acute asthma exacerbations in children.

Clinical assessment, although helpful, may underestimate the degree of airway obstruction in a child with acute asthma. The most commonly used pulmonary function test in pediatric acute care is the PEFR, which is the maximal rate of airflow during forced exhalation after a maximal inhalation. It is easily measured using an inexpensive handheld metering device. Children as young as 4 or 5 years old can be taught how to perform PEFR, although it is more difficult to learn in the setting of an acute exacerbation. Advantages of PEFR include ease of performance, low cost, and ability to track changes during therapy. However, PEFR is effort-dependent and cannot be performed by very young children. In addition, PEFR measures function in medium-sized and large airways, and much of the pathologic change in asthma occurs in medium-sized and small airways; thus, even PEFR can underestimate the severity of disease. Normal values depend on the age and height of the child (a table is usually included with the meter), as well as the severity of the underlying lung disease, so results should be expressed as a percentage of predicted value, or of the patient's usual best value, if known. A PEFR of at least 70% of the predicted value indicates *mild* disease, 40% to 70% of the predicted value is considered *moderate*, and less than 40% of the predicted value constitutes *severe* disease. Failure to achieve a PEFR of at least 40% of the predicted value is one indication for hospital admission.

Gorelick MH, Stevens MW, Schultz TR, Scribano PV: Difficulty in obtaining peak expiratory flow measurements in children with acute asthma. Pediatr Emerg Care 2004;20:22-26.

3. How should pulse oximetry be interpreted in acute asthma?

"Normal" values vary considerably between institutions and practitioners. An Sa_{O_2} (saturation of O_2 measured by pulse oximetry) of 95% correlates with a Pa_{O_2} of around 75 mm Hg, and an Sa_{O_2} of 90% is close to a Pa_{O_2} of 60 mm Hg, at the top of the steep portion of the oxygen desaturation curve. Most clinicians set a cutoff point for normal somewhere in between. Remember that although a low saturation may be worrisome, normal oxygen saturation does *not* rule out severe disease.

Keahey L, Bulloch B, Becker AB, et al: Initial oxygen saturation as a predictor of admission in children presenting to the emergency department with acute asthma. Ann Emerg Med 2002;40: 300-307.

4. Why does hypoxemia sometimes increase paradoxically after treatment with inhaled bronchodilators?

Hypoxemia in asthma is due to ventilation-perfusion mismatch. The acute deleterious effects of bronchodilators on oxygenation are believed to be due to their effects on the circulation rather than on the airways. After treatment, particularly when administered with high concentrations of oxygen, there is improvement in local alveolar oxygenation. This, in turn, causes a reversal of hypoxia-induced pulmonary vasoconstriction, with the result that areas previously neither ventilated nor perfused are now perfused but not ventilated, increasing the mismatch.

5. What is Poiseuille's law?

Poiseuille's law is represented by the following equation:

$$\text{Airway resistance (R)} = 8nl/\pi r^4$$

in which n = viscosity coefficient of the gas, l = length of the tube, and r = radius of the tube. Thus, resistance to airflow increases in inverse proportion to the fourth power of the radius of the air passages. The take-home message is that a little narrowing goes a long way toward blocking airflow.

6. What is pulsus paradoxus?

Pulsus paradoxus is an exaggeration of the normal decrease in systolic blood pressure during inspiration. In acute asthma, it has been shown to correlate well with pulmonary function tests, and it can be useful in assessing asthma severity and response to treatment. A pulsus paradoxus of less than 10 is considered normal; 10 to 20 indicates moderate obstruction; and greater than 20 indicates severe obstruction.

To measure pulsus paradoxus, inflate the cuff of a manual sphygmomanometer, then auscultate as pressure is gradually released. Initially, Korotkoff sounds are heard only during expiration and disappear with inspiration. The difference between the pressure at which the sounds are first heard and the pressure at which they cease with expiration is the pulsus paradoxus.

Other conditions that may cause an elevated pulsus paradoxus include severe pneumonia, pericardial effusion, constrictive pericarditis, and conditions affecting myocardial compliance (e.g., amyloidosis, endomyocardial fibroelastosis).

7. A 6-year-old with asthma is treated for acute wheezing in the emergency department (ED) with only partial relief and needs to be admitted to the hospital. Should a chest radiograph be obtained?

Chest radiography is of limited value in the evaluation of the patient with acute asthma. It may be useful in identifying those patients with air leak (i.e., pneumothorax or pneumomediastinum) or a concomitant pneumonia. However, routinely obtaining radiographs in all patients with acute asthma, or even all patients with exacerbation serious enough to require hospital admission, rarely leads to an unexpected finding that changes management. In patients with known asthma, limit chest radiographs to cases in which there is clinical suspicion of a radiographic abnormality, such as persistent rales or asymmetry of breath sounds, high fever, crepitus in the neck, or very poor response to therapy or deterioration after initial therapy.

8. What is the value of radiography in children with a first episode of wheezing?

Routine use of radiography in such children is of relatively low yield. However, some authorities recommend obtaining a radiograph in a child with a first episode of wheezing. A number of conditions other than reactive airways disease may present with wheezing, and they should be ruled out before a diagnosis of asthma is made (Table 41-1). Radiography is probably not necessary in children with clinical bronchiolitis of mild-to-moderate severity, or in children with a history and clinical course highly suggestive of asthma (e.g., family history of asthma, personal history of atopy, good response to inhaled bronchodilator treatment). If the diagnosis of asthma is in doubt, or there is a history suggestive of other disease (e.g., history of choking preceding the wheezing), a chest radiograph is prudent.

Roback MG, Dreitlin DA: Chest radiograph in the evaluation of first time wheezing episodes: Review of current clinical practice and efficacy. Pediatr Emerg Care 1998;14:181-184.

Table 41-1. Causes of Wheezing Other Than Reactive Airways Disease

INFLAMMATORY/ INFECTIOUS	INTRALUMINAL OBSTRUCTION	EXTRALUMINAL OBSTRUCTION
Bronchiolitis	Foreign body	Vascular ring
Aspiration (gastroesophageal reflux, tracheoesophageal fistula)	Tracheomalacia	Mediastinal mass
Bronchopulmonary dysplasia	Congestive heart failure	Cystic malformation of the lung
Cystic fibrosis	α_1-Antitrypsin deficiency Cholinergic poisoning (e.g., organophosphate)	Congenital lobar emphysema

9. What is the role of exhaled nitric oxide (NO) measurement in the assessment of asthma?

NO is generated from the action of NO synthase on L-arginine in the lung. NO synthase activity increases during the inflammatory process, producing increased concentrations of NO in exhaled air. Measurement of exhaled NO levels has been shown to be a sensitive, noninvasive means of measuring the degree of airway inflammation in adult and pediatric patients, and it correlates well with other markers of chronic asthma severity. Available data suggest, however, that exhaled NO levels correlate poorly with acute severity in ED patients.

Kwok MY, Walsh-Kelly C, Gorelick MH: The role of exhaled nitric oxide in evaluation of acute asthma in pediatric emergency department. Acad Emerg Med 2009; 16:21-28.

10. What is the preferred initial treatment for children with acute asthma?

Treatment is tailored to the severity of the exacerbation, but treatment with inhaled bronchodilators is the mainstay of initial therapy for all patients. For those with moderate to severe exacerbation (as determined by clinical status or PEFR), give three to four doses of an inhaled bronchodilator in the first hour of treatment until there is good response; those with mild disease may require less aggressive treatment. In addition, give systemic steroids immediately to severely ill patients, to those who are symptomatic despite appropriate bronchodilator therapy at home, and to all patients who fail to show complete response to one or two inhalation treatments in the ED.

Expert Panel on Management of Asthma: Guidelines for the Diagnosis and Management of Asthma: Clinical Practice Guidelines. Available at http://www.nhlbi.nih.gov/guidelines/asthma/asthgdln.htm.

11. Is nebulization a more effective means of delivering inhaled β-receptor agonists than a metered dose inhaler (MDI)?

No. Several studies and meta-analyses have shown that nebulizer and MDI are clinically equivalent, provided that an appropriate dose is given and that a spacer device is used with the MDI. Standard doses of nebulized β-receptor agonist (0.15 mg/kg per dose, maximum 5 mg) are substantially higher than for MDI. However, the particles produced by the MDI deliver medication far more effectively to the lungs. Current recommendations of ED dosages of albuterol for acute asthma are for 2 to 4 puffs of albuterol for young children, 4 to 6 puffs for older children, and 4 to 8 puffs for adolescents and adults. Spacer devices (e.g., Aerochamber) are available in a variety of sizes and configurations and can be used successfully even in young infants. In one sense the delivery systems are not equivalent: Studies have shown that use of MDIs is less expensive and leads to shorter ED stays.

Cates CJ, Crilly JA, Rowe BH: Holding chambers (spacers) versus nebulisers for beta-agonist treatment of acute asthma. Cochrane Database Syst Rev 2006;(2):CD000052.

12. What are the adverse effects of albuterol?

- Tachycardia; tachyarrhythmias (rare)
- Tremor
- Central nervous system (CNS) stimulation, hyperactivity
- Hypokalemia

Note that even with high-dose therapy, although mild degrees of hypokalemia are common (one third of children in one study), symptomatic hypokalemia requiring treatment is rare. For patients receiving high-dose continuous albuterol for more than 6 to 8 hours, monitor serum potassium.

13. What is levalbuterol?

Racemic (standard) albuterol is a mixture of two isomers. Levalbuterol (Xopenex) is the pure R-isomer, which is responsible for bronchodilation. Although some authors suggest anecdotally that levalbuterol produces fewer side effects than albuterol, most of the clinical evidence supports the notion that equipotent doses of albuterol and levalbuterol have similar bronchodilator efficacy and side effect profiles for acute use. There is some evidence that the S-isomer may increase airway reactivity, suggesting that levalbuterol may be preferred for chronic use.

Wilkinson M, Bulloch B, Garcia-Filion P, Keahey L: Efficacy of racemic albuterol versus levalbuterol used as a continuous nebulization for the treatment of acute asthma exacerbations: A randomized, double-blind, clinical trial. J Asthma 2011;48:188-193.

14. What is the mechanism of action of ipratropium?

Ipratropium bromide binds to cholinergic receptors, located primarily in the medium-sized and large airways, leading to bronchodilation. Because vagally mediated bronchodilation is much less marked than that produced by adrenergic stimulation, ipratropium is a much less potent bronchodilator than albuterol and should not be used alone. However, the concomitant administration of both leads to a synergistic effect. Ipratropium is structurally related to atropine, but it is a quaternary compound and therefore is poorly absorbed. Thus, systemic side effects are minimal.

15. Which patients should receive ipratropium?

Ipratropium bromide binds to cholinergic receptors in the airways; concomitant administration with β-receptor agonists leads to a synergistic bronchodilator effect. Pediatric studies are fairly convincing that addition of ipratropium to albuterol leads to significant improvement in patients with severe asthma exacerbation. The benefit in those with more moderate disease is less clear. However, ipratropium has very little downside; costs and adverse effects are both minimal. Give ipratropium to those patients with severe disease at presentation and those who fail or have failed to respond well to β-receptor agonists alone.

Plotnick L, Ducharme F: Combined inhaled anticholinergics and beta2-agonists for initial treatment of acute asthma in children. Cochrane Database Syst Rev 2000;(3):CD000060.

16. What is the optimal dose of ipratropium?

This dose has not yet been well established. The standard individual dose is 0.5 mg regardless of weight. Although multiple doses have been shown to be better than a single dose, the optimal schedule is not known. Most authorities recommend adding ipratropium to two or three of the first three doses of albuterol given in the ED, then every 2 to 4 hours. There is no evidence that ipratropium has additional benefit after initial ED therapy in hospitalized patients.

17. When should steroids be administered?

The sooner the better. Asthma is an inflammatory disease. Bronchodilators control only the symptoms, not the underlying disease. Therefore, nearly all children with acute asthma should receive systemic corticosteroids, preferably at the start of their ED treatment. That said, there is a trade-off in that too-frequent steroid administration may lead to problems. Patients with particularly mild disease, who require no or only a single treatment in the ED and have complete relief of symptoms with infrequent (no more than three to four times a day) treatments at home, may be managed without steroids.

18. What are the actions of corticosteroids in acute asthma?

Steroids have multiple actions:

- Inhibition of mediator release and synthesis
- Interference with mediator action
- Upregulation of β-adrenergic receptors

The last two help explain the observed onset of clinical effect within 2 hours of administration—during the time course of a typical ED visit. Peak effect of steroids requires interference with cellular synthetic pathways and occurs after 6 to 12 hours.

19. When are intravenous (IV) steroids preferred over the oral route for asthma?

Almost never. There are several pediatric studies, and more from the adult literature, showing oral and IV corticosteroids to be equally effective, even among severely ill patients. Reserve the IV route for those unable to take oral medicine (persistent vomiting, severe respiratory distress) and perhaps those patients ill enough to be admitted to an intensive care unit (ICU).

20. How can steroids be given to the child with asthma who is vomiting but does not require hospital admission?

Although pediatric data are sparse, a short course of high-dose inhaled steroids may be a useful alternative in children with mild exacerbation. Another option is the intramuscular route using dexamethasone. Three studies in children support this option; one used a dose of 0.3 mg/kg, another used 0.6 mg/kg, and the third used 1.7 mg/kg.

Edmonds ML, Milan SJ, Camargo CA Jr, et al: Early use of inhaled corticosteroids in the emergency department treatment of acute asthma. Cochrane Database Syst Rev 2012;(12):CD002308.

21. What are the contraindications to systemic steroids?

- **Absolute contraindications:** Active varicella or herpes infection
- **Relative contraindication:** Exposure to varicella in an unprotected child

Note that concomitant bacterial infection (e.g., otitis media, pneumonia) is *not* a contraindication to the use of steroids.

22. Describe the role of magnesium sulfate in acute asthma.

Magnesium sulfate produces bronchodilation via direct effect on smooth muscle because of calcium antagonism. It may provide added benefit when adrenergic and vagal receptors have been saturated. In children with severe exacerbations, the addition of magnesium sulfate to aggressive β-receptor agonist therapy and systemic steroids leads to greater clinical improvement and improved pulmonary function and Sa_{O_2}. The effectiveness in patients with less severe illness is equivocal.

Rowe BH, Bretzlaff J, Bourdon C, et al: Magnesium sulfate for treating exacerbations of acute asthma in the emergency department. Cochrane Database Syst Rev 2000;(1):CD001490.

23. What is the dose of magnesium sulfate for asthma?

Give 50-75 mg/kg (maximum 2 g) intravenously over 20 to 30 minutes.

24. What is heliox?

Heliox is a mixture of helium and oxygen, usually in a ratio of 60% to 70% He/30% to 40% O_2. Because helium is a smaller molecule than either nitrogen (the major component of air) or oxygen, heliox is less viscous than air. According to Poiseuille's law (Question 5), gas flow is inversely proportional to viscosity; thus, heliox may be better able to diffuse past constricted air passages than air mixtures or pure oxygen. The evidence for its effectiveness in status asthmaticus is mixed. Case series and uncontrolled trials have shown improvement among severely ill patients given heliox. However, controlled clinical trials have failed to show significant benefit in terms of pulmonary function or clinical score. Among the disadvantages are the relatively low oxygen concentration permitted and the need for a tight-fitting mask (which may limit compliance in younger children). It remains an investigational agent but may be considered in patients with severe illness that fails to respond to other treatment.

Rodrigo GJ, Pollack CV, Rodrigo C, Rowe BH: Heliox for non-intubated acute asthma patients. Cochrane Database Syst Rev 2006;(4):CD002884.

25. What are the indications for endotracheal intubation and mechanical ventilation in a patient with asthma?

- Failure of maximal pharmacologic therapy
- Hypoxemia unrelieved by O_2 therapy
- Hypercarbia with rising P_{CO_2}
- Deteriorating mental status
- Respiratory fatigue
- Respiratory arrest

26. Which medications should be used during endotracheal intubation of an asthmatic patient?

Use rapid sequence induction, including sedation and neuromuscular blockade. Ketamine is a good choice as a sedating agent because of its intrinsic bronchodilating properties. However, because it also increases airway secretions, use this in combination with an anticholinergic agent such as atropine or glycopyrrolate. Some medications, including opiates (morphine, meperidine) and muscle relaxants (curare, atracurium), potentiate histamine release and may worsen bronchospasm.

27. What are the two most common complications of mechanical ventilation?

- **Air leak**, including pneumomediastinum and pneumothorax, is common and due to barotrauma.
- **Hypotension** often occurs shortly after endotracheal intubation. It results from a combination of relative hypovolemia in severely ill patients and decreased venous return to the heart owing to positive intrapleural pressure.

Key Points: Treatment of Acute Asthma

1. Provide frequent (every 20 minutes) inhaled albuterol.
2. Provide early oral steroids (with or after first treatment).
3. Add ipratropium bromide to β-receptor agonists in case of incomplete response.
4. Monitor response to therapy with clinical assessment or PEFR.
5. Consider IV magnesium sulfate for severe or refractory cases.

28. What is bronchiolitis?

Bronchiolitis is a viral infection of the upper and lower respiratory tract (medium-sized and small airways). The peak incidence is in the winter. Children younger than 2 years of age are most commonly affected. Respiratory syncytial virus (RSV) accounts for approximately 80% of cases; parainfluenza, human metapneumovirus, adenovirus, influenza virus, and other respiratory viruses are less common causes.

29. Is there a test for RSV? How good is it?

RSV can be identified in nasal secretions by viral culture. Rapid antigen testing is also widely available in many hospital laboratories. The sensitivity of the commercially available tests is generally in the range of 80% to 90%, and specificity is greater than 90%. However, the clinical utility of these tests is limited, because treatment is directed toward the clinical manifestations rather than the etiologic agent. Treatment is generally the same regardless of the results of the test.

Bordley WC, Viswanathan M, King VJ, et al: Diagnosis and testing in bronchiolitis: A systematic review. Arch Pediatr Adolesc Med 2004;158:119-126.

30. What are the clinical characteristics and natural history of bronchiolitis?

Bronchiolitis begins as an upper respiratory infection, with coryza and cough. (In older children and adults, as well as many infants, RSV infection remains confined to the upper respiratory tract.) Over a period of several days, the lower tract becomes involved, with the development of wheezing and rhonchi, as well as signs of respiratory distress. Typically, signs and symptoms peak in severity on the third to fifth days of illness and then begin to wane.

The total duration of illness averages 14 to 15 days, although some infants may have a prolonged course. In one study, 25% of children were still symptomatic at 3 weeks. Central apnea can occur in young infants, usually at the onset of illness, before respiratory signs manifest.

Petruzella FD, Gorelick MH: Duration of illness in infants with bronchiolitis evaluated in the emergency department. Pediatrics 2010;126:e285-e290.

31. What is the role of albuterol in the management of bronchiolitis?

Because wheezing is a clinical hallmark of bronchiolitis, inhaled β-receptor agonists are often administered. However, multiple clinical trials, summarized in two meta-analyses, have shown mixed results. On average, the benefit of β-receptor agonists is small. It appears that a subset of infants responds well, but a larger group responds minimally or not at all. It is not currently possible to predict responsiveness; therefore, a trial of albuterol, either 0.25 to 0.5 mL of a 0.5% solution via nebulizer or two puffs from an MDI with a spacer/mask, is warranted. Those children who respond are candidates for further treatment. It is not necessary to continue treatment in the absence of clinical responsiveness. Oral albuterol has *not* been shown to be efficacious.

Gadomski AM, Brower M: Bronchodilators for bronchiolitis. Cochrane Database Syst Rev 2010;(12): CD001266.

32. A 3-month-old child with bronchiolitis has moderate distress and a pulse oximetry of 93% in room air. She does not respond to inhaled albuterol. Is there any other effective treatment?

Although several studies have found a superior clinical benefit of *inhaled epinephrine* compared to albuterol, more recent literature has shown that epinephrine and albuterol are similarly effective. It is not customary to administer inhaled epinephrine on an outpatient basis; a trial of epinephrine may be considered in those patients with more severe illness who are likely to require hospitalization. The usual dose is 3 mL of a 1:1000 solution via nebulizer. A systematic review of *inhaled hypertonic (3%)* saline suggests that this therapy may reduce severity of symptoms and length of stay in hospitalized infants with bronchiolitis, but it does not appear to have a role in ED management.

Hartling L, Bialy LM, Vandermeer B, et al: Epinephrine for bronchiolitis. Cochrane Database Syst Rev 2011; (6):CD003123.

Zhang L, Mendoza-Sassi RA, Wainwright C, Klassen TP: Nebulized hypertonic saline solution for acute bronchiolitis in infants. Cochrane Database Syst Rev 2008;(4):CD006458.

33. Is there a role for corticosteroids in bronchiolitis?

Most studies to date have shown no benefit of corticosteroids, either systemic or inhaled, in the treatment of bronchiolitis. Steroids may be of benefit in selected infants with wheezing suspected of having intrinsic reactive airways disease, such as those with a prior history of wheezing, or strong family or personal history of atopy and good response to inhaled β-receptor agonists.

Fernandes RM, Bialy LM, Vandermeer B, et al: Glucocorticoids for acute viral bronchiolitis in infants and young children. Cochrane Database Syst Rev 2010;(10):CD004878.

34. What are the best indicators of severe disease in bronchiolitis?

Although a number of factors appear to be associated with the need for hospitalization in infants with bronchiolitis, the ability to predict which children can be safely discharged to home is only moderate. In several multicenter studies, initial pulse oximetry of less than 94% was the best predictor of need for admission. Other risk factors include age (<2 months), tachypnea, greater clinical severity (as indicated by greater work of breathing or higher Respiratory Distress Assessment Index), and inadequate oral intake. Although the need for supplemental oxygen would seem to mandate hospital admission, several studies support the use of home oxygen therapy.

Corneli HC, Zorc JJ, Holubkov R, et al; The Bronchiolitis Study Group for the Pediatric Emergency Care Applied Research Network: Bronchiolitis: Clinical characteristics associated with hospitalization and length of stay. Pediatr Emer Care 2012;28:99-103.

Halstead S, Roosevelt G, Deakyne S, Bajaj L: Discharged on supplemental oxygen from an emergency department in patients with bronchiolitis. Pediatrics 2012;129:e605-e610.
Mansbach JM, Clark S, Christopher NC, et al: Prospective multicenter study of bronchiolitis: Predicting safe discharges from the emergency department. Pediatrics 2008;121:680-688.

35. What is palivizumab?

Palivizumab (Synagis) is a humanized mouse monoclonal antibody against RSV. Palivizumab prophylaxis has been shown to reduce the incidence and severity of RSV bronchiolitis in high-risk patients. It is administered as a monthly intramuscular injection.

Key Points: Management of Bronchiolitis

1. Care is mainly supportive.
2. Trial of inhaled β-receptor agonists may be useful, but most children do not respond.
3. Use of corticosteroids for bronchiolitis is not supported by evidence.
4. Typical course is progression of symptoms over first 3 to 5 days, with resolution over 2 to 3 weeks.

36. What is croup? How is it diagnosed?

Croup is a viral infection of the upper respiratory tract lasting 7 to 10 days. It is the most common cause of stridor in a febrile child. Parainfluenza virus can be recovered from the nasopharynx in over 90% of cases. The rest of the cases are caused by influenza, RSV, measles, or adenovirus. It is most often seen in the fall months with a peak in October, and it typically affects children between the ages of 6 and 36 months.

Croup usually begins with a fever (temperature 38-39° C) and rhinorrhea. The cricoid cartilage causes the mucosal swelling to occur inward, encroaching on the airway. A harsh, barky, "seal-like" cough begins within 12 to 48 hours. There may be associated tachypnea, stridor, and subcostal retractions in more severe cases.

37. What is the differential diagnosis of croup?

Conditions that may be mistaken for croup include bacterial tracheitis, retropharyngeal abscess, and epiglottitis (Table 41-2). Epiglottitis is a much less common entity since 1990, with the introduction of the Hib vaccine. In a 6- to 24-month-old afebrile child, an index of suspicion should also be kept for an aspirated foreign body. Often a clear history of choking is not obtained.

Table 41-2. Differential Diagnosis of Croup

VARIABLE	CROUP	EPIGLOTTITIS	RETROPHARYNGEAL ABSCESS	BACTERIAL TRACHEITIS
Anatomic area affected	Subglottic	Supraglottic	Retropharynx	Trachea
Age	6-36 mo	Any	6 mo-4 y	Any
Onset	Hours	1-3 days	1-3 days	3-5 days
Toxicity	None	Marked	Marked	Marked
Drooling	No	Yes	Yes	No
Voice	Hoarse	Muffled	Muffled	Normal
Cough	Barky	None	None	Painful
Radiographic finding	"Steeple"	"Thumb"	Widened retropharynx	"Shaggy"

Table 41-3. Westley Croup Score*

VARIABLE	0	1	2	3
Stridor	None	With stethoscope	At rest	
Retraction	None	Mild	Moderate	Severe
Air entry	Normal	Mild decrease	Marked decrease	

*Cyanosis = 4 points; altered mental status = 5 points.

38. When are diagnostic tests indicated (or of value) in croup?

Croup is diagnosed on the basis of presenting signs and symptoms. Children with characteristic disease do not require further diagnostic testing. Anteroposterior and lateral radiographs of the neck may be indicated in children with atypical features (e.g., age less than 6 months, recurrent or prolonged croup, toxicity on examination). The typical radiographic findings are shown in Table 41-2.

39. How do you assess the severity of croup?

Several scoring systems have been devised to assess croup severity. The most commonly used is the modified Westley score (Table 41-3). A score of less than 4 indicates mild disease, 4 to 6 indicates moderate disease, and a score of 7 indicates severe disease.

Kilic A, Ünüvar E, Sütçü M, et al: Acute obstructive respiratory tract diseases in a pediatric emergency unit. Pediatr Emerg Care 2012;28:1321-1327.

40. What are indications for admission in a child with croup?

Admit a child to the hospital with a croup score of 7, significant respiratory compromise, dehydration, an unreliable caretaker, or lack of adequate follow-up. For those severely ill patients not responding to therapy, an arterial blood gas may be helpful in deciding on admission to the general ward versus the pediatric ICU.

41. Is there any scientific evidence that humidified air is beneficial in the treatment of children with croup?

No. In theory, the humidified air moistens secretions and soothes the inflammation of the mucosa. However, despite the ubiquitous recommendations for the use of "the steamy bathroom" and "misty ox," controlled trials have failed to demonstrate a clinical benefit.

Nieto GM, Kentab O, Klassen TP, Osmond MH: A randomized controlled trial of mist in the acute treatment of moderate croup. Acad Emerg Med 2002;9:873-879.

Scolnik D, Coates AL, Stephens D, et al: Controlled delivery of high vs low humidity vs mist therapy for croup in emergency departments: A randomized controlled trial. JAMA 2006;295:1274.

42. Is there any scientific evidence that steroids are beneficial in the treatment of croup?

Yes. Although many of the randomized trials involving the use of steroids in croup have involved relatively small numbers of patients, meta-analyses of these trials have shown significant decreases in clinical croup scores and decrease in risk of intubation in patients with croup who receive steroids. In addition, one study has also demonstrated that steroids reduce symptom duration even in children with mild symptoms. In general, treat patients with more than minimal symptoms (e.g., a croup score = 1) with steroids.

Bjornson CL, Klassen TP, Williamson J, et al: A randomized trial of a single dose of oral dexamethasone for mild croup. N Engl J Med 2004;351:1306.

Russell K, Liang Y, O'Gorman K, et al: Glucocorticoids for croup. Cochrane Database Syst Rev 2011;(1): CD001955.

43. Which method of corticosteroid delivery is better: intramuscular, oral, or nebulized? What is the ideal dose?

Both dexamethasone (given orally or intramuscularly) and the synthetic glucocorticoid budesonide (inhaled) have been shown to be effective in the treatment of croup, although recent studies have suggested that systemic dexamethasone is superior. Comparisons of oral

and intramuscular dexamethasone have found similar rates of clinical improvement. The usual dose of dexamethasone for croup has traditionally been 0.6 mg/kg intramuscularly or orally, though doses of 0.15 and 0.3 mg/kg have been shown to be similarly effective.

44. Is racemic epinephrine superior to "regular" L-epinephrine in the treatment of croup?

No. Nebulized epinephrine has been shown to provide short-term benefit in the treatment of croup, presumably by reducing tracheal secretions and mucosal edema. Racemic epinephrine has been advocated traditionally, based on the belief that a mixture of D- and L-isomers would lead to less tachycardia. However, in randomized trials directly comparing the two drugs, they were equivalent in terms of efficacy and side effects. A dose of 5 mL of L-epinephrine 1:1000 is equal to 0.5 mL of the racemic (2%) mixture.

Bjornson C, Russell KF, Vandermeer B, et al: Nebulized epinephrine for croup in children. Cochrane Database Syst Rev 2011;(2):CD006619.

45. Do patients who receive nebulized epinephrine in the ED require admission to the hospital?

Because of the "rebound phenomenon"—a tendency to return to the baseline clinical picture after the epinephrine wears off—administration of nebulized epinephrine to children with croup in the ED became a major reason for admission to the hospital in the 1980s and early 1990s. However, studies have demonstrated a lack of adverse outcome in children who are discharged after a period of observation (typically 2-4 hours) after epinephrine. To be safely discharged, it is recommended that the child remain clinically stable (no stridor at rest, normal air entry, and oxygen saturation), and that the child receive a dose of dexamethasone prior to discharge.

46. How reliable are signs and symptoms in diagnosing pneumonia in young children?

Infants and young children with pneumonia often have subtle and nonspecific pulmonary findings. The issue of "occult" pneumonia—pneumonia in the absence of any pulmonary findings—has been a controversial one. Several authors have found that the risk of pneumonia is very low in the absence of any findings, but they caution that tachypnea may be a sole finding. Tachypnea, in fact, has the greatest negative predictive value. A study of highly febrile children with leukocytosis (white blood cell count > 20,000) found that 26% of such children in whom pneumonia was unsuspected clinically had infiltrates on chest radiograph. However, in this study tachypnea was evaluated qualitatively.

47. What are the most common bacterial causes of pneumonia in children?

The most common bacterial causes of pneumonia in children are listed in Table 41-4.

48. What organisms are most commonly associated with pleural effusion?

Pneumococci, *Staphylococcus aureus*, and *Mycobacterium tuberculosis* are the most common causes of effusion in children with pneumonia.

Table 41-4. Most Common Bacterial Causes of Pneumonia in Children

NEONATE (0-2 MO)	INFANT (2 MO-3 Y)	PRESCHOOL/SCHOOL AGE (>3 Y)
Group B streptococci	*S. pneumoniae*	*Mycoplasma pneumoniae*
Gram-negative bacilli	*S. aureus*	*S. pneumoniae*
Staphylococcus aureus	*Haemophilus influenzae**	*Chlamydia pneumoniae*
Chlamydia trachomatis		
Streptococcus pneumoniae		

*In unimmunized populations.

49. Are blood cultures necessary for children with community-acquired pneumonia?

There is no need for routine blood cultures in nontoxic, fully immunized children who will be managed as outpatients with community-acquired pneumonia. If the child does not improve clinically, deteriorates after initiation of antibiotic therapy, has complicated pneumonia, or is ill enough to require hospital admission, obtain a blood culture.

Bradley JS, Byington CL, Shah SS, et al: The management of community-acquired pneumonia in infants and children older than 3 months of age: Clinical Practice Guidelines by the Pediatric Infectious Diseases Society and the Infectious Diseases Society of America. Clin Infect Dis 2011;53:e25-e376.

50. What are the indications for admission to the hospital for an infant or child with community-acquired pneumonia?

Admit children with pneumonia if there is respiratory distress or hypoxemia (O_2 saturation $< 90\%$). Also, admit babies younger than 3 to 6 months of age with pneumonia and those with suspected infection due to community-associated methicillin-resistant *S. aureus* (CA-MRSA). Finally, admit infants and children when the parents are not compliant with therapy or when careful observation at home is unlikely.

Bradley JS, Byington CL, Shah SS, et al: The management of community-acquired pneumonia in infants and children older than 3 months of age: Clinical Practice Guidelines by the Pediatric Infectious Diseases Society and the Infectious Diseases Society of America. Clin Infect Dis 2011;53:e25-e376.

51. Which antibiotics are recommended for community-acquired pneumonia in infants and children?

Use amoxicillin as first-line therapy for a previously healthy, appropriately immunized child with mild to moderate pneumonia suspected to be bacterial in origin. If the child is ill enough to be admitted to the hospital, use IV ampicillin when local epidemiologic data indicate lack of substantial high-level penicillin resistance for invasive *Streptococcus pneumoniae*. It is recommended to order a third-generation cephalosporin (ceftriaxone or cefotaxime) when the admitted child or infant is not fully immunized, when there is suspected high-level penicillin resistance, or for those with life-threatening infection, such as empyema. Add a macrolide empirically for a hospitalized child if *Mycoplasma* or *Chlamydia* infection is suspected, and add vancomycin or clindamycin if infection with *S. aureus* is suspected.

Bradley JS, Byington CL, Shah SS, et al: The management of community-acquired pneumonia in infants and children older than 3 months of age: Clinical Practice Guidelines by the Pediatric Infectious Diseases Society and the Infectious Diseases Society of America. Clin Infect Dis 2011;53:e25-e376.

CHAPTER 42

TECHNOLOGY-ASSISTED CHILDREN—ACUTE CARE

Kate M. Cronan, Joel A. Fein, and Jill C. Posner

1. What are the most common reasons for tracheostomy tube placement in a child?

In children, tracheostomies are indicated for respiratory insufficiency due to a variety of causes, most commonly bronchopulmonary dysplasia and airway anomalies (e.g., congenital anomalies and subglottic stenosis). Other relatively common indications are neuromuscular disorders or central disorders, such as a brain tumor or Chiari malformation. A child with a tracheostomy may or may not require additional ventilatory assistance.

Al-Samri M, Mithell I, Drummond DS, Bjornson C: Tracheostomy in children: A population-based experience over 17 years. Pediatr Pulmonol 2010;45(5):487-493.

Hadfield PJ, Lloyd-Faulconbridge RV, Almeyda J, et al: The changing indications for paediatric tracheostomy. Int J Pediatr Otorhinolaryngol 2003;67:7-10.

Kremer B, Botos-Kremer AI, Eckel HE, Schlondorff G: Indications, complications, and surgical techniques for pediatric tracheostomies—An update. J Pediatr Surg 2002;37(11):1556-1562.

2. How do I determine the appropriate tracheostomy tube size?

A tracheostomy tube is sized by three dimensions: its inner diameter, its outer diameter, and its length. Ascertain the size of the tube by checking the flanges of the tube where the inner diameter, and in many cases the outer diameter, is imprinted. Many caretakers of technology-assisted children become experts in their child's technology and are excellent resources for medical personnel. Often, children will travel with emergency boards or "go bags," which contain all the equipment necessary to change a tracheostomy tube, including a replacement tube. If a patient presents without a tube in place and no replacement tube on hand, a reasonable estimate would be to use the formula for sizing an uncuffed endotracheal tube: (16 + age)/4 or 4 + (age/4).

Key Points: Three Dimensions of a Tracheostomy Tube

1. ID (inner diameter)
2. OD (outer diameter)
3. L (length)

3. What are the immediate management priorities for a child with a tracheostomy who presents in respiratory distress?

Assume this patient has an inadequate airway until proven otherwise. The tracheostomy tube may be obstructed or malpositioned. Assess airway patency and the adequacy of breathing through physical examination and the usual monitoring. Administer supplemental oxygen. Suction the tracheal tube to evaluate patency and to clear secretions, as this may help to alleviate symptoms. Do not be falsely reassured by a tube entering into the stoma, because it may actually descend into the soft tissues of the neck rather than into the trachea, especially if a prior tracheostomy tube change was attempted and unknowingly resulted in a false passage. An emergent tracheostomy tube change may be indicated if respiratory distress persists or the cannula is clearly dislodged.

4. What are the most common causes of respiratory distress in a child who has a tracheostomy tube?

Mechanical failure (cannula obstruction or dislodgement), infectious causes (pneumonia, tracheitis, viral respiratory infections), asthma, or reactive airway disease.

5. What are the causes of bleeding from the tracheostomy tube?

The most common cause of bleeding from the tracheostomy tube is drying and friability of the tracheal mucosa due to inadequate humidification. Anatomically, the tracheostomy tube inserts into the airway below the vocal cords. Therefore, inspired air bypasses the natural warming and humidification processes of the upper airway. Humidification is an important component of the ventilator circuit for patients with mechanical ventilation. For those patients who breathe independently from the ventilator, a heat-moisture exchanger device is attached at the opening of the tube. This plastic device contains a hydrophilic substance that captures the patient's own heat and humidity on exhalation so that it can be inhaled on inspiration. Other causes of bleeding include granuloma formation, infection (i.e., tracheitis), and erosion of the tube tip into a blood vessel.

6. How should I manage bleeding from the tracheostomy tube?

First, ensure the adequacy of the airway, breathing, and circulation. Suction frequently to avoid aspiration of blood. Minor bleeding usually indicates the need to increase inspired humidification. Evaluation by an airway specialist (e.g., otorhinolaryngologist) may be indicated for persistent minor bleeding to assess for intratracheal granuloma formation. A larger amount of blood may indicate that the tip of the tube has eroded into a blood vessel. Treat this as a surgical emergency with immediate notification of surgical, anesthesia, and operating room personnel. Obtain intravenous (IV) access with two large-bore catheters and initiate resuscitation with isotonic fluids or blood products. Overinflating the tracheostomy tube may tamponade the bleeding vessel, thereby providing a temporizing measure. Importantly, leave the tracheostomy tube in place, as this may be the only way to ensure an airway.

7. What can I do if I am unable to replace a dislodged tracheostomy tube?

When a tracheostomy tube becomes dislodged or decannulated, there is a tendency for the stoma to constrict, making it difficult to replace the tube. If stomal constriction prevents insertion of the replacement tube, several options are available: (1) insert a smaller tracheostomy tube; (2) insert a smaller endotracheal tube (being careful not to advance it into a mainstem bronchus), and then slowly dilate the stoma by inserting tubes of successively increasing size; (3) cover the stoma and use a bag-valve-mask device to ventilate by using traditional methods via the patient's upper airway; and (4) insert an oral tracheal tube. Oral intubation may be exceptionally difficult, especially if the original indication for tracheostomy was to overcome an airway anomaly. Therefore, use neuromuscular blocking agents with great caution and consider consulting specialists from anesthesiology or otolaryngology early.

Posner JC: Acute care of the child with a tracheostomy. Pediatr Emerg Care 1999;15:49-54.

Posner JC, Cronan K, Badaki O, Fein JA: Emergency care of the technology-assisted child. Clin Pediatr Emerg Med 2006;7:38-51.

8. What are the indicators for placement of a gastrostomy tube in a child?

- Inability to take in liquids and solids orally (e.g., neurologic conditions, prematurity)
- Decreased ability to keep up with metabolic demands through oral intake (e.g., patients with sepsis and other critical illnesses)
- Absorption abnormalities requiring changes in dietary intake (e.g., patients with chronic renal insufficiency, inflammatory bowel disease)

Collier S, Duggan C: Enteral nutrition in infants and children. UpToDate, June 2013. Available from www.uptodate.com.

9. What is the difference between a percutaneous endoscopic gastrostomy (PEG) tube and a surgically placed gastrostomy tube?

- A *PEG* procedure may be performed under sedation or general anesthesia. An endoscope is inserted into the esophagus, and a light source indicates the appropriate location on the stomach wall. An incision is made on the external abdominal wall, and the tube is placed via this aperture.
- A *surgical or open gastrostomy tube* is placed via laparotomy or laparoscopy under general anesthesia. A purse-string suture is made in the stomach to secure the tube, and the stomach is then sutured to the abdominal wall.

Brewster B, Weil B, Ladd A: Prospective determination of percutaneous endoscopic gastrostomy complication rates in children: Still a safe procedure. Surgery 2012;152(4):714-721.

10. What are some of the complications of gastrostomy tubes in children?

- Dislodgement (most common)
- Clogging
- Leaking
- Gastric outlet obstruction
- Bleeding
- Gastric ulceration
- Worsened gastroesophageal reflux
- Skin irritation and rash around the tube

Koulentaki M, Reynolds N, Steinke D, et al: Eight years' experience of gastrostomy tube management. Endoscopy 2002;34:941-945.

Saavedra H, Losek JD, Shanley L, Titus MO: Gastrostomy tube-related complaints in the pediatric emergency department: Identifying opportunities for improvement. Pediatr Emerg Care 2009;25 (11):728-732.

11. How is dislodgement of a gastrostomy tube managed in the emergency department (ED)?

The first step is to determine the age of the stoma. If the stoma is older than 8 weeks, the ED physician or nurse should take action. If the stoma is less than 8 weeks, contact the gastroenterologist or surgeon for input.

As soon as possible, stent the stoma open with a temporizing device, such as a Foley catheter, placed with lubricant. Consider the use of a mild sedative or analgesic. When the stoma is located, place the appropriate-size replacement gastrostomy tube in the stoma after removing the Foley catheter. If the tube has been dislodged for an extended period of time (hours), smaller gastrostomy tubes may be initially required to dilate the stoma. Always check the balloon's function before replacing the tube. Fill the balloon and check the tube to ensure that it is snug.

Key Points: Steps to Replace a Dislodged Gastrostomy Tube

1. Determine the age of the stoma and seek advice if the tube was recently placed.
2. Consider sedation and analgesics.
3. Stent the stoma open with a temporary device.
4. Check the balloon of the new gastrostomy tube.
5. Remove the temporary tube.
6. Place the new gastrostomy tube using lubricant.
7. Fill the balloon and check for adequate fit.

12. What is the danger of inserting a tube into a stoma that has recently been placed?

The stomach wall may not have adhered to the peritoneal lining and skin. The fistula tract can be disrupted, and a false lumen into the peritoneum may be formed. Formula inserted through the tube can lead to chemical or bacterial peritonitis.

13. What are some of the complications of replacing a gastrostomy tube?

- Accidental insertion into the peritoneal cavity can occur in specific scenarios.
- Lysis of adhesions of the stomach wall away from the abdominal wall may result in pneumoperitoneum.
- If a false lumen is created, formula may be installed into the space, causing chemical peritonitis.
- Inadequate advancement of the tip of the tube can result in the tube's staying in the fistula; the fistula can thus be disrupted when the balloon is dilated, and having the tip in the fistula can result in pain.
- Bleeding at the site of insertion can occur from trauma during insertion.
- A wound infection of the skin can occur.

Graneto J: Gastrostomy tube replacement. In Henretig FM, King C (eds): Textbook of Pediatric Emergency Medicine Procedures. Baltimore, Lippincott Williams & Wilkins, 2008, pp 832-833.

14. Is it necessary to perform a dye study in order to verify gastrostomy tube location after replacement?

Not usually. Aspiration of stomach contents after the gastrostomy tube is replaced confirms that the tube is in the stomach. Listening over the stomach for borborygmi when 15 mL of air is inserted into the tube's lumen is also confirmatory. If in doubt after these actions, take a plain radiograph after radiopaque contrast material is inserted through the tube.

Graneto J: Gastrostomy tube replacement. In Henretig FM, King C (eds): Textbook of Pediatric Emergency Medicine Procedures. Baltimore, Lippincott Williams & Wilkins, 2008, pp 832-833.

15. What is the best treatment for a clogged gastrostomy tube?

Warm water is the best irrigant and has been shown to be superior to vinegar, soda, and juices. Repeated gentle flushing is advised. Prevention is best accomplished with regular flushing before and after each gastrostomy tube use.

16. What are some complications that can occur in relation to the stoma?

- Peristomal infection
- Irritation
- Bleeding
- Stretching
- Granuloma formation

17. How is a stomal granuloma treated?

Application of silver nitrate to the granuloma is most often successful.

Graneto J: Gastrostomy tube replacement. In Henretig FM, King C (eds): Textbook of Pediatric Emergency Medicine Procedures. Baltimore, Lippincott Williams & Wilkins, 2008, pp 832-833.

18. Name the most common symptoms associated with cerebrospinal fluid (CSF) shunt obstruction.

Symptoms frequently include headache, vomiting, and some alteration of mental status, however subtle. Alteration in vital signs may reflect autonomic instability or brain herniation. Seizures are rarely the only manifestation of increased intracranial pressure and more likely represent an underlying seizure disorder. When a CSF shunt is obstructed, parents often accurately state, "This is what my child looked like when she/he had the last shunt obstruction."

Browd SR, Ragel BT, Gottfried ON, Kestle JR: Failure of cerebrospinal fluid shunts: Part I: Obstruction and mechanical failure. Pediatr Neurol 2006;34(2):83-92.

Key Points: Symptoms of Ventriculoperitoneal Shunt Obstruction

1. Headache
2. Vomiting
3. Change in mental status (may be subtle)
4. Alteration of vital signs

19. What is the best study to diagnose shunt malfunction?

Conventional wisdom (which is not always wise and need not always be conventional) suggests that easy depression of a shunt reservoir bubble signifies that the distal end is patent, and rapid refilling of this bubble signifies that the proximal shunt is patent. In reality, pumping the shunt identified fewer than 40% of obstructed shunts, and a finding of "normal filling" would miss between 10% and 35% of shunt obstructions. Not all shunts have a reservoir that can be pumped. Computed tomography (CT) is a tried and true adjunct to diagnose shunt obstruction, when placed together with other clinical information such as symptoms, prior history, and physical examination. When available, rapid T2-weighted magnetic resonance imaging (MRI) is a reasonable alternative and is becoming more common, especially in patients who do not require sedation. This has the advantage of no radiation exposure to the patient.

Lee P, DiPatri AJ: Evaluation of suspected cerebrospinal fluid shunt complications in children. Clin Pediatr Emerg Med 2008;9:76-82.

Piatt JH: Physical examination of patients with cerebrospinal fluid shunts: Is there useful information in pumping the shunt? Pediatrics 1992;89:470-473.

20. When should shunt infection be suspected? How can I differentiate CSF shunt infection from obstruction?

Suspect shunt infection in any febrile child with a shunt. However, in one series, fever was present in fewer than half of the patients with shunt infection. A history of surgical manipulation of the shunt in the prior 2 months should increase suspicion for infection; infections rarely occur more than 6 months after surgery. An age of less than 6 months at initial shunt placement or having had one or more revisions significantly increases the rate of subsequent infection. Symptoms of CSF shunt infection overlap considerably with those of CSF shunt obstruction, because the diagnoses often occur together. The higher viscosity of infected fluid may lead to a partial or complete obstruction of the small fenestrations on the shunt tubing.

Duhaime AC: Evaluation and management of shunt infections in children with hydrocephalus. Clin Pediatr 2006;45(8):705-713.

Simon TD, Whitlock KB, Riva-Cambrin J, et al: Revision surgeries are associated with significant increased risk of subsequent cerebrospinal fluid shunt infection. Pediatr Infect Dis J 2012;31(6):551-556.

Stone JJ, Walker CT, Jacobson M, et al: Revision rate of pediatric ventriculoperitoneal shunts after 15 years. J Neurosurg Pediatr 2013;11(1):15-19.

21. What studies are helpful in diagnosing CSF shunt infections?

Culture of an organism from the CSF is the most accurate method of diagnosing a shunt infection. Attention to the white blood cell count and a Gram stain from the shunt fluid are helpful but not foolproof methods of determining if a shunt is infected. The Gram stain is positive in a little more than half of all shunt infections. CSF that is aspirated from an infected shunt usually reveals a moderate pleocytosis but can be misleadingly normal, as can the CSF glucose level. Peripheral white blood cell counts and blood cultures are frequently normal in the presence of shunt infection. Consult neurosurgery before tapping the shunt. The neurosurgeon may prefer that the procedure not be done by ED personnel, as there is a risk for introducing infection during the procedure.

Anderson EJ, Yogev R: A rational approach to the management of ventricular shunt infections. Pediatr Dis J 2005;24(6):557-558.

22. What techniques can an emergency medicine practitioner use to treat the critically ill patient with a CSF shunt obstruction?

When a child with a suspected CSF shunt obstruction is critically ill with signs of increased intracranial pressure, emergent action is required to reduce that pressure. Conventional therapies, such as hyperventilation and mannitol administration, may be used, but if possible, relieve the pressure by accessing the shunt. If there is a distal obstruction or partial proximal obstruction, withdraw fluid in a sterile fashion using a 23-gauge butterfly needle and 20-mL syringe. This potentially lifesaving procedure is usually done by a neurosurgeon, but do not delay if the patient's condition is deteriorating—the procedure is well described in pediatric emergency procedure textbooks.

Wiley JF II, Duhaime AC: Ventricular puncture. In King C, Henretig FM (eds): Textbook of Pediatric Emergency Procedures, 2nd ed. Philadelphia, Lippincott Williams & Wilkins, 2008.

23. What are some complications that can result from accessing a central venous catheter (CVC) in the ED?

- Occlusion of the catheter
- Air embolus
- A break in the catheter
- Displacement of the catheter
- Infection

Fein JA, Cronan KM, Posner JC: Approach to the care of the technology-assisted child. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1729-1743.

24. What are some tips for accessing an indwelling venous catheter?

Use aseptic technique whenever you access an indwelling venous device. Do not use clamps or hemostats with teeth because they may damage the device. Do not use tincture of iodine to clean the area, as this may damage the catheter. Use povidone-iodine or 2% chlorhexidine instead. Do not use a small syringe (<3 mL), as this can generate significant pressure and cause the catheter to break. Flush the entire IV circuit before accessing the system. Always close the clamps when the system is open, and do not infuse medications or fluid until patency of the system has been established. Flush the catheter with 10 mL of saline between medications to reduce the chance of occlusion, and flush the cap or reservoir with heparin when the procedure is completed.

Fein JA, Cronan KM, Posner JC: Approach to the care of the technology-assisted child. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1729-1743.

DENTAL/PERIODONTAL EMERGENCIES

Susanne Kost

CHAPTER 43

1. What is the typical age at which an infant acquires the full complement of primary teeth?

Normal variation can range from 4 to 15 months for eruption of the first tooth, but most children acquire the full complement of 20 primary teeth by around their second birthday. A good mnemonic is the "7 + 4 rule," which refers to first tooth by 7 months, 4 teeth by 11 months, 8 teeth by 15 months, and so on until 20 teeth by 27 months.

2. Which are the first permanent teeth to erupt? Which teeth come next?

The first permanent teeth to erupt are usually the first permanent molars ("6-year" molars), followed by the mandibular central incisors, maxillary central incisors, lateral incisors, cuspids, and bicuspids. The mixed dentition phase concludes at about age 12 years with the eruption of the second permanent ("12-year") molars, but the adult complement of 32 teeth is not achieved until the eruption of the third molars ("wisdom" teeth) in late adolescence (Fig. 43-1). Up to 20% of people never develop third molars, with large genetic variation among populations.

3. What are Epstein's pearls? How do they differ from Bohn's nodules and dental laminal cysts?

Cystic lesions in the oral cavity of newborns are quite common, occurring in 80% of normal newborns. They are generally small and superficial and are caused by entrapment of bits of embryologic tissue within dental epithelium.

- **Epstein's pearls** are tiny 1- to 2-mm keratin-filled cystic lesions located along the midpalatine raphe.
- **Bohn's nodules** are small mucous gland cysts found on the alveolar ridges or posterior palate.
- **Dental laminal cysts** are larger, more lucent, fluctuant cysts consisting of remnants of dental laminal epithelium. They are usually single lesions, and they are found only on the crest of the alveolar mucosa.

Nelson LP, Shusterman S: Dental emergencies. In Fleisher GR, Ludwig S, Henretig FM (eds): Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 1538-1544.

4. What is ankyloglossia? What is the most common associated complication?

Ankyloglossia, or tongue-tie, is a condition affecting 4% to 10% of the population, with varying degrees of severity. A short, tight lingual frenulum can cause difficulty with breastfeeding and subsequent failure to thrive. Frenotomy, or clipping of the frenulum, can improve breastfeeding but in rare cases can cause bleeding or infection.

Kumar M, Kalke E: Tongue-tie, breastfeeding difficulties and the role of frenotomy. Acta Paediatr 2012;101 (7):687-689. Epub 2012 Apr 5.

5. Does teething cause fever?

No. Teething has been blamed for a number of symptoms in infants throughout time, ranging from fever, congestion, diarrhea, and rashes to seizures and death. A recent prospective descriptive study of 231 tooth eruptions in 47 children looked at parental reports of 13 "teething symptoms" daily for 8 months. The most frequent signs and symptoms that were statistically linked to the eruption of a tooth included mild temperature elevation (0.1° C tympanic on the day of eruption), irritability (median: 0.60; $P < 0.001$), increased salivation (median: 0.50; $P < 0.001$), runny nose (median: 0.50; $P < 0.001$), and loss of appetite (median: 0.50; $P < 0.001$).

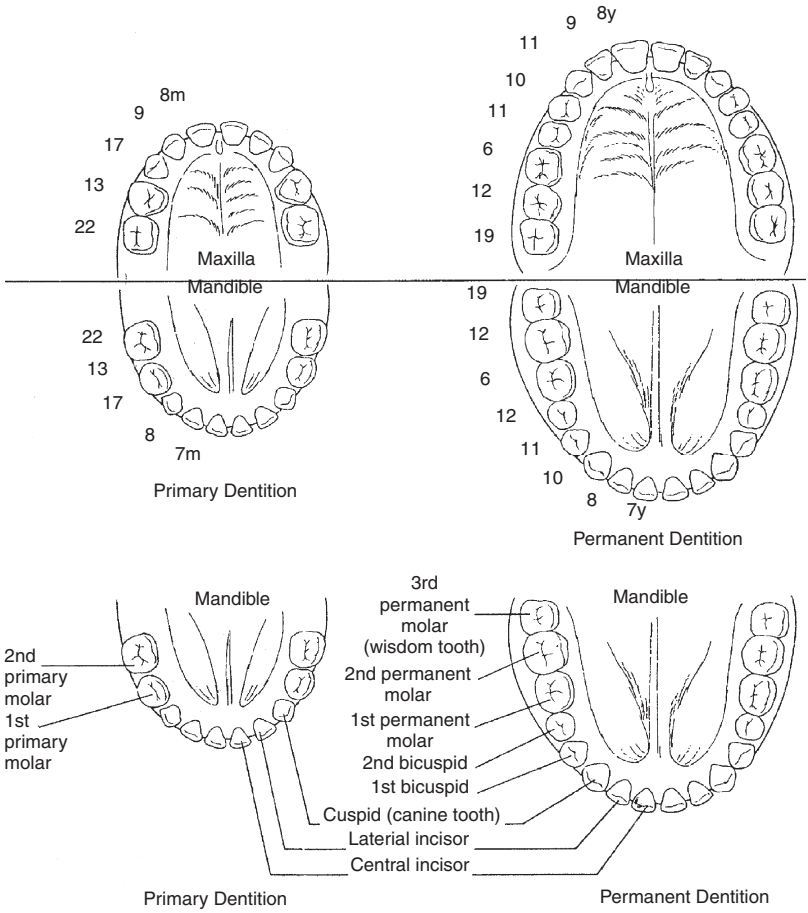

Figure 43-1. Primary and secondary tooth eruptions. (*From Nazif MM, et al: Oral disorders. In Zitelli BJ, McIntire SC, Nowalk AJ (eds): Atlas of Pediatric Physical Diagnosis, 6th ed. Philadelphia, Elsevier, 2012, Fig. 20.9*)

Ramos-Jorge J, Pordeus IA, Ramos-Jorge ML, Paiva SM: Prospective longitudinal study of signs and symptoms associated with primary tooth eruption. Pediatrics 2011;128(3):471-476. Epub 2011 Aug 8.

Key Points: Conditions for Which Teething Should Not Be Blamed

1. Common viral symptoms of young children
2. Upper respiratory tract infections
3. Significant fever

6. An 8-year-old presents with a blue-black cystic lesion over an erupting tooth. What is the cause?

This lesion is an eruption cyst. It is a nontender, fluid-filled cyst that sometimes forms over the crown of an erupting tooth. Occasionally, these cysts will fill with blood, creating an eruption hematoma. These cysts or hematomas are benign and self-limiting conditions that resolve when the tooth erupts.

7. How common is dental caries? What are the three main "ingredients" for dental caries to occur?

Dental caries is the most common disease process in children, affecting more than 25% of all school-aged children and disproportionately affecting indigent children. The disease of dental caries is multifactorial in origin, requiring a susceptible host (genetic predisposition), a cariogenic diet (sticky carbohydrates), and bacteria (most commonly *Streptococcus mutans*, which converts carbohydrate to acid that degrades dental enamel).

S. mutans has been shown to be transmitted both vertically and horizontally and can colonize the mouths of infants even prior to the eruption of teeth. Thus, practices such as "cleaning" a pacifier with maternal saliva before insertion into the infant's mouth should be strongly discouraged.

Berkowitz RJ: Mutans streptococci: Acquisition and transmission. Pediatr Dent 2006;28:106-109.
Centers for Disease Control: Untreated cavities in kids. Available at http://www.cdc.gov/features/dsuntreatedcavitieskids/index.html.

8. Is chewing gum good for your teeth?

Maybe. Chewing "sugarless" gum containing xylitol, a sugar alcohol, three to five times daily has been shown to significantly reduce plaque, *S. mutans* carriage, and caries.

9. Do you have to drink from a bottle to get "baby bottle caries"?

No. "Baby bottle caries" is a term used to describe severe caries resulting in necrosis and loss of the upper incisors of toddlers and young children. In recognition of the fact that severe caries at a young age is a multifactorial problem, and not only related to prolonged exposure to sugars from the bottle, the term "bottle caries" has been replaced by "early childhood caries," or ECC. Upper incisors are disproportionately affected by caries, most likely because they are among the first teeth to erupt, and thus the first to be exposed to bacteria, and because the lower incisors are protected by the tongue and better saliva flow.

Çolak H, Dülgergil CT, Dalli M, Hamidi MM: Early childhood caries update: A review of causes, diagnoses, and treatments. J Nat Sci Biol Med 2013;4(1):29-38.

10. What are the indications for hospital admission for a patient with a dental abscess?

Most dental abscesses can be managed on an outpatient basis with oral antibiotics and close dental follow-up for drainage or extraction of the affected tooth. Facial cellulitis resulting from an abscessed tooth can be managed with outpatient antibiotics also, unless the orbit is involved or the amount of swelling and pain compromises the airway or the ability to take fluids. Dentoalveolar abscesses are a common cause of facial cellulitis based on the anatomy of the alveolar bone. The outer (buccal) aspect of the bone is much thinner than the inner (lingual) aspect; the path of least resistance for drainage is through the fascial planes of the cheek and face. Admit children who are toxic-appearing, highly febrile, or lacking appropriate social support for follow-up.

Nelson LP, Shusterman S: Dental emergencies. In Fleisher GR, Ludwig S, Henretig FM (eds): Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 1538-1544.

Key Points: Areas into Which Untreated Dental Abscesses Can Extend

1. Sinuses
2. Orbits
3. Brain
4. Airway
5. Mediastinum

11. Describe common complications of wisdom tooth extraction.

Extraction of the third molars, or "wisdom teeth," is a common procedure in late adolescence and early adulthood. Complications resulting in a visit to the emergency department may include hemorrhage, infection, or alveolar osteitis ("dry socket"). The extraction site may normally ooze for 8 to 12 hours after the procedure; persistent oozing beyond 12 hours or frank bleeding at any point may require a coagulation profile. Most bleeding will be controlled with

pressure, best achieved by biting on gauze sponges for 15 to 30 minutes. Moistened tea bags placed over the socket may facilitate clotting, based on the coagulating properties of tannic acid. If pressure fails to control the bleeding, the socket may be packed with Gelfoam or closed with sutures. Infection of the extraction socket is rare but should be suspected in the presence of fever, swelling, or purulent exudate from the extraction site. Penicillin and pain control are the mainstays of treatment. Alveolar osteitis is a painful inflammatory process caused by disintegration of the clot in the tooth socket, typically occurring 3 days after extraction. Treatment consists of débridement and packing of the socket.

12. What are complications of oral piercings? Can they be life-threatening?

Complications of oral (lip and tongue) piercing include the following:

- Pain and swelling
- Localized infection
- Gingival recession and chipping of teeth
- Poor cosmetic outcome (keloids, bifid tongue)
- Embedded jewelry

Potentially life-threatening complications include disseminated infection, such as mediastinitis and endocarditis, and hemorrhage, especially in patients with an underlying predisposition.

Larzo MR, Poe SG: Adverse consequences of tattoos and body piercings. Pediatr Ann 2006;35:187-192.

13. What is meant by the term *geographic tongue*?

Geographic tongue, or benign migratory glossitis, is a painless condition notable for erythematous "islands" of denuded papillae surrounded by elevated whitish borders (Fig. 43-2). The islands appear to migrate over the surface of the tongue over time, akin to the movement of continents on the globe. The cause is unknown, with allergy, infection, and stress all implicated as potential contributors. It generally resolves without specific treatment over a period of weeks to months.

14. What is a ranula? How does it differ from a mucocele?

A ranula is a mucous retention cyst on the floor of the mouth, under the tongue (Fig. 43-3). A mucocele is a retention cyst located most commonly in the mucosa of the lower lip (Fig. 43-4). Both can range from several millimeters to a centimeter in diameter, are painless, and are felt to arise following trauma to the ducts of minor salivary glands. Treatment is surgical excision.

15. Give the systemic disease associated with the following mouth lesions:

1. Hyperpigmented macules on lips and buccal mucosa: Peutz-Jeghers syndrome (familial polyposis)
2. Early exfoliation (loss) of teeth: Langerhans cell histiocytosis

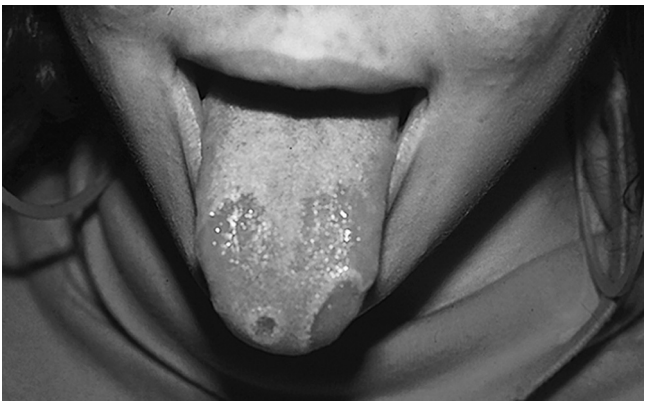

Figure 43-2. Geographic tongue. *(From Fitzpatrick JE, Morelli JG [eds]: Dermatology Secrets, 3rd ed. Philadelphia, Mosby, 2007, Fig. 58-5, p 467.)*

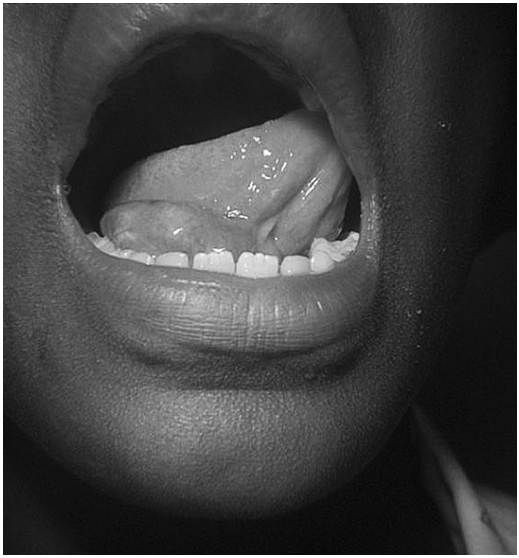

Figure 43-3. Ranula.

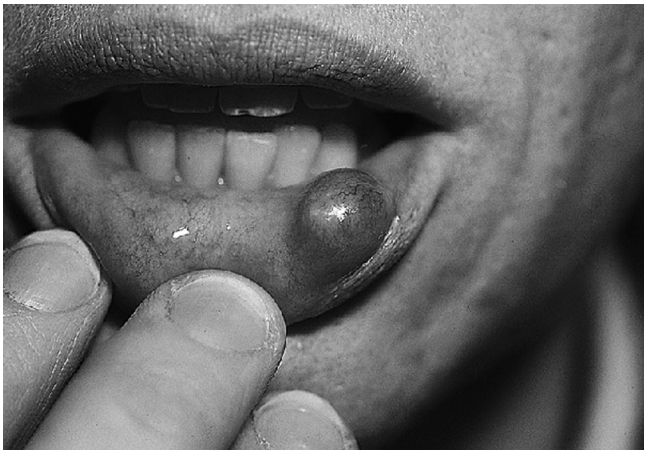

Figure 43-4. Mucocele. (*From Swartz MH: Textbook of Physical Diagnosis: History and Examination, 4th ed. Philadelphia, Saunders, 2002, Fig. 11-13, p 295).*

3. "Strawberry" lesions on the gingiva: Wegener's granulomatosis
4. Xerostomia (dry mouth) and enlarged parotid glands: Sjögren's syndrome
5. Leukoplakia: oral squamous cell carcinoma

16. What are the most common causes of stomatitis in young children?

Viral infections most commonly cause stomatitis in young children (<6 years old). Herpes simplex virus, usually type I, causes a syndrome of very painful vesicles and gingival

inflammation, sometimes to the point of bleeding, in the *anterior mouth and tongue*. Herpes stomatitis is often associated with high fevers (temperature of 39-40 ° C), headache, and cervical adenopathy. Coxsackieviruses cause a more benign vesicular inflammation of the *posterior pharynx*. Also known as herpangina, this infection causes pain, low-grade fever, and drooling. An acral viral exanthem (hand, foot, and mouth disease) is seen with some strains of coxsackievirus. Both infections are most common in the toddler age group, and both are treated symptomatically with topical or systemic analgesics. Some evidence suggests that acyclovir given early in the course of herpetic stomatitis may alleviate the symptoms.

17. Name three eponymous anaerobic infections associated with the mouth.

- **Vincent's disease**, also known as acute necrotizing ulcerative gingivostomatitis, or trench mouth, is a painful ulcerative gingivostomatitis caused by overgrowth of a spirochete, *Borrelia vincentii*. It is most common in adolescents. Treatment consists of vigorous oral hygiene, with the addition of penicillin in severe cases.
- **Ludwig's angina** is a life-threatening cellulitis of the sublingual and submandibular spaces. Rapid spread of infection into the neck may result in airway obstruction.
- **Lemierre's syndrome**, or postanginal sepsis, involves infection of the lateral pharyngeal space with septic thrombophlebitis of the jugular vein, potentially leading to septic embolization to the lungs or brain. The most commonly associated organism is *Fusobacterium*.

Fisher MC: Other anaerobic infections. In Behrmann RE, Kliegman RM, Jenson HB (eds): Nelson's Textbook of Pediatrics, 17th ed. Philadelphia, WB Saunders, 2004.

Websites

American Academy of Pediatric Dentistry. www.aapd.org.
American Academy of Pediatrics. http://www2.aap.org/ORALHEALTH/pact/.

GENERAL SURGERY EMERGENCIES

Kristine G. Williams and Dee Hodge III

CHAPTER 44

1. What is the most common acute surgical condition of the abdomen?
Appendicitis.

2. What is the classic presenting history in a child with suspected appendicitis?
If you are lucky, the child will have a history of periumbilical pain that migrated to the right lower quadrant (RLQ). However, pediatric patients rarely read the textbook. Usually the child presents with a history of diffuse abdominal pain, possibly associated with vomiting, and a low-grade fever. Some may have a history of anorexia. Unlike with adults, it is often hard to obtain an accurate history and to reliably examine children. The presenting symptoms of appendicitis are similar to those of many other childhood diseases. Gastroenteritis, urinary tract infections, streptococcal pharyngitis, constipation, and pneumonia are some of the illnesses that mimic appendicitis. Once peritoneal inflammation occurs, most older children with appendicitis will complain of pain in the RLQ.

3. Where else might a child with appendicitis localize pain?
If the appendix is located in the lateral gutter, the child may have tenderness in the lateral abdomen or flank. An appendix pointing toward the pelvis might cause pubic pain and diarrhea. A retrocecal appendix causes pain on deep palpation. This information is important, because not all children with appendicitis present with RLQ pain. In addition, it is important to consider the presentation of perforated appendicitis. These children often have fever, muscle guarding, and a longer duration of pain, and they tend to be younger.
Hung MH, Ling LH, Chen DF: Clinical manifestations in children with ruptured appendicitis. Pediatr Emerg Care 2012;28:433-435.

4. Is diarrhea a presenting symptom of appendicitis?
Yes. Diarrhea may delay accurate diagnosis if the symptom is mistakenly associated with gastroenteritis rather than appendicitis. Children less than 3 years old are the ones most likely to present with this symptom. A retrospective case series identified 63 children under the age of 3 who had an appendectomy for appendicitis. The mean age was 2.2 years. The mean delay from the onset of symptoms to presentation was 4.3 days, and 57% were misdiagnosed at initial presentation! Diarrhea was reported in one third of the patients. Although none of the children died, the morbidity rate was increased: 84% had perforation or gangrene.

You must continually keep serious and life-threatening disease processes in mind when a child, especially a young one, presents with symptoms that may look like simple gastroenteritis.
Horwitz JR, Gursoy M, Jaksic R, Lally KP: Importance of diarrhea as a presenting symptom of appendicitis in very young children. Am J Surg 1997;173(2):80-82.

5. Does a white blood cell (WBC) count of only 11.5 WBCs/mm^3 rule out appendicitis?
No. A child with a nonperforated appendicitis usually has a WBC count of 11,000 to 15,000 WBCs/mm^3 in the first 1 to 2 days of illness. A higher WBC count with a left shift may be found later in the course, as the appendix undergoes further degeneration, or if the appendix has ruptured. Although laboratory values may help to support your clinical diagnosis, they should not replace a good history and physical examination.

6. What laboratory findings are suggestive of appendicitis?
The two tests that should be done on all patients who are suspected of having appendicitis are a *complete blood count with differential* (CBC) and a *urinalysis* (UA). Elevated leukocyte counts and a left shift appear to be highly associated with both uncomplicated acute and

complicated acute appendicitis and are a good tool to aid in diagnosis. Although a normal or low WBC count may still be found in patients with appendicitis, this combination has been found to have a high negative predictive value. The UA may show ketosis due to decreased oral intake. If the inflamed appendix lies near the bladder or ureter, sterile pyuria may be seen as well. However, a normal CBC and a normal UA do not rule out appendicitis.

Wang LT, Prentiss KA, Simon JZ, et al: The use of white blood cell count and left shift in the diagnosis of appendicitis in children. Pediatr Emerg Care 2007;23:69-76.

7. Is C-reactive protein (CRP) helpful in diagnosing appendicitis?

Greatly elevated CRP levels (e.g., >50 mg/L) have been found to be associated with *perforated* appendicitis. A patient with a normal leukocyte count *and* a normal CRP level is unlikely to have appendicitis. Although many institutions do not routinely measure CRP in the diagnostic workup of appendicitis, there is increasing evidence that it may be an important consideration in assigning a clinical relative risk.

Gavela T, Cabeza B, Serrano A, et al: CRP and procalcitonin are predictors of the severity of acute appendicitis in children. Pediatr Emerg Care 2012;28:416-419.
Hennelly KE, Bachur R: Appendicitis update. Curr Opin Pediatr 2011;23:281-285.

8. Is a plain radiograph useful in diagnosing appendicitis?

Plain radiography should not be a routine part of the evaluation of a patient with clinically suspected appendicitis. It usually reveals nonspecific findings, unless a calcified appendicolith is present (<10% of patients). An obstructive series, in which the patient has radiographs taken in the supine and the left lateral decubitus positions, may suggest an RLQ process, although the process could be simple gastroenteritis. The logic of obtaining the left lateral decubitus film is that air is used as a contrast medium, allowing for better visualization of the RLQ. The presence of air-fluid levels in the RLQ suggests a localized ileus. Although this finding may heighten one's suspicion for appendicitis, beware! The same finding is also present in gastroenteritis. The bottom line is that the abdominal radiograph rarely changes the plan and so should not be a routine part of the evaluation of appendicitis.

Heller RM, Hernanz-Schulman M: Applications of new imaging modalities to the evaluation of common pediatric conditions. J Pediatr 1999;135(5):632-639.
Smith JE, Hall EJ: The use of plain abdominal x-rays in the emergency department. Emerg Med J 2009;26:160-163.

9. What are the pros and cons of ultrasound (US) in the diagnosis of appendicitis?

US has a reported sensitivity of approximately 80% and a reported specificity of approximately 93% for the diagnosis of appendicitis. It is quick and does not involve radiation. However, there are limitations to this technology:

- It requires an experienced technician.
- It may be uncomfortable for the patient due to a need for graded compression of overlying bowel.
- It can be difficult to perform on a crying, uncooperative, or obese child.
- The appendix can be located anywhere in the abdomen, making visualization difficult at times. If visualized, the inflammation may be localized to a small segment that cannot be seen. Thus, a positive result is highly suggestive, but a negative result should be followed with repeated physical examination and reassessment of your level of suspicion for the diagnosis.

10. What are the pros and cons of computed tomography (CT) in the diagnosis of appendicitis?

CT has a sensitivity of about 95% and a specificity of about 93% for the diagnosis of appendicitis. Drawbacks for CT include higher cost, the need for contrast, and a longer preparatory period prior to the study. However, radiation exposure is the greatest concern. In an effort to reduce the exposure to unnecessary radiation, many institutions have initiated protocols for evaluation of appendicitis, and there are several studies describing the protocols and outcomes. In most studies, when imaging is needed in the evaluation, US is the first choice. If US is equivocal or if it is negative and there is still a concern for appendicitis, most authors recommend either a CT scan or admission for serial abdominal examinations.

Ramarajan N, Krishnamoorthi R, Barth R, et al: An interdisciplinary initiative to reduce radiation exposure: Evaluation of appendicitis in a pediatric emergency department with clinical assessment supported by a staged ultrasound and computed tomography pathway. Acad Emerg Med 2009;16 (11):1258-1265.
Russell WS, Schuh AM, Hill JG, et al: Clinical practice guidelines for pediatric appendicits evaluation can decrease computed tomography utilization while maintaining diagnostic accuracy. Pediatr Emerg Care 2013;29(5):568-573.
Sivit CJ, Applegate KE, Stallion A, et al: Imaging evaluation of suspected appendicitis in a pediatric population: Effectiveness of sonography versus CT. AJR Am J Roentgenol 2000;175:977-980.

11. Can magnetic resonance imaging (MRI) be used instead of an abdominal CT scan to diagnose appendicitis?

MRI is an alternative to abdominal CT in children suspected of appendicitis when the patient has an inconclusive US. Sensitivity of MRI without contrast in diagnosing appendicitis is close to 100%, and specificity is 96%, with a positive predictive value of 88% and a negative predictive value of 100%. Although sedation of the child may be necessary to accomplish this study, there is no radiation involved, which makes MRI a very desirable study.

Herliczek TW, Swenson DW, Mayo-Smith WW: Utility of MRI after inconclusive ultrasound in pediatric patients with suspected appendicitis: Retrospective review of 60 consecutive patients. AJR Am J Roentgenol 2013;200:969-973.
Moore MM, Gustas CN, Choudhary AK, et al: MRI for clinically suspected pediatric appendicitis: An implemented program. Pediatr Radiol 2012;42:1056-1063.

12. Why does the pain of appendicitis typically start centrally (periumbilically) and then localize to the RLQ?

The initial pain of appendicitis is visceral: a dull, aching pain localized to the mid- or lower abdomen. *Visceral or splanchnic pain* originates in those abdominal organs, such as the appendix, that have visceral peritoneum. Increased hollow viscus wall tension and ischemia cause impulses to be sent from the organ to the spinal cord. In the case of the appendix, visceral afferent autonomic nerves enter the spinal cord at T8 to T10. These segments supply the periumbilical area, which explains the initial, poorly localized pain associated with appendicitis.

As the appendix becomes more inflamed, it causes inflammation of the serosa and parietal peritoneum. The inflammation triggers local somatic pain fibers. *Somatic pain* is mediated by afferent nerve fibers in segmental spinal nerves. Thus, the pain is sharper and more localized, usually to the RLQ.

Key Points: Common Findings in Acute Appendicitis

1. Diffuse or periumbilical abdominal pain
2. Low-grade fever
3. Vomiting or diarrhea
4. Anorexia

13. Describe the pathophysiologic mechanism that leads to appendicitis.

The appendix contains increasing amounts of lymphatic tissue as a child ages. Acute appendicitis occurs when the lumen of the appendix becomes obstructed. Sources of obstruction include fecaliths, lymphatic hypertrophy, worms, vegetable matter, and tumors. The appendix continues to secrete mucosal fluid, leading to distention of the viscus.

14. Which of these patients with appendicitis is most likely to present with a perforation? Why?

- A 2-year-old boy with abdominal pain and diarrhea for a few days
- A 12-year-old girl with severe pain for 24 hours
- An 8-year-old boy with a temperature of 40° C
- A 16-year-old with a WBC count of 18,000/mm^3

Although any one of the patients could present with a perforated appendix, the 2-year-old boy with pain for a few days and diarrhea is statistically most likely to have a perforation at

presentation. The rates of perforation are higher in younger children because of several factors: (1) the appendix is more thin-walled, predisposing them to early perforation, (2) younger children are less able to communicate clearly, resulting in prolonged symptoms before diagnosis, (3) the level of suspicion for appendicitis is often lower in younger age groups, leading to a delay in diagnosis, and (4) the signs and symptoms of appendicitis are often nonspecific in the younger child.

15. Why does perforated appendicitis progress to peritonitis more quickly in infants and children than in adults?

The omentum is not as well developed in infants and children as it is in adults. Because the omentum is primarily responsible for walling off the infection in a perforated appendix, its insufficiency contributes to the development of peritonitis. This complication of perforated appendicitis may play a role in the increased mortality risk in young children with appendicitis.

16. What conditions must be considered in a teenage girl with symptoms of appendicitis?

Gynecologic conditions and emergencies should be considered in the differential diagnosis of any teenage girl with RLQ pain. In particular, ovarian cysts, corpus luteal cysts, mittelschmerz, tubal pregnancy, ovarian torsion, and salpingitis can all present similarly to appendicitis.

17. Your 10-year-old patient with presumed appendicitis is in extreme pain. His tearful mother is asking if there is anything you can give him to make him feel better. The surgeons won't be available to examine him for another hour. What should you do?

Provide pain relief for this patient! Studies in adults have shown that administration of opiates decreases self-reported pain scores but does not hide evidence of peritoneal irritation. Studies in children are limited by small numbers of patients and serial observations by the same examiners but suggest that opiate administration provides pain relief without adversely affecting the examination or the ability to diagnose children with surgical conditions.

Despite the growing evidence that opiate administration will not affect diagnostic accuracy, some surgeons still prefer to examine patients prior to administration of pain medication. It is important to respect the practice in your institution, while remaining aware of the current literature. Reassure your patient's mother that you will address the issue, and you should give your surgical colleagues a call to let them know your plan.

Kim MK, Galustyan S, Sato TT, et al: Analgesia for children with abdominal pain: A survey of pediatric emergency physicians and pediatric surgeons. Pediatrics 2003;111:1122-1126.

Kim MK, Strait RT, Sato TT, Hennes HM: A randomized clinical trial of analgesia in children with acute abdominal pain. Acad Emerg Med 2002;9:281-287.

Kokki H, Lintula H, Vanamo K, et al: Oxycodone vs placebo in children with undifferentiated abdominal pain. Arch Pediatr Adolesc Med 2005;159:320-325.

18. What is the most common cause of acute pancreatitis in children?

Abdominal trauma.

19. What are the radiographic findings in a child with acute mechanical bowel obstruction?

Multiple dilated loops of bowel and an air-fluid level visible in the upright or lateral decubitus views on plain films are common.

20. What is the most common cause of acute intestinal obstruction in a child between 3 and 12 months of age?

Intussusception. This occurs when a proximal segment of bowel telescopes into a distal segment. It is commonly ileocolic, but small bowel may also telescope into itself.

21. Define "lead point."

The lead point is the area that is thought to initiate the intussusceptum. The causes of lead points differ by age. In infants, lead points are often hypertrophied Peyer's patches. In older children, there are many possibilities for focal lead points, including intestinal polyps, Meckel's diverticulum, and a tumor.

22. What causes intussusception?

There is no definitive cause, though there is an association between intussusception and a history of a preceding viral illness, diarrhea, or, perhaps, Henoch-Schönlein purpura (HSP). The association between the rotavirus vaccine and increased frequency of intussusception led to the recall of the original rotavirus vaccine.

23. How does the child with intussusception present?

The classic history is that the patient has crampy abdominal pain. Because this is a disease that mostly occurs in preverbal children, the parent may give the history that the child behaves normally or is slightly irritable but has episodic bouts of severe abdominal pain characterized by drawing the knees to the chest, laying still, or screaming inconsolably. Some children may present with profound lethargy. Intussusception should be a strong consideration in the differential diagnosis of any infant or toddler presenting with lethargy. Vomiting may become bilious as the small bowel becomes completely obstructed. There may be a history of rectal bleeding or bloody stool. On physical examination, the child's abdomen may be distended due to the partial or complete obstruction of the bowel. You may be able to palpate a sausage-like mass, typically in the right upper quadrant (RUQ).

Lockhead A, Jamjoon R, Ratnapalan S: Intussusception in children presenting to the ED. Clin Pediatr 2013;52:1029.

Mandeville K, Chien M, Willyard PA, et al: Intussusception: Clinical presentations and imaging characteristics. Pediatr Emerg Care 2012;28:842.

Shah S: An update on common GI emergencies. Emerg Med Clin North Am 2013;31:775-793.

24. What is the origin of pain in intussusception?

Intussusception is the result of invagination of a portion of proximal intestine into distal intestine. The intussuscepted mass can obstruct the intestinal lumen. Abdominal pain is due to distension and peristaltic rushes against the mass.

25. If there is no abdominal tenderness on examination, can you dismiss intussusception in the differential diagnosis?

No. Because intussusception is a dynamic process, i.e., the bowel can telescope in and out, abdominal pain may be intermittent. The classic signs and symptoms of intussusception—abdominal pain, palpable abdominal mass, and red currant jelly stool—are found in fewer than 50% of cases.

Lockhead A, Jamjoon R, Ratnapalan S: Intussusception in children presenting to the ED. Clin Pediatr 2013;52:1029.

Mandeville K, Chien M, Willyard PA, et al: Intussusception: Clinical presentations and imaging characteristics. Pediatr Emerg Care 2012;28:842.

26. What is "currant jelly" stool?

Currant jelly is made from a small, round, acidic berry that may be black or red. This term is used to describe the reddish, heme-positive stool of mixed mucus and blood associated with intussusception. It occurs when the edematous bowel causes compression of the mesenteric veins. It is a late sign of intussusception indicating mucosal damage and is present in only about one third of patients. Do not rule out the possibility of intussusception in the absence of currant jelly stool.

27. How do you diagnose intussusception?

Have a high index of suspicion for lethargic infants and toddlers and take a detailed history and physical examination! If there is a suspicion for intussusception, obtain an obstruction series. Those with positive radiographs should have US or go directly to enema. Those with equivocal radiographs should have US, if available. US can unequivocally identify intussusception and may help to identify the leading edge of the intussusceptum, the presence of an air contrast, and the presence of blood flow within the intussusceptum. The next step is enema.

Weihmiller SN, Buonomo C, Bachur R: Risk stratification of children being evaluated for intussusception. Pediatrics 2011;127:e296-e303.

Key Points: Classic Findings in Intussusception

1. Intense, intermittent abdominal pain
2. Currant jelly stool
3. Irritability
4. Vomiting
5. Lethargy

28. Why is air contrast enema recommended?

Air enema is preferred in most pediatric institutions, though some still use near-isotonic water-soluble media. Barium should not be used because perforation during the examination can result in barium and fecal spillage in the peritoneal cavity. Other contrast media, such as Gastrografin, should not be used because of the high osmolality; perforation could result in acute hypovolemic shock due to rapid intravascular depletion. It is prudent to inform your surgical colleagues of the case prior to performing the contrast enema, owing to the risk of perforation of potentially gangrenous bowel.

Hadidi AT, El Shal N: Childhood intussusception: A comparative study of nonsurgical management. J Pediatr Surg 1999;34:304-307.

29. What is a positive finding of intussusception on a radiograph?

A positive finding on obstructive series includes small bowel obstruction or the target or crescent sign. The target sign is a mass in the RUQ that may have a targetlike appearance or may just resemble a solid mass. The crescent sign is caused by the lead point protruding into a gas-filled pocket, which may result in a crescent-shaped finding on a radiograph.

Raskind CG, Kandar G, Ruzal-Shapiro CB, et al: Accuracy of plain radiographs to exclude the diagnosis of intussusception. Pediatr Emerg Care 2012;28:855.

30. What is the only absolute contraindication to enema in the diagnosis of intussusception?

The presence of free air or signs of peritoneal irritation are the only absolute contraindications.

31. How do you treat intussusception?

This is a trick question, as the diagnostic test (enema) is usually therapeutic as well. Intussusception can be reduced by enema in about 85% of cases. If the child is toxic, however, he or she may require surgical intervention. Recurrence rates are 5% to 8%, regardless of the means of reduction. There is still debate about whether antibiotics are necessary prior to enema reduction of intussusception. Recent evidence indicates antibiotics are probably not helpful.

Al-Tokhais, Hsieh H, Pemberton J, et al: Antibiotic administration before enema reduction of intussusception: Is it necessary? J Pediatr Surg 2012;47:928.

32. How do you explain intussusception to the parents of the child you just diagnosed?

Visual descriptions are helpful for families. One way to explain it is the sock model. In other words, if you want turn a sock inside out, you pull the toe of the sock through the cuff. This is similar to what the bowel does.

33. Why are inguinal hernias usually repaired when detected in young infants?

There is a high risk of intestinal incarceration in male infants and adnexal entrapment in female infants. Hernias that reduce spontaneously are generally repaired electively soon after detection. Those that require sedation and manual reduction are usually repaired 24 to 48 hours after reduction.

34. Is an incarcerated hernia more common in girls or boys?

Contrary to what you might think, it is more common in girls and usually involves the ovary rather than the intestine.

35. What is the difference between an incarcerated and a strangulated hernia?

Incarceration means that the intestine or ovary is nonreducible, but not necessarily gangrenous. However, if the hernia is not reduced, it can become strangulated. Once strangulated, venous and lymphatic obstruction occur, leading to occlusion of arterial supply. This sets the stage for necrosis and possible perforation.

36. On examination of a 3-month-old child brought in for a minor complaint, you notice a large umbilical hernia. The mother asks you what she should do about it. What do you tell her?

Umbilical hernias are common in infants and young children, especially African Americans. Incarceration of an umbilical hernia is rare. Your best advice is to reassure the parents (and often the grandparents, aunts, and uncles) that most of these hernias reduce spontaneously. If there is a *large* ring that has not diminished by age 2, or if an incarceration has occurred, the defect should be closed operatively. Otherwise, the hernia can be closed electively at age 5 or 6.

Key Points: Which Test Is Best?

1. Appendicitis: CT (US and MRI are also useful)
2. Pyloric stenosis: Ultrasonography
3. Intussusception: Air contrast enema (many use US initially for diagnosis)
4. Malrotation: Upper gastrointestinal (GI) series

37. A 10-month-old previously well child presents to the emergency department (ED) with acute onset of abdominal pain associated with bilious vomiting. The child is afebrile and ill-appearing and has a slightly distended abdomen that is diffusely tender. The rectal examination reveals blood on the examiner's finger. On routine abdominal radiograph, the emergency physician notes air-fluid levels and a few dilated loops of bowel. The upright film shows a "double-bubble" sign. An upper GI study reveals absence of the ligament of Treitz. What should the clinician do next?

The physician should call a surgeon and reserve the operating room ASAP! The "double-bubble" sign on the upright abdominal radiograph represents partial obstruction of the duodenum, causing distention of the stomach and the first part of the duodenum. The absence of the ligament of Treitz on the upper GI study almost clinches the diagnosis (malrotation of the bowel with volvulus), especially if it is associated with finding the duodenum to the right of the spine and a coiled appearance of the jejunum in the RUQ. *This is a true surgical emergency.*

38. What is the most common surgically correctable cause of vomiting in infants?

Pyloric stenosis. This condition is estimated to occur in about 1 in 400 births.

39. What is pyloric stenosis? How does it present?

Pyloric stenosis is a narrowing of the outflow tract of the stomach due to hypertrophy of the pyloric musculature. Pyloric stenosis is often found among first-born males, and males predominate by a factor of 4 to 5. There may be a family history for this condition. The infants are usually clinically normal at birth. In the typical history, the infant does well for the first few weeks of life, usually regaining birth weight. At about the third week of life, the infant starts to vomit, usually at the end of feedings. The vomiting eventually becomes projectile or "shoots across the room." It is typically nonbilious, though blood may be seen if there is an associated gastritis or esophagitis. Most patients present between 2 and 6 weeks of age. The infant is often described as hungry all the time, eating even after vomiting, because the infant cannot achieve adequate nutrition. In time, the infant may become profoundly dehydrated and emaciated.

Markowitz RI: Olive without a cause: The story of infantile hypertrophic pyloric stenosis. Pediatr Radiol 2014;44:202-211.

40. How do you examine an infant with suspected pyloric stenosis?

Examine the infant on his or her back, preferably on the parent's lap, and while he or she is quiet. Hold the infant's legs and flex them to 90 degrees at the hips to help relax the abdominal musculature. A bit of sugar or juice on a pacifier may help to relax the child even more. Begin the examination from below the liver, palpating in a rocking motion. A firm, ballotable mass may be felt in the region of the pylorus. This is the classic physical finding of pyloric stenosis—*the "olive."* If you cannot palpate an olive and still have a high suspicion for pyloric stenosis, US is the test of choice.

Markowitz RI: Olive without a cause: The story of infantile hypertrophic pyloric stenosis. Pediatr Radiol 2014;44:202-211.

41. Where is the "olive" usually found?

Usually it is found on abdominal examination to the right, just below the xiphoid.

42. What US findings are associated with pyloric stenosis?

Upper GI tract examination has long been used to diagnose pyloric stenosis. The diagnosis is made by documentation of delayed gastric emptying and visualization of a long pyloric channel. You cannot see the hypertrophied pylorus muscle. US, however, is quickly becoming the favored radiologic test for diagnosing pyloric stenosis. The reasons are that there is no radiation and no need to introduce barium into the GI tract of a potential surgical candidate who is at high risk for aspiration. Additionally, US allows visualization of the hypertrophied pylorus muscle. If the pyloric canal lengthening is greater than 16 mm and the wall thickness is more than 4 mm, suspect pyloric stenosis.

Sivitz AB, Tejani C, Cohen SG: Evaluation of hypertrophic pyloric stenosis by pediatric emergency physician sonography. Acad Emerg Med 2013;20:646-651.

43. What is the classic laboratory finding in a child with pyloric stenosis?

The classic finding is hypochloremic, hypokalemic metabolic alkalosis. Serum bicarbonate may be as high as 65 to 75 mEq/L. Acidosis usually signifies a more dangerous metabolic state due to extreme dehydration.

44. How does the metabolic derangement occur?

The metabolic alkalosis is due to loss of acid and retention of bicarbonate. Several mechanisms play a part in achieving this. Frequent vomiting leads to depletion of potassium chloride and hydrogen chloride. (Normally, H_2CO_3 [carbonic acid] in the villi of the stomach dissociates into H^+ and HCO_3^-. The hydrogen ions cross into the enterocyte and are transported from the stomach to the duodenum, where the entry of acid stimulates secretion of an equal amount of bicarbonate. This normal process is lost in pyloric stenosis because of the mechanical obstruction, and the decreased secretion of pancreatic HCO_3^- into the GI tract contributes further to the metabolic alkalosis created by the stomach acid lost through vomiting.) Additionally, as intravascular volume decreases with prolonged vomiting and dehydration, the concentration of HCO_3^- in the plasma increases, resulting in a contraction alkalosis. As plasma potassium levels fall due to gastric losses, potassium moves out of the cells to restore extracellular concentrations. Hydrogen ions then move into cells to maintain electroneutrality. The net result is a metabolic alkalosis. Although the serum potassium level may be normal or low, total body potassium is often depleted.

However, this is only part of the picture. The kidneys also play a role in the development of metabolic alkalosis. To maximize intravascular volume in the face of ongoing gastric losses, the kidneys resorb bicarbonate in the distal tubules despite alkalosis. If excess bicarbonate is excreted in the urine, it obligates sodium loss (think electroneutrality again). Remember that water follows sodium, which would result in further volume loss, and that a lot of chloride has been lost through vomiting. Decreased delivery of chloride to the macula densa of the kidneys results in renin release and secondary hyperaldosteronism, leading to increased distal hydrogen secretion and the paradoxical finding of aciduria in the presence of a metabolic alkalosis. Finally, in response to the total body depletion of potassium, the distal tubules reabsorb potassium in exchange for hydrogen, leading to further acid loss.

Dinkevich E, Ozuah PO: Pyloric stenosis. Pediatr Rev 2000;21(7):249-250.

Key Points: Findings That Require Immediate Surgical Consultation

1. Strangulated hernia on physical examination
2. Distended abdomen and "double-bubble" sign on upright abdominal radiograph
3. Bilious emesis, especially in the first few months of life
4. Absence of the ligament of Treitz on upper GI study
5. Ultrasonography findings consistent with ovarian torsion

45. How is pyloric stenosis treated?

Initially, correct the infant's fluid and electrolyte status with intravenous fluids. Once the infant is euvolemic and the electrolytes have returned to normal, a surgeon should perform a *pyloromyotomy*.

46. What are the most common surgically correctable causes of vomiting in the following infant age groups: First week of life? First month of life? After the neonatal period?

In the first week of life, consider anatomic malformations. Esophageal atresia, duodenal or jejunal atresia, duodenal stenosis, midgut malrotation, ileal atresia, and meconium ileus are the most common causes.

In the first month of life, consider pyloric stenosis and gastroesophageal reflux. As a general rule, gastroesophageal reflux can be managed medically. However, if medical management fails and the infant is failing to gain weight, bleeding, or aspirating, a surgical antireflux operation, such as a fundoplication, may be warranted. Other causes of vomiting in babies this age include Hirschsprung's disease, esophageal or intestinal webs, infection, increased intracranial pressure, and metabolic defects.

Beyond the neonatal period, consider intussusception, appendicitis, and bowel obstruction caused by malrotation, incarcerated or strangulated hernia, duplication cysts, or Meckel's diverticulum.

Moir CR: Abdominal pain in infants and children. Mayo Clin Proc 1996;71:984-989.

47. What are the common causes of rectal bleeding in the pediatric age group? How do they present?

- **Fissures**. Fissures are probably the most common cause. Often there is a history of constipation or passing a large, hard stool. The blood typically is bright red and found in streaks on the outside of the stool or on the toilet tissue. The diagnosis can be made by anal examination under a good light source. Treatment consists of sitz baths and lubrication of the rectal area with petroleum jelly. If the child suffers from constipation, address this as well.
- **Juvenile polyps**. Polyps occur in older infants and children in the lower part of the colon. They may be palpated on digital rectal examination and may bleed, especially if they break free. They are not premalignant, but they may serve as a lead point for intussusception.
- **Meckel's diverticulum**. Remember the rule of 2's! Two percent of the population is born with a Meckel's diverticulum. It is usually located about 2 feet proximal to the terminal ileum. And only 2% of people with a Meckel's diverticulum have any clinical problems. Meckel's diverticuli usually contain ectopic gastric mucosa, and the acid secretion produces erosion at the junction of the normal ileal mucosa and the Meckel's mucosa. It may present with painless rectal bleeding, perforation with peritonitis, diverticulitis, or intussusception.
- **Henoch-Schönlein purpura**. This vasculitis can cause symptoms ranging from painless rectal bleeding to abdominal pain and hematuria. The associated submucosal hemorrhage may also serve as a lead point for intussusception.
- **Other causes**. Intestinal vascular malformations, intussusception, inflammatory bowel disease, duplications, swallowed blood, bleeding peptic ulcer disease, bleeding varices, and trauma can cause rectal bleeding.

48. A 3-year-old child swallowed a safety pin that she found on the floor. You have located the object in her stomach on plain films, and the pin appears to be open. What do you do now?

Most foreign bodies that reach the stomach will pass completely through the GI tract and be evacuated in the stool. Even an open safety pin may be allowed to pass. It is advisable to repeat an abdominal radiograph in about 5 days to be sure the pin has moved. Occasionally, a foreign body may get stuck at the junction of the duodenum and jejunum at the ligament of Trietz. These are usually long, thin objects such as bobby pins or long nails. Because perforation may occur, surgical removal of objects that "catch" beyond the pylorus should be arranged. Occasionally, objects get caught in the appendix, necessitating appendectomy. Obtain a radiograph in all children who have a history of swallowing a foreign body, both for the already mentioned reasons and to make sure that it is not lodged above the level of the thoracic inlet, where it could be aspirated if the child coughs. Rates of nonoperative intervention vary from 10% to 20% across all age groups. Less than 1% require surgery.

American Society for Gastrointestinal Endoscopy: Guideline for the management of ingested foreign bodies. Gastrointest Endosc 2002;55(7):802-806.

49. Two months later the same 3-year-old swallows a button battery from a toy. Should this child be managed differently?

Button batteries present a special hazard. The 3-volt lithium batteries have an increased risk over alkaline batteries. The problem occurs with a reaction to the anode surface causing discharge of current, which creates tissue erosion and injury. Batteries in the esophagus can cause damage in 2 hours. Batteries with a 20 mm diameter are most frequently involved in injury. Obtain radiographs of the abdomen, esophagus, and neck. Batteries above the range of the radiograph have been missed. If battery diameter is unknown, estimate it from the radiograph, factoring out magnification (which tends to overestimate diameter). In general, all button batteries in the esophagus need immediate removal. If the battery is located in the stomach and the child is asymptomatic, the patient may be followed with radiographs every day to ensure passage. Management and identification help is available by consulting the National Battery Ingestion Hotline at 202-625-3333 or www.poison.org/battery/guideline.asp.

Dawe N, Puvanendran M, Flood L: Unwitnessed lithium ion disc battery ingestion: Case report and review of best practice management of an increasing clinical concern. J Laryngol Otol 2013;127:84-87.

Litovitz T, Whitaker N, Clark L, et al: Emerging battery-ingestion hazard: Clinical implications. Pediatrics 2010;125:1168-1177.

Sharpe SJ, Rochette LM, Smith GA: Pediatric battery-related emergency department visits in the United States, 1990-2009. Pediatrics 2012;129(6):1111-1117.

50. Does the ingestion of magnets cause serious problems? Do they need to be retrieved urgently?

The ingestion of multiple magnets can cause significant complications. The strong attraction force of the magnets can occur across the mucosal folds of the stomach or between loops of bowel, leading to bowel perforation, volvulus, ischemia of the bowel wall, and death. It may be difficult to determine if attached magnets are across the bowel wall. Surgical consultation is required.

Centers for Disease Control and Prevention: Gastrointestinal injuries from magnet ingestion in children—United States, 2003-2006. MMWR Morb Mortal Wkly Rep 2006;55(48):1296-1300.

Dutta S, Barzin A: Multiple magnet ingestion as a source of severe gastrointestinal complications requiring surgical intervention. Arch Pediatr Adolesc Med 2008;162(2):123-125.

Lee JH, Lee JS, Kim MJ, Choe YH: Initial location determines spontaneous passage of foreign bodies from the gastrointestinal tract in children. Pediatr Emerg Care 2011;27(4):284-289.

51. What is a vascular ring?

A vascular ring is a congenital condition that causes airway or esophageal obstruction. The obstruction is usually at the level of the trachea, but a ductus arteriosus or a pulmonary artery sling can also compress the bronchi. Many of these rings are caused by failure of involution of various segments of the six embryologic aortic arches. Infants who present with stridor, recurrent pneumonia, or a history of noisy breathing since birth should prompt consideration of this diagnosis.

52. What is the difference between a pilonidal dimple and a pilonidal sinus?

Both are located in the midline in the sacrococcygeal area. Close inspection reveals that the dimple does not have a *central pore*, but the sinus does. The sinus is a tract lined by stratified squamous epithelium that extends toward the spinal canal, but not into it. The sinus is asymptomatic until it becomes infected or obstructed, usually in adolescence. Predisposing factors are male gender, being overweight or hirsute, and a sedentary lifestyle. Once infection occurs, an abscess forms and expands deep to the skin surface. Patients usually complain of low back pain that is worse with sitting. They may also complain of localized tenderness. On examination, you will note a tender, indurated area in the sacrococcygeal region.

53. How is a pilonidal abscess treated?

Because these lesions typically expand inward, they must be incised and drained. Probe the abscess cavity to break up any loculations, and remove hair because it acts as a foreign body. Following incision and drainage, begin sitz baths and oral antimicrobial therapy (targeting staphylococcus, anaerobes, and fecal flora). Once the inflammation has resolved, arrange elective incision of the entire cyst and its sinus tracts.

54. What is omphalitis?

Omphalitis is an infection of a newborn's umbilical cord stump and the surrounding tissues, usually occurring in the first 2 weeks of life. Often, the infant initially presents with purulent, foul-smelling drainage from the umbilical stump and surrounding abdominal erythema, indicative of cellulitis. If the infection is not diagnosed and treated early, it can lead to more serious problems, such as peritonitis, liver abscess, or sepsis. The usual bacterial pathogens are *Streptococcus pyogenes*, *Staphylococcus aureus*, group B streptococcus, and gram-negative rods.

55. How is omphalitis treated?

If the findings are not clearly suggestive of omphalitis, i.e., there is minimal drainage or erythema, the infant can be treated at home with good skin/cord care (cleaning the cord with each diaper change and using topical antibiotics). Reevaluation in 24 hours is recommended. Instruct parents to return for reexamination if the child has a change in feeding, activity, or disposition. If the infant appears toxic or has obvious signs of omphalitis, presume a serious systemic infection. In this case, obtain appropriate laboratory evaluation for sepsis, admit, and treat with intravenous antibiotics.

CHAPTER 45

NEUROSURGICAL EMERGENCIES

Jillian Stevens Savage and Colette Mull

1. What is the basic pathophysiology of increased intracranial pressure (ICP)?

The cranium functions as a rigid compartment housing mainly brain tissue but also blood and cerebrospinal fluid (CSF). Based on the Monro-Kelli doctrine, any abnormal increase in volume of one of these components, without a corresponding decrease in one or both of the others, will result in increased ICP. In a healthy individual, the ICP will remain relatively stable with minor changes in volume. Once a critical ICP is reached, it becomes significantly more sensitive to even the smallest increase in volume.

Greenes DS: Neurotrauma. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 1423-1427.

2. How does increased ICP lead to cerebral ischemia?

Cell survival depends on oxygen delivery through adequate cerebral blood flow (CBF). Cerebral perfusion pressure (CPP) is the pressure gradient that generates CBF, thereby perfusing the brain. CPP is influenced by changes in mean arterial pressure (MAP) and ICP (CPP = MAP − ICP). CPP maintains adequate CBF only when the MAP is greater than 60 mm Hg and the ICP is less than 40 mm Hg. Outside these limits, CBF drops, resulting in suboptimal oxygen delivery, cell injury, and brain ischemia.

Greenes DS: Neurotrauma. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 1423-1427.

Kochanek PM: Guidelines for the acute medical management of severe traumatic brain injury in infants, children and adolescents. Chapter 5. Cerebral perfusion pressure thresholds. Pediatr Crit Care Med 2012;13(1):24-25.

3. What are the symptoms of increased ICP?

Headache is the most common early symptom of increased ICP. If left untreated, increased ICP leads to intellectual impairment, stiff neck, diplopia, vision loss, irritability, vomiting, and death.

4. What are the signs of increased ICP?

Cushing's triad—bradycardia, systemic hypertension, and irregular respirations—is highly suggestive of increased ICP. Skull percussion may elicit a *Macewen's sign*, which is a hyperresonant note thought to mimic the sound made by percussing a "cracked pot." The fontanel may be full or bulging. On ophthalmologic examination, papilledema, optic nerve atrophy, or retinal hemorrhages may be appreciated. Look for cranial nerve (CN) deficits; specifically, deficits of CN IV and CN VI produce head tilt and sunset eyes, respectively. Additional neurologic sequelae of increased ICP include change in mental status, hemiparesis, decorticate or decerebrate posturing, and meningismus. Be aware that a normal examination does not reliably rule out increased ICP; a strong clinical suspicion warrants inpatient observation with serial examinations.

Steele DW: Neurosurgical emergencies, nontraumatic. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 1587-1588.

5. What are the causes of increased ICP?

- Increased brain volume, or cerebral edema
  - Vasogenic causes: Tumor, abscess, hemorrhage

  - Cytotoxic causes: Hypoxia, ischemia, infections
  - Interstitial causes: Blockages in CSF absorption, shunt malfunction
- Increased CSF, or hydrocephalus
  - Congenital causes: Dandy-Walker cyst, Arnold-Chiari malformation, vein of Galen arteriovenous malformation
  - Acquired causes: Meningitis, tumor, leukemia, inflammatory response to hemorrhage
- Increased blood volume caused by trauma leading to space-occupying intracranial bleeding: Subdural or epidural hematoma
- Idiopathic: Idiopathic intracranial hypertension (pseudotumor cerebri)

Allen C, Ward J: An evidence based approach to management of increased intracranial pressure. Crit Care Clin 1998;14(3):485-486.

6. Describe the initial management of increased ICP.

- Emergent stabilization of circulation, airway, and breathing
- Prevention of hypoxemia, hypercarbia, and systemic hypotension
- Immobilization of the cervical spine in patients with suspected cervical spine trauma or unknown cause of increased ICP

Steele DW: Neurosurgical emergencies, nontraumatic. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 1587-1588.

7. What are the benefits and drawbacks of intubation in a patient with increased ICP?

- Early endotracheal intubation allows for airway maintenance, protection from aspiration, maximal oxygenation, and control over ventilation.
- Laryngoscopy alone can cause hypertension, bradycardia, and increased ICP. Rapid sequence intubation (RSI) can help prevent these side effects.

8. Discuss rapid sequence intubation in patients with increased ICP.

Each patient requires an intubation plan, easily recalled by the mnemonic SOAPEM:

- **S**taff: Assign health care providers roles according to their skills.
- **O**xygen
- **A**irway equipment
- **P**harmacologic agents

Lidocaine has traditionally been used to prevent the vagal responses of ICP associated with laryngoscopy and intubation. Evidence on the efficacy of lidocaine is limited and primarily in the adult literature. However, the decision to use this agent is still left to the clinician directing the intubation of the patient.

The choice of sedative in the setting of increased ICP is based on cardiovascular status. If the patient is hemodynamically stable, thiopental is an excellent induction agent. *Etomidate* is also commonly used, but be aware of the potential for adrenal suppression, even with one dose. In patients with hemodynamic instability, *midazolam* is a safe sedative choice. *Ketamine* is not ideal in the setting of increased ICP, as it may increase CBF.

- **E**nd-tidal CO_2 device
- **M**onitors

Thompson A: Airway management. In Fuhrman B, Zimmerman J (eds): Pediatric Critical Care, 4th ed. Philadelphia, Elsevier, 2011, pp 1607-1608.

Yamamoto L: Emergency airway management—rapid sequence intubation. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 74-80.

9. What nonpharmacologic treatments should be considered in the management of increased ICP?

- **Elevate the head of the bed** to 15 to 30 degrees to lower ICP without compromising CPP. Keep the patient's head in the midline position to promote venous drainage.

- **Hyperventilation** is recommended only for impending herniation. Hyperventilation generates cerebral vascular constriction, which in turn lowers CBF and therefore may lead to cerebral ischemia. In nonemergent situations, maintain the $Paco_2$ at 35 to 38 mm Hg.
- Seek **early neurosurgery consultation**. Consider CSF drainage in cases of increased ICP refractory to pharmacologic treatment.

Steele DW: Neurosurgical emergencies, nontraumatic. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 1587-1593.

10. What pharmacologic treatments are helpful in the management of increased ICP?

- **Mannitol** reduces ICP by two mechanisms. First, it immediately but transiently reduces blood viscosity. Onset of effect is rapid (1-5 minutes) and peak of effect ranges from 20 to 60 minutes after administration. Second, mannitol acts more slowly as an osmotic diuretic with onset of effect in 15 to 30 minutes and duration of effect up to 6 hours. It is contraindicated in patients with hypovolemia. Use it judiciously in patients with renal insufficiency.
- **Hypertonic saline** is another hyperosmolar treatment option. This medication is favored over mannitol in hypovolemic patients, as its diuretic properties are more attenuated. Other benefits include increased cardiac output and anti-inflammatory effects. Cautious use is advocated in patients with renal insufficiency.
- **Corticosteroids** are not indicated to treat increased ICP in the setting of infarction, hemorrhage, or head trauma, as multiple studies have been unable to show benefit from therapy. However, in the setting of vasogenic edema from mass effect (e.g., abscess or tumor), steroids may be indicated for an anti-inflammatory effect.
- Use **sedation, antipyretics, and analgesics** aggressively if agitation, fever, and pain are present. All three states increase cerebral metabolism, which in turn increases CBF and ICP. See prior discussion on sedative choice in the setting of increased ICP.
- **Muscle relaxants are not used** routinely because continuous neurologic assessment is imperative. Use only if the patient is shivering, which again increases cerebral metabolism, CBF, and ultimately ICP.
- **Antiepileptics** may be indicated in patients who are at increased risk for seizure in order to prevent an increase in ICP associated with seizure activity. Phenytoin and phenobarbital are typically used in the setting of severe traumatic injuries or significant cerebral parenchymal abnormalities. Benzodiazepines are recommended in the setting of breakthrough seizures.
- **Barbiturate coma** is a therapeutic modality used in patients with increased ICP refractory to conventional nonpharmacologic and pharmacologic treatments. By decreasing cerebral metabolism, this state decreases ICP. Barbiturates can depress cardiac function and cause hypotension. Judicious fluid management is indicated.

Kochanek PM: Guidelines for the acute medical management of severe traumatic brain injury in infants, children and adolescents. Chapter 8. Hyperosmolar therapy. Pediatr Crit Care Med 2012;13 (1):36-38.

Kochanek PM: Guidelines for the acute medical management of severe traumatic brain injury in infants, children and adolescents. Chapter 14. Corticosteroids. Pediatr Crit Care Med 2012;13 (1):61-63.

Steele DW: Neurosurgical emergencies, nontraumatic. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 1587-1588.

11. What imaging modalities can be utilized in the evaluation of increased ICP?

- Computed tomography (CT) is indicated for emergent detection of intracranial injury and masses.
- Magnetic resonance imaging (MRI) is considered superior to CT in detecting intracranial injury and is the imaging modality of choice if the patient is stable.

Kochanek PM: Guidelines for the acute medical management of severe traumatic brain injury in infants, children and adolescents. Chapter 7. Neuroimaging. Pediatr Crit Care Med 2012;13 (1):33-34.

Key Points: Increased Intracranial Pressure

1. Regardless of the cause, the first and most important step in the treatment of increased ICP is maintenance of the child's circulation, airway, and breathing.
2. Rapid sequence induction and endotracheal intubation allow for airway protection. Rapid sequence induction medications will limit further ICP elevation during intubation.
3. Reserve hyperventilation for cases of impending herniation.
4. Early consultation with the neurosurgery service is imperative.

12. Define hydrocephalus.

Hydrocephalus is defined as the presence of an increased volume of CSF with resultant ventricular dilatation, typically secondary to an imbalance between production and absorption of CSF. Although a new diagnosis of hydrocephalus is rarely made in the emergency department (ED), patients with hydrocephalus who have been treated with the placement of a ventricular shunt will often present with signs and symptoms of shunt complications and require immediate attention.

Garton HJ, Piatt JH: Hydrocephalus. Pediatr Clin North Am 2004;51:305-325.

13. What types of treatments are used for hydrocephalus?

- Benign and stable hydrocephalus does not need treatment. Ventricular shunts remain the gold standard for the treatment of progressive hydrocephalus. Most shunts have three components: a ventricular catheter, a unidirectional valve (including a reservoir system), and a distal catheter. The distal catheter is most commonly placed such that it drains into the peritoneum, but in select cases it will be placed to drain into the right atrium or lung pleura.
- Endoscopic third ventriculostomy (ETV) is a treatment option recently developed as an alternative to shunt placement. In this procedure, a small hole is made in the floor of the third ventricle, allowing continuous drainage of CSF into the basal cisterns, bypassing the obstructed or stenosed aqueduct of Sylvius. As there is no indwelling catheter, this procedure limits long-term complications, specifically infection. Short-term complications of malfunction are still possible, and approximately 30% of patients will ultimately still require shunt placement.

Kulkarni AV: Endoscopic third ventriculostomy in the treatment of childhood hydrocephalus. J Pediatr 2009;155(2):254-255.

14. What are the potential complications of CSF shunt placement in pediatric patients?

- **Shunt malfunction** is the most common complication of shunt placement. This is often a problem of decreased drainage due to obstruction, but increased drainage can also occur. The shunt can also malfunction secondary to valve dysfunction, catheter disconnection, or catheter migration.
- **Shunt infection** occurs in 5% to 10% of shunt placements, usually within the first 6 postoperative months. Eighty percent of infections occur within the first 3 months and 90% occur within the first 6 months after shunt placement.
- **Other complications** include scrotal or inguinal migration, small bowel obstruction, intussusception, omental cyst torsion, persistent hiccup, abdominal CSF collection or pseudocyst, volvulus, colon perforation, shunt nephritis, diaphragm perforation, intra-abdominal organ perforation, and subdural hemorrhage (from increased drainage and tearing of bridging veins or as a surgical complication).

Gerber JS, Zaoutis TE: Clinical syndromes of device-associated infections. In Long SS (ed): Principles and Practice of Pediatric Infectious Disease, 4th ed. Philadelphia, Elsevier, 2012, pp 101-102.

Piatt JH, Garton HJ: Clinical diagnosis of ventriculoperitoneal shunt failure among children with hydrocephalus. Pediatr Emerg Care 2008;24(4):201-210.

Prusseit J, Simon M, Brelie C, et al: Epidemiology, prevention and management of ventriculoperitoneal shunt infections in children. Pediatr Neurosurg 2009;45:325-333.

15. How common is shunt failure?

Mechanical shunt failure or shunt failure from an infection is quite common, occurring in up to 35% to 40% of shunt placements within the first postoperative year. Fifteen percent

will fail in the second postoperative year. Although rates decrease to less than 7% thereafter, failure remains a risk as long as the shunt is in place. Unfortunately, the lifetime risk of complication is approximately 60%.

Fein JA, Cronan KM, Posner JC: Approach to the care of the technology-assisted child. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 1498-1501.

Garton HJ, Piatt JH: Hydrocephalus. Pediatr Clin North Am 2004;51:305-325.

16. What imaging studies may be utilized in the evaluation of shunt malfunction?

- **SSFSE MRI** (single-shot fast spin-echo MRI), also referred to as "fast spin MRI," is increasingly being used over CT scan as the imaging method of choice to assess shunt position and the size of fluid-filled structures (e.g., ventricles). It minimizes radiation and scanning time, and it eliminates the need for sedation or anesthesia in young or behaviorally challenged children. Proponents of the technology also laud its ability to detect vascular anomalies missed by other imaging modalities. However, some researchers caution that poor contrast resolution and the use of only one type of pulse sequence may limit the detection of bleeding and of abnormalities such as venous sinus thrombosis. Research into refining SSFSE MRI protocols to maximize clinical yield is in progress. In the interim, be aware of the limitations of this newer imaging modality.
- **Noncontrast head CT** may be utilized when MRI is unavailable. CT shows brain anatomy, ventricle size, and shunt position. The availability of previous head CT scans is important for comparison purposes. It requires the use of sedation or anesthesia in young or behaviorally challenged children.
- **Shunt survey**, the plain radiography of the skull, neck, thorax, and abdomen, can be used to evaluate the integrity of the entire length of the shunt. This is an infrequently ordered study but may be utilized to supplement MRI or CT in the assessment of shunt connectivity.
- **Ultrasound** of the abdomen should be employed for patients with ventriculoperitoneal (VP) shunts and abdominal pain to evaluate for the presence of a pseudocyst.

Iskander BJ, Sansone JM, Medow J, Rowley HA: The use of quick-brain magnetic resonance imaging in the evaluation of shunt-treated hydrocephalus. J Neurosurg 2004;101(2 Suppl):147-151.

Rozovsky K, Ventureyra ECG, Miller E: Fast-brain MRI in children is quick, without sedation, and radiation-free, but beware of limitations. J Clin Neurosci 2013;20:400-405.

Steele DW: Neurosurgical emergencies, nontraumatic. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 1588-1589.

17. What is the treatment of CSF shunt malfunction?

Definitive treatment of shunt malfunction is surgical shunt revision. Temporizing bedside procedures include CSF shunt tap (effective for distal obstruction only) and burr hole puncture (effective for proximal obstruction refractory to medical management and for life-threatening symptoms). Temporizing medical management includes acetazolamide, dexamethasone, and hyperventilation, with the latter only in unstable patients.

Fein JA, Cronan KM, Posner JC: Approach to the care of the technology-assisted child. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 1499-1501.

18. List the symptoms, signs, and sites of shunt infection.

Symptoms of shunt infection are nonspecific and may include lethargy, irritability, nausea, vomiting, feeding difficulties, seizure, headache, and visual disturbances.

Signs include fever, cellulitis overlying the shunt reservoir or track, bulging fontanel, increased head circumference, papilledema, Macewen's sign, sunsetting eyes (sclera is visible between upper eyelid and iris), and abdominal peritoneal signs. Meningismus may be absent. Ventriculoatrial (VA) shunt infection may present with clinical evidence of the following complications: septic pulmonary emboli, pulmonary hypertension, and infective endocarditis. Classic signs and symptoms of VP shunt infection may be absent in patients with VA shunts. Pleural shunt infection may present with shortness of breath and chest pain from pleural fluid collection.

Sites of infection include skin and subcutaneous tissue overlying the shunt, the proximal surgical site, the shunt lumen, the ventricles, and the distal surgical site. Abdominal pseudocysts can result from infection or become secondarily infected.

Prusseit J, Simon M, Brelie C, et al: Epidemiology, prevention and management of ventriculoperitoneal shunt infections in children. Pediatr Neurosurg 2009;45:325-333.

19. What are the typical CSF findings in shunt infection?

Although CSF pleocytosis may be secondary to early postoperative inflammation, it is often present in the setting of shunt infections. Some evidence suggests that the combination of fever and CSF neutrophilia (defined as greater than 10% neutrophils) is highly specific (99%) and has high positive and negative predictive values, 93% and 95%, respectively, for detecting or excluding shunt infection.

McClinton D: Predictors of ventriculoperitoneal shunt pathology. Pediatr Infect Dis J 2001;20:593-595.
Prusseit J, Simon M, Brelie C, et al: Epidemiology, prevention and management of ventriculoperitoneal shunt infections in children. Pediatr Neurosurg 2009;45:325-333.

20. Describe CSF and serum culture results in patients with shunt infections.

Obtain CSF carefully, from the shunt reservoir, with the assistance of a neurosurgeon, as it is directly connected to the ventricular system. CSF sent for culture from lumbar puncture will likely remain sterile even in the face of known ventriculitis.

Blood cultures in patients with VP infections are rarely positive, as the shunt is not directly connected with the vascular system. However, in patients with VA shunt infections, bacteremia is commonly seen.

Flynn P: Infection associated with medical devices. In Kliegman RM, Stanton BF, St Geme JW III, et al (eds): Nelson Textbook of Pediatrics, 19th ed. Philadelphia, Elsevier, 2011, pp 903.

21. Which organisms cause shunt infections?

Most shunt infections are thought to result from intraoperative contamination by skin flora. Therefore, *Staphylococcus epidermidis* and *Staphylococcus aureus* (including methicillin-resistant strains) account for approximately 70% of infections and are the most likely pathogens in the early postoperative period.

Approximately 6 months after shunt placement, infections are typically caused by gram-negative pathogens including *Pseudomonas aeruginosa* and *Haemophilus influenzae*. They account for about 15% of shunt-related infections. Infections are associated with complications including bowel erosion, pressure necrosis from the shunt hardware, ascending infection from the distal shunt, and translocation of bacteria across the bowel wall.

Fein JA, Cronan KM, Posner JC: Approach to the care of the technology-assisted child. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 1499-1501.
Flynn P: Infection associated with medical devices. In Kliegman RM, Stanton BF, St Geme JW III, et al (eds): Nelson Textbook of Pediatrics, 19th ed. Philadelphia, Elsevier; 2011, pp 903.

22. What is the treatment of shunt infection?

Neurosurgical and infectious disease resources recommend combined surgical and medical treatment. Seek early consultation with a neurosurgeon, as the infected device may need to be removed or at least temporarily externalized until CSF cultures are negative and the patient shows clinical improvement.

Treat the infection empirically with broad-spectrum antibiotics, which can later be tailored when susceptibilities are available.

1. Treat with an antistaphylococcal antibiotic with good CSF penetration, e.g., vancomycin.
2. If examination of the abdomen is not normal, suspect a gram-negative organism infection, and add a broad-spectrum cephalosporin, e.g., add ceftazidime or cefepime to the antibiotic regimen.

Fein JA, Cronan KM, Posner JC: Approach to the care of the technology-assisted child. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 1501-1502.

Gerber JS, Zaoutis TE: Clinical syndromes of device-associated infections. In Long SS (ed): Principles and Practice of Pediatric Infectious Diseases, 4th ed. Philadelphia, Elsevier, 2012, pp 101-102.

23. What are some historical clues to identifying a patient with a brain tumor?

Brain cancers are the most common solid tumor of childhood and second most common pediatric cancer overall, making up about 25% of all childhood neoplasms. Early on, children with brain tumors present with nonspecific complaints, often delaying their diagnosis. Complaints include irritability, listlessness, failure to thrive, behavioral disturbances, vomiting, and anorexia. Later, these children have more classic symptoms of a space-occupying intracranial mass. Historical clues that should prompt the clinician to obtain neuroimaging include vomiting (especially without fever, abdominal pain, or alteration in stooling pattern), persistent headache, vision problems (blurred, loss, diplopic), loss of developmental milestones, seizures, neuroendocrine dysfunction (e.g., new-onset diabetes insipidus), motor weakness, sensory change(s), neck pain, and torticollis.

Fleming AJ: Brain tumors in children. Curr Probl Pediatr Adolesc Health Care 2012;42:80-85.

24. Headache is a very common pediatric symptom. What are the historical features of headache that should prompt concern for brain tumor?

Headaches concerning for brain tumors are typically recurrent, occur in the morning, awaken a child from sleep, are intense, last long, and become incapacitating. They are typically associated with vomiting, and they are exacerbated by bending, coughing, laughing, and the Valsalva maneuver. Of particular concern are headaches in the occipital region.

Fleming AJ: Brain tumors in children. Curr Probl Pediatr Adolesc Health Care 2012;42:80-85.

Key Points: Characteristics of Brain Tumor Headaches

1. Recurrent morning headaches
2. Prolonged incapacitating headaches
3. Changes in headache quality, frequency, and pattern

25. What clues from the physical examination suggest a brain tumor?

Cranial nerve deficits, visual abnormalities (loss of visual acuity, visual field loss, afferent papillary defect, or nystagmus), ataxia, loss of coordination or balance, and upper motor neuron signs. The latter include hyperreflexia, clonus, paraparesis, weakness, decreased or abnormal sensation, funduscopic evidence of increased ICP, and macrocephaly. These are important clues to recognize, and any one or more of them warrant investigation.

Steele DW: Neurosurgical emergencies, nontraumatic. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 1587-1593.

26. Which two specific syndromes should raise concern for intracranial disease?

- **Parinaud syndrome** suggests the presence of a tumor in the pineal region. Symptoms include vertical gaze limitation, pupillary dilatation reactive to accommodation but not to light, convergence-retraction nystagmus (on attempt of upward gaze, eyes move medially and globes retract), and eyelid retraction.
- **Diencephalic syndrome** raises concern for a tumor in the suprasellar or third ventricular region (diencephalon), typically involving the hypothalamus and thalmus. Symptoms include failure to thrive with emaciation, euphoria, and emesis.

Fleming AJ: Brain tumors in children. Curr Probl Pediatr Adolesc Health Care 2012;42:80-85.

27. What are the predisposing factors that may lead to brain abscess?

There are two pathophysiologic mechanisms behind the development of a brain abscess. Hematogenous spread of infection is the most common mechanism in children. Predisposing conditions or factors include cyanotic congenital heart disease (up to 6%),

endocarditis (especially left-sided), jugular venous thrombophlebitis associated with parapharyngeal infection (Lemierre's syndrome), pulmonary arteriovenous malformation, cystic fibrosis, and recent endoscopy leading to spinal venous plexus bacterial infection.

Less commonly, a brain abscess can arise from contiguous spread of infection from the oropharynx, middle ear, paranasal sinuses, or orbits. Risk factors for abscess formation by this mechanism are trauma (face, skull), recent neurosurgery, intracerebral hematoma, and neoplasm.

Harrison CJ: Focal suppurative infections of the nervous system. In Long SS (ed): Principles and Practice of Pediatric Infectious Diseases. New York, Churchill Livingstone, 2012, pp 321-327.

Honda H, Warren DK: Central nervous system infections: Meningitis and brain abscess. Infect Dis Clin North Am 2009;23:615-618.

28. What are some of the clues that can help identify brain abscesses?

Unfortunately, the early presentation of brain abscess in children is often nonspecific. Symptoms include headache, fever, and vomiting. Later, brain abscesses may manifest as seizure, mental status change, coma, focal neurologic deficits, papilledema, meningeal signs, and hemiparesis. A unilateral headache in a febrile child should raise concern for the presence of a brain abscess.

29. Describe the laboratory findings in focal suppurative intracranial infections.

Common laboratory indicators of infection and inflammation—e.g., leukocytosis, left shift, elevated erythrocyte sedimentation rate (ESR), elevated C-reactive protein (CRP)—are of limited value in diagnosing or excluding the presence of suppurative intracranial infections. Blood cultures are positive in only about 10% of cases.

Do not perform a lumbar puncture if a focal suppurative intracranial infection is confirmed. If CSF is obtained, it can be normal or nonspecifically abnormal (mild to moderately elevated white blood cell (WBC) count, neutrophil predominance, low glucose, and elevated protein). The rate of positive CSF cultures in the clinical setting of a brain abscess or subdural empyema may be as low as 10%. Rates are increased in the face of concurrent meningitis or abscess rupture into the subarachnoid space, but a culture of the pus collected intraoperatively is the gold standard for identification of the organism involved. Consider polymerase chain reaction (PCR) testing (e.g., enterovirus, herpes simplex virus [HSV]) of CSF if available.

Harrison CJ: Focal suppurative infections of the nervous system. In Long SS (ed): Principles and Practice of Pediatric Infectious Diseases. New York, Churchill Livingstone, 2012, pp 321-327.

30. What is the diagnostic modality of choice for focal suppurative central nervous system (CNS) infections?

Contrast-enhanced CT or MRI is indicated if the clinician suspects a focal suppurative CNS infection. Consult with a radiologist to help choose the correct study for a given patient.

31. What is the treatment for focal suppurative CNS infections?

A multidisciplinary approach, with involvement of consultants from the infectious diseases, neurosurgery, radiology, and critical care services, is critical to the prompt and successful treatment of a child with a suppurative CNS infection. As always, patient stabilization takes precedence in the management of increased ICP. Ideally, obtain cultures prior to the initiation of antibiotic treatment. It may be necessary to initiate antibiotic treatment prior to surgery (where the culture will be obtained). The choice of antibiotics should be guided by the clinician's knowledge of the patient's risk factors, as this can determine the likely pathogens involved. In the absence of such information, a reasonable choice is the combination of a third-generation cephalosporin, metronidazole, and vancomycin. Surgical treatment will be determined by the site of the infection. Corticosteroids are indicated only in the event that cerebral edema has resulted in increased ICP or neurologic deterioration. Also consider administration of antiepileptics.

Honda H, Warren DK: Central nervous system infections: Meningitis and brain abscess. Infect Dis Clin North Am 2009;23:615-618.

32. Compare hemorrhagic and ischemic strokes.

Contrary to the adult population, hemorrhagic strokes are more common in pediatrics than ischemic strokes. Hemorrhagic stroke refers to spontaneous intraparenchymal bleed and

nontraumatic subarachnoid hemorrhage. The most common risk factor is arteriovenous malformation. Other common risk factors of hemorrhagic stroke include coagulopathies (vitamin K deficiency, clotting factor deficiency, and thrombocytopenia), hemorrhage into a tumor, vascular malformations (sickle cell disease, berry aneurysms), and arterial hypertension secondary to renal disease, coarctation of the aorta, pheochromocytoma, or sympathomimetic drug use.

Ischemic stroke refers to a focal reduction in CBF leading to hypoxic damage to the brain. The most common risk factor is congenital heart disease (up to 25% of patients). Other risk factors include hypercoagulability, hyperviscosity, certain medications (e.g., oral contraceptive pills), acquired valvular defects, and vascular disease (sickle cell, homocystinuria, moyamoya, migraine).

Gorelick MH, Blackwell CD: Neurologic emergencies. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 1023-1024.

33. How may stroke present in the pediatric ED?

Fortunately, stroke is an uncommon diagnosis in the pediatric ED, but a low index of suspicion may lead to delayed diagnosis and treatment. As with many early presentations of neurosurgical conditions, stroke may present in a nonspecific fashion. Ischemic strokes often present with a focal neurologic deficit (e.g., hemiplegia), whereas hemorrhagic strokes may present with a variety of vague symptoms including headache, seizure, and change in mental status. With nonverbal children (e.g., infants, toddlers, children with special needs), diagnosis is even more challenging.

Steele DW: Neurosurgical emergencies, nontraumatic. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 1590-1592.

34. What is the initial radiographic evaluation for suspected stroke?

Noncontrast head CT is a reasonable first study in the ED because it will identify hemorrhage. Unfortunately, it may not detect an ischemic event in its early stages (12-24 hours after the event) and provides limited evaluation of the posterior fossa. MRI is a better study in both regards and should be performed if the noncontrast CT is normal.

35. What are the next steps in the diagnostic workup for stroke?

The workup that follows emergent neuroimaging is directed at identifying the cause for the hemorrhage or ischemia. Such an evaluation may include vascular studies (magnetic resonance angiography, magnetic resonance venography, transfontanel Doppler), cardiac evaluation (electrocardiography, chest radiography, echocardiography with or without bubble contrast), and laboratory evaluation (complete blood count, electrolytes, drug screen, prothrombin time/partial thromboplastin time/international normalized ratio, anticardiolipin antibodies, lupus anticoagulants, ESR, antinuclear antibody, and screening for antithrombin III, protein C and protein S deficiencies, factor V Leiden mutation, and prothrombin 20210 polymorphism).

Friess S, Ichord R: Stroke and intracerebral hemorrhage. In Fuhrman B, Zimmerman J (eds): Pediatric Critical Care, 4th ed. Philadelphia, Elsevier, 2011, pp 893-898.

Gorelick MH, Blackwell CD: Neurologic emergencies. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 1023-1024.

36. What is the treatment for stroke?

As always, cardiorespiratory stabilization remains the priority in the acute management of any patient in the ED. Obtain a dextrose level immediately and treat abnormalities aggressively, as both hypoglycemia and hyperglycemia have been found to worsen ischemic stroke. Antipyretics are recommended, as hyperthermia can similarly exacerbate ischemic stroke. Treat seizures with antiepileptics. In the case of hypotension, give fluids judiciously to avoid cerebral edema. Keep the head of the bed flat to avoid postural perfusion fluctuations.

Additional treatment depends on the type of stroke and identified cause. Ischemic strokes may be treated with anticoagulation after consultation with hematology and neurosurgery. At this time, there is little evidence to support thrombolytic therapy in pediatric patients

with stroke. Hemorrhagic strokes may require neurosurgical intervention if there is evidence of rapid hematoma expansion.

Friess S, Ichord R: Stroke and intracerebral hemorrhage. In Fuhrman B, Zimmerman J (eds): Pediatric Critical Care, 4th ed. Philadelphia, Elsevier, 2011, pp 893-898.

Gorelick MH, Blackwell CD: Neurologic emergencies. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 1023-1024.

37. What are the common causes, signs, and symptoms of nontraumatic spinal cord compression in the pediatric patient?

Nontraumatic spinal cord compression may be caused by primary neoplasms, vertebral metastases, abscesses, epidural hematomas, disk herniation, or tethered cord.

Back pain is rare in children and therefore should never be discounted without the performance of a careful and thorough history and physical examination. Weakness, increased or absent tendon reflexes, a Babinski reflex, symmetric loss of sensation, and sphincter abnormalities are common signs of spinal cord compression. Conus medullaris and cauda equina compression present in a similar fashion.

Aspesberro FP, Roberts JS, Brogan TV: Hematology and oncology problems in the intensive care unit. In Fuhrman B, Zimmerman J (eds): Pediatric Critical Care, 4th ed. Philadelphia, Elsevier, 2011, pp 1158-1159.

38. What is the approach to suspected nontraumatic spinal cord compression?

If spinal cord compression is suspected, search for the compressing entity or process. Emergent imaging with MRI is the cornerstone of the initial evaluation of such patients. Early consultation with a neurosurgeon is imperative. If a tumor is identified and there has been rapid progression of symptoms or there is a clear cord-level deficit, administer intravenous high-dose corticosteroids without delay and consult an oncologist immediately.

Schiff D: Spinal cord compression. Neurol Clin 2003;21:67-86.

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CHAPTER 46

OPHTHALMOLOGIC EMERGENCIES

Katie Giordano

1. List the eye emergencies that warrant ophthalmologic consultation.

- Ruptured globe
- Orbital hematoma
- Severe chemical burns
- Hyphema
- Retinal/vitreous detachment
- Intraocular foreign bodies
- Contact lens abrasions
- Complicated eyelid lacerations

2. How can you distinguish viral conjunctivitis from bacterial conjunctivitis?

Conjunctivitis is an inflammation of the conjunctiva (the thin translucent membrane lining the anterior sclera and undersurface of the eyelids). In this condition, there is dilation of the conjunctiva vessels, which leads to hyperemia and edema. Distinguishing bacterial conjunctivitis from a viral infection can be difficult. Viral conjunctivitis is the most common type overall and is seen more commonly in summer. Bacterial conjunctivitis accounts for 50% to 75% of conjunctivitis in children and is more common from December through April. Both are typically self-resolving in 1 to 2 weeks.

Viral conjunctivitis is highly contagious. From 65% to 90% of viral conjunctivitis is due to adenovirus, and discharge is typically watery. Treatment is supportive, although artificial tears or antihistamine eye drops may provide symptomatic relief. Simple handwashing can be preventive.

Bacterial conjunctivitis can be transmitted from an infected individual, overgrowth of native flora, or oculogenital transmission. The most common pathogens are staphylococcal species, *Streptococcus pneumoniae, and Haemophilus influenzae*, but in children *H. influenzae*, *S. pneumoniae* and *Moraxella catarrhalis* prevail. Discharge is typically purulent. Culture severe purulent discharge and consider gonococcal conjunctivitis.

Azari AA, Barney NP: Conjunctivitis: A systemic review of diagnosis and treatment. JAMA 2013;310 (16):1721-1729.

3. What is the treatment for bacterial conjunctivitis?

About 60% of culture-proven bacterial conjunctivitis cases are self-limiting in 1 to 2 weeks. When antibiotics are prescribed, any broad-spectrum topical antibiotic can be used. Use of an antibiotic may shorten the course of disease, decrease transmissibility, and allow for quicker return to school/day care. In a large Cochrane database analysis there was no difference in outcomes between those treated and those given placebo. Send a culture for those cases that are refractory to treatment.

Azari AA, Barney NP: Conjunctivitis: A systemic review of diagnosis and treatment. JAMA 2013;310 (16):1721-1729.

Sheikh A, Hurwitz B, van Schayck CP, McLean S, Nurmatov U. Antibiotic versus placebo for acute bacterial conjunctivitis. Cochrane Database Syst. Rev. 12, 9:CD001211.

4. What are special considerations for neonatal conjunctivitis?

In the first 3 days of life neonates can present with chemical conjunctivitis due to silver nitrate used for ocular prophylaxis. Most hospitals now apply erythromycin ointment or dilute betadine solutions to the eyes of newborns because of this complication. Prophylaxis does not completely eliminate the risk of chlamydial or gonoccocal infection in the neonatal period.

Chlamydia and *Neisseria gonorrhoeae* must be considered as a possible pathogen in neonatal conjunctivitis. A dramatically hyperemic conjunctiva with lid swelling and copious purulent

drainage is characteristic of gonococcal infection. There is a high risk of corneal perforation with gonococcal infections, and therefore, you must presume the infant has this infection and treat accordingly until proved otherwise. A Gram stain with the presence of gram-negative diplococci is helpful in determining the need to start treatment. Admit patients for parenteral antibiotic therapy with ceftriaxone and consult an ophthalmologist. Hourly saline lavage can limit the number of organisms that could penetrate the cornea.

The examination of infants with chlamydial conjunctivitis usually reveals hyperemic conjunctiva, mucopurulent discharge, and lymphoid follicle formation (conjunctival stippling). Send a culture of the discharge. The majority of chlamydial conjunctivitis cases are unilateral. About 50% of neonates with chlamydial conjunctivitis can have lung, nasopharynx, or genital infection. Treat with both topical and systemic oral antibiotics in culture-proven chlamydial conjunctivitis. The systemic treatment eradicates the carriage of *Chlamydia* in the nasopharynx and prevents the subsequent development of chlamydial pneumonitis.

Azari AA, Barney NP: Conjunctivitis: A systemic review of diagnosis and treatment. JAMA 2013;310 (16):1721-1729.

Levin AV: Ophthalmic emergencies. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1595-1602.

5. What additional concerns should be considered in contact-wearing patients with conjunctivitis?

There is a high risk of bacterial keratitis in those patients who wear contacts. Remove the contact lens immediately. Treat these patients with topical antibiotics and refer them to an ophthalmologist.

Azari AA, Barney NP: Conjunctivitis: A systemic review of diagnosis and treatment. JAMA 2013;310 (16):1721-1729.

6. How can you distinguish preseptal (periorbital) cellulitis from orbital cellulitis?

Both periorbital and orbital cellulitis may be associated with fever, pain, eyelid swelling, and eye redness. The hallmark signs of orbital cellulitis are decreased eye movement, proptosis, decreased vision, and signs of optic nerve involvement such as papilledema, decreased color vision, visual field deficits, or Marcus Gunn pupil.

Imaging studies are crucial in determining the extent of infection and appropriate management. Computed tomography (CT) scanning has been the modality of choice, as it is quick and provides distinct bony resolution. It can also be utilized in surgical image-guided procedures. Consider the use of magnetic resonance imaging (MRI) due to the risk of radiation exposure with CT. An abscess can potentially be seen on MRI without the use of contrast. MRI has been typically reserved for cases in which there is a concern for intracranial complications of orbital cellulitis. The limitation of MRI is the length of the study and the potential need for sedation in the younger population.

Bedwell J, Bauman NM: Management of pediatric orbital cellulitis and abscess. Curr Opin Otolaryngol Head Neck Surg 2011;19:467-473.

Levin AV: Ophthalmic emergencies. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1595-1602.

7. What is the treatment plan for orbital cellulitis?

After imaging has been obtained and orbital cellulitis or abscess has been diagnosed, begin intravenous (IV) antibiotics. Obtain blood cultures, and cerebrospinal fluid (CSF) testing when indicated, prior to initiation of antibiotics. A cephalosporin is a good broad-spectrum parenteral antibiotic. Consider adding clindamycin because of the high incidence of methicillin-resistant *Staphylococcus aureus* (MRSA). Initiate oral antibiotics once the patient is clinically improving. Loss of vision and extension of infection are complications of untreated orbital cellulitis. Orbital cellulitis associated with subperiosteal or retrobulbar abscesses typically responds to IV antibiotics and does not require surgical management. Immediate drainage is required in patients with pansinusitis, large abscesses with significant mass effect, or intracranial extension. When drainage is performed, the most common organism is *S. aureus*. Streptococcal species and *H. influenzae* have also been implicated.

Bedwell J, Bauman NM: Management of pediatric orbital cellulitis and abscess. Curr Opin Otolaryngol Head Neck Surg 2011;19:467-473.
Starkey CR, Steele RW: Medical management of orbital cellulitis. Pediatr Infect Dis J 2001;20: 1002-1005.

8. What are the key points in the initial management of an eye injury?

History will direct care of the patient and must include not only the present history of the injury/complaint but also the visual status of the patient prior to the injury. Ask if there is any history of eye surgeries or a prior known level of visual acuity, and whether the patient wears corrective lenses. Inquire about the mechanism of injury: blunt versus penetrating, suspected contaminants, risk of foreign body injury, and how hard, or the speed at which the eye was hit.

A crucial piece of the eye examination is visual acuity. At a minimum, evaluate light perception. This can be performed even through a closed, swollen eyelid. In an unresponsive patient, light perception can be demonstrated with reflex contraction of the closed eyelid. Next inspect the peripheral eye: observe eyelids, degree of swelling, ecchymosis, laceration, eye movement, and the anterior surface of the eye all without upsetting the patient. If during this portion of the examination a ruptured globe is suspected, stop the examination. Causing a patient to become upset in the presence of a ruptured globe can cause a Valsalva response and increases intraocular pressure.

Levin AV: Eye trauma. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 1448-1458.

9. What is the first step in the evaluation of eye trauma?

The first step is to rule out a ruptured globe and determine the extent of injury. A way to remember the examination has been referenced as I-ARM: **i**nspection, **a**cuity, **r**ed reflex, and **m**obility. If a ruptured globe is considered, stop the examination, place a protective shield over the eye, and consult ophthalmology emergently.

Wright KW: Sublux lens (ectopia lentis). Pediatric Ophthalmology for Pediatricians. Baltimore, Williams & Wilkins, 1999, pp 313-337.

Key Points: Evaluation of an Eye Injury

1. The mechanism of injury and timing of injury are critical points of information.
2. It is imperative to test visual acuity.
3. Investigate the possibility of a foreign body; metallic foreign bodies, if not removed, can stain the cornea.

10. Which signs indicate minor eye trauma?

- Visual acuity is 20/30 or greater
- Good red reflex
- Round pupil
- No pain
- No hyphema
- No significant conjunctival hemorrhage

If these criteria are present, the patient can be discharged home without further treatment.

11. How do you test the afferent pupillary reflex? What will you see if the patient has an afferent pupillary defect?

The pupillary light reflex is a reflex arc through the midbrain involving crossover innervation. It is assessed with the swinging light test: Shining a light in one eye should result in constriction of both pupils. No change in pupillary size should be noted when the light swings toward the other pupil. If a patient has an afferent pupillary defect, both pupils dilate when the light swings to the affected eye. The abnormal pupil, called a Marcus Gunn pupil, can result from retinal artery or venous occlusions, retinal detachment, tumors, or ischemic optic neuropathy.

Zitelli BJ, Davis HW: Atlas of Pediatric Physical Diagnosis, 5th ed. St. Louis, Mosby Elsevier, 2007.

12. What is the Bruckner test? How is it performed?

The Bruckner test is a simultaneous bilateral red reflex test that elicits both a corneal light reflex and a red reflex. View the patient's eyes from about 2 feet away with a broad beam of light that encompasses both eyes. You should see both a red reflex and a small white light reflex in each eye. The key to a normal examination is symmetry. An absent or dull red reflex may indicate vitreous hemorrhage, cataract, hyphema, opacity of the cornea, enophthalmos, or misalignment of the globe.

Garcia SE, Hickey R, Santamaria JP: Pediatric ocular trauma. Pediatr Emerg Med Rep 1998; October 87-98.

13. What are the causes of papilledema?

Papilledema is optic disk swelling. It is usually bilateral and is caused by anything that increases intracranial pressure. Unilateral papilledema suggests ipsilateral orbital trauma such as orbital hemorrhage, orbital tumor, or direct injury to the optic nerve. Ophthalmoscopic signs include blurring of the disk margins and disk edema. The disk may be hyperemic due to telangiectasis of the capillaries and small hemorrhages. The patient may have visual field deficits. With acute papilledema, vision is maintained. Common causes of papilledema are as follows:

- Increased or decreased CSF production
- Intracranial mass
- Obstruction of venous outflow
- Obstructive hydrocephalus
- Craniosynostosis
- Idiopathic intracranial hypertension (IIH) (pseudotumor cerebri)

Giovannini J: Papilledema. Available from www.emedicine.com/oph/topic187.htm.
Levin AV: Eye trauma. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1448-1458.
Zitelli BJ, Davis HW: Atlas of Pediatric Physical Diagnosis, 5th ed. St. Louis, Mosby Elsevier, 2007.

14. When should you be concerned about idiopathic intracranial hypertension (IIH)?

This condition usually occurs in obese teenage girls who present with severe headache and papilledema on fundoscopic examination. The papilledema is usually bilateral. Some patients with this condition also have diplopia, visual field restriction, and abducens nerve (sixth cranial nerve) palsy (unilateral or bilateral). Neurologic examination is otherwise normal.

Obtain neuroimaging when IIH is suspected. MRI (with and without contrast) is the imaging study of choice. With this condition, the MRI shows normal brain parenchyma with no mass, hydrocephalus, or structural lesion. Perform a lumbar puncture after the MRI, unless there is a clear source of increased intracranial pressure. With IIH the lumbar puncture will show elevated opening pressure and CSF will be otherwise normal. Consult an ophthalmologist or a neurologist to help with visual field testing and management.

15. What makes up the orbit?

- Frontal bone: Superior orbital ridge and the upper medial orbital ridge
- Zygoma: Lateral orbital rim
- Maxilla: Inferior and lower medial rim
- Maxillary sinus: Orbital floor
- Lacrimal bone: Separates the orbit from the nares
- Ethmoid bone: Medial wall and part of the posterior wall
- Sphenoid bone: Posterior wall

Widell T: Orbital fracture in emergency medicine. Available from www.emedicine.com/emerg/topic202.htm.

16. List and describe briefly the physical findings of orbital wall fractures.

- Medial wall fractures are caused by blows to the bridge of the nose. Physical findings include orbital emphysema, epistaxis, depressed nasal bridge, and enophthalmos. Excessive tearing may be seen if the lacrimal system is disrupted.
- Orbital floor ("blowout") fractures result when an object impacts the inferior lateral orbital rim. The impact causes increased intraorbital pressure and rupture of the orbital floor,

and the orbital contents can be prolapsed into the maxillary sinus. Entrapment of the inferior rectus muscle causes limitation of upward gaze. Infraorbital nerve injury causes hypesthesia of the ipsilateral cheek and upper lip. Traumatic optic neuropathy can complicate an orbital floor fracture with immediate loss of vision and afferent pupillary defect.

- Superior wall (orbital roof) fractures are less common than medial or floor fractures, but they are potentially life-threatening. They may be associated with central nervous system injury, pneumocephalus, or intracranial foreign body. Findings include CSF rhinorrhea and superior and lateral subconjunctival hemorrhage. Potential complications include brain abscess and meningitis.
- Tripod fractures involve the zygomatic arch and its lateral and inferior orbital rim articulations. Examination findings are similar to those of orbital floor fractures, along with limitation of mandibular movement and trismus.

Garcia SE, Hickey R, Santamaria JP: Pediatric ocular trauma. Pediatr Emerg Med Rep 1998;87-98.
Widell T: Orbital fracture in emergency medicine. Available from www.emedicine.com.

17. What are important historical questions regarding orbital fractures?

- Is there epistaxis or CSF rhinorrhea/otorhea? CSF rhinorrhea is seen with orbital fracture.
- Does the patient have vision changes such as blurry vision or double vision? Blurry vision can be seen with hyphema, retinal detachment, and vitreous hemorrhage.
- Is there diplopia with lateral or upward gaze? Diplopia is concerning for lens dislocation.
- Does the patient have pain with eye movement? Pain is indicative of extraocular muscle entrapment.
- Is photosensitivity present? Photophobia is concerning for iritis.
- Does the patient complain of flashes of light? Flashes of light are seen in retinal detachment.

Levin AV: Eye trauma. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1448-1458.
Widell T: Orbital fracture in emergency medicine. Available from www.emedicine.com/emerg/topic202.htm.

18. How are orbital fractures managed?

Address any life-threatening injuries before focusing on the eye injury. If globe rupture is suspected, stop the examination, keep the patient calm (crying can increase intraocular pressure), and consult ophthalmology. Therapeutic interventions for orbital fracture include antibiotic prophylaxis, nasal decongestants, and ice packs; some fractures may require surgical intervention. CT of the orbit, including axial and coronal views, is useful for diagnosing and delineating the extent of injury. Caution all patients with this injury to avoid blowing the nose.

Levin AV: Eye trauma. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1448-1458.
Widell T: Orbital fracture in emergency medicine. Available from www.emedicine.com/emerg/topic202.htm.

19. What is the Seidel test? How is it performed?

Fluorescein instilled into the eye at the lacerated area of the eye will show a stream of fluorescein, indicating an active leak of aqueous humor and full-thickness injury to the eye. A negative Seidel test does not exclude full-thickness injury, because small injuries can self-seal. Treat full-thickness lacerations with a rigid eye shield and emergent ophthalmologic consultation.

20. What is the treatment of corneal abrasions?

Current treatment recommendations include the use of topical antibiotics and pain control with either topical or oral analgesics. Patients using topical nonsteroidal anti-inflammatory drugs (NSAIDs) reported greater relief from pain and other symptoms. Examples of topical NSAIDs include diclofenac (Voltaren) and ketorolac (Acular). Some clinicians use prophylactic topical antibiotics for treatment of corneal abrasions. Ointments are more lubricating than drops, are better tolerated in young children, and do not need to be reapplied as often as drops. Antipseudomonal coverage is important in contact lens wearers, and they should discontinue lens use until the abrasion is healed and antibiotic therapy is complete. Topical anesthetics are not indicated after the initial examination. Continuing this treatment can

predispose the patient to subsequent injury. Patching is not recommended, as it can increase moisture and increase the risk of infection. Randomized, controlled trials have shown that topical mydriatics gave no improvement in pain control.

Weaver CS, Terrell KM: Evidence based emergency medicine update. Do ophthalmic nonsteroidal anti-inflammatory drugs reduce the pain associated with simple corneal abrasions without delaying healing? Ann Emerg Med 2003;41:134-140.
Wilson SA: Management of corneal abrasions Am Fam Physician 2004;70:123-128.

21. Which corneal abrasions are most worrisome? Why?

Corneal abrasions over the visual axis, abrasions in patients with a history of ocular herpes, and corneal abrasions in contact lens wearers are concerning. Those who wear contact lenses and develop a corneal abrasion are predisposed to fungal and bacterial infections and corneal ulceration.

22. What is a hyphema?

A hyphema is a collection of blood in the anterior chamber of the eye caused by blunt trauma to the eye and tearing of the anterior ciliary body. If there is no history of trauma, consider leukemia, hemophilia, juvenile xanthrogranuloma, retinoblastoma, or child abuse. Factors that place a person at risk for hyphema include a bleeding disorder, anticoagulation therapy, kidney disease, and sickle cell disease or trait. The higher the grade of the hyphema, the greater the risk for rebleeding. The likelihood of rebleeding is highest in the first 5 days after injury.

Levin AV: Eye trauma. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1448-1458.

23. What is the treatment for hyphema?

Identify and treat associated eye injuries. In particular, rule out a ruptured globe. Treatment of hyphema can be based on the grade of bleeding. Grading ranges start with microhyphema, which is circulating red blood cells seen on slit lamp examination only. Grade I is less than 33%, grade II is 33% to 40%, grade III is greater the 50%, and grade IV is 100%, or an "8-ball eye." There is only a 30% chance of recovery of visual acuity greater the 20/50 with a grade IV hyphema. Give special consideration when managing children and patients with hemoglobin S or hemophilia. Focus treatment on prevention of rebleeding and intraocular hypertension. Most recommend an eye shield, limited activity, elevation of bed to 45 degrees, control of nausea and vomiting, and pain control. Antifibrinolytic agents and corticosteroids may also be indicated but should be prescribed only after consultation with ophthalmology. Discharge the patient if there is no concern for medication compliance and follow-up. Consider hospital admission for those with hemoglobin S and hyphema greater than 50%, or grade III and above. The reported range for rebleeding is 0% to 38%; there is increased risk of loss of visual acuity when rebleeding occurs.

Chen A: Burns, ocular. Available from www.emedicine.com/emerg/topic736.htm.
Levin AV: Eye trauma. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1448-1458.
Sheppard J: Hyphema. Available from www.emedicine.com/oph/topic765.htm.

24. What are the symptoms of retinal detachment?

A retinal detachment is a separation between the sensory and pigment portions of the retina. Symptoms include "flashes," "floaters," and visual defects. Flashes of light are caused by traction at the peripheral retina. Floaters are caused by fibrous aggregates on the posterior portion of the vitreous that block light to the retina. Retinal tears may be asymptomatic for years, but if the macula is involved, the visual loss is severe. Consult an ophthalmologist to evaluate all patients who report flashes or floaters to prevent the progression of partial retinal tears to complete detachment. Traumatic damage to the retina often is located at the periphery of the retina, an area that is not easily examined with a direct ophthalmoscope.

Felter RA: Eye-visual disturbances. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 259-265.
Larkin GL: Retinal detachment. Available from www.emedicine.com/emerg/topic504.ht.

25. Which sports are associated with a high risk for eye injuries?

- Paintball and use of an air rifle or BB gun
- Basketball
- Baseball/softball
- Lacrosse
- Ice, field, and street hockey
- Squash and racquetball
- Fencing
- Boxing, wrestling, and full-contact martial arts

Committee on Sports Medicine and Fitness in Pediatrics: Protective eyewear for young athletes. Pediatrics 2004;113:619-622.

26. What are the recommendations for use of protective eyewear in athletes?

Appropriately fitted protective eyewear has been shown to decrease the incidence of injury by 90%. Almost 42,000 sports-related eye injuries occur each year in the United States, and more than one third involve children. The American Academy of Pediatrics and the American Academy of Ophthalmology recommend eye protection for all athletes and mandate it in functionally one-eyed patients and in patients whose ophthalmologist recommends eye protection after surgery or trauma. *Functionally one-eyed* is defined as best corrected visual acuity of less than 20/40 in the weaker eye.

Recommendations for the appropriate protective eyewear for each sport can be found in the American Academy of Pediatrics policy referenced here and on the website for the Protective Eyewear Certification Council.

American Academy of Ophthalmology: Think "protective eyewear" when playing sports. Available at www.sportseyeinjuries.com.
Hamill, MB. Academy of Pediatrics and Academy of Ophthalmology Joint Policy Statement: Protective Eyewear for Young Athletes. Ophthalmology Volume 111, Number 3, March 2004.
Prevent Blindness America. Available at www.preventblindness.org.
Protective Eyewear Certification Council. Available at www.protecteyes.org.

27. Why is a burn from an alkali more damaging to the eye than an acid burn?

The pH of the chemical that splashes in the eye is related to severity of injury. Alkali solutions tend to be more damaging because they penetrate more deeply. Acidic solutions coagulate proteins in the superficial layers of the eye, forming a protective barrier against further penetration. The severity of the injury is also related to the volume and duration of exposure.

Felter RA: Eye-visual disturbances. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 259-265.

28. What is the best solution to use for ocular decontamination after a chemical injury?

Traditional first aid teaches that tap water is an acceptable irrigant for immediate treatment of a foreign body or chemical in the eye. It is best for families to irrigate the eyes with tap water at home before coming to the emergency department. In the emergency department setting, the most common irrigant is normal saline solution (pH, 4.5-6.0). Other acceptable alternatives include lactated Ringer's solution (pH, 6.2-7.5), buffered normal saline (pH, 7.4), and balanced salt solution plus (BSS plus; pH, 7.4). Anesthetize the affected eye with drops as soon as possible (but do not delay irrigation). Continue irrigation for a minimum of 30 minutes. A Morgan lens is a helpful tool that can be connected to a bag of normal saline, and 1 to 2 L is used. If possible, check the pH of the eye before irrigation and continue irrigation until the pH is neutral. Be sure to examine and treat both eyes (if necessary) and not just the one most obviously injured.

Bhende MS, Tham E; Ocular irrigation and decontamination of conjunctiva. In Henretig FM, King C (eds): Textbook of Pediatric Procedures, 2nd ed. Baltimore, Williams & Wilkins, 2007, pp 555-558.
Butler FK Jr. The Eye in the Wilderness; Wilderness Medicine, 6th ed. St Louis, Mosby 2011, Ch 28.
Saidinejad M: Ocular irrigant alternatives in pediatric emergency medicine. Pediatr Emerg Care 2005;21:23-26.

29. While attempting to repair a superficial forehead laceration, some of the cyanoacrylate glue drips into the child's eye and his lashes are stuck together. How should this patient be managed?

Cyanoacrylate glue rarely causes a serious injury to the eye. The lashes can be gently separated by applying mineral oil or petroleum jelly to the glued area and allowing this to remain in place for 30 to 60 minutes. Do not forcefully pull the lids apart. Use of gentle friction with a cotton swab may be helpful. Avoid the use of acetone to dissolve the glue because acetone could injure the eye. Ophthalmologic consultation is rarely needed.

30. A teenager who was not wearing protective eyewear reports a "foreign body sensation" in his eye after hammering (metal on metal). How should this patient be managed?

With this history, the emergency physician must be suspicious of an intraocular foreign body that penetrated the cornea or sclera. The eye can heal quickly, and a laceration may not be visible. Subtle signs, such as red eye, pupil asymmetry, and decreased vision may be noted. However, small particles that rapidly penetrate the eye may produce few or no signs. Maintain a high index of suspicion.

Attempt to remove the foreign body after a thorough examination has been performed and visual acuity has been assessed. Patients are usually quite accurate in their localization of the foreign body based on sensation. Instill a topical anesthetic and fluorescein dye. A corneal foreign body will be surrounded by a circular area of fluorescein. Vertical abrasions are typical of a foreign body on the eyelid. Evert the eyelids and examine for foreign bodies. A slit lamp may assist in localizing the foreign body in a cooperative child. Shining the otoscope at an angle provides indirect lighting, and the foreign body may cast a shadow. Plain films, ultrasound, and CT maybe helpful to look for a foreign body. Avoid MRI if the foreign body might be metallic.

Irrigation is often used to easily remove superficial foreign bodies. Use a premoistened cotton swab to remove the foreign body from the bulbar or palpebral conjunctiva. If the patient is cooperative, secure the head against the slit lamp frame, and the physician's hand can rest on the forehead of the patient. Consult ophthalmology when the foreign body is embedded, removal attempts are unsuccessful, or there is concern for a penetrating foreign body/ruptured globe.

Neylan V, Eilbert WP: Ocular emergencies. Foresight 1998;43:1-8.
Selbst SM: Pediatric emergency medicine legal briefs. Pediatr Emerg Care 2002;18:133-136.
Whitaker WT, Pomerantx WJ; Ocular foreign body removal. In Henretig FM, King C (eds): Textbook of Pediatric Procedures, 2nd ed. Baltimore, Williams & Wilkins, 2007, pp 550-553.

Key Points: Eye Emergencies

1. Emergently treat neonatal conjunctivitis caused by *N. gonorrhoeae* with parenteral and topical antibiotics owing to risk of corneal perforation.
2. Always attempt to document the visual acuity in assessing a patient with a complaint related to the eye.
3. When a ruptured globe is considered, stop the examination, place a rigid shield over the eye, keep the patient calm, and emergently consult ophthalmology.
4. Appropriately fitted protective eyewear can reduce the risk of ocular injury by 90%.
5. Caution patients with potential orbital fractures to refrain from blowing the nose.
6. Prompt irrigation of the eye will prevent further damage from a chemical injury.

Acknowledgment

The author wishes to thank Dr. Kathy Palmer for her contributions to this chapter in the previous edition.

CHAPTER 47

ORTHOPEDIC EMERGENCIES

Christopher J. Russo

1. Describe the presentation of osteomyelitis.

Patients with osteomyelitis classically demonstrate fever and pain at the affected site. Other signs of systemic illness, such as anorexia, malaise, and vomiting, may be present. Infants often exhibit decreased feeding, irritability, or listlessness. When an extremity is involved, pain may manifest as pseudoparalysis. Examination findings typically include focal swelling, warmth, tenderness, or erythema, often metaphyseal in location. Neonates and younger children are more prone than older patients to present with secondary joint involvement. Tenderness may be out of proportion to the soft tissue findings. Some patients present with symptoms of severe illness, including signs of generalized sepsis and shock.

Frank G, Mahoney HM, Eppes SC: Musculoskeletal infections in children. Pediatr Clin North Am 2005;52:1083-1106.

Gutierrez KM: Osteomyelitis. In Long SS, Pickering LK, Prober CG (eds): Principles and Practice of Pediatric Infectious Disease, 4th ed. Philadelphia, Elsevier Churchill Livingstone, 2012, pp 469-476.

Pääkönen M, Peltola H: Bone and joint infections. Pediatr Clin North Am 2013; 60:425-436.

2. How are most cases of osteomyelitis acquired?

Hematogenous spread remains the most common source for osteomyelitis in children. Direct inoculation into the bone or local invasion from a contiguous infection can occur. Infection due to vascular insufficiency is rare in children.

Gutierrez KM: Bone and joint infections in children. Pediatr Clin North Am 2005;52: 779-794.

Kaplan SL: Osteomyelitis in children. Infect Dis Clin North Am 2005;19:787-797.

3. What laboratory tests are helpful in the diagnosis of osteomyelitis?

Diagnosis of osteomyelitis rests primarily on clinical findings; laboratory results and diagnostic imaging help confirm the diagnosis. The erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) are more sensitive indicators than peripheral white blood cell (WBC) count, which is unreliable and elevated only 20% of the time. ESR is elevated in up to 90% of cases of hematogenously acquired osteomyelitis. CRP is elevated in up to 98% of cases. Obtain a blood culture, though it may not identify a causative organism. The highest yield for isolating a causative organism is achieved when bone aspirate, joint aspirate, and blood are collected for culture. Together these cultures can establish a microbiologic diagnosis 50% to 80% of the time. With the exception of patients who are clinically unstable, obtain these cultures before initiation of parenteral antibiotics.

Polymerase chain reaction (PCR) may be helpful in identifying infections secondary to *Bartonella henselae* and *Kingella kingae*. Although more rapid and sensitive, PCR does not provide information regarding antibiotic sensitivity and resistance.

Faust SN, Clark J, Pallett A, Clarke NMP: Managing bone and joint infection in children. Arch Dis Child 2012;97:544-553.

Gutierrez KM: Bone and joint infections in children. Pediatr Clin North Am 2005;52:779-794.

4. Which imaging studies are indicated when osteomyelitis is suspected?

- **Plain radiography** is a reasonable initial imaging choice in the emergency department (ED) for a patient with suspected osteomyelitis. Plain films can rule out fracture or bone malignancy. Bone abnormalities in osteomyelitis (lytic lesions, periosteal elevation, and periosteal new bone formation) may not appear until 10 to 20 days after symptoms begin. Changes to adjacent soft tissues (deep soft tissue swelling and loss of normal tissue planes) may occur much earlier, as early as several days after symptoms begin.

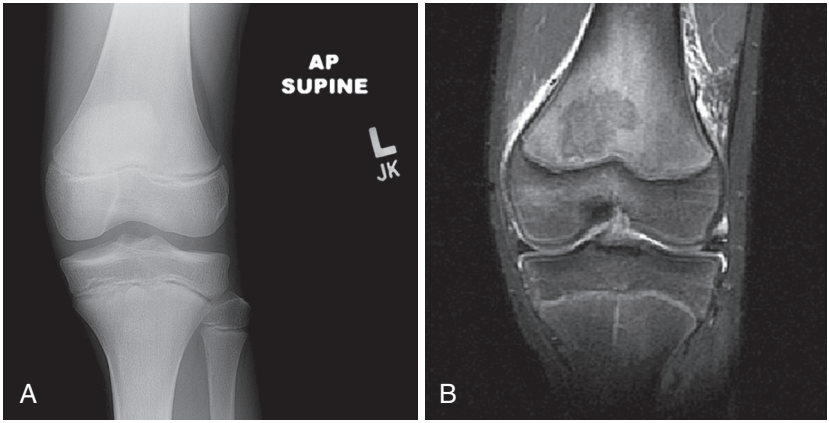

Figure 47-1. **A,** Plain radiograph of a knee with bone that registers as normal. **B,** Magnetic resonance image of the same knee with a positive reading of osteomyelitis.

- **Technetium-99 bone scanning** is more sensitive in the early diagnosis of osteomyelitis than plain radiography, with reported sensitivities of 80% to 100%. However, a bone scan can be normal in up to 20% of cases in the first few days of illness. Its specificity in differentiating osteomyelitis from other diagnostic considerations (malignancy, soft tissue cellulitis, septic arthritis, trauma, fracture, and infarction) is also limited.
- **Magnetic resonance imaging (MRI)** (Fig. 47-1) is now the imaging study of choice for evaluating a patient with suspected osteomyelitis. Sensitivity ranges from 92% to 100%. MRI helps differentiate osteomyelitis from cellulitis and can demonstrate myositis or pyomyositis contiguous to the site of bone involvement. As with bone scanning, malignancy, fracture, and infarction can appear similar to osteomyelitis on MRI.

Gutierrez KM: Osteomyelitis. In Long SS, Pickering LK, Prober CG (eds): Principles and Practice of Pediatric Infectious Disease, 4th ed. Philadelphia, Elsevier Churchill Livingstone, 2012, pp 469-476.
Kaplan SL: Osteomyelitis in children. Infect Dis Clin North Am 2005;19:787-797.

5. Which organisms are commonly seen in osteomyelitis?

Staphylococcus aureus is the most common causative organism. Other common organisms include *Streptococcus pneumoniae*, *Streptococcus pyogenes*, and *K. kingae*. In addition to *S. aureus*, group B streptococci and enteric gram-negative organisms are important organisms to consider in the neonatal period.

Consider *Neisseria gonorrhoeae* in sexually active adolescents. Anaerobic bacteria may be found in cases associated with sinusitis, mastoiditis, or dental abscess. *Serratia* and *Aspergillus* species are found in patients with granulomatous disease. Consider coagulase-negative staphylococci in patients who have undergone medical procedures. *Salmonella* species and gram-negative enteric organisms are found in patients with hemoglobinopathies. *Pseudomonas aeruginosa* is a pathogen found in puncture wounds to the foot. Exposure to kittens should prompt consideration of *Bartonella henselae*, and *Coxiella burnetii* should be considered in cases of exposure to farm animals. *Haemophilus influenzae* type b is only rarely seen since the advent of the *H. influenza*e type b vaccine but remains a consideration in an unimmunized child.

The increasing prevalence of methicillin-resistant *S. aureus* (MRSA) is worthy of particular attention and concern. Osteomyelitis caused by MRSA can be particularly virulent and is associated with multiple sites of bone involvement, myositis, pyomyositis, intraosseous and subperiosteal abscess formation, pulmonary involvement, and vascular complications (such as deep vein thrombosis and septic pulmonary emboli). These patients may be quite ill and require admission to the intensive care unit.

Mycobacterial and fungal infections are rare causes of osteomyelitis.

Frank G, Mahoney HM, Eppes SC: Musculoskeletal infections in children. Pediatr Clin North Am 2005;52:1083-1106.
Gutierrez KM: Osteomyelitis. In Long SS, Pickering LK, Prober CG (eds): Principles and Practice of Pediatric Infectious Disease, 4th ed. Philadelphia, Elsevier Churchill Livingstone, 2012, pp 469-476.
Kaplan SL: Osteomyelitis in children. Infect Dis Clin North Am 2005;19:787-797.

6. What historical features should raise suspicion for osteomyelitis (or septic arthritis) from *K. kingae*?

K. kingae is a gram-negative bacillus that colonizes the upper respiratory tract and plays an important role in osteomyelitis and septic arthritis in children. Infection with this organism typically occurs in children younger than 2 years of age and often follows an upper respiratory tract infection, pharyngitis, or stomatitis. It may be seasonal in occurrence (late summer through winter). Typically the course of illness for osteomyelitis is more subacute and may lead to delays in diagnosis. Outbreaks of invasive infections from this organism have been reported in day care populations.

Frank G, Mahoney HM, Eppes SC: Musculoskeletal infections in children. Pediatr Clin North Am 2005;52:1083-1106.
Gutierrez KM: Osteomyelitis. In Long SS, Pickering LK, Prober CG (eds): Principles and Practice of Pediatric Infectious Disease, 4th ed. Philadelphia, Elsevier Churchill Livingstone, 2012, pp 469-476.
Yagupsky P, Porsch E, St Geme JW III: *Kingella kingae*: An emerging pathogen in young children. Pediatrics 2011;127:557-565.

7. How should bone aspirate or synovial fluid be handled to improve isolation of *K. kingae*?

Recovery of *K. kingae* is improved when synovial fluid aspirate or bone aspirate is collected in aerobic blood culture medium. PCR assays substantially improve detection of this organism.

Kaplan SL: Osteomyelitis in children. Infect Dis Clin North Am 2005;19:787-797.
Ross JJ: Septic arthritis. Infect Dis Clin North Am 2005;19:799-817.
Yagupsky P, Porsch E, St. Geme JW III: *Kingella kingae*: An emerging pathogen in young children. Pediatrics 2011;127:557-565.

8. How is osteomyelitis treated?

Initiate antibiotic treatment as soon as appropriate cultures have been collected. Regardless of the patient's age, treatment should address the likelihood of infection with *S. aureus*. Given the increasing prevalence of MRSA in many communities, coverage of MRSA is very important. Many MRSA isolates are susceptible to clindamycin. In children, adolescents, and infants over 2 months of age, clindamycin is a good choice for initial antimicrobial coverage. Isolate testing for resistance to clindamycin should be performed. Vancomycin provides excellent coverage for MRSA. However, because of concerns about widespread use of vancomycin resulting in increasing antimicrobial resistance, reserve this for patients who are moderately to severely ill or who live in communities where significant resistance to clindamycin has been demonstrated. In infants younger than 2 months of age, additional antimicrobial coverage for group B streptococcus and enteric gram-negative bacteria is important. In this age group clindamycin or vancomycin plus cefotaxime or gentamycin are appropriate initial antimicrobial choices.

Special circumstances warrant consideration of antimicrobial coverage specific to the likely pathogens in such cases. For example, when considering infection with *K. kingae*, addition of a third-generation cephalosporin is prudent.

It is wise to consult your local antibiogram, because prevalence of community-acquired MRSA varies by geographic location and sensitivities to antibiotics vary as well.

Involve an orthopedic surgeon in cases of suspected or confirmed osteomyelitis. Surgical treatment is indicated in a number of circumstances. Moreover, surgical tissue is often helpful in identifying a causative organism to guide antimicrobial treatment.

Frank G, Mahoney HM, Eppes SC: Musculoskeletal infections in children. Pediatr Clin North Am 2005;52:1083-1106.

Gutierrez KM: Osteomyelitis. In Long SS, Pickering LK, Prober CG (eds): Principles and Practice of Pediatric Infectious Disease, 4th ed. Philadelphia, Elsevier Churchill Livingstone, 2012, pp 469-476.
Kaplan SL: Osteomyelitis in children. Infect Dis Clin North Am 2005;19:787-797.

9. Describe the signs and symptoms of septic arthritis.

Pain, fever, and decreased use of the involved limb are common symptoms of septic arthritis. Hip, knee, and ankle joints account for about 80% of cases of septic arthritis. Fever, malaise, and anorexia are seen in most patients. The involved joint is often held in a position of comfort. In the knee this is usually flexion. In the hip it is usually flexion, abduction, and external rotation. Passive range of motion away from the position of comfort is painful. In joints other than the hip, tenderness, swelling, warmth, and erythema may be seen and an effusion may be palpable. As in osteomyelitis, neonates may present with pseudoparalysis and tenderness of the affected limb.

Other diagnoses to consider in the patient with suspected septic arthritis include traumatic joint pain, transient synovitis ("toxic synovitis"), reactive arthritis, Lyme arthritis, juvenile rheumatoid arthritis, acute rheumatic fever, osteomyelitis, pyomyositis, necrotizing fasciitis, tumor, slipped capital femoral epiphysis, and Legg-Calvé-Perthes disease.

Gutierrez KM: Infectious and inflammatory arthritis. In Long SS, Pickering LK, Prober CG (eds): Principles and Practice of Pediatric Infectious Disease, 4th ed. Philadelphia, Elsevier Churchill Livingstone, 2012, pp 477-482.
Pääkönen M, Peltola H: Management of a child with suspected acute septic arthritis. Arch Dis Child 2012;97:287-292.
Ross JJ: Septic arthritis. Infect Dis Clin North Am 2005;19:799-817.

10. What laboratory tests help diagnose septic arthritis?

ESR and CRP levels are usually elevated in cases of septic arthritis. The WBC count appears to be less useful than ESR in differentiating septic arthritis from transient synovitis. A child with an ESR less than 20 mm/hour and CRP less than 20 mg/L (2 mg/dL) is very unlikely to have septic arthritis, but if either of these values is increased, consider arthrocentesis.

Blood culture and synovial fluid analysis (cell count, Gram stain, and culture) are essential in the evaluation of children with suspected septic arthritis. Ideally, perform these tests before the administration of antibiotics. Blood culture yields an organism in up to 40% and synovial fluid in up to 50% to 60% of cases. When combined, they yield an organism in up to 60% to 70% of cases. The yield of synovial cultures may be improved by inoculation of the fluid into blood culture media. The typical synovial fluid WBC count is over 50,000 cells/mm^3, with a predominance of polymorphonuclear cells (>75-90%). However, cell counts less than 50,000 cells/mm^3 can be seen in cases of septic arthritis, and counts over 50,000 cells/mm^3 are seen in juvenile rheumatoid arthritis. Synovial fluid glucose and protein levels are not reliable enough to differentiate septic arthritis from other infectious or inflammatory processes.

Pääkönen M, Peltola H: Management of a child with suspected acute septic arthritis. Arch Dis Child 2012;97:287-292.

11. Which imaging studies can help make the diagnosis of septic arthritis?

The initial imaging choice in the ED is plain radiography. Soft tissue swelling, joint space widening, and changes suggestive of osteomyelitis may be seen. In the hip, capsular swelling and loss of the gluteal fat planes may be seen as early evidence of infection in the joint. If hip infection is suspected, consider ultrasonography followed by joint aspiration. Technetium bone scanning and MRI are both sensitive modalities for detecting joint infection. MRI has the added advantage of delineating changes in the adjacent soft tissues and bone.

Frank G, Mahoney HM, Eppes SC: Musculoskeletal infections in children. Pediatr Clin North Am 2005;52:1083-1106.
Gutierrez KM: Infectious and inflammatory arthritis. In Long SS, Pickering LK, Prober CG (eds): Principles and Practice of Pediatric Infectious Disease, 4th ed. Philadelphia, Elsevier Churchill Livingstone, 2012, pp 477-482.
Ross JJ: Septic arthritis. Infect Dis Clin North Am 2005;19:799-817.

12. Which organisms are commonly seen in septic arthritis?

The organisms commonly responsible for septic arthritis are much the same as those commonly associated with osteomyelitis (see Question 5). Consider *N. gonorrhoeae* in neonates and in sexually active patients, especially those with multifocal joint involvement. Also consider *Candida* species in neonates.

Gutierrez K: Bone and joint infections in children. Pediatr Clin North Am 2005;52:779-794.
Gutierrez KM: Infectious and inflammatory arthritis. In Long SS, Pickering LK, Prober CG (eds): Principles and Practice of Pediatric Infectious Disease, 4th ed. Philadelphia, Elsevier Churchill Livingstone, 2012, pp 477-482.
Pääkönen M, Peltola H: Management of a child with suspected acute septic arthritis. Arch Dis Child 2012;97:287-292.

13. What is the treatment for septic arthritis?

Optimal treatment involves antibiotics and joint drainage. See Question 8 for discussion of antibiotics. Early consultation with an orthopedic surgeon is mandatory. Emergent surgical drainage of infected hip and neonatal shoulder infections is appropriate, whereas repeated nonsurgical aspiration may be reasonable for other joints. There is recent evidence that a short course of steroids may improve residual function and shorten the duration of acute symptoms.

Faust SN, Clark J, Pallett A, Clarke NMP: Managing bone and joint infection in children. Arch Dis Child 2012;97:544-553.
Frank G, Mahoney HM, Eppes SC: Musculoskeletal infections in children. Pediatr Clin North Am 2005;52:1083-1106.
Pääkönen M, Peltola H: Management of a child with suspected acute septic arthritis. Arch Dis Child 2012;97:287-292.

14. Reactive arthritis is in the differential diagnosis for the patients described earlier. What are the common organisms responsible for reactive arthritis?

Salmonella, *Shigella*, *Campylobacter* species, and *Yersinia enterocolitica* from the gastrointestinal tract; *N. gonorrhoeae* and *Chlamydia trachomatis* from the genitourinary tract; and *S. pyogenes*, *Mycoplasma pneumoniae*, and *Neisseria meningitidis* from the blood. Both *Neisseria* species and *S. pyogenes* can also cause septic arthritis.

Frank G, Mahoney HM, Eppes SC: Musculoskeletal infections in children. Pediatr Clin North Am 2005;52:1083-1106.
Gutierrez K: Bone and joint infections in children. Pediatr Clin North Am 2005;52:779-794.

15. Describe the typical presentation and evaluation of suspected pyomyositis.

Unfortunately, the diagnosis of pyomyositis is often not made until symptoms have been present for several weeks. Initial symptoms include crampy local muscle pain, most commonly involving large muscle groups like the thigh, calf, buttock, upper extremity, and iliopsoas muscles. Early in the course of the infection local edema is present, although the overlying skin is often normal. The edema has been described as having a woody or rubbery feel. Over time, induration, edema, erythema, and tenderness increase. Fever, increasing constitutional symptoms, toxicity, and signs of sepsis may develop if the infection progresses and is untreated. Presentation is often subacute. Pyomyositis is more common in tropical climates.

Laboratory test results are often nonspecific. Peripheral leukocytosis and elevated CRP levels and ESR may develop as the infection progresses. Collect blood cultures. Creatine kinase level is often normal. Ultrasonography remains a quick and inexpensive way to demonstrate pyomyositis. CT and MRI may demonstrate the presence of pyomyositis. MRI is more sensitive than CT.

Frank G, Mahoney HM, Eppes SC: Musculoskeletal infections in children. Pediatr Clin North Am 2005;52:1083-1106.
Small LN, Ross JJ: Tropical and temperate pyomyositis. Infect Dis Clin North Am 2005;19:981-989.

16. How is pyomyositis treated?

The organisms most commonly responsible for pyomyositis are *S. aureus* and *S. pyogenes* (often associated with varicella infection). As is the case in osteomyelitis and septic arthritis, MRSA must be considered in these patients and antimicrobial treatment directed appropriately. When an identifiable abscess is present, drainage is indicated. In the interest of isolating an organism, consider delaying the initiation of antibiotics until drained fluid can be collected for culture in stable patients. More extensive surgical intervention may be necessary if there is muscle necrosis.

Frank G, Mahoney HM, Eppes SC: Musculoskeletal infections in children. Pediatr Clin North Am 2005;52:1083-1106.

Small LN, Ross JJ: Tropical and temperate pyomyositis. Infect Dis Clin North Am 2005;19:981-989.

17. Describe the typical presentation and evaluation of suspected necrotizing fasciitis.

Necrotizing fasciitis is a rare, but rapidly progressive and potentially fatal, bacterial infection of the subcutaneous tissue and superficial fascia. Patients with necrotizing fasciitis often have soft tissue swelling and pain near a site of trauma. Initially, pain with manipulation may be out of proportion to the skin findings. Induration and edema follow, then blistering and bleb formation. The skin can become dusky or drain fluid. Subcutaneous pain and tenderness become severe—again, often out of proportion to the appearance of the overlying skin. There is often high fever and signs of systemic illness. The process may be quite fulminant, accompanied by toxic shock syndrome and multiorgan failure.

Laboratory evaluation and imaging, particularly MRI, may be helpful in establishing the diagnosis. However, because of the rapidly progressive nature of necrotizing fasciitis, do not delay initiation of treatment while obtaining laboratory studies and imaging. Collect wound, tissue, and blood cultures (aerobic and anaerobic).

Jamal N, Teach S: Necrotizing fasciitis. Pediatr Emerg Care 2011;27:1195-1202.

Leung AKC, Eneli I, Davis HD: Necrotizing fasciitis in children. Pediatr Ann 2008;37:704-710.

18. What is the treatment for necrotizing fasciitis?

The most important aspect of treatment for necrotizing fasciitis is immediate surgical débridement. Seek surgical consultation as soon as possible. Direct antibiotic treatment at the likely causative organisms, including *S. pyogenes* and *S. aureus* (including MRSA). A reasonable antibiotic choice would be clindamycin plus oxacillin. Many recommend vancomycin in patients who are very ill and in areas where MRSA is prevalent. Ampicillin-sulbactam and piperacillin-tazobactam are other possible antimicrobial choices. It cannot be overemphasized, however, that antibiotic treatment alone is insufficient for this severe, rapidly progressive, and often life-threatening infection. Antibiotic delivery to the affected tissues is often limited and surgical débridement remains essential.

Jamal N, Teach S: Necrotizing fasciitis. Pediatr Emerg Care 2011;27:1195-1202.

Leung AKC, Eneli I, Davis HD: Necrotizing fasciitis in children. Pediatr Ann 2008;37:704-710.

Martz C, Friedlander SF: Subcutaneous tissue infections and abscesses. In Long SS, Pickering LK, Prober CG (eds): Principles and Practice of Pediatric Infectious Disease, 4th ed. Philadelphia, Elsevier Churchill Livingstone, 2012, pp 454-461.

19. What concerns should the ED physician have when a child younger than 4 years presents with back pain?

Back pain is not common in young children, nor is it a particularly common expression of functional pain in young children. Concerning signs and symptoms in a young child with back pain include refusal to walk, limited range of motion in the back, persistent or increasing pain despite rest and analgesics or anti-inflammatory agents, fever, weight loss, signs of systemic illness, and abnormal neurologic signs. Consider serious causes in the young child including diskitis, vertebral osteomyelitis, and tumors.

Haidar R, Saad S, Khoury N, Musharrafieh U: Practical approach to the child presenting with back pain. Eur J Pediatr 2011;170:149-156.

Payne WK, Ogilvie JW: Back pain in children and adolescents. Pediatr Clin North Am 1996;43:899-918.

Rodriguez DP, Poussaint TY: Imaging of back pain in children. Am J Neuroradiol 2010;31:787-802.

Key Points: Orthopedic Emergencies

1. MRSA is an important organism to consider in bone, joint, and soft tissue infections. Provide adequate antimicrobial coverage if it is prevalent in your area. Consult your local antibiogram.
2. Overlying skin findings may not be very remarkable early in the course of pyomyositis and necrotizing fasciitis.
3. Surgical débridement without delay is the most important aspect of treating necrotizing fasciitis.
4. Back pain in a young child warrants serious consideration.

20. How do children with slipped capital femoral epiphysis (SCFE) present?

SCFE is the most common hip disorder in children. SCFE usually occurs during the rapid growth phase of adolescence and tends to occur more often in obese children. Males are affected almost twice as often as females. Black adolescents are affected about twice as often as white adolescents. SCFE may be chronic, acute, or acute superimposed on chronic in its presentation. Most (90%) are stable—those who are unable to bear weight are considered unstable. The presentation of SCFE is bilateral in approximately 20% of cases. Patients with SCFE often have pain in the anterior hip, groin, medial thigh, or knee and will also demonstrate limitation of hip motion. Because pain is localized to the distal thigh or knee, the presence of knee pain in a child or adolescent mandates a thorough examination of the hip. Patients who are ambulatory may have an antalgic gait with external rotation of the affected leg. Passive internal rotation of the hip is painful, and passive hip flexion is associated with compensatory external rotation.

Gholve PA, Cameron D, Millis MB: Slipped capital femoral epiphysis update. Curr Opin Pediatr 2009;12:39-45.

Kienstra A, Macias C: Slipped capital femoral epiphysis. UpToDate, 2014. Available at www.uptodate.com.

21. How can the diagnosis of SCFE be confirmed?

Plain radiography is the first step in the evaluation of patients with suspected SCFE. Films show displacement of the femoral capital epiphysis from the metaphysis through the growth plate. If the slip is chronic, metaphyseal remodeling may be seen. The anteroposterior view often demonstrates the presence of the slip. A line drawn tangent to the superior femoral neck should intersect the lateral aspect of the femoral head (Klein's line). In SCFE this line passes more lateral to the capital epiphysis. Cross-table lateral radiography of the hip can help define the extent of posterior epiphyseal displacement. Although a frog-leg view of the pelvis may reveal a subtle slip, avoid movement of the hip for radiography as it may cause further slippage. Although a slip may be symptomatic on one side only, radiographs often show bilateral SCFE. Because as many as 40% of patients have bilateral slips, comparing sides on plain films may give false reassurance and result in failure to diagnose both slips.

22. What is the approach to the patient with suspected SCFE in the ED?

Once the diagnosis is strongly suspected, obtain urgent orthopedic consultation. Do not allow the patient to bear weight. Most patients are admitted to the hospital for urgent surgical management.

Gholve PA, Cameron D, Millis MB: Slipped capital femoral epiphysis update. Curr Opin Pediatr 2009;12:39-45.

23. What are the features of "growing pains"?

- Pain occurs most commonly in the lower extremities and is bilateral.
- Pain occurs in the evening hours and is *better by morning*.
- Pain can become severe and is relieved by acetaminophen or ibuprofen.
- The physical examination is normal.
- There is often a family history of growing pains.
- Pain is chronic and may last for years.

Harel L: Growing pains: Myth or reality. Pediatr Endocrinol Rev 2010;8:76-78.

Wilking A: Growing pains. UpToDate, 2014. Available at www.uptodate.com.

24. What is the cause of growing pains?

The cause of growing pains remains unknown. Many causes have been suggested. Typically growing pains are not associated with fever or joint inflammation. Growing pains do not correlate with growth spurts and do not affect growth. They do not occur at the growth plate regions. Possible causes include fatigue, abnormal posture, restless legs syndrome, and overuse. Psychological stress and decreased pain threshold have been postulated as contributory. Growing pains are often seen in children with other types of recurrent pain, such as headaches and abdominal pain. They are slightly more common in girls.

Harel L: Growing pains: Myth or reality. Pediatr Endocrinol Rev 2010;8:76-78.
Wilking A: Growing pains. UpToDate, 2014. Available at www.uptodate.com.

25. What are some "red flags" to consider that suggest a disease other than growing pains?

- Pain that is persistent and unilateral (Consider imaging to rule out malignancy or fracture.)
- Fever and weight loss; hepatosplenomegaly or lymphadenopathy (Consider blood work to rule out an oncologic process.)
- Morning stiffness, back or groin pain, synovitis (Consider inflammatory joint disease.)
- Rashes suggesting vasculitis, systemic lupus erythematosis, or psoriasis
- Limp/abnormal gait is unusual in growing pains.

Harel L: Growing pains: Myth or reality. Pediatr Endocrinol Rev 2010;8:76-78.

26. When should one suspect sacroiliitis in children?

Consider sacroiliitis in a child with fever and hip, back, or gluteal pain. Sacroiliitis is relatively uncommon, accounting for approximately 1.5% of all cases of septic arthritis in children. These children are often initially misdiagnosed owing to the condition's rarity, low suspicion on the part of the diagnostician, and rare abnormal findings on plain radiographs. The clinical presentation varies widely, and the condition mimics other, more common disorders (septic arthritis of the hip, diskitis, and even appendicitis). Infection is thought to arise from hematogenous spread or, less commonly, from direct extension from adjacent joint or soft tissue.

Incidence is highest in the second decade of life, presumably because blood supply to the sacroiliac joint peaks at that time.

Grippi M, Zionts LE, Ahlmann ER, et al: The early diagnosis of sacroiliac joint infections in children. J Pediatr Orthop 2006;26(5):589-593.
Taylor ZW, Ryan DD, Ross LA: Increased incidence of sacroiliac joint infection at a children's hospital. J Pediatr Orthop 2010;30(8):893-898.

27. How is the diagnosis of sacroiliitis made?

High clinical suspicion based on physical examination should prompt further investigation. The **FABERE** (**f**lexion, **ab**duction, **e**xternal **r**otation, and **e**xtension) test or Patrick test can be helpful in diagnosis. The patient is placed supine; the hip is flexed, abducted, and externally rotated until the limit of passive motion is reached. The sacroiliac joint is then compressed by downward pressure on the medial aspect of the knee while stabilizing the pelvis. When this causes pain, the test is reported as positive. Also, straight leg raising as well as lateral compression of the pelvis often induces pain.

Laboratory testing is nonspecific: ESR and CRP are often elevated. Peripheral leukocytosis may be seen. Plain radiographs are usually not diagnostic; a bone scan may be negative early in the course of illness, and MRI remains the mainstay of diagnosis for these patients.

Pratte L, Docquier PL: Sacroiliitis in infants: A difficult diagnosis. 4 cases. Joint Bone Spine 2009;76(3):313-315.
Wada A, Takamura K, Fujii T, et al: Septic sacroiliitis in children. J Pediatr Orthop 2008;28(4):488-492.

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CHAPTER 48

OTORHINOLARYNGOLOGY EMERGENCIES

Megan Lavoie and Frances M. Nadel

1. From what part of the nose do most nosebleeds originate?

The vast majority of nosebleeds in children arise anteriorly (90%). On the anterior part of the septum, many capillaries converge, giving rise to Kiesselbach's plexus. Anterior nosebleeds tend to be slow and persistent. Posterior nosebleeds usually originate from branches of the sphenopalatine artery and ethmoidal arteries. As a result, the bleeding is more profuse.

2. What factors often contribute to nosebleeds in otherwise normal children?

Desiccation of the fragile nasal mucosa makes the tissue more friable. Inflammation from a viral upper respiratory tract infection or allergies may also make the mucosa more likely to bleed. Finally, local trauma from nose picking is often a major contributor.

3. How can one elicit a truthful answer about nose picking?

Ask the child which finger she uses to pick her nose!

4. What is "rhinitis sicca"?

This term refers to the drying of the nasal mucosa, which may make it more susceptible to bleeding. Rhinitis sicca often occurs during winter months, when dry hot air systems are being used to heat one's house. It can also occur in the setting of chronic allergic symptoms, or upper respiratory infection, or after sinus surgery, nasal trauma, or radiation.

5. Though most nosebleeds occur from the benign local conditions listed previously, what else should be considered in the differential diagnosis of nosebleeds?

See Table 48-1 for a list of what else should be considered in the differential diagnosis of nosebleeds.

6. What historical factors make one suspicious for a systemic cause of a nosebleed?

Children with a history of bleeding from other sources, easy bruising, weight loss, or other constitutional symptoms; a family history of a bleeding disorder; and severe or recurrent nosebleeds may need a more extensive workup or subsequent follow-up. Children with abnormal vital signs, who are pale, or who have diffuse adenopathy, nasal mass, or suspicion of a posterior source of the bleeding will likely need a more extensive evaluation as well.

Sandoval C, Dong S, Visintainer P, et al: Clinical and laboratory features of 178 children with recurrent epistaxis. J Pediatr Hematol Oncol 2002;24:47-49.

7. What is the most common age of children who present to the emergency department (ED) with nosebleeds?

Nosebleeds requiring an ED visit occur most commonly in children between the ages of 2 and 10 years old, and most have a readily identifiable local cause for their bleed. Nosebleeds in children less than 1 year old are rare. In this age group, consider more serious illness or injury, including child abuse.

McIntosh N, Mok JY, Margerison A, et al: The epidemiology of oro-nasal haemorrhage and suffocation in infants admitted to hospital in Scotland over 10 years. Arch Dis Child 2010;95:810-816.

Pallin DJ, Chng YM, McKay MP, et al: Epidemiology of epistaxis in US emergency departments, 1992 to 2001. Ann Emerg Med 2005;46:77-81.

8. What steps should be taken for a patient who has a nosebleed in the ED?

Calming the patient and family is often the most important step. For an anterior bleed, insert a roll of cotton saturated with a topical decongestant (oxymetazoline or adrenaline) in the affected side and squeeze the nose gently for 5 minutes to compress the cotton against the

Table 48-1. Differential Diagnosis of Epistaxis
Local Predisposing Factors
Trauma
Facial trauma
Direct nasal trauma
Nose picking
Local inflammation
Acute viral upper respiratory tract infection (common cold)
Bacterial rhinitis
Nasal diphtheria (rare),* usually a blood-tinged discharge
Congenital syphilis
Hemolytic streptococci
Foreign body
Acute systemic illnesses accompanied by nasal congestion: measles, infectious mononucleosis, acute rheumatic fever
Allergic rhinitis
Nasal polyps (cystic fibrosis, allergic, generalized)
Staphylococcal furuncle
Sinusitis
Leech infestation
Rhinitis sicca
Cocaine or heroin sniffing
Lobular capillary hemangioma (pyogenic granuloma)
Ectopic tooth
Telangiectasias (Osler-Weber-Rendu disease)
Juvenile angiofibroma*
Other tumors, granulomatosis (rare)*
Systemic Predisposing Factors
Hematologic diseases*
Platelet disorders
Quantitative: idiopathic thrombocytopenic purpura, leukemia, aplastic anemia
Qualitative: von Willebrand's disease, Glanzmann's disease, uremia
Other primary hemorrhagic diatheses: hemophilias
Clotting disorders associated with severe hepatic disease, disseminated intravascular coagulopathy, vitamin K deficiency
Anatomic: pseudoaneurysm of major vessel from infection/trauma
Drugs: aspirin, nonsteroidal anti-inflammatory drugs, warfarin, rodenticide, valproate
Vicarious menstruation
Hypertension*
Arterial (unusual cause of epistaxis in children)
Venous: superior vena cava syndrome or paroxysmal coughing seen in pertussis and cystic fibrosis

*Life-threatening condition.

Adapted from Nadel FN, Henretig FM: Epistaxis. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 236-239.

septum. If the bleeding site is visible, cauterize it with silver nitrate. Add a topical anesthetic to the cotton ball to greatly decrease the amount of discomfort of cautery. If the bleeding cannot be stopped with cautery, then pack the nose. Have bleeding that cannot be stopped with these measures evaluated by an otolaryngologist without delay.

9. What findings would make you more suspicious for a posterior nosebleed?

Suspect a posterior nosebleed when no obvious site is visible anteriorly. Bleeding from both nostrils and blood in the posterior pharynx suggest a posterior nosebleed, but can occur from brisk anterior epistaxis. Continued bleeding despite adequate anterior pressure is also concerning. Posterior bleeding may present more subtly in the form of hematemesis, hemoptysis, or melena. Posterior nasal bleeding is rare in children.

Kucik CJ, Clenney T: Management of epistaxis. Am Fam Physician 2005;71:305-311.

10. A teenage male presents with recurrent, profuse unilateral epistaxis. He reports that this same side of his nose has been progressively obstructed for 2 months. What other diagnosis should you consider?

Teenage males may develop *juvenile nasopharyngeal angiofibroma*. These uncommon tumors arise from the sphenopalatine region and can present with severe epistaxis. The diagnosis is made with nasal endoscopy or computed tomography (CT). These patients need a referral to an otolaryngology specialist for further surgical management.

11. A teenager presents with her fifth nosebleed in as many months. Her history and physical examination are otherwise unremarkable. What additional information regarding her nosebleeds may be helpful?

Ask her about the timing of nosebleeds in relation to her menstrual cycle. Some girls may experience "vicarious menstruation" in which they have a nosebleed at the same time as their menses.

Toppozada H, Michaels L, Toppozada M, et al: The human nasal mucosa in the menstrual cycle. J Laryngol Otol 1981;95:1237-1247.

12. Who should be tested for group A streptococcus pharyngitis with a throat swab?

Children with a sore throat and fever, with lymphadenopathy, palatal petechiae, and tonsillar exudate. Those with rhinnorhea, cough, hoarse voice, and other symptoms indicating a more likely viral cause need not be tested. Also, those younger than 3 years old typically do not need to be tested, as they are more likely to be carriers of group A streptococcus than to have true streptococcal pharyngitis, and the occurrence of acute rheumatic fever is rare in this age group.

Shulman ST, Bisno AL, Clegg HW, et al: Clinical practice guideline for the diagnosis and management of group A streptococcal pharyngitis: 2012 update by the Infectious Diseases Society of America. Clin Infect Dis 2012;55:1279-1282.

13. What are the common signs and symptoms of a retropharyngeal abscess (RPA)?

Commonly, there is a recent history of an upper respiratory tract infection, fever, and poor oral intake. Patients may be drooling, have a stiff neck or torticollis, and have tender cervical adenopathy. Respiratory symptoms of stridor, stertor, or dyspnea are usually late findings and are rarely described in the most current series of patients. A midline oropharyngeal mass can sometimes be seen in the posterior pharynx.

Hoffmann C, Pierrot S, Contencin P, et al: Retropharyngeal infections in children. Treatment strategies and outcomes. Int J Pediatr Otorhinolaryngol 2011;75:1099-1103.

14. Why is it unusual to see RPA in children older than 4 years of age?

The retropharyngeal nodes of Rouvière usually involute around 5 years of age, making RPA a rare disease in older children.

15. What is a parapharyngeal abscess?

A parapharyngeal or lateral space infection develops in the lateral aspect of the neck. This space is shaped like an inverted cone. It is divided into two compartments: (1) the anterior compartment contains no vital structures and is closely related to the tonsillar fossa; (2) the posterior compartment contains some of the cranial nerves and the carotid sheath. Infection in the lateral space results from dental infection, pharyngitis, tonsillitis, parotitis, and mastoiditis.

Parapharyngeal abscesses are less common than RPA, but symptoms are very similar.

16. What are the mechanisms of infection of the retropharyngeal space?

Usually, bacteria in the pharynx spread via lymphatic channels to the nodes of Rouvière, where they multiply and suppurate. Other possible mechanisms include oral trauma (causing a mucosal tear), hematogenous spread during bacteremia, or, rarely, extension from vertebral osteomyelitis.

17. Which pathogens are common in RPA?

RPA is usually a polymicrobial infection and may include *Streptococcus* spp., *Staphylococcus aureus* (including methicillin-resistant strains), and anaerobes. *Haemophilus influenzae* type b has become a less common pathogen since the introduction of the Hib vaccine. Less common pathogens such as *Klebsiella* and *Salmonella* spp., *Candida*, and *Mycobacterium tuberculosis* have been reported.

Abdel-Haq N, Quezada M, Asmar BI: Retropharyngeal abscess in children: The rising incidence of methicillin-resistant *Staphylococcus aureus*. Pediatr Infect Dis J 2012;31:696-699.

18. How wide is the normal retropharyngeal space on a lateral neck radiograph in children?

Prevertebral soft tissues at the level of C3 should measure less than two thirds of the anteroposterior width of the body of the third cervical vertebrae. This space is markedly widened in a child with RPA (Fig. 48-1).

Virk JS, Pang J, Okhovat S, et al: Analysing lateral soft tissue neck radiographs. Emerg Radiol 2012; 19:255-260.

19. List the causes of a false-positive lateral neck radiograph for RPA.

- Neck not in full extension
- Radiography done during expiration
- Not a true lateral position
- Crying child

Virk JS, Pang J, Okhovat S, et al: Analysing lateral soft tissue neck radiographs. Emerg Radiol 2012; 19:255-260.

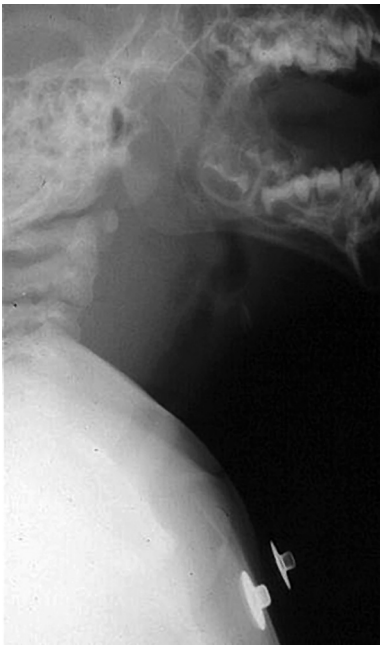

Figure 48-1. Widened retropharyngeal space on a lateral neck radiograph of a child with a retropharyngeal abscess.

20. How does CT assist in RPA management?

A hypodense region with rim enhancement and scalloping of the wall suggests an abscess, but a more heterogeneous mass without rim enhancement suggests retropharyngeal cellulitis only. However, the CT results should be clinically correlated, as the accuracy of CT reported in the literature varies greatly. CT can help demonstrate the extent of infection and the potential for airway compromise. The scan also helps the otolaryngologist decide whether intraoral drainage is feasible or whether an external approach is necessary.

In children with the clinical diagnosis of retropharyngeal infection without evidence of extension of infection beyond the retropharyngeal space, no signs of respiratory compromise, and a lateral neck consistent with a retropharyngeal process, a CT may not be necessary in the initial evaluation.

Grisaru-Soen G, Komisar O, Aizenstein O, et al: Retropharyngeal and parapharyngeal abscess in children—Epidemiology, clinical features and treatment. Int J Pediatr Otorhinolaryngol 2010; 74:1016-1020.

21. Do most patients with RPA require surgery?

The management of RPA varies greatly in the literature and by surgeon. Though it was previously the standard to drain all abscesses, more clinicians are willing to attempt a trial of intravenous (IV) antibiotics alone. Children more likely to need surgical management of their RPA include those with abscesses larger than 2.5 cm, persistent fever after 24 to 48 hours of IV antibiotics, and older age, as well as those with multiple abscesses, airway compromise, or signs of mediastinitis or meningitis.

Wong D, Brown C, Mills N, et al: To drain or not to drain: Management of pediatric deep neck abscesses: A case-control study. Int J Pediatr Otorhinolaryngol 2012;76:1810-1813.

22. How far inferiorly does the retropharyngeal space extend in the neck?

The retropharyngeal space extends from the base of the skull to the level of the second thoracic vertebra. An RPA can rupture into the prevertebral space, which communicates with the mediastinum and descends as far as the psoas muscles.

23. What are some of the complications of RPA?

Earlier recognition and treatment and improved diagnostic strategies have greatly reduced the morbidity and mortality risks of RPAs. However, a large RPA may obstruct the airway at the pharyngeal, laryngeal, or tracheal level, and a tracheostomy may be necessary. Aspiration of purulent fluid may occur from rupture of the abscess. Sepsis, mediastinitis, jugular vein thrombosis, Lemierre's syndrome, and carotid arteritis with subsequent rupture are rare complications.

24. What findings may herald a carotid artery rupture?

A pulsatile ecchymotic mass may be found anterior to the sternocleidomastoid muscle. Bleeding from the ear, nose, or mouth may occur.

Key Points: Dangers of Deep Neck Infections

1. Localized infections in the head and neck can spread regionally.
2. Intracranial extension may present with prolonged symptoms, change in mental status, or focal neurologic deficit.
3. Mediastinal extension can also be associated with prolonged symptoms, poor response to therapy, respiratory symptoms, and sepsis syndrome.

25. Define mastoiditis.

Mastoiditis is an infection of the mastoid air cells. Technically, a simple case of acute otitis media leads to some inflammation of the air cells, but true coalescent mastoiditis implies bony destruction. In younger children, mastoiditis has been found to develop via hematogenous spread, not as a complication of acute otitis media.

Groth A, Enoksson F, Hultcrantz M, et al: Acute mastoiditis in children aged 0-16 years—A national study of 678 cases in Sweden comparing different age groups. Int J Pediatr Otorhinolaryngol 2012;76:1494-1500.

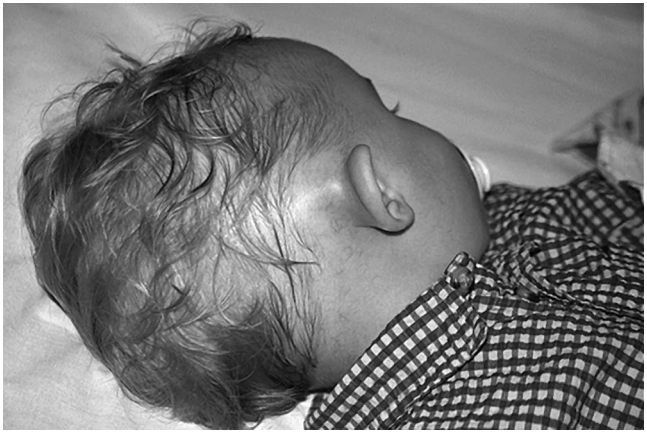

Figure 48-2. Protruding ear in a child with acute mastoiditis.

26. Why is it unusual to see mastoiditis in children less than 1 year old?

The mastoid air cells are not yet pneumatized.

27. How does a patient who has acute mastoiditis present?

Patients commonly present with persistent symptoms of acute otitis media, such as ear pain or drainage. Fever is common. The ear may protrude away from the head, and in younger children the ear is pushed down and out (Fig. 48-2). The postauricular skin is often tender, erythematous, fluctuant, or edematous. Otoscopy shows findings consistent with acute otitis media. One may see sagging of the posterosuperior external ear canal due to periosteal thickening.

Groth A, Enoksson F, Hultcrantz M, et al: Acute mastoiditis in children aged 0-16 years—A national study of 678 cases in Sweden comparing different age groups. Int J Pediatr Otorhinolaryngol 2012;76:1494-1500.

28. What are the common pathogens seen in acute mastoiditis?

Streptococcus pneumoniae, *Streptococcus pyogenes*, and *S. aureus* are common pathogens. Anaerobic enteric bacteria and *Pseudomonas* spp. are more often seen in subacute or chronic disease. *H. influenzae* is a rare pathogen.

Groth A, Enoksson F, Hultcrantz M, et al: Acute mastoiditis in children aged 0-16 years—A national study of 678 cases in Sweden comparing different age groups. Int J Pediatr Otorhinolaryngol 2012;76:1494-1500.

29. Does every child with acute mastoiditis need a CT for diagnosis?

No. If the clinical examination is sufficient to make the diagnosis and there are no concerns for associated complications, a CT may not be necessary. However, a CT with contrast may confirm your diagnosis in less obvious presentations, as well as show whether there is a subperiosteal abscess, intracranial extension, or sigmoid sinus thrombosis.

Tamir S, Schwartz Y, Peleg U, et al: Acute mastoiditis in children: Is computed tomography always necessary? Ann Otol Rhinol Laryngol 2009;118:565-569.

30. What are some of the central nervous system complications of mastoiditis?

The temporal bone's proximity to many important intracranial structures can lead to serious complications. Intracranial extension can include meningoencephalitis and intracerebral, epidural, or subdural abscesses. Progressive thrombophlebitis may lead to sigmoid or lateral sinus thrombosis. Any child with mastoiditis with focal neurologic symptoms should receive an immediate evaluation for intracranial spread. Other concerning findings include toxic appearance, stiff neck, persistent fever despite adequate antibiotic treatment, headache, vomiting, and unusual behavior.

Go C, Bernstein JM, de Jong AL, et al: Intracranial complications of acute mastoiditis. Int J Pediatr Otorhinolaryngol 2000;52:143-148.

31. What is a Bezold's abscess?

This is a type of mastoiditis that has ruptured through the mastoid tip and formed an abscess within the sternocleidomastoid muscle.

32. What is Gradenigo's syndrome?

Gradenigo's syndrome occurs from inflammation of the petrous apex of the temporal bone (petrositis), affecting cranial nerves V and VI. It presents as a suppurative otitis, pain around the eye and ear, abducens nerve paralysis (inability to move the eye laterally), and diplopia. Additional symptoms are facial paresis, vertigo, and fever.

33. What are the two types of postoperative tonsillectomy bleeding?

A common dictum states that bleeding occurs "in 7 minutes or 7 days":

- **Primary hemorrhage** occurs within 24 hours of surgery. This type of bleed is usually detected in the postoperative care unit and rarely will present to the ED.
- **Secondary or delayed hemorrhage** occurs anytime thereafter. It most commonly occurs 7 to 10 days after surgery but can occur as early as 3 days and as late as 21 days.

Liu JH, Anderson KE, Willging JP, et al: Posttonsillectomy hemorrhage: What is it and what should be recorded? Arch Otolaryngol Head Neck Surg 2001;127:1271-1275.

34. What percentage of children have delayed hemorrhage?

About 3% of children have delayed posttonsillectomy hemorrhage. Active bleeding or the presence of a clot in the tonsillar fossa requires a return to the operating room for hemostasis.

Walker P, Gillies D: Post-tonsillectomy hemorrhage rates: Are they technique dependent? Otolaryngol Head Neck Surgery 2007;136:27-31.

35. Is ibuprofen a safe form of pain control after tonsillectomy?

Postoperative pain is a significant source of morbidity after tonsillectomy. Ibuprofen has long been avoided perioperatively because of concern for its effect on platelet function causing increased risk of posttonsillectomy hemorrhage. More recently, however, it has been shown that ibuprofen and other similar nonsteroidal anti-inflammatory drugs (NSAIDs) offer good postoperative pain control without increasing the risk of posttonsillectomy hemorrhage.

Ozkiris M, Kapusuz Z, Yildirim YS, Saydam L: The effect of paracetamol, metamizole sodium and ibuprofen on postoperative hemorrhage following pediatric tonsillectomy. Int J Pediatr Otorhinolaryngol 2012;76:1027-1029.

36. How does the normal healing eschar of oropharyngeal tissue look after tonsillectomy?

The healing operative site will be covered with a grayish white exudate. Beneath the exudate there is usually beefy red granulation tissue. A blood clot will appear dark red, purple, or black and may be located inferiorly near the tongue.

37. What is the significance of seeing a clot on inspection of the oropharynx after tonsillectomy?

A blood clot not only shows you where the offending vessel is located; it also indicates that the vessel is in the earliest stages of hemostasis and can continue to bleed if provoked. *Never remove this blood clot in the ED, as brisk bleeding and loss of the airway can ensue!*

38. Why do children die from postoperative bleeding?

The usual mechanism of death is aspiration of blood, though some may die from hemorrhagic shock.

39. How should you manage a patient who is actively bleeding after tonsillectomy in the ED?

Assess the ABCs (airway, breathing, and circulation), with particular attention to obstruction of airway due to blood, as well as circulatory compromise due to blood loss. It is important to calm the patient and the family. Examination will show if any active bleeding is present. Leave any clot in the tonsillar fossa undisturbed! For severe, active bleeding, tamponade the site with gauze and digital pressure until the otolaryngologist arrives.

40. Is postadenoidectomy bleeding treated differently?

Bleeding from the adenoid bed poses less of a threat because the nasopharynx is supplied with fewer blood vessels than the tonsillar bed. Nonetheless, persistent nasopharyngeal hemorrhage still requires surgical intervention.

Key Points: Management of Bleeding After Tonsillectomy and Adenoidectomy

1. Attend to the ABCs.
2. Restore intravascular volume.
3. If there is a clot, leave it alone.
4. For severe active bleeding, tamponade the site with gauze and digital pressure.

41. What is quinsy?

Quinsy is derived from the Latin word *cynanche*, which means "sore throat," and refers to a peritonsillar abscess (PTA).

42. Define the classic presentation of PTA.

PTAs are most commonly seen in late adolescence but have been reported in young infants. Patients often present with a muffled voice, trismus, and odynophagia. Other symptoms may include fever, worsening sore throat, ear pain, and neck pain.

43. Which way does the uvula point when there is a PTA?

The abscess pushes the uvula away to the contralateral side of the oropharynx.

44. What other physical examination findings may you observe with PTA?

The tonsils and pharynx will be erythematous, and there may be an exudate on the tonsils, as in straightforward tonsillitis. One tonsil may be larger than the other, but by itself this asymmetry does not suggest PTA. Suspect a PTA when the soft palate is red and bulging on one side and the uvula is deviated to the contralateral side. Often there is trismus and ipsilateral cervical adenopathy.

45. What should be considered in your differential diagnosis of a PTA?

It is often difficult to differentiate a peritonsillar cellulitis from an abscess. The significant asymmetry of tonsil size, the presence of uvular deviation, and palatine fullness are more consistent with a PTA. CT scan and ultrasound may help distinguish peritonsillar cellulitis from PTA, reducing the rates of unnecessary drainage procedures. Children with mononucleosis also have very enlarged tonsils with exudate, though they are usually symmetrically enlarged. It is unusual for tonsillitis from other viruses to be confused with a PTA. If the patient has significant respiratory symptoms, one may be concerned about epiglottitis or RPA. Since the introduction of the Hib vaccine, epiglottitis is extremely rare. Though not always true, children with RPAs or epiglottitis are usually younger than 4 years of age, whereas PTAs are usually seen in teenagers.

Rarely, lymphoma may present as an asymmetric tonsillar enlargement, so long-term follow-up to make sure the asymmetry has resolved is important.

Baker K, Stuart J, Sykes J, et al: Use of computed tomography in the emergency department for the diagnosis of peritonsillar abscess. Pediatr Emerg Care 2012;28:962-965.

Tulin Kayhan F, Ozkul N: Case report: Extranodal non-Hodgkin's lymphoma of the parapharyngeal space. Auris Nasus Larynx 1999;26:201-205.

46. What are the common pathogens in PTA?

As one would imagine, *S. pyogenes* is the most common pathogen. *S. aureus* and oral anaerobes, especially *Fusobacterium necrophorum*, are common pathogens as well.

Rusan M, Klug TE, Ovesen T: An overview of the microbiology of acute ear, nose and throat infections requiring hospitalisation. Eur J Clin Microbiol Infect Dis 2009;28:243-251.

47. How is a PTA treated?

When physical examination suggests a PTA, aspirate the peritonsillar space with an otolaryngologist. Aspiration of pus confirms the diagnosis and often gives the patient immediate symptomatic relief. Some practitioners prefer a course of IV antibiotics, with aspiration reserved for treatment failures. Once drained, treat a PTA just like acute tonsillitis:

with antibiotics, hydration, and analgesics. Repeated aspirations are occasionally necessary. Penicillin usually provides adequate coverage. For patients who are allergic or have not responded to penicillin, clindamycin provides excellent coverage. The route of administration will depend on the patient's general appearance and ability to swallow.

Johnson RF, Stewart MG, Wright CC: The contemporary approach to the diagnosis and management of peritonsillar abscess. Otolaryngol Head Neck Surg 2005;13:157-160.

48. What is Lemierre's syndrome?

Lemierre's syndrome is a septic thrombophlebitis of the internal jugular vein. Pharyngitis and PTAs are the most common initial sites of infection, but it is also seen after other local infections such as otitis media, mastoiditis, or sinusitis.

49. What are the four findings of Lemierre's syndrome?

1. Pharyngitis/tonsillitis
2. Septicemia with at least one positive blood culture, usually of *F. necrophorum*
3. Evidence of internal jugular thrombosis
4. Metastatic focus of infection, often pulmonary

50. What findings would make you suspect Lemierre's syndrome?

Patients with recent or prolonged pharyngitis who have persistent fever, lateral neck pain/swelling, myalgias/arthralgias, and pulmonary decompensation should arouse suspicion. Patients are often quite ill-appearing. Suspect central nervous extension if the patient has headaches, meningismus, cranial nerve palsies, or seizures.

Kuppalli K, Livorsi D, Talati NJ, et al: Lemierre's syndrome due to Fusobacterium necrophorum. Lancet Infect Dis 2012;12:808-815.

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The authors wish to thank Dr. Douglas M. Nadel for his contributions to this chapter in the previous edition.

UROLOGIC EMERGENCIES

Kate M. Cronan

CHAPTER 49

1. How is the diagnosis of testicular torsion made?

- **Symptoms:** Scrotal pain. Pain may be referred to the abdomen. Other symptoms include nausea, vomiting, and diaphoresis.
- **Physical examination:** A torsed testis is typically swollen and tender and lies higher in the scrotum than that on the contralateral side. Erythema of the scrotal skin may or may not be present. Edema increases with time. In the case of complete torsion, the cremasteric reflex is absent. The presence of a brisk cremasteric reflex with a 2- to 3-cm testicular shift makes the diagnosis of testicular torsion very unlikely.
- **Studies:** Color Doppler ultrasonography has become the imaging study of choice. However, no clinical findings, laboratory tests, or radiographic studies can approach the sensitivity of surgical exploration.

2. How is testicular torsion differentiated from torsion of the appendix testis?

Although it can happen at any age, torsion of the appendix testis is most common from ages 7 through 12 years. Pain in torsion of the appendix testis is reportedly not as severe as that seen in testicular torsion. Classically, the *blue dot sign* on the scrotum indicates the site of the infarcted appendage, although as edema increases this may become obscured. In the early stages, pain may be localized initially to the upper pole of the testis, with the remainder of the testis nontender. Doppler ultrasonography reveals normal or increased blood flow to the affected testis in the case of torsion of the appendix testis, whereas arterial blood flow is absent or diminished in the case of testicular torsion. None of these studies is perfect, and changes in blood flow may be missed, especially in the case of intermittent torsion. Treatment for torsion of the torsed appendix includes analgesics or anti-inflammatory medications and rest. Pain usually resolves in 2 to 5 days.

3. Can testicular torsion be intermittent?

Yes. With intermittent testicular torsion the patient has symptoms compatible with testicular torsion: acute scrotal pain and swelling and in some cases nausea and vomiting. The pain is intermittent, with quick resolution. The clinical assessment may be normal, or there may be findings on physical examination such as swelling of the spermatic cord, mobile testes, or an anterior epididymis. Ultrasound results may be normal or show a pseudomass in the spermatic cord.

Those patients with a normal physical examination and ultrasound may be discharged with comprehensive instructions and a plan for close follow-up.

Eaton S, Cendron MA, Estrada CR, et al: Intermittent testicular torsion: Diagnostic features and management outcomes. J Urol 2005;174(4 Pt 2):1532.

4. Why does epididymitis occur more frequently in adolescent males?

Epididymitis indicates the presence of inflammation of the epididymis. It generally results from infection, which may lead to an enlarged and tender epididymis on palpation. Patients may also have symptoms of urinary tract infection (UTI), including dysuria and frequency. Most cases in young men are caused by sexually transmitted organisms, predominantly *Chlamydia trachomatis*, followed by *Neisseria gonorrhoeae* and *Ureaplasma urealyticum*. These organisms are common among adolescent boys. Treat patients for both chlamydial infection and gonorrhea. Epididymitis is extremely rare among prepubertal boys; if it is diagnosed, further investigation is warranted to rule out structural abnormalities of the urinary tract.

5. How is epididymitis treated?

Outpatient therapy is appropriate. Treatment for sexually transmitted disease–related epididymitis includes a single dose of ceftriaxone, 250 mg intramuscularly, and doxycycline,

100 mg orally twice daily for 7 to 10 days. The patient's sexual partner(s) need to be treated as well. Treat prepubertal children who have evidence of a UTI as outpatients with trimethoprim-sulfamethoxasole or a cephalosporin. Scrotal support and nonsteroidal anti-inflammatory drugs (NSAIDs) are also helpful to relieve pain.

Admit patients who are febrile and toxic-appearing to the hospital for treatment with intravenous (IV) antibiotics, and possibly for further diagnostic tests (e.g., testicular ultrasonography) to exclude a scrotal or testicular abscess.

Brenner J, Ojo A: Causes of scrotal pain in children and adolescents. UpToDate, [2013]. Available from www.uptodate.com. Accessed November 2013.

Workowski KA, Berman SM: Sexually transmitted diseases treatment guidelines, 2006. Centers for Disease Control and Prevention. MMWR Recomm Rep 2006;55(RR-11):1.

6. Describe the difference between phimosis and paraphimosis.

Both of these conditions are diagnosed in the uncircumcised male. *Phimosis* occurs when the distal foreskin is too tight to retract over the glans penis. *Paraphimosis* occurs when the foreskin is left in the retracted position, becomes swollen, and is then unable to be reduced (Fig. 49-1). Phimosis is not generally a problem in children, and is normal in boys younger than 6 months. Retraction of the foreskin should not be attempted routinely in infants. In contrast, reduce a paraphimosis in the emergency department (ED). This is generally accomplished by applying lidocaine gel to the swollen foreskin, followed by steady pressure on the glans, and then pulling the foreskin forward while pushing the glans backward with the thumb. A dorsal penile block with local anesthetic may make the patient more comfortable, but it is generally not needed.

7. What is balanoposthitis?

Balanoposthitis occurs in uncircumcised boys. It is an infection of the foreskin that may also affect the glans. If the foreskin is irritated as the result of poor hygiene, a break in the skin may occur, allowing bacteria to establish a skin infection. Treatment depends on the type of balanoposthitis. Voiding may be more comfortable while the patient is sitting in a tub of warm water. True phimosis may occur as a complication, resulting from

Figure 49-1. Paraphimosis. *(Photograph by John Loiselle, MD; used with permission.)*

scarring after the inflammatory reaction. Circumcision may be necessary to avoid recurrent infections. The term *balanitis* is used to describe an infection involving only the glans penis.

8. What are other causes of penile swelling in children?

Penile swelling is usually painful and results from infection, sickling of red blood cells (boys with hemoglobin SS), or trauma. Other considerations in the presence of isolated, nontender penile swelling include insect bite (look for the lesion), allergic reaction, or more generalized edematous states, including renal, cardiac, or hepatic problems.

9. How do a "communicating" and a "noncommunicating" hydrocele differ?

A hydrocele is an accumulation of fluid within the tunica vaginalis surrounding the testis. Although it may arise in the case of torsion, trauma, tumor, or epididymitis, a simple hydrocele may be present in the absence of any underlying testicular abnormality. Infants may be left with a simple noncommunicating hydrocele when fluid is trapped around the testis after the *processus vaginalis has closed* during development. The baby presents with painless scrotal swelling of constant size. Management consists of observation only, as most are resorbed by the age of 12 to 18 months.

If the history is of scrotal swelling that "comes and goes," especially with crying or exertion, the same fluid may be present, but *the processus vaginalis has not closed*. This allows "communication" between the scrotum and the abdominal cavity. Treatment in this case is surgical exploration with ligation of the processus vaginalis and drainage of the hydrocele.

10. How can a hydrocele be differentiated from an inguinal hernia in an infant?

The history given with an *inguinal hernia* is generally that the parents have observed a nonpainful, nonerythematous scrotal and inguinal swelling in males, or inguinal swelling in females. The swelling typically occurs when the baby is crying or straining. Sometimes bowel sounds can be heard over the mass. In contrast, the swelling of a *hydrocele*, though it may extend toward the inguinal canal, will not be felt as a mass at the internal inguinal ring.

Transillumination with a high-intensity light does *not* definitively make the distinction. Although the mass of a hydrocele generally does transilluminate, transillumination of a hernia can be variable.

11. What should be done to reduce an inguinal hernia?

First determine that the testes are bilaterally descended. Apply steady, gentle pressure to the mass in the direction of the inguinal ring. Often the mass will slip easily back into the abdominal cavity. It may take several minutes of steady pressure to accomplish this goal. If the hernia remains difficult to reduce, it may be necessary to sedate the child (e.g., with midazolam) and place him or her in the Trendelenburg position. This may be enough to allow the child to relax and the hernia to reduce.

12. What is a ureterocele?

A ureterocele is a dilation of the distal ureter that produces a cystic structure within the bladder or the urethra. Ureteroceles can prolapse out of the bladder neck, and in doing so may cause bladder outlet obstruction. In a female, a ball-valving ureterocele is the most common cause of bladder outlet obstruction. These patients will present with a painless pink cystic structure that is bulging out between the labia. This may be reduced by placing a feeding tube within the bladder. Ureteroceles should be managed by urologists, who will usually opt to carry out an endoscopic puncture and decompression of the obstructed system.

Pohl HG: Recent advances in the management of ureteroceles in infants and children: Why less may be more. Curr Opin Urol 2011;21(4):322-327.

13. How can one distinguish between a prolapsed ureterocele and a prolapsed urethra?

The differentiation of a prolapsed ureterocele and urethral prolapse can usually be made by physical examination. A urethral prolapse appears as a red or purplish doughnut-shaped mass. It is usually not tender and is 1 to 2 cm in diameter (Fig. 49-2). A small central dimple

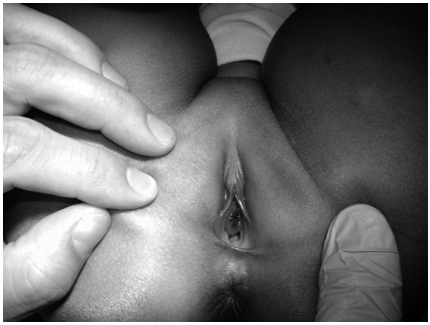

Figure 49-2. Urethral prolapse. *(Photograph by Steven M. Selbst, MD; used with permission.)*

indicates the lumen of the urethra. Rarely, there may be a dark ring of necrotic tissue around the edge of the prolapse. In contrast, the prolapsed ureterocele is a cystic swelling of the terminal ureter and appears as a cystic mass protruding from the urethra. It is often associated with ureteral duplication.

14. What is the most common cause of severe obstructive uropathy in children?

Posterior urethral valves are most common and occur only in boys. If the condition is not diagnosed in utero, the infant presents with a palpably distended bladder and a weak urinary stream. Urgent urologic consultation is indicated.

Caione P, Nappo SG: Posterior urethral valves: Long-term outcome. Pediatr Surg Int 2011;27 (10):1027-1035.

15. What is the first evaluation to do for a patient with "blood in the urine"?

First confirm that the red urine actually contains blood! Urine can appear red as the result of ingestion of a number of foods (blackberries, beets, red Kool-Aid drink, Fruit Loops cereal), substances (aniline dyes, urates), or drugs (phenazopyridine, phenolphthalein).

A urine dipstick test can quickly and easily determine whether blood is present. If the result is positive, send the urine for microscopic analysis to determine the presence or absence of red blood cells. Myoglobin will also give a positive result for blood on the dipstick. Consider the presence of more than 5 to 10 red blood cells per high-power field to be hematuria, often warranting further workup.

16. Describe the differences among bacteriuria, cystitis, pyelonephritis, and urethritis.

Bacteriuria refers to the presence of bacteria anywhere in the urinary tract. It may or may not cause symptoms.

Cystitis and pyelonephritis are part of the continuum of UTIs. *Cystitis* is bacteriuria with invasion of the bladder mucosa. Patients with cystitis present with urgency, frequency, and dysuria, and may on occasion develop a low-grade fever. *Pyelonephritis* occurs when the UTI has localized within the renal parenchyma and causes systemic symptoms; thus, it is the most serious of UTIs. Children present with fever (temperature often $> 39^{\circ}$ C), pyuria, and bacteriuria, often accompanied by nausea, vomiting, leukocytosis, flank pain, and tenderness.

Urethritis indicates inflammation or an infection localized to the urethra. There is usually a discharge, and in adolescents, urethritis generally is a manifestation of a sexually transmitted disease (*C. trachomatis* or *N. gonorrhoeae* infection). Urethritis can also result from noninfectious causes, including local irritation from detergents, fabric softeners, soaps, or bubble baths, as well as minor injury.

17. Is malodorous urine associated with UTI in infants and toddlers?

Yes. When malodorous urine is reported by parents of infants and toddlers, it increases the probability of UTI. However, the specificity and sensitivity are not high enough to definitively rule in or rule out a UTI. In a young child (>3 months) with foul-smelling urine, consider ordering a urine culture.

Gauthier M, Gouin S, Phan V, Gravel J: Association of malodorous urine with urinary tract infection in children aged 1 to 36 months. Pediatrics 2012;129(5):885-890.

18. What are the most common organisms in UTI?

Escherichia coli is isolated in about 80% of cases. Other less common organisms include *Enterobacter*, *Klebsiella*, and *Proteus* spp. *Enterococcus* sp. occasionally is isolated in patients of any age. *Staphylococcus saprophyticus* and *Staphylococcus epidermidis* are seen in adolescents, and group B streptococci are found in infants and during pregnancy. In immunocompromised and chronically ill patients, as well as patients with anatomic abnormalities or indwelling catheters, *Pseudomonas aeruginosa*, *Candida albicans*, and *Staphylococcus aureus* account for a small percentage of UTIs.

19. Which age groups most commonly get UTIs?

Overall, UTIs are more common in neonates and infants and in sexually active adolescent females. As many as 3% to 5% of febrile young infants presenting to the ED have a UTI. In neonates and infants under the age of 6 months, males have a higher incidence, perhaps because of a higher incidence of anomalies of the urinary tract. After this age, UTI becomes less common in males and is rare beyond the age of 1 year. In contrast, girls have a rate of UTI as much as 10-fold higher than that of boys between the ages of 6 months and 2 years, with the risk being greater in the first year than in the second.

20. List the variables that have optimal diagnostic accuracy in diagnosing UTI in young females.

- Age younger than 1 year
- Temperature higher than 39° C
- White race
- Fever for longer than 2 days
- Absence of another source of fever on history or examination

Note: The presence of three or more of the factors on the preceding list supports the best accuracy in making the diagnosis.

Heffner V, Gorelick M: Pediatric urinary tract infection. Clin Pediatr Emerg Med 2008;9(4):233-237.

21. How does the clinical presentation of UTI vary with the age of the patient?

Neonates and very young infants with UTI may present with fever or with nonspecific symptoms, including poor feeding, vomiting, diarrhea, irritability, jaundice, and even seizures. This is clearly why assessment of the urine is an integral part of the "full sepsis workup" performed on infants with any of these presenting symptoms.

Older infants and children up to the age of 2 years often have fever, and parents may notice such urinary symptoms as change in voiding pattern or foul-smelling urine. *Preschool and school-age children* describe specific urologic symptoms, such as frequency, urgency, dysuria, and enuresis. They may also report less specific symptoms, including abdominal pain and vomiting.

Key Points: Clinical Presentation of Urinary Tract Infections by Age

1. **Neonates:** Nonspecific symptoms, fever
2. **Infants up to age 2 years:** Fever, voiding issues, foul-smelling urine
3. **Preschool and school-age children:** Dysuria, frequency, urgency, abdominal pain

22. When should one get a catheterized urine specimen as opposed to a "clean-catch" specimen?

Examine the external genitalia for signs of inflammation or infection. Obtain a catheterized urine specimen (or in some instances a specimen by suprapubic tap) in any patient who lacks bladder control (e.g., infants and toddlers), has evidence of vaginitis, or is unable to provide an adequate midstream specimen. Putting a urine "bag" on any infant or toddler is not helpful if the specimen is being collected for culture, because the urine will probably be contaminated with perineal flora no matter how much preparation is done. This type of collection is reliable only if the culture has no growth. If you suspect that a child has a UTI, it is worth getting the most accurate specimen possible.

Finnell SM, Carroll AE, Downs SM; Subcommittee on Urinary Tract Infection: Technical report—Diagnosis and management of an initial UTI in febrile infants and young children. Pediatrics 2011;128(3):e749.

23. What is the significance of a positive test result for nitrites on the urine dipstick?

Most urinary pathogens (with the exception of *enterococcus*) are able to reduce urine nitrates to nitrite. Hence, a positive test result may indicate the presence of pathogenic bacteria.

24. What is the significance of a positive leukocyte esterase test result?

Leukocytes are able to convert indoxyl carboxylic acid to an indoxyl moiety. This test is highly specific for pyuria. Glucose or protein in the urine can cause a false-negative result for leukocyte esterase.

25. How should dipstick indicators be used to screen for UTI?

Many studies have been published regarding the usefulness of these tests in diagnosing UTI. Most of the studies have been performed in adult patients, and results of studies in children frequently differ. A meta-analysis by Gorelick and Shaw concludes that the presence of nitrites or leukocyte esterase or both on a dipstick test is almost as sensitive as a Gram stain in detecting UTI in children.

Gorelick MH, Shaw KN: Screening tests for urinary tract infection in children: A meta-analysis. Pediatrics 1999;104:e54.

Subcommittee on Urinary Tract Infection, Steering Committee on Quality Improvement and Management: Urinary Tract Infection: Clinical practice guideline for the diagnosis and management of the initial UTI in febrile infants and children 2 to 24 months. Pediatrics 2011;128:595.

Key Points: Screening for Infection via Urine Dipstick

1. **Nitrite positive:** Indicates probable pathogenic bacteria (does not screen for *enterococcus*)
2. **Leukocyte esterase positive:** Specific for pyuria

26. Do white blood cells in the urine always signify a UTI?

No, not always. Pyuria can be found in the absence of bacteriuria (i.e., "sterile pyuria"). Vaginitis can cause the leukocyte esterase test result to be positive, but this does not necessarily mean the patient has a UTI. In addition, an inflammatory process in the abdomen (e.g., appendicitis, inflammatory bowel disease, pelvic inflammatory disease) or Kawasaki disease can cause white blood cells to appear in the urine. A recent meta-analysis shows that the presence of white blood cells in the urine (either spun or unspun) is only about 75% sensitive for UTI.

Heffner V, Gorelick M: Pediatric urinary tract infection. Clin Pediatr Emerg Med 2008;9(4):233-237.

Roberts KB: Revised AAP guideline on UTI in febrile infants and young children. Am Fam Physician 2012;86(10):940-946.

Subcommittee on Urinary Tract Infection, Steering Committee on Quality Improvement and Management: Urinary Tract Infection: Clinical practice guideline for the diagnosis and management of the initial UTI in febrile infants and children 2 to 24 months. Pediatrics 2011;128:595-610.

27. Is a Gram stain of the urine helpful?

The presence of any bacteria on Gram stain of an uncentrifuged urine specimen is about 93% sensitive for detection of UTI, compared with culture as the reference standard. It does, however, require trained personnel to prepare the sample and read the results, and it may not be available to the emergency physician at all times. Fortunately, dipstick analysis has been shown to be almost as sensitive in detecting UTI and is a readily available, quick, easy, and inexpensive alternative.

Gorelick MH, Shaw KN: Screening tests for urinary tract infection in children: A meta-analysis. Pediatrics 1999;104:e54.

Subcommittee on Urinary Tract Infection, Steering Committee on Quality Improvement and Management: Urinary Tract Infection: Clinical practice guideline for the diagnosis and management of the initial UTI in febrile infants and children 2 to 24 months. Pediatrics 2011;128:595-610.

28. When should a urine culture be ordered?

An evidence-based review of the recent literature suggests that for children younger than 36 months of age who present with fever without source or UTI symptoms (e.g., dysuria, vomiting, abdominal pain), a urine culture should be obtained by using an age-appropriate method. If the child is 3 years of age or older, send a culture if the urine dipstick result is positive for nitrite or leukocyte esterase, or if the patient is symptomatic (even with a negative dipstick result).

29. Based on the most recent AAP Guidelines for the diagnosis of UTI, what is required for diagnosis?

A UTI is diagnosed when there are both pyuria and at least 50,000 colonies per milliliter of a single organism in a correctly collected specimen of urine.

Subcommittee on Urinary Tract Infection, Steering Committee on Quality Improvement and Management: Urinary Tract Infection: Clinical practice guideline for the diagnosis and management of the initial UTI in febrile infants and children 2 to 24 months. Pediatrics 2011;128:595-610.

30. Which patients should be treated with antibiotics?

Treat patients of any age presumptively if the dipstick result is positive for moderate leukocyte esterase or nitrite, if they have symptoms of UTI, or if follow-up is in question. For patients with a negative dipstick result (or trace leukocyte esterase but otherwise a negative result), no symptoms other than fever, and good follow-up, it is prudent to defer treatment while awaiting results of the culture.

Treat febrile infants younger than 1 month of age empirically with antibiotics because of the risk of serious bacterial illness. Thoroughly evaluate infants 1 to 2 months of age, and treat them if there is evidence of a UTI.

31. What should be the duration of treatment for UTI with fever?

A 7- to 14-day course is recommended for antibiotic therapy in children with UTI and fever. High failure rates have been documented for shorter courses of treatment.

Subcommittee on Urinary Tract Infection, Steering Committee on Quality Improvement and Management: Urinary Tract Infection: Clinical practice guideline for the diagnosis and management of the initial UTI in febrile infants and children 2 to 24 months. Pediatrics 2011;128:595-610.

32. Which factors in patients with UTI indicate they should be admitted for parenteral antibiotics?

- Age younger than 2 months
- Toxic appearance; signs of shock
- Immunocompromised patient
- Vomiting or not able to tolerate oral medication
- Suspicion for lack of adequate outpatient follow-up
- Failure of outpatient therapy

Shaikh N, Hoberman A: Urinary tract infections in infants and children older than one month: Acute management, imaging and prognosis. UpToDate, [2014]. Available from www.uptodate.com. Accessed March 2014.

33. Define urinary retention.

Urinary retention is the inability to pass urine from the bladder through the urethra. Because urine is being produced normally, the bladder may become largely distended, causing substantial discomfort to the patient.

34. What causes urinary retention in infants?

In the infant, most urinary retention is the result of obstruction. In males, the most common cause is obstruction of posterior urethral valves; other causes include urethral polyps,

strictures, diverticula, trauma, infection and, rarely, meatal stenosis. Urinary retention has been noted as a complication of the Plastibell device used for circumcision. Female infants may experience a prolapsing ureterocele, urethral prolapse, trauma, infection or a foreign body. Neurologic urinary retention may result from a spinal cord lesion.

35. How can sexual abuse contribute to urinary retention?

Always be aware that urinary retention or dysfunctional voiding may on rare occasions be the presenting symptom of sexual abuse. Children who have been sexually abused are often very fearful of any processes involving the genitalia and seek to avoid that portion of their anatomy. Furthermore, abusive acts may result in genital or urethral trauma or infection. Ask appropriate questions if no other cause for urinary retention can be found.

36. What should be done in the ED about urinary retention?

The approach depends on the cause. Refer newborn infants with ineffective voiding to a urologist for further evaluation. While waiting for the consultant, obtain a basic metabolic panel to check electrolytes, blood urea nitrogen (BUN), and creatinine. A suprapubic tap may be necessary to obtain a specimen for urinalysis. Consult a neurologist if a spinal cord lesion is suspected on the basis of physical examination. Older children who have previously voided normally may be catheterized for urinalysis if they are unable to void. Treat infections with appropriate antibiotics. Treat patients with retention from trauma to the urethra with frequent sitz baths (at least three times per day). Discontinue medications causing retention if possible. Refer patients with urinary retention suspected to be of psychosomatic origin or as the result of sexual abuse to psychiatric and sexual abuse management specialists as appropriate. If the patient is unable to void, catheterization may be necessary.

37. What causes renal stones in children?

Renal stones (calculi) are less common in children and adolescents than in adults. Seven percent of urinary calculi occur in children younger than 16 years of age. The cause of calculi depends in part on geography. In the United States, most stones are attributed to metabolic causes, and most contain calcium. Infectious stones are more common in European children, and uric acid stones occur in Southeast Asian children. Boys and girls are equally likely to be affected, and 94% of stones occur in white persons.

Rodig NM: Renal and electrolyte emergencies. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1099-1126.

38. What are the symptoms of urolithiasis in children?

Unlike the classic presentation of excruciating abdominal pain seen in adults, symptoms in children can be far less specific. The patient may appear uncomfortable and may report flank pain, abdominal pain, or costovertebral angle tenderness. Intense pain may cause an elevation in heart rate and blood pressure. Almost all children have some degree of hematuria, which may be microscopic or macroscopic. They may also have symptoms of dysuria, urinary frequency, or urinary retention. The cause of these symptoms needs to be distinguished from UTI.

39. How is the diagnosis of urolithiasis confirmed?

The first test to obtain is a urinalysis, to look for hematuria and for crystals in the urinary sediment. In recent years, spiral computed tomography has become the accepted gold standard study for the detection of urolithiasis, albeit with higher radiation exposure.

40. What treatment for urolithiasis should be initiated in the ED?

The foremost issue that must be addressed in the ED is pain management. NSAIDs and opioids are used to treat pain associated with acute nephrolithiasis. If the pain is not relieved, admit the patient to the hospital for further pain medication and IV fluids. Obtain a urologic consultation for any patient with evidence of obstruction, because endoscopic stenting may be indicated.

Lendyay T, Smith J, Stapleton F: Acute management of nephrolithiasis in children. UpToDate, [2014]. Available from www.uptodate.com. Accessed May 2014.

Rodig NM: Renal and electrolyte emergencies. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1099-1126.

41. Which patients with urolithiasis need to be admitted?

Admit any patient with evidence of obstruction for urologic consultation, because immediate intervention (e.g., endoscopic stenting or ureteroscopy with stone extraction) is necessary to relieve the obstructing stone. Also consider admission for patients with severe pain requiring parenteral analgesia, vomiting, or dehydration and inability to tolerate oral hydration. Also admit patients with renal insufficiency, structural abnormalities of the genitourinary tract, or a solitary kidney.

Bartosh S: Medical management of pediatric stone disease. Urol Clin North Am 2004;31:575-588.

42. What are the presenting symptoms of ureteropelvic junction (UPJ) obstruction in older children?

With UPJ obstruction there is a history of episodic pain that can occur in the abdomen, flank, or back. Nausea and vomiting may occur. The episodes of pain last from 30 minutes to several hours. UPJ obstruction presents more commonly on the left side and is bilateral in 10% of cases. The male-to-female ratio is 2:1. Examination may reveal an enlarged kidney and tenderness.

43. What is the most common cause of acute glomerulonephritis (AGN)?

There are numerous causes of AGN, but in children the most common is *postinfectious* or *poststreptococcal nephritis*. This syndrome occurs predominantly in school-aged children and is characterized by the sudden appearance of either grossly bloody or tea-colored urine, peripheral edema, and decreased urine output. Patients generally present 1 to 2 weeks after having had a sore throat, and sometimes 2 to 3 weeks after a case of impetigo. Other infectious causes include viral upper respiratory tract infections and mononucleosis syndromes.

44. What are the physical findings in AGN?

Children with AGN may develop peripheral edema. The edema is firm and initially appears around the eyes. Weight gain is frequently noted. Patients may have mildly elevated blood pressure. Some children have no symptoms other than abnormally colored, dark, or bloody urine. In others, hypertension can be significant and lead to complications requiring emergency intervention.

45. What laboratory tests should I order for suspected AGN?

The most important test is the urinalysis, with a microscopic examination of the urine. Dysmorphic red blood cells and red blood cell casts are diagnostic of this condition. Significant proteinuria is also characteristic. White blood cells may be present, and because hematuria and proteinuria may be presenting signs of a UTI, order a urine culture in the initial evaluation.

Other studies include complete blood count with differential, platelet count, erythrocyte sedimentation rate, serum electrolytes, calcium, BUN, creatinine, total protein, albumin, and complement (C3 and C4). Test for antistreptolysin O titer if poststreptococcal AGN is considered; antinuclear antibody and anti–double-stranded DNA should be done in the case of systemic lupus erythematosus. Order prothrombin time and partial thromboplastin time if there is concern about a bleeding diathesis. A throat culture may reveal group A streptococcal infection, and chest radiography may be important to evaluate heart size and to look for signs of congestive heart failure (CHF) in a hypertensive patient.

46. What are the goals of ED management of AGN?

The goal is aggressive treatment of life-threatening complications, particularly hypertension, CHF, and hyperkalemia. If the blood pressure is elevated but the patient is asymptomatic, oral antihypertensives (sublingual nifedipine or captopril) may be adequate. In the patient with acute neurologic changes, give antihypertensives (diazoxide or hydralazine) intravenously. Treat CHF by keeping the head of the bed elevated, providing supplemental oxygen, and promoting diuresis with furosemide (0.5-1.0 mg/kg via IV route). Tailor fluid management to restrict volume and sodium and replace only insensible losses plus urine output. Do not give potassium until the patient's urine output is established and hyperkalemia is resolved.

Admit children with AGN to the hospital.

47. What is the most common cause of nephrotic syndrome in children?

Nephrotic syndrome is a constellation of findings:

- Edema
- Proteinuria
- Hypoalbuminemia
- Hyperlipidemia

It can occur in children as a primary renal disorder or secondary to systemic disease, environmental toxins (heavy metals, bee venom), or medications. About 90% of pediatric patients between the ages of 1 and 10 years and 50% of those older than age 10 are associated with idiopathic nephrotic syndrome.

48. How is the diagnosis of nephrotic syndrome made?

After physical examination, evaluate urine for the presence of protein. Measure total serum protein, albumin, and cholesterol levels. Typical values in nephrotic syndrome are a urinary protein of 3 to 4+ on dipstick, serum albumin level lower than 3 g/dL, and elevated cholesterol level. Other laboratory values that may be important for management include the hematocrit (which may be elevated secondary to intravascular volume depletion), electrolytes (sodium is often low), and BUN/creatinine (BUN level may initially be elevated, reflecting the lowered intravascular volume; creatinine level may be normal or elevated depending on whether there is primary renal damage).

49. What are the five major complications from idiopathic nephrotic syndrome?

- Infection
- Thromboembolism
- Renal insufficiency
- Anasarca
- Hypovolemia

50. What management decisions need to be made in the ED for a child with nephrotic syndrome?

Complications requiring emergency management include the progression from hypovolemia to shock, hypercoagulability, and pleural effusions and ascites leading to difficulty walking, abdominal pain, or respiratory distress. The child must be evaluated for peritonitis, usually due to *Streptococcus pneumoniae*. Consider antibiotics if the child is febrile, or if peritonitis is otherwise suspected, after obtaining a blood culture. Give normal saline boluses (20 mL/kg) to restore circulation, if needed. For symptomatic massive edema, give furosemide orally, or in extreme cases, albumin followed by IV furosemide. Rarely is paracentesis necessary. Avoid deep venipunctures because of the increased risk of thromboembolic events.

51. Should all patients with nephrotic syndrome be admitted?

Admit those with the following:

- Newly diagnosed patients
- Patients in whom there is a possibility of infection
- Any who are symptomatic from dehydration or edema

Contact a pediatric nephrologist regarding steroid therapy and indications for renal biopsy.

52. What type of priapism is typically seen in patients with sickle cell disease?

Low-flow priapism is the most common type of priapism in these patients. Stasis develops, followed by hypoxia leading to acidosis of venous blood. This process occurs during a normal erection and results in sickled erythrocytes within the venous system of the corpora cavernosa. Venous outflow of the corporeal bodies is thus obstructed. The corpora cavernosa are firm and tender.

Donaldson JF, Rees RW, Steinbrecher HA: Priapism in children: A comprehensive review and clinical guideline. J Pediatr Urol 2014;10(1):11-24.

ABDOMINAL TRAUMA

Margarita S. Lorch

1. What are the most common mechanisms of injury for pediatric patients with abdominal trauma?

In the United States, blunt mechanisms account for 80% to 90% of abdominal trauma in children. The most common causes are motor vehicle collisions (children are injured as passengers or pedestrians) and falls.

2. Is the age of a patient a factor for abdominal injuries?

Yes. Children have compact torsos with a larger organ/body mass ratio than adults. Forces delivered to the abdomen dissipate over a smaller area, leading to increased risk of injury. Infants and toddlers have a larger portion of the liver and spleen exposed below a more flexible rib cage, placing those organs at greater risk for injury. In addition, young children involved in automobile crashes may have injuries related to lap belts if they are improperly restrained.

Saladino RA, Lund DP: Abdominal trauma. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, p 1271.

3. What is the "lap belt complex" seen with motor vehicle crash victims?

This complex refers to injuries sustained during a motor vehicle crash when the seat belt rides up from the pelvic bones and compresses the softer abdominal viscera against the spine in an improperly restrained child. This may result in soft tissue injury to the abdomen, Chance fractures (compression fractures of the lumbar spine), and intra-abdominal injury. A visible abdominal wall bruise or mark from the lap belt may be present and indicates a higher likelihood of significant intra-abdominal injury. Associated injuries include solid organ injury, bowel perforations, mesenteric injury, and bladder injury.

Davies KL: Buckled-up children: Understanding the mechanism, injuries, management, and prevention of seat belt related injuries. J Trauma Nurs 2004;11(1):16-24.

Sharma OP, Oswanski MF, Kaminski BP, et al: Clinical implications of the seat belt sign in blunt trauma. Am Surg 2009;75(9):822-827.

4. What is the significance of a blow to the abdomen from the handlebars of a bicycle?

A direct blow to the abdomen of a child from the handlebars of a bicycle can result in significant injury, often with limited external physical findings. Common injuries resulting from this mechanism are solid organ injury, intestinal perforation, and abdominal wall hernia.

Klimek PM, Lutz T, Stranzinger E, et al: Handlebar injuries in children. Pediatr Surg Int 2013;29(3):269-273.

5. When is abdominal injury suspicious for nonaccidental trauma?

Abusive abdominal injuries account for a large proportion of abdominal trauma among infants and toddlers. Consider child abuse in patients in this young age group who present with abdominal trauma unless the event has been reliably witnessed or there is a known history of a high-velocity mechanism, such as a motor vehicle collision.

Lane WG, Dubowitz H, Langenberg P, Dischinger P: Epidemiology of abusive abdominal trauma hospitalizations in United States children. Child Abuse Negl 2012;36(2):142-148.

6. What physical examination findings are useful for identifying abdominal injuries?

The physical examination during the secondary survey is intended to recognize that an intra-abdominal injury exists, rather than to identify a specific diagnosis. Physical examination findings, including absent or diminished bowel sounds, evidence of peritoneal irritation with involuntary guarding or rebound, abdominal distention or rigidity, abdominal wall abrasions or bruising, abdominal or flank tenderness, lower chest wall injury, or unexplained

hypotension may indicate the presence of an intra-abdominal injury. Evaluation for tenderness or instability of the pelvic bones is necessary, because pelvic fractures have been associated with an increased risk of intra-abdominal injury. Perform a rectal examination in patients with significant injury to assess for a palpable mass, lacerations, pelvic brim fracture, or blood.

7. Which laboratory studies are useful for evaluating patients with abdominal trauma?

Complete blood counts, transaminases, and urinalysis are useful in identifying possible injury. The utility of pancreatic enzymes is unclear but may be helpful in assessing for intra-abdominal injury. Serial blood counts in patients with abdominal pain or tenderness may be useful in identifying acute blood loss.

Unexplained anemia, gross or microscopic hematuria (>5 red blood cells/high-power field), or elevated serum transaminases may indicate the presence of intra-abdominal injury, even in the absence of other findings. Several studies attempting to describe cutoff values for transaminases have found differing results. An alanine aminotransferase (ALT) value above 104 IU/L was found in one study to have high sensitivity; however, a study looking at transaminases in the setting of suspected nonaccidental injury suggests using cutoff levels of 80 IU/L for both aspartate aminotransferase (AST) and ALT to prompt abdominal imaging.

Bevan CA, Palmer CS, Sutcliffe JR, et al: Blunt abdominal trauma in children: How predictive is ALT for liver injury? Emerg Med J 2009;26(4):283-288.

Cotton BA, Beckert BW, Smith MK, et al: The utility of clinical laboratory data for predicting intra-abdominal injury among children. J Trauma 2004;56:1068-1074.

Kumar S, Sagar S, Subramanian A, et al: Evaluation of amylase and lipase levels in blunt trauma abdomen patients. J Emerg Trauma Shock 2012;5(2):135-142.

Lindberg DM, Shapiro RA, Blood EA, et al: Utility of hepatic transaminases in children with concern for abuse. Pediatrics 2013;131(2):268-275.

8. Why is gastric distention following abdominal trauma of serious concern?

Children often develop gastric distention after abdominal trauma from crying and swallowing air. This distention can interfere with respiration by altering the motion of the hemidiaphragm. Young children are primarily diaphragmatic breathers, so this can be serious. Also, gastric dilatation increases the risk of vomiting. Children often have a full stomach when injured, and vomiting could lead to aspiration of stomach contents. Gastric distention also makes abdominal examination difficult. Place a nasogastric tube to decompress the stomach when there is distention.

Saladino RA, Lund DP: Abdominal trauma. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, p 1271.

9. What is the treatment for the child with an apparent abdominal injury and clinical instability?

The initial care for the unstable pediatric patient with abdominal trauma must begin with the ABCs (airway, breathing, circulation). Once airway and breathing are secure, address circulation with an initial infusion of an isotonic crystalloid solution (normal saline or lactated Ringer's solution) of up to 40 to 60 mL/kg via the intravenous (IV) route. For the patient who continues to be unstable, transfuse with packed red blood cells. If the transfusion requirement exceeds 40 mL/kg during the initial resuscitation or with separate transfusions during the hospital course, consider surgical exploration for ongoing bleeding. Intra-abdominal hemorrhage is the primary cause of early death in pediatric abdominal trauma.

Saladino RA, Lund DP: Abdominal trauma. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, p 1271.

10. What is the role of computed tomography (CT) in assessing pediatric abdominal trauma?

For the clinically stable pediatric patient with blunt trauma, CT continues to be the gold standard for abdominal injury diagnosis, with sensitivity, specificity, and accuracy all exceeding 95%. Advantages of CT include the ability to noninvasively assess anatomic organ injury, perfusion, renal function, the retroperitoneal space, and the lower chest. Disadvantages of CT include a limited utility for the unstable patient, radiation exposure for the patient, and limited sensitivity (60-80%) for the diagnosis of pancreatic or bowel injury.

A recent large multicenter prospective study derived a clinical prediction rule to identify children at very low risk for intra-abdominal injury who did not require emergent CT (negative predictive value [NPV] 99.9%). Indicators of very low risk were lack of abdominal or thoracic wall bruising or seat belt sign, Glasgow Coma Scale (GCS) score greater than 13, absence of abdominal pain or tenderness, normal breath sounds, and absence of vomiting.

Holmes JF, Lillis K, Monroe D, et al: Identifying children at very low risk of clinically important blunt abdominal injuries. Ann Emerg Med 2013;62(2):107-116. Epub Feb. 1, 2013.

Streck CJ, Jewett BM, Wahlquist AH, et al: Evaluation for intra-abdominal injury in children after blunt torso trauma: Can we reduce unnecessary abdominal computed tomography by utilizing a clinical prediction model? J Trauma Acute Care Surg 2012;73(2):371-376.

11. What is FAST? What is its role in the evaluation of pediatric abdominal trauma?

FAST is **f**ocused **a**bdominal **s**onography for **t**rauma. The FAST examination is helpful to identify intra-abdominal free fluid and parenchymal injury by rapidly imaging the right and left upper quadrants, subxiphoid region, and pelvis. It is advantageous because it is a rapid, convenient, inexpensive tool that does not expose a child to radiation. Also, it can be done at the bedside so the patient does not need to be transported.

In the unstable patient, a positive FAST examination may be an indication for operative intervention. In a stable patient, identification of intra-abdominal fluid on FAST examination indicates the need for an abdominal CT. A negative FAST, however, does not exclude intra-abdominal injury.

Fox JC, Boysen M, Gharahbaghian L, et al: Test characteristics of focused assessment of sonography for trauma for clinically significant abdominal free fluid in pediatric blunt abdominal trauma. Acad Emerg Med 2011;18(5):477-482.

12. What is the sensitivity and specificity of FAST in children? Which patients would benefit most from FAST?

Limited studies evaluating FAST's utility in children have shown low sensitivity but high specificity. A meta-analysis examining the test performance of abdominal ultrasound in children used for identification of intra-abdominal injury found a sensitivity of 66% and a specificity of 95% when only the most rigorous studies were included.

However, a study looking specifically at emergency department (ED) ultrasonography in hypotensive children with blunt abdominal trauma found a sensitivity of 100% and specificity of 100%. Therefore, although not necessarily recommended for routine use, there may be a role for FAST in the rapid evaluation of unstable patients with concern for intra-abdominal injury. FAST obtained in these patients may allow for more rapid disposition to CT versus the operating room for exploratory laparotomy.

Holmes JF, Brant WE, Bond WF, et al: Emergency department ultrasonography in the evaluation of hypotensive and normotensive children with blunt abdominal trauma. J Pediatr Surg 2001;36 (7):968-973.

Holmes JF, Gladman A, Chang CH: Performance of abdominal ultrasonography in pediatric blunt trauma patients: A meta-analysis. J Pediatr Surg 2007;42(9):1588.

13. What is the recommended ED management of splenic injury in children?

All children with splenic injury require admission to the hospital. Those with hemodynamic instability or anemia may require intensive care unit (ICU) admission. The majority (up to 90%) of patients are managed nonoperatively with monitoring of vital signs, hematocrit, and urine output. Transfuse with packed red blood cells as necessary for hemodynamic changes or significant anemia. Operative intervention should be dictated by clinical course rather than grade of injury. For those who are hemodynamically stable but require multiple transfusions, arterial embolization, which has been used frequently in adults but has limited experience in children, may provide an alternative to operative intervention.

The American Pediatric Surgery Association has established guidelines for resource utilization (including length of ICU and hospital stay and activity restriction) based on grade of injuries as seen on CT; however, at least one more recent study has shown a shorter length of stay and decreased resource use when hemodynamic stability and hematocrit were used as the basis for clinical decision making.

McVay M, Kokoska E, Jackson R, et al: Throwing out the "grade" book: Management of isolated spleen and liver injury based on hemodynamic status. J Pediatr Surg 2008;43(6):1072-1076.

Nance ML, Holmes JH, Wiebe DJ: Timeline to operative intervention for solid organ injuries in children. J Trauma 2006;61(6):1389.
Stylianos S: Evidence-based guidelines for resource utilization in children with isolated spleen or liver injury. The APSA Trauma Committee. J Pediatr Surg 2000;35(2):164.

14. Is there a role for diagnostic peritoneal lavage (DPL) in the evaluation of abdominal injury in children?

DPL is rarely performed in children, and there is controversy surrounding its role because of its low specificity along with the emergence and easy availability of CT and FAST. Its utility remains limited to the evaluation of a hemodynamically unstable patient requiring emergent surgery (such as craniotomy).

15. What are the indications for laparotomy for pediatric abdominal trauma?

Indications for emergency laparotomy are listed in Table 50-1.

16. What is the clinical approach to penetrating abdominal trauma?

Because of their high velocity and penetrating ability, more than 90% of gunshot wounds to the abdomen result in organ injury requiring laparotomy. Stab wounds are typically of lower velocity, with less penetrating ability, and can be managed more selectively. Triple-contrast CT, laparoscopy, clinical observation, and local wound exploration have all been advocated to identify peritoneal penetration or intra-abdominal organ injury in stable stab wound victims. Start broad-spectrum antibiotics for all patients with penetrating abdominal trauma. Findings of clinical instability, peritoneal irritation, pneumoperitoneum, hematuria, and rectal blood are all absolute indications for laparotomy in patients with gunshot or stab wounds.

17. What is meant by the nonoperative management of blunt abdominal trauma in children?

Despite the long list of indications for laparotomy mentioned previously, operative intervention is infrequently required for pediatric blunt abdominal trauma, and nonoperative management is the current standard of care for the majority of patients. With few exceptions, the need for an operation rests entirely with the patient's clinical condition, rather than with specifics of the intra-abdominal organ injuries or the grade or severity of organ injury. Studies indicate that appropriately selected patients (i.e., those without surgical indications) have excellent overall outcomes, low transfusion rates, and few complications.

However, nonoperative does not mean nonaggressive or nonsurgical management. These patients must be closely monitored, often in the ICU, with surgical supervision and

Table 50-1. Indications for an Emergent Laparotomy for the Pediatric Trauma Patient

Vital sign instability, despite fluid resuscitation
Blood transfusion requirement totaling greater than one half the child's blood volume or > 40 mL/kg in the first 24 hours after injury
Peritoneal irritation on physical examination
Gunshot wound to the lower chest or abdomen
Stab wound of the abdomen, with vital sign instability, peritoneal irritation, or clinical evidence of organ injury
Rectal or vaginal laceration
Evisceration of abdominal contents
Evidence of intestinal perforation (e.g., pneumoperitoneum on imaging studies, fecal content on diagnostic peritoneal lavage)
Multisystem injuries requiring craniotomy with intraperitoneal blood identified or strong history or physical examination findings to indicate intra-abdominal injury
Significant abdominal distention associated with hypotension

adequate laboratory, blood bank, radiologic, nursing, and medical support. An aggressive and continuing reevaluation of the patient's clinical condition is mandatory, with surgical exploration for any evidence of deterioration.

Stylianos S: Outcomes from pediatric solid organ injury; role of standardized care guidelines. Curr Opin Pediatr 2005;17:402-406.

Venkatsh KR, McQuay N Jr: Outcome of management in stable children with intra-abdominal free fluid without solid organ injury after blunt abdominal injury. J Trauma 2007;62:216-220.

18. What traumatic abdominal injuries are typically associated with a late presentation?

Pancreatic pseudocyst (presenting with epigastric pain, abdominal mass, and hyperamylasemia), duodenal hematoma (presenting with symptoms of intestinal obstruction), and hematobilia (presenting with abdominal pain and upper gastrointestinal bleeding) often have a delayed presentation after injury.

Saladino RA, Lund DP: Abdominal trauma. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, p 1271.

Key Points: Evaluation of Abdominal Trauma

1. The finding of a seat belt sign should prompt higher suspicion for significant intra-abdominal injury.
2. Laboratory tests (hematocrit, urinalysis, AST, ALT) are excellent adjuncts to the physical examination to screen for an abdominal injury in the stable pediatric patient.
3. Patients with no abdominal or thoracic bruising or tenderness, a GCS score greater than 13, a normal lung examination, and no vomiting are at very low risk for intra-abdominal injury and may not need a CT scan.
4. A negative FAST study does not exclude intra-abdominal injury.
5. Clinical instability is the most important indication for an emergent laparotomy in the child with an abdominal injury.

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The author wishes to thank Dr. Ronald A. Furnival for his contributions to this chapter in the previous edition.

CHAPTER 51

BURNS AND SMOKE INHALATION

John M. Loiselle

1. How common are burns and fire-related deaths among children?

Deaths related to injuries are the leading cause of death in children ages 1 to 14 years. Statistics from the Center for Disease Control and Prevention (2010) rate fire- and burn-related injuries as the fourth leading cause of injury-related deaths in this age group, behind motor vehicle–related deaths, drowning, and deaths from firearms. Burns and fire-related injuries were responsible for 430 childhood deaths in the United States in 2010.

National Center for Injury Prevention and Control: Burns and fire-related injuries. Available at http://www.cdc.gov/injury/wisqars/pdf/leading_causes_of_death_by_age_group_2011-a.pdf.

2. What functions of the skin do burns affect?

- Temperature control
- Protection from infection
- Pain and sensation
- Fluid homeostasis

3. How are different depths of burns classified?

Different depths of burns are classified in Table 51-1.

4. What are the common sources of burns in children?

Thermal or fire-related burns make up the majority of burn injuries treated in U.S. emergency departments (EDs) (Table 51-2). Children less than 6 years of age account for the majority of burn injuries. Scald burns are the second most common type of burns. Most of these occur when the child pulls over a hot liquid from the surface of a table or stove. Nonaccidental burns account for up to 14% of burns in children less than 1 year of age.

D'Souza AL, Nelson NG, McKenzie LB: Pediatric burn injuries treated in US emergency departments between 1990 and 2006. Pediatrics 2009;124:1424-1430.

Shah A, Srinivasan S, Thomas R, et al: Epidemiology and profile of pediatric burns in a large referral center. Clin Pediatr 2011;50(5):391-395.

5. Where do most burns occur?

Over 90% of childhood burns occur in the household. More than a third of all pediatric burn injuries occur in the kitchen. This statistic has important implications for preventive measures.

D'Souza AL, Nelson NG, McKenzie LB: Pediatric burn injuries treated in US emergency departments between 1990 and 2006. Pediatrics 2009;124:1424-1430.

Shah A, Srinivasan S, Thomas R, et al: Epidemiology and profile of pediatric burns in a large referral center. Clin Pediatr 2011;50(5):391-395.

6. How long must the skin be in contact with hot water to cause a burn?

The actual duration of contact and water temperature required to cause a partial-thickness burn depend on the location and thickness of the skin. Partial-thickness burns on the soles of the feet require a longer period of contact with the water because of the thick layer of skin. Scald burns occur more rapidly in the skin of young children than in adults, and partial- or full-thickness burns can occur in less time than listed here:

160° F: 1 second
150° F: 2 seconds
140° F: 5 seconds
130° F: 30 seconds
120° F: 300 seconds

Note: Hot chocolate and hot tea are often served at temperatures of 160° to 180° F.

Table 51-1. Classification of Burn Depth

WOUND DEPTH	LAYER INVOLVED	CLINICAL FINDINGS	COMMON CAUSES
First-degree (superficial)	Epidermis	Erythema	Sun exposure
Second-degree (partial-thickness)	Dermis	Erythema, blistering	Hot liquids
Third-degree (full-thickness)	Subcutaneous tissue	Pale or charred, waxy or leathery, does not bleed, insensate	Flame
Fourth-degree	Fascia, muscle, or bone	Tissue loss	Flame or high-voltage electricity

From Sheridan R: Outpatient burn care in the emergency department. Pediatr Emerg Care 2005;21:449-456.

Table 51-2. Common Sources of Burns in Children

HOSPITALIZED PATIENTS (%)		EMERGENCY DEPARTMENT PATIENTS (%)	
Flames	36	Contact	43.1
Scald	35	Scald	33.9
Immersion	14	Flames	11.0
Contact	9	Cigarette	5.5
Chemical	3.5	Electrical	2.8
Electrical	1.5	House fire	0.9
Other	2.7		

Tepas JJ III, Fallat ME, Moriarty TM: Trauma. In Gausche-Hill M, Fuchs S, Yamamoto L (eds): The Pediatric Emergency Medicine Resource, 4th ed. Boston, Jones & Bartlett, 2004, pp 268-323.

7. Why is it important to interview the paramedics who arrive with fire victims?

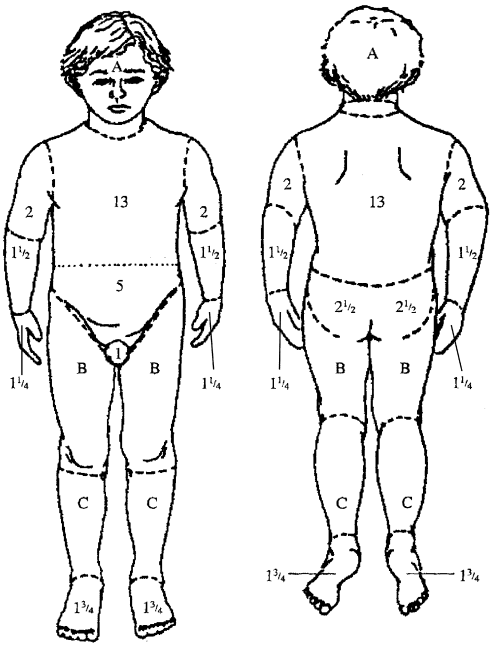
Paramedics can provide the answers to questions that influence therapy as well as prognosis. Important questions include the following:

- Did the fire occur in an open or enclosed area?
- Where was the child found?
- What was the duration of exposure to smoke?
- Did the child lose consciousness?
- How long was the transport?
- What therapy was instituted?
- Are associated injuries suspected?
- What materials were at the fire scene?
- Is additional toxic fume exposure a concern?

8. Name the methods commonly used to estimate the percentage of body surface area (BSA) damaged by burns in a child.

The distribution of BSA is different in children and adults. The standard "rule of nines" used in adults is not as accurate in children. The young child has a greater proportion of the BSA in the head and less in the lower extremities. Nagel and Schunk demonstrated that the entire palmar surface of a child's hand (including the fingers) is approximately 1% of BSA. The Lund and Browder chart (Fig. 51-1) provides useful estimates of larger contiguous burn areas in children younger than 10 years of age. First-degree burns are not generally included in the calculation of total BSA burned.

Lund C, Browder N: The estimation of areas of burns. Surg Gynecol Obstet 1944;79:352-358.
Nagel TR, Schunk JE: Using the hand to estimate the surface area of a burn in children. Pediatr Emerg Care 1997;13:254-255.



Lund and Browder Chart

Age (years)	0	1	5	10	15	Adult
A: Half of head	9 ½	8 ½	6 ½	5 ½	4 ½	3 ½
B: Half of thigh	2 ¾	3 ¼	4	4 ¼	4 ½	4 ¾
C: Half of leg	2 ½	2 ½	2 ¾	3	3 ¼	3 ½

Total area burned = second-degree surface area and third-degree surface area

Initial fluid maintenance: Ringer's lactate solution* given at:

$$\frac{\text{Surface area burned x weight (kg)}}{4} = \text{mL/hr}$$

*Some authors recommend adding sodium bicarbonate or 5% albumin to the solution.

Figure 51-1. Lund and Browder charts are somewhat more accurate to estimate the percentage of body surface burn than the rule of nines. Compared with adults, children have larger heads and smaller legs. Other areas are relatively stable through life. (*From Roberts JR, Hedges JR: Clinical Procedures in Emergency Medicine, 2nd ed. Philadelphia, WB Saunders, 1991, pp 614-615.*)

9. What is the initial treatment of major burns in a child?

1. Address and stabilize the airway, breathing, and circulation.
2. Remove clothing and any remaining hot or burning material.
3. Obtain intravenous (IV) access and begin fluid resuscitation, as needed, for severe burns.
4. Place a urinary catheter to monitor urine output.
5. Administer pain medication.
6. Monitor and maintain core temperature.
7. Assess the extent and depth of burns.
8. Irrigate the burns with lukewarm sterile saline.
9. Gently remove devitalized tissue with sterile gauze.
10. Perform escharotomies, as needed, for full-thickness circumferential burns.
11. Apply topical antibiotics to partial-thickness burns.
12. Cover large burn areas with sterile sheets.
13. Administer tetanus prophylaxis as indicated.
14. Consider transfer to a burn center.

Key Points: Major Threats to Children with Extensive Burn Injuries

1. Hypothermia
2. Hypovolemia
3. Infection

10. Name some recommended topical therapies for burns.

Multiple creams and ointments are appropriate for the treatment of burns. They perform several functions, including minimizing bacterial colonization, preventing desiccation, and reducing pain. Superficial burns require only a moisturizer. Commonly used topical therapies include the following:

- Bacitracin ointment is used for burns on the face.
- Erythromycin ophthalmic ointment is used for burns around the eye.
- 1% silver sulfadiazine cream is used for burns on the body.
- 11.1% mafenide acetate (Sulfamylon) is used for burns on the external ear. Mafenide acetate penetrates the burn eschar to reach and protect the cartilage of the ear.
- Synthetic membranes are also available to cover burn wounds. These dressings do not require daily changes but are much more expensive.

Karl SR: Trauma. In Fuchs S, Yamamoto L (eds): The Pediatric Emergency Medicine Resource, 5th ed. Boston, Jones & Bartlett, 2012, pp 249-257.

11. How should blisters be treated?

Intact blisters maintain a sterile environment below the surface, and unroofing the blisters allows for easier cleaning. Nonviable skin from a ruptured blister that is allowed to remain in the burn provides a medium for bacterial growth. Never aspirate blisters, as this predisposes to infection by disrupting the sterile environment and can introduce bacteria into the wound. Treatment is controversial, but in general, allow small blisters to remain intact. Unroof and débride open blisters, large blisters likely to rupture, and those crossing joints. Then clean the burn with mild soap and water.

Reed JL, Pomerantz WJ: Emergency management of pediatric burns. Pediatr Emerg Care 2005;21:118-129.

12. What is the approach to fluid management of a child with severe burn injuries?

Fluid resuscitation is typically required in children with partial- or full-thickness burns covering 15% or more of the total BSA. Fluid administration initially consists of lactated Ringer's solution or normal saline as needed for shock. Estimate early fluid replacement by following one of two formulas. The Parkland formula dictates that 4 mL/kg/% BSA burned should be administered over the first 24 hours. Infuse half of the total volume over the first 8 hours and half over the next 16 hours. Add maintenance requirements for children younger than 5 years of age in the form of 5% dextrose in normal saline. The Carvajal formula recommends 5000 mL/m^2/% BSA burned. Give half in the first 8 hours and the other half in the next 16 hours. Add maintenance fluids of 2000 mL/m^2/day to the total. Follow initial resuscitation

by fluids titrated to maintain urine output at 0.5 to 1 mL/kg/hour. Overhydration is poorly tolerated and may contribute to development of acute pulmonary edema. Consider placement of a central venous pressure monitor or Swan-Ganz catheter in severe cases.

Joffe MD: Burns. In Fleisher GR, Ludwig S, Henretig FM (eds): Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 1281-1288.

13. What are the criteria for outpatient management of burns?

1. Burns involve less than 10% of the BSA.
2. There is no airway or pulmonary involvement.
3. Burns do not cross major joints.
4. Burn is not circumferential.
5. Burns do not involve the face, hands, or genitals.
6. Abuse is not suspected.
7. An adult capable of managing wound cleaning and dressing changes is present in the home.

Fabia R, Groner JI: Advances in the care of children with burns. Adv Pediatr 2009;56:219-248.

14. What are the indications for referral to a regional burn center?

- Burns accompanied by respiratory injuries or major trauma
- Major chemical or electrical burns
- Partial-thickness and full-thickness burns covering over 10% BSA in children under 10 years of age, or over 20% in children older than 10 years
- Full-thickness burns over 5% BSA
- Burns that involve the face, eyes, ears, hands, feet, genitalia, perineum, or major joints
- Burn injury in patients with preexisting medical conditions affecting management or prognosis
- Burn injury in patients requiring special social, emotional, or long-term rehabilitative intervention

Committee on Trauma, American College of Surgeons: Advanced Trauma Life Support for Doctors, 8th ed. Chicago, American College of Surgeons, 2008, p 219.

15. Describe the patterns of burns commonly associated with child abuse.

- **Immersion burns**. This pattern of injury occurs as the result of a body part being dipped in a hot liquid. These burns are often circumferential with a sharply demarcated border. Burns in a glove-and-stocking distribution are a classic example.
- **Doughnut burn**. This term describes the sparing of the central area of the buttocks when it is held in contact with the cooler ceramic material of the bathtub floor. The surrounding areas of skin remain in contact with the water and sustain more severe burns. Burns involving the genitals, buttocks, or perineum are unlikely to be accidental.
- **Contact burns on the dorsum of the hand**. Children are more likely to sustain accidental burns by reaching for hot objects and grasping them with the palmar surface.
- **Markings consistent with an object**, such as a cigarette or iron, held to the skin.

In addition, burns that are inconsistent with the history or are attained in a manner beyond the developmental capacities of the child deserve further investigation.

Maguire S, Moynihan S, Mann M: A systematic review of the features that indicate intentional scalds in children. Burns 2008;34:1072-1081.

16. A 4-year-old boy sustains burns on the tip of his index finger and thumb when he places a bobby pin in an outlet. What is the recommended management of this child?

Household electrical outlets in the United States are generally 120 V or 240 V. These rarely result in serious injuries or cardiac dysrhythmias. Studies suggest that a patient who has not suffered a dysrhythmia or cardiac arrest at the scene and is asymptomatic at presentation to the ED is safe for discharge to home. These patients do not require an electrocardiogram (ECG) or inpatient monitoring. Patients with normal ECGs on presentation do not develop late-onset dysrhythmias.

Chen EH, Sareen A: Do children require ECG evaluation and inpatient telemetry after household electrical exposures? Ann Emerg Med 2007;49:64-67.

17. Name three potential causes of loss of consciousness in a fire victim.

- Carbon monoxide poisoning
- Head trauma
- Severe hypovolemia

18. What are the indications for intubation of the trachea of a fire victim?

- **Early-onset stridor.** The presence of stridor or hoarseness suggests upper airway injury that is likely to progress. Laryngeal edema does not peak until 2 to 8 hours after exposure. Intubation for this complication frequently requires an endotracheal tube with a smaller internal diameter than standard calculations suggest, because of airway swelling.
- **Severe burns of the face or mouth.** Patients are at significant risk for upper and lower airway injury.
- **Progressive respiratory insufficiency.** Respiratory insufficiency may be diagnosed clinically or by the finding of a widened arterial-alveolar gradient or rising levels of partial pressure of carbon dioxide. Hypercarbia may result from depressed mental status, pain associated with chest wall movement, restriction of chest wall movement secondary to burns, pulmonary restrictive or obstructive injury, or upper airway swelling and obstruction.
- **Inability to protect the airway** due to coma or profuse tracheobronchial secretions.
- **Carboxyhemoglobin levels higher than 50%.** Intubation and active ventilation provide increased oxygen concentrations and help to decrease levels more rapidly.

The presence of soot in the nares or carbonaceous sputum in isolation is not an indication for intubation.

A cuffed endotracheal tube is recommended, as decreased pulmonary compliance in smoke inhalation victims may require higher ventilator pressures.

19. What acute findings are common on the chest radiograph of a child with smoke inhalation?

Initial chest radiographs are usually normal. Radiographic findings lag behind physical symptoms. Chest radiographs are thus an insensitive means of determining lung injury and rarely dictate ED management. A normal chest radiograph does not exclude the presence of significant pulmonary injuries. Early findings are suggestive of severe injury. Diffuse interstitial infiltrates are consistent with significant smoke inhalation. Focal infiltrates in the first 24 hours indicate atelectasis. Bronchopneumonia as the result of smoke inhalation does not typically occur until 3 to 5 days after injury. Pulmonary edema typically follows aggressive fluid resuscitation and does not appear until 6 to 72 hours after exposure. Pneumothorax is often the result of barotrauma after intubation and positive-pressure ventilation.

Clark WR, Bonaventura M, Myers W: Smoke inhalation and airway management at a regional burn unit: 1974 to 1983. Part I. Diagnosis and consequences of smoke inhalation. J Burn Care Rehabil 1989;10:52-62.

20. What precautions must be taken when interpreting an arterial blood gas sample from a victim of smoke inhalation?

Partial pressure of oxygen in arterial blood (Pa_{O_2}) measures only dissolved oxygen in the blood. It is unaffected by the presence of carbon monoxide. Pa_{O_2}, therefore, provides a falsely reassuring measure of oxygenation. In addition, it is important to know whether the blood gas report includes a measured or calculated oxygen saturation. A calculated oxygen saturation does not take into account the presence of carboxyhemoglobin, whereas a measured oxygen saturation does. A calculated oxygen saturation registers falsely elevated readings in the setting of carbon monoxide poisoning.

21. Describe the pathophysiologic effects of carbon monoxide toxicity.

Carbon monoxide binds hemoglobin with 230 times the affinity of oxygen. The production of carboxyhemoglobin significantly reduces the oxygen-carrying capacity of blood. The binding of carbon monoxide to the hemoglobin molecule shifts the oxygen dissociation curve to the left. This shift inhibits the release of oxygen at the tissue level. Carbon monoxide also has toxic effects on the cellular level. Carbon monoxide binds cytochrome oxidase and effectively blocks cellular respiration. This blockade facilitates free radical production and disrupts mitochondrial function. The results are tissue hypoxia and metabolic acidosis.

22. How is the half-life of carboxyhemoglobin affected at different oxygen concentrations?

- Room air (21%, 1 atmosphere [1 atm]): half-life of 4 hours
- 100% O_2, 1 atm: half-life of 60 to 90 minutes
- Hyperbaric oxygen (100% oxygen, 2.5 atm): half-life of 15 to 30 minutes

Key Points: Pathophysiologic Effects of Smoke Inhalation

1. Airway burns and edema from superheated gases and particles
2. Hypoxia due to carbon monoxide
3. Disruption of cellular respiration by carbon monoxide and cyanide
4. Bronchospasm from gases and particulate matter

23. How is a pulse oximeter reading affected by the presence of carboxyhemoglobin?

The pulse oximeter essentially measures the absorption of two separate wavelengths of light. These wavelengths correspond to the peak absorption spectra of oxygenated and deoxygenated hemoglobin. Carboxyhemoglobin absorbs light at a wavelength similar to oxygenated hemoglobin and therefore does not affect the overall reading. The result is a falsely high oxygen saturation reading. Standard pulse oximeter readings are unreliable measures of true oxygen saturation in patients with smoke inhalation. Pulse CO-oximeters have been developed to determine carboxyhemoglobin levels but are not as accurate as laboratory measurements.

Fidkowski CW, Fuzaylov GM, Sheridan RL, et al: Inhalation burn injury in children. Pediatr Anesth 2009;19:147-154.

Touger M, Birnbaum A, Wang J, et al: Performance of the RAD-57 CO-oximeter compared with standard laboratory carboxyhemoglobin measurement. Ann Emerg Med 2010;56(4):382-388.

24. What are the indications for hyperbaric oxygen therapy?

The use of hyperbaric oxygen in the setting of smoke inhalation is highly controversial. Studies to date have not demonstrated reduced mortality rates or improved neurologic outcomes with treatment after smoke inhalation. When indications for a particular patient remain unclear, seek further consultation from hyperbaric experts. To be considered a candidate for hyperbaric oxygen therapy, the patient must be deemed stable enough for transport. The potential benefits should outweigh the risks of transferring the patient to the chamber, especially when long distances are involved.

Buckley NA, Juurlink DN, Isbister G, et al: Hyperbaric oxygen for carbon monoxide poisoning. Cochrane Database Syst Rev 2011;(4);CD002041.

25. What are the criteria for hyperbaric oxygen therapy?

Generally accepted criteria:

- Syncope
- Severe neurologic symptoms on presentation (seizures, focal neurologic findings, coma)
- Myocardial ischemia diagnosed by history or electrocardiography
- Cardiac dysrhythmias (ventricular, life-threatening)
- Persistent neurologic symptoms and signs (mental confusion, visual disturbance, ataxia) after several hours of 100% oxygen therapy at ambient pressure
- Pregnancy (symptomatic, carboxyhemoglobin level > 15%, evidence of fetal distress)

Criteria for consideration:

- Carboxyhemoglobin level higher than 20% to 25%
- Abnormal results on neuropsychological examination
- Age less than 6 months with symptoms (lethargy, irritability, poor feeding) or involved in same exposure as adults with any of the preceding criteria
- Children who have underlying diseases (i.e., sickle cell anemia) for whom hypoxia may have deleterious effects

Liebelt EL: Hyperbaric oxygen therapy in childhood carbon monoxide poisoning. Curr Opin Pediatr 1999;11:259-264.

26. What is the evidence for cyanide toxicity in relation to house fires?

A study performed in France measured cyanide concentrations in blood samples obtained from fire victims by ambulance physicians at the scene. Cyanide levels were found to be significantly higher than those in one control group of patients with no fire exposure and a second control group with isolated carbon monoxide poisoning. Mean cyanide levels in fire victims declared dead at the scene were over 110 μmol/L. Mean cyanide levels in survivors were 21.6 μmol/L. Lethal cyanide levels are generally considered to be those exceeding 40 μmol/L.

Baud FJ, Barriot P, Toffis V, et al: Elevated blood cyanide concentrations in victims of smoke inhalation. N Engl J Med 1991;325:1761-1766.

27. What are the indications for treatment of cyanide poisoning in fire victims?

Cyanide levels are not immediately available, and the administration of antidote cannot be delayed. Treatment for cyanide poisoning is indicated in fire victims with evidence of significant smoke inhalation and altered neurologic status. Elevated lactate levels correlate with the presence of cyanide and are typically available sooner than a cyanide level.

Borron SW, Baud FJ, Barriot P: Prospective study of hydroxocobalamin for acute cyanide poisoning in smoke inhalation. Ann Emerg Med 2007;49:794-801.

28. What treatments are available for cyanide poisoning?

- **Sodium thiosulfate** can be administered intravenously as an infusion of a 25% solution of sodium thiosulfate at a dose of 1.65 mL/kg administered over 10 minutes. It is the rate-limiting compound in the conversion of cyanide to thiocyanate, which is excreted harmlessly in the urine. It has a slow onset of action.
- **Amyl nitrate** and **sodium nitrate** convert hemoglobin to methemoglobin, which preferentially binds cyanide in the form of cyanmethemoglobin. This therapy is generally not recommended for smoke inhalation because it further reduces the level of normal hemoglobin in fire victims, who already have a significant amount of dysfunctional hemoglobin.
- **Hydroxocobalamin** binds cyanide to form cyanocobalamin (vitamin B_{12}), which is excreted in the urine. Hydroxocobalamin is administered intravenously in a dose of 70 mg/kg to a maximum dose of 5 g over 10 minutes. It has a rapid onset of action without serious adverse effects and is the preferred treatment for pediatric fire victims with concern for cyanide poisoning.

Mintegi S, Clerigue N, Tipo V, et al: Pediatric cyanide poisoning by fire smoke inhalation: A European expert consensus. Pediatr Emerg Care 2013;29:1234-1240.

29. What is the role of steroids in the setting of smoke inhalation?

Steroids may benefit patients with acute bronchospasm due to smoke inhalation. No evidence indicates that steroids are beneficial in the acute treatment of airway edema or inflammatory processes due to smoke inhalation. Randomized trials have demonstrated an association of steroid use with worse outcomes in terms of infection and death.

Ruddy RM: Smoke inhalation injury. Pediatr Clin North Am 1994;41:317-336.

30. What preventive measures will reduce morbidity and mortality risks from burns and smoke inhalation?

Anticipatory guidance and preventive care have the greatest potential to reduce deaths from burns and house fires. It is estimated that over 50% of fire-related deaths could be avoided with the proper use of smoke detectors. Over 90% of childhood deaths from fires occur in homes without properly functioning smoke detectors, and most children in house fires die from smoke inhalation rather than burns. Educate families to install smoke detectors on every level of the house and test them regularly. Tell them to replace batteries twice a year. Instruct families about the planning of escape routes, evaluating fire risks in the house, and the use and storage of fire extinguishers. Also encourage parents to do the following: Avoid careless (or any) cigarette use. Lower the setting on the water heater to deliver water at a maximum temperature of 120° F (48.8° C), as this substantially increases the time it takes for direct exposure to induce a full-thickness burn (see Question 6). Place boiling liquids on the back burners of the stove, where they are out of the reach of young children. Do not allow children to operate or remove objects from microwave ovens. Dress children in flame-retardant sleepwear.

Websites

National Center for Injury Prevention and Control: www.cdc.gov/ncipc.
Emergency Medical Services for Children: www.ems-c.org.

CHAPTER 52

CHILD ABUSE

Stephen Ludwig

1. What four elements should you consider in making a diagnosis of suspected child abuse?

1. Detailed history of injury
2. Physical findings and their correspondence with the history
3. Laboratory and radiographic information
4. Observed interaction between parent-child and health care team members

Combining these four elements should help to determine whether you have sufficient grounds to institute a report of suspected abuse.

Bar-Merritt MH, Lane WG: An evidence based approach to the evaluation and treatment of child physical abuse. Pediatr Emerg Med Pract 2011;8:1-23.

2. What forms of abuse are defined by most state child abuse laws?

- Physical abuse
- Physical neglect
- Sexual abuse
- Psychological/emotional abuse

3. Which form of abuse is reported most often? What form occurs most often?

Physical abuse is usually the most disabling, with sexual abuse a close second. However, psychological abuse occurs most often and has the most disabling long-term consequences.

Tenney-Soeiro R, Wilson C: An update on child abuse and neglect. Curr Opin Pediatr 2004; 16:233-237.

4. What injury accounts for most deaths due to child abuse?

Head injury accounts for most child abuse–related deaths, including children who suffer direct trauma and shaking-impact injuries. Abdominal trauma causes most other child abuse–related deaths.

5. Abuse occurs most often in which ethnic group?

The rate of abuse by ethnic groups roughly matches the ethnic distribution in the general population. Thus, white children are abused most often.

6. True or false: Most cases of abuse are reported by medical professionals.

False. Most cases are reported by nonphysicians and non–health care institutions. Abuse is rarely reported by primary care physicians (probably less than 2% of reported cases nationwide).

7. What has happened to the rates of hospitalization for child abuse over the past 10 years?

The rates of hospitalizations have remained the same from 1997 to 2009, but rates of abuse such as abusive head trauma have gone up in the midst of the economic crisis of the last 6 years.

Berger RP, Fromkin JB, Stutz H, et al: Abusive head trauma during a time of increased unemployment: A multicenter analysis. Pediatrics 2011;128:637-643.

Farst K, Ambadwar PB, King AJ, et al: Trends in hospitalization rates and severity of injuries from abuse in young children, 1997-2009. Pediatrics 2013;131(6):e1796-e1802. Epub May 20, 2013.

8. How many abuse victims are killed each year in the United States?

Approximately 1400 children annually. This rate has been fairly constant.

Sirotnak AP, Grigsby T, Krugman RD: Physical abuse of children. Pediatr Rev 2004;25:264-277.

U.S. Department of Health and Human Services, Administration for Children and Families, Administration on Children, Youth and Families, Children's Bureau: Child Maltreatment 2012. Washington, DC, USDHHS, 2013. Available from http://www.acf.hhs.gov/programs/cb/research-data-technology/statistics-research/child-maltreatment.

9. Can the color of bruises on a child's body be used to date the injury definitively?

Despite findings noted in most standard textbooks, the color of the bruise does not indicate the age. The color of a bruise is determined by location, nature of the traumatic force, amount of subcutaneous tissue, and other factors. Aging of bruises based on color is highly unreliable.

Bariciak ED, Plint AC, Gaboury I, et al: Dating of bruises in children: An assessment of physician accuracy. Pediatrics 2003;112:804-807.

10. In a child with multiple bruises and a normal neurologic examination, is computed tomography (CT) of the head indicated?

At least one study indicated a high yield of positive CT findings, even in children with normal neurologic examinations. CT screening appears to be more helpful than routine ophthalmologic examinations.

Rubin DM, Christian CW, Bilaniuk LT, et al: Occult head injury in high-risk abused children. Pediatrics 2003;111:1382-1386.

11. What are the most common skin lesions mistaken for abuse?

Mongolian spots, which most commonly occur on the back, at the base of the spine, and on the buttocks are often mistaken for abuse. However, they also may occur on other skin surfaces. Also, areas of denuded skin that result from drug reactions such as toxic epidermal necrolysis are often misinterpreted as scald burns. There are many other acute dermatologic conditions that may mimic signs of abuse.

Asanti DP, Singh S, Sharma VK: Dermatoses misdiagnosed as deliberate injuries. Med Sci Law 2012;52:198-204.

12. What conditions may lead to easy bruisability?

Ehlers-Danlos syndrome and other connective tissue disorders may lead to easy bruisability. Hemophilia usually results in deep soft tissue and joint bleeding rather than superficial skin bruises. Idiopathic thrombocytopenia purpura and other platelet abnormalities lead to petechiae and mucous membrane bleeding rather than bruises. Henoch-Schönlein purpura also may be initially confused with bruising.

Bechtel K: Identifying the subtle signs of pediatric physical abuse. Pediatr Emerg Med Rep 2001;6:57-67.

13. What developmental skill is associated with bruising?

Walking or learning to walk may be associated with bruises. However, bruises found on children who have not yet learned to walk should be considered a sign of possible abuse. Children who do not cruise or walk generally do not bruise.

Sugar NF, Taylor JA, Feldman KW, et al: Bruises in infants and toddlers: Those who don't cruise rarely bruise. Arch Pediatr Adolesc Med 1999;153:399-403.

14. A 2-week-old infant, seen in the emergency department (ED) for crying, has several fractures at varying stages of healing. What may you conclude from these findings?

Although this scenario may indicate abuse, the young age of the infant and the possibility that fractures may have occurred in utero make a metabolic bone disease more likely.

15. What findings make osteogenesis imperfecta more likely than child abuse?

Blue scleras, dental abnormalities, hearing loss, wormian bones, and radiographs showing osteopenia and healing with abundant callus formation.

16. Which specific fractures have a high probability of being caused by child abuse?

Rib fractures, metaphyseal chip fractures (Fig. 52-1), spine and scapula fractures, and complex skull fractures.

17. Which specific fractures have a low specificity for abuse?

Linear skull fractures, clavicle fractures, single-bone transverse fractures, and spiral fractures of the tibia (toddler's fracture).

18. What type of force is consistent with the finding of a metaphyseal fracture?

Metaphyseal fractures occur when an extremity is pulled in the direction of its long axis. Stress on the tight periosteal attachments along the metaphysis results in a chip or corner fracture.

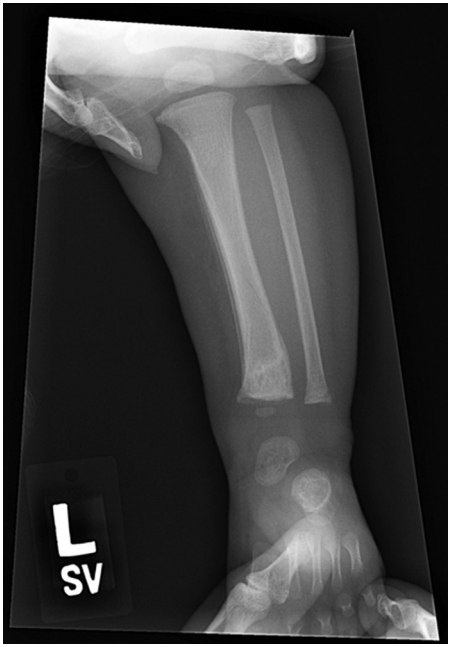


Figure 52-1. Metaphyseal chip fracture.

19. A child appears to be tender when his upper humerus is palpated. Plain radiographs fail to show any injury. What other studies may be helpful?

Bone scanning or a skeletal magnetic resonance imaging (MRI) scan may be more sensitive in demonstrating bony injury.

Mandelstam SA, Cook D, Fitzgerald M, et al: Complementary use of radiologic skeletal survey and bone scintigraphy in detection of bony injuries in suspected child abuse. Arch Dis Child 2003; 88:387-390.

20. In evaluating child abuse, a skeletal survey is required for which children?

According to the AAP Statement on Diagnostic Imaging of Child Abuse, "The skeletal survey is mandatory in all cases of suspected physical abuse in children younger than 2 years. Its utility diminishes thereafter." Beyond 5 years of age the utility markedly diminishes, leaving the necessity for children aged between 2 and 5 years open for clinical judgment.

AAP Section on Radiology: Diagnostic Imaging of Child Abuse. Pediatrics 2012;123(5):1430-1435.

21. What tests should be used to differentiate rickets from child abuse as the cause of skeletal abnormality?

- Bone density (appearance of bones)
- Serum calcium and phosphorus
- Serum alkaline phosphatase
- X-rays–the best radiograph to demonstrate rickets is that of the distal radius and ulna (wrist).

22. What history of injury matches a spiral fracture?

A spiral injury is caused by torque or twisting (Fig. 52-2). The extremity is twisted, or the child's body is twisted while the extremity is held in a fixed position. If the mechanism of injury does not match the nature of the injury, suspect abuse.

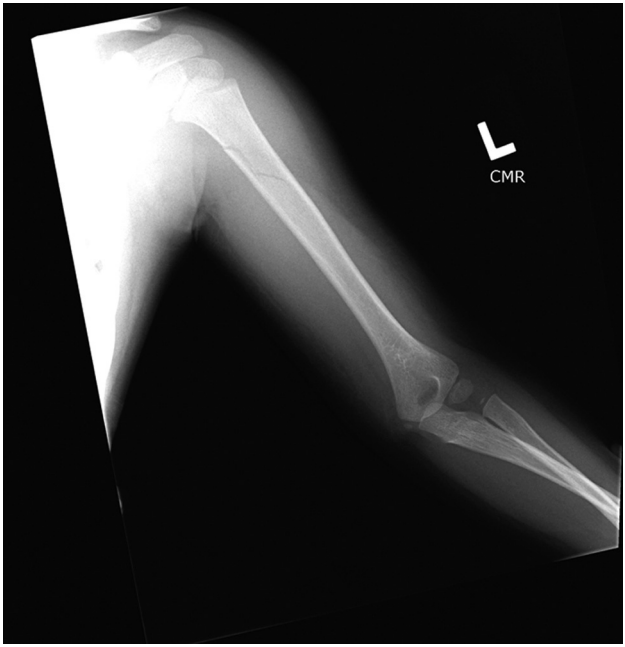


Figure 52-2. Spiral fracture of humerus.

23. What study has the highest sensitivity and specificity for diagnosing a suspected skull fracture?

A skull radiograph best shows a skull fracture, although a CT scan of the head often reveals a fracture. If the fracture line is horizontal, it may be missed by the horizontal cuts of the CT scan.

24. What is the preferred imaging technique for acutely injured children suspected to have neurotrauma as the result of abuse?

The head CT is the preferred modality, as it is readily available in most centers and will identify most significant injuries. The CT should be followed by an early MRI, and diffusion weighted imaging (DWI) may help in the detection of ischemic injury, timing, and prognosis.

AAP Section on Radiology: Diagnostic imaging of child abuse. Pediatrics 2012;123(5):1430-1435.

25. A 6-month-old boy is brought to the ED for new onset of seizures. You note retinal hemorrhages. A CT scan of the brain is read as normal. What is your conclusion?

CT may not pick up all central nervous system (CNS) injuries. Your next step is to order an MRI scan. MRI may detect small punctate hemorrhages or subdural collections that are isodense on CT. A new onset of seizures and retinal hemorrhages has a high probability of being caused by child abuse.

Care M: Imaging in suspected child abuse: What to expect and what to order. Pediatr Ann 2002;31:651-659.

26. What preimagery signs may be clues to shaken baby syndrome?

Retinal hemorrhages are present in 75% to 80% of cases. Signs of external trauma are minimal or absent. The child may present with hypothermia or respiratory difficulties. Xanthochromic fluid may be present in the cerebrospinal fluid obtained to rule out sepsis. Bruising may be seen on the upper extremities or chest wall in the place where the child was grasped.

27. True or false: Nonabusive head trauma may result in retinal hemorrhage.

True. Other causes of trauma may be associated with retinal hemorrhage, but with much lower frequency than shaking. If retinal hemorrhages are present with no history of trauma, suspect an incorrect or hidden history.

Bechtel K, Stoessel K, Leventhal JM, et al: Characteristics that distinguish accidental from abusive injury in hospitalized young children with head trauma. Pediatrics 2004;114:165-168.

Feldman K, Bethel R, Shugerman R, et al: The cause of infant and toddler subdural hemorrhage: A prospective study. Pediatrics 2001;108:636-646.

Reese RM, Sege R: Childhood head injuries: Accidental or inflicted? Arch Pediatr Adolesc Med 2000;154:11-15.

Shiau T, Levin AV: Retinal hemorrhages in children: The role of intracranial pressure. Arch Pediatr Adolesc Med 2012;166:623-628.

28. True or false: In a comparison between inflicted head trauma and noninflicted head trauma, the neurologic findings are similar.

False. Children with inflicted head trauma have more severe head injury, which requires more ED management and longer hospital stays and is associated with poorer outcomes.

Bechtel K, Stoessel K, Leventhal JM, et al: Characteristics that distinguish accidental from abusive injury in hospitalized young children with head trauma. Pediatrics 2004;114:165-168.

Chadwick DL, Bertocci G, Castillo E, et al: Annual risk of death rate resulting from short falls among young children: Less than 1 in a million. Pediatrics 2008;121:1213-1224.

29. How often have children with head trauma due to child abuse been injured only once?

Rarely. Studies demonstrate that by the time head trauma due to child abuse is diagnosed, many earlier episodes probably have gone undiagnosed and unreported. Chronic effusions from old blood or cerebral atrophy may be present.

30. What four types of retinal hemorrhages are associated with abuse?

1. Simple flame hemorrhage
2. Dot-and-blot hemorrhage
3. Surface hemorrhage, obscuring vessels
4. Retinoschisis

31. Which metabolic disease is associated with CNS hemorrhage?

Glutaric acidemia has the features of CNS hemorrhage and retinal hemorrhage. Usually, other signs of metabolic derangement and mental retardation are present. Glutaric acidemia is extremely rare, unlike abuse.

32. How can you tell a "bloody tap" from a CNS hemorrhage?

The differentiation can be difficult. With a bloody lumbar puncture, the blood usually clears as you collect more fluid. When the collection tube is spun, the cerebrospinal fluid of the CNS hemorrhage is xanthochromic; the bloody tap is clear.

33. What are the classic CT findings in shaken baby syndrome?

The classic findings are subdural hemorrhage, particularly in the intrahemispheric fissure; injury to the frontal or occipital lobe; loss of gray-white matter differentiation; and basal ganglia injury. Evidence of old CNS injury is another alerting sign.

Preer G, Sorrentino D, Ryznar E, Newton AW: Child maltreatment: Promising approaches and new directions. Curr Opin Pediatr 2013;25(2):268-274.

34. A child is taken to a babysitter. Six hours later the child collapses, has seizures, and is brought to the ED, where a serious head injury is found. The babysitter claims that the injury occurred at home before the parent dropped off the child. What is the likely determination?

With the exception of an epidural hematoma, symptoms develop just after the trauma is inflicted. There is no lucid or normal interval. The findings suggest that the babysitter or someone in the babysitter's house inflicted the trauma.

35. Raccoon eyes are a sign of what kind of injury?
Raccoon eyes are consistent with basilar skull fracture. More common than true raccoon eyes is bilateral infraorbital ecchymosis due to a midline forehead hematoma and tracking of blood into the infraorbital position.

36. If a child is slapped forcefully, what skin finding is typical?
A slap mark shows the outline of the shape of the fingers in petechiae or bruise lesions—not the imprint of the fingers themselves.

37. What does a cigarette burn look like?
Cigarette burns are often talked about but rarely seen. When a cigarette is extinguished on a child's skin, the mark is usually circular and the width slightly larger than the cigarette (roughly 0.8-1 cm). The second-degree burn should be uniform throughout the circular lesion, and the edges of the circle are raised by 1 to 2 mm. Inadvertent contact between a child and a cigarette (nonintentional) produces a simple partial-thickness bullous lesion.

38. At what temperature does water produce a burn in a child?
There is a relationship between water temperature and time of exposure. Exposure for 20 seconds to 125° F water is sufficient to cause a second-degree or partial-thickness burn.

39. What factors help to differentiate an inflicted bathtub scald burn from an accidental burn?
If the child is of toilet-training age, if there was a delay (>1-2 hours) in seeking care, and if the person who brings the child to the ED is not the person who was supervising the child when the burn occurred, the likelihood of abuse increases.
Andronicus M, Oates RK, Peat J, et al: Non-accidental burns in children. Burns 1998;24:552-558.

40. If a child is immersed in hot water while wearing some articles of clothing, what should physical examination of the burn reveal?
The clothed areas have increased burn severity because clothes hold the hot water closer to the skin. On the other hand, areas of thicker skin (e.g., palms and soles) may be relatively spared.

41. What is a "boxed ear"? How do you recognize it?
A boxed ear is a common injury caused by abuse. It results when the child receives a blow to the side of the head, including the ear. The finding to look for is ecchymosis on the inside surface of the pinna. This area is not exposed to other forms of injury.

42. What is the significance of a duodenal hematoma in the evaluation of child abuse?
A duodenal hematoma in children younger than 2 years is highly suspicious for abuse. Other causes such as handlebar injuries and lap belt trauma do not occur in children this young.
Sowrey L, Lawson KA, Garcia-Fillon P: Duodenal injuries in the very young child: Child abuse? J Trauma Acute Care Surg 2013;74:136-142.

43. What is "bottle-jamming"?
Trauma to the upper gum line and frenulum, which results when a frustrated parent forcefully jams a bottle into the child's mouth.

44. A child with multiple bruises is brought to the ED. Other than documentation of normal coagulation, what laboratory studies should be obtained?
Assess amylase, lipase, and liver enzymes. Studies have shown that the rate of intra-abdominal injury is higher than what may be apparent. Also order a urinalysis to look for signs of renal bleeding.

45. A 10-month-old baby is brought to the ED, essentially dead on arrival. No marks, bruises, or ecchymoses are found on the child's body, and there is no history of illness. Is sudden infant death syndrome (SIDS) the most likely diagnosis?
Most cases of SIDS occur at ages 2 to 5 months. Once a child is older than 6 months, you should be highly skeptical. Accurate diagnosis of SIDS requires a complete autopsy and death-scene investigation.

Key Points: Suspected Child Abuse

1. Child abuse occurs frequently. Consider child abuse as a possible mechanism in every traumatic injury.
2. Physicians and nurses are mandated to report the suspicion of abuse.
3. A level of suspicion is built by compiling elements of history, physical examination, laboratory and radiographic data, and observed interactions of the family.
4. Injuries that kill children are head injuries from both direct trauma and shaking and abdominal injuries.
5. The more similar the family is to you in such characteristics as age, race, socioeconomic status, and education level, the more difficult it will be to suspect abuse.

46. What metabolic disease may present to the ED as an apparent case of SIDS?
Deficiency of medium-chain acyl-CoA dehydrogenase, a defect in fatty acid metabolism.

47. What are the characteristics of a parent who may be involved in Münchausen syndrome by proxy?
- Mother
- Medical background or experience as a patient
- Articulate and cooperative
- Unusual or unexplainable disorder that you are asked to diagnose
- Always present when episode, event, or finding is discovered
- Distant relationship with the child's father

48. A child is brought to the ED for spitting up blood. The mother brings in a bib with a large blood spot. She reports that it has happened before and the child underwent an extensive workup at a nearby medical center. What should you do?
Check the type of the blood on the bib to confirm that it is the same as the baby's. Get the records from the other hospital to see what the physician's impressions were. Check with the child's primary care physician. Collect as much background data as possible.

49. What chief complaint is associated most often with Münchausen syndrome by proxy?
Complaints of apnea and near-SIDS have been documented as Münchausen syndrome by proxy in many case series. It is an important element in the differential diagnosis of apnea or acute life-threatening events.

50. A child comes alone to the ED with abdominal pain. She does not want her mother called for permission and states that she is afraid of her mother. Is this a case of child abuse?
This is a difficult case. It may meet the criteria for emotional abuse, and you should attempt to report it. The diagnosis of abuse usually rests on documentation of an injury. You may need a mental health consultation to document the nature and extent of the fear.

51. Does failure to thrive (FTT) always indicate abuse or neglect?
No. Some cases of FTT are based on lack of proper child-rearing practices, others are due to an organic condition, and still others result from the combination of a child who is difficult to feed and a family without proper skills and resources. Cases that result from abuse, of course, must be reported.

52. What are some red flags that indicate that a patient with FTT may have an underlying medical cause?
Some of the red flags include cardiac findings, developmental delay, dysmorphic features, organomegaly, lymphadenopathy, recurrent infections, and other symptoms such as diarrhea, vomiting, dehydration. Most children with FTT on a psychosocial basis have few signs or symptoms and are just not receiving adequate calories.

53. What forms of neglect are included in the definition of most state laws?
- Physical neglect
- Educational neglect
- Medical neglect
- Emotional neglect
- Abandonment

54. What are the indications for the immediate ED evaluation of a sexually abused child?

If bleeding or other specific symptoms are present or if the suspected abuse has occurred in the past 72 hours, most specialists suggest that the child be seen immediately. Otherwise, the patient and family may be referred to a special center for child sexual abuse.

Adams JA, Kaplan RA, Sterling SP, et al: Guidelines for medical care of children who may have been sexually abused. J Pediatr Adolesc Gynecol 2007;20:161-172.

Mollen CJ, Goyal MK, Frioux SM: Acute sexual assault. Pediatr Emerg Care 2012;28:584-590.

55. Should vaginal, pharyngeal, and rectal cultures be performed on every child undergoing a sexual abuse evaluation?

Studies clearly show that cultures should be performed only if the child has symptoms (e.g., vaginal discharge, genital injury) and if the perpetrator or sibling of the patient has a sexually transmitted disease.

Atabaki S, Paradise JE: The medical evaluation of the sexually abused child: Lessons from a decade of research. Pediatrics 1999;104:178-186.

Fortin K, Jenny C: Sexual abuse. Pediatr Rev 2012;33(1):19-32.

56. Does a Gram stain showing gram-negative intracellular diplococci indicate gonorrheal infection and possible sexual abuse?

Although *Neisseria gonorrhoeae* has these characteristics, other normal flora may be visualized in the same way. Do not base a diagnosis of suspected sexual abuse on smear findings. Cultures are essential, and results must be confirmed by at least two different laboratory methods.

57. Which sexually transmitted diseases are highly specific for sexual abuse?

Gonorrhea and syphilis are transmitted sexually. If they are found in a prepubertal child beyond infancy, you can be almost certain that sexual abuse has occurred.

58. Which sexually transmitted diseases are less specific because they have other modes of transmission?

Herpes simplex virus, *Trichomonas* spp., condylomata, *Gardnerella* spp., and *Chlamydia* spp. may indicate sexual abuse, but all may be transmitted nonsexually and thus have lower specificity.

Centers for Disease Control and Prevention: Sexually transmitted diseases treatment guidelines. MMWR Morb Mortal Wkly Rep 2006;55(RR-11):1-94.

59. In examining a child for possible sexual abuse, how helpful is the width of the intralabial distance?

Although at one time this finding was thought to be important, subsequent research has negated its diagnostic usefulness.

Fortin K, Jenny C: Sexual abuse. Pediatr Rev 2012;33(1):19-32.

60. A child comes to the ED for bloodstains in her underpants. Physical examination reveals a red doughnut-shaped mass just inferior to the clitoris. What is the likely diagnosis?

Urethral prolapse, a finding often misidentified as child abuse. The mucosa of the urethra is friable and bleeds. It is treated with sitz baths and time. It has no association with sexual abuse.

61. True or false: The physical examination of a recently sexually abused child will be abnormal.

False. The physical examination of most sexually abused children is expected to be normal.

Hariton TN: Sexual assault in prepubertal girls: It is normal to be normal—Or is it? Med Sci Law 2012;52:193-197.

62. What are SARTs and what has been their impact on identifying and treating child sexual abuse?

Sexual assault response teams (SARTs) are community- and hospital-based efforts to address the problem of child sexual abuse. Many teams are multidisciplinary and include sexual assault nurse examiners (SANEs) who are trained to perform the initial examination, coordinate care, enhance interprofessional communication, and improve legal outcomes. These teams are working well in many communities, but controlled data about their effectiveness are still being questioned.

Greeson MR, Campbell R: Sexual assault response teams (SARTs): An empirical review of their effectiveness and challenges to successful implementation. Trauma Violence Abuse 2013;14:83-95.

63. Should the examination for child sexual abuse be performed with sedation?

It is important to gain the confidence of the child in order to perform the examination, whenever possible. Use of dissociative medication is to be avoided. When acute severe trauma must be ruled out, for example, in the situation of ongoing vaginal bleeding, there is some justification for the examination being conducted under sedation or anesthesia.

Fortin K, Jenny C: Sexual abuse. Pediatr Rev 2012;33(1):19-32.

64. What are the two types of child abuse cases in the court system? What are the differences?

The two types are criminal and civil. Civil cases are brought in violation of specific child abuse laws. They require only proof that injury occurred through nonaccidental means. The ultimate penalty is removal of the child from the family. The rules of evidence tend to be more lenient. In criminal cases one must prove that a specific perpetrator committed a specific crime—a violation of the criminal code. The penalty is incarceration, and the rules of the courtroom are strictly enforced.

65. What are the differences between a fact witness and an expert witness?

A fact witness may testify to what he or she saw, heard, and did in a given case. An expert witness can interpret the facts of the case into an expert opinion. The expert may not have had first-hand contact with the child but bases opinions on record review.

66. In testifying in a child abuse case, what is the role of the ED physician?

The physician must be fair, objective, and reasonable in the presentation of facts and medical data. There is no room for testimony that presents feelings, sentiment, or emotion. The role of the emergency physician is to offer and explain medical information.

67. What is the most important step in managing a child abuse case?

The most important step is to have a high degree of suspicion for every traumatic injury; to build a level of suspicion with objective findings and observations; and when the level of suspicion is high, to report all cases of suspected abuse.

68. Does child abuse affect children only during their childhood?

Many studies have proved that child abuse and even the witnessing of abuse of others may have long-term (lifelong) consequences.

Lee C, White HR: Effects of childhood maltreatment on violent injuries and premature death during young adulthood among urban high-risk men. Arch Pediatr Adolesc Med 2012;166:814-820.

Mills R, Scott J, Alari R, et al: Child maltreatment and adolescent mental health problems in a large birth cohort. Child Abuse Negl 2013;37(5):292-305.

DENTAL INJURIES

Steven Chan and Evaline A. (Evie) Alessandrini

CHAPTER 53

1. How frequently do health care practitioners encounter pediatric dental injuries?

Pediatric dental injuries occur in approximately 50% of all children at some time during childhood with either primary or secondary teeth. Most injuries occur during the summer months. Injuries to the primary teeth typically involve displacement in the alveolar bone, resulting in luxations. The incidence is equal in males and females, and most injuries are caused by falls, usually in the home. Injuries to the permanent dentition most often involve trauma to the hard dental structures, resulting in crown fractures. In this age group, males incur injuries more commonly than females. Trauma to the permanent dentition typically occurs on playgrounds, during sporting events, or as a result of motor vehicle and pedestrian accidents.

Dale RA: Dentoalveolar trauma. Emerg Med Clin North Am 2000;18:521-538.

Wilson S, Smith GA, Preisch J, Casamassimo PS: Epidemiology of dental trauma treated in an urban pediatric emergency department. Pediatr Emerg Care 1997;13:12-15.

2. What is the hardest substance in the human body?

Enamel, which covers the entire crown of the tooth.

3. What are the other components of the tooth?

Other components of the crown of the tooth include dentin, a softer, microtubular structure, and pulp, which provides the tooth's neurovascular supply (Fig. 53-1). The root of the tooth, which anchors it to the alveolar bone, consists of cementum, the periodontal ligament, and the alveolar bone.

4. Why is it important to distinguish primary from permanent teeth?

Management strategies for most dental injuries differ according to the type of tooth.

5. How do I make the distinction between primary and permanent teeth?

- **Primary (deciduous) teeth** begin to erupt at about 6 months of age and are complete by 3 years. A full complement of primary teeth consists of 10 mandibular and 10 maxillary teeth, including four central incisors, four lateral incisors, four canines, and eight molars. Usually, mandibular teeth erupt before their maxillary counterparts (Fig. 53-2).
- **Permanent teeth** typically begin to erupt at 6 years of age and are complete by 16 years of age. A full complement of permanent teeth consists of 16 mandibular teeth and 16 maxillary teeth, including four central incisors, four lateral incisors, four canines, eight bicuspids (premolars), and 12 molars (see Fig. 53-2).

If in doubt, parents usually can distinguish the child's primary from permanent teeth. If a parent is unavailable, two other hints are helpful:

- Primary teeth are often much smaller than permanent teeth.
- The occlusive or chewing surface of the permanent tooth is ridged, whereas the occlusive surface of the primary teeth is smooth.

Helpin ML, Alessandrini EA: Dental trauma. In Schwartz MW, Curry TA, Sargent AJ, et al (eds): Pediatric Primary Care: A Problem-Oriented Approach, 3rd ed. St. Louis, Mosby Yearbook, 1997, pp 777-782.

Nelson LP, Needleman HL, Padwa BL: Dental trauma. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1289-1297.

6. How do I accurately describe which tooth is injured?

There are two ways to describe an injured tooth. The first way is to divide the mouth into quadrants: right maxillary, right mandibular, left maxillary, and left mandibular. Then describe the type of tooth and the quadrant in which it is located. For example, the terms *right maxillary central incisor* and *left mandibular canine* denote both the type of tooth and the quadrant of

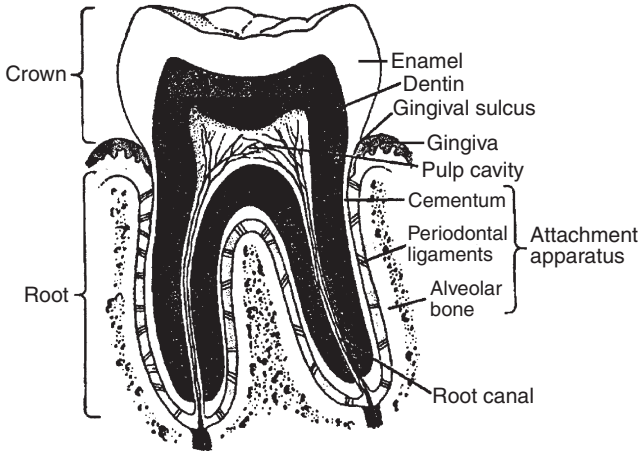


Figure 53-1. Anatomy of a tooth.

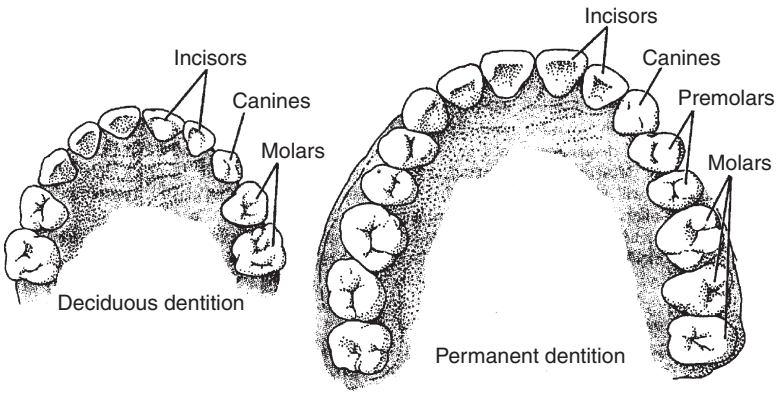


Figure 53-2. Deciduous and permanent dentition of the upper jaw.

the mouth in which it is found (see Fig. 53-2). The second way is the lettering (primary teeth) and numbering (permanent teeth) system. Teeth are assigned starting from the right-most maxillary tooth then proceeding to the left-most maxillary tooth (A through J for primary; 1 through 16 for permanent). The mandibular teeth are assigned starting from the left-most tooth then proceeding to the right-most tooth (K through T for primary; 17 through 32 for permanent) (Fig. 53-3).

Zitelli BJ, McIntire SC, Nowalk AJ: Atlas of Pediatric Physical Diagnosis, 6th ed. Saunders Elsevier, 2012, Chap. 20, pp 775-802.

7. How are broken or fractured anterior teeth classified?

In the Ellis classification system, *class I fractures* involve only the enamel and result in jagged tooth edges but no other sequelae. *Class II fractures* break through both the enamel and dentin of the crown. The yellowish dentin is visible within the pearly white enamel. Class II fractures are often sensitive to heat, cold, and air. *Class III fractures* involve the pulp of the tooth. The pink and bleeding neurovascular bundle of the tooth is exposed, along with the

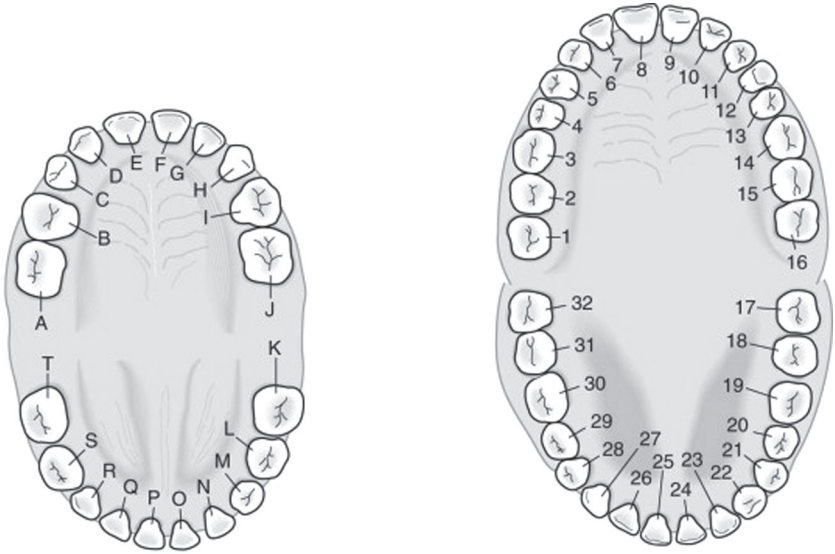


Figure 53-3. Lettering and numbering system of deciduous and permanent dentition.

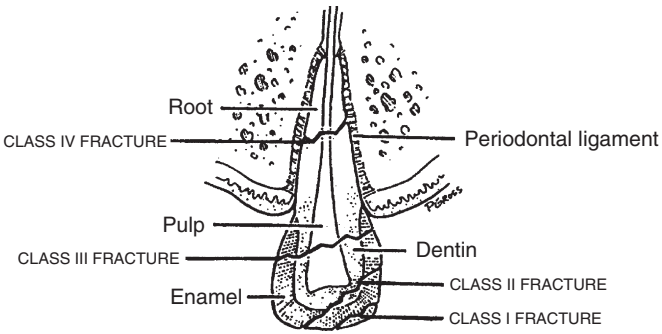


Figure 53-4. Ellis classification of fractures of the anterior teeth.

dentin. Pain is often severe. *Class IV fractures* involve the root. The diagnosis must be confirmed by a dental radiograph or panoramic radiograph (Fig. 53-4).

Diangelis AJ, Andreasen JO, Ebeleseder KA: IADT Guidelines for the management of traumatic dental injuries: 1. Fractures and luxations of the permanent teeth. Dent Traumatol 2012;28(1):2-12.

8. How are fractured teeth treated? How soon does a dentist need to be consulted?

Treatment depends on the classification of the tooth fracture:

- **Class I fractures** require filing of sharp tooth edges to prevent oral soft tissue injury. The tooth fragment can be bonded to the tooth if it is available.
- **Class II fractures** require prompt treatment. Cover the fractured tooth with dental foil (aluminum foil with an adhesive coating) or a calcium hydroxide coating made with commercially available products, such as Dycal. Mix a base and an accelerator and apply this

to the dry tooth. Instruct the patient to eat a soft diet, take analgesics for pain, and see a dentist within 48 hours. Correct treatment of class II fractures decreases the need for root canal therapy.

- Treatment of **class III fractures** is almost identical to that of class II fractures. Delay in dental treatment may result in severe pain and tooth abscess. In the immature tooth, pulp capping or partial pulpectomy is performed in an attempt to preserve the pulp. In the mature tooth, total removal of pulpal tissue (root canal) is preferred. This is followed by cosmetic tooth restoration.
- **Class IV fractures** also require prompt treatment. These fractures may require dental radiographs to diagnose if tooth is not avulsed. If tooth is displaced, repositioning is attempted. Tooth is splinted and healing is monitored to determine if a pulpectomy is necessary.

Diangelis AJ, Andreasen JO, Ebeleseder KA: IADT Guidelines for the management of traumatic dental injuries: 1. Fractures and luxations of the permanent teeth. Dent Traumatol 2012; 28(1):2-12.

9. How are root fractures diagnosed?

Classified by their location along the root, these fractures are identified as coronal, midroot, or apical and are seen most commonly in teeth with complete root formation, approximately 2 to 3 years after eruption. The coronal fractures may be associated with crown displacement and, therefore, are usually the easiest to diagnose clinically. Such fractures with displacement often require immediate dental consultation for splinting. The midroot and apical fractures, however, may only be suspected by the presence of bleeding from the gingival sulcus after a traumatic event and require follow-up intraoral dental radiographs for confirmation.

Nelson LP, Needleman HL, Padwa BL: Dental trauma. In Fleisher GR, Ludwig S, Henretig FM (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2010, pp 1289-1297.

10. When should I suspect an alveolar ridge fracture?

Alveolar ridge fractures occur in less than 10% of all dentoalveolar injuries. They are most commonly associated with anterior teeth and may be either single or segmental. Identification of subtle fractures may be possible by palpating the gingiva and looking for any evidence of crepitus or step-offs. These fractures are commonly seen with luxation injuries of adjacent teeth. Malocclusion is a common finding. The most important management strategy involves the repositioning and splinting of the affected area; therefore, immediate dental consultation is often necessary. Oral antibiotics may also be utilized, although little evidence is available regarding the effectiveness of this strategy.

McTigue DJ: Diagnosis and management of dental injuries in children. Pediatr Clin North Am 2000;47:1067-1084.

Key Points: Tooth Fractures

1. Tooth fractures most commonly occur in the anterior dentition and have four classes: Class I involves enamel only; class II involves enamel and dentin; class III involves enamel, dentin, and pulp; class IV involves enamel, dentin, pulp, and root.
2. Class II and III fractures will have excellent outcomes if they receive proper treatment within 48 hours.
3. Suspect alveolar ridge fractures when palpation of the gingiva reveals crepitus or step-offs, there are large gingival lacerations, or several teeth are luxated en bloc.

11. How are concussion and subluxation injuries diagnosed and treated?

Concussion is defined as a traumatic injury to the tooth and supporting structures without displacement or mobility. Signs and symptoms include exaggerated tooth sensitivity during percussion, chewing, occlusion, or mobility testing. Treatment includes a soft diet and analgesics for 7 to 10 days with dental follow-up to monitor tooth viability.

Subluxation is defined as trauma to the tooth with minor mobility but no displacement. Signs and symptoms include tooth sensitivity to percussion, minor mobility on tooth examination, and blood in the gingival sulcus. Treatment is the same as for tooth concussion, but dental follow-up is more important—not only to monitor tooth viability but also to rule out a root fracture. Subluxation injuries may be splinted for patient comfort.

Diangelis AJ, Andreasen JO, Ebeleseder KA: IADT Guidelines for the management of traumatic dental injuries: 1. Fractures and luxations of the permanent teeth. Dent Traumatol 2012; 28(1):2-12.

12. What is a luxation injury? Define the five types.

Luxation injuries result in physical displacement of the tooth within the alveolar socket, tearing of the periodontal ligament, and possible injury to the alveolar bone. Luxation injuries occur in any of five directions:

1. **Intrusion** describes a tooth impacted into the alveolar socket.
2. **Extrusion** describes a tooth that is vertically dislodged from the socket.
3. **Lingual luxation** is displacement of the tooth toward the tongue. It is common because of the frequency of injuries from an anterior force on the anterior teeth.
4. **Labial luxation** is tooth displacement toward the lips.
5. **Lateral luxations** occur within the plane of the tooth.

International Association of Dental Traumatology: Dental Trauma Guide, 2011. Available at www.iadt-dentaltraumaguide.org. Last accessed June 30, 2014.

13. How are luxation injuries of primary teeth treated?

Although treatment of intrusions differs, treatment of the other four types of luxation injuries is the same. Treatment decisions are based on the degree of displacement and mobility. If the tooth is loose enough to become avulsed and possibly aspirated, then extraction is the treatment of choice. In most circumstances, emergency physicians can extract primary teeth with firm pressure applied manually with the aid of gauze. If the tooth is not too loose, it may be repositioned within the alveolar socket and splinted. A dentist generally does this procedure within 48 hours.

Malmgren B, Andreasen JO, Flores MT: IADT Guidelines for the management of traumatic dental injuries: 3. Injuries in the primary dentition. Dent Traumatol 2012;28(3):174-182.

14. How are luxation injuries of permanent teeth treated?

Luxation injuries of the *permanent dentition* require immediate treatment, usually by a dentist, if malocclusion or significant tooth mobility is present. Otherwise, patients should seek dental care within 48 hours. Therapy for a luxated permanent tooth usually necessitates a local nerve block, followed by repositioning in the alveolar socket and splinting for 10 to 14 days or, if an alveolar fracture is present, 6 weeks. Temporary splinting may be done in the emergency department (ED) with a Coe pack. Zinc oxide and a catalyst are mixed and then applied to the dry injured tooth and one or two teeth on either side for stabilization. The mixture is carefully placed to the gingival line and between the teeth for best results and allowed to dry. The patient must see a dentist within 24 hours for more permanent splinting. In the meantime, the patient may take analgesics and is restricted to a soft diet.

Diangelis AJ, Andreasen JO, Ebeleseder KA: IADT Guidelines for the management of traumatic dental injuries: 1. Fractures and luxations of the permanent teeth. Dent Traumatol 2012;28(1):2-12.

15. How is an intrusion injury to a primary tooth treated?

Treat patients who sustain an intrusion injury to a primary tooth with analgesics in the ED, and instruct them to eat a soft diet and to see their dentist within the next 48 hours. Management of an intruded primary tooth varies. Some dentists obtain a dental radiograph. If the underlying tooth bud is uninjured, they allow the primary tooth to re-erupt. If the permanent tooth bud is injured, the intruded primary tooth is extracted. Others allow the tooth to re-erupt spontaneously. Ninety percent of all intruded primary teeth re-erupt after 6 months.

Kenny DJ, Barrett EJ, Casas MJ: Avulsions and intrusions: The controversial displacement injuries. J Can Dent Assoc 2003;69:308-313.

16. How is an intrusion injury to a permanent tooth treated?

Patients with intruded permanent teeth can be managed conservatively in the ED and instructed to see their dentist within the next 48 hours. In adolescents and patients with more thorough development of the root, the injured tooth is submerged, realigned, and splinted, as described earlier. In younger patients with an immature root, the tooth usually is allowed to

re-erupt spontaneously. If re-eruption has not occurred within 6 weeks, the intruded tooth is submerged, realigned, and splinted.

Kenny DJ, Barrett EJ, Casas MJ: Avulsions and intrusions: The controversial displacement injuries. J Can Dent Assoc 2003;69:308-313.

17. What affects the outcome for intruded permanent teeth?

Intrusion injuries, with displacement of the tooth into the alveolar bone and crushing of the periodontal ligament, can result in significant long-term damage to the dentition. Intrusion injuries to permanent teeth can be especially difficult to manage. The most important factor in outcome is the amount of intrusion present. Several studies have reported that intrusions less than 3 mm have an excellent prognosis, but those greater than 6 mm have a universally poor outcome secondary to inflammatory root resorption and pulp necrosis.

Al-Badri S, Kinirons M, Cole B, Welbury R: Factors affecting resorption in traumatically intruded permanent incisors in children. Dent Traumatol 2002;18:73-76.

Kinirons MJ, Sutcliffe J: Traumatically intruded permanent incisors: A study of treatment and outcome. Br Dent J 1991;170:144-146.

18. What is an avulsion injury?

An avulsion injury is complete displacement of the tooth (crown and root) from the alveolar socket.

19. What storage medium is best for an avulsed tooth?

It is imperative to store the tooth in an appropriate solution to keep the periodontal ligament alive and to ensure successful reimplantation. Appropriate fluids, in descending order of preference, are ViaSpan or Hank's Balanced Salt Solution (HBSS), cold milk, saliva, and saline. Adolescents may place the avulsed tooth under their tongue until they are seen by an emergency physician or dentist. This option is risky with younger children, who may swallow or aspirate the tooth. HBSS is a pH-balanced cell-culture fluid, commercially marketed by 3M as Save-a-Tooth; it can store an avulsed tooth and preserve the periodontal ligament for nearly 24 hours. It is useful for patients with multiple traumas who have more life-threatening injuries that must be addressed first.

Sigalas E, Regan JD, Kramer PR, et al: Survival of human periodontal ligament cells in media proposed for transport of avulsed teeth. Dent Traumatol 2004;20:21-28.

20. What is the procedure for reimplanting an avulsed permanent tooth?

1. Hold the crown of the tooth, and rinse the root with HBSS or saline. To avoid injury to the periodontal ligament, do not handle or scrub the root of the tooth.
2. Insert the root of the tooth into the alveolar socket. The concave part of the tooth should face the tongue.
3. Splint the tooth for 10 to 14 days.
4. If the tooth has been avulsed for a while and the alveolar socket is filled with blood, it may be necessary to perform a local nerve block and irrigate the socket with saline prior to successful tooth reimplantation.
5. Prescribe a soft diet, analgesics, and antibiotics (penicillin or amoxicillin).

Klein BL, Larson BJ: Reimplanting an avulsed permanent tooth. In King C, Henretig FM (eds). Textbook of Pediatric Emergency Medicine Procedures. 2nd edition, Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2008, pp. 669-673.

21. Why is it so important to reimplant a permanent tooth within 30 minutes?

There is a 90% likelihood of tooth survival if a permanent tooth is reimplanted within 30 minutes. The survival rate declines rapidly with time, by approximately 1% for each minute beyond 30 minutes. A tooth reimplanted after 60 minutes of extraoral dry time is almost never viable.

Anderrson L, Adreasen JO, Day P: IDAT Guidelines for the management of traumatic dental injuries: 2. Avulsion of permanent teeth. Dent Traumatol 2012;28(2):88-96.

Klein BL, Larson BJ: Reimplanting an avulsed permanent tooth. In King C, Henretig FM (eds). Textbook of Pediatric Emergency Medicine Procedures. 2nd edition, Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2008, pp. 669-673.

22. Why not reimplant an avulsed primary tooth?

An avulsed primary tooth should never be reimplanted because of a risk of ankylosis—a bony fusion of the tooth with the alveolar bone that may result in facial deformities. In addition, reimplantation of a primary tooth may interfere with eruption of the underlying permanent tooth. Temporary prosthetic devices can be made if a cosmetic effect is desired.

International Association of Dental Traumatology: Dental Trauma Guide, 2011. Available at www.iadt-dentaltraumaguide.org. Last accessed June 30, 2014.

23. When is reimplantation potentially harmful?

Reimplanting an avulsed permanent tooth is potentially harmful in high-risk patients. Give special consideration and take caution in patients with severe dental caries, noncooperating patients, immunocompromised patients, and patients with severe cardiac disease.

Key Points: Tooth Avulsions

1. Never reimplant avulsed primary teeth because of the risk of ankylosis and resultant facial deformities.
2. Fluids suitable to store avulsed permanent teeth, in descending order, are HBSS, milk, saliva, and saline.
3. An avulsed permanent tooth that is reimplanted within 30 minutes has a 90% survival rate.
4. The prognosis for avulsed permanent teeth is highly time-dependent. Complete the reimplantation as soon as possible.

24. When should a chest radiograph be obtained in patients with dental injury?

If the patient has a fractured or avulsed tooth and the entire missing segment has not been found by the medical team, obtain a chest radiograph. Patients requiring lobectomies from aspirated teeth have been reported.

25. When should prophylactic antibiotic therapy be prescribed for patients with dental injuries?

Although this topic is controversial, most authorities agree that prophylactic antibiotics should be prescribed for 3 to 5 days in patients who have sustained an injury to the periodontal ligament. For patients older than 12 years of age, tetracycline is the first choice. Penicillin and amoxicillin are appropriate alternatives. Examples include avulsed permanent teeth, significant luxation injuries, and alveolar ridge fractures. Several studies have suggested that this may decrease the risk of inflammatory root resorption and may, therefore, improve outcome.

Andreasen JO, Andreasen FM, Skeie A, et al: Effect of treatment delay upon pulp and periodontal healing of traumatic dental injuries—A review article. Dent Traumatol 2002;18:116-128.

Finucane D, Kinirons MJ: External inflammatory and replacement resorption of luxated and avulsed replanted permanent incisors: A review and case presentation. Dent Traumatol 2003; 19:170-174.

26. Summarize patient discharge instructions after dental trauma.

- Avoid contact sports.
- Take appropriate analgesics (e.g., acetaminophen, ibuprofen, cold compresses) as needed.
- Eat a soft diet.
- Use chlorhexidine mouth rinse.
- Use a soft toothbrush if able to brush teeth.
- For certain injuries, restrict the use of straws/pacifiers.

27. Summarize the appropriate timing of dental consultation for patients with dental trauma.

Immediate dental consultation:

- Avulsed permanent teeth
- Luxation injuries with malocclusion or significant tooth mobility
- Root fractures with crown displacement
- Alveolar ridge fractures

Urgent dental consultation (within 48 hours):
- Class II and III tooth fractures
- Tooth subluxation
- Tooth intrusion
- Luxation injuries not included earlier

Nonurgent dental consultation (within 1 week):
- Class I tooth fractures
- Tooth concussion

Andreasen JO, Andreasen FM, Skeie A, et al: Effect of treatment delay upon pulp and periodontal healing of traumatic dental injuries—A review article. Dent Traumatol 2002;18:116-128.

28. What is recommended to prevent sports-related dental injuries?

Sports-related dental injuries occur frequently during adolescence. Dental injuries are increased sixfold to eightfold in high school basketball players who do not wear mouthguards. To support this finding, it has been noted that in football, where mouthguards are always worn, 0.07% of the injuries are orofacial, while in basketball, where mouthguards are not routinely worn, 34% of the injuries are orofacial.

Multiple types of these guards are available on the market, including stock, boil and bite, and custom-fitted. It is generally felt that the better-fitted mouthguards, primarily those that are custom-fitted, will be more comfortable and provide better protection against dental injuries.

American Academy of Pediatric Dentistry: Policy on prevention of sports-related orofacial injuries. Pediatr Dent 2010;32(special issue):55-58. http://www.aapd.org/media/Policies_Guidelines/P_Sports.pdf.

Andreasen JO, Andreasen FM, Skeie A, et al: Effect of treatment delay upon pulp and periodontal healing of traumatic dental injuries—A review article. Dent Traumatol 2002;18:116-128.

Key Points: Pediatric Dental Trauma

1. Consider each patient with dental trauma to be a trauma patient like any other.
2. A unique consideration in the evaluation of children with dental injuries is the need to understand normal dental development, as proper identification of primary versus permanent dentition may affect diagnosis and treatment.
3. Primary teeth may be distinguished from permanent teeth because they are usually found in children younger than 6 years of age, are smaller in size, and have a smooth, not ridged, occlusive surface.
4. It is possible to prevent many sports-related dental injuries with the use of properly fitted mouthguards.

Acknowledgment

The authors wish to thank Dr. Linda L. Brown for her contributions to this chapter in the previous edition.

Websites

American Dental Association: Mouthguards. Available at http://www.ada.org/en/member-center/oral-health-topics/mouthguards.

International Association of Dental Trauma: http://www.iadt-dentaltrauma.org/for-patients.html.

National Guideline Clearinghouse: American Academy of Pediatric Dentistry, http://www.guideline.gov/browse/by-organization.aspx?orgid=874.

EXTREMITY INJURIES

John M. Loiselle

CHAPTER 54

1. How do the pediatric musculoskeletal system and its response to stress differ from those in the adult?

- The pediatric skeleton is less densely calcified than the adult version. It is composed of a higher percentage of cartilage. Pediatric bones are lighter and more porous than adult bones, with haversian canals making up a greater percentage.
- The pediatric musculoskeletal system is an actively growing structure. Long bones contain growth plates or physes that are the primary site of this growth. The ends of the bones contain a chondro-osseous segment termed the *epiphysis*, or secondary site of ossification.
- The bones in a child are surrounded by a thick and very active periosteum. This structure provides additional support as well as a high capacity for remodeling injured bone.
- The relative strengths of the different musculoskeletal components differ from child to adult. In the child, the ligaments and periosteum are stronger than the bone itself and less likely to give way under stress. The physis is the weak link. As a result, fractures tend to be relatively more common than sprains or ligamentous injuries in children.
- The degree of ossification, the thickness of the periosteum, and the width of the growth plate vary with age. Therefore, the age of the child and the corresponding anatomy dictate the response of the musculoskeletal system to trauma.

Key Points: Unique Characteristics of the Pediatric Musculoskeletal System

1. Less densely calcified
2. Thick periosteum
3. Presence of the growth plate
4. Relatively strong ligaments

2. What unique categories of fractures are commonly seen in pediatrics as a result of these differences?

- Physeal or Salter-Harris fractures
- Plastic deformation fractures
- Avulsion fractures

Bachman D, Santora S: Musculoskeletal trauma. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1335-1375.

3. Describe the Salter-Harris classification of fractures.

- **Salter I:** A fracture within the growth plate. The fracture line itself is not visible on radiographs, but a widening of the physis or displacement of the epiphysis may suggest such a fracture.
- **Salter II:** A fracture that extends through the growth plate and metaphysis
- **Salter III:** An intra-articular fracture that extends through the growth plate and the epiphysis
- **Salter IV**: An intra-articular fracture that involves the metaphysis, growth plate, and epiphysis
- **Salter V:** A compression fracture of the growth plate. This injury is also unlikely to be detected initially by radiographs and often becomes evident only as the result of eventual growth arrest in the affected limb.

Salter RB, Harris WR: Injuries involving the epiphyseal growth plate. J Bone Joint Surg 1963;45A:587-622.

4. Why is the Salter-Harris classification important?

The Salter-Harris classification of fractures categorizes fractures that involve the growth plate. The classification has implications for both prognosis and treatment. Fractures categorized within the higher numbers of the classification are more likely to affect joint congruity and disrupt blood supply to the growth centers of the bone, leading to greater potential for future growth disturbance.

5. What are the four zones in the physis? Through which zone do fractures commonly occur?

The four zones in the physis are found in the following order from the epiphysis to the metaphysis:

1. The zone of resting cells
2. The zone of proliferating cells
3. The zone of hypertrophic/maturing cells
4. The zone of provisional calcification

Fractures typically occur through the zone of hypertrophic/maturing cells. The blood supply enters the bone through the epiphysis and is most crucial to the resting and proliferating cell layers.

6. At what age are physeal injuries most likely to occur?

Eighty percent occur between 10 and 16 years of age, with a median age of 13 years. Hypertrophy of the physis during the growth spurt and increased participation in physical activity and sports at this age are responsible for the increased incidence.

Peterson CA, Peterson HA: Analysis of the incidence of injuries to the epiphyseal growth plate. J Trauma 1972;12:275-281.

7. How does a mallet finger deformity differ in children and adults?

The adult mallet finger is the result of disruption of the extensor tendon. In young children, the underlying injury involves a fracture through the growth plate—a Salter I fracture with displacement of the epiphysis. In older children or teenagers, the injury is an avulsion or Salter III fracture through the epiphysis. Such injuries may cause difficulty in reducing the finger and may require open reduction.

8. In what order do the growth centers in the elbow ossify? Why is this clinically important?

The mnemonic CRITOE is a useful reminder of the sequence in which these ossified growth centers appear:

- **C:** Capitellum
- **R:** Radial head
- **I:** Internal (medial) epicondyle
- **T:** Trochlea
- **O:** Olecranon
- **E:** External (lateral) epicondyle

An ossified fragment that is present out of its expected order suggests a fracture rather than a normal ossification center.

Yamamoto L: Radiology cases in pediatric medicine: Elbow ossification centers in a child. Radiology in Pediatric Emergency Medicine. Available at www.hawaii.edu/medicine/pediatrics/pemxray/v1c11.html. Accessed 10/5/2014.

9. What are plastic fractures?

Plastic fractures result from the pliability of the bones in childhood. Pediatric bones respond to compressive and transverse forces through plastic deformation. With a small amount of force, the bone is capable of bending slightly and then returning to its natural state. If excessive force is applied, the bone eventually exceeds its capacity for full elastic recoil and deforms. Depending on the degree of force and how it is applied, one of the three plastic fractures of childhood may result.

10. Name the three types of plastic fractures of childhood.

1. Buckle or torus fracture
2. Greenstick fracture
3. Bowing or bending fracture

11. Why is it important to reduce a forearm bowing fracture?

These fractures do not include injury to the periosteum and therefore do not undergo as vigorous stimulation to remodel. This may result in permanent angulation of the bone, which can have deleterious effects on the normal range of motion and function of the involved forearm.

Vorlat P, De Boeck H: Bowing fractures of the forearm in children. Clin Orthop Relat Res 2003;413:233-237.

Key Points: Pediatric Fractures

1. Any fracture can be the result of abuse, although certain extremity fractures are specific for abuse.
2. Because of its increased pliability, the pediatric bone can go through several stages of deformity prior to fracture.
3. Salter-Harris fractures are those involving the growth plate.

12. In what situations are comparison films most useful?

- Suspicion of a bowing fracture
- Suspicion of a nondisplaced Salter I fracture
- Discriminating a secondary or accessory site of ossification from a fracture
- Discriminating a possible anatomic variant from a fracture

13. How do you evaluate distal nerve function in an uncooperative or preverbal child following extremity or digit injury?

Placing the fingers in a bowl of warm water for several minutes results in wrinkling of the skin on the finger pads if distal nerve function is intact.

Antevy PM, Saladino RA: Management of finger injuries. In King C, Henretig FM (eds): Textbook of Pediatric Emergency Procedures, 2nd ed. Baltimore, Wolters Kluwer/Williams & Wilkins, 2008, pp 939-953.

14. What is a FOOSH? What is its significance to pediatric extremity injuries?

A **FOOSH** is a **f**all **o**n an **o**utstretched **h**and. It is the most common mechanism of forearm, elbow, and wrist injuries in children.

Perron AD, Miller MD, Brady WJ: Orthopedic pitfalls in the ED: Pediatric growth plate injuries. Am J Emerg Med 2002;20:50-54.

15. What name is associated with a Salter III fracture of the distal tibia? How is this fracture related to the age and growth of a child?

A Salter III fracture of the distal tibia is called a Tillaux fracture (Fig. 54-1). It classically occurs in teenagers shortly before growth plate closure. The medial segment of the distal tibial physis fuses last, and the medial aspect of the distal epiphysis remains anchored to the fibula by the anterior tibiofibular ligament. External rotation of the foot with sufficient force produces an avulsion fracture through the unfused medial segment of the growth plate and down through the epiphysis.

Ayyagari S, Bancroft LW: Diagnosis: Juvenile Tillaux fracture. Orthopedics 2010;33:207-208.

16. What is the difference between Galeazzi and Monteggia fractures?

Both injuries involve a combination forearm fracture and dislocation. The Galeazzi fracture is a fracture of the radius with dislocation of the radioulnar joint. The Monteggia fracture is an ulnar fracture with radial head dislocation. The dislocations associated with these fractures are at risk of being overlooked once the fracture has been identified.

17. What is a toddler's fracture?

A toddler's fracture is described most commonly as a hairline, nondisplaced spiral or oblique fracture of the distal third of the tibia. It occurs, as its name suggests, in ambulatory children up to approximately 4 years of age. It often results from relatively minor force, such as a fall from a step or the end of a sliding board. Others have expanded the definition to include any subtle or occult injury such as a distal buckle fracture of the tibia.

John SD, Moorthy CS, Swischuk LE: Expanding the concept of the toddler's fracture. Radiographics 1997;17:367-376.

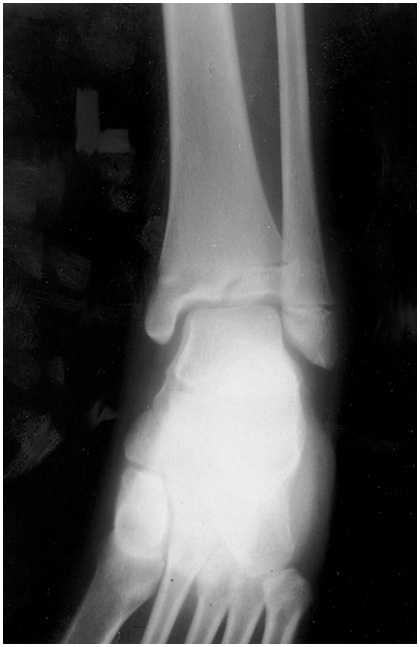


Figure 54-1. Salter III fracture of the distal tibia, commonly called a Tillaux fracture.

18. How do you differentiate an avulsion fracture at the proximal fifth metatarsal from the normal secondary site of ossification?

Avulsion injuries of the proximal fifth metatarsal occur during inversion of the ankle. Stress is transmitted through the peroneus brevis ligament, which inserts on the proximal fifth metatarsal. The resulting fracture line is transverse to the foot, whereas the secondary site of ossification (os vesalianum) occurs in a longitudinal orientation. A comparison view of the normal foot may be helpful. Palpating for tenderness over the area is a simpler means of making the distinction.

19. What is the mechanism of injury in a "boxer's fracture"?

Fractures of the fourth or fifth metacarpal ("boxer's fractures") are sustained when a person strikes an object with a closed fist. Destructive infections of the hand and metacarpophalangeal joint can occur when one person strikes another in the teeth and mouth flora are introduced into the resulting lacerations.

20. Describe the initial approach to the trauma victim with a significantly deformed extremity fracture.

The initial approach to any trauma patient should focus on potential life-threatening injuries. The primary survey consists of evaluation and stabilization of the airway, breathing, and circulation. Do not be distracted by the more obvious extremity injuries, which are rarely life-threatening. Extremity fractures are more appropriately addressed as part of the secondary survey.

21. Describe pain management for pediatric extremity injuries.

Several studies demonstrate inadequate attention to analgesia for pediatric extremity injuries. Provide oral or parenteral analgesics early in the visit. Intravenous administration of morphine (0.1-0.15 mg/kg, max 10 mg) is recommended for displaced fractures. The superiority of oral opiates over ibuprofen has not been proved. Early immobilization of the injured extremity through application of a splint results in significant reduction in pain scores.

Drendel AL, Gorelick MH, Wisman SJ, et al: A randomized clinical trial of ibuprofen versus acetaminophen with codeine for acute pediatric arm fracture pain. Ann Emerg Med 2009;54:553-560.
Koller DM, Myers AB, Lorenz DM, et al: Effectiveness of oxycodone, ibuprofen or the combination in the initial management of orthopedic injury-related pain in children. Pediatr Emerg Care 2007;23:627-633.

22. What factors determine the need for fracture reduction?

Pediatric bones have a high capacity to remodel. As a general rule, remodeling should not be relied upon to correct all deformities. Fractures should be immobilized as close to the anatomic position as possible. No degree of deformation is considered acceptable in all cases. The decision to actively reduce a particular fracture or to rely on remodeling depends on several factors:

- **Patient's age:** The older the child, the less time there is for growth and remodeling.
- **Location of the fracture:** Fractures farther from the physis have less capacity to remodel.
- **Bone involved:** Different bones are subjected to different muscular stresses that in part determine their growth and healing capacity.
- **Type of fracture:** In general, the greater the disruption of periosteum, the greater the stimulation for repair and remodeling.
- **Degree and direction of disruption:** Rotational deformities are particularly poor at remodeling.

23. What pediatric extremity injuries require emergent orthopedic consultation?

- Femur fracture
- Complete fractures of the tibia or fibula
- Open fracture
- Fractures associated with neurovascular compromise
- Dislocation of a large joint, with the possible exception of the shoulder
- Fractures with significant displacement
- Fractures involving a large joint
- Displaced supracondylar fractures

Bachman D, Santora S: Musculoskeletal trauma. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1335-1375.

24. List the key information that should be communicated when an orthopedic consultation is requested.

- Age and sex of the patient
- Mechanism of injury
- Bone or bones involved in the injury
- Type of fracture
- Neurovascular status of the extremity
- Presence and amount of displacement
- Presence and degree of angulation
- Presence or absence of an open fracture

25. Describe the appropriate acute management for the vast majority of extremity injuries.

The mnemonic PRICE summarizes the key elements:

- **P:** Pain control
- **R:** Rest
- **I:** Ice
- **C:** Compression
- **E:** Elevation

The various components of this therapy help to reduce hemorrhage, posttraumatic edema, and pain. Immobilization prevents extension of soft tissue and neurovascular injury. The lack of consistent attention to pain management is a major problem in the care of injured children.

Cimpello LB, Khine H, Avner JR: Practice patterns of pediatric versus general emergency physicians for pain management of fractures in pediatric patients. Pediatr Emerg Care 2004;20:228-232.

26. What is the acute management and recommended follow-up of a distal radius buckle fracture?

Buckle fractures of the distal radius are stable injuries with a high potential for complete remodeling. They are common injuries accounting for 40% of distal radius and ulna fractures in young children. Standard practice has been splinting with follow-up in 1 week in an orthopedic clinic and subsequent application of a short arm cast for 2 to 3 weeks. Shorter periods of immobilization in a splint or removable brace have been shown to provide more rapid functional recovery and greater convenience and are preferred by patients and parents. This management can be monitored in a primary care setting.

Plint AC, Perry JJ, Correll R, et al: A randomized, controlled trial of removable splinting versus casting for wrist buckle fractures in children. Pediatrics 2006;117:691-697.

27. How do you reduce a dislocated patella?

A dislocated patella almost always reduces spontaneously when the leg is placed in extension; it remains dislocated only when the patient maintains the leg in flexion. Flexion of the hips to relax the quadriceps muscles may aid in the reduction. Occasionally a small amount of pressure applied on the medial side of the patella is necessary. Sedation is often needed before movement of the extremity. After reduction, immobilize the joint and obtain radiographs to rule out associated fractures. Whether prereduction radiographs are useful or simply prolong the patient's discomfort remains controversial.

Johnson FC, Okada PJ: Reduction of common joint dislocations and subluxations. In King C, Henretig FM (eds): Textbook of Pediatric Emergency Procedures, 2nd ed. Baltimore, Williams & Wilkins, 2008, pp 962-990.

28. What clinical findings suggest a compartment syndrome?

In addition to a tense, swollen area at the injury site, clinical findings may include the 5 P's:

- Pain
- Paresthesia
- Paresis
- Pallor
- Pulselessness

Pain out of proportion or distal to the injury is the earliest and most sensitive sign of compartment syndrome and is frequently the only one present. One study found pain and an increasing requirement for pain medications in 90% of children with compartment syndrome. Of the other P's, only paresthesia was present in greater than 50% of patients. Pain is increased with passive extension of involved muscles. Notably, the absence of a distal pulse is not necessary for the diagnosis.

Bae DS, Kadiyala RK, Waters PM: Acute compartment syndrome in children: Contemporary diagnosis, treatment and outcome. J Pediatr Orthop 2001;21:680-688.

Newton EJ, Love J: Acute complications of extremity trauma. Emerg Med Clin North Am 2007;25:751.

29. What extremity injuries are at the highest risk for compartment syndrome?

Compartment syndrome can occur in the hand, foot, forearm, thigh, or leg but most frequently involves the anterior tibial and peroneal compartments of the leg and the deep flexor compartment of the arm. Most compartment syndromes in children involve fractures (75-85%). Open fractures carry twice the risk of compartment syndrome of closed fractures. Specific injuries associated with compartment syndrome include:

- Displaced supracondylar fractures
- Forearm crush injuries or midshaft radius and ulna fractures
- Proximal tibia fractures
- Elbow dislocations
- Distal femoral physeal fractures with anterior displacement

Grottkau BE, Epps HR, Discala CD: Compartment syndrome in children and adolescents. J Pediatr Surg 2005;40:678-682.

30. What is Volkmann's contracture?

Volkmann's contracture is the deformation that results from ischemia of the muscles and other soft tissues. It occurs most commonly in the forearm as the result of a compartment syndrome. The predisposing injury in children is frequently a supracondylar fracture.

31. What is a gunstock deformity?

A gunstock deformity or cubitus varus deformity occurs when a supracondylar fracture heals with poor alignment, resulting in medial displacement of the distal humerus. It is the most common complication of supracondylar fractures. Cubitus varus is most often a cosmetic rather than a functional deformity.

Labelle H, Bunnell WP, Duhaime M, et al: Cubitus varus deformity following supracondylar fractures of the humerus in children. J Pediatr Orthop 1982;1:539-546.

32. What is the most common nerve injury resulting from a supracondylar fracture, and how do you clinically assess for it?

Median, radial, and ulnar nerve injuries can all occur as a complication of a supracondylar fracture. The anterior interosseous branch of the median nerve is most commonly injured in extension type supracondylar fractures. This nerve may be injured in up to 50% of patents with type III supracondylar fractures. A nerve palsy of the anterior interossesous nerve results in isolated motor loss and is often missed due to lack of sensory changes. The injury results in loss of the ability to tightly approximate the tips of the index finger and thumb while maintaining flexion of the distal interphalangeal joint of the index finger and the interphalangeal joint of the thumb. Alternatively the patient is unable to extend the interphalangeal joint of the thumb against resistance. The majority of nerve injuries are neuropraxias that resolve spontaneously over several months.

Babal JC, Mehlman CT, Klein G: Nerve injuries associated with pediatric supracondylar humeral fractures: A meta-analysis. J Pediatr Orthop 2010;30:253-263.

33. Describe the diagnosis and management of metaphyseal chip fractures.

The radiograph in Figure 54-2 depicts metaphyseal chip or "corner" fractures. The finding is highly suggestive of child abuse. The likely mechanism is vigorous pulling or shaking of the limbs. Corner fractures are most common in the distal femur and proximal tibia in children under 1 year of age. Treatment consists of splinting and pain management as well as a search for other evidence of abuse, including a skeletal survey. Involve social work services and child welfare for these patients.

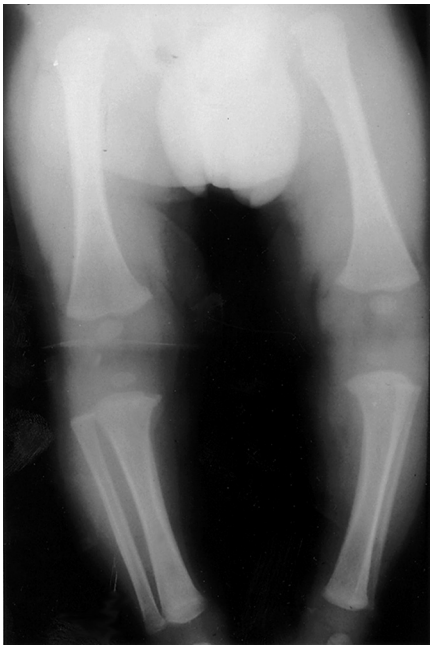


Figure 54-2. Radiograph of a metaphyseal chip or "corner" fracture. This finding is highly suggestive of child abuse. The likely mechanism is vigorous pulling or shaking of the limbs. Corner fractures are most common in the distal femur and proximal tibia.

34. What extremity fractures are associated with abuse?

Up to 10% of all extremity injuries in children are inflicted. Almost any extremity injury may be the result of abuse. Certain classic extremity fractures are more specific for abuse:

- Metaphyseal chip fractures/corner fractures/bucket handle fractures
- Multiple fractures in different stages of healing
- Fractures inconsistent with the history or developmental abilities of the child
- Femur fractures in children under a year of age or in preambulatory children
- Spiral long bone fractures in preambulatory children
- Fractures in association with other injuries suggestive of abuse

35. Explain the term *nursemaid's elbow.*

The term refers to the most common mechanism for obtaining a radial head subluxation injury. A caregiver (nursemaid) may abruptly lift a young child by the wrist while crossing a street or attempting to prevent a stumbling child from falling. Traction is applied to the forearm with the elbow extended and the forearm in pronation. A nursemaid's elbow can occur without a history of this classic mechanism.

36. What is the typical clinical presentation for a nursemaid's elbow?

Children typically do not appear to be in pain but refuse to use the affected arm. The arm is held in pronation and slight flexion at the side of the body. The examiner detects no swelling, point tenderness, bruising, or warmth over the joint. The child may hold the wrist on the affected side to support the extremity, often leading the parents to believe that the wrist is involved. Mild dependent edema of the hand may appear as swelling and further confuse the picture. There is no point tenderness over the elbow, but resistance to supination, flexion, and pronation is noticeable.

37. Name the classic radiographic findings for a radial head subluxation (nursemaid's elbow).

There are no diagnostic radiographic findings for a radial head subluxation. Radiographs appear normal. They should be obtained only when the diagnosis is in question after the history and physical examination. In this setting, radiography is performed to rule out alternative causes of elbow pain.

Swischuk LE: Emergency Imaging of the Acutely Ill or Injured Child, 4th ed. Philadelphia, Lippincott Williams & Wilkins, 2000.

38. Describe two accepted maneuvers for reducing a radial head subluxation.

In both procedures, the child is seated in the parent's lap, facing the examiner. The examiner grasps the elbow and provides stabilization with the nondominant hand. With the dominant hand, the examiner holds the wrist or hand of the child's affected side and either (1) extends the elbow and hyperpronates the forearm or (2) supinates the forearm and then flexes the elbow in one smooth motion so that the child's hand comes up to the ipsilateral shoulder. A palpable "pop" may be appreciated at the elbow as the reduction occurs. The hyperpronation maneuver has a higher success rate and is perceived to be less painful.

Krul M, van der Wouden JC, van Suijlekom-Smit LW, Koes BW: Manipulative interventions for reducing pulled elbow in young children. Cochrane Database Syst Rev 2012;1:CD007759.

Key Points: Findings in Radial Head Subluxation

1. History of forearm traction in pronation
2. Refusal to use arm
3. Lack of swelling or bruising
4. Negative radiographs (if obtained)

39. What radiologic findings on a lateral elbow film suggest the presence of an occult fracture?

A visible posterior fat pad or outward displacement of the anterior fat pad away from the humerus is evidence of an elbow effusion. Assume a fracture to be present when an elbow effusion is seen in the setting of elbow trauma.

A malaligned anterior humeral line (one that does not pass through the middle or posterior third of the capitellum) suggests the presence of a supracondylar fracture with dorsal displacement of the capitellum and distal humerus.

Malalignment of the radiocapitellar line, as determined by a line through the center of the radius that does not pass through the center of the capitellum on either the anteroposterior or lateral radiograph, is evidence of a dislocated radius.

Swischuk LE: Emergency Imaging of the Acutely Ill or Injured Child, 4th ed. Philadelphia, Lippincott Williams & Wilkins, 2000.

40. What is the most frequently fractured bone in a pediatric elbow injury?

In one study of children with elbow fractures, 81% of elbow fractures involved the distal humerus. Radial head fractures accounted for 9% of elbow fractures, and proximal ulnar fractures, for 10%. Of the fractures involving the humerous, 68% were supracondylar fractures, 23% were lateral condyle fractures, and 9% were medial epicondyle fractures.

John SD, Wherry K, Swischuk LE, et al: Improving detection of elbow fractures by understanding their mechanics. Radiography 1996;16:1443-1460.

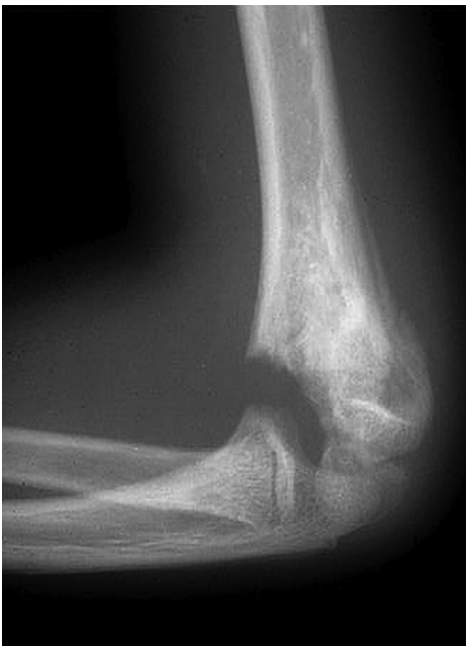
41. What are the three classifications of supracondylar fractures?

- **Type I:** Nondisplaced fracture
- **Type II:** Mild displacement with intact posterior cortex
- **Type III:** Displaced fracture with no anterior and posterior cortical contact

All type III fractures and most type II supracondylar fractures (Fig. 54-3) require percutaneous pinning to obtain appropriate reduction and to reduce complications.

Clifford R. Wheeless, III, MD: Pediatric supracondylar fractures of the humerus. Wheeless' Online Textbook of Orthopedic Surgery. Available at www.wheelessonline.com/ortho/pediatric_supracondylar_fractures_of_the_humerus. Last updated 3/4/2013.

Figure 54-3. Supracondylar fracture.



Key Points: Elbow Trauma

1. The most common mechanism of pediatric elbow injury is a fall on an outstretched hand.
2. The presence of a posterior fat pad or elevated anterior fat pad is evidence of a supracondylar fracture until proved otherwise.
3. Type II and type III fractures typically require surgical pinning for adequate reduction.
4. Secondary ossification centers in the elbow appear in a predictable order.

Websites

Radiology cases in pediatric medicine. Available at www.hawaii.edu/medicine/pediatrics/pemxray/pemxray.html.
Reviews and radiology cases in pediatric orthopedics. Available at www.pemdatabase.org/ortho.html.
Wheeless' Online Textbook of Orthopedic Surgery. Available at www.wheelessonline.com.

EYE INJURIES

Martha W. (Molly) Stevens

CHAPTER 55

1. How common are eye injuries in the pediatric population?

Eye injuries are a very common pediatric problem. It is estimated that there are 70,000 children (<15 years) with eye injuries annually. Eye trauma is the leading cause of unilateral blindness in the United States. Boys are four times more likely to have an eye injury than girls. Many eye injuries are related to sports, and many are preventable with use of proper eye protection.

2. Name two injuries that are ophthalmologic emergencies.

Injuries that lead to incremental vision loss without early recognition and prompt initiation of treatment are ophthalmologic emergencies. Two such injuries include chemical burns and globe rupture. Both warrant emergent (same-day) ophthalmologic consultation after emergent initiation of treatment/stabilization in the emergency department (ED).

3. List the important aspects of the history in all patients with eye injuries.

- Change in visual acuity
- Change in appearance of eyes
- Discomfort or pain (including photophobia)
- History of trauma, including details of the mechanism (blunt/sharp, significant impact, soil contamination, foreign-body risk)
- Corrective lenses (contacts currently in place?)
- Medications
- Eye surgeries
- History of ocular problems
- Systemic disease (sickle cell, connective tissue, or rheumatologic disease; hypertension; diabetes; human immunodeficiency virus [HIV] infection)
- Tetanus immunization history

4. What should be included in the routine physical examination for all pediatric patients with eye injuries (except when temporarily deferred in absolute emergencies)?

- Visual acuity
- Extraocular muscles
- Pupillary reactions
- External examination (including lids and bulbar/palpebral conjunctivae)
- Direct ophthalmoscopy/red reflex
- Other tests as indicated: visual field testing, slit-lamp examination, intraocular pressure (palpation or tonometer). The last two tests usually are performed by an ophthalmologist for pediatric patients.

5. What are the common pitfalls in evaluating children with eye injuries?

- Failure to treat life-threatening injuries before the more obvious (but less serious) eye injury
- Failure to examine both eyes
- Failure to consider globe injury (after finding a more superficial injury)

Levin AV: Eye trauma. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1448-1458.

6. How do you test visual acuity in infants?

Check pupillary reaction to light and fixation reflex. Infants should be able to fix on a high-contrast object held in their central vision, such as the caretaker's or examiner's face or a bright toy. By 2 to 3 months of age, they should be able to fix on and start to follow an object or light, and by about 6 months, to fix and follow to all visual fields. Central, steady, maintained fixation is estimated to be equivalent to 20/40 vision, unsteady fixation equivalent to 20/100, and eccentric wandering gaze equivalent to less than 20/400.

7. How do you test visual acuity in preschool- and school-aged children?
- 2½ to 3½ years old: Picture chart (Allen object recognition)
- 3½ and older: Picture chart, tumbling E chart, or Sheridan-Gardner visual acuity test
- School age: Snellen chart (letters in rows of diminishing size)

8. What pearls should be kept in mind for visual acuity testing in children?
- Have the child identify the objects on the chart up close before testing at a distance.
- Test one eye at a time, "bad" eye first.
- Use a card or the child's palm to cover the other eye; children "peek" between fingers.
- Always test with corrective glasses if available.

9. For patients without their glasses or contact lenses, how can you differentiate between decreased acuity and baseline refractive error?
Pinhole testing—with a premade device, a card with a hole punched with an 18-gauge needle, or multiple pinholes in a rigid eye shield—corrects refractive errors to about 20/30.

10. How do you record decreased acuity in patients unable to visualize a chart?
- Ability to count fingers from a specified number of feet
- Ability to perceive hand motion
- Light perception (with or without directionality)

11. What are the causes of an irregularly shaped pupil?
- Ocular rupture/corneal or other globe perforation
- Prior ocular surgery
- Scarring from previous iritis

12. What are the causes and expected course of subconjunctival hemorrhage?
Subconjunctival hemorrhage, the localized rupture of small subconjunctival vessels, may be spontaneous or may be due to direct trauma or increased intrathoracic pressure (e.g., coughing, nose-blowing, vomiting). It usually is benign and resolves in 2 to 3 weeks without treatment or sequelae. Very large hemorrhages or those that fully encircle the iris raise concern for an underlying injury, such as globe perforation, or a bleeding diathesis.

13. What is the most common pediatric eye injury?
Corneal or conjunctival abrasion.

14. Which eye injuries are seen when a child is hit by an airbag in a car crash?
Pediatric eye injuries from airbag deployment are caused by blunt force trauma and chemical burns. Injuries can be monocular or binocular and include eyelid and corneoscleral abrasions and lacerations, lens dislocation, iris detachment, hyphemas and vitreous hemorrhages, globe rupture, retinal injuries, and alkaline burns (from the alkaline combustion byproduct of sodium azide). Wearing glasses at the time of the crash protects against the chemical injury but increases the risk of penetrating trauma to the eyes.

Eliott D, Hauch A, Kim RW, Fawzi A: Retinal dialysis and detachment in a child after airbag deployment. J AAPOS 2011;15:203-204.

Salan T, Stavrakas P, Wickham l, Bainbridge J: Airbag injury and bilateral globe rupture. Am J Emerg Med 2010;28:982.

Scarlett A, Gee P: Corneal abrasion and alkali burn secondary to automobile air bag inflation. Emerg Med J 2007;24:733-734.

Subash M, Manzouri B, Wilkins M: Airbag-induced chemical eye injury. Eur J Emerg Med 2010;17:22-23.

15. How does a child with corneal abrasion typically present?
The common presentation is a red eye with epiphora (tearing), intense pain, resistance to eye opening, and, less frequently, lid swelling or photophobia. The child often does not know what caused the injury and may complain of a foreign body sensation.

16. How is a corneal abrasion diagnosed?
- Larger abrasions can be seen with tangential light (surface irregularity or dry-appearing area).
- Fluorescein staining and cobalt blue light (or Wood's lamp) best delineate abrasions.
- Use a topical anesthetic to greatly facilitate the examination and aid in the diagnosis (pain will be fully alleviated with an ocular surface problem—superficial foreign body, abrasion, or

superficial ulcer). Persistent pain after a topical anesthetic suggests injury of deeper structures.

- Check thoroughly for a retained foreign body when evaluating corneal or conjunctival abrasions.

Pediatric pearl: Consider application of a topical anesthetic *before* your first examination attempt in suspected corneal or conjunctival abrasions. In young children, this is most easily accomplished by letting the child keep the eye shut, having the child lie in a supine position, and pooling several drops of the topical anesthetic medially at the corner of the eye. Gentle traction caudally on the cheek will crack apart the lids, and the drops will run onto the eye surface. Let the child sit up, and in a few seconds he or she will usually spontaneously open the eye and your examination can proceed without a fight!

17. Describe the management of a corneal or conjunctival abrasion.

Most abrasions heal quickly without sequelae. Antibiotic ophthalmic ointment or artificial tears provide lubrication and some pain relief. Small lesions do not need to be patched and, if asymptomatic in 24 hours, do not require follow-up. Large abrasions and those involving the visual axis are treated with antibiotic ointment; consider soft eye patching and daily follow-up until they have healed. Instruct patients to return or see an ophthalmologist for pain or foreign body sensation that persists for more than 2 or 3 days, or if pain and redness worsen at any time. Instruct those who wear contact lenses to discontinue their use until the patient is seen by an ophthalmologist.

Turner A, Rabiu M: Patching for corneal abrasion. Cochrane Database Syst Rev 2006;(2):CD004764.

18. Describe the management of the child with "something in my eye."

- Most patients with an external foreign body (conjunctival or superficial corneal) report just that.
- If there is no suspicion of a perforated globe or deep corneoscleral laceration, instill a topical anesthetic, which will relieve pain fully and allow thorough examination.
- Thoroughly examine the bulbar conjunctival surface, cornea, palpebral conjunctivae, and superior and inferior fornices.
- Perform a full conjunctival surface evaluation: Evert (flip up) the upper lid to visualize the inner lid surface; exert outward traction on the lower lid with upward gaze to visualize the lower bulbar and palpebral conjunctivae.
- The foreign body usually can be loosened and removed with saline irrigation or a moistened sterile swab or gauze. If the foreign body is still adherent, consider removal under slit-lamp magnification or ophthalmologic referral. Be sure to complete a full eye examination after removal of the foreign body; treat associated abrasions.
- Seek urgent ophthalmology consultation for a corneal or scleral laceration or an imbedded foreign body.

19. What is a hyphema?

A hyphema is bleeding into the chamber between the cornea and iris or lens after blunt or sharp trauma. It may range from microscopic bleeding to a full-chamber or "eight-ball" hyphema.

20. How are hyphemas diagnosed and treated?

Suspect a hyphema (Fig. 55-1) in any child with a tearing, painful eye and injected bulbar conjunctiva immediately after blunt or lacerating eye trauma. It is often diagnosed by careful

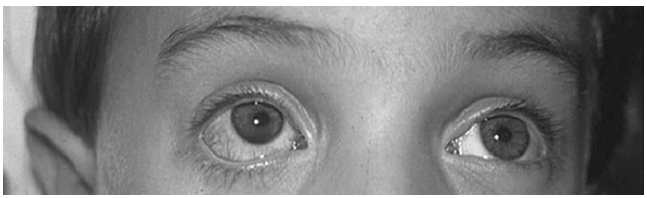


Figure 55-1. Hyphema. Note layering of blood in right eye.

physical examination; very small and microscopic hyphemas require microscopic slit-lamp examination to visualize. All hyphemas require urgent ophthalmologic consultation for evaluation, management, and follow-up. With treatment, 66% to 97% of isolated hyphemas will fully resorb without complication.

Children with large or complex hyphemas are occasionally admitted to the hospital for management, particularly if the patient is young or has sickle cell hemoglobinopathy or hemophilia, or if inadequate follow-up is a concern. Most children with hyphemas are managed at home with frequent outpatient follow-up after the initial visit. Therapies include cycloplegics, topical or systemic steroids, analgesics, antifibrinolytic agents, and restricted activity or bed rest.

Recchia FM, Saluja RK, Hammel K, et al: Outpatient management of traumatic microhyphema. Ophthalmology 2002;109:1465-1470.

SooHoo JR, Davies BW, Braverman RS, et al: Pediatric traumatic hyphema: A review of 138 consecutive cases. J AAPOS 2013;17(6):565-567.

21. What is the major potential vision-threatening sequela of hyphemas? Who is at risk?

Rebleeding with development of acute glaucoma or corneal staining is the most serious sequela. Rebleeding is most likely within 3 to 5 days of the initial injury. Patients with sickle cell disease are at particularly high risk of ocular complications; a screening test or hemoglobin electrophoresis is recommended for patients at risk for an undiagnosed hemoglobinopathy.

22. Why are pateints with sickle cell disease at high risk for complications secondary to a hyphema?

Sickle cell patients are at higher risk for complications from a hyphema because of the relatively hypoxic and acidic environment of the anterior chamber that predisposes erythrocytes to sickle. These abnormally shaped cells are then slow to exit through the filtration angle and may block exit of blood, leading to elevation of intraocular pressure. Sickle cell patients are also more likely to develop optic nerve atrophy at lower intraocular pressure than other children.

Bloom JN: Traumatic hyphema in children. Pediatr Ann 1990;19(6):368-375.

23. When and how does traumatic iritis present?

Traumatic iritis (Fig. 55-2), which may accompany other ocular injury or be the sole manifestation of blunt eye trauma, is characterized by a delayed presentation, typically 24 to 72 hours after injury. Physical examination reveals a painful, red eye (perilimbal conjunctival injection), tearing, and pain with pupillary constriction (on accommodation or concentric constriction to light). The affected eye also may have slight miosis and a decreased or sluggish pupillary response. The pain is secondary to inflammation in the anterior chamber. Slit-lamp evaluation is diagnostic when it reveals white blood cells and a protein "flare" in the aqueous humor.

24. How is traumatic iritis treated?

In children, request an ophthalmology consultation for full evaluation and initial management, which includes mydriatics for pain control, topical steroids, and close outpatient follow-up.

25. What is an orbital "blow-out" fracture?

An orbital blow-out fracture involves one of the bony orbital walls and is usually caused by blunt trauma to the face. The presentation may include periorbital ecchymosis, facial

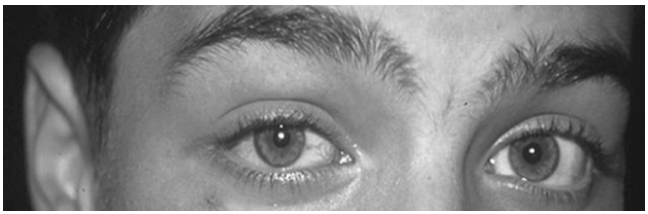


Figure 55-2. Iritis.

asymmetry, lid swelling or ptosis, proptosis or enophthalmos, ophthalmoplegia, localized anesthesia of the face, and, in rare cases, orbital emphysema. The patient may have entrapment or restriction of the extraocular muscle adjacent to the fracture site and restriction of extraocular movement on examination. In particular, the patient may be unable to look up with the affected eye. The most common fracture sites are the inferior and medial walls (rarely lateral). Superior wall fractures have the potential to communicate with the intracranial space. Computed tomography (CT) or magnetic resonance imaging typically delineates the fracture. A full ocular examination is needed to rule out other eye injuries, including retinal trauma.

26. What is the major pitfall when diagnosing an orbital wall fracture?

Failure to consider and appropriately rule out other eye injuries. The incidence of concomitant globe injury with orbital fractures has been reported in up to 20% of cases.

27. What are the indications for obtaining a CT scan of the orbits when a patient has a blow to the face and presents with pain, periorbital swelling, and ecchymosis?

- Evidence of fracture on examination
- Limitation of extraocular motility
- Decreased visual acuity
- Severe pain
- Inadequate examination of the eye (soft tissue swelling prevents eye examination)
- Altered mental status

Neuman MI, Bachur RG: Orbital fractures. UpToDate, 2014. Available from http://www.uptodate.com.

28. What physical examination findings are consistent with a ruptured or perforated globe?

- Significantly decreased visual acuity and severe pain
- Characteristic tear-drop pupil, pointing toward perforation
- Mucoid, fleshy, or pigmented-appearing mass on the ocular surface (extrusion through the perforation or rupture by vitreous or aqueous humor, choroid, or iris; do not mistake this for a foreign body!)
- Large, overlying subconjunctival hemorrhage (often fully around iris) or hyphema
- An unusually shallow anterior chamber

Levin AV: Eye trauma. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1448-1458.

Key Points: Reasons to Suspect Globe Perforation or Rupture

1. History of hammering/grinding metal
2. Significant eye trauma causing decreased vision
3. Physical examination findings of a subconjunctival hemorrhage encircling the iris or cornea; large hyphema; posttraumatic corneal conjunctival edema; enophthalmos; extruded vitreous, iris, or choroid; irregularly shaped pupil
4. The more difficult it is to examine the patient after trauma (because of pain, edema, hyphema, or vitreous hemorrhage), the greater the concern for globe rupture.

29. What aspects of the history or physical examination place patients at high risk for globe perforation or rupture?

- Hammering/grinding metal
- Significant eye trauma causing decreased vision
- Subconjunctival hemorrhage encircling the iris or cornea
- Large hyphema
- Posttraumatic corneal conjunctival edema
- Enophthalmos
- Irregularly shaped pupil
- Extruded vitreous, iris, or choroid

Note: It is sometimes difficult to see a globe rupture, especially if the cause was a rapidly penetrating foreign body (from hammering). The more difficult it is to examine the patient after trauma (because of pain, edema, hyphema, or vitreous hemorrhage), the greater the likelihood of globe rupture.

Tok O, Tok L, Ozkaya D, et al: Epidemiological characteristics and visual outcome after open globe injuries in children. J AAPOS 2011;15(6):556-561.

30. What are the initial treatments/stabilization for globe rupture?

- Stop the examination; place a rigid eye shield.
- Have the patient rest in bed with the head elevated.
- Keep the patient calm; treat pain and nausea/vomiting as needed (to avoid increased pressure to the globe, which can cause further extrusion of vitreous or aqueous humor).
- Consider administration of tetanus and intravenous (IV) antibiotic prophylaxis.
- Order imaging studies as indicated (concern for fracture, retained foreign body, intraorbital hematoma).

Key Points: Management of Globe Rupture/Laceration

1. Stop your examination.
2. Place shield over affected eye.
3. Keep patient at rest, with head elevated.
4. Avoid agitation of the child; treat pain and nausea/vomiting if present.
5. Ensure that patient consumes nothing by mouth.
6. Consult ophthalmologist urgently.
7. Consider IV antibiotics.

31. What are the important considerations in evaluating lacerations of the eyelids?

- Evaluate for full-thickness laceration and underlying orbital/globe injury.
- Check the inner (conjunctival) surface (superficial-appearing lacerations may be full thickness).
- Assess the integrity of the levator muscle (both lids raise equally with upward gaze).
- Lacerations of the medial third of the upper or lower lid potentially involve the lacrimal canaliculi.
- Adipose tissue exposed/extruding in an upper lid laceration indicates full-thickness lid laceration (upper lid has no adipose; this is orbital fat).
- Full-thickness lacerations, lacerations through the lid margin or tarsal plate, and lacerations involving lacrimal canaliculi require ophthalmologic consultation for multilayer repair.
- Consider consultation for lacerations with ptosis or significant avulsions or if you are unable to rule out other ocular/globe injury.

32. Describe the emergency initial management of chemical burns to the eye.

- Recommend that parents put the child under running water immediately, to clean the eyes prior to transport to the ED.
- Upon ED arrival, *irrigate immediately* (begin in triage if possible) and copiously with normal saline or lactated Ringer's solution, at least 1 to 2 L via IV tubing set.
- Consider a similar, perhaps less severe, injury to the other eye.
- Use topical anesthetic early and every 20 minutes; retract lids (with gauze or retractors) to ensure thorough irrigation; remove particulate matter from fornices.
- Monitor pH of conjunctival fossa after irrigation and recheck 10 to 20 minutes later. Continue irrigation until a pH of 7.4 to 7.6 is maintained on rechecks.
- Arrange emergent (same day) ophthalmologic consultation for patients with evidence of a significant burn (such as persistent pain, corneal lesions, or decreased acuity).

Blende MS: Irrigation of conjunctivae. In King C, Henretig FM (eds): Textbook of Pediatric Emergency Procedures, 2nd ed. Baltimore, Williams & Wilkins, 2008, pp 555-558.

33. Which are more severe—alkali or acid burns?

Alkali burns are potentially more severe. Alkaline substances penetrate more deeply than acids, which coagulate with surface proteins. Common alkaline agents include lye, lime, ammonia, and aluminum-containing or magnesium hydroxide–containing fireworks (flares and sparklers). Automobile airbag deployment is a reported cause of alkali eye burns (combustion byproduct of sodium azides).

34. An emergency physician attempts to repair a forehead laceration of a young child using cyanoacrylate glue. Some of the glue drips down the child's face and the child's eyelashes are noted to be stuck together. The child's parent is quite concerned. Which eye injuries are likely?

Usually none. The eyelashes may be stuck together by the glue, but the eye is very rarely injured in this scenario. Parents may be concerned, as the child cannot open his/her eye. Avoid forceful attempts to pry open the eyelids. Instead, place petroleum jelly on the eyelashes and wait patiently or massage with a cotton swab until the glue dissolves.

CHAPTER 56

HEAD TRAUMA

Sara A. Schutzman

1. How common and important is head trauma in children?

Head trauma accounts for more than 600,000 emergency department (ED) visits and 50,000 hospitalizations per year. Traumatic brain injury is the most common cause of death and disability in childhood.

Faux M, Xu L, Wald MM, Coronado VG: Traumatic Brain Injury in the United States: Emergency Department Visits, Hospitalizations and Deaths 2002-2006. Atlanta, Centers for Disease Control and Prevention, National Center for Injury Prevention and Control, 2010.

2. What kinds of head injuries commonly present to the ED?

Head trauma includes injuries to the scalp, skull, and intracranial contents. Although most injuries are minor, there is a wide spectrum ranging from simple contusion to lethal brain injury. Lacerations and contusions are common scalp injuries. Injuries to the cranial vault result in skull fractures, and intracranial injuries (ICIs) include concussion, cerebral contusion, hematoma (epidural, subdural, subarachnoid, and intracerebral), and acute brain swelling. Most head injuries result from blunt head trauma, but penetrating injuries do sometimes occur and are caused by bullets, teeth (e.g., dog bites), or other sharp objects (e.g., dart, pellet, pencil).

3. How is head trauma severity defined?

There is no standard definition for minor head trauma, which accounts for about 80% of injuries evaluated. Many definitions of minor head trauma have been based on the Glasgow Coma Scale (GCS) score; however, there has been no consistency among the various sources, which include children with GCS scores of 13 to 15, 14 to 15, or 15. The American Academy of Pediatrics defined children with minor head injury as those who have normal mental status at the initial examination, a normal neurologic examination, and no physical evidence of skull fracture. Moderate head trauma is typically defined as a GCS score of 9 to 12, and severe head trauma, as a GCS score of 3 to 8.

Committee on Quality Improvement, American Academy of Pediatrics: The management of minor closed head injury in children. Pediatrics 1999;104:1407-1415.

4. How many children evaluated for minor head trauma have ICIs?

Approximately 3% to 7% of children with minor head injury who present for evaluation have an ICI noted on computed tomography (CT). Approximately 0.1% to 0.6% require surgical intervention. Overall, about 50% of ICIs occur in children with minor head trauma.

Dunning J, Daly JP, Lomas JP, et al: Derivation of the children's head injury algorithm for the prediction of important clinical events decision rule for head injury in children. Arch Dis Child 2006;91:885.

Kuppermann N, Holmes JF, Dayan PS, et al: Identification of children at very low risk of clinically-important brain injuries after head trauma: A prospective cohort study. Lancet 2009;374:1160.

Osmond MH, Klassen TP, Wells GA, et al: CATCH: A clinical decision rule for the use of computed tomography in children with minor head injury. CMAJ 2010;182:341.

5. What are clinically important ICIs?

Researchers have focused more recently on patients deemed to have injuries that were of significant clinical importance (i.e., not just a small abnormality noted on CT requiring no intervention). One large study published by the Pediatric Emergency Care Applied Research Network (PECARN, Kuppermann and associates) defined clinically important injuries as those resulting in death, neurosurgery, intubation for more than 24 hours, or hospital admission for 2 or more nights. A large Canadian study (CATCH, Osmond and colleagues) defined clinically important injuries as those requiring neurologic intervention, neurosurgery, monitoring of intracranial pressure (ICP), or intubation or resulting in death.

Kuppermann N, Holmes JF, Dayan PS, et al: Identification of children at very low risk of clinically-important brain injuries after head trauma: A prospective cohort study. Lancet 2009;374:1160.
Osmond MH, Klassen TP, Wells GA, et al: CATCH: A clinical decision rule for the use of computed tomography in children with minor head injury. CMAJ 2010;182:341.

6. Name the ways in which infants differ from older children with regard to head trauma.

Children younger than 1 to 2 years of age differ in several ways, making a low threshold for head imaging prudent:

- Clinical assessment is more difficult.
- ICI is frequently asymptomatic (i.e., they may have swelling but no symptoms of brain injury).
- Skull fractures and ICIs may result from relatively minor trauma.
- Inflicted injury occurs more often.

However, these youngest children are also the most sensitive to the effects of ionizing radiation (from CT), because they have a longer lifetime to manifest cancer and have increased radiation sensitivity of developing tissues.

Brenner DJ, Elliston CD, Hall EJ, et al: Estimated risks of radiation-induced fatal cancer from pediatric CT. AJR 2001;176:289-296.
Brenner DJ, Hall EJ: Computed tomography—An increasing source of radiation exposure. N Engl J Med 2007;357:2277-2284.
Schutzman SA, Barnes P, Duhaime AC, et al: Evaluation and management of children younger than two years of age with apparently minor head trauma: Proposed guidelines. Pediatrics 2001;107:983-993.

7. How common is head trauma in infants and young children related to abuse?

Although most head trauma is accidental, the incidence of abusive head trauma for children younger than 1 is 30 per 100,000 person-years; 20% to 30% of young children admitted for head trauma are victims of intentional injury, and abusive head trauma is the most common cause of traumatic death in children. Because these children are preverbal and abusive caretakers are rarely forthcoming, have a heightened sense of awareness to diagnose nonaccidental trauma. This is important to guide appropriate therapy and to prevent further trauma.

Christian C: Child abuse: Epidemiology, mechanisms, and types of abusive head trauma in infants and children. UpToDate, 2013. Available from www.uptodate.com.
Jenny C, Hymel KP, Ritzen A, et al: Analysis of missed cases of abusive head trauma. JAMA 1999; 281:621-626.

8. Describe the different types of skull fractures.

Skull fractures are described in terms of location and characteristics. They may involve the frontal, parietal, occipital, and temporal bones of the skullcap (calvarium). The skull base consists of portions of the temporal and occipital bones, along with the maxillary, sphenoid, and palatine bones. Fractures in this area are referred to as basilar skull fractures.

Fractures may be linear, depressed (if the inner table of the skull is displaced by more than the thickness of the entire bone), or diastatic (traumatic separation of the cranial bones at one or more suture sites). Compound fractures communicate with lacerations, and comminuted fractures are those with several fragments.

9. Why are skull fractures important?

Skull fractures are one of the best predictors of ICI. Depressed and basilar fractures may have significant complications related to the fracture. Simple linear fractures develop complications in only a small percentage of cases. In certain circumstances fractures may also be important evidence of child abuse.

Dunning J, Daly JP, Lomas JP, et al: Derivation of the children's head injury algorithm for the prediction of important clinical events decision rule for head injury in children. Arch Dis Child 2006;91:885.
Kuppermann N, Holmes JF, Dayan PS, et al: Identification of children at very low risk of clinically-important brain injuries after head trauma: A prospective cohort study. Lancet 2009;374:1160.

Osmond MH, Klassen TP, Wells GA, et al: CATCH: A clinical decision rule for the use of computed tomography in children with minor head injury. CMAJ 2010;182:341.

10. Are skull films indicated/helpful in evaluating children with head trauma?

CT is the imaging modality of choice to evaluate for acute injury, because skull radiography gives no direct information about ICI. However, skull radiographs may occasionally be useful to screen for fracture and avoid the risk of higher radiation and sedation from CT in selected asymptomatic patients 3 to 24 months of age with concerning scalp hematomas. Skull radiography offers the advantage of requiring no sedation and having significantly less radiation than a CT scan. Skull radiographs (if ordered) may be challenging to interpret, and it is important that a skilled radiologist be available to read such films. Obtain a head CT if a fracture is identified.

Consider skull radiography in:

- Alert, asymptomatic infants with scalp hematomas: These infants are at risk for harboring occult ICIs, and skull fractures are one of the best predictors for ICI.
- Possible nonaccidental injury: Skull radiography sometimes detects fractures missed by CT and is indicated (as part of a skeletal survey) for the evaluation of possible abuse.
- Suspicion of possible depressed fracture, penetrating trauma, or foreign body.

Chung S, Schamban N, Wypij, et al: Skull radiograph interpretation of children less than age two: How good are pediatric emergency physicians? Ann Emerg Med 2004;43:718-722.
Tang PH, Lim CC: Imaging of accidental paediatric head trauma. Pediatr Radiol 2009;39:438.

11. Name the most important complications of basilar skull fractures.

- **ICI:** From 10% to 40% of patients with basilar skull fractures have an associated ICI, and about 20% of alert children with basilar skull fractures and a normal neurologic status have an ICI.
- **Cerebrospinal fluid (CSF) leak:** An associated dural tear may lead to CSF leak through the nose or ear and occurs in approximately 15% to 30% of children with basilar skull fractures.
- **Meningitis:** Meningitis occurs in 0.7% to 5% of children with basilar skull fractures (due to CSF leak and exposure to microorganisms); the rate is less than 1% for children with a GCS score higher than 13 and no ICI.
- **Cranial nerve impairment:** This complication occurs in 1% to 23% of cases, with cranial nerves VI, VII, and VIII most commonly injured. The impairment may be transient or permanent.
- **Hearing loss:** Hearing loss occurs in up to half of patients with basilar skull fractures; it can be conductive (from hemotympanum or otic canal disruption) or sensorineural.

Kadish HA, Schunk JE: Pediatric basilar skull fracture: Do children with normal neurologic findings and no intracranial injury require hospitalization? Ann Emerg Med 1995;26:37-41.

12. How are CSF leaks treated?

Most CSF leaks through dural tears resolve spontaneously without complications within 1 week, and are thus managed conservatively. Consider operative intervention for persistent leaks. Given the potential for developing meningitis with CSF leaks, there has been significant controversy regarding the use of prophylactic antibiotics. Current data indicate that routine use of prophylactic antibiotics does not significantly reduce the incidence of meningitis.

13. Define primary and secondary brain injury.

Primary brain injury is the neural damage sustained at the time of trauma. Secondary brain injury is neuronal damage sustained after the initial traumatic event to cells not initially injured, and results from numerous causes including hypoxia, hypoperfusion, and metabolic derangements. Because many causes of secondary brain injury are potentially preventable, the clinician's main goal is to monitor for and attempt to prevent these complications in order to limit further neuronal damage.

14. What is a concussion?

An international multidisciplinary conference defined *concussion* as a complex pathophysiologic process affecting the brain, induced by traumatic biomechanical forces. It typically results in the rapid onset of short-lived impairment of neurologic function that resolves spontaneously and reflects a functional disturbance rather than a structural injury. Concussions are associated with grossly normal neuroimaging, if obtained. Concussions result in a graded set of clinical symptoms that may or may not involve loss of consciousness (LOC).

For the clinician, a simpler, perhaps more useful working definition is a trauma-induced alteration in mental status that may or may not involve LOC. From a practical standpoint, *concussion* often is used to refer to a head injury when the GCS score is 14 to 15, the patient has some symptoms (e.g., headache, dizziness, vomiting, amnesia, or confusion), there is no evidence of a fracture, there are no focal neurologic deficits, and neuroimaging is normal (if obtained).

McCrory P, Meeuwisse WH, Aubry M, et al: Consensus statement on concussion in sport: The 4th International Conference on Concussion in Sport held in Zurich, November 2012. Br J Sports Med 2013;47(5):250-258.

Meehan WP, Bachur RG: Sport-related concussion. Pediatrics 2009;123:114-123.

15. What is the second impact syndrome?

The second impact syndrome, a very rare event, is acute, usually fatal brain swelling that occurs when a second concussion is sustained before complete recovery from a previous concussion. The pathophysiology of the second impact syndrome is not well understood; the initial blow may be associated with alteration in cerebral blood flow and failure of normal autoregulation, making the second impact so much more devastating, with resulting brain swelling and increased ICP.

Evans R: Concussion and mild traumatic brain injury. UpToDate, 2013. Available from www.uptodate.com.

16. What imaging modality is recommended for acute injuries?

CT identifies essentially all significant ICIs requiring intervention.

17. If CT identifies major complications, why not image all children with head injuries?

Although a very useful imaging modality, CT carries disadvantages, most importantly exposure to ionizing radiation. Children are at higher risk of radiation-induced cancer because they have a longer lifetime to manifest cancer and have increased radiation sensitivity of developing tissues. The greatest lifetime risk occurs in the youngest patients, and risk decreases as age increases. The estimated rate of lethal malignancies from CT is between 1 in 1000 and 1 in 5000 pediatric cranial CT scans, with higher risk in younger patients. Therefore, use head CT selectively.

Brenner DJ: Estimating cancer risks from pediatric CT: Going from the qualitative to the quantitative. Pediatr Radiol 2002;32:228-231.

Brenner DJ, Elliston CD, Hall EJ, et al: Estimated risks of radiation-induced fatal cancer from pediatric CT. AJR 2001;176:289-296.

Brenner DJ, Hall EJ: Computed tomography—An increasing source of radiation exposure. N Engl J Med 2007;357:2277-2284.

18. Which children are at high risk for ICI and should undergo CT?

Evidence indicates that children with a GCS score lower than 15, abnormal neurologic examination, or evidence of skull fracture are at high risk for ICI and should undergo CT. Additional criteria for infants include irritability, bulging fontanel, or suspected abuse.

Dunning J, Daly JP, Lomas JP, et al: Derivation of the children's head injury algorithm for the prediction of important clinical events decision rule for head injury in children. Arch Dis Child 2006;91:885.

Kuppermann N, Holmes JF, Dayan PS, et al: Identification of children at very low risk of clinically-important brain injuries after head trauma: A prospective cohort study. Lancet 2009;374:1160.

Osmond MH, Klassen TP, Wells GA, et al: CATCH: A clinical decision rule for the use of computed tomography in children with minor head injury. CMAJ 2010;182:341.

19. Which children are at low risk for significant head injury and may not need a CT scan?

One large study (PECARN) published a decision rule identifying children at low risk for clinically important ICI, for whom imaging is not indicated. For children older than 2 years with no altered mental status, LOC, vomiting, severe headache, signs of basilar skull fracture, or significant mechanism of injury (e.g., fall >5 feet) the likelihood of having a clinically important injury was less than 0.05%. For children younger than 2 years, the low-risk predictors were having normal mental status, no nonfrontal scalp hematoma, no LOC, no palpable skull fracture, no significant mechanism (e.g., fall >3 feet), and acting normally per parents.

Patients in an intermediate risk group have some risk, but no high-risk findings. Not all patients in this group require imaging. Consider imaging if symptoms are more intense, worsening, or of longer duration or if more than one symptom is present. For children with mild symptoms, careful observation in the ED or at home may be an alternative approach, with reevaluation and CT for persistent or worsening symptoms.

Kuppermann N, Holmes JF, Dayan PS, et al: Identification of children at very low risk of clinically-important brain injuries after head trauma: A prospective cohort study. Lancet 2009;374:1160.

20. Are there additional factors to consider for alert children younger than 1 to 2 years old with a nonfocal examination?

The PECARN study identified low-risk predictors for children younger than 2 years, as previously mentioned. Nonfrontal hematomas can be markers for either skull fractures or more significant trauma and are useful for identifying infants and young children with "occult ICIs" (i.e., injuries with no signs of brain injury). Hematomas of greatest concern are those that are larger in size, nonfrontal in location, and in younger children. The youngest infants (particularly those younger than 2-3 months of age) may have no signs or symptoms with ICI; therefore, a low threshold for imaging (unless trivial trauma) is prudent. Although not specifically identified as predictors by the PECARN decision rule, other studies have found infants with irritability, persistent vomiting, or bulging fontanel and those concerning for nonaccidental trauma to be at higher risk for ICI.

Dunning J, Daly JP, Lomas JP, et al: Derivation of the children's head injury algorithm for the prediction of important clinical events decision rule for head injury in children. Arch Dis Child 2006;91:885.

Greenes DS, Schutzman SA: Clinical indicators of intracranial injury in head-injured infants. Pediatrics 1999;104:861-867.

Greenes DS, Schutzman SA: Clinical significance of scalp abnormalities in head-injured infants. Pediatr Emerg Care 2001;17:88-92.

Schutzman SA, Barnes P, Duhaime AC, et al: Evaluation and management of children younger than two years of age with apparently minor head trauma: Proposed guidelines. Pediatrics 2001; 107:983-993.

21. What is cerebral perfusion pressure (CPP)?

CPP is the difference between the mean arterial pressure (MAP) and the ICP (CPP = MAP – ICP). Therefore, significant decreases in MAP or significant increases in ICP can lead to inadequate CPP, with resultant cerebral ischemia and secondary brain injury.

22. How does increased ICP occur?

After infancy (when the cranial sutures fuse), the cranial vault becomes stiff and poorly compliant. The normal intracranial contents include brain, blood, and CSF. Because intracranial volume is fixed, any increase in the volume of one of the components (e.g., cerebral edema, expanding epidural hematoma) must be accompanied by a proportional decrease of the others; otherwise, ICP will increase.

Key Points: Intracranial Injury

1. Most head injuries are minor; most children with minor head injury don't have ICI, but about 5% of those with minor head injury presenting to EDs do, and approximately 1% will have clinically important ICIs and 0.1% to 0.6% will require neurosurgery.
2. The clinician's goal is to identify children with ICI in order to avoid further neuronal injury while limiting unnecessary neuroimaging procedures.

Key Points: Intracranial Injury (*Continued*)

3. Altered mental status, focal neurologic examination, and skull fractures are clear predictors for increased risk of ICI.
4. Other signs and symptoms (including LOC, headache, vomiting) have been variably predictive for ICI.
5. Children older than age 2 with a nonsevere injury mechanism, no LOC, no vomiting, no severe headache, no signs of basilar skull fracture, and normal mental status have an extremely low likelihood of having a *clinically important* ICI.
6. Children younger than age 2 with a nonsevere injury mechanism, no LOC, no scalp hematoma (except frontal), and no palpable skull fracture who have normal mental status (acting normally per parents) have an extremely low likelihood of having a *clinically important* ICI.
7. For all head-injured children who are discharged from the ED, give clear discharge instructions to a responsible adult who is able to return if the child develops concerning signs/symptoms of possible ICI.

23. What is cerebral herniation syndrome?

The cranial cavity is separated by dural folds and bony prominences into the anterior, middle, and posterior fossa. Cerebral herniation occurs when increased ICP causes the brain parenchyma to shift into an anatomic area that it does not normally occupy.

Uncal herniation is the most common form, in which the uncus (inferomedial-most structure of the temporal lobe) slides through the tentorial notch, passing from the middle to the posterior fossa. Initial symptoms include headache and decreased level of consciousness, followed by ipsilateral pupillary dilatation and contralateral hemiplegia. Altered respirations, bradycardia, and systemic hypertension may ensue, with decerebrate posturing or flaccid paresis. If the process continues unchecked, ultimately brainstem failure with respiratory arrest, cardiovascular collapse, and death will occur.

24. Outline the ED treatment for increased ICP.

- Manage the ABCs (airway, breathing, and circulation). This is essential to avoid hypoxia and hypercarbia, and to maintain adequate CPP.
- Avoid secondary brain injury from other metabolic causes, including hypoglycemia, hyperthermia, and seizures.
- Ensure appropriate positioning, with elevation of the head of the bed 30 degrees and the neck midline. This promotes venous drainage and can lead to a significant decrease of ICP.
- Obtain emergent head CT to identify mass lesions that require surgical evacuation.
- Consider osmotic agents: Mannitol and 3% saline can be used to lower the ICP.
- Use sedation: Conscious patients who are paralyzed for intubation require sedation.

25. Isn't hyperventilation used to treat elevated ICP?

Although hyperventilation had been a mainstay of emergency treatment for increased ICP, its use has become controversial. Carbon dioxide is one of the main determinants of cerebrovascular tone, with low levels producing vasoconstriction and decreased blood flow, thus lowering ICP. However, if cerebral blood flow diminishes too much, substrate delivery of oxygen and glucose is impaired and CPP may be inadequate, resulting in ischemic injury. Consider mild hyperventilation (maintaining P_{CO_2} of 30-35 mm Hg) for periods of intracranial hypertension.

Mazzola CA, Adelson PD: Critical care management of head trauma in children. Crit Care Med 2002;30 (11 Suppl):S393-S401.

26. Are steroids indicated for treating increased ICP?

Steroids have not been shown to improve outcome for patients with head trauma; therefore, their use is not recommended.

27. Which children need to be admitted following acute head trauma?

Children with ICIs or depressed or basilar skull fractures usually require hospital admission. Consult a neurosurgeon regarding their management and disposition. Consider hospital admission for patients with persistent neurologic deficits (despite normal CT scan), significant

extracranial injuries, or unremitting vomiting, or if the caretakers are unreliable or unable to return when necessary. Strongly consider hospital admission for any child with suspected nonaccidental trauma.

28. Why are discharge instructions important in patients with head trauma?

Even well-appearing children with head trauma who have no evidence of complications (either clinically or radiographically) have a small chance of subsequent deterioration. Therefore, it is mandatory to have competent caretakers observe the child at home. Educate them about the signs and symptoms of complications of head trauma, and instruct them to bring the child to medical care for reevaluation should concerning symptoms arise.

29. When can a child return to sports after a concussion?

The majority (80-90%) of concussions resolve in a short period (7-10 days), although the recovery time frame may be longer in children and adolescents. Children with concussion should have physical and cognitive rest until symptoms resolve and then follow a graded program of exertion prior to medical clearance and return to play. The current evidence evaluating the effect of rest and treatment following a sports-related concussion is sparse. An initial period of rest may be of benefit. However, further research to evaluate the long-term outcome of rest, and the optimal amount and type of rest, is needed.

McCrory P, Meeuwisse WH, Aubry M, et al: Consensus statement on concussion in sport: The 4th International Conference on Concussion in Sport held in Zurich, November 2012. Br J Sports Med 2013;47(5):250-258.

MINOR TRAUMA

Sabina B. Singh and Magdy W. Attia

CHAPTER 57

1. What are the major considerations in wound assessment?

- **Local factors** include location, mechanism of injury, wound age, possibility of foreign body, and degree of contamination. Soil contamination with organic matter has the highest rate of wound infection if not properly cleansed. Consider the possibility of a retained foreign body in wounds caused by broken glass or other debris. Obtain imaging studies if a foreign body is suspected.
- **Host factors** include disease states (diabetes, immunocompromised state, etc.), tetanus immunization, allergies, sedation, and pain control.

2. How does location of injury affect assessment?

- **Injuries over a joint or adjacent to tendons** should be checked for crepitus, which may signify disruption of the joint capsule. Loss of function may indicate tendon injury.
- **Lacerations close to the neurovascular bundle** may cause nerve damage. Assess capillary refill and pulses and conduct a careful neurologic examination of motor and sensory functions.
- Wounds in proximity to **areas of high bacterial concentration**, such as the perineum, axilla (particularly in adolescents), and exposed parts (hands, feet) are at a higher risk for infection.

3. Why are bites and crush injuries of special concern?

Bites and crush injuries are more difficult to repair. They are often associated with devitalized tissue and are more likely to become infected. Manage an injury to the skin over a metacarpophalangeal joint from a fist punch to the mouth as if it were a human bite.

4. What is the ideal time frame for repair of wounds?

Ideally wounds should be repaired within 6 hours of injury. Clean wounds can be closed within 12 to 24 hours after injury. Consider facial and scalp wounds for primary closure for best results. Delayed closure is usually carried out 3 to 5 days after the original injury for wounds with high potential for infection (heavily contaminated wounds, bite wounds, puncture wounds) and wounds in areas of low cosmetic concern in immunocompromised patients.

5. Which wounds are allowed to heal by secondary intention?

Allow infected wounds and small puncture wounds in areas of low cosmetic concern to close by secondary intention. A small wick of iodoform gauze may be placed in the wound to decrease chances of infection.

6. How should the emergency physician approach sedation and pain control?

Discuss options, risks, and benefits with parents and older children. Parents often can predict the level of cooperation expected from their child. Psychosocial intervention, local anesthetics, and midazolam (intranasal or oral) reduce the pain associated with procedures.

7. When should consultation be obtained for wound care?

Consider consultation with a surgical or orthopedic specialist for the following types of wounds:

- Fracture or violation of a joint cavity
- Injury to a tendon, nerve, or large vessel
- Wounds requiring complex repair located in areas of high cosmetic concern

Selbst SM, Attia M: Minor trauma. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1256-1270.

8. When are radiographic studies indicated as part of wound management?

Obtain radiographs for wounds associated with fractures or joint disruption or if a foreign body is suspected. Plain radiographs identify fragments of metal and glass if they are larger than 0.5 cm. Wood is identified in only 15% of cases. Ultrasonography has 90% sensitivity and specificity for nonopaque foreign bodies if the location is not near a bony or gaseous structure. Consider computed tomography or magnetic resonance imaging (MRI) if a surgical approach is planned for the removal of a foreign body near vital structures.

9. How can you decrease the chances of missing a retained foreign body in a wound?

A detailed history of the mechanism and circumstances surrounding the injury is important. Good exploration with visualization of the base of the wound significantly reduces the risk. In one study, wounds less than 0.5 cm deep had a low risk of an embedded foreign body. Glass fragments can penetrate deeply and have the potential of not being visualized. Persistent or extreme pain, pain on passive movement, and a mass with discoloration at the site of a wound are all highly suggestive of a retained foreign body.

10. Should a retained foreign body be removed?

In general, foreign bodies should be removed; however, limit removal attempts to 30 minutes. The following factors need to be considered prior to starting removal:

- **Location:** Intra-articular, intravascular, or in close proximity to vital structures or has the potential to migrate toward vital structures (e.g., lung, spleen). Consult a specialist in orthopedics, plastic surgery, etc.
- **Material:** Risk of toxicity (e.g., lead, venom from spines)
- **Tissue response:** Production of inflammatory response (e.g., organic matter, silica that causes large granuloma formation), persistent pain, infection, or cosmetic disfigurement

Explore foreign bodies associated with penetrating injuries to the abdomen, neck, or chest and arrange for removal in the operating room. Small, deeply embedded foreign bodies may be difficult to locate without ultrasound guidance. They can be left in place with close follow-up or removed electively.

11. Should hair be removed from a wound site before repair?

The presence of hair usually does not interfere with repair of the wound. Petroleum jelly can be used to keep hair away from the wound site during repair. If hair removal is needed, use clippers because they have a lower rate of infection than do razors. *Do not remove eyebrows during wound repair, because regrowth can be slow or absent.*

12. Outline the steps in preparing a child for wound closure.

- Follow standard precautions.
- Prepare the child and the family. An honest and caring explanation of the various steps is important to reduce anxiety. Use distraction techniques to decrease anxiety.
- Determine whether sedation is required.
- Cleanse and irrigate the wound.
- Use local anesthesia.
- Use sterile draping and technique.

Gursky B, Kestler LP, Lewis M: Psychosocial intervention on procedure related distress. J Dev Behav Pediatr 2010;31:217-222.

13. How is irrigation and cleansing of the wound best achieved?

Irrigation is best achieved with normal saline or tap water under pressure using a 20- or 30-mL syringe on a 19- or 22-gauge plastic intravascular catheter and 50 to 100 mL of fluid for irrigation per centimeter of laceration. Although studies have shown no difference in outcome between irrigation with sterile water and tap water, sterile water is used most commonly for irrigation. Splash shields decrease splattering of irrigation fluid. Avoid excessive pressure during irrigation, because it can distort the anatomy and damage the tissues. Remove road rash (tar), dirt, and foreign material gently with irrigation and mild scrubbing to prevent tattooing. Limit scrubbing because it can damage tissues, leading to poor cosmetic outcome. Several studies have shown that use of povidone-iodine affects fibroblasts and hence wound defenses. Irrigation is best done after analgesia is obtained.

Gravett A, Stener S, Clinton JE, et al: A trial of povidone-iodine in the prevention of infection in sutured lacerations. Ann Emerg Med 1987;16:167.

Valente JH, Forti RJ, Freundlich LF, et al: Wound irrigation in children: Saline solution or tap water? Ann Emerg Med 2003;41:609-616.

14. What agents are used for local anesthesia?

Topical anesthetics such as LET (**l**idocaine 4%, **e**pinephrine 1:1000, **t**etracaine 0.5%) are very useful. LET is applied to the wound locally in the form of a gel for 15 to 20 minutes and produces anesthesia with 97% efficacy. Avoid topical agents in areas of end-blood supply, such as the fingers, ears, nose, toes, and penis (do not use epinephrine for end-arterial structures). Occasionally, deeper wounds require infiltration with another agent, such as lidocaine 1% with or without epinephrine. Bupivacaine, which has a longer duration of action, is an alternative in lidocaine-allergic patients.

15. What is the maximum dose of lidocaine in local anesthetic medications?

The dose of lidocaine with epinephrine should not exceed 7 mg/kg body weight; the dose of lidocaine alone should not exceed 4 mg/kg.

Ernst AA, Gershoff L, Miller P, et al: Warmed versus room temperature saline for laceration irrigation—A randomized clinical trial. South Med J 2003;96:436-439.

Key Points: Tips to Reduce Pain of Anesthetic Infiltration

1. Prior application of LET gel (time permitting)
2. Rubbing the skin near the site of injection (stimulates other nerve endings and thereby decreases the perception of pain)
3. Buffering lidocaine with 8.4% sodium bicarbonate (ratio, 9:1)
4. Warming the buffered lidocaine to 40° C
5. Using a 27- or 30-gauge needle to slow the rate of injection
6. Injecting from inside the wound through devitalized tissue

16. What is the best method of obtaining hemostasis during wound repair?

A tourniquet, such as a rubber band or blood pressure cuff, and a local anesthetic with epinephrine can decrease the amount of bleeding during wound repair. Inflate the blood pressure cuff to just above systolic blood pressure. Use a tourniquet for only 20 to 30 minutes at a time. Release it periodically for 2 to 3 minutes to allow reperfusion. Prolonged continuous use can lead to nerve and vascular damage, with subsequent thrombosis and gangrene.

17. What are the key points for proper wound approximation?

- Place the first suture at the midpoint.
- Slight eversion of the wound edges prevents tethering of the scar line.
- Remove clot from the wound to help prevent infection and allow for better approximation.
- Minimize tension on the scar line by doing a layered closure if needed.

18. What are the appropriate types of sutures and techniques for closure of various wounds?

For skin closure, use fast-absorbing gut or synthetic nonabsorbable monofilament nylon (Ethilon or Dermalon). Fine absorbable synthetic sutures (e.g., coated Vicryl or Dexon) are used for deeper layers or nail bed repairs. Suture size depends on the site and size of the wound. Use smaller needles (e.g., PS or P1) for wounds that require fine cosmetic outcome. Table 57-1 lists suggested suture types for various locations.

Key Points: Achieving Ideal Wound Approximation

1. Plan ahead.
2. Consider placing the first suture at the midpoint for good alignment.
3. Ensure slight eversion of the wound edges.
4. Remove clots from gaping wounds.
5. Keep wound tension to a minimum.
6. In layered closures, close each layer individually.

19. How do alternative wound care techniques compare?

See Table 57-2 for a comparison of alternative wound care techniques.

Table 57-1. Suggested Suture Types for Various Locations

WOUND SITE	SUTURE TYPE	RECOMMENDED TECHNIQUE	REMOVAL (DAYS)
Scalp	4-0 or 5-0 SNA	Simple interrupted	7
Face	6-0 FA or SNA for skin 5-0 AS for deeper layers	Simple interrupted or continuous for skin; interrupted for deeper layers	3-5
Extremity	3-0 or 4-0 SNA 4-0 or 5-0 AS	Simple interrupted mattress or continuous for skin; interrupted for deeper layers	7-10
Hands or feet	5-0 SNA 4-0 AS	Simple interrupted or continuous for skin; interrupted for deeper layers	7-10
Joints	3-0 or 4-0 SNA 4-0 AS	Simple interrupted mattress or continuous for skin; interrupted for deeper layers	10-14

AS, absorbable synthetic; FA, fast absorbing gut; SNA, synthetic nonabsorbable.

Table 57-2. Comparison of Alternative Wound Care Techniques

TYPE OF CLOSURE	WOUND TYPE	EASE OF APPLICATION	COSMETIC RESULT	PAIN	COST	NEED FOR REMOVAL
Suture	Any	Poor	Excellent	High	High	Yes
Staple	Scalp*	Good	Poor	High	Medium	Yes†
Surgical glue	Small	Good	Good	None	Medium	None
Steri-Strips	Small‡	Good	Fair	None	Low	Yes

*Should be avoided in areas of cosmetic concern or in patients needing computed tomography or magnetic resonance imaging.
†Requires special removal forceps.
‡Small, straight lacerations or located over joints, hair-bearing areas, or moist areas.
Data from Farion K, Osmond MH, Hartlng L, et al: Tissue adhesives for traumatic lacerations in children and adults. Cochrane Database Syst Rev 2002;(3):CD003326; Karounis H, Gouin S, Eisman H, et al: A randomized, controlled trial comparing long-term cosmetic outcomes of traumatic pediatric lacerations repaired with absorbable plain gut versus nonabsorbable nylon sutures. Acad Emerg Med 2004;11:730-735.

20. When can tissue adhesives be used?

Tissue adhesives are nearly painless, do not require local anesthesia or removal, and have cosmetic results similar to those of sutures. Use tissue adhesives for wounds with straight edges that are less than 5 to 6 cm long and no wider than 2 to 4 mm. During application, ensure good approximation of the wound, and take care to prevent the adhesive from dripping into the wound, eyes, or eyelashes. The presence of adhesive in the wound acts like a foreign body and can produce intense inflammation. Instruct parents to avoid the use of ointments or cream at the site of the wound because they lead to premature peeling of the adhesive. Tissue adhesive (cyanoacrylate) polymerizes on the surface of the approximate wound edges, forming a strong bond to maintain the achieved approximation. Routine follow-up is not necessary, and no removal is needed because the glue will slough off after 7 to 10 days.

Zempsky WT, Parrotti D, Grem C, Nichols J: Randomized controlled comparison of cosmetic outcomes of simple facial lacerations closed with Steri-Strip skin closure or Dermabond tissue adhesive. Pediatr Emerg Care 2004;20(8):519-524.

21. When should tissue adhesives be avoided?

Do not use tissue adhesives alone for deep wounds. Also, do not use tissue adhesives for wounds on the hands (the glue is likely to wear off) or for those subject to great tension (located over a joint). It may be best to avoid use of tissue adhesives in hair-bearing areas.

22. What are the complications of tissue adhesives?

Occasionally, skinfolds are inadvertently glued together, the glove of the operator may become glued to the patient's skin, or adhesive drips into the patient's eyelashes. If these complications occur, massage petroleum jelly or eye ointment into the site to help dissolve the glue. Refrain from clipping the eyelashes, because their regrowth is uncertain. The strength of the wound after repair with an adhesive is 10% to 15% less than that of wounds closed by sutures. Dehiscence occurs in 1% to 5% of all wounds closed by adhesives. If the wound dehisces, leave it to heal by secondary intention unless the result will be cosmetically unacceptable.

23. What are some important considerations in the evaluation of puncture wounds?

Puncture wounds account for 3% to 5% of all injuries. The most common site is the forefoot (50%). The rate of complications is directly proportional to the depth of penetration. Infection occurs in 6% to 10% of all puncture wounds, with staphylococci, streptococci, and anaerobes being the most common organisms. Injuries penetrating through a foam or rubber insole of a sneaker are often contaminated by *Pseudomonas aeruginosa*. The most common offending object in puncture wounds is a nail (>90%). It is essential to examine the wound well for the possibility of damage to deeper structures and for retained foreign bodies. Puncture wounds have a greater incidence of infectious complications, such as cellulitis, soft tissue abscess, pyarthrosis, osteomyelitis (0.4-0.6% of wounds), and foreign body granuloma.

Racz RS, Ramanujan Cl, Zgonis T: Puncture wound of the foot. 2010;27(4):523-534.

24. A 10-year-old boy presents with persistent pain in his right forefoot after sustaining a puncture wound from a nail 10 days ago. Some redness and swelling are seen at the site. What are your major concerns and management course?

Osteomyelitis is a major concern with a puncture wound when persistent pain is accompanied by local signs of infection, even without systemic involvement. Obtain a complete blood count and erythrocyte sedimentation rate, along with imaging studies. Bone scanning or MRI is more sensitive in detecting a periosteal reaction or bony destruction than radiography in the first week. Treat osteomyelitis with intravenous (IV) antistaphylococcal and possibly antipseudomonal antibiotics. If no improvement is noted in 48 hours, surgical débridement may be necessary.

25. What is coring of a puncture wound?

Coring removes a 2-mm circular rim of the puncture track. Prepare the area with povidone-iodine, local anesthetic, or regional nerve block, followed by irrigation and packing with iodoform gauze. Instruct the patient to avoid weight-bearing for 5 days. Consider coring in wounds that are grossly contaminated or have a foreign body in an area of low cosmetic concern and high risk for infection, such as the foot.

26. How are forehead lacerations generally repaired?

Superficial transverse lacerations of the forehead are easy to repair. They can be repaired using tissue adhesive if superficial with minimal tension or with interrupted or continuous cuticular sutures using 6-0 absorbable suture materials (fast absorbing gut), which has the same cosmetic result as nonabsorbable sutures. The absorbable suture material has the added advantage of not requiring suture removal. Repair deeper transverse lacerations involving the deep fascia, frontalis muscle, or periosteum in layers. If the deeper tissue planes are not closed, the function of the frontalis muscle (eyebrow elevation) may be compromised. Vertical forehead lacerations tend to have wider scars because they traverse the tension lines if proper tension and coaptation are not achieved. Forehead lacerations are rarely associated with skull fractures, but it is advised to rule out facial, neck, and intracranial injuries.

Luck R, Treadway T, Flood R, et al: Comparison of cosmetic outcomes of absorbable versus non-absorbable sutures in pediatric facial lacerations. Pediatr Emerg Care 2013;29(6):691-695.

27. Describe the appropriate technique for repair of eyebrow lacerations.

Do not shave the eyebrow for wound preparation, because it serves as a landmark during repair. In addition, eyebrow regrowth is unpredictable; it may be slow or incomplete, leading to poor cosmetic outcome. Avoid inverting the hair-bearing edges into the wound. It is also important to properly align both ends along the eyebrow wound.

28. How are lacerations of the eyelid repaired?

Lacerations of the eyelid are mostly simple transverse wounds of the upper eyelid just inferior to the eyebrow. Repair does not require special skills. Consider ophthalmology referral for complicated lacerations potentially involving the levator palpebrae muscle or medial canthal ligament or those close to the lacrimal duct. Evaluation for associated injuries to the globe is imperative.

29. How are lacerations of the external ear treated?

To avoid necrosis and auricular deformity, minimize débridement and cover the perichondrium with the lacerated thin but vascular skin. Simple closure with the least possible tension is usually advised. Include the perichondrium in the sutures to ensure that the suture material does not tear through the friable cartilage and to restore nutrient and oxygen supply. If ear trauma has led to auricular hematoma, drain this promptly to avoid necrosis of the cartilage, which leads to a deformed auricle or cauliflower ear. After repair of ear lacerations or evacuation of an auricular hematoma, apply a pressure dressing. Follow-up in 24 hours is recommended to evaluate vascular integrity.

30. What is the recommended technique for repair of lip lacerations?

Proper repair of lip lacerations is important because they are highly visible. The vermilion border is a relatively pale line that identifies the junction of the dry oral mucosa and facial skin. It is an important landmark for proper repair. Avoid the use of epinephrine with local anesthesia, because it causes swelling that distorts the border. A mental nerve block provides good anesthesia while preserving the landmarks. Lacerations involving the vermilion border must be aligned precisely. Warn parents that although the lip is still anesthetized, the child might bite off the sutures. They should distract the child from doing so. After local anesthesia has worn off, the site typically is sore enough that the child does not attempt to manipulate the area.

31. Describe the proper approach to lacerations of the tongue and buccal mucosa.

- Small isolated lacerations of the buccal mucosa, usually due to teeth impaction after falls, require no suturing. Lacerations greater than 2 to 3 cm in length or with flaps are best closed with simple interrupted stitches using absorbable material.
- Tongue lacerations often bleed excessively at the time of injury, but the bleeding usually ceases quickly as the lingual muscle contracts. Most tongue lacerations can be left alone with good results. Repair large lacerations involving the free edge, large flaps, and bleeding lacerations. Full-thickness repair with interrupted 4-0 absorbable sutures is recommended.
- Local or regional anesthesia is often sufficient.
- Pay attention to potential airway problems during repair.
- Hold the mouth open with a padded tongue depressor. The tongue can be maintained in the protruded position by a gentle pull by using a towel clip or by placing a suture through the tip.
- As in lip lacerations, children may chew off the stitches; warn parents of this possibility. They should attempt to distract the child, at least until the local anesthesia has worn off.
- A recent case report describes successful repair of a tongue laceration using a tissue adhesive.

Kazzi MG, Silverberg M: Pediatric tongue laceration repair using 2-octyl cyanoacrylate (Dermabond). J Emerg Med 2013;45(6):846-848.

32. How are fingertip avulsions repaired?

Most fingertip avulsions are contused lacerations or partial avulsions. Sharp injuries are more common in older children and less likely to be associated with fractures. Evaluate for associated nail bed injury and obtain radiographs for possible fracture of the phalanx. In general, this type of injury is managed conservatively, as tissue regeneration is excellent in preadolescent children. For complete amputation reattachment, skin flap and grafting are options used by the hand specialist. Give antibiotic coverage if the bone is exposed or fractured in the fingertip injury.

33. Describe the approach to nail bed injuries.

Trauma to the distal fingers is often associated with nail and nail bed (matrix) injuries. Nail avulsion may be partial or complete, and may be associated with nail bed laceration and/or underlying fracture of the distal phalanx. Injury to the fingertip often is associated with subungual hematoma. Unrepaired nail bed lacerations may permanently disfigure new nail growth from the cicatrix nail bed. If the nail is partially avulsed but is firmly attached to its bed, exploring the nail bed is not warranted. Good outcome is expected, because the nail holds the underlying lacerated nail bed tissues in place.

When the nail is completely avulsed or attached loosely, lift the nail and assess the nail bed for a laceration. There is a recent case report of successful use of ultrasound to identify nail bed injury. If the nail bed is lacerated, repair it by using 6-0 or smaller absorbable material. After its soft proximal portion is cleansed and trimmed, replace the nail between the nail bed and nail fold (eponychium) and then anchor it in place with a few stitches. This technique splints the nail fold away from the nail bed and prevents obliteration of the space between the two. Preserving this space allows the new nail to grow undisturbed. The preferred method of local anesthesia for nail bed repair is digital block or sedation for a young child. The use of a finger tourniquet during repair allows a bloodless field. Apply a finger splint after repair, especially in patients with an associated fracture.

Soyuncu S: Nail bed injury detected by ultrasound. Am J Emerg Med 2012;30(7):1323.e5-1323.e6.

34. How should a subungual hematoma be managed?

A subungual hematoma is a collection of blood between the nail plate and nail bed. It is commonly seen with blunt and crush fingertip injuries and presents with throbbing pain and discoloration of the nail. It may be associated with nail bed injury or fracture of the distal phalanx. Drain a subungual hematoma involving 50% or more of the nail. Drainage of the hematoma relieves pain and prevents deformity of the nail bed. Local anesthesia is not required for a simple trephination by cauterization of the nail (Fig. 57-1). Postdrainage care includes elevation of the hand and warm soaks for a few days. Discuss the possibility of nail deformity in the future with the family.

When the injury is more involved, digital block is advised. If the hematoma is large and extends to the tip of the nail, separation of the nail from the nail bed allows drainage. In the presence of a distal phalangeal fracture, be careful not to transform a closed fracture into an open fracture by communicating a subungual hematoma to the exterior surface of the nail. If this possibility exists, antibiotic coverage and close follow-up are appropriate.

Dean B, Becker G, Little C: Management of acute traumatic subungual hematoma: A systematic review. Hand Surg 2012;17(1):151-154.

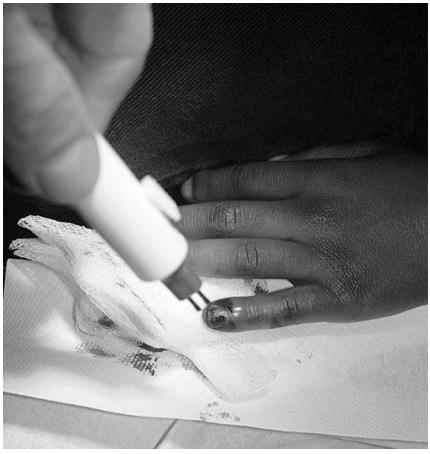


Figure 57-1. Trephination of a subungual hematoma by cauterization.

35. What are the signs of wound infection?

Signs of wound infection include marked or worsening pain or tenderness at the wound site beyond that expected from the initial trauma. Local erythema, warmth, swelling, and discharge, such as pus or serosanguineous fluid, also are signs of infection. In severe infections, fever, phlebitis (streaking), regional lymphadenopathy, and other systemic manifestations may be seen.

36. For which wounds should prophylactic antibiotics be considered?

- Human bites
- Cat bites
- Crush injuries
- Extensive wounds
- Exposed cartilage
- Open fractures
- Joint cavity violated
- Hand and foot wounds
- Moist areas (axilla, perineum)
- Contaminated wounds
- Immunocompromised host

Use a first-generation cephalosporin to cover staphylococci and streptococci or use clindamycin or trimethoprim-sulfamethoxazole if methicillin-resistant *Staphylococcus aureus* is a concern. Use amoxicillin–clavulanic acid for bites to provide additional coverage against anaerobes. Clindamycin is a suitable alternative.

37. What are the key elements of postrepair wound care?

- Apply antibiotic cream to reduce infection by preventing scab formation.
- Elevate the affected area to decrease edema.
- Immobilize wounds over joints with a splint or apply a bulky dressing.
- Provide discharge instructions regarding wound care, signs of infection, and follow-up.
- Instruct the patient to avoid bathing for 24 to 48 hours; after that time, the wound should be washed with mild soap and water and gently dried.
- Recheck patient in 24 to 48 hours for healing and signs of infection.
- Remove sutures in a timely manner to prevent scars from suture tracks.
- Instruct the patient to use sunscreen for 6 to 12 months after injury to prevent hyperpigmentation of the scar.

38. When does a wound regain its strength?

A wound regains 5% of its strength in 2 weeks, 30% in 1 to 2 months, and full strength by 6 to 8 months. The scar achieves its final appearance in 6 to 12 months.

39. What are the indications for tetanus prophylaxis?

Tetanus is a potential risk in all wounds. There were an average of 29 cases of tetanus per year from 2001 to 2008 in the United States, of which 13% were fatal. Wounds at a higher risk for tetanus are those contaminated by soil or feces and those with devitalized tissue (e.g., puncture, crush, missile, or avulsion wounds). Burns and frostbite are also prone to tetanus. Prophylaxis depends on the type of wound and the patient's immunization status (Table 57-3).

Centers for Disease Control and Prevention: Vaccines and Immunizations. Chapter 16: Tetanus. Manual for the surveillance of vaccine-preventable diseases. Available at http://www.cdc.gov/vaccines/pubs/surv-manual/chpt16-tetanus.html.

40. A young boy is brought to the ED after being stuck by a needle on the playground. The needle has crusted blood along the metallic edge. What are your concerns? What is the best course of management?

A needlestick injury raises concerns about exposure to tetanus and blood-borne pathogens, such as hepatitis B virus (HBV), hepatitis C virus (HCV), and human immunodeficiency virus (HIV). Give tetanus prophylaxis after considering the potential for contamination and the immunization status of the child. HBV can survive on fomites for several days, and prompt postexposure prophylaxis reduces the risk of transmission. Table 57-4 summarizes the recommendations for hepatitis prophylaxis. The risk of infection with HIV causes great

Table 57-3. Guidelines for Tetanus Prophylaxis

		Clean Wound		*All Other Wounds*	
DOSE OF TETANUS TOXOID	**TIME SINCE LAST DOSE**	DTaP/ Tdap/Td*	TIG	DTaP/ Tdap/Td	TIG
>3	<5 y	No	No	No	No
	5-10 y	No	No	Yes	No
	>10 y	Yes	No	Yes	No
<3 or unknown		Yes	No	Yes	Yes

DTaP, diphtheria, tetanus, acellular pertussis toxoid for children younger than 7 years of age; Td, tetanus toxoid, reduced diphtheria; Tdap, tetanus toxoid, reduced diphtheria toxoid, acellular pertussis vaccine; TIG, tetanus immunoglobulin.
*Tdap is preferred to Td for adolescents who have never received Tdap.

Table 57-4. Hepatitis Prophylaxis After a Needlestick Exposure

NUMBER OF DOSES OF HBV VACCINE ALREADY RECEIVED	IMMUNOPROPHYLAXIS
>3	None
1-3	Additional dose of HBV vaccine; complete the rest of the schedule with or without HBIG
None	Begin vaccination series + HBIG

HBIG, hepatitis B immunoglobulin; HBV, hepatitis B.

anxiety to the family. However, the risk of transmission of HIV from a discarded needle is less than 0.3%. Testing the syringe is neither practical nor reliable. Testing the patient for HIV is controversial, but if testing is elected, it should be done at the time of injury and at 6 weeks, 12 weeks, and 6 months after exposure. Consult a specialist in HIV before using prophylaxis. Start antiretroviral therapy if the syringe had fresh blood.

The risk of transmission of HCV from a discarded needle is low. Testing is done if there is known exposure or high risk of HCV. Assess for anti-HCV antibodies by enzyme immunoassay at the time of injury and 3 to 6 months after exposure. There is no prophylaxis for HCV.

American Academy of Pediatrics: Red Book Online. Available from http://aapredbook.aappublications.org.

41. When is rabies prophylaxis indicated?

The rabies virus is shed in the saliva of infected animals for 10 to 14 days before they become symptomatic. Postexposure guidelines are summarized in Table 57-5. Bites from animals such as squirrels, hamsters, guinea pigs, gerbils, chipmunks, rats, mice, rabbits, hares, and other rodents rarely require rabies prophylaxis. There is no wildlife rabies in the state of Hawaii.

American Academy of Pediatrics: Red Book Online. Available from http://aapredbook.aappublications.org.

42. What is the regimen for postexposure rabies prophylaxis?

Active immunization with rabies human diploid cell vaccine (HDCV) is given as soon as possible after the exposure, starting on the day of injury and on days 3, 7, and 14. Immunocompromised patients should receive a fifth dose on day 28. Administer the vaccines as a 1.0-mL intramuscular injection in the deltoid. In small infants, use the gluteal area. Give rabies immunoglobulin (RIG) along with the first dose of rabies vaccine, but at a different site. The dose of RIG is 20 IU/kg body weight. Infiltrate as much as possible of the total dose of RIG at the wound site and inject the remainder intramuscularly. Give RIG as soon as possible, and within 7 days after exposure. For patients previously immunized with rabies vaccine give only two doses of HDCV, on days 0 and 3.

Table 57-5. Guidelines for Rabies Prophylaxis

TYPE OF ANIMAL	AVAILABILITY OF ANIMAL FOR OBSERVATION	POSTEXPOSURE PROPHYLAXIS
Dog or cat	Healthy or can be observed for 10 days	Only if animal develops signs of rabies
	Suspected to be rabid or unknown	RIG + HDCV
Livestock, ferrets, rodents	Consider individually	As per advice of public health official
Skunks, raccoons, bats, foxes, woodchucks, other carnivores	Consider rabid unless geographic area is known to be free of rabies	RIG + HDCV

HDCV, human diploid cell vaccine; RIG, rabies immunoglobulin.

43. What is the best course of management for a fishhook embedded in soft tissue?

Fishhooks have a straight shank and a curved belly that has an eyelet with a barb pointed away from the tip. Do not attempt removal in the ED if the fishhook is buried near vital structures. If the fishhook is not in a dangerous location, consider using a digital block or local infiltration. Wear protective eyewear to avoid injury during the removal. Remove lures and additional hooks first with a hemostat and wire cutter.

There are several methods of removal. The push-through method is most effective when the barb is close to the skin. Push the barb forward (antegrade) through the skin. Then clip it with a wire cutter and pull the rest of the barb back through the original wound.

With the string technique, loop a long piece of string around the hook at the point of entry and around the clinician's finger. While the clinician applies pressure downward over the straight part of the fishhook to disengage the barb, pull the hook away rapidly. Local anesthetics are rarely needed with this technique.

44. What is the best way to remove a ring that is stuck on a child's finger?

If vascular compromise is present, remove the constricting ring as soon as possible. Risk of gangrene is present in any obstruction that persists beyond 10 to 12 hours. A ring cutter is useful. It is easiest to cut the thinnest part of the ring, which is usually on the palmar surface of the hand. Once cut, separate the ends manually. The string-pull method is best used for broad or metallic bands. One end of a suture is slipped under the band. After application of lubricant, grasp the string with a hemostat and pull in a circular motion until it slides off.

45. Is ultrasound helpful for removal of splinters and other foreign bodies?

Yes. Ultrasound can help identify a subcutaneous foreign body, especially when it cannot be detected by physical examination or radiograph. Ultrasound can pinpoint the location of the foreign body by imaging in multiple planes. Ultrasound can also aid in foreign body removal, but this is technically challenging. Moderate experience with ultrasound and dexterity is needed. Alternatively, find the foreign body with ultrasound, mark its location, and remove it in the standard fashion.

Levy J: Ultrasound. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1729-1743.

MULTIPLE TRAUMA

Laurie H. Johnson and Richard M. Ruddy

CHAPTER 58

1. What is the importance of trauma to the health of children?

Injury is the leading cause of death and disability in children older than 1 year. Although injury death rates in the U.S. population have continued to decline since 2002, trauma is still the number one killer of children. Trauma is responsible for about 10,000 deaths per year in children age 19 years and younger. Most injury is responsible for intermediate morbidity, with significant differences observed for specific mechanisms of injury based on age, injury severity, and mortality risk. Hospital admissions in the 0- to 14-year-old age group for trauma are estimated to exceed 250,000 per year (>51 per 100,000 population). The sequelae from these injuries can often have a substantial impact on the mental and physical functioning of children as they mature; the number of permanently disabled may approach over 100,000 per year.

Centers for Disease Control and Prevention (CDC): National trends in injury hospitalizations: 1971-2001. Available at http://www.cdc.gov/nchs/data/injury/InjuryChartbook79-01.pdf. Accessed on July 9, 2013.

Centers for Disease Control and Prevention (CDC): Vital signs: Unintentional injury deaths among persons aged 0-19 years—United States, 2000-2009. MMWR Morb Mortal Wkly Rep 2012;61:270-276.

Peclet MH, Newman KD, Eichelberger MR, et al: Patterns of injury in children. J Pediatr Surg 1990;25 (1):85-91.

2. What specific mechanisms of injury are seen typically in children?

- Motor vehicle traffic–related crashes are the leading cause of death from injuries in children, both as passengers and as pedestrians struck.
- Drowning is the second leading cause of pediatric injury death nationally.
- Deaths from burns and smoke inhalation have declined but remain fourth.
- Mortality rates are highest for gunshot wounds, especially in teens, but also in the young.
- Falls are the most common mechanism; severity of injury is minor except in falls greater than 10 to 20 feet.

Centers for Disease Control and Prevention (CDC), National Center for Injury Prevention and Control: Web-based Injury Statistics Query and Reporting System (WISQARS). Available at http://www.cdc.gov/Injury/wisqars/pdf/National_Estim_10_Leading_Causes_Nonfatal_Injuries_Tx_Hospital-ED_US2010-a.pdf. Accessed on July 9, 2013.

Key Points: The Challenge of Pediatric Trauma

1. "Multiple trauma" is injury to two or more body areas.
2. Patients with severe head injuries are at high risk of poor outcome or death.
3. Lack of cooperation with examination due to age or fear, initially occult injuries, altered mental status due to alcohol or illicit substances, and nonaccidental trauma may interfere with rapid determination of isolated versus multiple trauma.

3. What injury patterns are expected with specific mechanisms?

In addition to the details of the mechanism (speed of a vehicle, height of a fall, protective equipment worn), the age and size of the pediatric patient are factors in predictable patterns of injury (Table 58-1).

4. Discuss the importance of sports-related injuries.

Sports-related injuries are common but do not often lead to death. Neck injury due to falls in equestrian sports or football spearing is an important cause of severe injury and death despite a fairly low incidence. Blunt trauma from sports can lead to serious head, intra-abdominal solid organ, and eye injury. In terms of visits for emergency care, sports injuries are responsible for large numbers of musculoskeletal injuries, fractures, and joint injuries.

Table 58-1. Common Mechanisms and Associated Patterns of Injury in Pediatric Trauma

MECHANISM	COMMON INJURY PATTERNS
Pedestrian struck	Low speed: Lower extremity fractures High speed: Multiple trauma, head and neck injuries, lower extremity fractures
Automobile occupant	Unrestrained: Multiple trauma, head and neck injuries, scalp and facial lacerations Restrained: Chest and abdomen injuries, lower spine fractures
Fall from a height	Low height: Upper extremity fractures Medium height: Head and neck injuries, upper and lower extremity fractures High height: Multiple trauma, head and neck injuries, upper and lower extremity fractures
Fall from a bicycle	Without helmet: Head and neck lacerations, scalp and facial lacerations, upper extremity fractures With helmet: Upper extremity fractures Striking handlebar: Internal abdominal injuries

Reprinted from American College of Surgeons, Committee on Trauma: ATLS, Advanced Trauma Life Support for Doctors, 8th ed. Chicago, American College of Surgeons, 2008.

5. Describe the prehospital care capability for children with potentially serious injuries.

Emergency medical technicians have vast experience in advanced life support for adults, but important skills, such as endotracheal intubation and intravenous (IV) access, can be challenging in younger patients. Early studies reported successful field intubation rates of 50% in infants younger than 1 year of age and 64% in children younger than 18 years of age. Rates of success with IV access have been fair in infants, good in preschoolers, and excellent in adolescents. Even with programs conducted to improve these rates (such as procedures in the operating room and field courses), in settings with less experienced providers and short transit distances, the best procedure is safe extrication/preparation and immediate transfer to the hospital.

Dieckmann RA, Brownstein D, Gausche-Hill M, et al: Trauma. In: Dieckmann RA, editor: Pediatric education for prehospital professionals (PEPP), 2nd ed., Sudbury, MA, 2006, Jones & Bartlett, pp 128–155.

Gausche M, Lewis RJ, Stratton SJ, et al: Effect of out-of-hospital pediatric endotracheal intubation on survival and neurological outcome: A controlled clinical trial. JAMA 2000;283(6):783-790.

Losek JD, Szewczuga D, Glaeser PW: Improved prehospital pediatric ALS care after an EMT-paramedic clinical training course. Am J Emerg Med 1994;12(4):429-432.

Key Points: Abnormal Vital Signs in the Injured Child

1. Tachycardia in an injured patient may be due to pain or loss of blood volume.
2. Carefully evaluate the tachycardic trauma patient for the possibility of compensated shock.
3. An older child in compensated shock may be deceivingly responsive and alert.
4. Treat shock in the trauma patient with a rapidly infused 20mL/kg bolus of normal saline or lactated Ringer's solution, repeated once if shock persists.
5. If the patient is refractory to treatment with crystalloid, rapidly infuse packed red blood cells and emergently seek operative intervention.

6. Describe the initial approach to children with potentially serious injuries.

The ABCDEs are used during the primary assessment (called the primary survey) to define underlying injury and to reverse potential life-threatening problems. The team must assess and stabilize each step in order (e.g., control of airway always precedes control of circulation).

- **A**irway management: Asses and secure airway and maintain cervical spine control.
- **B**reathing: Maximize oxygen delivery.
- **C**irculation: Establish vascular access, control hemorrhage, and restore circulatory volume.
- **D**isability: Assess potentially critical injury to the central nervous system.
- **E**xposure: Visualize every part of the patient to assess for injury and control body temperature (especially important in young infants and children).

Dieckmann RA, Brownstein D, Gausche-Hill M, et al: Trauma. In: Dieckmann RA, editor: Pediatric education for prehospital professionals (PEPP), 2nd ed., Sudbury, MA, 2006, Jones & Bartlett, pp 128–155.

7. How can one appropriately "clear the cervical spine" in a pediatric trauma patient?

In the alert patient with no distracting injury and no midline cervical pain with palpation, the cervical spine can be cleared clinically by assessing active range of motion. Maintain proper immobilization and obtain plain film images if any of the following are present: cervical spine tenderness to palpation, altered mental status or neurologic deficits (presence or history of numbness, tingling sensation, decreased sensory or motor function), or distracting injury (such as an extremity fracture or abdominal pain). Desired radiographs include anteroposterior, lateral, and (if possible due to patient age and cooperation) odontoid films of the cervical spine.

Chung S, Mikrogianakis A, Wales PW, et al: Trauma association of Canada Pediatric Subcommittee National Pediatric Cervical Spine Evaluation Pathway: Consensus guidelines. J Trauma 2011; 70(4):873-884.

Slack SE, Clancy MJ: Clearing the cervical spine of paediatric trauma patients. Emerg Med J 2004; 21(2):189-193.

8. What factors raise suspicion for possible cervical spine injury in a pediatric trauma patient?

A recent large case-control study of 540 cases of children less than 16 years of age with cervical spine injury identified eight factors associated with cervical spine injury with 98% sensitivity (confidence interval [CI] 96-99%) and 26% specificity (CI 23-29%). These factors were altered mental status, focal neurologic findings, neck pain, torticollis, substantial torso injury, underlying physical conditions predisposing to cervical spine injury that are observable on physical examination (such as patients with Down syndrome, osteogenesis imperfecta, juvenile rheumatoid arthritis, Marfan syndrome), diving, and high-risk motor vehicle crash (head-on collision, rollover, ejection from vehicle, death in the same crash, speed >55 mph).

Leonard JC, Kuppermann N, Olsen C, et al: Factors associated with cervical spine injury in children after blunt trauma. Ann Emerg Med 2011;58(2):145-155.

9. Is hypertonic saline beneficial in the fluid resuscitation of the multiply injured trauma patient with severe head injury?

Studies performed in pediatric and adult trauma patients in the intensive care unit setting have demonstrated the safety and efficacy of hypertonic saline for acutely decreasing intracranial pressure compared to traditional therapies. The proposed mechanism for use of hypertonic solutions is that increased serum osmolality decreases intracranial pressure via the osmotic pressure gradient. Initial fluid resuscitation with hypertonic saline may therefore be helpful in supporting blood pressure as well as decreasing intracranial pressure.

Adelson PD, Bratton SL, Carney NA, et al: Guidelines for the acute medical management of severe traumatic brain injury in infants, children, and adolescents. Chapter 11. Use of hyperosmolar therapy in the management of severe pediatric traumatic brain injury. Pediatr Crit Care Med 2003;4(3 Suppl): S40-S44.

Scaife ER, Statler KD: Traumatic brain injury: Preferred methods and targets for resuscitation. Curr Opin Pediatr 2010;22(3):339-345.

Key Points: Initial Management of Potentially Serious Injuries

1. Always assess the airway first.
2. Once airway is secure, stabilize breathing.
3. Confirm or establish stable circulation.
4. Always maintain cervical spine immobilization and control.

10. Explain the secondary survey.

The secondary survey is a detailed head-to-toe physical examination that follows the initial ABCDE survey and resuscitation. Survey the head, neck, and face first for evidence of blood or occult injury while maintaining control of the cervical spine. Assessment of maxillary and mandibular stability, eyes, ears, and oropharynx follows. Careful assessment of the bony thorax, lungs, and cardiovascular system is next, followed by assessment of the abdomen, pelvis, and external genitourinary tract. Logroll the child in a neutral position to assess the back, posterior chest, and spine. Lastly, assess the extremities carefully for obvious and occult injury, along with neurovascular status. Evaluation of the central nervous system may be conducted during each part of the examination or performed at the end.

American College of Surgeons, Committee on Trauma: ATLS, advanced trauma life support for doctors, 8th ed, Chicago, 2008, American College of Surgeons.

11. What components of the neurologic examination should be conducted during the primary and secondary surveys?

A rapid neurologic evaluation is indicated rather than a detailed examination. It is not appropriate to assess the child's reflexes or fine-motor coordination during the primary survey. Instead, evaluate pupillary size, symmetry, and reactivity. Assess the child's level of consciousness by the AVPU mnemonic:

- **A:** **A**lert
- **V:** Responds to **V**oice
- **P:** Responds to **P**ain
- **U:** **U**nresponsive

The secondary survey should ascertain whether there is disability from a "quick" neurologic assessment based on the child's responsiveness.

American College of Surgeons, Committee on Trauma: ATLS, advanced trauma life support for doctors, 8th ed, Chicago, 2008, American College of Surgeons.

12. What initial radiographic and laboratory studies are important in trauma?

In unstable or high-risk patients, the most important studies to be obtained in the first 5 to 10 minutes include a complete blood count with differential, type and cross-match for packed red blood cells, coagulation studies (prothrombin time, partial thromboplastin time), and levels for the aminotransferases and amylase. Obtain initial radiographs including lateral cervical spine, chest, and pelvis views. Obtain a urinalysis to look for hematuria, evidence of a genitourinary injury.

In patients with mild to moderate trauma, screening blood studies and radiographs, although standard in the past, are no longer routine and are usually of low utility. It is always appropriate to reassess the patient and obtain studies later if needed.

13. Should one remove a penetrating foreign body?

Leave any foreign body to the neck, chest, abdomen, or back, or a foreign body that has the possibility of neurovascular compromise in any other location, in place during the initial assessment. Only remove this object in a controlled setting by a surgeon.

14. List the major signs of intra-abdominal bleeding caused by organ rupture.

- Abdominal tenderness
- Abdominal distention that does not improve after nasogastric decompression
- Shock
- Bloody nasogastric aspirate

Karl SR: Trauma. In Fuchs S, Yamamoto L, editors: APLS: the pediatric emergency medicine resource, 5th ed, Burlington, MA, 2012, Jones & Bartlett Learning, pp 204–261.

15. Describe the presentation and treatment of tension pneumothorax. Define open pneumothorax. How is this injury managed?

Tension pneumothorax results from penetrating chest trauma or acute barotrauma during blunt injury. Air is trapped behind a one-way flap-valve defect in the lung. The child presents with moderate to severe respiratory distress and contralateral tracheal deviation. Systemic perfusion is often compromised significantly by obstructed venous return. Treatment requires needle decompression followed by chest tube placement.

Open pneumothorax, or a "sucking chest wound," which may occur after a penetrating chest wound, allows free bidirectional flow of air between the affected hemithorax and surrounding atmosphere. Sucking chest wounds are extremely rare in children. Management centers on positive-pressure ventilation and covering the wound with an occlusive dressing.

Table 58-2. Therapeutic Classification of Hemorrhagic Shock in Pediatric Patients

MEASUREMENT	CLASS I	CLASS II	CLASS III	CLASS IV
Blood volume loss*	≤15%	15%-30%	30%-40%	≥40%
Pulse rate	Normal	Mild tachycardia	Moderate tachycardia	Severe tachycardia
Blood pressure	Normal/ increased	Normal/ decreased	Decreased	Decreased
Capillary blanch test	Normal	Abnormal	Abnormal	Abnormal
Respiratory rate	Normal	Mild tachypnea	Moderate tachypnea	Severe tachypnea
Urine output	1-2 mL/kg/hr	0.5-1.0 mL/kg/hr	0.25-0.5 mL/kg/hr	Negligible
Mental status	Slightly anxious	Mildly anxious	Anxious/confused	Confused/ lethargic
Fluid replacement (3:1 rule)	Crystalloid	Crystalloid	Crystalloid, blood	Crystalloid, blood

*Assume blood volume to be 8% to 9% of body weight (80-90 mL/kg).
Reprinted from Waltzman ML, Mooney DP: Major trauma. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1244-1255.

16. In a child who has suffered blunt abdominal trauma, what class of hemorrhage results in diminished pulse pressure, prolonged capillary refill, and normal systolic blood pressure?

These findings are associated with a class II hemorrhage (15-30% blood loss). This level of blood loss is associated with tachycardia, tachypnea, minimal decrease in urine output, decreased pulse pressure, prolonged capillary refill, and normal blood pressure. Hypotension is a late finding, associated with ongoing hemorrhage, and indicates that the child is near decompensation (Table 58-2).

Waltzman ML, Mooney DP: Major trauma. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1244-1255.

17. What is the "3-for-1" rule in trauma fluid resuscitation?

The 3-for-1 rule is a rough guideline for the total amount of crystalloid required in the acute management of hypovolemia. Replace each milliliter of lost blood with 3 mL of crystalloid fluid. This amount allows replacement of plasma volume lost into the interstitial space. The patient's response to fluid therapy remains the most important guideline for further fluid resuscitation (Fig. 58-1).

18. Which visceral injuries are more common in children?

Duodenal hematoma and blunt pancreatic injury may occur when a handle bar strikes the child in the right upper quadrant, due to undeveloped abdominal muscular tone. Small bowel perforations at or near the ligament of Treitz and mesenteric small bowel avulsion injuries are more common in children. The shallowness of the pelvis in a child leads to more frequent occurrences of bladder rupture. Because of the proximity of the peritoneum to the perineum, intraperitoneal injuries occur more commonly after straddle injuries in children.

Key Points: Pitfalls in Resuscitation of Injured Children

1. Failure to recognize impending respiratory failure or potential airway obstruction
2. Failure to recognize early shock when the patient has a "normal" blood pressure with other signs of hemorrhage (e.g., tachycardia, delayed capillary refill, diaphoresis, altered mental status)
3. Failure to recognize impending neurologic deterioration from unsuspected head injury

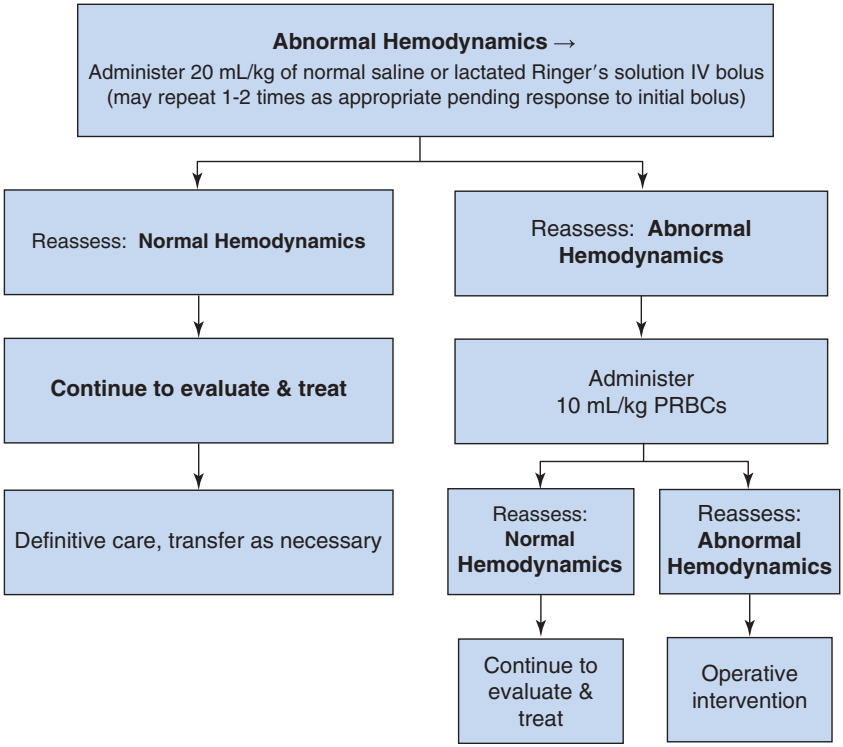


Figure 58-1. Fluid resuscitation in the pediatric trauma patient. IV, intravenous; PRBCs, packed red blood cells. *(Adapted from American College of Surgeons, Committee on Trauma: ATLS, Advanced Trauma Life Support for Doctors, 8th ed. Chicago, American College of Surgeons, 2008.)*

19. True or false: To open the airway in a pediatric trauma patient, the head-tilt/chin-lift maneuver is the recommended procedure.

False. The cervical spine must be immobilized in a neutral position. This goal is best accomplished with the *jaw-thrust spinal-stabilization maneuver*. Place two fingers on each side of the lower jaw and lift the jaw upward and outward. This maneuver can be performed without extension of the neck. The head-tilt/chin-lift maneuver may cause manipulation of the neck, which can convert an incomplete spinal cord injury to a complete one.

20. What are the factors that may interfere in the detection of multiple injuries in the pediatric patient?

- Lack of cooperation of the child due to any of the following: Preverbal or nonverbal, fearful, in pain, or with altered mental status due to injury or ingested substances
- No trauma team activation
- Occult injuries, which are not initially apparent
- Presentation without trauma as the history (physical abuse/nonaccidental trauma)

21. What is the most appropriate way to intubate the trachea of a seriously injured child?

The orotracheal route is preferred. The nasotracheal route is contraindicated because it can be very difficult without direct visualization of the airway. The patient's neck must remain in the neutral position and *should not be hyperextended* during the procedure. Always precede tracheal intubation with ventilation by a bag-valve-mask device. Ideally, one rescuer should stabilize the neck (bimanually) while another performs the endotracheal intubation.

The Sellick maneuver (cricoid pressure) is now controversial. It may be considered before the intubation if the child is unresponsive, but the potential benefit of using this method to prevent gastric distention and aspiration must be weighed against the possibility of impaired gas exchange and ventilation. Have a rigid suction device immediately available. In the child who is conscious, especially if increased intracranial pressure is a concern, administer a short-acting neuromuscular blocking agent (succinylcholine) and a sedative or anesthetic (thiopental) before intubation. Sedatives are a risk if the patient is hypotensive or "on the edge."

Ellis DY, Harris T, Zideman D: Cricoid pressure in emergency department rapid sequence tracheal intubations: A risk-benefit analysis. Ann Emerg Med 2007;50(6):653-665.

22. What can lead to gastric distention in an injured child? Why is gastric distention important?

Gastric distention can occur when the child swallows air at the time of injury. It also may be due to ventilation with a bag-valve-mask device or a leak around an endotracheal tube. Gastric distention is important because it can compromise ventilation. It limits diaphragmatic motion, reduces lung volume, and increases the risk of vomiting and aspiration.

23. How should gastric distention in an injured child be managed?

Place a nasogastric tube as soon as the airway is controlled, and decompress the stomach. Use an orogastric tube instead of nasogastric decompression if the child has significant facial trauma or a maxillofacial or basal skull fracture. This route prevents possible intracranial placement of the tube.

24. Which signs indicate shock in injured children?

Shock may be difficult to recognize because the signs are subtle and mimic those of fright or pain. Injured children with loss of about 15% of blood volume exhibit tachycardia, cool extremities, delayed capillary refill beyond 2 seconds, and possibly weak pulses. Confusion and clammy skin also may be present. Hypotension may not be evident until the child has lost 25% to 40% of blood volume and is thus a late finding.

25. List the current child passenger safety recommendations.

1. All infants and toddlers should be in a rear-facing car safety seat until the age of 2 years (or until the maximum height/weight limit has been reached for a specific car safety seat as listed by the manufacturer).
2. Children 2 years of age or older should use a forward-facing car safety seat with a harness until the maximum height/weight limit for a car seat has been reached.
3. Children whose height or weight is greater than the forward-facing limit of a car seat should use a belt-positioning booster seat until they reach a height of 4 feet 9 inches tall (and are between 8 and 12 years of age).
4. Children younger than 13 years of age should be properly restrained with a lap-and-shoulder seat belt in the rear seat of the vehicle.

Committee on Injury, Violence, and Poison Prevention, Gardner HG, Baum CR, Dowd MD, et al: Child passenger safety. Pediatrics 2011;127(4):788-793.

26. How might airbags and pediatric restraint devices in vehicles influence motor vehicle crash–related injuries?

Children younger than 12 years of age seated in the front seat of the car are at risk for injuries from airbag deployment, such as facial burns, lacerations, contusions, abrasions, and eye injuries. More devastating injuries such as spinal cord transection and blunt head and neck trauma have also been reported in children who were restrained in the front seat. Children restrained in the rear seat are one half to two thirds as likely to be injured as those restrained in the front seat. Unrestrained and improperly restrained children are at risk of being ejected during a motor vehicle collision. Age-appropriate restraint devices have been shown to decrease rates of mortality and significant injury in children 6 years old and younger.

Arbogast KB, Kallan MJ, Durbin DR: Front versus rear seat injury risk for child passengers: Evaluation of newer model year vehicles. Traffic Inj Prev 2009;10(3):297-301.

National Highway Traffic Safety Administration (NHTSA). Available at http://www.nhtsa.gov/. Accessed on July 9, 2013.

Scheidler MG, Shultz BL, Schall L, et al: Risk factors and predictors of mortality in children after ejection from motor vehicle crashes. J Trauma 2000;49(5):864-868.

Tyroch AH, Kaups KL, Sue LP, et al: Pediatric restraint use in motor vehicle collisions: Reduction of deaths without contribution to injury. Arch Surg 2000;135(10):1173-1176.

27. Can the intraosseous (IO) route be used to establish vascular access in an injured child?

Yes. The IO route is expeditious and may be lifesaving if an IV line cannot be established quickly. It can be used to deliver crystalloid fluids, blood, or medications. Placement sites include the proximal tibia, proximal humerus, distal ulna, and distal femur. Placement of an IO line is contraindicated in a fractured long bone, an extremity with a vascular injury, an area with an overlying skin infection or burn, or an extremity where an IO line was recently attempted or placed. Patients with underlying bone fragility syndromes (such as osteogenesis imperfecta) are not good candidates for IO placement. IO access currently is underutilized as an emergent access technique.

Karl SR: Trauma. In Fuchs S, Yamamoto L, editors: APLS: the pediatric emergency medicine resource, 5th ed, Burlington, MA, 2012, Jones & Bartlett Learning, pp 204–261.

Luck RP, Haines C, Mull CC: Intraosseous access. J Emerg Med 2010;39(4):468-475.

Voigt J, Waltzman M, Lottenberg L: Intraosseous vascular access for in-hospital emergency use: A systematic clinical review of the literature and analysis. Pediatr Emerg Care 2012;28(2):185-199.

28. When is a blood transfusion indicated for an injured child?

A blood transfusion is necessary in children when signs of shock from hemorrhage persist despite two boluses of crystalloid (20 mL/kg each). Administer blood as a bolus of packed red blood cells in aliquots of 10 mL/kg. Warming the blood to body temperature is recommended to increase the speed of transfusion and prevent hypothermia. Give type-specific, cross-matched blood; O-negative blood may be imperative if shock is present and type-specific blood is not available. Patients requiring more than 40 mL/kg of packed red blood cells receive benefit from balanced blood-product transfusions, with weight-based fixed ratios of fresh frozen plasma, packed red blood cells, platelets, and cryoprecipitate as administered in a massive blood transfusion protocol to help prevent coagulopathies.

Diab YA, Wong EC, Luban NL: Massive transfusion in children and neonates. Br J Haematol 2013; 161:15-26.

Hendrickson JE, Shaz BH, Pereira G, et al: Implementation of a pediatric trauma massive transfusion protocol: One institution's experience. Transfusion 2012;52(6):1228-1236.

29. What is the most likely injury in a child with blunt head trauma and no evidence of external bleeding who presents with signs of shock?

The child most likely has a serious injury of the chest, abdomen, or pelvis with internal bleeding. Isolated head trauma rarely results in shock, although a significant scalp laceration can produce excessive blood loss. Femur fractures from high-impact injuries in adolescents may lead to shock.

30. How does the anatomy of a child relate to the type of injury sustained?

Because of their smaller size, children sustain injuries to multiple organ systems more commonly than do adults exposed to the same mechanism. The immature skeletons of children result in less frequent bone injuries and more frequent soft tissue injuries and internal organ damage. Bone injury may be more subtle because children have open growth plates at the epiphyses. Owing to a flexible and less muscular chest wall, rib fractures and flail chest are less common, but forces are more easily transmitted to internal organs. Likewise, the solid organs in the abdomen are disproportionately larger and more exposed than in adults. Children have larger heads relative to their bodies and are more likely to land on their heads when they fall. The large head also contributes to cervical spine injuries at a higher level (C2-C3) in children than in adults. Because children have more skin surface area in relation to their overall size than adults, they can lose heat quickly after injury, resulting in hypothermia.

American College of Surgeons, Committee on Trauma: ATLS, advanced trauma life support for doctors, 8th ed, Chicago, 2008, American College of Surgeons.

31. What are the important aspects of medical history in seriously injured children?

In managing a critically injured child, it is not appropriate to divert attention to a long, detailed history. Instead, the American College of Surgeons recommends an AMPLE history:

- **A: A**llergies
- **M: M**edications
- **P: P**ast illnesses
- **L: L**ast meal
- **E: E**vents surrounding the injury

32. What are the indications for endotracheal intubation of the injured child?

- Inability to ventilate with a bag-valve-mask device
- Need for prolonged airway control
- Prevention of aspiration in a comatose child
- Severe traumatic brain injury (Glasgow Coma Scale [GCS] score of 8 or less)

33. What are the criteria for admission to a pediatric trauma center?

- GCS score of 12 or less
- Decompensated shock (low systolic blood pressure for age)
- Shock unresponsive to fluid resuscitation
- Abnormal respiratory rate (adjusted for age)

34. When a trauma patient requires transfer to a trauma center, what are the responsibilities of the transferring physician?

- Identification of the patient requiring transfer
- Initiation of the transfer process via phone contact with the receiving doctor
- Maximal stabilization using the capabilities of the local institution
- Determination of the best mode of transport
- Assurance that the level of care allows the patient to remain stable and not deteriorate
- Transfer of all relevant records, results, and radiographs to the receiving facility

Waltzman ML, Mooney DP: Major trauma. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, Chap. 105, pp 1244-1255.

CHAPTER 59

NECK AND CERVICAL SPINE TRAUMA

Nadine Smith and Howard Kadish

1. What are the differences in neck anatomy between children and adults?

Compared to adults, children have a larger head and mandible and a shorter neck in proportion to the rest of their body. These differences protect the child's anterior neck at the time of injury; therefore the head and face absorb the major force of impact.

In infants and young children, not only is the larynx smaller in overall size, but it also has smaller relative dimensions compared with adults. In children, the arytenoids are larger, the epiglottis has an omega shape, and the larynx has a funnel shape, which is narrowest in the subglottic region. In adults, the narrowest point of the trachea is located at the level of C7, whereas in children it is at the cricoid cartilage ring at the level of C4. These anatomic differences, along with the ring-like cricoid cartilage, result in a narrowed laryngeal inlet in children. Adolescents and adults can tolerate up to 50% narrowing of the airway without obvious respiratory distress, whereas infants and children experience significant respiratory embarrassment with this degree of restriction.

Jagannathan J, Dumont AS, Prevedello DM, et al: Cervical spine injuries in pediatric athletes: Mechanisms and management. Neurosurg Focus 2006;21(4):1-5.

Khanna G, El-Khoury GY: Imaging of cervical spine injuries of childhood. Skeletal Radiol 2007; 36(6):477-494.

Lustrin ES, Karakas SP, Ortiz AO, et al: Pediatric cervical spine: Normal anatomy, variants, and trauma Radiographics 2003;23(3):539-560.

McCall T, Fassett D, Brockmeyer D: Cervical spine trauma in children: A review. Neurosurg Focus 2006;20 (2):1-8.

Woodward GA: Neck trauma. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1376-1421.

2. Describe the three anatomic zones of the neck.

Zone I is the area in between the thoracic inlet and the cricoid. Zone II extends from the cricoid to the angle of the mandible. Zone II is the most common site for penetrating trauma. Zone III includes the area above the angle of the mandible. Awareness of these landmarks and the anatomy present within each are valuable when evaluating and managing neck injuries.

Woodward GA: Neck trauma. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1376-1421.

3. What are some preexisting conditions that put some children at higher risk for cervical spine trauma?

- Down syndrome
- Achondroplasia
- Morquio syndrome (odontoid hypoplasia)
- Klippel-Feil syndrome (congenital fusion of the cervical spine)
- Cervical stenosis
- Chiari malformation
- Rheumatic disease

Woodward GA: Neck trauma. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1376-1421.

4. Name the major signs and symptoms of a laryngotracheal injury.

- Neck pain/tenderness
- Drooling
- Stridor
- Crepitus
- Odynophagia (painful swallowing)
- Retractions
- Aphonia (loss of voice)
- Dysphagia (difficulty swallowing)
- Airway obstruction
- Subcutaneous emphysema
- Hoarseness
- Pneumothorax
- Cough
- Neck deformity
- Dysphonia (change in voice)
- Pneumomediastinum

Demetriades D, Salim A, Brown C, et al: Neck injuries. Curr Probl Surg 2007;44(1):13-85.
Kadish H, Schunk J, Woodward GA: Blunt pediatric laryngotracheal trauma: Case reports and review of the literature. Am J Emerg Med 1994;12(2):207-211.
Losek JD, Tecklenburg FW, White DR: Blunt laryngeal trauma in children: Case report and review of initial airway management. Pediatr Emerg Care 2008;24(6):370-373.
Woodward GA: Neck trauma. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1376-1421.

5. How should I manage a patient with a blunt or penetrating neck injury?

The goals of management follow trauma guidelines with strict attention to airway, breathing, and circulation (ABCs). After the airway is assessed and breathing stabilized, control any hemorrhage and maintain good cervical spine precautions rapidly. Stabilize all penetrating objects in the neck, but keep them in place until they can be removed under surgical care in the operating room. Obtain routine trauma laboratory studies, including type and crossmatch of blood for packed red blood cells. Minimal radiographic evaluation includes cervical spine films and chest radiograph.

Abujamra L, Joseph MM: Penetrating neck injuries in children: A retrospective review. Pediatr Emerg Care 2003;19(5):308-313.
Losek JD, Tecklenburg FW, White DR: Blunt laryngeal trauma in children: Case report and review of initial airway management. Pediatr Emerg Care 2008;24(6):370-373.

6. How should the airway and breathing be managed in a child with a neck injury?

Give all patients supplemental oxygen and treat them as if they have a cervical spine injury until proved otherwise. Include cervical spine stabilization for any airway manipulation.

Elective endotracheal intubation is not recommended unless backup measures, such as a surgical airway or fiberoptic intubation equipment, are available. If endotracheal intubation is to be completed, take special care to avoid hyperextension by using inline neck immobilization and gentle cricoid pressure. Always intubate with two providers in order to ensure cervical spine safety.

Attempted placement of an endotracheal tube through an already injured airway may cause a small mucosal laceration to progress to complete transection. In cases of laryngotracheal separation, the transected ends of the trachea may separate by as much as 8 cm. Successful passage of an endotracheal tube across this distance is difficult and may delay or preclude airway control. Trauma to the airway also may produce a blind path and inability to pass an endotracheal tube successfully. In the unstable airway, blind nasotracheal intubation is not recommended. If orotracheal intubation needs to be performed emergently, ensure that backup measures, such as surgical and anesthesia consultation, fiberoptic bronchoscopy, cricothyrotomy, and tracheostomy, are available in case complications occur.

Abujamra L, Joseph MM: Penetrating neck injuries in children: A retrospective review. Pediatr Emerg Care 2003;19(5):308-313.

Demetriades D, Salim A, Brown C, et al: Neck injuries. Curr Probl Surg 2007;44(1):13-85.
Losek JD, Tecklenburg FW, White DR: Blunt laryngeal trauma in children: Case report and review of initial airway management. Pediatr Emerg Care 2008;24(6):370-373.

7. What are the major indications for surgical evaluation in patients with neck trauma?

- Unstable vital signs
- Hematemesis
- Exposed cartilage
- Hemothorax
- Cord paralysis
- Neurologic deficits
- Dysphagia
- Foreign bodies
- Airway obstruction
- Hemoptysis
- Displaced fracture
- Pneumothorax
- Active bleeding
- Decreased level of consciousness
- Dysphonia
- Gun, rifle, or explosion wounds

Abujamra L, Joseph MM: Penetrating neck injuries in children: A retrospective review. Pediatr Emerg Care 2003;19(5):308-313.
Demetriades D, Salim A, Brown C, et al: Neck injuries. Curr Probl Surg 2007;44(1):13-85.
Losek JD, Tecklenburg FW, White DR: Blunt laryngeal trauma in children: Case report and review of initial airway management. Pediatr Emerg Care 2008;24(6):370-373.

8. What are the major differences between pediatric and adult cervical spines?

Children have various anatomic differences from adults that make them more prone to cervical spine fractures. Their relatively large heads, hypermobile spines, and weak neck muscles (specifically the underdeveloped paraspinous muscles) make them particularly susceptible to upper cervical fractures.

Higher fulcrum locations in children's necks also contribute to an increased incidence of upper cervical fractures, specifically those fractures located from the occiput to C2. In infants, the fulcrum is present at C2-C3. By the age of 5 to 6 years, the fulcrum shifts to C3-C4. It is not until the age of 8 years that the fulcrum is similar to those of adults, being located at C5-C6. The higher fulcrum location in combination with weak neck muscles and poor protective reflexes makes children more susceptible to upper cervical injuries.

Jagannathan J, Dumont AS, Prevedello DM, et al: Cervical spine injuries in pediatric athletes: Mechanisms and management. Neurosurg Focus 2006;21(4):1-5.
Khanna G, El-Khoury GY: Imaging of cervical spine injuries of childhood. Skeletal Radiol 2007; 36(6):477-494.
McCall T, Fassett D, Brockmeyer D: Cervical spine trauma in children: A review. Neurosurg Focus 2006;20 (2):1-8.
Platzer P, Jaindl M, Thalhammer G, et al: Cervical spine injuries in pediatric patients. J Trauma Inj Infect Crit Care 2007;62(2):389-396.
Woodward GA: Neck trauma. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1376-1421.

9. How common are cervical spine injuries in children?

Cervical spine injuries are rare, occurring in only 1% to 2% of pediatric patients with injuries due to blunt trauma. Motor vehicle accidents account for most cervical spine injuries; however, falls, sports, firearms, nonaccidental trauma, and obstetric complications also can account for such injuries. Because a cervical spine injury can result in permanent disability or death, carefully evaluate patients with suspicious mechanisms of injury.

Dormans JP: Evaluation of children with suspected cervical spine injury. J Bone Joint Surg 2002; 84(1):124-132.

McCall T, Fassett D, Brockmeyer D: Cervical spine trauma in children: A review. Neurosurg Focus 2006;20(2):1-8.

10. Which methods are helpful in immobilizing a patient with suspected cervical spine injury?

Soft cervical collars offer no stability to a potentially unstable cervical spine. Even hard collars (Philadelphia, The Stif-Neck, Miami J) allow a significant amount of motion. Place patients with a suspected cervical spine fracture in a rigid cervical collar and immobilize on a hard spine board. Sizing of the collar will depend on both the age of the patient and the type of collar used. Generally, apply the longest collar that does not hyperextend the neck. It should also not interfere with airway management but should provide support to the chin and jaw. Even with adequate immobilization, the patient may be able to flex or extend the neck approximately 20% to 60% depending on the type of collar used. Contraindications to placement of a cervical collar include cervical swelling or the need for surgical intervention of the airway.

Anderson RC, Scaife ER, Fenton SJ, et al: Cervical spine clearance after trauma in children. J Neurosurg Pediatr 2006;105(5):361-364.

McCall T, Fassett D, Brockmeyer D: Cervical spine trauma in children: A review. Neurosurg Focus 2006;20 (2):1-8.

Woodward GA: Neck trauma. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1376-1421.

11. What type of cervical spine radiographs should I order for a child with a suspected neck injury?

At a minimum, order three views—lateral, anteroposterior, and odontoid (open mouth view). However, the odontoid view is a difficult view to obtain and may not be useful in children less than 5 years of age. Be sure the initial three views adequately demonstrate all cervical vertebrae, including the relationship of C7 on T1. If C7-T1 is not visualized, obtain a swimmer's view.

Alone, a lateral cervical spine view misses approximately 20% of cervical spine injuries, but when combined, the three views miss approximately 3% to 6% of cervical spine injuries. Pediatric cervical spine films can also be difficult to interpret due to a large amount of cartilage and variability of the retropharyngeal space related to the child's crying or respiratory patterns. Therefore, if a patient is still symptomatic, consider further imaging tests such as cervical spine computed tomography (CT) or magnetic resonance imaging (MRI) of the cervical spine.

Anderson RC, Scaife ER, Fenton SJ, et al: Cervical spine clearance after trauma in children. J Neurosurg Pediatr 2006;105(5):361-364.

Khan SN, Erickson G, Sena MJ, et al: Use of flexion and extension radiographs of the cervical spine to rule out acute instability in patients with negative computed tomography scans. J Orthop Trauma 2011;25 (1):51-56.

Khanna G, El-Khoury GY: Imaging of cervical spine injuries of childhood. Skeletal Radiol 2007; 36(6):477-494.

Swischuk LE, John SD, Hendrick EP: Is the open-mouth odontoid view necessary in children under 5 years? Pediatr Radiol 2000;30(3):186-189.

12. What about flexion-extension views? Are they helpful?

The clinical value of flexion-extension views is questionable, and their use in the acute evaluation of neck injuries is controversial. Do not obtain these routinely. For alert patients with neck pain, normal plain films (three views), and no neurologic deficits, flexion-extension views are sometimes ordered. These x-rays are obtained while the patient independently flexes and extends the neck, only until the child feels pain or discomfort.

Woodward GA: Neck trauma. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1376-1421.

13. When should CT or MRI of the cervical spine be ordered for a suspected cervical spine injury?

CT identifies fractures but is not ideal for evaluation of the spinal cord or ligaments. It is useful in the secondary evaluation of a possible cervical spine fracture when plain radiographs are difficult to obtain or to interpret or noted to be abnormal, or if there is a high suspicion for injury despite normal radiographs. Given the low incidence of pediatric cervical spine injuries and the increased radiation exposure with CT, do not use CT as a primary radiographic evaluation in patients with a possible cervical spine fracture.

MRI is not ideal for identifying bony fractures. If a cervical spine injury is diagnosed, obtain an MRI to evaluate the ligaments and spinal cord for potential injury such as disk herniation, spinal cord edema, hemorrhage, compression, or transection. MRI is also valuable in the obtunded, intubated, or uncooperative patient as well as the child with persistent or delayed neurologic symptoms.

Anderson RC, Scaife ER, Fenton SJ, et al: Cervical spine clearance after trauma in children. J Neurosurg Pediatr 2006;105(5):361-364.

Flynn JM, Closkey RF, Mahboubi S, Dormans JP: Role of magnetic resonance imaging in the assessment of pediatric cervical spine injuries. J Pediatr Orthop 2002;22(5):573-577.

Lustrin ES, Karakas SP, Ortiz AO, et al: Pediatric cervical spine: Normal anatomy, variants, and trauma Radiographics 2003;23(3):539-560.

14. What is the best way to read cervical spine radiographs?

Use a systematic approach, such as the ABCS method:

- **A: A**lignment (Four lines are assessed: anterior vertebral line, posterior vertebral line, spinolaminar line, spinous process line.)
- **B: B**ones (Evaluate for fractures of all vertebrae.)
- **C: C**artilage (Because cartilage is radiolucent on cervical spine radiographs, evaluate the intervertebral space where cartilage is present. Compression or widening of the intervertebral space may indicate a cartilage disruption.)
- **S: S**oft tissues (Because a child's spinal column contains a significant amount of cartilage, prevertebral soft tissue swelling may be the only clue to cartilage or ligament injury. The prevertebral space at C2 or C3 should not be greater than half the width of the adjacent vertebral body. Abnormal swelling of the prevertebral space may be due to blood or edema.)

Khanna G, El-Khoury GY: Imaging of cervical spine injuries of childhood. Skeletal Radiol 2007; 36(6):477-494.

Woodward GA: Neck trauma. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1376-1421.

15. What is the NEXUS?

The National Emergency X-Radiography Utilization Study (NEXUS) was a multicenter, prospective, observational study of emergency department (ED) patients with blunt trauma for whom cervical spine imaging was ordered. The results of the study suggested which patients would benefit from imaging and which would not. According to the NEXUS, patients must meet *all five* of the following criteria to be considered low risk, and therefore not need cervical spine imaging:

1. No midline cervical tenderness
2. No evidence of intoxication
3. No altered level in mental status
4. No focal neurologic deficit
5. No distracting or painful injury

If a patient does not meet all five of these criteria, evaluate him or her for possible cervical spine injury with radiographic imaging. Do not use this rule in children younger than 8 years old, as the original study included only a small population of children under this age.

Chung S, Mikrogianakis A, Wales PW, et al: Trauma association of Canada pediatric subcommittee national pediatric cervical spine evaluation pathway: Consensus guidelines. J Trauma Acute Care Surg 2011;70 (4):873-884.

Hoffman JR, Mower WR, Wolfson AB, et al: Validity of a set of clinical criteria to rule out injury to the cervical spine in patients with blunt trauma. N Engl J Med 2000;343(2):94-99.

Hoffman JR, Wolfson AB, Todd K, Mower WR: Selective cervical spine radiography in blunt trauma: Methodology of the National Emergency X-Radiography Utilization Study (NEXUS). Ann Emerg Med 1998;32(4):461-469.
Viccellio P, Simon H, Pressman BD, et al: A prospective multicenter study of cervical spine injury in children. Pediatrics 2001;108(2):e20.

16. What is the Canadian C-spine rule?

The Canadian C-spine rule was developed from a prospective cohort study in Canada evaluating patients with head or neck trauma. If there is a high-risk factor (dangerous mechanism, paresthesias), perform radiography. A low-risk factor (simple rear-end motor vehicle collision, sitting position in ED, ambulatory at any time since injury, delayed onset of neck pain, or absence of midline cervical spine tenderness) would allow for the clinician to assess the patient's range of motion. If the patient is able to actively rotate his or her neck 45 degrees to the left and right, then cervical spine radiography may not be needed.

Chung S, Mikrogianakis A, Wales PW, et al: Trauma association of Canada pediatric subcommittee national pediatric cervical spine evaluation pathway: Consensus guidelines. J Trauma Acute Care Surg 2011;70(4):873-884.
Stiell IG, Wells GA, Vandemheen KL, et al: The Canadian C-spine rule for radiography in alert and stable trauma patients. JAMA 2001;286(15):1841-1848.

17. What is pseudosubluxation of C2 on C3? How do you differentiate it from anterior subluxation of C2 on C3?

Pseudosubluxation is a normal variant seen in approximately 25% of patients up to 16 years of age. It is due to ligamentous laxity and horizontal positioning of facet joints. Anterior subluxation of C2 on C3 is usually caused by a hyperflexion injury following a hyperextension injury with possible associated spinal cord injury.

Swischuk's "posterior cervical line" (Fig. 59-1) is helpful in distinguishing pseudosubluxation from anterior subluxation on a lateral cervical film. A line is drawn from the

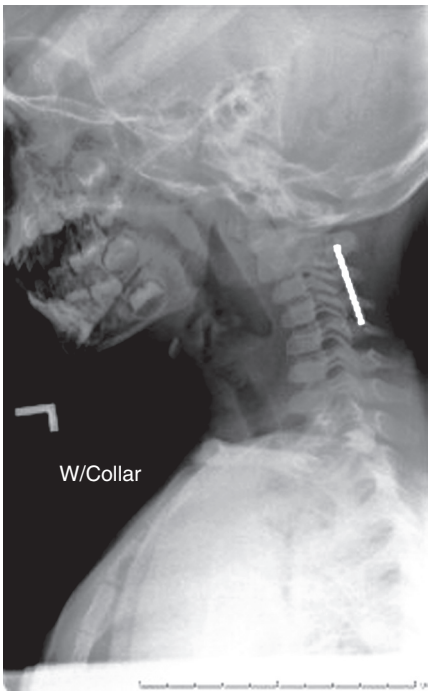


Figure 59-1. Posterior cervical line. *(From Easter JS, Barkin R, Rosen CL, Ban K: Cervical spine injuries in children, Part II: Management and special considerations. J Emerg Med 2011;41(3):252-256.)*

cortex of the spinous process of C1 to the cortex of the spinous process of C3. If the line is located more than 1.5 to 2.0 mm anterior to the cortex of the spinous process of C2, suspect a fracture of C2.

Khanna G, El-Khoury GY: Imaging of cervical spine injuries of childhood. Skeletal Radiol 2007; 36(6):477-494.

McCall T, Fassett D, Brockmeyer D: Cervical spine trauma in children: A review. Neurosurg Focus 2006;20 (2):1-8.

Swischuk L: Emergency Radiology of the Acutely Ill or Injured Child, 3rd ed. Baltimore, Williams & Wilkins, 1994.

Woodward GA: Neck trauma. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1376-1421.

18. What is a Jefferson fracture?

A Jefferson fracture (Fig. 59-2) is a burst fracture of C1 secondary to an axial load of the occipital condyles of the skull on the lateral masses of C2 (such as with a diving injury). Patients may present with severe neck pain, especially with rotation. Although patients rarely have neurologic symptoms because the fracture bursts outward and does not impair the spinal cord, a Jefferson fracture is unstable and needs to be immobilized immediately. The odontoid view may show the lateral masses offset or overriding the vertebral body of C2. Radiographic criteria include a lateral offset of the lateral masses of C1 greater than 1 mm from the vertebral body of C2.

This fracture can be confused with a pseudo-Jefferson fracture, which is a normal variant due to the child's growth. Children's lateral masses of C1 grow faster than the vertebral body of C2; often the lateral masses override C2 either unilaterally or bilaterally. A pseudo-Jefferson fracture can be present in up to 90% of children under the age of 2 years but usually normalizes by the age of 4 years. If in doubt, CT of the cervical spine at C1 and C2 is helpful in making the diagnosis.

Khanna G, El-Khoury GY: Imaging of cervical spine injuries of childhood. Skeletal Radiol 2007; 36(6):477-494.

Woodward GA: Neck trauma. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1376-1421.

19. What is a hangman's fracture?

A hangman's fracture (Fig. 59-3) results from severe hyperextension of the neck. The name refers to a similar injury that results from execution-style hangings. During hyperextension, there is compression of the posterior elements of C2, which can cause a fracture of the pars interarticularis of C2. This type of fracture can also have ligamentous damage secondary to hyperflexion after the hyperextension. Ligamentous damage can lead to anterior subluxation of

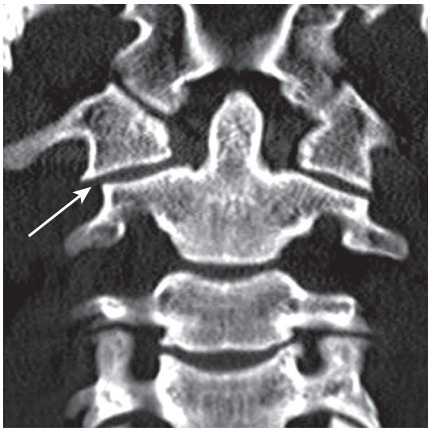


Figure 59-2. Jefferson fracture (*arrow*) with offset of lateral masses. *(From Jo K, Park I, Hong JT: Motion-preserving reduction and fixation of C1 Jefferson fracture using a C1 lateral mass screw construct. J Clin Neurosci 2011;18(5):695-698.)*

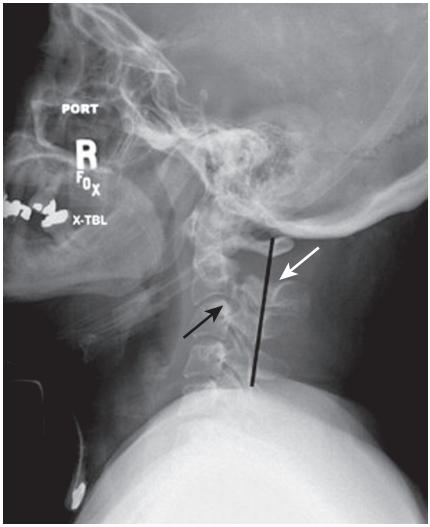


Figure 59-3. Hangman's fracture: Fracture of the pars interarticularis of C2. Anteriolisthesis of C2 on C3 *(black arrow)*. Posterior displacement of C2 *(white arrow)*. Spinolaminar line *(black line)*. *(From Giauque AP, Bittle MM, Braman JP: Type I hangman's fracture. Curr Probl Diagn Radiol 2012;41 (4):116-117.)*

C2 on C3. Use Swischuk's "posterior cervical line" in distinguishing pseudosubluxation from a subtle hangman's fracture on a lateral cervical film.

Patients with this injury usually present after a motor vehicle crash, and they may have neck pain but often no neurologic findings. Immobilize the cervical spine of these patients.

Khanna G, El-Khoury GY: Imaging of cervical spine injuries of childhood. Skeletal Radiol 2007; 36(6):477-494.

Woodward GA: Neck trauma. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1376-1421.

20. Why should patients with Down syndrome receive cervical spine radiographs before participating in sports?

Approximately 15% of patients with Down syndrome have atlantoaxial subluxation (AAS) secondary to atlantoaxial instability (AAI), and many with this condition will be asymptomatic. AAI occurs because of transverse ligament laxity (the most common reason in Down syndrome patients) or when a fractured dens allows movement between C1 and C2. If AAI progresses to AAS, it will be demonstrated on the lateral cervical spine radiograph with a widening of the predental space. A normal predental space should be less than 3 mm in adults and less than 4 to 5 mm in children. Neurologic symptoms such as abnormal gait, loss of sphincter control, and/or paralysis may be seen if the predental space is 7 to 10 mm. In addition to Down syndrome, AAI has been associated with minor trauma in patients with connective tissue disorders, arthritis, and pharyngitis.

Ali FE, Al-Bustan MA, Al-Busairi WA, et al: Cervical spine abnormalities associated with Down syndrome. Int Orthop 2006;30(4):284-289.

Elliott S, Morton RE, Whitelaw RA: Atlantoaxial instability and abnormalities of the odontoid in Down's syndrome. Arch Dis Child 1988;63(12):1484-1489.

21. What is SCIWORA?

SCIWORA (**S**pinal **C**ord **I**njury **W**ith**O**ut **R**adiographic **A**bnormality) usually is seen in children younger than 8 years because of the increased amount of cartilage and elastic nature of the younger child's spinal column. The child with a spine injury may present with an abnormal neurologic examination caused by vascular or ligamentous injury and no evidence of a fracture on plain radiography or CT. Neurosurgical consultation is recommended for this type of injury, as well as MRI to further evaluate the cervical spine and spinal cord.

Hendey GW, Wolfson AB, Mower WR, Hoffman JR; National Emergency X-Radiography Utilization Study Group: Spinal cord injury without radiographic abnormality: Results of the National

Emergency X-Radiography Utilization Study in blunt cervical trauma. J Trauma Inj Infect Crit Care 2002;53(1):1-4.
Khanna G, El-Khoury GY: Imaging of cervical spine injuries of childhood. Skeletal Radiol 2007; 36(6):477-494.
Pang D: Spinal cord injury without radiographic abnormality in children, 2 decades later. Neurosurgery 2004;55(6):1325-1343.
Yucesoy K, Yuksel KZ: SCIWORA in MRI era. Clin Neurol Neurosurg 2008;110(5):429-433.

22. Describe the appropriate treatment of a child with a spinal cord injury.

First, focus on the ABCs of trauma management. Children may present with hypotension, bradycardia, and warm or flushed peripheral extremities (spinal shock) because of the loss of sympathetic input. They need fluid resuscitation and inotropic support (dopamine). A common mistake is to treat patients with spinal shock as if they have hypovolemic shock and to overload them with fluids. If the child is not improving after aggressive fluid resuscitation, consider spinal shock and use inotropic support to stabilize their blood pressure.

23. Why is the administration of steroids in pediatric spinal cord injuries controversial and no longer recommended?

Experts in emergency medicine and neurosurgery have questioned the potential benefit of steroids for a traumatic cervical spine injury. Children younger than 13 years of age were excluded from the studies, and some experts feel the potential risks of steroids are greater than the potential neurologic benefits. The administration of high-dose steroids is no longer recommended in most types of spinal cord injury. Give steroids only after consultation with a neurosurgeon or trauma surgeon.

In 2013, based on available evidence, the American Association of Neurological Surgeons and the Congress of Neurological Surgeons said glucocorticoids are not recommended for acute spinal cord injury.

Hurlbert RJ, Hadley MN, Walters BC: Pharmacolgical therapy for spinal cord injury. Neurosurgery 2013;72 (S2):93-105.

Key Points: Neck and Cervical Spine Trauma

1. Cervical spine injuries occur in only 1% to 2% of pediatric patients.
2. Obtain radiographs for any pediatric trauma patient with an altered mental status, abnormal neurologic examination, point tenderness of the cervical spine, or pain with rotation. Use CT and MRI as adjuncts and not for first-line imaging.
3. One view is no view—at a minimum, for patients with suspected cervical spine injury, order lateral, anteroposterior, and odontoid radiographs.
4. The administration of high-dose steroids is no longer recommended in most types of spinal cord injury. Consult a neurosurgeon or trauma surgeon before giving steroids.
5. When in doubt, immobilize, obtain imaging studies, and consult a neurosurgeon or trauma surgeon.

24. How do you differentiate benign torticollis from atlantoaxial rotary subluxation?

Benign torticollis is due to a muscular spasm of the sternocleidomastoid muscle (SCM). On examination the patient's head will be tilted toward the affected SCM and the chin will be rotated away. If there is a history of trauma prior to the development of pain, plain radiographs may help to differentiate benign torticollis from other serious causes. Treatment generally includes soft collar placement, rest, and analgesia.

Atlantoaxial rotary subluxation is due to a rotational displacement of C1 on C2 and can occur after minor trauma, upper respiratory tract infections, or pharyngeal surgery or with certain congenital cervical malformations. These patients look similar to those with benign torticollis; however, the spasm of the SCM is on the same side toward which the chin points. With this condition, odontoid views can be helpful. They may demonstrate asymmetry of the lateral masses of C1 in relation to the odontoid. Specifically, one of the lateral masses of C1 is medially offset and will appear rotated forward, wider, and closer to the midline. The other lateral mass is laterally offset and will appear more narrow and farther from midline. Reconstructive CT views can help evaluate the location of C1 upon C2, but if there are neurologic changes on examination, MRI is the preferred test of choice to evaluate for cord compression. Treatment includes management of the underlying cause and neck

immobilization. Treatment for mild rotary subluxation includes soft collar and analgesia, although more severe cases require neurosurgical consultation and may require further immobilization, traction, and possibly fusion of the cervical spine.

Chih YK, Szu-Kai H, Chang J, et al: Pediatric atlantoaxial rotary subluxation after minor trauma and chiropractic manipulation. Fu-Jen J Med 2013;11:53-58.
Khanna G, El-Khoury GY: Imaging of cervical spine injuries of childhood. Skeletal Radiol 2007; 36(6):477-494.
Muñiz AE, Belfer RA: Atlantoaxial rotary subluxation in children. Pediatr Emerg Care 1999;15(1):25-29.
Pharisa C, Lutz N, Roback MG, Gehri M: Neck complaints in the pediatric emergency department: A consecutive case series of 170 children. Pediatr Emerg Care 2009;25(12):823-826.
Woodward GA: Neck trauma. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1376-1421.

25. What are the main differences between anterior cord syndrome, central cord syndrome, Brown-Séquard syndrome, and complete cord syndrome?

Anterior cord syndrome is due to hyperflexion injuries and anterior cord compression. Compression of the anterior cord involves injury to the spinothalamic tract, which is responsible for motor function and the body's response to pain, light touch, and temperature sensation. Injury of the anterior cord results in paralysis and loss of pain, light touch, and temperature sensation below the level of vertebral injury. Proprioceptive functions, pain, and vibratory sense remain intact.

Central cord syndrome is usually seen in older patients with degenerative spinal disease; however, it can be seen in children with hyperextension injuries. Injury of the central cord results in weakness that is greater in the upper extremities in comparison to the lower extremities. Patients can also have a transient burning sensation in their hands.

Brown-Séquard syndrome is caused by hemisection of the spinal cord, most often secondary to penetrating trauma. Injury to this area results in contralateral loss of pain and temperature sensation with ipsilateral loss of motor function, light touch sensation, and proprioception below the level of vertebral injury.

Complete cord syndrome is due to complete transection of the spinal cord, most often from penetrating or blunt trauma. It results in complete loss of all neurologic function below the level of injury. This type of injury is most often responsible for neurogenic shock.

Jagannathan J, Dumont AS, Prevedello DM, et al: Cervical spine injuries in pediatric athletes: Mechanisms and management. Neurosurg Focus 2006;21(4):1-5.
Saleh J, Raycroft JF: Hyperflexion injury of cervical spine and central cord syndrome in a child. Spine 1992;17(2):234-237.
Wagner R, Jagoda A: Spinal cord syndromes. Emerg Med Clin North Am 1997;15(3):699-711.
Woodward GA: Neck trauma. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1376-1421.

CHAPTER 60

PELVIC TRAUMA AND GENITOURINARY INJURY

Timothy Brenkert and Javier A. Gonzalez del Rey

1. Why are children more likely to sustain renal injuries than adults?

A child's kidneys are disproportionately larger in relation to body size and have less perinephric fat, more lobulations, and a more intra-abdominal location. Because children have a weaker anterior abdominal wall and a less well ossified thoracic cage with a more flexible thoracolumbar spine, the kidneys and other genitourinary structures are more susceptible to blunt trauma. Preexisting congenital renal abnormalities have been documented in up to 20% of patients evaluated for renal trauma. Approximately 10% of trauma patients have urogenital injuries. Common associated injuries include head injuries, fractures (extremities, pelvis, ribs, spine, and skull), spinal cord injuries, and liver or spleen injuries. Blunt trauma accounts for up to 90% of renal injuries.

Brown SL, Elder JS, Spirnak JP: Are pediatric patients more susceptible to major renal injury from blunt trauma? A comparative study. J Urol 1998;160:138-140.

2. Describe the immediate and delayed complications of renal trauma.

Contusions and minor cortical lacerations usually heal without sequelae. More severe injuries may be associated with delayed bleeding, renal failure, abscess formation, urinary extravasation with sepsis, and ureteral obstruction secondary to clot formation. Late complications include hypertension, arteriovenous fistulas, hydronephrosis, pseudocyst formation, and renal calculi. Children with renal trauma (radiologically or operatively confirmed) should be followed closely with serial scans for at least 1 year after injury to evaluate renal anatomy and to ensure prompt diagnosis and treatment of possible complications.

Garcia CT, Thompson VT: Genitourinary trauma. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010.

3. Which is the cardinal laboratory marker of renal injury?

Hematuria is the most important laboratory evidence of renal injury. Gross hematuria is the hallmark sign of severe injury. However, this finding may be absent in up to 50% of patients with vascular pedicle injuries and in 29% of patients with penetrating injuries. Radiographic evaluation is needed for children with gross hematuria, microscopic hematuria of more than 50 red blood cells per high-powered field (RBCs/HPF) in blunt trauma or more than 5 RBCs/HPF in penetrating trauma, microscopic hematuria with shock, or clinical findings indicative of renal trauma.

Buckley JC, McAninch JW: Pediatric renal injuries: Management guidelines from a 25-year experience. J Urol 2004;172:687-690.

4. How are renal injuries classified?

The Organ Injury Scaling Committee of the American Association for Surgery of Trauma developed a scaling system that unifies previous classifications for research purposes:

- **Grade I:** Contusions or nonexpanding subcapsular hematomas with associated hematuria
- **Grade II:** Hematomas confined to the retroperitoneum or lacerations smaller than 1 cm in depth without urinary extravasation
- **Grade III:** Lacerations extending into the perinephric fat larger than 1 cm in depth without involvement of the collecting system or extravasation
- **Grade IV:** Deep lacerations into the collecting system and vascular injuries with contained hemorrhage
- **Grade V:** Fractured or completely shattered kidneys and avulsion of the renal pedicle with organ devascularization

Tinkoff G, Esposito TJ, Reed J, et al: American Association for the Surgery of Trauma Organ Injury Scale 1: Spleen, liver, and kidney, validation based on the National Trauma Data Bank. J Am Coll Surg 2008;207 (5):646-655.

5. Which imaging modality is considered the diagnostic test of choice for evaluation of renal injuries in stable pediatric patients?

Contrast-enhanced computed tomography (CT) is considered the diagnostic test of choice for detection of renal injury in stable pediatric patients with trauma. It not only delineates the degree of renal parenchymal injury with high accuracy but also assesses other intra-abdominal, retroperitoneal, and pelvic injuries simultaneously. However, this test is relatively expensive, subjects the child to radiation, and requires a hemodynamically stable patient who can tolerate transport to the radiology suite. CT is useful for identifying genitourinary injury in children with penetrating trauma to the abdomen or other significant injuries.

Smith JK, Kenney PJ: Imaging of renal trauma. Radiol Clin North Am 2003;41(5):1019-1035.

Key Points: Indications for Radiologic Evaluation of the Genitourinary Tract in Pediatric Trauma

1. Gross hematuria
2. Hematuria of more than 50 RBCs/HPF in blunt trauma or more than 5 RBCs/HPF in penetrating trauma
3. Microscopic hematuria with shock or clinical findings indicative of renal trauma

6. Which diagnostic modality can be used in unstable patients?

Consider intravenous (IV) pyelography for the evaluation of renal trauma or isolated urogenital trauma in unstable patients. This study is rarely used now, but it is available in most institutions, is relatively inexpensive, and provides evidence of renal function (including contralateral function in patients with penetrating injuries) and visualization of the calyceal system or extravasation. One-shot IV pyelography can be performed in the emergency department or operating room, so it is useful for patients too unstable to go for a CT scan.

Stevenson J, Battistella FD: The "one-shot" intravenous pyelogram: Is it indicated in unstable trauma patients before celiotomy? J Trauma 1994;36:828-833; discussion, 833-834.

7. Should IV pyelography be used routinely to evaluate patients with possible renal injury?

No. Intravenous pyelography may give false-negative results in patients with pedicle injuries and correlates in only 50% to 75% of patients with injuries found at exploratory laparotomy. Owing to the wide availability of CT and a low sensitivity of IV pyelography in detecting and characterizing injuries, IV pyelography no longer has a place in the routine evaluation of the trauma patient. Its use has been relegated to situations in which CT may not be available or as an intraoperative adjunct.

Ramchandani P, Buckler PM: Imaging of genitourinary trauma. Am J Roentgenol 2009;192(6):1514-1523.
Stevenson J, Battistella FD: The "one-shot" intravenous pyelogram: Is it indicated in unstable trauma patients before celiotomy? J Trauma 1994;36:828-833; discussion, 833-834.

8. Which are the most commonly found symptoms in bladder injuries?

Although symptoms may be nonspecific, those most commonly found with bladder injuries include hematuria, suprapubic or abdominal pain, and tenderness and difficulty with voiding or inability to void. During childhood, the bladder has a higher abdominal location, which renders the organ more susceptible to injury. Radiologic evaluation is indicated in patients with pelvic or lower abdominal trauma who experience difficulty in voiding or gross hematuria. However, avoid catheterization if physical examination reveals blood at the urethra or a high-riding prostate on rectal examination.

Guttmann I, Kerr HA: Blunt bladder injury. Clin Sports Med 2013;32:239-246.

9. What clinical findings should make you suspect ureteral injury?

Signs and symptoms of ureteral injuries are nonspecific and often overlooked. Fewer than 50% of patients are diagnosed within 24 hours because the presentation may develop gradually. An enlarging flank mass in the absence of retroperitoneal bleeding suggests the possibility of urinary extravasation. Hematuria is not a reliable sign because it may be absent in more than 40% of patients with ureteral injuries. Common symptoms in delayed presentation include

fever, chills, ileus, lethargy, pyuria or bacteriuria, and flank pain. Ureteral injuries usually are discovered during evaluation of other traumatic injuries.

10. Describe the most common mechanisms for ureteral injuries.

Ureteral injuries are uncommon in children, accounting for less than 1% to 3% of patients with genitourinary tract injury. They may result from blunt trauma (involving the ureteropelvic junction) or penetrating trauma (gunshot wounds), but most ureteral injuries are iatrogenic (resulting from urologic, gynecologic, colonic, and vascular surgery procedures). Iatrogenic injury is less common in children than adults because of the nature of the procedures and easier identification of the ureters because of lack of retroperitoneal fat.

Tarman GJ, Kaplan GW, Lerman SL, et al: Lower genitourinary injury and pelvic fractures in pediatric patients. Urology 2002;59:123-126.

11. What clinical findings indicate urethral injuries?

The presence of blood at the meatus has been reported in more than 90% of patients with anterior urethral injury. Such injuries are commonly due to motor vehicle crashes, straddle injuries (bulbar urethra), or instrumentation. Patients may present with hematuria, difficulty or inability to void, swelling, and ecchymosis or hematoma of the perineum or penis. Anterior urethral injuries may produce extravasation of blood and urine into the abdominal wall, scrotum, or perineum.

12. What are the indications for urethrography in pediatric trauma patients?

Retrograde urethrography is the radiologic method of choice for the diagnosis of urethral injuries. It should be performed in any child with penetrating trauma and suspected genitourinary injuries; perineal trauma with hematuria; inability to void or to advance a urinary catheter; vaginal laceration or bleeding; swelling, hematoma, or ecchymosis of the perineum; blood at the urethral meatus; and high-riding or boggy prostate.

Avery LL, Scheinfeld MH: Imaging of male pelvic trauma. Radiol Clin North Am 2012;50(6):1201-1217.

Key Points: Indications for Retrograde Urethrography in Pediatric Trauma Patients

1. Perineal trauma (including swelling, hematoma, or ecchymosis) with hematuria
2. Inability to void or to advance a urinary catheter
3. Vaginal laceration or bleeding
4. Blood at the urethral meatus
5. High-riding or boggy prostate

13. How does traumatic testicular dislocation present?

Forceful displacement of the testicle from its anatomic position is uncommon in children because of their brisk cremasteric reflex; however, a straddle-type injury may produce enough force to dislocate the testicle. Patients may present with nausea, vomiting, scrotal pain, and absence of the testicle in the involved hemiscrotum or a testis palpated in another location. The dislocated testicle may be found in various ectopic locations: superior inguinal, pubic, abdominal, penile, acetabular, or perineal. Associated injuries, such as pelvic fractures, are common.

14. How does testicular rupture present?

The tunica albuginea ruptures, and testicular contents extravasate into the scrotal sac. This is a surgical emergency, because there is a higher chance of testicular salvage if exploration is done within 24 hours after the injury.

15. Describe the management for zipper entrapment of the penis or foreskin.

Numerous methods to release zipper entrapment of the foreskin have been utilized. Most commonly described is the use of bone or wire cutters. The tool is used to split the median bar of the zipper fastener, thus allowing the two sides of the zipper to release the entrapped tissue (Fig. 60-1). Alternatively, mineral oil can be liberally applied to the involved tissue and allowed to soak for 15 minutes. The trapped skin may then be easily released. Additionally, the head of a small flat-head screwdriver may be inserted between faceplates of the fastener opposite the median bar from the site of entrapped tissue. With a rotating motion the faceplates

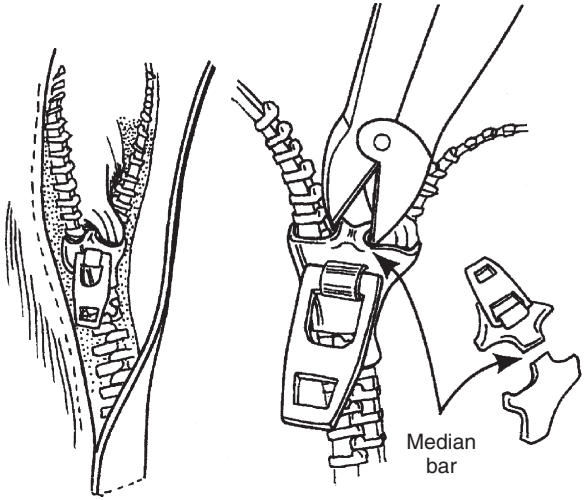


Figure 60-1. Release of entrapped foreskin from zipper.

can be separated from one another, releasing the injured tissue. Warm soaks may be used after the procedure to control edema. Some children may require sedation or local anesthetics to assist with pain control.

Fein J, Zderic SA: Management of zipper injuries. In Henretig FM, King C (eds): Textbook of Pediatric Emergency Procedures, Baltimore, Williams & Wilkins, 2007.

Raveenthiran V: Releasing of zipper-entrapped foreskin: A novel nonsurgical technique. Pediatr Emerg Care 2007;23(7):463-464.

16. Is a pelvic fracture predictive of a genitourinary injury in children?

A review of the literature has found that associated genitourinary injury is seen in 11% to 12% of pediatric pelvic fractures. This incidence increases to 40% to 50% with multiple fractures to the pelvic ring. The presence of multiple fractures is also the best predictor of abdominal injury in these patients.

Gansslen A, Hildebrand F, Heidari N, Weinberg AM: Pelvic ring injuries in children. Part 1: Epidemiology and primary evaluation. Acta Chir Orthop Traumatol Cech 2012;79(6):493-498.

17. What are the most common diagnostic findings in children with pelvic fractures?

Most patients with pelvic fractures have a history of a major mechanism of injury and associated multiple trauma. Some patients present with unstable vital signs due to internal hemorrhage and massive retroperitoneal hematomas. This presentation is most common in older children and adults. Pain and instability elicited by pelvic rocking (while holding the anterior iliac spines) suggest fractures in the pelvic ring. Pressure over the pubis also may produce pain and crepitus. Pelvic tenderness is by far the most common physical examination finding in patients with pelvic fractures (73%). Iliac contusions (30%) or abrasions (15%) are also frequently seen. Other findings, such as obturator, femoral, and sciatic nerve injuries, are infrequent in children because of the flexibility of the pelvic ring.

Junkins EP, Furnival RA, Bolte RG: The clinical presentation of pediatric pelvic fractures. Pediatr Emerg Care 2001;17(1):15-18.

18. When is CT indicated in pediatric pelvic fractures?

Plain radiographs have limited sensitivity for detecting pediatric pelvic fractures (54%). Consider a CT scan for further evaluation of pelvic ring fractures seen on radiographs in order to better assess the extent of the fracture and to evaluate the presence of associated injuries.

Guillamondegui OD, Mahboubi S, Stafford PW, et al: The utility of the pelvic radiograph in the assessment of pediatric pelvic fractures. J Trauma 2003;55(2):236-239; discussion, 239-240.

19. What musculoskeletal differences account for the different injury pattern seen in children versus adults?

Pediatric bones have more pliability and contain a thicker periosteum when compared to adult bones. Additionally, pediatric ligaments and tendons are relatively stronger than their adjacent bones and apophyses, respectively. These differences contribute to a higher incidence of avulsion fractures in the pelvis as well as increased isolated pelvic ring fractures when compared to adults.

20. Describe the most common mechanisms causing blunt perineal injury in females.

Due to the infrequent occurrence of such injuries in pediatric multisystem trauma patients, the diagnosis of perineal injury is often overlooked. Following compression against the bony pelvis, blunt trauma to the perineum can result in a wide array of urogenital injuries. Motor vehicle collisions result in the majority of blunt perineal injuries in the adolescent age group, causing up to 92% of blunt perineal injuries seen in patients 15 to 16 years old. Straddle injuries, when the subject straddles a long, horizontal object such as a bicycle bar or the ledge of a pool or bathtub, predominate in the younger child, accounting for more than 80% of cases in children 1 to 14 years old. Sexual assault is another mechanism. It's incidence is highest in children 0 to 4 years of age and is responsible for 17% of blunt perineal injuries in this population.

Scheidler MG, Shultz BL, Schall L, et al: Mechanisms of blunt perineal injury in pediatric patients. J Pediatr Surg 2000;35:1317-1319.

Spitzer RF, Kives S, Caccia N, et al: Retrospective review of unintentional female genital trauma at a pediatric referral center. Pediatr Emerg Care 2008;24:831-835.

21. When should I consider the possibility of sexual abuse in a child with a vaginal injury?

Young girls frequently suffer nonintentional straddle injuries from a bicycle or a fall. However, injury to the perineum in which the history fails to explain the findings, a history of possible abuse reported by the child or another source, or the presence of other suspicious injuries on physical examination obligates a report to appropriate agencies.

22. Describe the management of a 9-year-old female with pain and bleeding of the genitalia following a straddle injury on a bicycle.

The evaluation of a child with genital trauma can frequently be limited secondary to fear and pain experienced by the patient. Often these patients require more thorough examination under sedation or anesthesia. The external genitalia and unestrogenized mucosa of these patients are highly vascularized, with profuse bleeding seen after even minor injuries. Initially, focus on identification of external injuries and assessment and control of active bleeding. Straddle injuries typically result in vulvar trauma as the soft tissues are compressed between the object and the pubic symphysis and pubic rami. Hematomas are usually self-limited and can be managed with local ice packs and bed rest as long as the patient can demonstrate the ability to void. If urination is obstructed, place a Foley catheter until swelling has sufficiently improved. (Consider topical anesthetic gels for catheter placement to minimize pain.) Manage minor abrasions and lacerations conservatively due to rapid healing of the tissues. Consult a surgical subspecialist for possible further evaluation and repair in the operating room if there is any evidence of vaginal bleeding, anorectal involvement, or inability to obtain hemostasis of a vulvar laceration.

Jones JG, Worthington T: Genital and anal injuries requiring surgical repair in females less than 21 years of age. J Pediatr Adolesc Gynecol 2008;21:207-211.

Merritt DF: Genital trauma. In Altcheck A, Deligdisch L (eds): Pediatric, Adolescent and Young Adult Gynecology. Hoboken, NJ, John Wiley & Sons, 2009.

PEDIATRIC SPORTS INJURIES

Maria Carmen G. Diaz

CHAPTER 61

1. Which sports are associated with the highest injury rates in children, and what are the most common types of injuries seen?

In boys, most injuries are seen with football. In girls, soccer accounts for the highest injury rates. Other sport activities that commonly lead to the emergency department evaluation of pediatric injuries include basketball, baseball/softball, rollerblading, and hockey.

The most common types of acute injuries are soft tissue injuries, including sprains, strains, and contusions. Fractures and lacerations occur less frequently. However, in adolescents, overuse injuries are more common than acute injuries.

Patel DR, Nelson TL: Sports injuries in adolescents. Med Clin North Am 2000;84:983-1007.
Taylor BL, Attia MW: Sports-related injuries in children. Acad Emerg Med 2000;7:1376-1382.

Key Points: Acute Sports Injuries

1. Patients with concussions require complete cognitive and physical rest.
2. Children are more prone to commotio cordis than adults because of their thin, flexible chest wall.
3. Shoulders most commonly dislocate anteriorly.
4. Pelvic avulsion fractures result from sudden muscular contractions and are usually associated with vigorous running or jumping.

2. What are overuse injuries and how are they managed?

Overuse injuries are chronic injuries that are related to constant high levels of physiologic stress without sufficient recovery time. These injuries are characterized by repetitive microtrauma and the development of inflammation, leading to significant pain and loss or limitation of function. Overuse injuries are treated with rest and supportive bracing. Additionally, encourage the athlete to partake in proper training regimens that emphasize flexibility and stretching.

Brenner JS: Overuse injuries, overtraining, and burnout in child and adolescent athletes. Pediatrics 2007;119:1242-1245.

Key Points: Overuse Injuries

1. Overuse injuries are injuries that occur due to repetitive stress or trauma to an area that has had inadequate time for recovery between injuries.
2. Rest and proper conditioning regimens are crucial elements in the management of overuse injuries.
3. Overuse injuries are the most common sports injuries seen in adolescents.
4. Osgood-Schlatter disease is the most common overuse injury in young athletes.

3. What is little league shoulder and how does it present?

Little league shoulder, also known as proximal humeral epiphysitis, is most often seen in high-performance pitchers between 11 and 16 years of age. It may also be seen in tennis players, swimmers, and gymnasts. It is caused by overuse and subsequent inflammation of the proximal humeral physis. The chief complaint is gradual pain localized to the proximal humerus during the act of throwing or serving. The physical examination may be negative, or there may be tenderness to palpation of the proximal humerus. Radiographs may reveal a widening of the proximal humeral physis. Treatment is almost always nonsurgical and includes a 3-month rest period and physical therapy.

Osbahr DC, Kim HJ, Dugas JR: Little league shoulder. Curr Opin Pediatr 2010;22:35-40.

4. What is little league elbow?

Little league elbow, or medial epicondylar apophysitis, results from stress to the medial epicondyle of the humerus. It is most commonly seen in children between the ages of 9 and 12 years who are baseball pitchers, infielders, or tennis players. An excessive number of pitches thrown and a sidearm pitching style have both been implicated as causes. The athlete presents with gradual onset of pain in the medial elbow and proximal forearm while throwing.

Radiographs may be normal or reveal medial epicondylar apophyseal widening. Treatment is complete rest from throwing activities, ice, nonsteroidal anti-inflammatory drugs (NSAIDs), and physical therapy. After the 4- to 6-week rest period, asymptomatic athletes may participate in a 4- to 8-week progressive throwing program.

Greiwe RM, Saifi C, Ahmad CS: Pediatric sports elbow injuries. Clin Sports Med 2010;29:677-703.

5. Name the two types of apophysitis syndromes that occur surrounding the patellar tendon.

1. Osgood-Schlatter disease—an apophysitis at the anterior tibial tubercle due to traction by the inferior aspect of the patellar tendon.
2. Sinding-Larsen-Johansson disease—an apophysitis of the inferior pole of the patella due to traction by the superior aspect of the patellar tendon. Treatment is analogous to Osgood-Schlatter disease. Sinding-Larsen-Johansson disease is differentiated from "jumper's knee" in that jumper's knee is not an apophysitis, but rather an inflammation within the proximal patellar tendon itself.

Soprano JV, Fuchs SM: Common overuse injuries in the pediatric and adolescent athlete. Clin Pediatr Emerg Med 2007;8:7-14.

6. Which is the most common overuse injury in the young athlete?

Osgood-Schlatter disease, which is an apophysitis at the anterior tibial tubercle, commonly affects adolescents during periods of rapid growth. Symptoms include tenderness and a prominence at the tibial tubercle, with pain worsened by high impact sports, kneeling, or squatting. Plain radiographs will demonstrate enlargement or fragmentation at the anterior tibial tubercle due to the traction force at the insertion of the patellar tendon. Management includes ice, NSAIDs, and stretching regimens to increase flexibility of the quadriceps and hamstrings. The problem usually resolves with closure of the physis, but children may rarely develop ossicles within the patellar tendon that require surgical removal.

Adirim TA, Cheng TL: Overview of injuries in the young athlete. Sports Med 2003;33(1):75-81.

7. Do you need radiographs to diagnose Osgood-Schlatter disease?

Generally, no. Osgood-Schlatter disease is usually a clinical diagnosis. However, if there are unilateral findings, consider other serious conditions such as osteogenic sarcoma. This malignancy presents in young teens, like Osgood-Schlatter disease, and radiographs will be reassuring if normal. Bilateral malignancy is very rare, so Osgood-Schlatter disease can be diagnosed without radiographs in most cases when the child has bilateral complaints.

8. What is the recommended management for Sever's disease?

Sever's disease, an apophysitis resulting from repetitive stresses on the calcaneal ossification center by the Achilles tendon, is often seen in young runners. The athlete will complain of heel pain and often has had a recent growth spurt and increase in activity level. Management includes rest, NSAIDs, Achilles tendon stretching, and the use of heel cups in cleats or sneakers. It usually resolves in 2 to 4 months' time without the need for surgery.

Ganley TJ, Lou JE, Pryor K, Gregg JR: Sports medicine. In Dormans JP, Bell LM (eds): Pediatric Orthopedics and Sports Medicine: The Requisites in Pediatrics. St. Louis, Mosby, 2004, p 280.

Simons SM, Sloan BK: Foot injuries. In Birrer RB, Griesemer BA, Cataletto MB (eds): Pediatric Sports Medicine for Primary Care. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2002, pp 443-444.

9. List the most common locations for stress fractures in pediatric athletes.

- Tibia (51%)
- Fibula (20%)
- Pars interarticularis (15%)
- Femur (3%)
- Metatarsal (2%)
- Tarsal navicular (2%)

Coady CM, Micheli LJ: Stress fractures in the pediatric athlete. Clin Sports Med 1997;16:225-238.

10. In which sports are participants most likely to sustain stress fractures?

- Running
- Basketball
- Gymnastics
- Football
- Ice skating

Saperstein AL, Nicholas SJ: Pediatric and adolescent sports medicine. Pediatr Clin North Am 1996;43:1013-1033.

11. Describe the most likely diagnosis in a gymnast who complains of gradual onset, recurrent, activity-associated midline low back pain.

The most likely diagnosis is spondylolysis, a defect of the pars interarticularis that occurs with repetitive axial loading of the lumbar spine. The most common site is at L5. Radiographs may be normal or may show a pars defect in the oblique view often described as a "Scotty dog with a collar" appearance (Fig. 61-1). Treatment requires restriction of activity and an aggressive rehabilitation regimen that includes abdominal strengthening and flexion exercises. Bracing is reserved for athletes who are unresponsive to these measures.

Haus BM, Micheli LJ: Back pain in the pediatric and adolescent athlete. Clin Sports Med 2012;31:423-440.

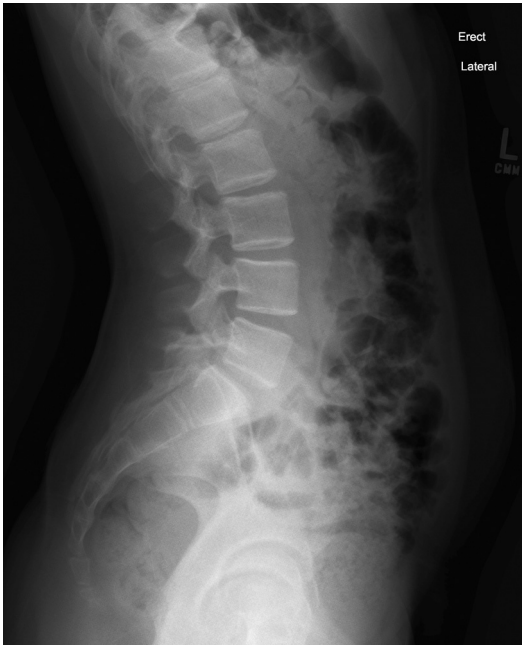


Figure 61-1. Spondylolysis, a pars interarticularis defect ("Scotty dog with a collar").

12. You diagnose a teenage football player with his first concussion. When may he return to play?

A patient with a concussion requires complete cognitive and physical rest. Because each patient follows a variable time course to recovery, make individualized plans for stepwise return to play. The patient should be symptom free before beginning the return to play stepwise protocol (Table 61-1). Patients may proceed to the next level when asymptomatic at the current level. If symptoms recur, then the patient should go back to the previous asymptomatic step and reattempt progression after 24 hours of rest. The patient should have a normal physical examination and be asymptomatic at rest and with exertion before returning to play.

Patel DR, Reddy V: Sports related concussions in adolescents. Pediatr Clin North Am 2010;57:649-670.

Table 61-1. Return to Play Stepwise Protocol

1. No activity; complete physical and cognitive rest
2. Light aerobic exercise (walking, stationary cycling, keeping intensity <70% maximal predicted heart rate, and no resistance training)
3. Sport-specific exercise (skating in hockey, running in soccer)
4. Noncontact training drills (progression to more complex training drills, e.g., passing drills in football; may start resistance training)
5. Full-contact practice following medical clearance
6. Return to unrestricted sport participation

Patel DR, Reddy V: Sports related concussions in adolescents. Pediatr Clin North Am 2010;57:649-670.

13. What is the most dreaded complication of returning to sports too soon following concussion?

Second impact syndrome. This results from acute brain swelling when a second head trauma is sustained prior to full recovery from an initial concussion. Second impact syndrome is thought to occur because the athlete reinjures the head during a period of disordered cerebral autoregulation following the initial head injury. Second impact syndrome, though quite rare, is not treatable and is often fatal.

Cantu RC: Second impact syndrome. Clin Sports Med 1998;17(1):37-44.
Patel DR, Reddy V: Sports related concussions in adolescents. Pediatr Clin North Am 2010;57:649-670.

14. Does the occurrence of a seizure directly after head trauma increase the risk of intracranial injury?

Posttraumatic seizure (PTS) can be divided into three categories based on when it occurs in relation to the injury: immediate (within seconds), early (within 1 week), and late (>1 week). Most PTSs occur within 24 hours of injury, and they substantially increase the risk of intracranial injury. Obtain computed tomography (CT) of the head for these patients to evaluate for traumatic brain injury. There is debate as to whether children with negative CT scans and normal neurologic examinations may be safely discharged to home. Immediate PTSs rarely recur, whereas early PTSs and late PTSs are more likely to recur.

Holmes JF, Palchak MS, Conklin MS, Kupperman N: Do children require hospitalization after immediate post-traumatic seizure? Ann Emerg Med 2004;43(6):706-710.

15. What is commotio cordis?

Commotio cordis is a sudden cardiac arrest in a patient without preexisting heart disease following a relatively minor blow to the chest. The pathophysiology involves the mechanical force of the blow leading to a depolarization of the myocardium on the T wave of the cardiac cycle, precipitating cardiac arrest. It is the second leading cause of sudden death in the young athlete and occurs most frequently in the sport of baseball. In animal models, commotio cordis is seen most often with balls thrown between 30 and 50 miles per hour, the speed of most Little League pitches. Chest protectors and softer-core balls have been marketed in an attempt at prevention, but most leagues do not mandate their use.

Maron BJ: Sudden death in young athletes. N Engl J Med 2003;349:1064-1075.

16. Why are children at greater risk for commotio cordis than adults?

Children are more vulnerable than adults because of their thin and flexible chest walls and their lower agility.

17. What is the most common cause of sudden death in the young athlete?

Hypertrophic cardiomyopathy (HCM). This is a congenital heart deformity resulting in left ventricular outflow obstruction with asymmetric septal hypertrophy. It predisposes the athlete to arrhythmias that are likely to cause syncope or death. Unfortunately, most athletes are asymptomatic before the development of the lethal event and preparticipation screening examinations (even with electrocardiography) may be normal.

Maron BJ, Epstein SE, Roberts WC: Causes of sudden death in competitive athletes. J Am Coll Cardiol 1986;7(1):204-214.

18. What are some clinical characteristics of burners/stingers?

A stinger is a brachial plexus injury following a traumatic stretching or compression force on the neck. Symptoms include unilateral arm pain or burning, especially in the C5 to C6 nerve root distribution. There may be associated weakness. The symptoms often resolve within minutes but may persist. Of note, if the symptoms are persistent, bilateral, involve the lower extremities, or are accompanied by neck pain, more significant spinal cord injury may have occurred.

Maron BJ, Epstein SE, Roberts WC: Causes of sudden death in competitive athletes. J Am Coll Cardiol 1986;7(1):204-214.

19. What is the most commonly injured nerve in acute shoulder dislocation?

The axillary nerve. This nerve innervates the upper arm and must be carefully examined with acute shoulder dislocation. Shoulder dislocation is most likely to occur when the arm is abducted and externally rotated, is far more likely to be dislocated anteriorly than posteriorly, and is often recurrent.

Ganley TJ, Lou JE, Pryor K, Gregg JR: Sports medicine. In Dormans JP, Bell LM (eds): Pediatric Orthopedics and Sports Medicine: The Requisites in Pediatrics. St. Louis, Mosby, 2004, pp 283-284.

20. Describe the mechanism of injury in a gamekeeper's thumb.

Gamekeeper's thumb, also known as skier's thumb, is a sprain of the ulnar collateral ligament of the metacarpophalangeal joint of the thumb. The injury is the result of a forced abduction and extension of the thumb that occurs when falling on an outstretched hand or colliding with an object. These patients exhibit point tenderness over the ulnar collateral ligament and have no radiographic findings. Treatment includes immobilization and compression with a thumb spica splint. If the patient has an open physis, this mechanism of injury will produce a Salter III fracture of the proximal phalanx.

Bielak KM, Kafka J, Terrell T: Treatment of hand and wrist injuries. Prim Care Clin Office Pract 2013;40:431-451.

21. What is the difference between a mallet finger and a jersey finger?

The difference between a mallet finger and a jersey finger is shown in Table 61-2.

Bielak KM, Kafka J, Terrell T: Treatment of hand and wrist injuries. Prim Care Clin Office Pract 2013;40:431-451.

22. How do pelvic avulsion fractures occur?

Avulsion fractures result from sudden muscular contractions and are usually associated with vigorous running or jumping. These are seen in sports that require rapid acceleration or deceleration, or quick changes of direction. Consider this condition when a well-conditioned, muscular athlete falls to the ground, writhing in pain, during a race or competition.

Table 61-2. Mallet Finger Versus Jersey Finger

FEATURE FOR COMPARISON	MALLET FINGER	JERSEY FINGER
Injury	Extensor digitorum tendon disruption	Flexor digitorum tendon disruption
Physical examination	Forced flexion of an extended distal interphalangeal joint	Forced extension of a flexed distal interphalageal joint
Mechanisms	Finger struck on the tip by a ball	Football player grabs another player's jersey
	Forcefully tucking in a bedspread	Lifting a latch on a car door
	Pushing off a sock with an extended finger	

23. What are the most common locations of pelvic avulsion fractures, and what muscle attachment contributes to the fracture?

An image of a pelvic avulsion fracture can be seen in Figure 61-2, and the muscle attachment that contributes to the fracture is in Table 61-3.

24. A running football player was struck in the thigh by another player's helmet. Radiographs are negative, yet the patient has marked pain at the site of the injury and with ambulation. What is the diagnosis and what treatment should be initiated?

This patient probably sustained a quadriceps contusion. Initial treatment involves halting the hemorrhage and preventing spasm by rest, ice, compression, and elevation for 24 to 48 hours. Bracing with 90 degrees of knee flexion may be needed in cases of severe swelling. The patient should then undergo progressive isometric strengthening and active range-of-motion exercises.

Avoid vigorous massage and passive stretching, because both have been associated with myositis ossificans, a condition in which heterotopic bone growth occurs within the quadriceps muscle.

25. What is the typical mechanism for an injury to the anterior cruciate ligament (ACL) in children?

As with adult patients, injury to the ACL results from a sudden deceleration accompanied by hyperextension and a rotatory force on a planted foot. This is often accompanied by a popping sensation and the acute onset of hemarthrosis. Whereas adults and older adolescents will

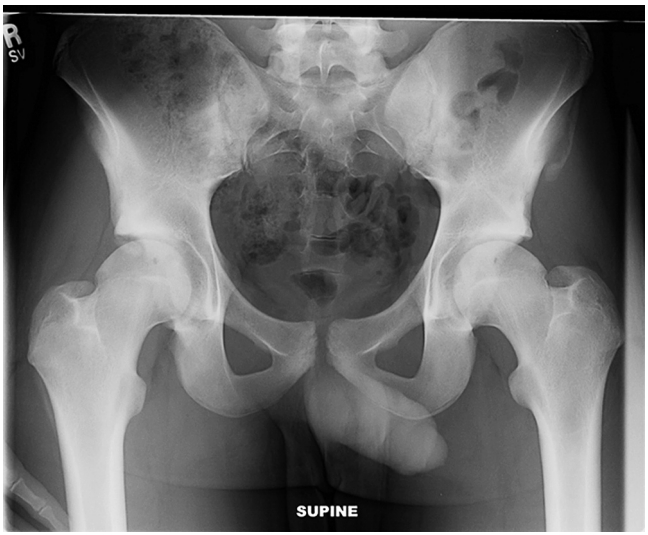


Figure 61-2. Image of pelvic avulsion fracture, left anterior superior iliac spine.

Table 61-3. Pelvic Avulsion Fractures

LOCATION	INVOLVED MUSCLE
Ischial tuberosity	Hamstrings and adductors
Anterior superior iliac spine	Sartorius
Anterior inferior iliac spine	Rectus femoris
Lesser trochanter of the femur	Iliopsoas

probably tear the ligament itself, younger children may avulse a bony segment of tibia at the ACL insertion site. Plain radiography followed by magnetic resonance imaging is used to delineate the specifics of the injury. Management of the ACL injury is complicated by the open physes in skeletally immature children, requiring careful consideration of operative versus nonoperative treatment plans by the orthopedist.

Frank JS, Gambacorta PL: Anterior cruciate ligament injuries in the skeletally immature athlete: Diagnosis and management. J Am Acad Orthop Surg 2013;21:78-87.

26. What is the rate of recurrence of acute patellar dislocation?

Approximately 1 in 6 pediatric patients with patellar dislocation will be affected by recurrent patellar instability. The patient most prone to recurrence will have an increased Q angle. The Q angle is created by two lines drawn from the anterior superior iliac spine to the center of the patella, and from the center of the patella to the center of the tibial tubercle. Q angles greater than 20 degrees predispose to instability by increasing the lateral force of the patella during knee extension.

Ganley TJ, Lou JE, Pryor K, Gregg JR: Sports medicine. In Dormans JP, Bell LM (eds): Pediatric Orthopedics and Sports Medicine: The Requisites in Pediatrics. St. Louis, Mosby, 2004, pp 285-287.

27. What are the limitations to the Ottawa Ankle Rules in children?

The Ottawa Ankle Rules are criteria developed for predicting ankle fractures based on examination features in adults with the goal of reducing unnecessary radiographs. They allow, however, for "insignificant fractures" (such as small avulsion fractures) to be undiagnosed. They are not universally applicable to the pediatric population due to the questionable significance of Salter I and avulsion fractures on children's future growth and development. A 2003 meta-analysis of 15,581 adults and children from 27 studies demonstrated an overall sensitivity and specificity for the Ottawa Ankle Rules of 97.6% and 31.5%, respectively. In a 2009 pooled analysis of 12 studies representing 3130 children with 671 fractures, the authors reported a sensitivity of 98.5% and a negative likelihood ratio of 0.11. The data estimated a missed fracture rate of 1.2% in children.

Bachmann LM, Kolb E, Koller MT, et al: Accuracy of Ottawa Ankle Rules to exclude fractures of the ankle and mid-foot: Systematic review. BMJ 2003;326:417.

Dowling S, Spooner CH, Liang Y, et al: Accuracy of Ottawa Ankle Rules to exclude fractures of the ankle and midfoot in children: A meta-analysis. Acad Emerg Med 2009;16:277-287.

28. What are some unique characteristics of young athletes that make them more prone to heat-related illness than adults?

The larger body surface area allows children to absorb more external heat, and they have a limited ability to produce sweat as compared to adults. They are also less able to cognitively appreciate the need to adequately hydrate and may not ask for hydration breaks often enough. Other risk factors for heat illness include obesity, dehydration, deliberate water restriction in order to "make weight," use of laxatives, practices early in the season without adequate acclimatization, and the use of dark clothing or heavy equipment such as padding.

Gambrell RC: Environmental conditions and youth sports. In Birrer RB, Griesemer BA, Cataletto MB (eds): Pediatric Sports Medicine for Primary Care. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2002, pp 95-96.

29. What measures can be used to prevent the occurrence of pediatric sports injuries?

A thorough preparticipation sports evaluation by a physician can detect potential risks and determine possible medical contraindications to participation in certain sports.

Start a carefully planned conditioning regimen 6 to 8 weeks before the athletic season begins. Emphasize overall fitness rather than focusing on skills specific to only one athletic activity. The athletes should slowly adapt to increases in intensity and frequency of exercise. Equipment and playing sites must be of appropriate size and in satisfactory condition.

Carter CW, Micheli LJ: Training the child athlete for prevention, health promotion, and performance: How much is enough, how much is too much? Clin Sports Med 2011;30:679-690.

Acknowledgment

The authors wish to thank Dr. Lauren Daly for her contributions to this chapter in the previous edition.

Website

Centers for Disease Control and Prevention: http://www.cdc.gov/concussion/headsup.

CHAPTER 62

THORACIC TRAUMA

Howard Kadish

1. How common are thoracic injuries and why are they important?

Among injured children, thoracic trauma is uncommon. About 4% to 8% of pediatric trauma victims have a thoracic injury. Blunt mechanisms account for 85% of these injuries. Thoracic injuries are important because the mortality rate is high (15-26%) for children with such trauma.

2. What are the most common injuries in blunt and penetrating thoracic trauma?

- **Blunt thoracic trauma:** Lung contusions (53%), pneumothorax or hemothorax (38%), and rib fractures (38%)
- **Penetrating thoracic trauma:** Pneumothorax or hemothorax (64%), diaphragmatic lacerations (15%), cardiac injuries (13%), and vascular injuries (10%)

Kadish HA: Thoracic trauma. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1459-1477.

3. How does thoracic trauma differ in children and adults?

Pediatric thoracic trauma differs from adult thoracic trauma in mechanism of injury, type of injury, and other organ systems involved. Falls are the most common mechanism of injury in infants and children. Older children are often injured as pedestrians or unrestrained passengers in motor vehicle accidents. Adolescents are more likely to be occupants in motor vehicle–related accidents and to experience penetrating injuries secondary to violence.

Lung contusion is the most common pediatric thoracic injury, with intrapleural injury second. Only 30% of children, compared with 50% to 75% of adults, sustain rib fractures because of increased compliance in the pediatric thoracic cage due to increased cartilage content and greater elasticity of the bones. Thus, a child may have an internal injury (lung contusion) without external evidence of trauma (rib fracture, laceration, bruising). The pediatric trachea has a smaller internal diameter than the adult trachea; therefore, a small amount of obstruction secondary to blood, secretions, or edema can cause significant respiratory distress and hypoxia. The younger pediatric patient is also more sensitive to hypoxia and may develop reflex bradycardia or asystole. In evaluating pediatric patients with thoracic trauma, consider head, neck, and intra-abdominal injuries, because approximately 80% of cases of thoracic trauma in children are part of multisystem injury. Thoracic trauma is routinely associated with abdominal trauma in children because the chest and abdominal cavities lie in close proximity.

4. How do I evaluate a patient with thoracic trauma?

The ABCs (airway, breathing, circulation) of trauma management apply regardless of the organ system injured. Evaluate the injured child according to the primary survey of trauma management. Indications for endotracheal intubation in thoracic trauma patients include depressed neurologic status, inadequate oxygenation or ventilation, compromised circulatory status, and unstable airway (including burns). If a patient has an abnormal examination but appears to be oxygenating and ventilating well and is not in shock, chest radiography is indicated. If breathing is inadequate after endotracheal intubation, with asymmetry of breath sounds, intervention is required prior to chest radiography. Consider pericardial tamponade, tension pneumothorax, or hemothorax in poorly perfused patients with signs of shock in whom other sources of blood loss have been excluded and volume resuscitation has not improved clinical status. Once the patient is stabilized and immediate life-threatening injuries, such as airway obstruction, tension pneumothorax, hemothorax, and pericardial tamponade, are treated, chest radiography and thoracic computed tomography (CT) provide valuable information about other potentially life-threatening and operative injuries.

5. What are the predictors of thoracic injury in children sustaining blunt trauma?

- Low systolic blood pressure
- Elevated respiratory rate
- Abnormal results of thoracic examination
- Abnormal chest auscultation findings
- Glasgow Coma Scale score less than 15

6. Which thoracic injuries require operative intervention?

- Tracheal/bronchial rupture
- Laceration of lung parenchyma, internal mammary artery, or intercostal artery
- Esophageal disruption
- Diaphragmatic hernia
- Pericardial tamponade
- Great vessel laceration

7. Describe signs and symptoms related to each operative thoracic injury.

See Table 62-1 for the signs and symptoms of thoracic injuries.

8. What signs and symptoms are associated with pulmonary contusion?

Most pulmonary contusions result from blunt trauma, generally a motor vehicle crash or a child hit by a car. Patients with moderate to severe pulmonary contusions may be tachypneic, with abnormal breath sounds and an oxygen requirement secondary to shunting within the lung. In one study of patients with mild pulmonary contusions, a finding of tachypnea, tenderness, or abnormal breath sounds was 100% sensitive for all positive radiographs in trauma. However, the clinical features of pulmonary contusion may be subtle, especially just after the injury. Even the initial chest radiograph can appear normal or show only minimal abnormality. Carefully observe the patient's respiratory status after thoracic trauma.

Gittelman M, Gonzalez-del-Rey J, Brody A, et al: Clinical predictors for the selective use of chest radiographs in pediatric blunt trauma evaluations. J Trauma 2003;55:670-676.

Ruddy R: Trauma and the paediatric lung. Paediatr Respir Rev 2005;6:61-67.

9. Are rib fractures important?

Rib fractures occur in about one third of children admitted with thoracic trauma. A single rib fracture does not correlate with severity of injury, but if multiple ribs are fractured the likelihood of multisystem and intrathoracic injury increases. Occasionally a rib fracture

Table 62-1. Signs and Symptoms of Thoracic Injuries

INJURY	SIGNS AND SYMPTOMS
Tracheal/bronchial rupture	Active chest tube air leak
Lung parenchyma	Chest tube bleeding >2-3 mL/kg/hr
Internal mammary or intercostal artery laceration	Hypotension unresponsive to transfusions
Esophageal disruption	Abnormal esophagogram (leak) or esophagoscopic result
	Gastric contents in the chest tube
Diaphragmatic hernia	Abnormal gas pattern in hemithorax
	Displaced nasogastric tube in hemithorax
Pericardial tamponade	Positive pericardiocentesis
Great vessel laceration	Widened mediastinum
	Tracheal or nasogastric tube deviation
	Blurred aortic knob
	Abnormal aortogram (gold standard)

may lead to hemothorax if the rib lacerates a nearby vessel. A pneumothorax is also possible when a direct blow causes the rib to fracture inward and it punctures the pleural cavity. Fractures of the first rib are more important because more force is required to injure this protected rib. With this injury, maintain a high index of suspicion for vascular disruption or tracheal laceration. (The patient is very likely to be symptomatic with these injuries.) Children with rib fractures will likely splint and hypoventilate secondary to pain. Adequate pain control is therefore very important in the management of children with rib fractures. Otherwise, most isolated rib fractures will heal without complications in about 6 weeks.

Kadish HA: Thoracic trauma. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 1459-1477.

10. What signs and symptoms are associated with pneumothorax?

Patients may be asymptomatic, report pleuritic chest pain, have tachypnea, or be in severe respiratory distress. Physical examination may be normal or reveal diminished or absent breath sounds, crepitus, or hyperresonance to percussion on the side of the pneumothorax.

11. How do I diagnose and treat patients with pneumothorax?

In asymptomatic or mildly symptomatic patients, a chest radiograph is helpful in diagnosing and determining type of treatment. If the pneumothorax is small and the patient asymptomatic, hospital observation and administration of 100% oxygen are all that is necessary. A small pneumothorax is classically described as smaller than 15%, although it is common to underestimate the size of a pneumothorax on plain films and to find a much more extensive lesion on a CT scan. If the patient is in respiratory or cardiovascular distress, the diagnosis is made clinically. Place a chest tube or a pigtail catheter when this is noted. Use a pigtail catheter only with a pure pneumothorax (no blood). Chest tube or pigtail placement is also indicated in patients undergoing positive-pressure ventilation or requiring air transport. An asymptomatic patient may rapidly become symptomatic if a small, simple pneumothorax progresses to a tension pneumothorax.

12. Should thoracic CT replace chest radiography in the initial management of blunt trauma in the pediatric patient?

No. Even though CT has been shown to be more sensitive than chest radiography in diagnosing thoracic injuries, thoracic trauma makes up less than 20% of all pediatric traumas. Performing thoracic CT on all pediatric trauma patients would increase costs and patient radiation dose, with little clinical improvement. Obtain a thoracic CT only in patients with thoracic injury suspected from clinical examination or from chest radiograph (see Question 5).

Moore MA, Wallace EC, Weston SJ: The imaging of paediatric thoracic trauma. Pediatr Radiol 2009;39:485.

Patel RP, Hernanz-Schulman M, Hilmes MA, et al: Pediatric chest CT after trauma: Impact on surgical and clinical management. Pediatr Radiol 2010;40:246.

13. What is the role of bedside thoracic ultrasound in the initial management of blunt trauma in the pediatric patient?

Thoracic ultrasound can also be used in the evaluation of thoracic trauma and is sensitive in identifying pleural air or fluid. It can be done quickly in the emergency department (ED) with the absence of radiation. The only disadvantage of bedside ultrasound is that it is operator dependent. CT is still the gold standard for the evaluation of underlying lung parenchyma injury.

14. What is the most common complication of an intrapleural injury?

Tension pneumothorax is the most common complication of an intrapleural injury, developing in up to 20% of children after simple pneumothorax. Tension pneumothorax occurs with progressive accumulation of air within the pleural cavity, which not only collapses the ipsilateral lung but also compresses the contralateral lung. Patients may present in severe respiratory distress, with decreased breath sounds on the side of the pneumothorax and a shift of mediastinal structures to the contralateral side. Because the inferior vena cava is relatively fixed in place and cannot shift as much as the superior

vena cava, venous return to the heart is reduced. The patient may appear tachycardic, peripherally vasoconstricted, and in hypotensive shock. Whenever a trauma patient suddenly deteriorates, the treating physician must reassess the airway and breathing before focusing on circulation.

15. How do I treat a patient with a tension pneumothorax?

Initial treatment consists of needle decompression performed in the midclavicular line, at the level of the second intercostal space of the ipsilateral side. With a tension pneumothorax, an immediate release of air should be noted. If this sign is positive, needle decompression is only a temporizing measure and must be followed by tube thoracotomy. Tube thoracotomy or insertion of a pigtail catheter usually is done in the midaxillary line at the level of the fifth intercostal space (nipple level). Obtain chest radiography only after insertion of the chest tube/pigtail catheter. Do not use a radiograph to diagnose tension pneumothorax in symptomatic patients. One of the advantages of bedside thoracic ultrasound is that it can be done quickly in the ED while prepping the patient for a chest tube. If significant air leak continues after chest tube placement, consider a tracheobronchial rupture.

16. How can I tell if a patient has a tracheobronchial injury?

Injury to the tracheobronchial tree in children is rare (incidence <1%). The mortality rate for these injuries is very high, and many patients with a tracheobronchial injury will die at the scene. This injury is caused most commonly by acceleration or deceleration forces. The mechanism of injury (fall, crush, and direct blow) provides an important clue. Clinical signs include cyanosis, hemoptysis, tachypnea, and subcutaneous emphysema (cervical, mediastinal, or both). A continued air leak after insertion of a thoracostomy tube should alert the physician to the possibility of a bronchial tear. In the absence of pneumothorax, suspect tracheal rupture if pneumomediastinum or cervical emphysema is present. Immediately consult a surgeon when this injury is suspected.

17. How is a tracheobronchial injury treated?

Treatment includes initial airway stabilization followed by bronchoscopic evaluation of the airway. According to numerous reports in the literature, a partial tracheal tear may become complete after endotracheal intubation. Therefore, if the airway is stable, perform orotracheal intubation in the operating room under bronchoscopic guidance. If the airway is unstable and emergent endotracheal intubation is needed, prepare for backup measures, such as cricothyroidotomy, tracheostomy, or fiber-optic bronchoscopy.

18. How can I tell if a patient has an esophageal injury?

Esophageal injury is very rare in children but presents a diagnostic challenge when it does occur. The most common cause for esophageal perforation in children is iatrogenic, followed by penetrating trauma (gunshot or stab wound). Patients with an esophageal rupture in the cervical region may report neck stiffness or neck pain. They may regurgitate bloody material and have cervical subcutaneous emphysema or odynophagia. In the thoracic region, patients may present with abdominal spasms and guarding, chest pain, subcutaneous emphysema, tachycardia, or dyspnea. A lateral neck radiograph may show retroesophageal emphysema. A chest radiograph may show pneumothorax, pneumomediastinum, or an air-fluid level in the mediastinum. Esophagography, esophagoscopy, or both can make the diagnosis of esophageal perforation.

19. How are esophageal injuries treated?

Treat patients who have suspected esophageal perforation with adequate volume resuscitation, placement of a nasogastric tube, and antibiotics covering gram-positive, gram-negative, and anaerobic organisms. Once the diagnosis is made, prompt surgical correction is mandatory. If the diagnosis is made within 24 hours, the mortality rate is approximately 5%. Diagnosis delayed for more than 24 hours after injury is associated with a mortality rate of 70%.

20. On which side is a diaphragmatic injury seen more commonly?

Approximately 80% of diaphragmatic injuries occur on the left. The left diaphragm is relatively unprotected, whereas the liver protects the right side. Right-sided diaphragmatic injuries are associated with increased mortality rate; patients usually have a greater physiologic insult and more associated injuries.

21. How is the diagnosis of a traumatic rupture of the aorta (TRA) made?

CT with contrast may be obtained quickly and is widely available in the ED. Consider this study when aortic injury is suspected clinically or by chest radiograph. Thoracic CT is only 55% to 65% accurate but helps to diagnose associated injuries. The gold standard for diagnosing TRA is aortography. In one study, transesophageal echocardiography was shown to be highly sensitive and specific for detecting injury to the thoracic aorta. If the patient is stable and TRA is a concern, perform aortography. Evaluate for life-threatening intracranial, thoracic, or intra-abdominal injuries and stabilize the patient before aortography. If the patient is unstable, transesophageal echocardiography can be performed in the operating room while other life-threatening injuries are treated. Early diagnosis is imperative in patients with TRA. Morbidity and mortality rates increase threefold if operative intervention is delayed more than 12 hours.

22. How do patients with pericardial tamponade present?

Pericardial tamponade initially may be difficult to diagnose because associated injuries obscure the clinical signs and symptoms. Patients may present with distant heart sounds, low blood pressure, poor perfusion, narrow pulse pressure, or electromechanical dissociation. Pulsus paradoxus, with blood pressure falling more than 10 mm Hg during inspiration, occurs in less than half of patients with pericardial tamponade. Do not rely on this to make the diagnosis. Chest radiography may show an enlarged heart, and an electrocardiogram may show low-voltage QRS waves. Neither of these tests is diagnostic for pericardial tamponade. Do not delay treatment (pericardiocentesis) in unstable patients. In stable patients, an echocardiogram can demonstrate fluid within the pericardial sac.

23. How concerned should I be about blunt cardiac injury in patients with thoracic trauma?

Consider blunt cardiac injury, commotio cordis, in any patient with thoracic trauma (e.g., a child hit by a fast-pitched baseball) who develops cardiac arrhythmia, a new murmur, or congestive heart failure. Monitor patients with suspected blunt cardiac injury in the ED or hospital; if no arrhythmias develop on electrocardiography, they can safely be sent home. Cardiac biomarkers (creatine kinase[CK]-MB, troponin) have a high false-positive rate and are not a helpful screening tool. Consider transesophageal echocardiography for patients with thoracic trauma who have an abnormal electrocardiogram, arrhythmia, or new heart murmur. It is more sensitive in detecting myocardial injury than transthoracic echocardiography. A child with suspected blunt cardiac injury who is hemodynamically stable and has no arrhythmias is unlikely to develop a serious life-threatening arrhythmia or pump failure. Any patient with suspected blunt cardiac injury who is hemodynamically unstable or has arrhythmias should undergo transesophageal echocardiography and be admitted to the intensive
care unit.

24. What are the four conditions that lead to sudden circulatory arrest after a nonpenetrating blow to the chest (commotio cordis)?

1. The blow must strike the chest in the area of the heart with force.
2. The object must have sufficient mass (baseball, hockey puck, knee).
3. The blow must strike the chest during the ventricular vulnerable period (T wave).
4. The ventricles must have a large enough mass (>3.5 kg).

Link MS: Pathophysicology, prevention and treatment of commotio cordis. Curr Cardiol Rep 2014;16:495.
Maron BJ, Estes NA: Commotio cordis. N Engl J Med 2010;362:917-927.

Key Points: Thoracic Trauma

1. The most common injuries in blunt thoracic trauma are lung contusions (58%), pneumothorax or hemothorax (38%), and rib fractures (38%).
2. Thoracic CT should be part of the initial evaluation of pediatric trauma patients if a lung contusion, pneumothorax, or a hemothorax is suspected or if the cause of the patient's respiratory distress is unknown. Thoracic ultrasound can be used at the bedside to acutely diagnose a pneumothorax or hemothorax.
3. Myocardial contusion is the most common, and ventricular rupture is the most lethal of blunt cardiac injuries.
4. Patients with a TRA may present with hypotension, paraplegia, anuria, or absent/diminished femoral pulses.
5. Radiographic findings of a TRA may include a widened mediastinum, blurred aortic knob, pleural cap, or tracheal or nasogastric tube deviation.
6. Suspect commotio cordis and ventricular fibrillation in any patient who has become unconscious immediately after a blow to the chest.

BITES AND STINGS

Kate M. Cronan

1. Describe the bite of the imported fire ant. How is it treated?

Imported fire ants are nonwinged Hymenoptera found in the Southeastern and South Central United States. They bite with their jaws, then pivot around and sting at multiple sites in a circular pattern. Within 24 hours a sterile pustule develops at the site of the stings. This lesion can be diagnostic of a fire ant sting. Stings often occur on the legs and feet of children. Treatment consists of local wound care, antihistamines, and steroids for severe cases.

Hodge D: Bites and stings. In Fleisher GR, Ludwig S (eds): The Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 671-689.

2. True or false: Stings from imported fire ants *(Solenopsis invicta)* can cause severe systemic reactions.

True. Local reactions can consist of a wheal, a pustule, or a large local reaction. Systemic reactions may range from urticaria to life-threatening anaphylaxis.

LaShell MS, Calabria CW, Quinn JM: Imported fire ant field reaction and immunotherapy safety characteristics: The IFACS Study. J Allergy Clin Immunol 2010;125:1294.

3. How common are dog bites?

Each year, there are approximately 4.5 million dog bites in the Unites States. Young children, especially males, are at particular risk of serious injury due to dog bites. Children are more likely to receive medical attention for these bites, and children 5 years of age or younger may be at increased risk for hospital admission related to a dog bite. Young children may be more likely to unintentionally provoke a dog to attack. Dog bites to children occur most often within the home or involve a dog that is familiar to the child. Thus, children who live in a home with a dog are at higher risk. Usually, the dog involved has not bitten anyone previously. The dog's owner can be identified 85% to 90% of the time. Many dog owners are unaware of the risk of injury to a young child.

Daniels DM, Ritzi RB, O'Neil J: Analysis of non-fatal dog bites in children. J Trauma 2009;61:S17.
Gielen A, Stepnitz R, McDonald E, et al: Dog bites: An opportunity for parent education in the pediatric emergency department. Pediatr Emerg Care 2012;28:966-970.
Hodge D: Bites and stings. In Fleisher GR, Ludwig S (eds): The Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 671-689.

4. A 2-year-old child is attacked by a large dog and she has multiple significant facial lacerations. Besides the obvious wounds, what other injuries should you consider?

When a small child is attacked by a large dog, consider a fracture of the skull or facial bones. The powerful jaws of the dog exert strong force and can encircle the child's head. This can cause significant damage to a child, and fractures are common. Consider evaluating the child with radiographs of the skull or a computed tomography (CT) scan of facial bones and skull.

5. What is the rate of wound infection after common animal bites?

- Cat bites: as high as 50%
- Dog and human bites: 10% to 15%

6. What are the common microbiologic organisms involved with dog, cat, and human bites?

- **Dog bites**: *Staphylococcus aureus*, streptococci, *Pasteurella canis*
- **Cat bites**: *Pasteurella multocida*, *S. aureus*, streptococci
- **Human bites**: *Streptococcus viridans*, *S. aureus*, anaerobes, *Eikenella corrodens*

Note: Methicillin-resistant *S. aureus* (MRSA) is a potential bite wound pathogen.

Pickering LK (ed): Red Book: 2012 Report of the Committee on Infectious Diseases, 29th ed. Elk Grove Village, IL, American Academy of Pediatrics, 2012, pp 203-206.

7. Which bites should be treated with prophylactic antibiotics?

- All dog bites involving hands and feet
- All human bites
- All cat bites
- Puncture wounds
- Bites that penetrate a joint
- Bites for which treatment has been delayed for more than 24 hours
- Bites in immunosuppressed patients

8. True or false: Wounds due to some animal bites should be closed with cyanoacrylate tissue adhesive.

False. Avoid use of tissue adhesive in treating animal bites because of the risk of infection.

Hodge D: Bites and stings. In Fleisher GR, Ludwig S (eds): The Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 671-689.

Key Points: Dog Bites

1. Frequently involve young children who may have unintentionally provoked the dog
2. Could involve fractures of the facial bones and skull if a small child is attacked by a large dog
3. Must be carefully irrigated with saline to prevent infection
4. Often require closure with sutures for cosmetic reasons
5. Should not be closed if at high risk for infection

9. How can one tell if a human bite was inflicted by an adult or a child?

If the intercanine distance is greater than 3 cm, the bite is most likely caused by an adult. This should raise concerns about child abuse. However, this measurement is not completely reliable. It is best to take pictures of the bite and consider involvement of a forensic dentist.

Kemp A, Maguire SA, Sibert J, et al: Can we identify abusive bites on children? Arch Dis Child 2006;91:951 (letter).

10. What is the preferred hand position for evaluation of a fist injury from a bite?

Evaluate the wounds when the hand is in a *clenched* position. This permits more accurate assessment of the injuries. Once the hand is relaxed, it is difficult to assess the extent of injury because the deeper tissues form a closed space, permitting the growth of bacteria.

11. Which organisms cause rat-bite fever?

Streptobacillus moniliformis and *Spirillum minus*. In the United States *S. moniliformis* is most common and is transmitted by rats, other rodents, and rodent-eating animals. Transmission to humans occurs via bites, scratches, handling infected animals, or ingestion of food or water contaminated by excreta. *S. minus* is transmitted by the bites of rats and mice.

Pickering LK (ed): Red Book: 2012 Report of the Committee on Infectious Diseases, 29th ed. Elk Grove Village, IL, American Academy of Pediatrics, 2012, pp 608-609.

12. What are the symptoms of rat-bite fever?

Patients with *S. moniliformis* infection present with fever, chills, myalgia, headache, and vomiting. Within a few days of the onset of fever, a rash develops and is typically found on the extremities including the palms and soles. The bite area heals quickly and is followed by migratory polyarthritis in approximately 50% of patients. Complications of infection such as pneumonia, endocarditis, myocarditis, and meningitis may occur.

In patients with *S. minus* infection, the bite area heals and is followed by local ulceration, lymphangitis, and lymphadenopathy. A rash consisting of red or purple plaques follows. Arthritis is rare.

13. What is the best multipurpose insect repellent?

DEET (*N,N*-diethyl-m-toluamide) is the best multipurpose insect repellent and is the active ingredient in most commercial insect repellents. It is available in various forms,

including sprays, gels, liquids, and sticks, and in concentrations of 4% to 100%. DEET can be applied to skin or clothes. It should not be applied under clothes. It does not work against stinging insects but is quite effective against mosquitoes, biting flies, chiggers, fleas, and ticks. It is important to note that application of DEET may reduce the efficacy of sunscreen. Protection with DEET is shortened by swimming, washing, rainfall, sweating, and wiping.

14. What are some disadvantages of DEET?

DEET can be toxic, and use of low concentrations (10%) is recommended in children. Avoid application to areas around the eyes, mouth, and open skin lesions. For children, apply DEET once per day. Avoid this completely for children younger than 2 months of age. Oral ingestion of large quantities of DEET may cause seizures, coma, or death. For those concerned about the possible toxicity of DEET, citronella, a plant-derived insect repellent, is a good alternative.

Committee on Environmental Health: Pesticides. In Etzel RA, Balk SJ (eds): Pediatric Environmental Health, 3rd ed. Elk Grove Village, IL, American Academy of Pediatrics, 2012, pp 515-548.

15. What is the role of permethrin in helping to avoid insect bites?

Permethrin is not an insect repellent, but it causes toxicity to the nervous system of insects. It is available as a spray and should be applied to clothing or bedding. Do not apply this to the skin for the purpose of preventing insect bites.

16. What is papular urticaria?

Papular urticaria is a skin condition that consists of intensely pruritic wheals and papules. These lesions are attributable to arthropod bites in specifically sensitized children. Sensitization may occur in infancy, but the lesions do not appear until the second year of life in many cases. Various arthropods have been identified as culprits: dog lice, mosquitoes, bedbugs, fleas, mites, and chiggers. The wheals represent an immediate hypersensitivity reaction to insect bites. Wheals then evolve into papules, which represent a delayed hypersensitivity reaction. New lesions form continually, and the eruption may persist for many months. The lesions usually appear on exposed areas, such as the extremities, head, neck, and shoulders. The differential diagnosis includes scabies, varicella, pediculosis, urticaria, and insect bites without papular urticaria.

17. How is papular urticaria treated?

Treatment should focus on a multipurpose insecticide for the entire home. Include household pets in the treatment. When the cause of the lesions is removed, clinical symptoms should improve greatly, but supportive treatment is necessary. Oral antihistamines and topical steroid creams help to control the itching.

18. Name the tick-borne infectious diseases that occur in the United States.

- **Bacterial:** Lyme disease, tularemia, relapsing fever
- **Rickettsial:** Rocky Mountain spotted fever, ehrlichiosis
- **Viral:** Colorado tick fever
- **Protozoal:** Babesiosis

Pickering LK (ed): Red Book: 2012 Report of the Committee on Infectious Diseases, 29th ed. Elk Grove Village, IL, American Academy of Pediatrics, 2012, pp 620-622.

19. In penicillin-allergic patients who present with a human bite or a high-risk animal bite, what antibiotic regimen is recommended?

Prophylaxis and treatment for penicillin-allergic children are challenging problems. Erythromycin and tetracycline activity against *S. aureus* and anaerobes is unreliable. Tetracycline is effective against *Pasteurella* spp., but consider the risk of dental staining in children younger than 8 years. Therefore, combination treatment with oral or parenteral trimethoprim-sulfamethoxazole (effective against *S. aureus*, *E. corrodens*, and *Pasteurella* spp.) and clindamycin (effective against anaerobes, streptococci, and *S. aureus*) is used for preventing or treating bite wound infections. Ceftriaxone may be used if the patient can tolerate cephalosporins. Metronidazole provides excellent anaerobic coverage and may be used as an alternative to clindamycin.

Key Points: Mammalian Bites at High Risk for Infection

1. Puncture wounds
2. Minor wounds of the hands or feet
3. Wounds with care delayed more than 12 hours
4. Cat and human bites
5. Wounds in immunocompromised children

20. Explain the Hymenoptera sting.

A sting consists of an injection of venom via a tapered shaft that projects from the venom sac located in the abdomen of females of the *Hymenoptera* species. Stings usually occur in warm weather.

21. Which group of the order *Hymenoptera* is considered aggressive—apids or vespids?

Apids include honeybees and bumblebees; vespids include yellow jackets, hornets, and wasps. Apids tend to be docile and sting only when provoked. Bees, wasps, and hornets are responsible for many of the anaphylactic reactions to venomous insects in the United States. The bumblebee is large, slow-moving, and very noisy. Honeybees may cause problems among beekeepers.

Vespids are aggressive; the yellow jacket is the most aggressive in this family. Yellow jackets are thought to be the principal cause of allergic reactions to insect stings in the United States.

22. How are reactions to bee stings categorized?

- The most common reaction is a **small local reaction** that presents as a painful pruritic lesion smaller than 5 cm at the site of the sting and lasts briefly (several hours).
- A **large local reaction** occurs in approximately 10% of people after a sting and manifests with swelling and erythema in the area of the sting. It is usually larger than 5 cm in diameter, often is very painful and itchy, and lasts for longer than 24 hours, sometimes up to 1 week. The features of this reaction may be confused with cellulitis, but cellulitis rarely develops after a bee sting.
- **Systemic reactions**, which occur in approximately 0.5% of *Hymenoptera* stings, begin within minutes to hours after the sting and can be mild or severe.
  - **Mild systemic reactions** most commonly consist of dermal signs and symptoms: generalized urticaria, pruritus, flushing, and angioedema. Gastrointestinal symptoms, such as abdominal pain, nausea, and diarrhea, may also occur.
  - **Severe systemic reactions** include life-threatening signs, such as upper airway edema with hoarseness and stridor; shock, manifested by pallor and fainting; and bronchospasm, characterized by coughing, dyspnea, and wheezing.
- **Unusual reactions** to bee stings include vasculitis, nephrosis, encephalitis, and serum sickness. Symptoms usually occur several days to several weeks after the sting and tend to last for a long time.

23. What is the best way to remove a stinger after a bee sting?

If a stinger remains in the skin after a bee sting, swipe it sideways away from the direction in which it points. A credit card or other similar card works well to do the swiping. Grasping and pulling the stinger may squeeze the remaining venom from the venom sac into the sting site. Remove the stinger as soon as possible to avoid further deposition of venom.

24. What is the treatment for anaphylaxis due to an insect sting?

The treatment is the same as for anaphylaxis from other causes. First, pay attention to airway, breathing, and circulation. Give intramuscular epinephrine in a 1:1000 dilution immediately; repeat the dose every 30 minutes as needed. Intravenous (IV) epinephrine is rarely indicated except in the case of profound shock. Antihistamines, such as diphenhydramine or hydroxyzine, given parenterally, may reduce urticaria and pruritus. Some recommend that steroids, such as methylprednisolone, be administered early in the treatment plan. Depending on the severity of the reaction, other treatment modalities may be indicated (vasopressors, supplemental oxygen, IV fluids, bronchodilators).

After recovery, prescribe an emergency kit for self-administration of epinephrine. Provide detailed instructions about technique and appropriate use to the patient and family.

Vankawala H, Park R: Bee and Hymenoptera stings. Available from www.emedicine.com/emerg/topic55.htm.

25. What is the significance of the Southwestern desert scorpion, *Centruroides exilicauda*?

It is the only scorpion species of medical importance in the United States. Also called the Arizona bark scorpion, it is found mainly in Arizona, Texas, and California. Arizona bark scorpions are yellow or brown and are usually 5 cm in length. Their appearance resembles that of the shrimp. Their long, very mobile tails contain the stinger. They tend to reside in brush areas and trees and sting mostly at night. Children are usually stung on their extremities.

26. How does the venom of the Arizona bark scorpion work?

The venom is mostly neurotoxic. It overstimulates the parasympathetic and sympathetic nervous systems and results in agitation, tachycardia or bradycardia, hypertension, dysrhythmias, and increased secretions. Cranial nerve dysfunction may cause rapid dysconjugate eye movements and contractions of muscles of the face and tongue. Peripheral motor neuron involvement presents as muscle contractions and uncontrolled jerking movements of the extremities, which may be mistaken for seizures. Respiratory distress may result from decreased pharyngeal tone and uncoordinated contraction of respiratory muscles.

27. How are scorpion envenomations treated?

Treatment of scorpion envenomation is usually supportive; place focus on airway control. Provide local wound care along with tetanus prophylaxis. Admit children with systemic signs and symptoms to the hospital. Administer analgesics and benzodiazepines for pain and agitation. Many authorities prefer a continuous infusion of midazolam. A hyperimmune goat serum antivenin is known to be effective but has not been approved by the Food and Drug Administration. It is therefore available only in Arizona, where the Arizona State Board of Pharmacy has approved its use.

Bush S, Gerardo C: Scorpion envenomations. Available from www.emedicine.com/emerg/topic524.htm.

28. What are the four kinds of sharks in the United States that are responsible for most shark bites?

Gray reef, great white, blue, and mako sharks. Bites from these sharks can cause extensive injury, including fractures, amputations, and penetration of internal organs.

Hodge D: Bites and stings. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 671-689.

29. Which two spiders in the United States cause significant envenomations?

The black widow spider *(Latrodectus mactans)* and the brown recluse spider *(Loxosceles reclusa)* are the only two spiders known to cause significant envenomations. Brown recluse spider bites occur much more frequently than black widow spider bites. Brown recluse spiders are nonaggressive and usually bite only when threatened. They have a brown or yellow violin-shaped marking on the dorsal thorax and are found most commonly in the Southern and Midwestern states. They prefer dark, warm, protected areas, such as closets and garages. Black widow spiders inhabit all states except Alaska and are found most commonly in the South, Ohio Valley, Southwest, and West Coast. They are found in attics, barns, trash piles, and other dimly lit areas. Bites are more frequent in the warmer months. The black widow spider is jet black, with an hourglass-shaped red mark on the underside of the abdomen.

30. What are the symptoms of the bite of a brown recluse spider?

At the time of the bite, a stinging sensation may be felt. Increased pain and pruritus may develop within several hours. The clinical course ranges from mild local irritation to large necrotic skin lesions and systemic reactions. Most bites go unnoticed or result in only a mild red papule that heals quickly. A central vesicle with surrounding erythema is discovered at the site of the bite. In some scenarios, vasospasm and hemorrhage in the first few hours after the bite account for the red, white, and blue discoloration. Systemic signs

and symptoms occur in approximately 40% of envenomations, including fever, nausea, vomiting, headache, and arthralgias. A scarlatiniform rash may occur simultaneously. Systemic loxoscelism is rare and seen almost exclusively in children. It is characterized by anemia, hemolysis and thrombocytopenia, shock, and renal failure.

31. How is the bite of a brown recluse spider treated?

Treatment consists of local wound care; extensive dermal injury may require skin grafting. Analgesics and tetanus immunization are mainstays of therapy. If extensive intravascular hemolysis occurs, it is crucial to maintain high urinary flow rates with alkalinization of the urine via sodium bicarbonate infusion.

Stibich A, Schwartz R: Spider envenomation, brown recluse. Available from www.emedicine.com/DERM/topic598.htm.

32. What is latrodectism?

This term applies to systemic symptoms due to the spread of the neurotoxin from black widow spider bites. There are three phases of latrodectism:

- The **exacerbation** phase, which occurs from less than 6 up to 24 hours after the bite and is characterized by muscle spasms at the site of the bite or elsewhere
- The **dissipation** phase, which occurs 1 to 3 days after the bite and is a time when symptoms subside
- The **residual** phase, which occurs weeks to months after the bite and may consist of persistent tremors and weakness and fatigue

Isbister GK, Fan HW: Spider bite. Lancet 2011;378:2030.

33. How is the bite of a black widow spider treated?

Treatment consists of analgesia, including oral narcotics for milder cases and IV morphine for more severe cases. Benzodiazepines can relieve anxiety. An antivenin is available, but it is indicated only for the most severe cases unresponsive to the preceding measures. Life-threatening tachycardia and hypertension are indications for this antivenin. Death is extremely uncommon.

34. What infectious agent should be considered when treating an iguana bite?

Iguanas are becoming popular pets. They can inflict injury via scratching, biting, and tail whipping. They frequently carry unusual subtypes of fecal salmonella. The Centers for Disease Control and Prevention recommends that children under age 5 and those who are immunocompromised should not have iguanas and other salmonella-carrying pets, such as turtles, in the home.

Centers for Disease Control and Prevention (CDC): Reptile-associated salmonellosis—Selected states, 1998-2002. MMWR Morb Mortal Wkly Rep 2003;52(49):1206-1209.

35. What are the poisonous snakes indigenous to the United States?

Snakes in the Crotalidae family (pit vipers) and Elapidae family (coral snakes) are poisonous. Rattlesnakes, copperheads, and water moccasins are pit vipers and account for 99% of poisonous snakebites. The coral snake accounts for less than 1% of poisonous snakebites.

36. How can pit vipers be distinguished from nonpoisonous snakes?

Pit vipers have two pits, one on either side of their head, and their pupils are elliptical and vertically oriented. The head is triangular, and they have two curved fangs that are widely spaced. The scales caudal to the anal plate continue in a single row. Nonpoisonous snakes have oval heads with round pupils. The anal plate has a double row of subcaudal plates.

Hodge D: Bites and stings. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 671-689.

37. What causes tularemia?

Francisella tularensis, a gram-negative pleomorphic coccobacillus, is the causative agent. Sources of this organism include many species of wild mammals, some domestic animals (e.g., sheep, cattle, cats), and the blood-sucking arthropods that bite these animals (e.g., ticks and mosquitoes). In the United States, rabbits and ticks are major sources of infection.

38. How does tularemia present? How is it treated?

Most patients present with an abrupt onset of fever, chills, myalgia, and headache. There are several tularemic syndromes. The most common is the ulceroglandular syndrome, which presents with a painful maculopapular lesion at the bite site and painful, acutely inflamed lymph nodes. Other, less common syndromes are the glandular, oropharyngeal, oculoglandular typhoidal, intestinal, and pneumonic presentations.

The treatment is streptomycin. Gentamicin is an effective alternative.

American Academy of Pediatrics: Red Book: 2012 Report of the Committee on Infectious Diseases, 29th ed. Elk Grove, IL, American Academy of Pediatrics, 2012, pp 768-769.

39. Which type of jellyfish sting is highly toxic?

Stings from the common purple jellyfish (*Pelagia noctiluca*) and the sea nettle jellyfish (*Chrysaora quinquecirrha*) are only mildly toxic. Local skin irritation is the usual clinical manifestation. However, stings from *lion's mane* (*Cyanea capillata*) are highly toxic. This jellyfish is found along both coasts of the United States. Contact with the tentacles of lion's mane causes severe burning; prolonged exposure results in muscle cramps, and respiratory failure may ensue.

40. How are toxic jellyfish stings treated?

Treatment of toxic jellyfish stings focuses on relieving pain, alleviating effects of the venom, and providing supportive therapy. The first goal is to remove the tentacles to avoid further nematocyst discharging. It is possible to inactivate the remaining nematocysts by topical application of vinegar (acetic acid) for 30 minutes. However, vinegar may cause pain and exacerbation of nematocyst discharge in the majority of species. Hot water immersion (as tolerated) and topical lidocaine appear more widely beneficial in improving pain symptoms and are preferentially recommended. Unfortunately, they may be difficult to obtain at the site of envenomation, such as the beach or diving sites. In these instances, removing the nematocysts and washing the area with saltwater may be considered. Remove the residual tentacles with instruments or gloved hands. No antivenin is available. Antihistamines, steroids, and analgesics may be indicated. In some cases cardiac and respiratory support may be required.

Marcus E, Isbister G: Jellyfish stings. UpToDate, 2014. Available from http://www.uptodate.com. Accessed on Feb. 14, 2014.

Ward NT, Darracq MA, Tomaszewski C, Clark RF: Evidence-based treatment of jellyfish stings in North America and Hawaii. Ann Emerg Med 2012;60:399-414.

41. How should one remove a tick embedded in the skin?

Grasp the tick by the head or the mouth with blunt angled forceps as close to the skin as possible. This action should be perpendicular to the patient's skin. With steady traction, pull the tick and do not twist as you pull back. It is best not to squeeze, puncture, or crush the tick, as it could regurgitate material into the wound during removal. If any residual parts are noted on the skin, remove them with forceps. Alternatively, use a large-gauge needle to swipe residual parts away. Cleanse the bite wound after removal. Do *not* apply petroleum jelly, a hot match, alcohol, or other irritants.

American Academy of Pediatrics: Red Book: 2012 Report of the Committee on Infectious Diseases, 29th ed. Elk Grove, IL, American Academy of Pediatrics, 2012, pp 207-209.

42. How important is the duration of attachment of ticks to the skin?

Duration of attachment to the host plays a major role in the transmission of pathogens. *Borrelia burgdorferi* from an infected tick has a higher likelihood of transmission after 24 hours, and this likelihood increases after 72 hours. On the other hand, *Ehrlichia chaffeensis* requires a shorter period for transmission.

43. What are the most effective precautions to avoid Lyme disease?

- **Personal protection consists of wearing appropriate clothes:** Wear long sleeves that are buttoned and long pants tucked in when in at-risk areas.
- **Use of repellants:** Apply DEET to the skin and clothes. Consider the use of permethrin applied to the clothes in conjunction with DEET.
- **Inspection:** Parents should inspect their children after times spent in risky areas. If a tick is found on the skin, washing can be effective before attachment occurs.
- **Modification of outdoor environment:** Replace brush with wood chips.

44. What is skeeter syndrome?

Skeeter syndrome refers to large (several cm to 10 cm) local reactions to mosquito bites. The reactions are itchy and sometimes painful with redness, swelling, and warmth and develop within hours of the bite. The reactions progress over the next 8 to 12 hours. These large reactions are caused by an allergic reaction to the saliva of mosquitoes. They resolve with or without treatment over 3 to 10 days. Treatment is based on symptoms and can include antihistamines and in some cases glucocorticoids.

45. How soon after an exposure should immunoprophylaxis for rabies begin?

After thorough wound care is completed, passive and active immunoprophylaxis is required for optimal therapy. Begin prophylaxis as soon as possible after exposure, ideally within 24 hours. However, a delay of several days or more may not compromise effectiveness; begin prophylaxis, if indicated, regardless of the time of exposure.

American Academy of Pediatrics: Red Book: 2012 Report of the Committee on Infectious Diseases, 29th ed. Elk Grove, IL, American Academy of Pediatrics, 2012, pp 606-607.

46. What is the recommendation for rabies prophylaxis after a bite from a bat?

The risk for rabies resulting from an encounter with a bat is often difficult to assess because of the sometimes imperceptible bite inflicted by this mammal. Consider rabies prophylaxis when direct contact between a human and a bat has occurred. Also, consider rabies prophylaxis for a child who has been in a room with a bat and who may be unable to rule out any physical contact. Such patients might include (1) a sleeping child who awakens to find a bat in the room, (2) an unattended child, (3) a mentally disabled person, or (4) an intoxicated person. Rabies prophylaxis is not necessary if the person was aware of the bat at all times while in an enclosed space and is certain that there was no bite, scratch, or mucous membrane exposure. If the bat is captured and can be tested for rabies, rabies prophylaxis can await the results of prompt testing. If uncertainty about the need for prophylaxis still exists, consult the local public health authority.

DROWNING

Linda Quan and Derya Caglar

CHAPTER 64

1. What is drowning?

Drowning is "the process of experiencing respiratory impairment from submersion or immersion in liquid." The term drowning does not imply death or any morbidity associated with the event. Drowning causes anoxic injury to end organs. Terms such as "near," "wet," "dry," "secondary," "silent," "passive," and "active" drowning have been abandoned.

Idris AH: Recommended guidelines for uniform reporting of data from drowning: The "Utstein style." Resuscitation 2003;59:45-57.

Papa L, Hoelle R, Idris A: Systematic review of definitions for drowning incidents. Resuscitation 2005;65 (3):255-264.

2. Who is at highest risk for drowning? How does it happen?

Children aged 1 to 4 years and 15 to 19 years have the highest drowning rates in the United States. Drowning is second only to motor vehicle injury as a leading cause of death from unintentional injury. The majority of drownings in children younger than 5 years occur in residential swimming pools or in the bathtub when unsupervised for even as little as 5 minutes. Adolescents mostly drown in natural waters, like lakes and rivers. Alcohol use, lack of personal flotation device (PFD) use, black race, and male gender significantly increase drowning risk in this age group.

Centers for Disease Control and Prevention (CDC): Drowning—United States, 2005-2009. MMWR Morb Mortal Wkly Rep 2012;61:344.

Cohen RH, Matter KC, Sinclair SA, et al: Unintentional pediatric submersion-injury-related hospitalizations in the United States, 2003. Inj Prev 2008;14:131-135.

Irwin CC, Irwin RL, Ryan TD, Drayer J: The legacy of fear: Is fear impacting fatal and non-fatal drowning of African American children? J Black Stud 2011;42(4):561-576.

Mao SJ, McKenzie LB, Xiang H, et al: Injuries associated with bathtubs and showers among children in the United States. Pediatrics 2009;124:541-547.

3. What is a cold-water drowning?

Cold water is lower than 65° F (<17° C). Cold water immersion induces a brief bradycardia, this diving reflex superceded by supraventricular tachyarrhythmias and hypertension. More importantly, cold water induces a reflexive gasp followed by uncontrollable hyperventilation. These involuntary respiratory and cardiac responses can cause drowning. A recent study showed cold water temperature was not associated with survival after drowning.

Quan L, Mack CD, Schiff MA: Association of water temperature and submersion duration and drowning outcome. Resuscitation 2014;85(6):790-794.

4. Is hypothermia protective?

We should first distinguish between those patients who are hypothermic on presentation after a drowning event and therapeutic hypothermia, the medical intervention of cooling a drowning victim with hopes of improving neurologic outcome. Hypothermia can dramatically decrease the cerebral metabolic rate, with a resultant decrease in cerebral oxygen requirements and greater tolerance of hypoxia. However, to be protective, hypothermia must precede the onset of hypoxic injury. The very rapid cooling required to produce protective hypothermia is usually not accomplished in non–icy water drowning. Scattered case reports of good neurologic recovery after prolonged arrest following a drowning incident mostly describe survivors submerged in freezing water (<5° C) who presented with core body temperatures lower than 30° C. These cases are extremely rare. The vast majority of patients who present with hypothermia (<34° C) have high mortality and morbidity rates; hypothermia in the emergency department (ED) predicts a poor outcome.

There is no evidence that therapeutic hypothermia is beneficial for improving outcomes following pediatric cardiopulmonary arrest. Pediatric intensive care experience

with therapeutic hypothermia in the 1970s and recent studies evaluating extracorporeal membrane circulation, rewarming, and therapeutic hypothermia in pediatric and adult drowned patients showed no significant improvement in neurologic outcome or mortality rates.

Dean R, Mulligan J: Management of water incidents: Drowning and hypothermia. Nurs Stand 2009;24 (7):35-39.
Kochanek PM, Fink EL, Bell MJ, et al: Therapeutic hypothermia: Applications in pediatric cardiac arrest. J Neurotrauma 2009;26(3):421-427.
Quan L, Mack CD, Schiff MA: Association of water temperature and submersion duration and drowning outcome. Resuscitation 2014;85(6):790-794.
Suominen PK, Vallila NH, Hartikainin LM, et al: Outcome of drowned hypothermic children with cardiac arrest treated with cardiopulmonary bypass. Acta Anaesthesiol Scand 2010;54(10):1276-1281.

5. Should an icy cold water drowning be managed differently from a warm water drowning?

Manage a patient who has had a true icy water drowning aggressively because of the possibility of intact survival. Prolonged and maximal resuscitative efforts, including cardiopulmonary bypass or extracorporeal membrane oxygenation, may be indicated for the icy water submersion. However, studies are equivocal on benefits and predictors and likelihood ratios for survival from icy water submersions.

For non–icy water drownings, failure to restore a circulating cardiac rhythm within about 30 minutes after rewarming to 32° to 35° C suggests that further efforts are unlikely to be successful or may lead to significant neurologic devastation with minimal quality of life.

Dyson K, Morgans A, Bray J, et al: Drowning related out-of-hospital cardiac arrests: Characteristics and outcomes. Resuscitation 2013;84(8):1114-1118.
Wollenek G, Honarwar N, Golej J, Marx M: Cold water submersion and cardiac arrest in treatment of severe hypothermia with cardiopulmonary bypass. Resuscitation 2002;52:255-263.

6. Is there a clinical difference between a salt water and fresh water drowning?

There is none. A large clinical study showed no real difference in the clinical presentation of adults who drowned in salt or fresh waters, thus invalidating the mythic physiologic changes reported in one animal study. Clinical experience dictates that both salt water– and fresh water–drowned patients may develop pulmonary edema, anemia is rare, and electrolytes are normal. The explanation may be that humans do not aspirate large volumes of water when they drown. Furthermore, the pulmonary response to drowning, especially the development of pulmonary edema, relates to other factors besides the salinity of the aspirated fluid.

Modell JH: Drowning. N Engl J Med 1993;328:253-256.

7. Is cardiopulmonary resuscitation (CPR) indicated for every patient in cardiac arrest following a drowning?

Survival rates from cardiac arrest following drowning are among the highest, 20% to 31%, reported in pediatric cardiac arrest, although it is unclear why. To be effective, resuscitation should begin as soon as possible after retrieval from the water, at the scene. However, survival from prolonged (>25 minutes) non–icy water drownings is essentially zero, as is survival following CPR for longer than 25 minutes without return of spontaneous circulation (a perfusing blood pressure). There are clearly some patients who are dead at the scene for whom efforts are futile.

Orlowski JP: Prognostic factors in pediatric cases of drowning and near-drowning. JACEP 1979;8:176-179.
Quan L, Mack CD, Schiff MA: Association of water temperature and submersion duration and drowning outcome. Resuscitation 2014;85(6):790-794.

8. What are the tenets of management in resuscitation of the unresponsive drowning patient?

The injury of drowning is hypoxia, and the goal of resuscitation is directed toward the ABCs (airway, breathing, and circulation). Focus initial resuscitation on rapidly restoring oxygenation, ventilation, and adequate circulation. Initiate positive-pressure ventilation and supplemental oxygen as soon as possible. This may restore the patient's own spontaneous

respiratory drive or the patient may continue to need support, at which time endotracheal intubation is appropriate. Begin CPR in any patient who is pulseless, bradycardic, or severely hypotensive. Poor perfusion may be the result of significant myocardial hypoxia. Treat this first with one to two boluses of normal saline (20 mL/kg body weight) for the possibility of hypovolemia; if this fails to restore adequate perfusion, treat cardiac dysfunction with inotropic agents.

Recognition and treatment of hypothermia are the unique aspects of cardiac resuscitation in the drowning victim. Evaluate core temperature on primary assessment, because moderate to severe hypothermia (<32° C) can depress myocardial function and cause arrhythmias. Remove wet clothing to prevent ongoing heat loss; however, in the hemodynamically stable patient, initiate rewarming in the controlled environment of the receiving ED or pediatric intensive care unit (PICU). Pay special attention to avoid overheating and hyperthermia. After successful resuscitation, direct attention toward preventing secondary brain injury by monitoring for hypoxia, hypercapnia, and hypothermia.

Zuckerbraun NS: Pediatric drowning: Current management strategies for immediate care. Clin Pediatr Emerg Med 2005;6:49-56.

Key Points: Drowning

1. Failure to restore a perfusing rhythm within about 30 minutes of resuscitation in a patient with a core temperature of 32° C to 35° C, following a non–icy water drowning, suggests that further efforts are unlikely to be successful.
2. Patients who experienced a drowning but are asymptomatic after 6 to 8 hours of observation can be safely sent home if the social situation permits.
3. Social work involvement is important in the evaluation of any drowning patient for possible abuse, homicide, suicide, or risk-taking behaviors, especially alcohol use.

9. Should prophylactic antibiotics be given to the drowning patient?

No. The only indication for the prophylactic use of antibiotics is drowning in grossly contaminated water (e.g., sewage, retention pond). Instead, initiate antibiotics if signs of sepsis or pneumonia become apparent.

Orlowski JP: Drowning. Rescue, resuscitation, and reanimation. Pediatr Clin North Am 2001;48:627-646.

10. Which tests are helpful in managing the drowning patient?

The patient's clinical examination should guide what tests are needed. At a minimum, perform serial monitoring of vital signs and oxygenation by pulse oximetry, repeated pulmonary examination, and neurologic assessment in all drowning victims. For the alert patient with no or mild respiratory distress, oximetry monitoring may be all that is needed and is a more reliable treatment guide than chest radiography. Several studies have recommended against using an abnormal radiograph as the sole criterion for hospital admission. Reserve blood gas, chest radiography, complete blood count, and electrolytes for the unresponsive patient who is at great risk for complications and bad outcome. Additionally, include a glucose level because it is a useful prognostic factor. Hyperglycemia (glucose level > 250 mg/dL) portends a poor outcome. It is unclear if hyperglycemia should be treated. On the other hand, hypoglycemia must be treated.

Wollenek G, Honarwar N, Golej J, Marx M: Cold water submersion and cardiac arrest in treatment of severe hypothermia with cardiopulmonary bypass. Resuscitation 2002;52:255-263.

11. What is the most common cause of death and disability in hospitalized drowning victims?

Cerebral edema secondary to hypoxic ischemia is the usual cause of death and becomes a clinical challenge at 6 to 18 hours after the drowning event, as may other end-organ failure. Hypoxic encephalopathy in survivors of severe drownings usually results in severe neurologic sequelae.

Suominen PK, Vähätalo R, Sintonen H, et al: Health-related quality of life after a drowning incident as a child. Resuscitation 2011;82(10):1318-1322.

12. Can some drowning patients be sent home from the ED?

Yes. Some postsubmersion patients arrive cheery but wet. They have usually had a short submersion interval (<5 minutes), have minimal signs (mild metabolic acidosis) of a hypoxic insult, and are asymptomatic. Observe these patients in the ED for a minimum of 6 to 8 hours with the focus on neurologic and pulmonary status, including cardiorespiratory monitoring with pulse oximeter. Some initially asymptomatic, alert patients may develop mild respiratory distress (tachypnea, mild hypoxia) and cough at the scene or within a few hours of the submersion. In many cases, their symptoms respond to a brief period of low-flow oxygen and do not mandate hospital admission if they are asymptomatic at 8 hours.

Causey AL: Predicting discharge in uncomplicated near-drowning. Am J Emerg Med 2000;18:9-11.

13. Does every drowning patient need evaluation for cervical spine injury?

No. Routine cervical spine imaging is not indicated for the overwhelming majority of patients. Cervical spine injury is very rare (<1%) in drowning patients and is almost never seen in preadolescent children. It is typically associated with motor vehicle, boating, or personal watercraft accidents; diving; or surfing. In two large studies of over 2000 patients, all cases of cervical spine injury had a history consistent with a high-speed mechanism or clinical signs of serious injury on physical examination. A careful history and examination of the neck are sufficient screening for cervical spine injury for most patients.

Hwang V: Prevalence of traumatic injuries in drowning and near drowning in children and adolescents. Arch Pediatr Adolesc Med 2003;157:50-53.

Watson RS: Cervical spine injuries among submersion victims. J Trauma 2001;51:658-662.

14. Who will survive drowning?

Intact survival or mild neurologic impairment is primarily seen in young children with a submersion duration less than 5 minutes and resuscitation duration less than 10 minutes. Children with normal cardiac and neurologic examinations and responsiveness at the scene virtually always have good outcomes. High morbidity is seen in those with deep coma, apnea, submersion durations longer than 10 minutes, and failure of response to CPR given for longer than 20 minutes. Young children are more likely to survive than adolescents, who are more likely to be declared dead at the scene. This difference is explained by their different submersion scenarios. Young children usually drown where supervision is nearby but has lapsed briefly (often <5 minutes); they also tend to drown in bodies of water, such as bathtubs or swimming pools, where rescue is easy. Conversely, adolescents and adults are more likely to be in larger, deeper, murkier bodies of water, such as lakes and rivers, where rescue is more difficult and the submersion duration is lengthy. When these factors are controlled for, age is not a predictor of outcome.

Quan L: Characteristics of drowning by different age groups. Inj Prev 2003;9:163-168.

15. What are predictors of outcome in drowning?

The key predictor is the patient's mental status after the drowning. Patients who are alert on arrival at the ED or at hospital admission will survive with normal neurologic status. For patients arriving unresponsive to the ED, neurologic outcome is more difficult to predict. Factors upon presentation that will identify patients who will remain in a persistent vegetative state or die include duration of submersion longer than 10 minutes, absent pupillary reflexes, hyperglycemia after resuscitation (glucose level > 250 mg/dL), acidosis (pH < 7.10), and duration of CPR greater than 25 minutes until return of spontaneous circulation. A poor prognosis is most likely when all of these bad prognostic factors are present. Early Glasgow Coma Scale (GCS) assessments fail to adequately distinguish children who will survive intact from those with major neurologic injury. Because no single factor or combination of factors at the time the patient arrives in the ED has achieved greater than 96% accuracy in predicting poor outcome, some recommend resuscitation attempts for all drowning victims on arrival in the ED.

Neurologic examination and progression of symptoms during the first 24 to 72 hours are currently the best prognosticators of neurologic outcome. Children who regain consciousness and neurologic function within 48 to 72 hours, even after prolonged resuscitation, are unlikely to have serious neurologic sequelae. Survivors with good outcomes typically have spontaneous purposeful movements and normal brainstem function within 24 hours; most of those who remain comatose at 24 hours will die or survive with severe neurologic sequelae.

Causey AL: Predicting discharge in uncomplicated near-drowning. Am J Emerg Med 2000;18:9-11.
Dyson K, Morgans A, Bray J, et al: Drowning related out-of-hospital cardiac arrests: Characteristics and outcomes. Resuscitation 2013;84(8):1114-1118.
Suominen PK, Vähätalo R, Sintonen H, et al: Health-related quality of life after a drowning incident as a child. Resuscitation 2011;82(10):1318-1322.

Key Points: Poor Prognostic Signs after Drowning

1. Duration of submersion longer than 10 minutes
2. Absent pupillary reflexes
3. Hyperglycemia (glucose level > 250 mg/dL)
4. Acidosis (pH < 7.1)
5. No spontaneous circulation after 25 minutes of CPR
6. Prolonged coma

16. How does one recognize child abuse as the cause for drowning?

Drowning as child abuse or homicide is most often recognized in the young child whose submersion occurred in the bathtub. Drowning may be the primary injury, as well as a secondary injury when the abuser attempts to revive the child or to conceal other physical injury by placing the child in the tub. The key to recognition of intentional trauma is usually in the history. A caregiver's explanation for the injury that is not compatible with the child's developmental status or a changing or vague history should prompt consideration of child abuse. The physical examination must include a careful search for signs of physical abuse and signs of multiple traumas. Radiographs for fractures are usually not helpful. A critical part of any childhood drowning, regardless of the concern for abuse, is evaluation by social services.

Fugiwara T, Barber C, Schaechter J, Hemenway D: Characteristics of infant homicides: Findings from a US multisite reporting system. Pediatrics 2009;124:e210-e217.
Gillenwater JM: Inflicted submersion in childhood. Arch Pediatr Adolesc Med 1996;150:298-303.

17. How can pediatric drowning be prevented?

Drowning is a multifaceted problem, but several preventive strategies are effective: appropriate supervision of children, formal swim lessons, the presence of lifeguards, barriers to swimming pools, and use of PFDs. Adult supervision of children around any water is essential and can prevent all bathtub-related drownings. Swim lessons that are developmentally appropriate and aimed at the individual child's readiness and skill level may start at an early age and have been shown to provide some level of protection to young children. Four-sided isolation fencing with self-closing, self-latching gates around residential pools is effective in preventing more than 50% of childhood drownings.

The use of U.S. Coast Guard–approved lifejackets (PFDs) is advised with all families spending time around open water, not just those who consider themselves boaters. For adolescents, drownings occur while swimming with friends or while boating. This emphasizes the need for them to learn not just to swim but also water survival skills and to avoid alcohol while swimming.

Brenner RA, Taneja GS, Haynie DL, et al: Association between swimming lessons and drowning in childhood: A case-control study. Arch Pediatr Adolesc Med 2009;163:203-210.
Committee on Injury, Violence, and Poison Prevention: Policy statement—Prevention of drowning. *Pediatrics* 2010;126:178–185.
Quan L, Liller K, Bennett E: Water-related injuries of children and adolescents. In Liller K (ed): Injury Prevention for Children and Adolescents: Research, Practice and Advocacy, 2nd ed. Washington, DC, American Public Health Association, 2012.
Rahman F, Bose S, Linnan M, et al: Cost-effectiveness of an injury and drowning prevention program in Bangladesh. Pediatrics 2012;130(6):e1621-e1628.

18. What medical conditions can increase the risk of drowning?

Seizures are the most common predisposing event for drowning in all age groups, and drowning is the major cause of death in patients with epilepsy. This patient group has 10 times the risk for drowning and fatal drowning compared to those without seizures. The bathtub actually is the highest risk site. Health care providers should advise patients with seizures to take showers, not baths. Involvement in water sports should include

wearing of life jackets when near or in the water and swimming in pools where lifeguards supervise and are notified of the patient's increased risk.

Several recent studies have also identified cardiac abnormalities as risk factors for drowning. Cardiac causes, including arrhythmias, myocarditis, and prolonged QT syndromes, have been found in some children who die suddenly in the water, particularly in those with a family history of cardiac arrest, prior drowning, or QT prolongation.

Kenny D, Martin R: Drowning and sudden cardiac death. Arch Dis Child 2011;96:5-8.

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ELECTRICAL AND LIGHTNING INJURIES

Amanda Pratt

CHAPTER 65

1. What characteristics of electrical energy lend to severe injury?

- In general, the higher the voltage and current, the worse the injury.
- Alternating current (AC) causes intense muscle contractions, thus prolonging the exposure.
- Direct current (DC) can cause significant trauma by throwing the victim from the source.
- AC is three times more dangerous than DC of the same voltage.

2. Which body tissues offer the highest resistance?

From greatest to least resistance: bone > fat > tendon > skin > muscle > nerve. The most important resistor of current flow is skin, because it is typically the site of first contact. Dry skin over palms and soles can have a resistance of 100,000 ohms. When skin is wet, the resistance drops to 2500 ohms; when skin is immersed in water, resistance can be as low as 1500 ohms. Lowered resistance allows more current to penetrate deeper tissue.

Fish RM, Geddes LA: Conduction of electrical current to and through the human body: A review. ePlasty 2009;9:e44.

3. How common is death from electrical injury?

There are about 1000 deaths per year in the United States from electrical injuries.

Lee RC. Injury by electrical forces: pathophysiology, manifestations, and therapy. Curr Probl Surg 1997;34 (9):677-764.

4. Who are the most likely victims of electrical injury?

Small children, teenagers, and working-age adults. Children are exposed primarily at home; teens are more likely to have high-voltage injuries.

Glatstein MM, Ayalon I, Miller E, Scolnik D: Pediatric electrical burn injuries: Experience of a large tertiary care hospital and a review of electrical injury. Pediatr Emerg Care 2013;29:737-740.

5. How does the pathway of the current through the body affect the degree of injury?

The path that a current takes determines the number of organs affected and thus the severity of injury. A vertical path parallel to the body involves almost all vital organs and is thus the most dangerous. A horizontal path from hand to hand spares the brain but can still be fatal because it can involve the heart, spinal cord, or respiratory muscles. A horizontal path through the lower body may cause severe local damage but is not typically fatal.

Bailey B, Gaudreault P, Thivierge RL: Cardiac monitoring of high risk patients after an electrical injury: A prospective multicenter study. Emerg Med J 2007;24:348-352.
Koumbourlis AC: Electrical injuries. Crit Care Med 2002;30:S424-S430.

6. Is a small electrical burn reassuring?

No. The visible burn of an electrical injury only represents a small portion of the tissue damage. Entry and exit wounds are poor predictors of internal damage. Tissue that is initially viable may become ischemic over time.

Glatstein MM, Ayalon I, Miller E, Scolnik D: Pediatric electrical burn injuries: Experience of a large tertiary care hospital and a review of electrical injury. Pediatr Emerg Care 2013;29:737-740.

7. What are the cardiac manifestations of electrical injuries?

- Sinus tachycardia, premature ventricular contractions, reversible QT prolongation, and other arrhythmias have been reported.
- Low-voltage AC may cause ventricular fibrillation.
- High-voltage AC or DC is likely to cause ventricular asystole.

8. How can electrical injuries affect other internal organs?

- Paralysis or tetany of the respiratory muscles and diaphragm may occur.
- Primary central nervous system dysfunction at the respiratory center of the brain can induce apnea.
- Myoglobinuria with subsequent renal failure is possible, but direct renal injury is rare.

9. Describe the neurologic effects of electrical injuries.

- Acute findings may include altered mental status, loss of consciousness, seizures, and paralysis.
- Delayed findings (days to years) may include depression, memory loss, motor neuropathy, and transverse myelitis.

10. What are the dermatologic manifestations of electrical injuries?

- Entrance and exit burns may be seen and should be evaluated to determine the path of current.
- Burns across joints at the flexor creases on both flexor surfaces may be seen, known as "kissing burns."
- Mouth commissure burns are a unique problem in children; other issues are associated with their management (see Question 16).

11. How do I manage patients with significant electrical exposure?

Perform rapid trauma assessment and resuscitation for patients with significant electrical exposure (i.e., high-voltage, DC, or AC injury with respiratory, hemodynamic, or neurologic sequelae). Provide the ABCs of trauma care (airway, breathing and circulation). In patients with DC exposure, immobilize the cervical spine until further clinical and radiographic evaluations are done. Initiate aggressive fluid resuscitation to treat presumed myonecrosis and prevent renal failure. Assess for compartment syndrome due to myonecrosis. Provide local burn care and tetanus prophylaxis. Obtain laboratory data: complete blood count, seven-panel chemistry study, creatine phosphokinase, urinalysis, urine for myoglobin, electrocardiogram (12 lead), and appropriate radiographs. If severe injury or suspected abdominal trauma is present, add liver function tests, amylase, prothrombin time, partial thromboplastin time, and type and crossmatch.

Glatstein MM, Ayalon I, Miller E, Scolnik D: Pediatric electrical burn injuries: Experience of a large tertiary care hospital and a review of electrical injury. Pediatr Emerg Care 2013;29:737-740.

Key Points: Initial Management of Victims of Significant Electrical Injury

1. Treat the victim as a trauma patient.
2. Initiate the ABCs.
3. Provide cervical spine immobilization.
4. Initiate aggressive fluid resuscitation.
5. If the physical examination reveals bruises, distorted long bones, or tenderness, obtain appropriate radiographs.

12. What complications can occur from significant (high tension) electrical injury?

High-voltage injuries may be associated with additional trauma from a fall or from being thrown. About 30% of survivors of high-tension injuries require amputation of some body part.

O'Brien SP, Billmire DA: Prevention and management of outpatient pediatric burns. J Craniofac Surg 2008;19:1034-1039.

13. How should fluid resuscitation be approached in victims of electrical injury?

Do not base fluid resuscitation on the rule of nines as in thermal burns, because there is typically more extensive internal injury than is manifested by the skin findings. Give fluids to patients with rhabdomyolysis to maintain urine output of 1 to 1.5 mL/kg/hour. If there is no evidence of blood in the urine, the goal is urine output of 0.5 to 1 mL/kg/hour.

14. How do I approach, evaluate, and manage the typical child with short-sustained exposure to household current?

Most pediatric electrical exposures are minor. A tailored evaluation with specific attention to cardiac and neurologic issues, as well as local wound care, is warranted. For household, low-voltage exposures (120-240 volts), monitor any patient with loss of consciousness, history of tetany, wet skin, or current path across the heart (hand-to-hand contact) for 4 hours for delayed cardiac rhythm disturbances before making a disposition. If injury is regarded as minor, risk factors are absent, and the general physical examination is normal, brief observation, local wound care, and discharge with appropriate follow-up are sufficient.

15. What are the admission criteria for cardiac monitoring in children who sustain electrical injuries?

Electrocardiogram and hospital admission may not be mandatory if there is minimal injury related to low voltage. Obtain an electrocardiogram if there is a history of tetany or decreased skin resistance by water or burns, or if it is an unwitnessed event. Admit patients to the hospital for 24 hours for cardiac monitoring if there is an abnormal electrocardiogram, past cardiac history, loss of consciousness, or voltage greater than 240 volts.

Bailey B, Gaudreault P, Thivierge RL: Cardiac monitoring of high risk patients after an electrical injury: A prospective multicenter study. Emerg Med J 2007;24:348-352.

Claudet I, Marechal C, Debuisson C, et al: Risk of arrhythmia and domestic low voltage electrical injury. Arch Pediatr 2010;17:343-349.

16. What are the special concerns in children with mouth commissure burns sustained from biting an electric cord?

Such burns occur most frequently in children ages 6 to 36 months. The burn is caused by direct contact with the electrical source as well as an arc burn in contact with the wet mouth surface. Patients are often stable on initial presentation, but as the eschar begins to loosen (after approximately 7-14 days), labial artery bleeding may occur. Local care, along with use of an acrylic oral splint, is the mainstay of treatment. Discharge instructions should include an understanding of the risk of rebleeding during the following 1 to 2 weeks. For the best cosmetic results, delay surgical reconstruction until the burn has fully healed (at least 6-9 months).

Yeroshalmi F, Sidoti EJ, Adamo AK, et al: Oral electrical burns in children—A model of multidisciplinary care. J Burn Care Res 2011;32:e25-e30.

17. List the common pitfalls in evaluation and treatment of electrical injuries.

- Rescuer injuries at the scene due to inappropriately secured active electrical lines
- Failure to immobilize the cervical spine and perform the ABCs of trauma assessment
- Failure to consider occult blunt trauma injuries in children with DC exposure
- Underestimating fluid requirements for the severity of the burn, specifically depth of thermal injury

18. What are the four mechanisms of a lightning strike?

- **Direct strike** (to either the victim or an object held by the victim) has the highest mortality rate.
- **Side flash**, the most common form, involves a lightning strike that jumps from a primary strike area through the surrounding air to the nearby victim.
- **Ground strike** impacts the ground near the victim. The current enters the victim usually through one foot and exits the other.
- **Blast phenomenon** can occur when rapid cooling of air superheated from lightning causes an explosion with enough force to create blunt trauma, including concussive symptoms.

19. What is the voltage potential in a lightning strike?

A typical lightning strike usually exceeds 1 million volts, but its duration is short (on the order of 1/1000 to 1/10,000 of a second). Because of the extreme voltage potential, the mortality rate is approximately 30% and the morbidity rate is approximately 70% for all lightning injuries.

Whitcomb D, Martinez JA, Daberkow D: Lightning injuries. South Med J 2002;95:1331-1334.

20. How successful is cardiopulmonary resuscitation in victims of a lightning strike?

If resuscitation is initiated promptly, the survival rate is significantly higher than in other causes of cardiopulmonary arrest. Because of the high voltage and DC, asystole is the usual initial rhythm. Provide aggressive and persistent efforts.

21. Discuss the rules of triage for lightning victims.

Lightning injuries frequently involve more than one victim, because people tend to seek shelter in groups. The rules of triage are reversed for lightning injuries, and attention should first be given to those not breathing. Victims with signs of life at the scene are highly likely to survive and should not be treated first. Give victims with cardiac or respiratory arrest the greatest efforts, because they have a high likelihood of survival with aggressive cardiopulmonary resuscitation.

Spies C, Trohman RG: Narrative review: Electrocution and life-threatening electrical injuries. Ann Intern Med 2006;145:531-537.

22. Explain feathering burns.

Feathering burns are microburns due to the electron shower that results from the lightning exposure. They are seen within several hours of injury and resolve within 24 hours. These are also called Lichtenberg figures and are pathognomonic for lightning injuries.

23. How are hearing and vision affected by a lightning strike?

Perforated tympanic membranes and sensorineural hearing loss have been reported in up to 50% of victims. Fixed, dilated pupils can result from autonomic effects of the lightning strike and are not a reason to terminate resuscitative efforts. Retinal detachment and optic nerve injury also have been reported.

Key Points: Approach to Victims of a Lightning Strike

1. Reverse the rules of triage. Treat those who are not breathing first.
2. Pursue aggressive cardiopulmonary resuscitation.
3. Remember that a blown pupil is not always an ominous sign; continue resuscitation.

24. Define *keraunoparalysis*.

Keraunoparalysis is the vasospastic paralysis of limbs unique to lightning strikes. It may last several hours before resolution. *Keraunos* is Greek for "thunderbolt" and is used as a prefix to describe lightning-related phenomena.

25. What are the risks of sustaining a lightning injury indoors?

The dangers of lightning injury to persons who are indoors are not well known. The two potential risks are telephone and plumbing lines. A current surge on a telephone line, due to a direct lightning strike or, more commonly, to current traveling along the ground, may result in a large voltage difference between telephone apparatus and user. Deaths are rare. Acoustic trauma is the most common form of injury. Current also may travel in grounded pipes to bathtubs and showers. Such injuries have high morbidity and mortality rates, because the ground current is easily conducted by the water in the bathtub or shower.

26. Fact or fiction: The rubber tires on a vehicle are what protect an individual inside the automobile from injury after a lightning strike.

Fiction. The protective effect comes from the metal-roofed vehicle, which allows electrical energy to travel over the car body. Therefore, soft-topped jeeps and convertibles are not safe shelters during a storm.

27. What is the "30-30 rule"?

This rule is an easy way to remember how to avoid lightning injury. If the time between seeing lightning and hearing thunder is less than 30 seconds, an individual should seek shelter. Outdoor activities should not resume until 30 minutes after the last sound of thunder or sighting of lightning. Another easy reminder that can be taught to children is this: "If you see it, flee it; if you hear it, clear it."

O'Keefe Gatewood M, Zane RD: Lightning injuries. Emerg Med Clin North Am 2004;22:369-403.

HEAT-RELATED ILLNESS

Brenda J. Bender

CHAPTER 66

1. What is in the spectrum of heat-related illness?

Heat-related illness includes heat cramps, heat exhaustion, and heat stroke.

Key Points: Prevention of Heat-Related Illness

1. Wear loose-fitting, lightweight clothing.
2. Rest often.
3. Seek shade.
4. Avoid exercise or strenuous activity, especially during the hottest part of the day.
5. Drink frequently.

2. What is the difference between heat exhaustion and heat stroke?

Heat exhaustion and heat stroke are on a continuum of heat-related illnesses that occur when the body's heat loss mechanisms are overwhelmed or insufficient to respond to environmental demands. Heat exhaustion is the less severe of the two conditions and is believed to represent reversible heat overload, whereas heat stroke is characteristically associated with irreversible tissue damage.

Key Points: Symptoms of Heat Exhaustion

1. Severe thirst
2. Excessive sweating
3. Dilated pupils
4. Lightheadedness
5. Nausea/vomiting

3. Why does heat-related illness occur?

High ambient temperature and humidity contribute. Humidity limits cooling and sweat evaporation, and high ambient temperatures prevent heat dissipation by radiation or convection.

Ewald MB, Baum CR: Environmental emergencies. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 791-794.

4. Who is at increased risk for heat-related illness?

People at increased risk include those involved in extreme activities in a hot, humid atmosphere, people with an underlying illness or obesity, those with decreased fluid intake, and those who are using alcohol or certain drugs such as cocaine, salicylates, amphetamines, phenothiazines, antihistamines, and anticholinergics.

Heat stroke typically occurs in extremes of age after excessive exertion in hot weather such as heat waves. Young children who rely on others for liquid intake are at higher risk. Neonates are perhaps at greatest risk because they have poorly developed thermoregulatory mechanisms and depend on others to remove them from a hot environment and provide adequate hydration.

Ewald MB, Baum CR: Environmental emergencies. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 791-794.

5. Which diseases increase the risk for heat-related illness?

- Diabetes insipidus, diabetes mellitus (associated with excessive fluid loss)
- Skin diseases
- Cystic fibrosis (salt losses in sweat)
- Mental retardation (inadequate drinking)
- Spina bifida (sweat gland dysfunction, suboptimal sweating)
- Hypothyroidism
- Cardiovascular disease (excessive sweating in some cyanotic heart disease)
- Anorexia nervosa (abnormal hypothalamic function)
- Obesity
- Sickle cell trait (see Question 6)

Mistry RD: Heat illness. In Hoffman RJ, Wang VJ, Scarfone RJ (eds): Fleisher and Ludwig's 5-Minute Pediatric Emergency Medicine Consult. Philadelphia, Lippincott Williams & Wilkins, 2012, pp 468-469.

6. How can having sickle cell trait impact a patient with heat-related illness?

Although sickle cell trait is often thought of as a benign condition, deaths from *exertional heat stroke* have been reported in adolescent athletes with sickle cell trait. Patients with sickle cell trait can have increased sickling with hypoxemia, acidosis, hyperthermia, and red blood cell dehydration. Patients with sickle cell trait are at increased risk for rhabdomyolysis or sudden death during exercise, even if not in an extremely warm environment. This danger is especially likely when the exercise is intense, done by unconditioned people, and done at high altitudes. These factors are all made worse if the individual is hyperthermic or dehydrated, but this is not always the case with exertional heat stroke (see Question 28).

The exact pathophysiology is not known, but rhabdomyolysis in these patients is most likely related to the sickling of the red blood cells in the exercising muscle. Recent research has shown that the skeletal muscle capillary structure in patients with sickle cell trait may differ when compared to control subjects. The biopsies from muscles of patients with sickle cell trait demonstrate a higher proportion of larger microvessels and a decrease of capillary density and degree of vascular tortuosity. These changes affect muscle perfusion and contribute to exercise-related acidosis and rhabdomyolysis.

Connes P, Reid H, Hardy-Dessources MD, et al: Physiological responses of sickle cell trait carriers during exercise. Sports Med 2008;38(11):931-946.

Eichner ER: Sickle cell trait in sport. Curr Sport Med Rep 2010;9(6):347-351.

Key NS, Derebail VK: Sickle-cell trait: Novel clinical significance. Hematol Am Soc Hematol Educ Program 2010;2010:418-422.

Vincent L, Feasson L, Oyono-Enguelle S, et al: Remodeling of skeletal muscle microvasculature in sickle cell trait and alpha-thalassemia. Am J Physiol Heart Circ Physiol 2010;298(2):H375-H384.

7. Why are children at increased risk for heat-related illnesses?

Children may be less cognizant of the need to stay hydrated when exercising or playing in warm weather. Children are also less efficient at thermoregulation: they have a slower speed of acclimatization, have a lower sweating rate, produce more metabolic heat per kilogram of body weight, and have a higher set point.

Ciorciari AJ: Environmental emergencies. In Crain EF, Gershel JC (eds): Clinical Manual of Emergency Pediatrics, 5th ed. New York, Cambridge University Press, 2010, pp 205-207.

8. How is heat produced?

Heat is produced as a byproduct of basal metabolism, through muscle activity (shivering), or through the effects of thyroxine and sympathetic stimulation on cellular processes.

9. How is heat lost?

Heat is lost through *conduction* to objects and air, *convection* through air or liquid that surrounds tissues, *evaporation*, and *radiation* of infrared energy.

10. How does sweat production increase?

Exercise and emotional states cause the release of epinephrine and norepinephrine, which cause increased sweat production.

11. What is acclimatization?

Acclimatization is the natural physiologic adjustment to heat exposure that usually occurs over 7 to 14 days. This adjustment allows for improved sweating and cardiac performance.

Mistry RD: Heat illness. In Hoffman RJ, Wang VJ, Scarfone RJ (eds): Fleisher and Ludwig's 5-Minute Pediatric Emergency Medicine Consult. Philadelphia, Lippincott Williams & Wilkins, 2012, pp 468-469.

12. What happens to the body's salt production in the heat?

Initially, with heat exposure a child will lose a lot of salt, up to 15 to 20 g/day. Over time, aldosterone production increases, causing more NaCl reabsorption in the sweat gland ducts, and salt production normalizes to 3 to 5 g/day.

Ewald MB, Baum CR: Environmental emergencies. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 791-794.

13. What is the cause of heat cramps?

Heat cramps occur when a teenager loses excessive salt from heavy sweating after strenuous physical activity, leading to electrolyte depletion.

Heller JL: Heat emergencies. Medline Plus Medical Encyclopedia. Available at http://www.nlm.nih.gov/medlineplus/ency/article/000056.htm.

14. What are the symptoms of heat cramps?

Symptoms of heat cramps include sudden onset of intermittent, brief severe cramps or spasm, usually in large muscles such as the hamstrings or gastrocnemius, or in the abdomen. Pain usually occurs after exercise, when the child is relaxing or showering.

Ciorciari AJ: Environmental emergencies. In Crain EF, Gershel JC (eds): Clinical Manual of Emergency Pediatrics, 5th ed. New York, Cambridge University Press, 2010, pp 205-207.

Ewald MB, Baum CR: Environmental emergencies. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 791-794.

Key Points: Symptoms of Heat Cramps

1. Sweating
2. Muscle spasm or cramps
3. Weakness
4. Lightheadedness

15. Are there any abnormal laboratory values with heat cramps?

With heat cramps, blood levels of sodium and chloride are often decreased and there is absent urinary sodium.

16. Who most often suffers from heat cramps?

Heat cramps are most often seen in highly conditioned persons who are acclimatized. They usually have good water replacement, but inadequate salt replacement.

17. How are heat cramps treated?

A patient suffering from heat cramps should rest in a cool place, drink fluids, and increase salt intake.

18. What causes heat exhaustion?

Heat exhaustion is caused by dehydration—excessive sweating with inadequate water and salt intake. The two types of heat exhaustion are water depletion and salt depletion.

19. What causes water depletion heat exhaustion and what are the symptoms?

Water depletion heat exhaustion occurs in the unacclimatized person who exerts himself in the heat and has poor water replacement. Symptoms typically include high body temperature (38° C to 39° C), thirst, headache, vomiting, hypotension, lethargy, tachycardia, myalgias, muscle cramps, and dizziness. If unattended, heat exhaustion may progress to heat stroke.

Ewald MB, Baum CR: Environmental emergencies. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 791-794.

20. Which laboratory abnormalities may be present with water depletion heat exhaustion?

With water depletion heat exhaustion laboratory abnormalities include hypernatremia, hyperchloremia, elevated hematocrit, and elevated urine specific gravity.

21. What causes salt depletion heat exhaustion and what are the symptoms?

Salt depletion heat exhaustion is seen in an unacclimatized person who exerts himself in hot weather and who has poor salt replacement. It is also seen in patients with cystic fibrosis. Symptoms include a temperature of 39° C to 40° C, weakness, fatigue, headache, nausea, vomiting, muscle cramps, tachycardia, and orthostatic hypotension.

Ewald MB, Baum CR: Environmental emergencies. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 791-794.

22. Which laboratory abnormalities are found with salt depletion heat exhaustion?

In salt depletion heat exhaustion you may find hyponatremia, elevated hematocrit, and severely decreased urinary sodium.

Key Points: Physical Examination Findings of Heat Stroke

1. Anhidrosis
2. Fever
3. Red skin
4. Constricted pupils
5. Rapid shallow breathing
6. Central nervous system (CNS) dysfunction

23. How is heat exhaustion treated?

Treat a patient with heat exhaustion with rest in a cool location and rehydration—either orally or intravenously. Increase salt intake, and avoid alcohol and caffeine. In severe salt depletion from heat exhaustion, consider giving intravenous hypertonic saline.

Ewald MB, Baum CR: Environmental emergencies. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 791-794.

24. What is heat stroke?

Heat stroke is a life-threatening emergency. This condition has a greater than 50% mortality rate. It is the third most common cause of exercise-related death among U.S. high school athletes (after head injury and cardiac disorders). Heat stroke is characterized by a body temperature higher than 40° C, flushed appearance, red and hot skin, change in mental status with severe CNS dysfunction, and anhidrosis. Often, sweating stops before the onset of heat stroke.

The patient may have circulatory collapse, rhabdomyolysis, renal failure, lightheadedness, muscle cramps, nausea, vomiting, tachycardia, tachypnea, and CNS dysfunction (see Question 25).

Ciorciari AJ: Environmental emergencies. In Crain EF, Gershel JC (eds): Clinical Manual of Emergency Pediatrics, 5th ed. New York, Cambridge University Press, 2010, pp 205-207.
Ewald MB, Baum CR: Environmental emergencies. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 783-804.
Glazer JL: Management of heatstroke and heat exhaustion. Am Fam Physician 2005;71(11):2133-2140.
Mistry RD: Heat illness. In Hoffman RJ, Wang VJ, Scarfone RJ (eds): Fleisher and Ludwig's 5-Minute Pediatric Emergency Medicine Consult. Philadelphia, Lippincott Williams & Wilkins, 2012, pp 468-469.

Key Points: Treatment of Heat-Related Illness

1. Remove clothing.
2. Move the patient to a cooler location.
3. Increase salt intake.
4. Provide fluid replacement and maintenance.
5. Make active cooling available.
6. Give cardiovascular support.

25. What are the CNS manifestations of heat stroke?

CNS dysfunction can be manifested in many ways, including a sense of impending doom, headache, dizziness, weakness, confusion, euphoria, gait disturbance, combativeness, seizures, posturing, and coma. The early clinical symptoms are sometimes overlooked because they often are perceived to be the normal result of exertion. Often the severity of the problem is realized only when the victim collapses suddenly.

Glazer JL: Management of heatstroke and heat exhaustion. Am Fam Physician 2005; 71:2133-2142.

26. What cardiac findings are present in heat stroke?

In heat stroke initially there is a rapid and full pulse and an elevated pulse pressure. As heat stroke progresses, peripheral vascular resistance decreases and vasodilation occurs to increase cardiac output.

Ewald MB, Baum CR: Environmental emergencies. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 791-794.

27. List the possible serious complications of heat stroke.

Acidosis, electrolyte imbalance, adynamic ileus, arrhythmias, shivering, seizures, rhabdomyolysis, disseminated intravascular coagulation, renal failure, hepatic damage, permanent neurologic sequelae, and death may occur.

Glazer JL: Management of heatstroke and heat exhaustion. Am Fam Physician 2005;71:2133-2142.

28. What are the two types of heat stroke and how do they differ?

Classic heat stroke usually involves a child with poor water intake in a very warm climate. It has a slow onset and there is no hypoglycemia or rhabdomyolysis. *Exertional heat stroke* typically affects a child who has been involved in prolonged activity, usually during the initial phases of training. Exertional heat stroke is less common and is usually seen in younger individuals. It is not associated with a markedly elevated core temperature or anhidrosis. In many cases the ambient temperature is not as high as that seen in cases of classic heat stroke. Exertional heat stroke has a rapid onset, and there is often lactic acidosis, hypoglycemia, hypocalcemia, and rhabdomyolysis.

Ciorciari AJ: Environmental emergencies. In Crain EF, Gershel JC (eds): Clinical Manual of Emergency Pediatrics, 5th ed. New York, Cambridge University Press, 2010, pp 205-207.

Pretzlaff RK: Death of an adolescent athlete with sickle cell trait caused by exertional heat stroke. Pediatr Crit Care Med 2002;3:308-310.

29. What factors determine if a patient will survive heat stroke?

The most important prognostic sign is duration of the heat stroke. A patient is most likely to survive if that person can maintain cardiac output.

Ewald MB, Baum CR: Environmental emergencies. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 791-794.

Ciorciari AJ: Environmental emergencies. In Crain EF, Gershel JC (eds): Clinical Manual of Emergency Pediatrics, 5th ed. New York, Cambridge University Press, 2010, pp 205-207.

30. Which laboratory abnormalities are seen in heat stroke?

In heat stroke sodium and chloride can be normal or elevated, and glucose can be normal or decreased. Calcium is usually decreased, and serum creatinine phosphokinase (CPK) is typically elevated.

31. What are the priorities of treating heat stroke?

The two priorities of treating heat stroke are eliminating hyperpyrexia and supporting the cardiovascular system. First, bring the patient into a cool location and remove all clothing. Actively cool the patient by spraying him with lukewarm water and positioning fans to blow air across the body, and apply ice packs to the neck, groin, and axilla. In extreme cases consider iced peritoneal lavage. Use 5% dextrose in 0.5 normal saline for maintenance fluid and bolus with normal saline or lactated Ringer's solution (20 mL/kg) as needed for resuscitation. Use other fluids as needed to correct electrolyte abnormalities. Monitor the child's temperature using a rectal probe, heart rate, electrocardiogram (ECG), blood pressure, pulses, perfusion,

urine output, and CNS function. Treat shivering with lorazepam. Consider extracorporeal membrane oxygenation (ECMO) in extreme situations.

Ciorciari AJ: Environmental emergencies. In Crain EF, Gershel JC (eds): Clinical Manual of Emergency Pediatrics, 5th ed. New York, Cambridge University Press, 2010, pp 205-207.
Ewald MB, Baum CR: Environmental emergencies. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 791-794.
Glazer JL: Management of heatstroke and heat exhaustion. Am Fam Physician 2005;71(11):2133-2140.

32. What medication should you use for cardiovascular support for patients with heat stroke?

Use dobutamine at 5 to 20 μg/kg/minute as needed to support the cardiovascular system. Dobutamine is a β-receptor agonist that increases myocardial contractility and maintains peripheral vasodilation.

Ewald MB, Baum CR: Environmental emergencies. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 791-794.

33. How do you treat myoglobinuria related to heat stroke?

Treat myoglobinuria with diuresis. Maintain urine output at more than 1 mL/kg/hour, and if needed use furosemide at 1 mg/kg. Some recommend giving mannitol at 0.25 to 1 g/kg.

Ewald MB, Baum CR: Environmental emergencies. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 791-794.

34. Which laboratory values need to be assessed in heat stroke?

For patients with heat stroke, check the following: complete blood count (CBC), prothrombin time/partial thromboplastin time (PT/PTT), electrolytes, blood urea nitrogen (BUN), creatinine, calcium, phosphorus, CPK, urinalysis (including myoglobin), and arterial blood gas.

35. Which medications should be avoided in managing patients with heat stroke?

Avoid α-agonists, such as epinephrine and norepinephrine, because they cause peripheral vasoconstriction leading to poor heat dissipation. Do not use atropine and other anticholinergic drugs because they inhibit sweating. There is no role for ibuprofen or acetaminophen with this condition.

36. What happens to an infant left in a car on a very hot day?

Every year, infants die when left unattended in unventilated automobiles in the sun. Temperatures can reach 60° C within 15 minutes when a car is left in the sun at 30° C to 40° C.

The average temperature increase in a closed vehicle is 3.2° F for every 5-minute interval, with 80% of the temperature rise happening during the first 30 minutes. Unfortunately, keeping the vehicle windows slightly opened DOES NOT decrease the rate of temperature rise or decrease the maximum temperature reached in the vehicle.

McLaren C, Null J, Quinn J: Heat stress from enclosed vehicles: Moderate ambient temperatures cause significant temperature rise in enclosed vehicles. Pediatrics 2005;116;e109-e112.

37. How can heat-related illnesses be avoided?

To prevent heat-related illness, encourage children to wear loose-fitting, lightweight, and light-colored clothing in hot weather and during exertion, with as much skin exposed as possible to allow evaporative dissipation of heat. Instruct children to rest often and seek shade in warm weather. Tell them to modify physical activity in the face of high ambient temperature and humidity and to drink frequently. Assure that athletes are well hydrated before initiating physical activity and that they continue to consume cold water throughout the exercise period and after exercise has been completed to avoid dehydration. In warm weather, ensure at least a 2-hour break between same-day events. Athletes should participate in a preseason conditioning program and allow a period of acclimation when exercising in the hot summer months.

Bergeron MF, Devore C, Rice SG: Climatic heat stress and exercising children and adolescents. Council on Sports Medicine and Fitness and Council on School Health. Pediatrics 2011;128;e741-e747.
Glazer JL: Management of heatstroke and heat exhaustion. Am Fam Physician 2005;71:2133-2142.
Heller JL: Heat emergencies. Medline Plus Medical Encyclopedia. Available at http://www.nlm.nih.gov/medlineplus/ency/article/000056.htm.

38. How much should a child drink during physical activity to stay well hydrated?

A child aged 9 to 12 years should drink 100 to 250 mL (3-8 oz) every 20 minutes. The adolescent needs to drink 1 to 1.5 L (34-50 oz) each hour.

Bergeron MF, Devore C, Rice SG: Climatic heat stress and exercising children and adolescents. Council on Sports Medicine and Fitness and Council on School Health. Pediatrics 2011;128;e741-e747.

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CHAPTER 67

HYPOTHERMIA

Howard M. Corneli

1. Are children more prone to hypothermia than adults?

Yes. A primary reason is that children's bodies have larger surface-to-mass ratios. Children may also possess less insulation, a faster metabolism, and smaller energy reserves.

2. Why is hypothermia so often overlooked in pediatric patients in the emergency department (ED)?

Some clinical thermometers only measure as low as 94° F (34° C). Clinicians may think of hypothermia only with severe environmental exposure, when in fact it may occur after prolonged exposure in less cold conditions, after any immersion in cold or warm water, with any major illness or trauma, after resuscitation or transport, and with inapparent causes such as drug ingestion or child abuse.

3. But don't physicians recognize cold patients?

Not always. Only in the early stages of hypothermia do patients appear cold. In severe hypothermia, signs such as shivering, pallor, cyanosis, and agitation are replaced by flushing, edema, and muscular rigidity, so that the coldest patients are the most likely to have their hypothermia overlooked. These are the same patients, though, who sometimes benefit from hypothermic protection.

4. List some risk factors for hypothermia.

Trauma
Severe illness
Immersion or submersion
Exposure to wind or cold air
Central nervous system illness or injury
Hypothalamic dysfunction
Endocrine disease
Metabolic impairment
Iatrogenic causes
Rescue
Transport
Resuscitation
Intoxication, especially with alcohol, barbiturates, or phenothiazines
Burns and weeping dermatoses
Child abuse (e.g., cold water immersion)

5. Is there an electrocardiogram (ECG) clue to hypothermia?

The J (Osborne) wave (Fig. 67-1) is diagnostic but is seen in only 10% to 80% of cases.
Nolan J, Soar J: Images in resuscitation: The ECG in hypothermia. Resuscitation 2005;64:133.

6. How should temperatures be taken to detect hypothermia?

A low-reading thermometer must be used. Oral, axillary, and skin scanning thermometers are unreliable, and tympanic temperatures have not been studied. Rectal temperatures must be taken deep in the rectum for at least several minutes, and may still be subject to damping, artifact, and time lags.

In critical patients or those with marked hypothermia, flexible temperature probes in the bladder, esophagus, or nasopharynx best reflect core temperatures and provide the vital ability to track temperature over time. These should be readily available in EDs.
Brown DJ, Brugger H, Boyd J, et al: Accidental hypothermia. N Engl J Med 2012;367(20):1930-1938.

7. Define the approximate stages of hypothermia.

- **Mild hypothermia:** Core temperatures range from approximately 32° to 35° C, and the body attempts to combat heat loss by shivering, increasing metabolism, and vasoconstriction.
- **Moderate hypothermia:** Core temperatures range from 28° to 32° C, and compensatory mechanisms begin to fail. Mental status, respiration, and circulation may diminish.

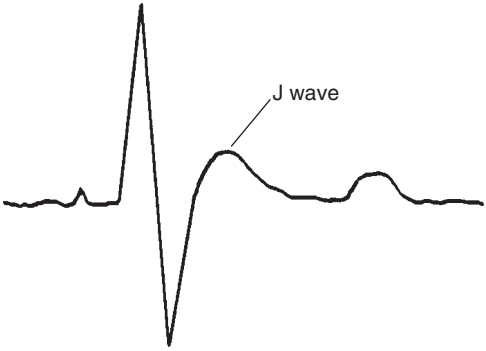

Figure 67-1. Electrocardiogram tracing showing Osborne or J wave.

- **Severe hypothermia:** Core temperatures lower than 28° C lead to failed thermoregulation, metabolic shutdown, and paradoxical vasodilatation accompanied by hypovolemia, decreased perfusion, and stupor or coma.

8. Describe methods for converting Fahrenheit degrees to Celsius degrees.

The upper limit of hypothermia, 35° C, is 95° F. The threshold of severe hypothermia, 28° C, can be inverted to yield 82° F. Starting with these landmarks, an approximate 2:1 ratio between degrees Fahrenheit and degrees Celsius gives workable conversions. Online conversion tools are readily available, for example, by typing a phrase such as "82 F in C" into the search field of popular search engines.

9. Why do patients die from hypothermia during rescue and resuscitation?

Afterdrop is the continued cooling of the body after removal from cold locations. It increases markedly with exertion or with peripheral rewarming.

Rewarming shock is almost universal in severe or prolonged hypothermia, as hypovolemia is caused by profound diuresis and by fluid shifts out of the vasculature, worsened by sludging of cold, thick blood, and augmented by cardiac collapse, vascular dilation, and loss of autoregulation. Shock and death often result unless copious warmed intravenous (IV) fluids can be provided.

Asystole and *ventricular fibrillation* are common end points in severe hypothermia. Profound bradycardia and hypotension may be adequate to maintain the minimal metabolic needs of patients with core temperatures below 28° C, but further cooling, rewarming shock, and patient exertion have all been linked to deterioration from fibrillation.

10. Are advanced life support protocols altered by hypothermia?

At one time, opinion suggested that endotracheal intubation or the performance of cardiopulmonary resuscitation (CPR) should be withheld in some cases of severe hypothermia and that all medications and electrical cardioversion should be avoided.

Today, evidence endorses gentle endotracheal intubation and standard CPR if indicated. In severe hypothermia, focus efforts mostly on rapid, effective rewarming and patient support. Medications including vasopressors and antiarrhythmics may be tried judiciously; their half-life may be prolonged. Cardioversion may be attempted at standard energy settings. Frequent repetition or increases in drug dosage or energy delivery, however, may be deferred. Warming alone may restore the heart rate and blood pressure and has produced spontaneous conversion from asystole to fibrillation to a perfusing rhythm.

CPR and intubation may be difficult because of muscle rigidity. Gentle intubation may be aided by adjuncts such as video laryngoscopy. CPR is performed using standard techniques and rates.

Vanden Hoek TL, Morrison LJ, Shuster M, et al: Part 12: Cardiac arrest in special situations: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation 2010;122(18 Suppl 3):S829-S861.

11. Name some standard treatments that are safe and effective in hypothermia.
Administration of oxygen and maintenance of the airway are basic. Almost all hypothermic patients are severely volume depleted, and aggressive volume replacement with warmed isotonic IV fluid is vital to prevent rewarming shock. Infusion of dextrose is indicated if the blood glucose is low or unknown.

12. What common treatments are *not* routinely recommended for hypothermia?
Precautionary antibiotics or corticosteroids are not beneficial in hypothermia. Acidosis is physiologic in the hypothermic state, and the use of bicarbonate has provoked dangerous alkalosis after rewarming. Insulin for hyperglycemia is contraindicated; the condition will usually resolve with rewarming, and the insulin will begin to work only at that time.

13. Isn't room-temperature IV fluid thermally neutral?
No. At approximately 70° F (21° C), room-temperature IV fluid is colder than all but the coldest hypothermia patients. The infusion of room-temperature IV fluid has been associated with further cooling of patients and also with shock, ventricular fibrillation, cardiac arrest, and death. Any rapid infusion or bolus fluids must be heated effectively to approximately 40° to 44° C.

14. What is the best way to deliver warm IV fluids in the ED?
Unless warm fluids are given very rapidly through specifically designed short, large-bore IV tubing, they cool further before reaching the patient. Effective warming requires countercurrent blood warmers designed for rapid warming and infusion, such as those used in trauma care. These warmers use short, large-bore, insulated or plumbed tubing to deliver warm fluid to the patient.

15. Compare three rewarming strategies.
Passive rewarming involves only a warm room and dry blankets. This method is associated with increased morbidity and mortality risks and has no demonstrated role in pediatrics.

Active external rewarming traditionally involved measures such as warmed blankets or baths, plumbed pads, chemical hot packs, and heat lamps. Modern methods employ forced-air warming. Although available in many settings, these techniques can promote the dumping of cold, acidotic blood from the periphery onto the core and increase metabolic demand and circulatory work prior to volume expansion or cardiac rewarming. Thus, they potentiate afterdrop, rewarming shock, and fibrillation. These methods may be especially harmful in severe hypothermia and are also less effective as circulation fails.

Core rewarming methods range from warmed IV fluids and heated, humidified oxygen to warmed irrigation of the bladder, stomach, or, most effectively, left pleural cavity, and finally to rewarming using heart-lung pumps or extracorporeal membrane oxygenation (ECMO). In the order listed, these methods are increasingly rapid and effective and provide increasing support for patients. All tend to decrease the risk of afterdrop and rewarming shock. Core rewarming is preferred in moderate to severe hypothermia; the more aggressive methods are indicated especially for circulatory compromise or cardiac arrest.

The benefits of extracorporeal rewarming for severe hypothermia, especially in the presence of circulatory failure, warrant advance planning to deploy a pediatric team capable of these measures, or to transport children with severe hypothermia to regional centers with such teams. If such methods are completely unavailable, clinicians are reminded that remarkable survivals from severe hypothermia are still reported with less advanced rewarming methods.

Walpoth BH, Walpoth-Aslan BN, Mattle HP, et al: Outcome of survivors of accidental deep hypothermia and circulatory arrest treated with extracorporeal rewarming. N Engl J Med 1997;337:1500-1505.

16. If active external rewarming is indicated, what is the preferred method?
Current evidence suggests that forced-air warming using devices such as the Bair Hugger (Arizant Healthcare, Eden Prairie, MN) is the most effective method and may be associated with less afterdrop in patients. There is little evidence or logic to suggest that any external rewarming method would work well in patients with absent or severely depressed circulation.

17. What is the ideal rewarming rate?

Rapid or profound cooling warrants rapid rewarming. Most children suffering from acute hypothermia will tolerate rapid rewarming if given aggressive volume expansion and other support. The previously mentioned core methods are listed in approximate order of increasing effectiveness. If the method chosen does not produce rapid rewarming, employ a more effective method.

Key Points: Hypothermia in Children

1. Children are particularly prone to hypothermia, especially with serious illness or injury.
2. Serious hypothermia often goes undetected by clinicians lacking a high degree of suspicion.
3. Hypothermia is generally harmful, although there is occasional remarkable recovery after prolonged resuscitation.
4. Experts no longer recommend extensive modifications to standard resuscitation in severe hypothermia.
5. Effective rewarming therapy is vital, yet often difficult.

18. Discuss the ED treatment of frostbite.

After core rewarming to at least 35° C and only after any risk of further chilling is past, rapidly and definitely rewarm frostbitten body parts. Do this in a bath carefully maintained at 40° to 42° C for 15 to 30 minutes, until flushing indicates reperfusion. Narcotic analgesia is indicated. Update tetanus immunization and elevate the affected body part.

Seek consultation regarding management from specialists in burn, plastic, or trauma surgery. Thrombolytic therapy has been shown to markedly reduce the risk of later amputation, and other drugs may be helpful.

Bruen KJ, Ballard JR, Morris SE, et al: Reduction of the incidence of amputation in frostbite injury with thrombolytic therapy. Arch Surg 2007;142(6):546-551; discussion 551-553.

Murphy JV, Banwell PE, Roberts AH, et al: Frostbite: Pathogenesis and treatment. J Trauma 2000;48:171-178.

Key Points: Treatment of Frostbite

1. Rapidly rewarm frostbitten parts in a bath of water at 40° to 42° C.
2. Give narcotic analgesics.
3. Consult surgical colleagues regarding management and admission to the hospital.

19. Does hypothermia alter usual prognostic indicators?

Yes. Remarkable survivals occur due to cerebral protection in severe hypothermia. Intact survival has been reported after submersion for up to 66 minutes, immersion for hours, exposure for days, core temperatures as low as 14° C, and CPR for up to 9 hours. Neurologic recovery is often slow.

20. Are there exceptions to the rule that "no one is dead until he's warm and dead"?

Yes. This does not apply when there are injuries that are incompatible with life, if a patient cannot be rescued from a cold environment in the first place, or if maximal available rewarming methods are ineffective and transport is not an option. Standard time limits may be employed if there is no return of spontaneous circulation within 30 minutes of warming to 32° to 35° C.

Drowning in water that is not "icy cold" (defined as <50° F or 10° C) confers no cerebral protection. Caution is required if (1) immersion may have preceded submersion, (2) submersion times are unclear, or (3) the exact temperature of cold water is unknown.

Laboratory markers of nonsurvivability remain unproved. Although some experts have suggested that a serum potassium level greater than 10 to 12 mmol/L may be incompatible with life, at least one child has survived hypothermia with a potassium level of 11.8 mmol/L.

Brown DJ, Brugger H, Boyd J, et al: Accidental hypothermia. N Engl J Med 2012;367(20):1930-1938.

Dobson JA, Burgess JJ: Resuscitation of severe hypothermia by extracorporeal rewarming in a child. J Trauma 1996;40(3):483-485.

Vanden Hoek TL, Morrison LJ, Shuster M, et al: Part 12: Cardiac arrest in special situations: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation 2010;122(18 Suppl 3):S829-S861.

CHEMICAL AND BIOLOGIC TERRORISM

Frederick M. Henretig and Theodore J. Cieslak

CHAPTER 68

1. Why is terrorism (particularly chemical and biologic terrorism) relevant to those treating children in the emergency department (ED)?

Increasingly, children are the victims of acts of terrorism. Highlighted by Timothy McVeigh's references to child fatalities as "collateral damage" during the Oklahoma City bombing of April 1995, the intentional targeting of children has become tragically commonplace, as evidenced by attacks such as the one that occurred at a school in Beslan, Russia, in September 2004. That incident, which resulted in 334 fatalities (including 186 children), preceded additional attacks targeting children at an Amish school in Pennsylvania in 2006; at a camp for teenagers in Utoya, Norway, in 2011; and at an elementary school in Newtown, Connecticut, in 2012, among others. Coincident with the intentional targeting of children is, perhaps, a trend toward the use of "unconventional" weapons of terror, or so-called weapons of mass destruction. In 1984, members of the Rajneeshee cult used *Salmonella Typhi* in a series of intentional poisonings that affected 751 persons in The Dalles, Oregon, including 142 teenage patrons of a popular pizza parlor. In 1995, the Aum Shinrikyo cult killed 12 and caused thousands to become ill by intentionally releasing sarin nerve agent in the Tokyo subway system (see Question 7). Finally, in October 2001, a troubled scientist disseminated anthrax spores via the U.S. mail, killing 5 and injuring 17 in an attack upon a nation deeply troubled by the 9/11 attacks of the previous month.

American Academy of Pediatrics: Chemical-biological terrorism and its impact on children—A subject review. Pediatrics 2000;105:662-670.

Cieslak TJ, Henretig FM: Biological and chemical terrorism. In Kliegman RM, Stanton BF, Schor NF, St. Gene JW III, Behrman RE (eds): Nelson's Textbook of Pediatrics, 19th ed. Philadelphia, Saunders Elsevier, 2011, pp 2454-2457.

Scarfone RJ, Henretig FM, Cieslak TJ, et al: Emergency department recognition and management of victims of biological and chemical terrorism. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 125-152.

2. Why might terrorists choose biologic and chemical weapons?

The term *weapons of mass destruction* (WMD) has been used to denote weapons using nuclear, biologic, or chemical agents in devices intended to kill and injure large numbers of victims. Biologic and chemical weapons are believed by U.S. military and counterterrorism experts to constitute a likely mode of WMD attack on civilian populations by terrorist groups. These agents are relatively inexpensive and technically less difficult to produce and deploy than nuclear weapons, the raw materials for their production are less regulated, and there is wide access to biologic and chemical agent information via the Internet.

Chemical and biologic weapons can be deployed easily, using relatively simple devices, such as garden sprayers, aerial crop dusting equipment, and insect control vehicles. In addition, a conventional attack on factories, chemical production facilities, or tank cars may result in the release of toxic industrial chemicals, with effects similar to those of an attack by military chemical warfare agents. Biologic agents may involve aerosol dispersal or possible contamination of food or water supplies.

3. What can the recent events in Syria teach us?

There is ample evidence that the same agent employed by the Aum Shinrikyo cult (and by Iraq's Saddam Hussein against Kurdish civilians), namely the nerve agent sarin, has recently

been used against Syrian civilians with devastating effect. Although both sides in the ongoing Syrian civil war blame each other for the release of this agent, it is widely known that the Syrian military stockpiled large quantities of sarin, as well as mustard gas. Recent reports demonstrate the following: (a) the potential morbidity of chemical agents; (b) the difficulty in maintaining their security; (c) the potential for state-supported weapons to fall into unintended hands; (d) the difficulties inherent in destroying a weapons arsenal once it has been created; (e) the vulnerability of unprotected civilians to such agents; (f) the problems encountered with attribution; and (g) the ability of a large-scale release of a chemical agent, with its victims tightly clustered in time and space, to rapidly overwhelm emergency medical systems.

Tapper J, Castillo M: First on CNN: Videos show glimpse into evidence for Syria intervention. Available at http://www.cnn.com/2013/09/07/politics/us-syria-chemical-attack-videos/index.html.

4. What did we learn from the anthrax outbreak of 2001?

This outbreak was characterized by 22 confirmed or suspected cases (11 inhalational, 11 cutaneous), with five deaths resulting from known or presumed exposure to anthrax-contaminated mail. At least five letters containing anthrax spores were sent to government and business offices in Florida, Washington, DC, and New York City from Trenton, New Jersey. This means of dispersal represents one mode of attack, but many bioterrorism defense experts express greater fear over the possibility of a widespread aerosol release (e.g., from a small crop duster–type airplane) that could potentially sicken hundreds of thousands. As it was, even the 2001 attack resulted in enormous public anxiety and major demands for medical care and public health resources. Antibiotic prophylaxis was prescribed for over 30,000 persons, and decontamination of the Hart Senate Office Building alone took months and cost more than $20 million. In 2008, a government scientist under investigation for the crime committed suicide before he was arrested, and although the FBI insisted he was the most likely perpetrator, there remains some controversy over the validity of that conclusion.

Centers for Disease Control and Prevention: Update: Investigation of bioterrorism-related anthrax and interim guidelines for exposure management and antimicrobial therapy, October 2001. MMWR Morb Mortal Wkly Rep 2001;50:909-919.

Kortan MP: The anthrax investigation: The view from the F.B.I. New York Times, Oct. 27, 2011. Who mailed the anthrax letters? New York Times, Oct. 17, 2011 (editorial).

5. Why are such incidents of particular importance to ED personnel?

Such incidents illustrate the potential for biologic or chemical terrorist attacks, and thus drive the imperative that health care workers, particularly first responders and ED personnel, be familiar with the expedient diagnosis and management of chemical and biologic terrorist events. Although biologic agents have incubation periods of days to weeks, making it possible that some victims will seek care through primary care providers, chemical agents are likely to produce effects immediately upon exposure, and ED personnel are almost certain to be called upon to respond to attacks with these agents.

6. What are the potential medical consequences of a chemical attack?

Chemical attacks probably would combine elements of traditional mass disasters (e.g., earthquakes), in which large numbers of casualties occur almost immediately, and traditional hazardous materials (HAZMAT) incidents. However, they have the potential to be more catastrophic for several reasons:

- Intent to inflict mass casualties
- Hazardous materials of extreme lethality
- HAZMAT site that is extremely toxic to rescue workers
- Less information about agent(s) involved
- Potentially overwhelming numbers of patients requiring emergency medical services (EMS)
- Even larger numbers of mildly affected or “worried-well” patients self-transporting to EDs and placing additional demands on the health care system
- Mass hysteria and panic

Henretig FM, Cieslak TJ, Eitzen EM Jr: Biological and chemical terrorism. J Pediatr 2002;141:311-326.

7. What were the consequences of the 1995 Tokyo sarin attack?

One ED close to the scene received more than 500 patients, including three in cardiopulmonary arrest. Citywide, over 5000 persons sought emergency medical treatment at more than 200 facilities within a few hours, and about 25% required hospitalization. Of note, 90% of the victims went to hospitals by taxi, private vehicles, or on foot rather than by formal EMS transport, further compounding the initial chaos. Until the identity of the agent was known, significant efforts at patient decontamination were lacking, resulting in many symptomatic exposures to hospital staff, although most were mild.

Okumura T, Suzuki K, Fukuda A, et al: The Tokyo subway sarin attack: Disaster management, Part 2: Hospital response. Acad Emerg Med 1998;5:618-624.

8. How would a biologic attack differ from a chemical attack?

Owing to the intrinsic incubation periods of biologic agents, attacks with these substances must be viewed differently than conventional and chemical terrorist attacks. The scenario is more like that of a public health crisis than an EMS or HAZMAT emergency. Exposed persons may be unaware of the attack and disperse from the site of initial exposure. Many diseases caused by high-threat agents begin with nonspecific febrile syndromes. The first indication of a biologic attack is likely to be an epidemic of an unusually large number of persons presenting to diverse locations, possibly several days after exposure, with either early nonspecific clinical findings or later findings of severe disease. Thus, patients may present to various medical offices and EDs in piecemeal fashion, reporting, for example, flulike symptoms. This situation was clearly illustrated by the mail-borne anthrax outbreak in the fall of 2001.

When such features are noted, PEM providers should immediately report suspicion of such an attack to appropriate public health authorities.

Scarfone RJ, Henretig FM, Cieslak TJ, et al: Emergency department recognition and management of victims of biological and chemical terrorism. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 125-152.

9. What clues suggest a biologic attack?

Pediatricians and emergency physicians must maintain an index of suspicion if a biologic attack is to be diagnosed in time for useful measures to be undertaken. Several epidemiologic features may suggest a bioagent attack. When these are noted, immediately report suspicion of such an attack to appropriate public health authorities:

- Epidemic presentation in a relatively compressed time frame (because most persons are exposed at the same time)
- Diseases that are rare or are not endemic in the area of exposure
- Especially high infection rate among exposed persons
- More respiratory forms of disease than usual
- Particularly high morbidity and mortality rates
- Several epidemics occurring simultaneously
- Infection rates lower in persons sheltered from the suspected route of exposure
- Infected or dying animals
- Discovery of suspicious actions or potential delivery systems

Scarfone RJ, Henretig FM, Cieslak TJ, et al: Emergency department recognition and management of victims of biological and chemical terrorism. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 125-152.

10. Why may children be disproportionately affected by both chemical and biologic agents?

- In the event of an intentional release, it is expected that exposure is most likely to occur via the aerosol route. Children have higher minute ventilation rates for body size and live closer to the ground, which enhances respiratory exposure, especially for agents denser than air.
- Children have thinner and more permeable skin, allowing greater injury from vesicant or corrosive chemicals and in some cases faster systemic absorption (e.g., nerve agents).
- Children possess a larger surface area/body weight ratio, further compounding the dermal absorption problem. Moreover, their smaller relative blood volumes make children more susceptible to the volume losses associated with enteric infections such as cholera and to gastrointestinal intoxications due to staphylococcal enterotoxins.

- Their immature blood-brain barriers may heighten the risk of central nervous system toxicity (e.g., from nerve agents) among children.
- Children have age-related developmental vulnerabilities that may hamper their ability to escape exposure from a contaminated site.
- Children may suffer unique psychological trauma in the context of separation from parents or witnessing the death of family members.
- Pediatric experience with several of the relevant antibiotics, antidotes, and vaccines is limited; in several cases, thoughtful use of treatments usually considered contraindicated in children may be necessary. The health care response to children would be hampered by the usual decrements in the ability of EMS systems to handle pediatric patients.
- All procedures are more difficult if providers are garbed in protective gear; this phenomenon would be exaggerated with small children.
- Massive numbers of pediatric casualties would require the definitive treatment of numerous children in centers that normally rely on expeditious interhospital transport to relieve them of the long-term responsibility for critical pediatric patients.
- Pediatric centers probably would be overwhelmed with both indigenous patients and those transferred.

Henretig FM, Cieslak TJ, Eitzen EM Jr: Biological and chemical terrorism. J Pediatr 2002;141:311-326.

Rotenberg JS, Newmark J: Nerve agent attacks on children: Diagnosis and management. Pediatrics 2003;112:648-658.

Scarfone RJ, Henretig FM, Cieslak TJ, et al: Emergency department recognition and management of victims of biological and chemical terrorism. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 125-152.

Key Points: Reasons That Chemical and Biologic Agents Disproportionately Affect Children

1. Children have higher minute ventilation rates and live closer to the ground, which enhances respiratory exposure of aerosolized agents.
2. Children have thinner and more permeable skin, allowing greater injury from vesicant or corrosive chemicals and possibly faster systemic absorption of nerve agents.
3. Children possess a larger surface area/body weight ratio, further compounding the dermal absorption problem.
4. Children have smaller relative blood volumes, making them more susceptible to volume losses associated with enteric infections such as cholera.
5. Children have immature blood-brain barriers, which may heighten the risk of central nervous system toxicity from nerve agents.
6. Children have age-related developmental vulnerabilities that may hamper their ability to escape exposure from a contaminated site.
7. Children may suffer unique psychological trauma in the context of separation from parents or witnessing the death of family members.
8. Pediatric experience with several of the relevant antibiotics, antidotes, and vaccines is limited.
9. Procedures are more difficult, especially with small children, if providers are garbed in protective gear.
10. EMS systems may be unable to handle pediatric patients.
11. Massive numbers of pediatric casualties would overwhelm hospital transport teams.
12. Pediatric centers probably would be overwhelmed with both indigenous patients and those transferred.

11. What are the principal biologic agent threats, and what is the general approach to their management?

The major biologic agents of concern are categorized by the Centers for Disease Control and Prevention (CDC) as Category A agents. The diseases caused by these agents are summarized in Table 68-1, which outlines the principal clinical findings, appropriate diagnostic measures, and specific antimicrobial or antitoxin therapies. Regarding initial ED management, in most circumstances, patients will present after a significant time interval from their exposure, and thus specific decontamination procedures are unnecessary. In the event of an announced attack, with patients presenting immediately after exposure to the ED, consider

Table 68-1. Critical Biologic Agents

DISEASE	CLINICAL FINDINGS	INCUBATION PERIOD (DAYS)	ISOLATION PRECAUTIONS	INITIAL TREATMENT	PROPHYLAXIS
Anthrax (inhalational)*	Febrile prodrome, then rapid progression to mediastinal lymphadenitis and mediastinitis, sepsis, shock, and meningitis	1-5	Standard	Ciprofloxacin 10-15 mg/kg IV q12h OR levofloxacin 8 mg/kg IV/PO q12h OR doxycycline 2.2 mg/kg IV q12h AND clindamycin§ 10-15 mg/kg IV q8h AND penicillin G‖ 400-600 kU/kg/d IV in divided doses q4h	Ciprofloxacin 10-15 mg/kg PO q12h OR doxycycline 2.2 mg/kg PO q12h
Plague (pneumonic)	Febrile prodrome, then rapid progression to fulminant pneumonia, hemoptysis, sepsis, disseminated intravascular coagulation	2-3	Droplet (for first 3 d of therapy)	Gentamicin 2.5 mg/kg IV q8h OR doxycycline 2.2 mg/kg IV q12h OR ciprofloxacin 15 mg/kg IV q12h OR levofloxacin 8 mg/kg IV/PO q12h	Doxycycline 2.2 mg/kg PO q12h OR ciprofloxacin 20 mg/kg PO q12h OR levofloxacin 8 mg/kg PO q12h
Tularemia	Pneumonic: abrupt onset of febrile, fulminant pneumonia Typhoidal: fever, malaise, abdominal pain	2-10	Standard	Gentamicin 2.5 mg/kg IV q8h OR doxycycline 2.2 mg/kg IV q12h OR ciprofloxacin 15 mg/kg IV q12h Patients who are clinically stable after 14 d may be switched to a single oral agent (ciprofloxacin or doxycycline) to complete a 60-d course.†	Doxycycline 2.2 mg/kg PO q12h OR ciprofloxacin 20 mg/kg PO q12h

Continued on following page

Table 68-1. Critical Biologic Agents (*Continued*)

DISEASE	CLINICAL FINDINGS	INCUBATION PERIOD (DAYS)	ISOLATION PRECAUTIONS	INITIAL TREATMENT	PROPHYLAXIS
Smallpox	Febrile prodrome, then synchronous, centrifugal, vesiculopustular exanthema	7-17	Airborne (+contact)	Supportive care	Vaccination may be effective if given within the first several days after exposure
Botulism	Afebrile, then descending symmetrical flaccid paralysis and with cranial nerve palsies	1-5	Standard	Supportive care; antitoxin (see text) may halt the progression of symptoms but is unlikely to reverse them	None
Viral hemorrhagic fevers	Febrile prodrome, then rapid progression to shock, purpura, and bleeding diatheses	4-21	Contact (consider airborne in cases of massive hemorrhage)	Supportive care; ribavirin may be beneficial in select cases	None

[*] In a mass casualty setting, in which resources are severely constrained, consider substitution of oral therapy for the preferred parenteral option.
[†] Assuming the organism sensitivity, children may be switched to oral amoxicillin (80 mg/kg/d in divided doses q8h) to complete a 60-d course. We recommend that the first 14 d of therapy or postexposure prophylaxis, however, include ciprofloxacin, levofloxacin, or doxycycline, regardless of age.
[§] Rifampin or clarithromycin may be an acceptable alternative to clindamycin as a drug that targets bacterial protein synthesis. If ciprofloxacin, levofloxacin, or another quinolone is used, doxycycline may be used as a second agent, because it also targets protein synthesis.
[‖] Ampicillin, imipenem, meropenem, and chloramphenicol may be acceptable alternatives to penicillin as drugs with good central nervous system penetration.
Adapted from Henretig FH, Cieslak TJ, Eitzen EM: Biological and chemical terrorism. J Pediatr 2002;141:311-326.

decontamination issues. In most cases, simple removal of outer garments and washing with soap and water provide for the safe removal of biologic agents.

Scarfone RJ, Henretig FM, Cieslak TJ, et al: Emergency department recognition and management of victims of biological and chemical terrorism. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 125-152.

Key Points: Category A Biologic Agents

1. Variola virus
2. *Bacillus anthracis*
3. *Yersinia pestis*
4. *Francisella tularensis*
5. Botulinum toxin
6. Filoviruses and arenaviruses (viral hemorrhagic fevers)

12. What is the most important biologic threat?

Because the motivation of terrorists is often obscure and their choice of weapons may be influenced by opportunity, public health officials often refer not to likelihood of use but rather to the magnitude of the problems that would be encountered if a given agent were used. The most problematic biologic threat is believed to be *inhalational anthrax*. This disease is caused by aerosol exposure to anthrax spores, which were weaponized by the United States (before 1969, when the United States renounced the use of biologic weapons and destroyed all existing stockpiles), as well as by the Soviet Union and Iraq. The causative bacterium is *B. anthracis*, a sporulating gram-positive rod. Endemic anthrax occurs in cutaneous and gastrointestinal as well as inhalational forms; generally it is contracted by close contact with hides, wool, or meat of infected herbivores (especially sheep, cattle, and goats).

Bravata DM, Wang E, Holty JE, et al: Pediatric anthrax: Implications for bioterrorism preparedness. Evid Rep Technol Assess (Full Rep) 2006;141:1-48.

Inglesby TV, O'Toole T, Henderson DA, et al: Anthrax as a biological weapon, 2002: Updated recommendations for management. JAMA 2002;287:2236-2252.

13. What are the signs and symptoms of inhalational anthrax exposure?

After inhalation of anthrax spores, infection begins with pulmonary macrophage uptake and subsequent carriage to mediastinal lymph nodes, where necrotizing lymphadenitis and sepsis ensue. *Inhalational anthrax* has an incubation period of 1 to 6 days, and then begins with a flulike illness characterized by fever, myalgia, headache, cough, and chest "tightness." A brief period of improvement is sometimes seen 1 to 2 days later, but is followed by rapid deterioration with high fever, dyspnea, cyanosis, and shock. Hemorrhagic meningitis is present in up to 50% of cases. Chest radiographs obtained late in the course of illness may demonstrate a widened mediastinum or prominent mediastinal lymphadenopathy; infiltrates or pleural effusions may also be present. Consider the diagnosis with this clinical picture and a chest radiograph demonstrating a widened mediastinum, and confirm this with positive blood cultures. The prognosis for patients with inhalational anthrax is grave; classically, death was universal in untreated cases, and still may occur in as many as 95% of treated cases if therapy is begun more than 48 hours after symptom onset. Even in the 2001 outbreak, with modern intensive care and the availability of latest generation antibiotics, only 6 of 11 patients with inhalational anthrax survived.

14. What are the signs and symptoms of cutaneous anthrax exposure?

Cutaneous anthrax occurs when organisms gain entry into skin, particularly through abrasions or cuts. It is characterized by the development of a papule at the inoculum site, which then progresses over days to a vesicle, then an ulcer, and finally to a depressed, black eschar. The surrounding tissue becomes markedly edematous, but not particularly tender, distinguishing this infection from more typical staphylococcal or streptococcal cellulitis. It is quite amenable to therapy with a variety of antibiotics; with timely institution of treatment, it is rarely fatal. In the fall 2001 outbreak, all 11 patients with cutaneous anthrax survived. The only pediatric

victim of that attack was a 7-month-old boy with cutaneous anthrax on his arm, presumably contracted after a brief visit to a New York City television news studio that had received contaminated mail. He was initially suspected of having a brown recluse spider bite, and the correct diagnosis was confirmed only after the discovery of anthrax contamination at another television studio. Of note, he also developed hemolysis, thrombocytopenia, and renal insufficiency, features not usually observed in otherwise uncomplicated cases of cutaneous disease. This case raised the possibility of particular vulnerability in infancy.
Roche KJ, Chang MW, Lazarus H: Cutaneous anthrax. N Engl J Med 2001;345:1611.

15. What problems are associated with management of children exposed to anthrax?

First, the anthrax vaccine is approved only for adults. Second, both quinolones and tetracyclines have relative contraindications in children, although dental staining seen with tetracyclines usually requires multiple courses, and the risk of cartilage problems associated with quinolones is theoretical. Current experience with the use of both classes of antibiotics in children suggests that short-term use is safe. It seems reasonable that the extreme danger posed by anthrax exposure warrants use of any or all of these modalities in exposed children. In fact, as of 2013, the FDA (Food and Drug Administration) has approved ciprofloxacin for use in the prophylaxis of anthrax after inhalational exposure in children during a terrorist attack, and doxycycline and levofloxacin are now licensed as well in children for the same indication. Anthrax has little potential for person-to-person transmission; thus, standard precautions are adequate for health care workers.

16. Why is smallpox of special concern?

Because smallpox is highly contagious, protection of health care workers and prevention of secondary transmission are important factors. Smallpox was globally eradicated in 1980, and children are no longer vaccinated. Most American adults and all children are susceptible. Smallpox produces a febrile prodrome followed by a characteristic centrifugal rash that progresses synchronously from macules to papules to vesicles to pustules; fatality rates approach 30% in unimmunized patients. Smallpox mandates the use of the airborne isolation technique. Treatment is primarily symptomatic, though a few promising antiviral agents are currently under investigation, including cidofovir and ST-246.

17. What are the concerning features of plague?

Like smallpox, plague is highly contagious. Inhalational exposure causes a severe, hemorrhagic pneumonia (pneumonic plague) characterized by respiratory distress and hemoptysis. Plague is highly lethal in untreated patients. Antibiotics, including aminoglycosides and doxycycline, are effective if begun within 24 hours of onset of illness. Droplet precautions are necessary in managing patients with plague.

18. How dangerous is ricin?

Ricin-containing letters were mailed to a U.S. Senate office building in 2004, and again to President Obama and New York City Mayor Bloomberg in 2013, although fortunately no persons were sickened in either attack. Ricin is a biologic protein toxin (toxalbumin) derived from the castor bean plant (*Ricinus communis*) that inhibits ribosomal protein synthesis. It is very toxic in animal studies when inhaled, and may result in the delayed onset of respiratory distress, pulmonary edema, and acute respiratory failure. A case series of eight persons from the 1940s described a febrile respiratory illness after inhalational exposure. If injected, ricin may cause a sepsis-like syndrome that may progress to multiorgan system failure. Ingestion can lead to severe gastroenteritis.
Audi J, Belson M, Patel M, et al: Ricin poisoning: A comprehensive review. JAMA 2005;294:2342-2351.

19. What are the primary chemical threats?

The primary chemical threats are summarized in Table 68-2.

20. Describe the general approach to management and decontamination of chemical threats.

The general approach to these exposures begins with stabilization of life-threatening effects ("ABCs" [airway, breathing, and circulation]), followed as soon as possible by appropriate decontamination. Decontamination capability should be available on a short set-up time basis,

Table 68-2. Critical Chemical Agents

AGENT	TOXICITY	CLINICAL FINDINGS	ONSET	DECONTAMINATION*	MANAGEMENT
Nerve Agents					
EG, sarin, soman, VX	Anticholinesterase: muscarinic, nicotinic, central nervous system effects	Vapor: miosis, rhinorrhea, dyspnea Liquid: diaphoresis, vomiting Both: coma, paralysis, seizures, apnea	Vapor: seconds Liquid: minutes-hours	Vapor: fresh air, remove clothes, wash hair Liquid: remove clothes, wash skin and hair with copious soap and water, ocular irrigation	ABCs Atropine: 0.05 mg/kg IV†, IM‡ (min 0.1 mg, max 5 mg), repeat q2-5min PRN for marked secretions, bronchospasm Pralidoxime: 25 mg/kg IV, IM§ (max 1 g IV; 2 g IM), may repeat within 30-60 min PRN, then again q1h for one or two doses PRN for persistent weakness, high atropine requirement Diazepam: 0.3 mg/kg (max 10 mg) IV; lorazepam: 0.1 mg/kg IV, IM (max 4 mg); midazolam: 0.2 mg/kg (max 10 mg) IM PRN for seizures or severe exposure
Vesicants					
EG: Mustard	Alkylation	Skin: erythema, vesicles Eye: inflammation Respiratory tract: inflammation	Hours	Skin: soap and water Eyes: water (effective only if done within minutes of exposure)	Symptomatic care
Lewisite	Arsenical		Immediate pain		Consider British antilewisite (BAL) 3 mg/kg IM q4-6h for systemic effects of lewisite in severe cases
Pulmonary Agents					
Chlorine, phosgene	Liberate hydrochloric acid, alkylation	Eye, nose, and throat irritation (especially chlorine)	Minutes: eye, nose, and throat irritation, bronchospasm	Fresh air Skin: water	Primarily symptomatic care (see text)

Continued on following page

Table 68-2. Critical Chemical Agents (*Continued*)

AGENT	TOXICITY	CLINICAL FINDINGS	ONSET	DECONTAMINATION	MANAGEMENT
		Respiratory: bronchospasm, pulmonary edema (especially phosgene)	Hours: pulmonary edema		
Other					
Cyanide	Cytochrome oxidase inhibition: cellular	Tachypnea, coma, seizures, apnea Anoxia, lactic acidosis	Seconds	Fresh air Skin: soap and water	ABCs, 100% oxygen Sodium bicarbonate PRN for metabolic acidosis Hydroxycobalamin 70 mg/kg IV (max 5 g) OR Nitrite/thiosulfate, given as follows: Sodium nitrite (3%) dose (mL/kg) (max 10 mL) / Estimated hemoglobin concentration (g/dL) 0.27 / 10 0.33 / 12 (estimated for average child) 0.39 / 14 followed by: Sodium thiosulfate (25%): 1.65 mL/kg (max 50 mL)

ABCs, airway, breathing, and circulatory support; max, maximum; min, minimum; PRN, as needed.

*Decontamination, particularly for patients with significant nerve agent or vesicant exposure, should be performed by health care providers garbed in adequate personal protective equipment. For emergency department staff, recommended equipment consists of a nonencapsulated, chemically resistant body suit, boots, and gloves with a full-face air purifier mask/hood.

†Intraosseous route is likely equivalent to intravenous.

‡Atropine might have some benefit via endotracheal tube or inhalation, as might aerosolized ipratropium. See also Table 68-3.

§Pralidoxime is reconstituted to 50 mg/mL (1 g in 20 mL water) for IV administration, with the total dose infused over 30 min, or it may be given by continuous infusion (loading dose 25 mg/kg over 30 min, and then 10 mg/kg/h). For IM use, see also Table 68-3.

Adapted from Henretig FH, Cieslak TJ, Eitzen EM: Biological and chemical terrorism. J Pediatr 2002;141:311–326.

typically using an outdoor facility with multiple patient stations, arranged so that parallel lines of ambulatory and nonambulatory patients may be processed simultaneously. One problem with outdoor decontamination units is that it may be challenging to protect victims from inclement weather in temperate climate zones, an issue especially important in the management of young children. An alternative is the use of an enclosed facility, optimally adjacent to, but separate structurally from, the main ED, with a separate, high-volume ventilation system vented directly outdoors.

Medical personnel in appropriate personal protective equipment (PPE) should staff an initial triage station at the entrance to the decontamination structure. Such PPE that is generally recommended for hospital personnel is OSHA (Occupational Safety and Health Administration) "Level C," which consists of a nonencapsulated, chemically resistant body suit, gloves, and boots, with a full-face air purifier mask containing a cartridge with both an organic-vapor filter for chemical gases and vapors and a HEPA (high-efficiency particulate air) filter to trap aerosols of biologic and chemical agents. These triage personnel facilitate rapid identification of patients requiring immediate antidotal or other lifesaving intervention, as well as diversion of nonambulatory patients to the appropriate area with medical assistance. Instruct ambulatory patients who are old enough in self-decontamination. Obviously, young children require assistance or may be accompanied by parents if they are present. An outdoor facility must optimally provide adequate water, some temperature control during environmental extremes, and measures to maintain personal modesty, such as curtains or other barriers separating shower lines for males from lines for females.

Decontamination efforts should favor physical and mechanical removal over chemical decontamination. For vapor-exposed patients, decontamination is effected primarily by removing clothes and washing hair and skin with soap and water. In contrast, those patients with liquid agent dermal exposure require disrobing and thorough skin and hair decontamination. Any agent present on their skin or in their clothing poses a serious threat to ED personnel. Carefully remove and double-bag clothing. Ocular exposure requires copious eye irrigation with saline or water. Wash the child's skin and hair thoroughly with soap and tepid water. Another approach that has been developed for military field use is that of "reactive skin decontamination lotion," which is supplied to soldiers as an impregnated sponge and functions by both physical removal and neutralization of chemical agents. However, civilian, and particularly pediatric, experience with this product is currently limited.

Scarfone RJ, Henretig FH, Sullivan FJ: Decontamination and the use of personal protective equipment. Pediatr Emerg Care 2006;22(6):445-453.

21. Which chemical weapons are most feared?

Nerve agents are the most potent and prompt-acting and, thus, the most feared chemical weapons. These organophosphate compounds, similar to many pesticides, are toxic by inhalation, ingestion, and topical absorption and can result in profound muscarinic (cholinergic syndrome), nicotinic (initial muscle fasciculations, then paralysis), and central nervous system effects (seizures, coma, apnea). The clinical picture varies slightly by route of exposure, as noted in Table 68-2. Severe cases require antidotal therapy with atropine (adults, 2-5 mg; children, 0.02-0.05 mg/kg), pralidoxime (adults, 1-2 g; children, 25-50 mg/kg), and usually diazepam for seizure control (adults, 5-10 mg; children, 0.1-0.3 mg/kg). Administer these agents at precise mg/kg dosing by an intravenous (IV) route in individual cases or for relatively small numbers of patients. For mass casualties, intramuscular (IM) injection of agents in the prehospital setting may be appropriate, as noted in the Table 68-3.

Rotenberg JS, Newmark J: Nerve agent attacks on children: Diagnosis and management. Pediatrics 2003;112:648-658.

22. What are the effects of cyanide exposure?

Cyanide is a cellular poison with multiorgan system clinical manifestations. The efficacy of cyanide as a chemical terrorism agent may be limited by its volatility in open air and relatively low lethality in comparison with nerve agents. Released in a closed room, however, cyanide would have devastating effects, as evidenced by its use in the Nazi gas chambers during World War II. Cyanide interferes with normal mitochondrial oxidative metabolism by inhibiting cytochrome a_3, leading to cellular anoxia and lactic acidosis. Initially, cyanide toxicity would likely manifest as tachypnea and hyperpnea, progressing rapidly to apnea in cases with large exposure. Additional early findings among cyanide victims include tachycardia, flushing,

Table 68-3. Pediatric Autoinjector Recommendations

Atropine Autoinjector Therapy

ESTIMATED AGE RANGE	APPROXIMATE WEIGHT (KG)	AUTOINJECTOR SIZE (MG)
<6 mo	<7.5	0.25
6 mo-4 yr	7.5-18	0.5
5-10 yr	18-30	1.0
>10 yr	>30	2.0

Pralidoxime Autoinjector Therapy

APPROXIMATE AGE RANGE (YR)	APPROXIMATE WEIGHT (KG)	NUMBER OF AUTOINJECTORS	PRALIDOXIME DOSE (MG/KG)
3-7	13-25	1	24-46
8-14	26-50	2	24-46
>14	>50	3	<35

*Consider autoinjector use for mass casualties when IV (intravenous) access or more precise mg/kg IM (intramuscular) dosing is logistically impractical. The initial dose using atropine autoinjectors is one autoinjector of the recommended size. The initial dose using pralidoxime autoinjectors is the recommended number of (adult-intended, 600 mg) autoinjectors. These pralidoxime autoinjectors may also be injected into an empty sterile vile; the contents redrawn through a filter needle into a small syringe may then provide a ready source of concentrated (300 mg/mL) pralidoxime solution for IM injection in infants. Recently, adult-intended autoinjectors that combine both atropine and pralidoxime have become available and might find use in children older than age 3 for both medications, as per the guidelines for pralidoxime-only autoinjectors above.

From Henretig FM, Mechem C, Jew R: Potential use of autoinjector-packaged antidotes for treatment of pediatric nerve agent toxicity. Ann Emerg Med 2002;40:405-408.

dizziness, headache, diaphoresis, nausea, and vomiting. As exposure increases, seizures, coma, apnea, and cardiac arrest may follow within minutes. An elevated anion gap metabolic acidosis, with increased serum lactate, is typically present, and decreased peripheral oxygen utilization leads to an elevated mixed venous oxygen saturation value.

23. How is cyanide exposure best treated?

Therapy for cyanide exposure includes removal of the patient to fresh air and intensive supportive care. In significant cases, administer specific antidotes. The classic cyanide antidote utilizes a nitrite as a methemoglobin-forming agent, with sodium thiosulfate, a compound used as a substrate by the hepatic enzyme rhodanese, which converts cyanide to thiocyanate, a less toxic compound excreted in the urine. However, a newer antidote available in the United States is hydroxocobalamin, which exchanges its hydroxy group for cyanide, forming harmless cyanocobalamin (vitamin B_{12}). This is subsequently excreted by the kidneys. Hydroxocobalamin use is not complicated by the potential for nitrite-induced hypotension or severe methemoglobinemia, and it has low toxicity. Although no human controlled trials are currently available to compare hydroxocobalamin with nitrite/thiosulfate-based therapies, many experts believe that hydroxocobalamin's efficacy and safety profile make it the cyanide antidote of choice, especially for children. See Table 68-2 for details.

Geller RJ, Barthold C, Salers JA, Hall AH: Pediatric cyanide poisoning: Causes, manifestations, management and unmet needs. Pediatrics 2006;118:2146-2158.

EMERGENCY MEDICAL SERVICES AND PREHOSPITAL CARE

CHAPTER 69

Hazel Guinto-Ocampo

1. What are the links in the American Heart Association's pediatric chain of survival?

Prevention → early cardiopulmonary resuscitation (CPR) → prompt access to the emergency response system → rapid pediatric advanced life support (PALS) → integrated postcardiac arrest care.

Berg MD, Schexnayder SM, Chameides L, et al: Part 13: Pediatric basic life support: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation 2010;122:S862-S875.

2. What is enhanced 911?

Enhanced 911 automatically provides computerized identification of the telephone number and location of the caller, regardless of the quality of information provided.

3. What percentage of 911 calls are made from a cellular phone?

It is estimated that about 70% of 911 calls are placed from wireless phones, and that percentage is growing.

Federal Communications Commission: Guide: 911 wireless services. Available at http://www.fcc.gov/guides/wireless-911-services.

4. How does enhanced 911 work with calls made from a cellular phone?

A Federal Communications Commission (FCC) rule requires all wireless service providers to transmit all 911 calls to a public safety answering point (PSAP) regardless of whether or not the caller subscribes to the provider's service.

Phase I Enhanced 911 rules require wireless service providers to provide the PSAP with the telephone number of the originator of a wireless 911 call and the location of the cell site or base station transmitting the call.

Phase II Enhanced 911 rules require wireless service providers to provide the caller's latitude and longitude, which must be accurate to within 50 to 300 m.

Federal Communications Commission: Guide: 911 wireless services. Available at http://www.fcc.gov/guides/wireless-911-services.

5. What is an emergency medical dispatcher (EMD)?

An EMD is a specialized operator who gathers essential information regarding the location, nature, and severity of the emergency and relays this information to the emergency medical services (EMS) system for dispatch of a first responder or ambulance. When critical conditions are identified, the EMD may assist the caller in providing interventions prior to EMS arrival, such as opening the airway, providing CPR, controlling hemorrhage, and assisting with childbirth.

6. What are dispatcher protocols?

Dispatcher protocols are written guidelines, often computerized, that are developed and utilized so that dispatchers can accurately instruct callers in pediatric CPR, relief of foreign-body airway obstruction, and essential first aid maneuvers until prehospital personnel arrive. Their use reduces the variability of the information provided by the dispatchers and ensures that accurate emergency information is provided to every caller.

7. Name the three general categories of prehospital personnel.

1. First responders
2. Basic life support (BLS) providers
3. Advanced life support (ALS) providers

The categories vary in levels of training and degrees of capabilities. At the federal level, the National Highway Traffic Safety Association (NHTSA) has developed the National Standard Curricula for certification for each category, but state or local requirements supersede these standards. Intermediate levels of providers with varied capabilities have evolved, as many jurisdictions offer supplemental training modules.

Blackwell TH: Emergency medical services: Overview and ground transport. In Marx JA, Hockberger RS, Walls RM (eds): Rosen's Emergency Medicine Concepts and Clinical Practice, Vol. 2, 7th ed. Philadelphia, Mosby Elsevier, 2010, pp 2461-2468.

8. What is a first responder?

First responders have limited but significant lifesaving capabilities. By definition, first responders are typically the first to arrive at the scene of an incident. The Department of Transportation (DOT) recommends a 40-hour didactic curriculum for certification and 16 to 36 hours for refresher training. Most are trained to help clear an obstructed airway, control hemorrhage, use an automated external defibrillator (AED), and administer first aid or CPR until the arrival of more advanced personnel. Spine immobilization and oxygen and medication administration are typically beyond the first responder's capabilities. Except in some rural EMS systems, first responders usually do not provide ambulance transport as the primary caregiver. This level of providers is referred to as emergency medical responders (EMRs) in the new NHTSA model.

Woodward GA, King BR, Garrett AL, et al: Emergency medical services and transport medicine. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 85-124.

9. What are BLS providers?

BLS providers are usually called emergency medical technicians–basic (EMT-Bs). They have capabilities that exceed those of the first responders. EMT-B training requires 100 or more hours and observation time in the emergency department (ED). EMT-Bs are capable of patient assessment, spinal immobilization, noninvasive ventilatory assistance, and defibrillation with AEDs. They are trained to recognize respiratory distress, altered mental status, shock, mechanisms of injury, and death, as well as to recognize and treat pulselessness, apnea, upper airway obstruction, and extremity deformity. EMT-Bs are referred to as emergency medical technicians or EMTs in the new NHTSA model.

Woodward GA, King BR, Garrett AL, et al: Emergency medical services and transport medicine. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 85-124.

10. What are ALS providers?

ALS providers are usually referred to as emergency medical technicians–paramedics (EMT-Ps). They can administer a high level of care in the field. They undergo 1000 to more than 3000 hours of training, internship, and clinical hospital time. They are trained to perform advanced resuscitation techniques, such as ventilatory support, vascular access, and drug administration. They are capable of general diagnostic skills, rhythm disturbance recognition and treatment, and advanced airway management, including endotracheal intubation. In some areas, they can perform medication-assisted intubations using sedatives and paralytics and can place emergent surgical airways. Many ALS provider educational programs have advanced to a 2-year associate or a 4-year baccalaureate degree. EMT-Ps are classified as paramedics in the new NHTSA model.

Woodward GA, King BR, Garrett AL, et al: Emergency medical services and transport medicine. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 85-124.

11. What is an EMT-intermediate provider (EMT-I)?

EMT-Is, besides having met EMT certification, undergo supplemental training for more advanced skills, such as placement of vascular access or performance of advanced airway management. They possess some skills beyond those of an EMT, but less than those of a paramedic. This intermediate classification has developed in response to perceived needs of

local jurisdictions. This level of providers is classified as advanced emergency medical technicians or AEMTs in the new NHTSA model.

Woodward GA, King BR, Garrett AL, et al: Emergency medical services and transport medicine. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 85-124.

Key Points: Categories of Prehospital Personnel

1. First responders (EMRs)
2. BLS providers (EMTs)
3. ALS providers (paramedics)

12. Where can I find a list of standard pediatric equipment for BLS and ALS units?

A list of equipment in ambulances to safely manage pediatric emergencies was collaboratively developed and endorsed by the American College of Surgeons (ACS) Committee on Trauma, the American Academy of Pediatrics (AAP), the American College of Emergency Physicians (ACEP), the National Association of EMS Physicians (NAEMSP), and the Pediatric Equipment Guidelines Committee—Emergency Medical Services for Children (EMSC) Partnership for Children Stakeholder Group. These lists were published on the websites listed below:

American Academy of Pediatrics: Policy Statement—Equipment for Ambulances, July 2009. Available at http://pediatrics.aappublications.org/cgi/reprint/124/1/e166.

American College of Surgeons, Committee on Trauma, American College of Emergency Physicians: Equipment for ambulances. Bull Am Coll Surg 2009;94:23-29.

13. What is medical command?

Medical command is the entity responsible for the supervision of the EMS of the community. In general, prehospital personnel are assigned to a specific operational base or base station, and these base stations are responsible to medical command. *Medical control* provides medical direction to prehospital personnel. Medical control can be off-line/indirect or on-line/direct.

14. Differentiate off-line/indirect and on-line/direct medical control.

- **Off-line/indirect medical control** consists of patient care protocol development, personnel education and training, prospective and retrospective patient care review, and other process improvement activities.
- **On-line/direct medical control** involves real-time interaction between a physician or designee and a field provider. On-line medical control can be centralized or decentralized. In a *centralized* system, a designated hospital is responsible for all direct medical control orders and notification, regardless of the receiving facility. In a *decentralized* system, each receiving hospital provides direction to prehospital personnel transporting patients to their facility.

15. What are standing orders?

Standing orders are policies or protocols issued by a medical director of an EMS system that authorizes EMTs to perform particular skills in certain situations.

Limmer DJ, O'Keefe MF, Grant HT: Medical, legal, and ethical issues. In Limmer DJ, O'Keefe MF, Grant HT: Emergency Care, 12th ed. Saddle River, NJ, Pearson Health Science, 2012, pp 74-91.

16. Describe the difference between single-tiered and multitiered EMS systems.

- In a **single-tiered** system, every response, regardless of the call type, receives the same level of personnel expertise and equipment (all BLS or ALS). The advantage of this design is the provision of an advanced level of care to all calls, and it overcomes under- or over-triaging by EMS dispatchers.
- **Multitiered** systems respond with an ALS- or BLS-level unit depending on the nature of the call. This design reserves ALS units for higher-priority calls and aims to ensure that an ALS unit is always available for potentially critical responses.

The ideal EMS system design provides quality patient care in the briefest possible period of time. Therefore, the efficiency of adopting a single-tiered versus multitiered response is affected by the availability of BLS and ALS providers in the community and the distance from the scene of the incident to the nearest receiving hospital. Regardless of the response design, EMS systems

usually include first responder services, often provided by the police or fire department, as part of the response.

17. What is EMSC?

EMSC is a federally funded program administered by the Health Resources and Services Administration's Maternal and Child Health Bureau and NHTSA that provides grant funding to states or medical schools in all 50 states, the District of Columbia, and five territories, to support pediatric emergency care initiatives at the state and local levels. Funding supports all the components of the program, namely, education and training, systems development, data analysis and research, and public policy and future planning. EMSC partners with numerous national organizations involved in the care of acutely ill or injured children.

Emergency Medical Services for Children. Available at http://www.childrensnational.org/emsc/.

18. What resources are available through EMSC?

Numerous education and training resources regarding emergency care are available through EMSC. These resources are geared toward various users, such as children, parents and families, school and child care professionals, prehospital care providers, physicians and nurses. These resources are available, mostly for free, through the EMSC website.

Emergency Medical Services for Children. Available at http://www.childrensnational.org/emsc/.

19. Where can I find examples of pediatric EMS protocols?

The EMSC National Resource Center provides links to model pediatric protocols.

Emergency Medical Services for Children: Pediatric Protocol Resource Toolkit. Available at http://www.bcm.edu/pediatrics/texasemsc/index.cfm?pmid=23492.

20. What is family-centered prehospital care?

Family-centered prehospital care is a systematic approach to building collaborative relationships between prehospital personnel and the patients' families during on-site treatment, transport, and transition of care. The goal is to provide the best outcome for the patient through collaboration with his or her family members.

Emergency Medical Services for Children. Available at http://www.childrensnational.org/emsc/.

21. How should a prehospital care provider respond to an advance directive?

Advance directives, such as do not resuscitate orders, are utilized for many pediatric patients. Most states require that prehospital care providers honor legitimate advance directives unless the legal guardian allows resuscitation. The legal guardian may revoke the advance directive at any time. If there is an advance directive (e.g., a certified do not resuscitate order or the patient is wearing a "do not resuscitate" medical bracelet), the prehospital provider with support from on-line medical control should try to communicate with the legal guardian to determine their current wishes. If an advance directive cannot be verified, transportation with all appropriate emergency treatment measures is advised.

Woodward GA, King BR, Garrett AL, et al: Emergency medical services and transport medicine. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 85-124.

22. Is it appropriate to use an AED in a pediatric patient?

Yes. AEDs can recognize rhythms amenable to shock and direct the user to deliver the shock to a pediatric patient. Some are equipped with a pediatric attenuator, which decreases the energy delivered, and is preferred for use in infants and children younger than 8 years of age. If this is not available, an AED without a dose attenuator may still be used. AEDs without a dose attenuator may deliver higher energy doses, but they have been successfully used in infants with minimal myocardial damage and good neurologic outcomes.

Berg MD, Schexnayder SM, Chameides L, et al: Part 13: Pediatric basic life support: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation 2010;122:S862-S875.

23. What is the lowest infant weight in which an AED can be used?

The lower limit of infant size or weight below which an AED should not be used is not known.

Kleinman ME, deCaen AR, Chameides L, et al: Part 10: Pediatric basic and advanced life support: 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Circulation 2010;122:S466-S515.

24. What is the recommended method of shock delivery for treatment of rhythm abnormalities in infants?

The recommended devices for shock delivery in infants, in order of preference, are as follows:

1. Manual defibrillator
2. AED with dose attenuator
3. AED without dose attenuator

Kleinman ME, deCaen AR, Chameides L, et al: Part 10: Pediatric basic and advanced life support: 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Circulation 2010;122:S466-S515.

25. What is the doctrine of implied consent?

This doctrine permits the treatment of minors without parental consent when an emergency exists. A minor with a condition that threatens either life or limb is viewed as an emergency and must be treated and transported.

Limmer DJ, O'Keefe MF: Medical, legal, and ethical issues. In Limmer DJ, O'Keefe MF, Grant HT: Emergency Care, 12th ed. Saddle River, NJ, Pearson Health Science, 2012, pp 74-91.

26. What is the "good Samaritan law"?

This law provides immunity from liability to an individual who helped in an emergency, provided the rescuer acted in good faith and provided care to the level of his training and to the best of his ability.

Limmer DJ, O'Keefe MF: Medical, legal, and ethical issues. In Limmer DJ, O'Keefe MF, Grant HT: Emergency Care, 12th ed. Saddle River, NJ, Pearson Health Science, 2012, pp 74-91.

27. How should prehospital care providers respond if the legal guardian refuses transport?

Prehospital care providers and on-line medical control should try to persuade caregivers to allow hospital transport to a hospital. Most children whose caretaker refuses transport receive medical care within a week of the refusal and typically have good outcomes. However, about 10% are admitted to the hospital. Hospital transport cannot be refused if the legal guardian is intoxicated or otherwise incompetent, if child abuse is suspected, or if the child appears to be in imminent danger.

If the guardian refuses transport, prehospital care providers should complete a medical screening evaluation, contact medical command, follow all local regulations, document carefully, and have the legal guardian sign an "against medical advice" form.

Seltzer AG, Vilke GM, Chan TC, et al: Outcome study of minors after parental refusal of paramedic transport. Prehosp Emerg Care 2001;5:278.

28. What action should be taken if a prehospital care provider suspects child abuse?

When a prehospital care provider suspects child abuse, the medical control physician must ensure that appropriate treatment and transport occur. Notify the receiving facility of the concern for child abuse. Prepare a mandatory report to child protection agencies in accordance with prevailing laws of the jurisdiction. Do not allow the caregiver to refuse transport when child abuse is suspected. Police protection may be required to ensure the safety of the prehospital provider and to ensure that medical transport occurs.

Committee on Pediatric Emergency Medicine and Committee on Bioethics: Consent for emergency medical services for children and adolescents. Pediatrics 2011;128:427.

CHAPTER 70

PATIENT SAFETY IN THE EMERGENCY DEPARTMENT

Melanie Pitone and Steven M. Selbst

1. How extensive is the problem of medical errors?

The Institute of Medicine (IOM) estimated in 1998 that 44,000 to 98,000 deaths per year are due to medical errors. These data make medical errors the eighth leading cause of death. Medical errors in the United States cost $2 billion each year. This number of deaths has been disputed, and some of the adverse events included in this report may not have been due to medical error. However, the problem is significant. It is not known how many errors in the IOM report involved delivery of care to children in the emergency department (ED). Many of these errors are preventable.

Bates DW, Spell N, Cullen DJ, et al: The costs of adverse drug events in hospitalized patients. Adverse Drug Events Prevention Group. JAMA 1997;277:307-311.

Kohn LT, Corrigan JM, Donaldson MS (eds): To Err Is Human: Building a Safer Health System. Washington, DC, Institute of Medicine, National Academy Press, 1998.

2. What can we learn about errors in the pediatric ED from incident reports?

It is well known that safety incidents are grossly underreported through voluntary incident report systems. However, one study collected more than 3000 incident reports from 18 different pediatric EDs in 1 year. The most common incidents reported are laboratory errors (25%), medication errors (20%), and process variances (14%) (e.g., delay in care). Only 15% of reported events resulted in harm to the patient. Almost 70% of incidents were related to human factors, and most of these to lack of adherence to established protocols. Very few reports involve major severity (death or permanent injury). It is possible these events are rare and reported through other mechanisms.

Incident reports can help with investigation of many "near misses" in the ED and thus may be valuable in reducing future errors. ED staff must be encouraged to report incidents, and many hospitals have on-line systems to do so.

Chamberlain JM, Shaw KN, Lillis KA, et al; for the Pediatric Emergency Care Applied Research Network: Creating an infrastructure for safety event reporting and analysis in a multicenter pediatric emergency department network. Pediatr Emerg Care 2013;29:125-130.

3. What are near misses? How can these help the ED?

A near miss is an unplanned event that did not result in injury, illness, or damage—but had the potential to do so. Only a fortunate break in the chain of events prevented an injury, fatality, or damage.

The current process is for internal reporting of "near misses," keeping the learned lessons within an organization. By developing a system to share those findings among a network of EDs treating pediatric patients, Shaw and associates discovered that trends in latent systems issues can be learned and potential future errors can be prevented.

Pruitt CM, Liebelt EL: Enhancing patient safety in the pediatric emergency department. Pediatr Emerg Care 2010;26:942-951.

Selbst S, Levine S, Mull C, et al: Preventing medical errors in pediatric emergency medicine. Pediatr Emerg Care 2004;20:702-709.

Shaw KN, Lillis KA, Ruddy RM, et al: Reported medication events in pediatric emergency research network: Sharing to improve patient safety. Emerg Med J 2013;30:815-819.

4. Which factors inherent to the ED contribute to medical errors?

- Time pressures
- Incomplete medical and drug histories available
- Unscheduled care
- Inconsistency of patient arrival

- High-risk patients (high acuity)
- Stressful/demanding environment
  - ED overcrowding
  - Fatigue (nurses and physicians)
  - Miscommunication (with patients or among staff)
- Environment in flux—patients have varied locations (rooms, x-ray department, hallway)
- 24-hour activity in the ED
  - No time to restore order and "reset" the environment
  - Circadian rhythm of staff is challenged
- Transition of patient care among staff (consultants and change of shift)

Schenkel S: Promoting patient safety and preventing medical error in emergency departments. Acad Emerg Med 2000;7:1204-1219.

5. Why are children in the ED at particular risk for error?

- Variety of patient size and age
- Need to calculate most medication doses
- Limited time for pharmacist review of medication orders

Seriously ill pediatric patients are at greatest risk for error in the ED.

American Academy of Pediatrics, Committee on Pediatric Emergency Medicine. Patient safety in the pediatric emergency care setting. 2007;120:1367-1375.

Chamberlain JM, Slonim A, Joseph JG: Reducing errors and promoting safety in pediatric emergency care. Ambul Pediatr 2004;4:55-63.

Selbst S, Levine S, Mull C, et al: Preventing medical errors in pediatric emergency medicine. Pediatr Emerg Care 2004;20:702-709.

6. What is a latent error?

Latent errors are design flaws or failures in the tools or systems and work environment that produce circumstances in which a worker (nurse, physician) is likely to err. Latent errors may persist for long periods of time before they are discovered and corrected.

7. What are potential causes of patient misidentification?

- Similar or same patient names
- Language barriers
- Patient answers to wrong name
- Identification (ID) bands removed
- ID bands illegible or incorrect
- Lack of patient ID process or failure to comply

O'Neill K, Shinn D, Starr K: Patient misidentification in a pediatric emergency department: Patient safety and legal perspectives. Pediatr Emerg Care 2004;20:487-492.

8. How common are medication errors in the ED?

Medication-related problems have been reported in up to 11% of ED visits. The volume of medications given in the ED makes it ripe for errors. The weight-based dosing and calculations required for prescribing medications to children make medication errors in a pediatric ED a real issue. In a study by Kozer and colleagues, 10% of all patients seen in a pediatric ED had a prescribing error. In a study by Taylor and coworkers, almost 60% of prescriptions written in a pediatric ED contained an error.

Kozer E, Scolnick D, Macpherson A, et al: Variables associated with medication errors in pediatric emergency medicine. Pediatrics 2002;110:737-742.

Taylor BL, Selbst SM, Shah AEC: Prescription writing errors in the pediatric emergency department. Pediatr Emerg Care 2005;21:822-827.

9. What role do nurses and pharmacists play in medication errors in the ED?

In 2009, Shaw and associates surveyed 21 pediatric EDs and found that 75% of medications are prepared by a nurse, with the remaining 25% prepared by a pharmacist. This increases the risk for error, as ED nurses are often multitasking and may be distracted during medication preparation. They are also administering the medications most of the time, which means they play a critical role in the hospital's "safety net." Medication errors thus impact patients but also the nurses, who feel the brunt of medication errors. Nurses have identified the electronic health record (EHR) and bar coding to be important aids in reducing medication errors. In a recent study, having an ED pharmacist review the discharge prescriptions reduced some prescription errors and optimized therapy for 8.5% of adult prescriptions and 23.6% of pediatric prescriptions.

Cesarz JL, Steffenhagen AL, Svenson J, et al: Emergency department discharge prescription interventions by emergency medicine pharmacists. Ann Emerg Med 2013;61:209-214.
Mattei JL, Gillespie GL: Pediatric emergency nurses self-reported medication safety practices. J Pediatr Nurs 2013;28:596-602.
Shaw KN, Ruddy RM, Olsen CS, et al: Pediatric patient safety and emergency departments: Unit characteristics and staff perceptions. Pediatrics 2009;124:485-491.

10. What are the most common types of medication errors in a pediatric ED?

Medication dosing errors are the most common type. Pediatric medicines, for the most part, have weight-based dosing. Incorrect recording of weight or incorrect calculation of dose results in the most errors. This can lead to severe morbidity, because often the mistake is a "10-fold error" because of a misplaced decimal point. Giving an incorrect drug is the next most common type of medication error. This mistake is usually due to similar packaging of drugs or medication names that sound alike. Administration of a medication to a child with a known allergy to the medication is another common type of medication error. The most common outcome of medication errors is that no harm is done. There is, however, potential risk of prolonged hospital stay, additional care required, and death.

American Academy of Pediatrics, Committee on Pediatric Emergency Medicine. Patient safety in the pediatric emergency care setting. 2007;120:1367-1375.
Kozer E, Scolnick D, Macpherson A, et al: Variables associated with medication errors in pediatric emergency medicine. Pediatrics 2002;110:737-742.
Selbst S, Fein JA, Osterhoudt K, et al: Medication errors in a pediatric emergency department. Pediatr Emerg Care 1999;15:1-4.

Key Points: Medication Errors in the Pediatric Emergency Department

1. The largest threat to children in the ED is medication errors, most of which are dosing errors.
2. The ED environment is a challenge, largely because of its unstructured and hurried environment, and patients of varied sizes that present with unpredictable issues, with different levels of urgency, at unscheduled times.
3. Better ED systems, communication, and teamwork can reduce errors. Knowing the risk of errors is a big first step.

11. What is a transition? What makes it a risk in emergency medicine?

Transition is the transfer of care between care providers. Examples of transitions in the ED are "change of shift," a visit by a specialty consultant, admission of the patient to an inpatient unit, and a resident presenting the patient to an attending physician. Transitions interrupt continuity of care and are a source of potential error. Staff should be educated on the risks created by the transfer of a patient's care, and best practices for safe transitions should be promoted. Protocols for appropriate transitions are beginning to become part of residency training. Medication reconciliation—the process of comparing a patient's medication list before and after an encounter and correcting discrepancies—when coupled with a safe approach to transitions can improve patient care and decrease errors.

A transition also takes place when a patient is leaving medical care to go home to be cared for by the family. Clear communication is essential to ensure the care continues safely at home. One tactic that is proving helpful is postdischarge phone calls, during which a nurse can give support to family members transitioning care to home, answer questions, and clarify instructions.

Bucaro PJ, Black E: Facilitating a safe transition from the pediatric emergency department to home with a post-discharge phone call: A quality-improvement initiative to improve patient safety. J Emerg Nurs 2014;40:245-252.
Cheung DS, Kelly JJ, Beach C, et al: Improving handoffs in the emergency department. Ann Emerg Med 2010;55:171-180.
Kwan JL, Lo L, Sampson M, et al: Medication reconciliation during transitions of care as a patient safety strategy: A systematic review. Ann Intern Med 2013;158:397-403.
Samuels-Kalow ME, Stack AM, Porter SC: Effective discharge communication in the emergency department. Ann Emerg Med 2012;60:152-159.

12. What information is essential for a handoff in the ED?

- Relevant medical and surgical history
- Patient course, current condition
- Studies obtained and pending
- Suspected diagnosis
- Anticipated disposition

Cheung DS, Kelly JJ, Beach C, et al: Improving handoffs in the emergency department. Ann Emerg Med 2010;55:171-180.

Dhingra KR, Elms A, Hobgood C: Reducing error in the emergency department: A call for standardization of the sign-out process. Ann Emerg Med 2010;56:637-642.

13. How common are interruptions in the ED?

There is an association between interruptions in work flow and medical errors. A recent study by Berg and colleagues showed an interruption rate of 5.1 interruptions an hour for all staff and providers in an ED. Some other studies report even higher rates. A study from a level 1 trauma center found physicians had 10 interruptions per hour and nurses had 12 interruptions per hour. The staff workers were interrupted by other people, phones, and pagers. The ED staff members performed between one and eight other activities before returning to the original task.

Interruptions during the preparation of medication are common and create a risk for medication errors. Clinicians do not always perceive interruptions as a negative thing. Some interruptions are unavoidable, as in the case of deteriorating patients or critical findings.

Berg LM, Kallberg AS, Goranson KE, et al: Interruptions in emergency department work: An observational and interview study. BMJ Qual Safety 2013;22:656-663.

Brixey JJ, Tang Z, Robinson DJ, et al: Interruptions in a level 1 trauma center: A case study. Int J Med Inform 2008;77:235-240.

Pruitt CM, Liebelt EL: Enhancing patient safety in the pediatric emergency department. Pediatr Emerg Care 2010;26:942-951.

14. Why are children with special health care needs vulnerable to error in emergency care?

Children with special health care needs are a growing population representing 16% to 18% of children in the United States. They are at risk for medical error in the ED for several reasons:

- Some of their problems are occult and difficult to recognize, thus delaying care.
- Some conditions are recognizable but refractory to standard therapy, thus delaying receipt of the best care for that particular patient.
- Their baseline condition (e.g., vital signs, mental status) may not be known to the ED provider. Thus, severity of illness can be underestimated or overestimated.
- They often have rare conditions that are unfamiliar to the emergency care provider.
- Many require technologic devices that are not available to the ED providers.

Sacchetti A, Sacchetti C, Carraccio C, et al: The potential for errors in children with special health care needs. Acad Emerg Med 2000;7:1330-1333.

15. What can be done to make children with special health care needs safer when receiving emergency care?

An emergency information form, promoted and conceptualized in part by the American College of Emergency Physicians and the American Academy of Pediatrics, can help ED physicians provide more efficient and appropriate care to children with special health care needs. The form may contain contact information, past medical history and procedures, common presenting problems, and suggestions for management strategies. Coupled with medical ID jewelry and electronic information transfer, the emergency information form has the potential to improve safety for this at-risk group.

Sacchetti A, Sacchetti C, Carraccio C, et al: The potential for errors in children with special health care needs. Acad Emerg Med 2000;7:1330-1333.

16. When caring for a patient with limited English proficiency in the ED, would a family member interpreter be sufficient?

No, it would not be ideal. In one pediatric study, one clinical encounter generated 31 errors in medical interpretation. Most errors were of omission and had potential for clinical consequences. Errors committed by ad hoc interpreters (hospital staff, family members, other patients in the ED) as opposed to hospital interpreters were more likely to have clinical consequences. The legal liability of not ensuring appropriate interpretation is large.

A $71 million lawsuit was generated because of one misinterpreted word in the ED. One family was erroneously separated by social services for misinterpretation in a child abuse case. Efforts should be made to provide reliable, professional language interpretation for ED patients and families.

Flores G, Laws MB, Mayo SJ, et al: Errors in medical interpretation and their potential clinical consequences in pediatric encounters. Pediatrics 2003;111:6-14.

17. Why would physicians conceal a medical error?

- The medical profession values perfection.
- The doctor may feel shame or guilt.
- The doctor may fear damage to his/her reputation and decreased income.
- There is a need to maintain trust with the patient/family.
- There may be pressure from administration or other parties.
- There is often fear of punishment.
- There is often fear of a malpractice lawsuit.

American Academy of Pediatrics, Committee on Pediatric Emergency Medicine. Patient safety in the pediatric emergency care setting. 2007;120:1367-1375.

Finkelstein D, Wu AW, Holtzman NA, et al: When a physician harms a patient by a medical error: Ethical, legal and risk management considerations. J Clin Ethics 1997;8:330.

Selbst SM: The difficult duty of disclosing medical errors. Contemp Pediatr 2003;20:51-63.

18. Are physicians obligated to disclose medical errors to patients?

The American Medical Association Code of Ethics guidelines for professional conduct state: "The physician is ethically obligated to inform the patient of all the facts necessary to ensure understanding of what has occurred when a patient experiences a significant medical complication from a mistake." Disclosure regarding minor errors with little consequence is not specifically addressed. Several studies have found that patients and parents want the physician to disclose a medical error. Patients want the physician to apologize for an error. Families should be assured that everything is being done to discover how the error occurred and to prevent it from happening again. Many hospitals now support a full disclosure policy when an error has been found.

American Medical Association: Code of Medical Ethical Current Opinions. Chicago, IL, AMA, 2003, Sect 8.121.

Gallagher TH, Waterman AD, Ebers AG, et al: Patients' and physicians' attitudes regarding the disclosure of medical errors. JAMA 2003;289:1001-1007.

19. Is disclosure of a medical error likely to lead to a malpractice lawsuit?

There is no evidence for this. Most patients, when asked, would be more likely to distrust a doctor, take their care elsewhere, or bring a lawsuit against a physician who conceals an error as opposed to one who is truthful and forthcoming with information. In Hobgood's survey of parents who presented to an ED with a child, 99% wanted disclosure of hypothetical errors presented to them in scripted scenarios. Only 39% said they wanted the doctor to be reported to a disciplinary board because of the hypothetical error. Thirty-six percent said they were less likely to sue if the physician informed them of the error. However, if parents thought the error was severe, their desire for legal action was less amenable to reduction by disclosure.

Hobgood C, Tamayo-Sarver JH, Elms A, Weiner B: Parental preferences for error disclosure, reporting, and legal action after medical error in the care of their children. Pediatrics 2005;116:1276-1286.

Whitman AB, Park DM, Hardin SB: How do patients want physicians to handle mistakes? Arch Intern Med 1996;156:2565-2569.

20. How should an ED physician approach a family after an error is discovered?

- Investigate the problem first: Make sure there was an error.
- Follow hospital policy. Discuss the case with risk management according to policy.
- Find an appropriate time (when the family might be less stressed) and place (quiet area) to talk with the family.
- Sit with the family and speak at eye level.
- If an error is uncertain, advise the family that the event will be investigated.
- If a mistake is certain, apologize. Say you are sorry.
- Be plausible; do not mislead the parents or patient.
- Avoid placing blame on others.
- Reassure the family that any effects of the error will be managed to mitigate harm to the child.

21. What can be done to prevent errors in a pediatric ED?

Errors and error prevention must be addressed instead of hidden. Creating a culture in which patient safety is part of an open process is a very important step toward error reduction. An environment that encourages staff to detect and report errors is crucial. An environment that blames individuals hinders the reporting process. Physicians and administrators must focus on systems that lead to error. Medical errors are rarely the result of one individual and are most commonly due to the ED process. In order to make the ED more safe, leaders must also address workforce fatigue issues, ensure quiet areas for calculation and drawing-up of medications, develop a system to allow for independent double-checking of medications, carefully mentor and monitor trainees, and analyze near misses (Table 70-1).

22. How important is good communication among staff in reducing errors in the ED?

Effective communication and team leaders who are receptive to feedback can decrease the likelihood of an error reaching the patient. Authority gradients between health care workers (authority of attending over residents, residents over students, physicians over staff, and between physicians of different disciplines) can obstruct free dialogue and impede posing a question that could protect a patient's safety. Standardized communication strategies can help level the playing field so that all concerns can be heard. One framework for sharing critical information is represented by the acronym SBAR:

- Situation (What is going on?)
- Background (What is the context?)
- Assessment (What do I think the problem is?)
- Recommendation (What do I think should be done?)

23. How well does the ED staff communicate with patients in the ED?

Several studies show that ED staff members do not always communicate clearly with patients in the ED, and this can lead to medical errors and poor care. In one study, patients were surveyed after an ED visit about their knowledge of four domains: (1) diagnosis and cause of their condition, (2) ED care they received, (3) post-ED care recommended, and (4) return instructions. In this study, 78% of those surveyed did not understand one of these domains and 51% did not understand two or more of them.

In another study, ED staff members were recorded while giving discharge instructions to patients. This study revealed that patients in the ED often had minimal opportunities to ask questions or confirm their understanding of the instructions. Only 34% were told which symptoms should prompt a return to the ED.

Table 70-1. Preventing Medication Errors in the Emergency Department (ED)

1. Educate physicians, nurses, pharmacists, and manufacturers about potential errors.
2. Create systems in the ED to make errors less likely.
3. Remove dangerous medications that are rarely used from ED shelves. For example, because tetanus immunoglobulin is rarely needed and sometimes confused with tetanus toxoid, do not keep this drug in the ED. When needed, order it from the hospital pharmacy.
4. Reduce the noise level in the area where medications are drawn.
5. Reduce calculations for drug ordering as much as possible. Use length-based tapes, books, or computer programs with precalculated doses based on the patient's weight.
6. Avoid abbreviations (such as qd) in drug ordering as much as possible.
7. Always place a zero before a decimal point (such as 0.5 mg), and avoid use of a terminal zero (such as 5.0 mg). Misinterpretation could result in a 10-fold error.
8. Highlight patient drug allergies in the record.
9. Record weights of pediatric patients clearly and only in kilograms.
10. Note the child's weight in an easily located area of the medical record, perhaps near where orders are written.
11. Consider computerized order entry to help reduce computational errors and errors related to patient allergies.
12. Welcome a nurse or pharmacist who questions an order. Do not act defensively or refuse to investigate a possible error.
13. Consider bar coding of medications.
14. Consider using an automated drug-dispensing system.

Finally, one study found that only 60% of guardians for pediatric patients complied with discharge instructions to follow-up with a physician after leaving the ED. Of course, the guardians in this study may have just disregarded their instructions, but it is also possible that follow-up instructions were not made clear at the time of discharge.

Engel KG, Heisler M, Smith DM, et al: Patient comprehension of ED care and instructions. Ann Emerg Med 2009;53:454-461.

Vashi A, Rhodes KV: "Sign right here and you're good to go," a content analysis of emergency department discharge instructions. Ann Emerg Med 2011;57:315-322.

Wang N, Kiernan M, Golzari M, et al: Characteristics of pediatric patients at risk of poor emergency department aftercare. Acad Emerg Med 2006;13:840-847.

24. What lessons learned from aviation can help the ED?

Aviation is a field similar to medicine in that the stakes are high, the environment is stressful, and errors can cost lives. Twenty years of research in aviation has shown that effective teamwork and communication are essential. The IOM and the Agency for Healthcare Research and Quality (AHRQ) have suggested adopting principles of aviation known as CRM (crew resource management) into health care to enhance patient safety. CRM improves safety through improving operational reliability and reducing the risk of error by using all resources at hand. CRM emphasizes six key areas: (1) managing fatigue, (2) creating and managing teams, (3) recognizing adverse situations, (4) cross-checking and communication, (5) developing and applying shared mental models for decision making (known in health care as "best practices"), and (6) giving and receiving performance feedback. The evidence is growing that CRM principles are increasing patient safety in the ED setting.

Pruitt CM, Liebelt EL: Enhancing patient safety in the pediatric emergency department. Pediatr Emerg Care 2010;26:942-951.

25. How does computer provider order entry (CPOE) affect patient safety?

CPOE, the process by which orders for patient care are directly entered into the computer by the treating provider, can improve patient safety by making medication ordering safer. The software can provide decision support, check allergies and weights, and eliminate transcription errors. CPOE avoids omitted information on generated prescriptions, and the prescriptions can be sent electronically to a given pharmacy, enhancing efficiency. Implementation of CPOE in one intensive care unit setting decreased medication errors by 99% and adverse drug events by 40%. Unfortunately, there can be new types of errors inherent in the system. The frequency of inexact or clinically insignificant drug alerts causes "alert fatigue" and potentially important warnings may be overridden. There can also be adverse events if allergy information is not updated, or if the patient's weight is entered incorrectly. Wrong order–wrong patient errors are created by selecting a chart or laboratory result that is not for the intended patient prior to placing an order. Since 2006 there has been an increase in publications about the impact of CPOE in the ED. There is no consistency in the findings except that CPOE increased physician computer time and did not decrease patient time, but did decrease medication errors. The U.S. Emergency Medicine Information Technology Consensus Conference concluded that if there is a weakness in a CPOE system, it will first fail in the ED because of the complex, high-risk, fast-paced setting.

Farley HL, Baumlin KM, Hamedani AG, et al: Quality and safety implications of emergency department information systems. Ann Emerg Med 2013;62(4):399-407.

Georgiou A, Prgomet M, Paoloni R, et al: The effect of computerized provider order entry systems on clinical care and work processes in emergency departments: A systematic review of the quantitative literature. Ann Emerg Med 2013;61:644-653.

Hsieh T, Kupperman G, Jaggi T, et al: Characteristics and consequences of drug allergy alert overrides in a computerized physician order entry system. J Am Med Inform Assoc 2004;11:482-491.

Potts A, Barr F, Gregory D, et al: Computerized physician order entry and medication errors in a pediatric critical care unit. Pediatrics 2004;113:59-63.

26. What are the implications and challenges of information technology in the ED?

The Health Information Technology for Economic and Clinical Health Act of 2009 encourages hospitals to use EHRs. Penalties will be imposed on hospitals that do not comply by 2015. In addition, new needs for reporting quality metrics have been created by the Centers for Medicare and Medicaid Services "meaningful use" incentives. These pressures have galvanized hospitals to adopt electronic medical records (EMRs) and ED information systems (EDISs).

These systems are challenged to aid in the delivery of emergency medical care by the variety and complexity of care, fast pace, and inability to control processes such as patient arrival. EMRs and EDISs are challenged further by the pediatric emergency patient by the need for weight-based dosing and the lack of standard documentation of the physical examination due to different findings at different ages (i.e., older children do not have a fontanel to document).

Farley HL, Baumlin KM, Hamedani AG, et al: Quality and safety implications of emergency department information systems. Ann Emerg Med 2013;62(4):399-407.

Hoffman JM, Zorc JJ, Harper MB: IT in the ED: Past, present, future. Pediatr Emerg Care 2013;29:402-405.

27. How can simulation help in the pediatric emergency environment?

Pediatric codes and resuscitations are much less frequent than those for adults. Simulation allows for practice of skills and improved comfort during difficult and rare situations. One study showed improved adherence to pediatric advanced life support (PALS) guidelines during mock codes when high-fidelity simulation was used instead of traditional mannequins. Simulation aids in training for rare resuscitation events as well as procedures such as fiberoptic intubation or placement of central lines, nasogastric tubes, Foley catheters, and chest tubes. Assessment and testing of skills as well as practice of teamwork and communication are possible through simulation. Patterson and coworkers demonstrated improved safety attitudes and teamwork behaviors in a pediatric ED after multidisciplinary simulation-based training emphasizing teamwork and communication.

Dull KE, Bachur RG: Simulation in the pediatric emergency department. Clin Pediatr 2012; 51(8):711-717.

Griswold S, Ponnuru S, Nishisaki A, et al: The emerging role of simulation education to achieve patient safety: Translating deliberate practice and debriefing to save lives. Pediatr Clin North Am 2012;59:1329-1340.

Patterson MD, Geis GL, LeMaster T, et al: Impact of multidisciplinary simulation-based training on patient safety in a paediatric emergency department. BMJ Qual Safety 2013;22:383-393.

28. What are the advantages of simulation training?

Simulation enables training for especially rare situations or procedures. It provides active learning, repeat experience, immediate feedback, control of degree of difficulty, and a safe environment without the fear of harming patients. Errors that occur during the early stages of learning procedures and nontechnical skills can be resolved on simulated rather than real patients. The more authentic the simulation, the better the learning experience. The most real simulators can even evoke the clinicians' response to a stressful patient event. Elevated heart rates and salivary cortisol have been demonstrated in users of high-fidelity simulation compared to trainees using traditional means of learning.

29. What are the challenges of simulation?

- Cost: A high-fidelity simulator ranges in cost from $30,000 to $250,000. Starting a simulation center can cost $200,000 to $1.6 million.
- Realism: Even with functioning monitors and a simulator that mimics breathing, some physiologic parameters (breaths sounds, mental status, skin color, heart tones) seem synthetic. Chest wall movement and palpable pulses have been reported as the most helpful simulated features.

Dull KE, Bachur RG: Simulation in the pediatric emergency department. Clin Pediatr 2012;51(8):711-717.

Griswold S, Ponnuru S, Nishisaki A, et al: The emerging role of simulation education to achieve patient safety: Translating deliberate practice and debriefing to save lives. Pediatr Clin North Am 2012;59:1329-1340.

30. What resources are available to learn about patient safety?

- AHRQ (www.ahrq.gov/qual): A division of the U.S. Department of Health and Human Services with a mission to improve the quality, safety, efficiency, and effectiveness of health care for all Americans
- National Guideline Clearinghouse (www.guideline.gov): A public resource for evidence-based clinical practice guidelines and an initiative of the AHRQ. Search terms can be entered.
- National Patient Safety Foundation (www.npsf.org): Nonprofit organization devoted to understanding patient safety issues and how to improve them, as well as promoting public awareness and fostering communication

- Institute for Safe Medication Practices (www.ismp.org): Nonprofit organization devoted entirely to medication error prevention
- The Leapfrog Group (www.leapfroggroup.org)
- The American Hospital Association (www.hospitalconnect.com/aha/about/index.html)
- Institute for Healthcare Improvement (www.ihi.org)
- Joint Commission on Accreditation of Healthcare Organizations (www.jcaho.org)

RISK MANAGEMENT AND LEGAL ISSUES

Steven M. Selbst

CHAPTER 71

1. Which diagnoses involving pediatric patients in an emergency department (ED) are most likely to result in malpractice suits?

Most malpractice suits involving children in an ED result from failure to diagnose meningitis, appendicitis, fractures, and testicular torsion. In addition, failure to recognize sepsis (especially meningococcemia), medication errors, errors in wound management, and failure to diagnose slipped capital femoral epiphysis, myocarditis, dehydration, pneumonia, and child abuse are among the common sources of lawsuits in pediatric emergency medicine.

Carroll AE, Buddenbaum JL: Malpractice claims involving pediatricians—Epidemiology and etiology. Pediatrics 2007;120:10-17.

McAbee GN, Donn SM, Mendelson RA, et al: Medical diagnoses commonly associated with pediatric malpractice lawsuits in the United States. Pediatrics 2008;122:e1282-e1286.

Selbst SM, Friedman MJ, Singh SB: Epidemiology and etiology of malpractice lawsuits involving children in US emergency departments and urgent care centers. Pediatr Emerg Care 2005;21:165-169.

2. Why are emergency physicians at high risk for involvement in a malpractice suit?

Whenever the outcome is poor, an emergency physician is likely to be the subject of a malpractice suit, especially if the family is angry during their visit to the ED. Anger is a major force in initiating a lawsuit. Families in the ED may be dissatisfied before they even interact with the physician. They may be angry because of a long waiting time to see the physician. Sometimes they are angry with a discourteous staff member. The impersonal setting in the ED may be another contributing factor. It is often difficult to establish rapport with a family during a brief visit to the ED, which also puts the physician at a disadvantage if the outcome is poor.

Selbst SM, Koren JB: Preventing Malpractice Lawsuits in Pediatric Emergency Medicine. Dallas, American College of Emergency Physicians, 1999, pp 1-196.

3. Why are cases of testicular torsion often the subject of malpractice lawsuits?

- Other conditions mimic testicular torsion (trauma to the scrotum, torsion of the appendix testes, epididymitis).
- Some patients have atypical presentations (complain of abdominal pain rather than scrotal pain).
- Minor trauma to the scrotum can confuse the picture.
- Examination findings may be inconsistent (cremasteric reflex is not 100% reliable).
- No single test is always diagnostic (not even Doppler ultrasound).
- The child may not be completely undressed—the physician may fail to examine the genitalia when the child complains of abdominal pain.
- Physicians may not realize testicular torsion is found in young infants as well as older boys.
- Delay in consultation or delay in action by the urologist can lead to a bad outcome (time is of the essence).

4. Why are cases of appendicitis frequently the subject of malpractice lawsuits?

- Numerous conditions can cause abdominal pain and mimic appendicitis.
- Young children may have nonspecific signs of pain such as irritability, lethargy, poor feeding.
- Appendicitis may not be considered in a young child.
- Physical examination of a child can be challenging because of lack of cooperation.
- Features of "classic" appendicitis may be absent; atypical cases are common.
- Diarrhea may confuse the picture with gastroenteritis.
- The examination can be misleading; tenderness in the right lower quadrant may be absent. (An appendix in the lateral gutter can cause flank pain and lateral abdominal

tenderness. An appendix that lies toward the left can cause hypogastric tenderness. Pain may be elicited only on deep palpation if the appendix is retrocecal.)
- Diagnostic studies may not be helpful. (Leukocytosis and pyuria are nonspecific; the appendix may not be visualized with ultrasound.)
- The physician may fail to observe the child or arrange follow-up.
- The physician may fail to document findings.

5. Which physicians have the highest average settlement per case? Why?

According to the Physician Insurers Association of America (1985-2011), pediatricians have the fourth highest average indemnity per case compared with physicians of 28 fields of medicine. Only those cases from neurology, neurosurgery, and obstetrics/gynecology have higher mean payouts. The average payment for pediatric claims is about $282,000 ($384,000 per case for the years 2005-2009).

A recent study, with data from a single insurer covering physicians in every state, showed that pediatricians had the *highest* mean indemnity of all specialties, $521,000. This compares to a mean of $275,000 for all specialties and $180,000 for emergency medicine physicians. Pediatricians are perhaps less likely to be sued than other specialties (3.1% sued annually compared to 7.4% of all physicians sued annually), but when there is a payout, the pediatric cases are generally high.

Pediatricians likely face more substantial claims because payouts are assessed over the patient's lifetime of lost earnings and medical costs—a much longer period for an injured child. Also, juries tend to be sympathetic to the family when there is an injured child.

Jena AB, Seabury S, Lakdawalla D, et al: Malpractice risk according to physician specialty. N Engl J Med 2011;365:629-636.

Physician Insurers Association of America. Available from www.thepiaa.org.

6. How is the "standard of care" defined?

The standard of care is defined as care that a reasonable physician in a particular specialty would give to a similar patient under similar circumstances. Physicians generally are held to the same standard of care across the country—a national level of competence. It is assumed that all physicians have the same knowledge of current procedures, treatments, and practices. Although not every doctor has the same access to specialists and technology, it is usually expected that an emergency physician will recognize a medical condition and attempt to get the proper care for the child as soon as reasonably possible.

7. In addition to civil charges, can criminal charges be brought against an emergency physician in a malpractice setting?

Very rarely. In some cases, such as failure to report a case of drug abuse, child abuse, or injury by a weapon, an emergency physician may be charged with a misdemeanor. Such charges usually are not made unless it is believed that the doctor was deliberately uncooperative. If a patient has died, the doctor can be charged with manslaughter if it is believed that an extreme or unusual breach of the physician's duty took place. The court may determine that the physician's actions were reckless or disregarded the rights and safety of others. Again, this event is very rare.

8. What is the statute of limitations?

The statute of limitations sets the length of time in which a person may bring a lawsuit for an alleged injury. Each state sets its own statute of limitations, but most states set a limit for adult patients of 2 to 3 years from the time that an injury due to alleged negligence is discovered or should have been discovered. After that time has passed, a malpractice suit cannot be initiated, regardless of the merits of the case. In a malpractice case involving a child, the time period does not begin until the child has reached the age of majority (18-21 years of age) in many states, because the child is unable to initiate legal action on his or her own behalf. Thus, it is possible for a lawsuit to be filed 18 to 20 years after the alleged injury occurred.

Selbst SM, Korin JB: Malpractice and emergency care—Doing right by the patient and yourself. Contemp Pediatr 2000;17:88-106.

Shea KG, Scanlan KJ, Nilsson KJ, et al: Interstate variability of the statute of limitations for medical liability: A cause for concern? J Pediatr Orthop 2008;28(3):370-374.

Zane RD: The legal process. Emerg Med Clin North Am 2009;27:583-592.

9. What percentage of malpractice lawsuits is brought to a jury for a verdict?

Only about 10% of malpractice cases reach a jury verdict. Most are settled out of court, and some are dropped altogether. Nonetheless, a malpractice lawsuit, once initiated, is a long and stressful event for the physicians involved. The duration of claims against physicians lasts 11 to 43 months. Physicians undergoing litigation stress often feel isolation, sadness, anger, disbelief, and a sense of betrayal.

Jena AB, Seabury S, Lakdawalla D, et al: Malpractice risk according to physician specialty. N Engl J Med 2011;365:629-636.

10. How are plaintiffs rewarded in malpractice lawsuits involving children in the ED?

Although millions of dollars are paid out in malpractice lawsuits each year, money is actually paid to the plaintiffs who initiate the lawsuit in less than one third of closed claims. The overwhelming majority of payments occur in pretrial settlements. If a case proceeds to trial, the plaintiff is rarely rewarded. Less than 2% of cases result in judgment for the plaintiff.

Physician Insurers Association of America. Available from www.thepiaa.org.
Selbst SM, Friedman MJ, Singh SB: Epidemiology and etiology of malpractice lawsuits involving children in US emergency departments and urgent care centers. Pediatr Emerg Care 2005;21:165-169.

11. What role does good communication with patients and families play in reducing malpractice suits?

Good communication with patients and families is crucial. A lawsuit is more likely when there is poor communication between family members and the medical staff. One study showed a direct correlation between good communication skills of physicians and fewer malpractice suits. A patient or family must perceive that the physician has a caring attitude, professional integrity, openness, and high standards of excellence. Another study of families who had sued physicians found that most mothers were dissatisfied with the doctor-patient communication. About 13% reported that the doctor would not listen to them, 32% noted that the doctor would not talk openly, 48% believed that the doctor attempted to mislead them, and 70% believed that the doctor did not warn them about long-term neurodevelopmental problems.

It is important to explain your thought process to families and acknowledge uncertainty about a diagnosis when appropriate. Tell families that symptoms of serious illness (e.g., appendicitis or meningitis) can develop later, and encourage them to seek care if the child's condition worsens. Unfortunately, communication at the time of discharge from the ED is not always adequate.

Hickson GB, Clayton EW, Githens PB, et al: Factors that prompted families to file medical malpractice claims following perinatal injuries. JAMA 1992;267:1359-1363.
Hickson GB, Federspiel CF, Pichert JW, et al: Patient complaints and malpractice risk. JAMA 2002;287:2951-2957.
Vashi A, Rhodes KV: "Sign right here and you're good to go." A content analysis of audio taped emergency department discharge instructions. Ann Emerg Med 2011;57:315-322.

12. What are the leading factors in medical errors and missed diagnoses that end in malpractice lawsuits?

The leading contributing factors in missed diagnoses related to malpractice lawsuits in the ED are cognitive factors, patient-related factors (atypical presentations, poor history reporting, nonadherence to instructions), lack of appropriate supervision, inadequate handoffs, and excessive workload. Trainees involved in errors that result in malpractice lawsuits most often made errors in judgment, but teamwork breakdowns (including handoff problems, lack of supervision) and lack of technical competence also play important roles.

Kachalia A, Gandhi TK, Puoolo AL, et al: Missed and delayed diagnosis in the emergency department: A study of closed malpractice claims from 4 liability insurers. Ann Emerg Med 2007;49:196-205.
Singh H, Thomas EJ, Petersen LA, et al: Medical errors involving trainees: A study of closed malpractice claims from 5 insurers. Arch Intern Med 2007;167(19):2030-2036.

13. How important is it to arrange for a follow-up examination of a child seen in the ED?

Physicians have been found liable when they failed to instruct parents, instructed them inadequately, or could not prove that they gave instructions to return to the ED or to seek care

elsewhere. In one case, a patient successfully sued because a doctor did not document that an incidental finding of hypertension had been addressed and follow-up arranged. The child eventually developed end-stage renal disease. Discharge instructions for patients or parents should include specific information about when to seek follow-up care with the child's primary care physician or specialist or when to return to the ED.

One study showed that even when instructions are given, only 60% of guardians for pediatric patients complied with discharge instructions to follow up with a physician after leaving the ED.

Samuels-Kalow ME, Stack AM, Porter SC: Effective discharge communication in the ED Ann Emerg Med 2012;60:152-159.

Wang N, Kiernan M, Golzari M, et al: Characteristics of pediatric patients at risk for poor emergency department aftercare. Acad Emerg Med 2006;13:840-847.

Yu KT, Green RA: Critical aspects of emergency department documentation and communication. Emerg Med Clin North Am 2009;77:641-654.

14. What is the physician's obligation to inform parents about a procedure or treatment?

Physicians are obligated to obtain informed consent from parents. Most states follow a "patient-focused" concept of informed consent. That is, the doctor must give the consenting person (guardian) a description of the procedure and the risks and alternatives so that a reasonably prudent person would be able to make an informed decision about the procedure. A physician must speak in "lay language" so that the guardian understands the information. A guardian or patient is entitled to know the diagnosis, nature of the proposed treatment or procedure, risks and side effects of the procedure, available alternatives, and risks involved with alternatives. Guardians or patients also are entitled to know the prognosis with and without treatment. Disclose all but the most remote risks if the outcome may be serious, but only common risks if they are likely to result in only minor harm to the child.

American Academy of Pediatrics Committee on Pediatric Emergency Medicine and Committee on Bioethics: Consent for emergency medical services for children and adolescents. Pediatrics 2011;128:427-433.

15. How important is it to get parents to sign a consent form for a procedure?

A signed consent form has some value. It provides documentation that an attempt was made to educate the guardian about a procedure. However, signing a form does not always equate with informed consent. A parent may still claim that risks and benefits were not adequately explained. The consent form must be a clearly written document in the language that most parents would understand. If a signed consent form is not used, the medical record should document that specific risks and benefits were explained to the guardian before the procedure was performed.

16. When is it permitted to treat a minor in the ED without consent from a parent or guardian?

Any minor can and should receive medical care in the event of a true emergency. Most states define an emergency in vague terms such as a situation that "threatens life and limb" or the "life and health" of a child. Such vague definitions are meant to protect a well-meaning physician. In reality, because an emergency is often difficult to define, perform a medical screening exam for every patient who presents to the ED, even if parents or guardians are not available to give consent. Furthermore, parental consent for treatment is generally not needed if a child presents to the ED with a chief complaint related to venereal disease, pregnancy, testing for HIV (human immunodeficiency virus), or drug and alcohol abuse. However, many states require that the child be over the age of 14 before treatment can be rendered for these conditions without parental consent.

American Academy of Pediatrics Committee on Pediatric Emergency Medicine and Committee on Bioethics: Consent for emergency medical services for children and adolescents. Pediatrics 2011;128:427-433.

Key Points: Communicating with Parents and Families in the Emergency Department

1. Demonstrate compassion and professionalism (try to appear unhurried).
2. Apologize for wait time.
3. Be a good listener.
4. Sit in the examination room, at eye level with the family.
5. Speak clearly in language the family will understand.
6. Hide your own frustrations.
7. Keep the family informed, and tell them what to expect.
8. Use a translator if needed.

17. What is an emancipated minor?

An emancipated minor is one who can seek and receive medical care without the consent of parents. In almost all 50 states, an emancipated minor is defined as one who:

- Is over the age of 18 years
- Has been married or self-employed
- Has graduated from high school
- Has served in the armed forces
- Is otherwise independent of parental care or control

Many states consider a pregnant minor to be emancipated and allow her to give consent for care for herself and her unborn baby.

Anderson SL, Shaechter J, Brosco JP: Adolescent patients and their confidentiality: Staying within legal bounds. Contemp Pediatr 2005;22:54-64.

18. What is the "mature minor doctrine"?

This law, recognized by less than half of all states, allows a minor over the age of 14 years (not necessarily emancipated) to consent to medical or surgical treatment even if it is not a true emergency. In the judgment of the treating physician, the minor must be "sufficiently mature to understand the nature of the procedure and its consequences." The treatment must be intended to benefit the minor rather than someone else and must not involve serious risks. For example, the mature minor doctrine was applied to the case of a 17-year-old girl who consented to receive treatment for a minor finger laceration without parental consent.

American Academy of Pediatrics Committee on Pediatric Emergency Medicine: Consent for emergency medical services for children and adolescents. Pediatrics 2003;111:703-706.

Kassutto Z, Vaught W: Informed decision making and refusal of treatment. Clin Pediatr Emerg Med 2003;4:124.

19. In which situations do parents *not* have the right to refuse treatment for their child?

If a life-threatening situation exists and the emergency physician believes that it is unsafe for a patient to leave the ED to seek care elsewhere, a family cannot sign out against medical advice or refuse care for their child. Likewise, if the patient or parent is under the influence of drugs or alcohol and cannot understand the risks and benefits of receiving or refusing care, the patient cannot be permitted to leave the ED. Finally, if child abuse is suspected, a guardian may not refuse care for the child.

20. Can parents refuse emergency care for their child based on their religious beliefs?

Usually not. Most courts in the United States will not allow a parent to impose his or her religious beliefs on a minor, especially in a life-threatening situation. In such situations it is generally unclear whether the child has the same religious beliefs and is "autonomous" or free from coercion or manipulation by the family. A young child may also be unable to comprehend the risks of refusing recommended treatment. Therefore, if a blood transfusion is essential for treatment, give the blood. If parents object, report the situation as medical neglect to the appropriate agency and obtain a court order for permission to treat, while simultaneously delivering the emergency care. When it is unclear if an emergency situation exists, err on the side of treatment.

21. What is the impact of the electronic medical record (EMR) on medical malpractice?

The answer to this question is not yet known. One study of office-based physicians showed users of EMRs were less likely to pay malpractice claims over a 10-year period than nonusers (6.1% versus 10.8%), but this difference was not statistically significant. The EMR likely improves medical care and reduces errors in the ED. With EMRs, information about patients is readily available and clinicians can locate the previous ED visits and treatments given by specialists within the hospital system. Medication errors may be less likely because of computer order entry. However, EMRs are not without risk. EMRs provide more discoverable evidence for the plaintiff. Also, ED physicians can make errors using EMRs by clicking on templates quickly for items that are not correct for the patient. Drop-down lists can lead to errors, and copying and pasting erroneous material can perpetuate incorrect information. Finally, the EMR may lead to "information overload", and clinicians may therefore skip information that could turn out to be important.

Malgalmurti SS, Murtagh L, Mello MM: Medical malpractice liability in the age of electronic health records. N Engl J Med 2010;363:2060-2067.

Virapongse A, Bates DW, Shi D, et al: Electronic health records and malpractice claims in office practice. Arch Intern Med 2008;168:2362-2367.

22. List guidelines for documentation in the medical record of a child who presents to the ED.

- Always document the child's chief complaint, even if it seems trivial.
- Be sure to record the child's medical history, allergies, immunization status, and current medications.
- Carefully describe the child's general appearance (active, playful) and state of hydration.
- Include important positive and negative findings, rather than just noting that the examination was "normal."
- Avoid derogatory or self-serving statements in the record.
- Make sure that the chart looks neat and professional.
- When appropriate, record a "progress note" to indicate a child has improved prior to discharge.
- *Never enhance or alter the record* after the child leaves the ED. Additions should be made cautiously. Handwritten notes must be timed and dated so as not to appear misleading. With an EMR, it is immediately known when the record was entered and what information was added or deleted.

Selbst SM, Koren JB: Preventing Malpractice Lawsuits in Pediatric Emergency Medicine. Dallas, American College of Emergency Physicians, 1999, pp 1-196.

Yu KT, Green RA: Critical aspects of emergency department documentation and communication. Emerg Med Clin North Am 2009;77:641-654.

23. How should an ED physician proceed if the referring primary care doctor disagrees with the ED physician's assessment and plan for the child?

It is not uncommon for a referring physician to have a specific plan in mind for a pediatric patient. However, once the child arrives in the ED, the emergency physician probably will have some liability if the outcome is poor. The ED physician may be considered to be in a better position to determine the child's need for treatment and hospital admission, especially if the referring doctor has not actually examined the patient. Thus, do not automatically defer to the referring doctor on the telephone. Try to reach an agreement about the child's care without compromising what you believe is the best plan for the child. If the referring doctor does not agree with the decision to admit a child, it is best for that doctor to come to the ED (if he or she has staff privileges) to examine the child and assume responsibility for the disposition. Do not delay emergency care while waiting for a primary care physician or specialist requested by the primary care physician to arrive in the ED. Hospital policies should be in place to guide the staff in such situations.

24. What role do consultants play in malpractice lawsuits in the ED?

Malpractice suits that originate in the ED frequently involve consultants, especially radiologists. The ED and hospital radiologists must have a system in place in which the ED is contacted whenever there is a discordant reading or a change in the official reading of the radiograph. Likewise, ED physicians and specialists/consultants must communicate clearly during a telephone consultation. There is legal risk whenever information is given or requested

by telephone. Finally, sometimes an ED physician disagrees with the recommendations of a specialist. The ED physician can be sued with the specialist if the patient has a bad outcome. When an ED physician has concerns about telephone advice of the consultant, he/she should insist that the specialist come to the ED to evaluate the patient directly. For all phone consultations, record the essence of the phone conversation with the specialist in the patient's chart.

The ED physician is not obligated to accept the advice of a consultant, but the recommendations of the specialist should not be rejected without careful consideration and discussion. The recommendations of the specialist will have great weight in court. When applicable, document why the consultant's recommendations are not followed.

25. What is the Emergency Medical Treatment and Active Labor Act (EMTALA)?

EMTALA states that all hospitals that receive Medicare funds and have an ED must provide an "appropriate medical screening examination" to all patients who present to the ED to determine whether a medical emergency exists. EMTALA was developed to protect patients without medical insurance from being "dumped" by some hospitals, but the law applies to patients with insurance as well as to those who belong to a managed care plan. Many experts recommend that an emergency physician perform the medical screening examination rather than a nurse. However, EMTALA has been updated, and hospitals may allow nonphysicians to perform the screenings as long as hospital policies define in writing that such individuals are authorized to perform the screenings. The acuity of the patient's illness may indicate whether a physician should perform the screening. A medical screening examination may range from a brief history and physical examination to a complex process involving ancillary studies and procedures. The screening examination must include all appropriate ancillary tests and services normally available to any patient. The tests must be ordered regardless of the patient's insurance if they are needed to determine whether an emergency exists.

Bitterman RA: EMTALA and the ethical delivery of hospital emergency services. Emerg Med Clin North Am 2006;24(3):557-577.

Testa PA, Gang M: Triage, EMTALA, consultations and prehospital medical control. Emerg Med Clin North Am 2009;27:627-640.

Key Points: Tips for Preventing Malpractice Lawsuits in Pediatric Emergency Medicine

1. Use caution in treating children with fever and abdominal pain.
2. Use caution if the patient is unable to ambulate upon discharge without a good explanation.
3. Use caution in performing a lumbar puncture (LP) in a small infant, especially if the baby is in respiratory distress (risk of apnea when the baby is curled up for an LP).
4. Remember that care given by others in the hospital (and prehospital) will affect the liability of the ED staff.
5. Communicate carefully with colleagues, especially around change of shift.
6. Consider pathologic examination beyond the gastrointestinal tract if a child has vomiting.
7. Do not allow consultants to avoid cases when their help is needed.
8. Ask for help when managing complex wounds.
9. Read the notes of others involved in the child's care.
10. Document patient improvement in a discharge note.

26. When is transfer of a child from the ED in compliance with EMTALA?

A patient may be transferred from the ED if the transfer is medically indicated and the patient needs a level of care that is not available at the transferring hospital. A patient is considered stable when no material deterioration of the emergency medical condition is likely, within reasonable medical probability, to result from or occur during the transfer of the individual from the facility. An unstable child may be transferred if the patient or parents make the request. Informed consent should be obtained if the patient or parent requests the transfer. If the transfer is medically indicated, the emergency physician must document that the benefits of transfer outweigh the risks and must arrange for an "appropriate" or safe transfer.

Before transfer of the patient, obtain agreement to accept the patient from a physician or responsible individual at the receiving hospital. When a patient requires a higher level of care than that which can be provided at the transferring facility, a hospital with the capability and

capacity to provide a higher level of care may not refuse any request for transfer. Send an appropriate medical summary and other pertinent records with the patient to the receiving facility, or have these records electronically transferred as soon as is practical.

Testa PA, Gang M: Triage, EMTALA, consultations and prehospital medical control. Emerg Med Clin North Am 2009;27:627-640.

Woodward GA: Legal issues in pediatric interfacility transport. Clin Pediatr Emerg Med 2003;4:256-264.

27. Is a pediatric ED responsible for an adult with an emergency according to EMTALA?

If a patient is within 250 yards of the hospital, EMTALA rules apply. Thus, if a person (child or adult) collapses on the parking lot or sidewalk outside the ED, or in the hospital gift shop, the hospital is obligated to screen and appropriately stabilize the individual, regardless of the person's age or ability to pay. Pediatric ED staff should screen and attempt to stabilize the adult patient within their capabilities. Staff should not just call emergency medical services without first starting to treat the adult. The hospital should generate a medical record for the adult patient, and the physician may wish to certify that benefits of transfer to a hospital for adults outweighs the risks of continuing treatment in the pediatric hospital, which has limited capabilities.

28. What is the policy of the American College of Emergency Physicians (ACEP) about providing telephone advice from the ED?

The ACEP policy (revised 2/2013) states that most medical conditions cannot be accurately diagnosed by telephone. Each ED should have a procedure in place to identify the nature of all incoming calls. Give persons making calls about mental health emergencies, or life- or limb-threatening medical emergencies, information about how to access the emergency medical services system. Manage calls from patients recently discharged from the ED by prearranged protocols that include the circumstances in which the patient should return to the ED.

In the interest of good patient care (and except for telemedicine or toxicology services), the ACEP recommends that EDs not attempt medical assessment or management by telephone. Advise callers that EDs are available at all times to assess their condition.

American College of Emergency Physicians. Available from www.acep.org.

29. What is the recommendation of ACEP for managing telephone orders called in to the ED by outside physicians?

ACEP policy (reaffirmed 10/2008) states that telephone orders for ED patients that are dictated by a physician from outside the ED can adversely affect the quality of medical care that patients receive and create legal liability for physicians. ACEP endorses the following principles:

- Hospital policy should specify the criteria for accepting telephone orders in the ED.
- Hospital policy should specify that all patients who come to the hospital for emergency care should be provided with an appropriate medical screening examination.
- Telephone orders directed to ED personnel should be subject to the review and approval of the emergency physician on duty.

It should also be noted that the Drug Enforcement Administration regulations prohibit dispensing controlled substances from ED stocks for treatment of patients by telephone order.

American College of Emergency Physicians. Available from www.acep.org.

30. What are the most important factors in patient satisfaction in the ED?

Patient satisfaction depends primarily on prompt treatment, the caring nature of the emergency nurses and physicians, and the degree of organization of the medical staff.

31. What are the most common sources of complaints resulting from pediatric visits to the ED?

Most patient or parent complaints concern waiting time, quality of medical care (including an incomplete medical examination or failure to order enough tests), attitude of the ED staff, and misdiagnosis. In a general ED, complaints about billing are frequent, but billing accounts for only about 20% of complaints from a pediatric ED. Other complaints from families involve an unclean appearance and lack of privacy in the ED. Complaints from parents in the ED are inevitable, but many are preventable. It generally requires less work to prevent a complaint than to manage a family after they have formally complained about their ED visit. Developing rapport with families is essential.

Table 71-1. Tips for Preventing Complaints in the Pediatric Emergency Department

1. Meet or exceed the expectations of the patient and family.
2. Introduce yourself to the family.
3. Pay attention to the patient and family.
4. Tell them why they are waiting.
5. Make the patient and family comfortable while they wait.
6. Defuse potential complaints during the visit.

From Cronan K: Pediatric complaints in a pediatric emergency department: Averting lawsuits. Clin Pediatr Emerg Med 2003;4:235-242.

32. What are some tips for preventing complaints in the pediatric ED?

Tips for preventing complaints in the pediatric ED are listed in Table 71-1.

Cronan K: Pediatric complaints in a pediatric emergency department: Averting lawsuits. Clin Pediatr Emerg Med 2005;4:235-242.

33. What are "good Samaritan statutes"?

Good Samaritan statutes exist in almost every state and provide immunity to physicians and others who err while administering emergency medical care to ill or injured people. The good Samaritan laws were passed to encourage physicians and others to stop at the scene of an emergency and offer assistance without fear of a malpractice suit. These statutes require that aid be given without compensation. They generally apply to care at the scene of an accident rather than in the ED or elsewhere in the hospital. Physicians are obligated to act in good faith but are not expected to put their own lives or their families in danger. Many statutes exclude "gross negligence" or "willful misconduct," but a physician who makes an honest mistake is likely to be protected. A lawsuit still may be initiated if the patient suffers a poor outcome after treatment at the scene of an accident, but the suit is very unlikely to be successful if the physician acted in good faith.

34. What should you do if named in a malpractice lawsuit?

Do not panic. However, if you receive a letter of *Complaint* from a plaintiff's attorney, take this matter seriously. Even if you disagree with everything in the Complaint (charges are often exaggerated), *do not ignore this*. The Complaint may list statements that are demoralizing or insulting, but remember they are only unproven accusations. Contact your insurance carrier as soon as possible about any potential lawsuit, even a threatening letter from an attorney. Early notification allows the insurance carrier to investigate the claim and assign an attorney to assist you and prepare a defense. Your attorney, once assigned to the case, will send an *Answer* to the Complaint (generally denying the allegations) within a certain time frame. It is possible that a *default judgment* against the physician may result if the doctor fails to notify his or her carrier and the defense team is not given adequate time to answer a claim.

Tell your attorney everything, and help him/her develop the case. Make some recommendations for a possible expert witness for your case. Do not discuss the case with colleagues. Do not call the patient or his/her family.

35. Which actions are reported to the National Practitioner Data Bank (NPDB)?

- Any adverse licensure action, whereby a physician is denied a medical license because of professional incompetence or misconduct, or has a license revoked
- Any action by a professional society, hospital, or other health care facility that adversely affects a physician's clinical privileges; includes voluntary surrender of clinical privileges while under investigation or in return for not conducting an investigation
- Criminal convictions related to health care
- All medical malpractice payments (payors must submit reports to the NPDB about payments to settle a claim within 30 days of payment)

36. Who is permitted to access information from the NPDB?

Hospitals *must* request information when screening an applicant for medical staff appointment or granting new clinical privileges and every 2 years for medical staff reappointment. State licensing boards and other health care entities (health plans) *may* request information when

screening applicants for licenses or privileges. Professional societies also *may* request information when a physician applies for membership. In some cases, a plaintiff's attorney *may* request information from the NPDB if there is evidence that a hospital failed to check on the status of a staff physician involved in a malpractice case. A physician may self-query, but currently, the lay public *may not* access information from the NPDB.

37. Does the presence of family members at the resuscitation of a relative increase the risk of litigation for emergency physicians?

No data exist on family member presence and litigation. However, most families who witness a resuscitation report favorable opinions of the medical personnel involved. This is true even though the actual survival rate from resuscitation is far lower than the perception of the public. Family satisfaction after witnessing cardiopulmonary resuscitation may actually lower the risk of a malpractice lawsuit, even in the event of a poor outcome. Family members build trust in health care professionals when they are present for resuscitation in the ED. Also, health care providers are more likely to consider the privacy of a patient and pain management when family members are present. Attention to these details may also reduce the risk of malpractice lawsuits.

Leske JS, McAndrew NS, Brasel KJ: Experiences of families when present during resuscitation in the emergency department after trauma. J Trauma Nurs 2013;20(2):77-85.

Porter JE, Cooper SJ, Sellick K: Family presence during resuscitation (FPDR): Perceived benefits, barriers and enablers to implementation and practice. Int Emerg Nurs 2014;22(2):69-74.

SEDATION AND ANALGESIA

Richard J. Scarfone

CHAPTER 72

1. Why is the term *conscious sedation* considered obsolete?

In the past, conscious sedation has been defined as a medically controlled state of depressed consciousness in which:

- Protective reflexes are maintained.
- The airway remains patent.
- The patient responds purposefully to physical or verbal stimuli.

This term is now considered obsolete and has been replaced with the phrase *moderate sedation and analgesia*. However, for most clinical indications, emergency department (ED) physicians attempt to achieve a deeper level of altered consciousness for their patients, referred to as *deep sedation and analgesia*. This state is characterized by:

- Partial or complete loss of protective reflexes
- An inability to independently maintain a patent airway
- No purposeful response to stimulation

American Society of Anesthesiologists: Continuum of depth of sedation: Definition of general anesthesia and levels of sedation/analgesia. Available from www.asahq.org/publicationsAndServices/standards/20.pdf.

EMSC Grant Panel on Pharmacologic Agents Used in Pediatric Sedation and Analgesia in the Emergency Department: Clinical policy: Evidence-based approach to pharmacologic agents used in pediatric sedation and analgesia in the emergency department. Ann Emerg Med 2004;44:342-377.

2. What are some reasons for the mismanagement of pain in children?

- Lack of available data in children. The Food and Drug Administration studies and approves medications for use in adults. Physicians must extrapolate this information for pediatric patients. Until the past decade, few clinical trials assessed the safety and efficacy of sedatives or analgesics in children.
- Fear of addiction from opioids. In both adult and pediatric patients, physicians have been overly concerned about inducing addiction with the use of opioid analgesics. In fact, addiction is a rare consequence of the legitimate use of opioids for medical purposes in children.
- Belief that neonates and young children do not experience pain to the same degree as adults because of their immature nervous systems. Any physician who has attempted to intubate the trachea of an awake neonate or to perform a lumbar puncture (LP) in a struggling toddler can testify to the contrary.

Young children cannot understand the purpose of a painful procedure or comprehend its time-limited nature. Therefore, they are likely to experience a greater degree of pain and anxiety compared to older children or adults and are more likely to benefit from the liberal use of procedural sedation and analgesia (PSA).

Berde CB, Sethna NF: Analgesics for the treatment of pain in children. N Engl J Med 2004;347:1094-1101.

Zempsky WT, Cravero JP; Committee on Pediatric Emergency Medicine and Section on Anesthesiology and Pain Medicine: Relief of pain and anxiety in pediatric patients in emergency medical systems. Pediatrics 2004;114:1348-1356.

3. What are tolerance, physical dependence, and addiction?

A child with sickle cell anemia who has been treated at home with acetaminophen and codeine for 2 weeks will probably require higher than usual doses of parenteral narcotics for management of a vaso-occlusive crisis. Such a child may have developed *tolerance*, causing diminished effectiveness of the drug with repeated administration. A child with leukemia and bone pain who abruptly discontinues daily oral morphine after 10 days may experience headaches, sweating, tachycardia, and tachypnea. Such a child may have developed *physical dependence*. In contrast, *addiction* is a psychological syndrome marked by compulsive drug-seeking behavior and is associated with the desire for euphoric effects.

4. Describe methods of reducing the pain of administration of local anesthetics.

The administration of local anesthetics by injection through intact skin or into an open wound can cause considerable pain. Techniques that may reduce the pain of injection include using a needle of small caliber, buffering the lidocaine with bicarbonate, warming the drug, injecting slowly, and providing counterstimulation to the adjacent skin. Many physicians employ topical formulations, such as lidocaine, epinephrine, and tetracaine (L.E.T.), either in a liquid or a gel form to anesthetize a wound. This form may be administered, without using a needle, to exposed mucosa. Do not use these drugs in any regions of the body in which epinephrine is contraindicated, such as fingertips or ears. More recently, needle-free injection systems have been shown to compare favorably to needle injection of local anesthetics.

In addition, liposomal lidocaine (LMX) and a mixture of lidocaine and prilocaine (EMLA [eutectic mixture of local anesthetics cream]) may each be applied to intact skin to reduce the pain of venipuncture. Physicians have also found that these topical mixtures alleviate the pain associated with LP, incision of abscesses, and insertion of intravenous (IV) lines.

Eichenfield LF, Funk A, Fallon-Friedlander S, Cunningham BB: A clinical study to evaluate the efficacy of ELA-Max (4% liposomal lidocaine) as compared with eutectic mixture of local anesthetics cream for pain reduction of venipucture in children. Pediatrics 2002;109:1093-1099.

Ferayorni A, Yniguez R, Bryson M, Bulloch B: Needle-free jet injection of lidocaine for local anesthesia during lumbar puncture: A randomized controlled trial. Pediatr Emerg Care 2012;28:687-690.

Scarfone RJ, Jasani M, Gracey EJ: Pain of local anesthetics: Rate of administration and buffering. Ann Emerg Med 1998;36:36-40.

Zempsky WT: Pharmacologic approaches for reducing venous access pain in children. Pediatrics 2008;122: S140.

Zempsky WT, Cravero JP; Committee on Pediatric Emergency Medicine and Section on Anesthesiology and Pain Medicine: Relief of pain and anxiety in pediatric patients in emergency medical systems. Pediatrics 2004;114:1348-1356.

5. Describe the key elements of the medical history for a child about to receive PSA.

Before drug administration, determine the time since the child's last meal, current medications, allergies, pregnancy status, comorbid conditions, and history of complications with sedation or general anesthesia, and conduct a complete review of systems. If the child was transported from another institution, verify whether he or she received sedation or analgesia at the referring hospital and, if so, what was given and when. In addition, ask questions pertinent to the specific medication that is about to be given. For example, consider active laryngotracheobronchitis a contraindication to the use of ketamine.

6. Describe the key elements of the physical examination for a child about to receive PSA.

Clinicians must know and carefully monitor the patient's vital signs (especially blood pressure), mental status, and cardiac and pulmonary status and perfusion. Many medications employed for sedation and analgesia cause hypotension. Use these cautiously or not at all for a child with traumatic injuries if those injuries may cause hypovolemia or for a child with severe dehydration or sepsis. Ketamine, which causes a catecholamine release, would be a good choice for those with hypotension or hypovolemia. Similarly, agents that may cause hypoventilation and hypoxemia are poor choices for any child with respiratory distress.

7. How much time should elapse between the last oral intake of food or liquid and PSA?

This is an area of great controversy in the administration of PSA to children. The following are the published fasting guidelines for elective procedures developed by the American Society of Anesthesiologists (ASA):

Minimum fasting periods for the following ingested material:

- Clear liquids: 2 hours
- Breast milk: 4 hours
- Infant formula, nonhuman milk, light meal: 6 hours

8. What are the concerns about fasting guidelines for PSA?

- They are arbitrary. For example, what evidence exists to support a longer fasting time after formula intake compared to breast milk? How does the age of the patient or the volume ingested influence these recommendations? In fact, the ASA states that

"the literature does not provide sufficient evidence to test the hypothesis that preprocedural fasting results in a decreased incidence of adverse outcomes."
- They were written for fasting prior to general anesthesia.
- Physicians working in busy EDs with time and space constraints find these guidelines prohibitively conservative and difficult to adhere to.
- Aspiration following moderate or deep sedation is extremely rare.

Newer evidence suggests that prolonged fasting may not lead to fewer adverse events. In a recent report of over 1000 children receiving PSA, about half were not fasted as per the ASA guidelines. These children did not experience a greater incidence of aspiration or other adverse outcomes compared to the fasted group. A second study of over 2000 children and young adults found no correlation between preprocedural fasting time and adverse outcomes.

A more recently published clinical practice advisory recommends weighing several factors when deciding what is the appropriate fasting for elective procedures. These factors include the overall health of the patient, the desired length and depth of sedation and analgesia, and the urgency of the procedure.

Agrawal D, Manzi SF, Gupta R, Krauss B: Preprocedural fasting state and adverse events in children undergoing procedural sedation and analgesia in a pediatric emergency department. Ann Emerg Med 2003;42:636-646.

American Society of Anesthesiologists: Practice guidelines for sedation and analgesia by non-anesthesiologists. American Society of Anesthesiologists Task Force on sedation and analgesia by non-anesthesiologists. Anesthesiology 2002;96:1004-1017.

Green SM: Fasting is a consideration—not a necessity—for emergency department procedural sedation and analgesia. Ann Emerg Med 2003;42:647-650.

Green SM, Roback MG, Miner JR, et al: Fasting and emergency department procedural sedation and analgesia: A consensus-based clinical practice advisory. Ann Emerg Med 2007;49(4):454-461.

Roback MG, Bajaj L, Wathen JE, Bothner J: Preprocedural fasting and adverse events in procedural sedation and analgesia in a pediatric emergency department: Are they related? Ann Emerg Med 2004;44:454-459.

9. What is the ASA physical status classification?

To determine the health status of the patient before initiating sedation, the ASA has assigned five classes:
- **Class I:** Normally healthy patient
- **Class II:** Patient with mild systemic disease (e.g., mild asthma)
- **Class III:** Patient with severe systemic disease (e.g., poorly controlled diabetes mellitus)
- **Class IV:** Patient with severe systemic disease that is a constant threat to life
- **Class V:** Moribund patient who is unlikely to survive without the operation

10. During PSA, which equipment is needed to monitor the patient and to be immediately available at the bedside?

The risks of sedating children are significant and include hypoventilation, apnea, airway obstruction, hypotension, and aspiration. Ensure that the following equipment is immediately available at the bedside:
- Cardiorespiratory monitor
- Pulse oximeter
- Capnograph to measure exhaled carbon dioxide
- Blood pressure cuff
- Suction catheters
- Oxygen source
- Airway equipment, such as self-inflating breathing bags with masks, oropharyngeal and nasopharyngeal airways

In addition, be sure advanced airway equipment, such as laryngoscopes and endotracheal tubes, are available in the ED.

American Academy of Pediatrics and American Academy of Dentistry: Guidelines for monitoring and management of pediatric patients during and after sedation for diagnostic and therapeutic procedures: An update. Pediatrics 2006;118:2587-2602.

American Society of Anesthesiologists: Practice guidelines for sedation and analgesia by non-anesthesiologists. American Society of Anesthesiologists Task Force on sedation and analgesia by non-anesthesiologists. Anesthesiology 2002;96:1004-1017.

11. What is the role of capnography during PSA?

Capnography allows clinicians to monitor the concentration of carbon dioxide (CO_2) during the inspiratory and expiratory phases of respiration of a sedated patient, plotted over time. In particular, measurement of exhaled CO_2 allows for an indirect assessment of the partial pressure of arterial CO_2; these values closely approximate one another. Although pulse oximetry indicates oxygen saturation, it does not assess ventilation. In contrast, capnography provides a graphic display of the effectiveness of ventilation. In particular, a common practice is to administer supplemental oxygen to a deeply sedated patient. This practice can delay the detection of hypoventilation or even apnea in a patient who is not being monitored by capnography. In one study assessing PSA in children, the use of capnography allowed for an earlier detection of arterial oxygen desaturation resulting from hypoventilation.

Lightdale JR, Goldmann DA, Feldman HA, et al: Microstream capnography improves patient monitoring during moderate sedation: A randomized, controlled trial. Pediatrics 2006;117(6):e1170-e1178.

12. What is the optimal staffing for PSA?

Ideally, a physician experienced in pediatric advanced life support should administer the medications and closely observe the child's response. The risk of respiratory depression is minimized by administering agents slowly, over about 60 seconds. The physician should remain at the bedside at least until the period of peak sedation and cardiorespiratory side effects has passed (typically about 20 minutes, depending on the agent administered). A second physician should perform the procedure. In this way, a single physician is not dividing attention between two important tasks. A nurse should document the patient's response to medications and be available to assist in suctioning or administering oxygen or reversal agents.

13. How frequently should the child be monitored? How should this be documented?

Supplement the patient's medical record with a sedation and analgesia flowchart to provide a complete and accurate account of the encounter. Record medications, times administered, doses, routes, and name of the person who gave the drugs. Monitor the patient's vital signs, oxygen saturation, exhaled CO_2, and mental status continuously, with recordings documented every 5 minutes for the first 30 minutes after drug administration. Provide one-on-one nursing staff for clinical monitoring until the child is well into the recovery phase. The frequency of monitoring beyond the initial half-hour and the duration of observation depend on which agents are given and the depth of sedation.

14. Describe the characteristics of the ideal agent for sedation and analgesia.

- Painless administration
- Onset of action within minutes
- Adequate and predictable sedation, analgesia, anxiolysis, and amnesia
- Excellent safety profile
- Duration of action longer than procedure time
- Rapid recovery
- Ready reversibility

In fact, no agent has all of these desirable characteristics. Clinicians must decide what agent or agents can safely and efficiently achieve the goals for a particular clinical situation. For example, do not choose a pure sedative hypnotic for a patient needing potent analgesia.

15. What are the expected rates of success and complications for PSA in children?

Studies have consistently demonstrated that when PSA is provided to children by pediatric emergency medicine physicians in a tertiary care children's hospital, success rates are very high and serious adverse outcomes are rare. In one report, over 1200 children received PSA in such a setting, with a variety of different medications employed. For more than 98% of the children, the procedure was successfully completed while the patient remained minimally responsive. The rate of complications was 18%, with hypoxia as the most common complication. In a separate study assessing more than 30,000 pediatric sedation/anesthesia events, there were no deaths, cardiopulmonary resuscitation was required once, and oxygen desaturation was uncommon.

Cravero JP, Blike GT, Beach M, et al: Incidence and nature of adverse events during pediatric sedation/anesthesia for procedures outside the operating room: Report from the Pediatric Sedation Research Consortium. Pediatrics 2006;118(3):1087-1096.
Newman DH, Azer MM, Pitetti RD, Singh S: When is a patient safe for discharge after procedural sedation? The timing of adverse effect events in 1,367 pediatric procedural sedations. Ann Emerg Med 2003;42:627-645.
Pitetti RD, Singh S, Pierce MC: Safe and efficacious use of procedural sedation and analgesia by nonanesthesiologists in a pediatric emergency department. Arch Pediatr Adolesc Med 2003;157:1090-1096.

16. What are the advantages and disadvantages of transmucosal drug administration?

The main advantage of transmucosal (oral, intranasal, rectal), as compared to parenteral (IV, intramuscular), drug administration is its painless nature. It seems counterintuitive to cause pain with a needle puncture in an attempt to ultimately relieve pain. However, important disadvantages of transmucosal administration include delayed onset of action, less predictable results, and difficulties in titrating the dose to the desired effect. It can be frustrating to the child, parents, and doctor if the child is fully alert 40 minutes after an oral sedative has been administered. Atomizers convert liquid medications to aerosols. When employed to administer drugs intranasally, they result in enhanced tissue delivery and more rapid onset of action of drugs such as midazolam or fentanyl. However, for most ED patients, the parenteral (IV) route of administration is preferred because it allows finer minute-to-minute control of depth of sedation and analgesia.

Lane RD, Schunk JE: Atomized intranasal midazolam use for minor procedures in the pediatric emergency department. Pediatr Emerg Care 2008;24:300-303.

17. Discuss the role of propofol for PSA.

Propofol is a nonbarbiturate ultra–short-acting hypnotic agent. It has an extremely short onset (within 1 minute) of effect and duration of action, necessitating delivery by continuous IV infusion or frequent readministration for most procedures. Respiratory depression, hypoxemia, hypotension, and injection pain are common side effects. Its use requires vigilance on the part of physicians because of the potential for adverse effects. Given the depth of sedation achieved with propofol, in many cases it is not necessary to add an analgesic even for painful procedures such as joint relocations or fracture reductions. However, propofol would not be a good choice for sedating a child who will be leaving the ED, such as for a radiologic study. Even in the hands of practitioners experienced with its use, two thirds of children experienced decreased systolic blood pressure below the fifth percentile, and 12% had partial airway obstruction.

Chiaretti A, Benini F, Pierri F, et al. Safety and efficacy of propofol administered by paediatricians during procedural sedation in children. Acta Paediatr 2014;103:182.
Kost S, Roy A. Procedural sedation and analgesia in the pediatric emergency department: A review of sedative pharmacology. Clin Ped Emerg Med 2010;11:233.
Mallory MD, Baxter AL, Yanosky DJ, et al. Emergency physician administered propofol sedation: A report of 25,433 sedations from the pediatric sedation research consortium. Ann Emerg Med 2011;57:462.
Miner JR, Burton JH. Clinical practice advisory: Emergency department procedural sedation with propofol. Ann Emerg Med 2007;50:182.

18. Discuss the role of etomidate for PSA.

Etomidate is a nonbarbituate hypnotic without analgesic properties that has been used extensively as an anesthetic induction agent. It has a very rapid onset of action, short duration of action, and minimal effects on cardiovascular status. Myoclonus and injection pain are common side effects. There is increasing experience with its use for PSA in children. In a recent study, it compared favorably to pentobarbital for sedating children for computed tomography (CT) scans. Similarly, it has been shown to be superior to midazolam for fracture reductions.

Baxter AL, Mallory MD, Spandorfer PR, et al: Etomidate versus pentobarbital for computed tomography sedations: Report from the Pediatric Sedation Research Consortium. Pediatr Emerg Care 2007;23(10):690-695.
Liddo LD, D'Angelo A, Nguyen B, et al: Etomidate versus midazolam for procedural sedation in pediatric outpatients: A randomized controlled trial. Ann Emerg Med 2006;48(4):433-440.
Miner JR, Danahy M, Moch A, Biros M: Randomized clinical trial of etomidate versus propofol for procedural sedation in the emergency department. Ann Emerg Med 2007;49(1):15-22.

Key Points: Sedative Hypnotic Agents Used in Sedation

1. Midazolam
2. Propofol
3. Barbiturates
4. Etomidate

19. Can opioids be used safely in neonates?

The greatest experience with the use of opioids in neonates is with the use of morphine. Morphine may be indicated to reduce the pain associated with a strangulated inguinal hernia or from an inflicted injury, such as a fracture. Because a smaller proportion of an administered dose of morphine is protein-bound in young infants compared to older children, the proportion of drug reaching the brain is increased and the elimination half-life is prolonged. Thus, very young infants are particularly susceptible to apnea and respiratory depression with morphine. For those younger than 6 months of age, use a starting dose that is one quarter to one third the dose recommended for older infants and children. Age from birth, rather than duration of gestation, determines how premature and full-term infants metabolize narcotics. Thus, a 4-month-old infant who was born at term metabolizes narcotics at the same rate as a 4-month-old infant who was born prematurely. The clinician must judge the infant's facial expressions, heart rate, and blood pressure to determine whether to administer additional morphine for the desired effect.

Berde CB, Sethna NF: Analgesics for the treatment of pain in children. N Engl J Med 2002;347:1094-1101.

20. A bead is located in the ear canal of an 8-year-old boy. Initial attempts to remove it cause considerable anxiety and discomfort, preventing successful removal. What are some sedation options for this patient?

Because the boy does not have an inherently painful condition, and because he does not require an IV line for any other reason, a pure sedative hypnotic such as midazolam given orally may be a reasonable option. However, it will take about 30 minutes for the full effects to be realized, the depth of sedation after oral administration is not predictable, and midazolam alone does not offer him analgesia. Intranasal midazolam administered with an atomizer is likely to result in more rapid and more reliable sedation than that given orally. Another option would be nitrous oxide, an odorless gas that patients inhale. Nitrous oxide causes mild analgesia, anxiolysis, sedation, and amnesia. Its ease of delivery and rapid onset of action make it ideal for sedation before foreign body removal. However, at a ratio of 50% nitrous oxide to 50% oxygen, it is not likely to be effective for more painful procedures, such as incision and drainage of an abscess.

21. A 3-year-old girl fell down five cement steps and struck her head on the pavement. She has vomited repeatedly since the injury but is fully alert and awake in the ED. You want to perform CT of the head to rule out an intracranial injury. What are some sedation options for this patient?

Because the desired goal is to prevent the child from struggling and moving during the study, you wish to achieve sedation rather than analgesia. Pure sedative hypnotics in common use in EDs include propofol, benzodiazepines, barbiturates, and etomidate. In addition to providing sedation before imaging studies, these drugs may also be useful as adjuncts to narcotics prior to painful procedures. For this patient, midazolam or pentobarbital may be the best choices. Their safety profiles and longer durations of action offer advantages over propofol and etomidate.

Midazolam may be administered by the IV, intramuscular, oral, nasal, or rectal route. Its onset is within minutes after IV administration, and clinical effectiveness usually lasts about 30 minutes. One may expect mild reductions in blood pressure and dose-related respiratory depression. Significant hypoventilation is rare unless the drug is given concurrently with a narcotic or pushed too rapidly. Its effects may be reversed with the competitive antagonist flumazenil.

Pentobarbital is a short-acting barbiturate that produces sedation within 5 minutes, lasting 30 to 60 minutes. As with midazolam, hypoventilation may occur but responds readily to gentle stimulation. Hypotension is a common side effect; do not use the drug in children with possible cardiovascular compromise.

Boswinkel JP, Litman RS: Sedating patients for radiologic studies: Some procedures call for medication to reduce movement, anxiety, or pain. Pediatr Ann 2005;34(8):650-656.

22. An adolescent girl needs an LP to rule out aseptic meningitis. What agent(s) should be used for PSA?

IV sedation alone does not provide adequate analgesia for the painful LP. She will need a local anesthetic in the lumbar region, and administration of this will be painful. Topical anesthetics alone do not provide a great enough depth of analgesia for this indication. In response to the injection of the local anesthetic, an anxious patient may arch her back and move enough that it will be nearly impossible to perform the LP.

For this situation, some physicians have had success combining midazolam with fentanyl, which is 100 times more potent than morphine. Fentanyl's onset and duration of action closely parallel those of midazolam, making this combination particularly potent. Fentanyl provides analgesia and potentiates the sedative effect of midazolam. Respiratory depression is common with this combination but may be minimized by administering each agent over 60 seconds with a 60-second interval in between and by titrating doses carefully. Gentle stimulation of the patient and supplemental oxygen almost always prevent hypoxemia.

Fentanyl has a more rapid onset and shorter duration of action than morphine, making it a better choice for most procedural analgesia. When more prolonged analgesia is desired (e.g., for vaso-occlusive crisis), morphine is the better choice. The combination of midazolam and fentanyl is also useful in the settings of laceration repair, burn care, and reduction of minimally displaced or angulated fractures.

23. A 10-year-old boy has displaced and angulated fractures to his ulna and radius that require closed reduction. What agent(s) should be used for PSA?

Clearly, the primary goal in this case is to achieve potent analgesia for what is anticipated to be a very painful procedure. With the midazolam/fentanyl combination it will be difficult to achieve the depth of analgesia required without producing significant hypoventilation. In this case, ketamine is a good choice. It causes dissociation between the cortical and limbic systems, resulting in potent sedation, analgesia, and amnesia. Unlike most other agents, it does not cause cardiovascular depression. In fact, patients typically experience increased heart rate and blood pressure with its use. Onset of action after IV administration is 3 to 5 minutes, with return of coherence in 30 to 45 minutes. It may also be administered intramuscularly. Two uncommon but potentially serious side effects are hallucinatory emergence reactions and laryngospasm. Many clinicians believe that the incidence of emergence reactions can be minimized with the concurrent administration of midazolam, but the results of recent studies have failed to prove this. Rather than giving midazolam concurrently with ketamine, many clinicians choose to provide it for the rare patient who experiences an emergence reaction as ketamine effects wane. Recent studies have determined that the coadministration of atropine or glycopyrrolate, although resulting in less hypersalivation, does not decrease the incidence of laryngospasm. In a study assessing the ED use of intramuscular ketamine among more than 1000 children, over 90% achieved acceptable sedation within 10 minutes, and 4 young children experienced transient laryngospasm. The expected rate of laryngospasm with the use of ketamine is 1 in 250.

Green SM, Krauss B: Clinical practice guideline for emergency department ketamine dissociative sedation in children. Ann Emerg Med 2004;44:1-21.

Green SM, Rothrock SG, Lynch EL, et al: Intramuscular ketamine for pediatric sedation in the emergency department: Safety profile in 1,022 cases. Ann Emerg Med 1998;31:688-697.

Heinz P, Geelhoed GC, Wee C, Pascoe EM: Is atropine needed with ketamine sedation? A prospective, randomized, double-blind study. Emerg Med J 2006;23(3):206-209.

Migita RT, Klein EJ, Garrison MM: Sedation and analgesia for pediatric fracture reduction in the emergency department. Arch Pediatr Adolesc Med 2006;160(1):46-51.

Roback MG, Wathen JE, MacKenzie T, Bajaj L: A randomized, controlled trial of IV versus IM ketamine for sedation of pediatric patients receiving emergency department orthopedic procedures. Ann Emerg Med 2006;48:605-612.

Sherwin TS, Green SM, Khan A: Does adjunctive midazolam reduce recovery agitation after ketamine sedation for pediatric procedures? A randomized, double-blind, placebo-controlled trial. Ann Emerg Med 2000;35:239-244.
Wathen JE, Roback MG, MacKenzie T, Bothner JP: Does midazolam alter the clinical effects of intravenous ketamine sedation in children? A double-blind, randomized, controlled, emergency department trial. Ann Emerg Med 2003;36:579-588.

24. What are contraindications to the use of ketamine?

Absolute contraindications to the use of ketamine are age younger than 3 months (because of increased risk of laryngospasm) and psychosis. Other factors are considered to be relative contraindications because of the concern for laryngospasm, and physicians must weigh the risk/benefit ratio for individual patients. These include age younger than 12 months, intraoral procedures, upper airway problems (such as moderate upper respiratory tract infection, croup, tracheomalacia, or anatomic abnormalities), and active asthma. Other relative contraindications include coronary artery disease, increased intracranial pressure, and glaucoma.

Green SM, Krauss B: Clinical practice guideline for emergency department ketamine dissociative sedation: 2011 update. Ann Emerg Med 2011;57:449-461.
Green SM, Roback MG, Krauss B, et al: Predictors of airway and respiratory adverse events with ketamine sedation in the emergency department: An individual-patient data meta-analysis of 8,282 children. Ann Emerg Med 2009;54(2):158-168.
Melendez E, Bachur R: Serious adverse events during procedural sedation with ketamine. Pediatr Emerg Care 2009;25:325-328.

25. For what period of time should a child be observed in the ED after PSA before being discharged home?

A recent report of over 1300 sedation events in children found that 92% of adverse events occurred during the procedure and serious adverse events rarely occurred after 25 minutes from the final medication administration. In another study of over 1000 children who had received intramuscular ketamine, four experienced laryngospasm. Onset of this complication ranged from 15 to 25 minutes after ketamine administration.

Ideally, a child should return to his or her baseline verbal and motor skills and mental status before ED discharge. However, if the drugs are administered to a young child in the late evening or beyond, this state may not be achieved until the following morning. More practical end points are the ability to maintain normal spontaneous respirations and oxygen saturation for a period beyond the peak effect of the drugs and easy arousability. For most of the agents discussed previously, the child will be ready for discharge 45 to 90 minutes after drug administration.

Green SM, Rothrock SG, Lynch EL, et al: Intramuscular ketamine for pediatric sedation in the emergency department: Safety profile in 1,022 cases. Ann Emerg Med 1998;31:688-697.
Newman DH, Azer MM, Pitetti RD, Singh S: When is a patient safe for discharge after procedural sedation? The timing of adverse effect events in 1,367 pediatric procedural sedations. Ann Emerg Med 2003;42:627-645.

26. What are the top 10 pitfalls in administering sedation and analgesia to children in the ED?

1. Undersedation
2. Oversedation
3. Using reversal agents to speed recovery
4. Choosing a short-acting narcotic when prolonged pain relief is required
5. Combining two opioids or two sedative agents
6. Choosing a sedative when analgesia is required or vice versa
7. Choosing an improper route of administration
8. Failure to document appropriately
9. Failure to have proper equipment immediately available
10. Not including parents in the discussion about the need for sedation and analgesia

Key Points: Requirements for Safe Use of Procedural Sedation and Analgesia

1. Focused patient history
2. Careful physical examination
3. Available emergency equipment
4. Knowledge of sedative agents to be used
5. Appropriate staff
6. Careful monitoring

27. What are the recommended starting doses for sedatives and analgesics commonly used in children?

See Table 72-1 for recommended starting doses.

Table 72-1. Recommended Starting Dose

DRUG	ROUTE	DOSE (MG/KG)
Midazolam	IV	0.1
Midazolam	IM	0.2
Midazolam	IN	0.4
Midazolam	PO, PR	0.5
Pentobarbital	IM	4
Pentobarbital	IV	2
Propofol	IV	1-3 load, 25-100 μg/kg/min
Etomidate	IV	0.1-0.2
Ketamine	IV	1-2
Ketamine	IM	4
Morphine	IV	0.1 (lower in neonates)
Fentanyl	IV	1 μg

IM, intramuscular; IN, intranasal; IV, intravenous; PO, oral; PR, rectal.
*All drugs should be titrated to desired effect.

CHAPTER 73

TRANSPORT MEDICINE

George A. Woodward

1. List the goals of interfacility transport.

Goals of interfacility transport are as follows:

- To meet the unique needs of ill and injured infants and children
- To meet the needs of the medical community
- To allow for regionalization
- To provide high-quality care
- To deliver the patient to the receiving center in stable or improved condition

Woodward GA, Garrett AL, King BR, Baker MD: Prehospital care and transport medicine. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 85-124.

Woodward GA, Insoft RM, Pearson-Shaver AL, et al: The state of pediatric interfacility transport: Consensus of the second national pediatric and neonatal interfacility transport medicine leadership conference. Pediatr Emerg Care 2002;18:38-43.

Woodward GA, Kirsch R, Trautman MS, et al: Stabilization and transport of the high-risk neonate. In Gleason CA, Devaskar S (eds): Avery's Diseases of the Newborn, 9th ed. Philadelphia, Elsevier Saunders, 2011, pp 341-356.

2. What issues should be considered at the time of transport?

- Current level of care
- Stability of the patient
- Options available to provider and patient
- Type of care that the patient requires and urgency of need for advanced medical care

Stroud MH, Prodhan P, Moss MM, Anand KJS: Redefining the golden hour in pediatric transport. Pediatr Crit Care Med 2008;9(4):435-437.

3. List the advantages and disadvantages of the seven tiers of ground transport.

The advantages and disadvantages of the seven tiers of ground transport are listed in Table 73-1.

4. True or false: When choosing an ambulance service or interfacility transport provider, the receiving hospital is legally responsible for ensuring the adequacy of transport service.

False. Under Emergency Medical Treatment and Active Labor Act (EMTALA) regulations, the *referring hospital* and clinicians are responsible for ensuring that the quality of care during transport does not diminish and that an unstable patient is not placed in a less sophisticated environment. The transporting system is responsible for ensuring that the medical care it delivers meets the defined standard of care.

Woodward GA: Legal issues in pediatric interfacility transport. Clin Pediatr Emerg Med 2003;4:256-264.

5. What are the advantages and disadvantages of ambulance transport?

Advantages include the ambulance leaving from a referring facility and traveling directly to the receiving facility. It has the ability to stop or redirect if problems arise during transport. Team composition changes and personnel additions are easy and safe. Ambulance transport is relatively inexpensive, family member(s) can accompany the patient, backup vehicles are often available, and there are few weather restrictions. Disadvantages include noise, vibration, motion sickness, road conditions, traffic, detours, delays, and accident risk.

Hankins D: Air versus ground transport studies. Air Med J 2010;29(3):102-103.

6. Describe the advantages and disadvantages of transport by helicopter.

Helicopters offer the advantage of rapid transport (often one third to one half of ground transport time to the same location). They have the ability to access difficult locations and avoid traffic. A major disadvantage is the need for a local landing zone or helipad, without

Table 73-1. Seven Methods of Ground Transport: Advantages and Disadvantages

METHOD	ADVANTAGES	DISADVANTAGES
Private vehicle	Immediate transport	Potential for no direct transport, no medical care en route
Taxi	Direct transport to hospital	No medical care
Volunteer ambulance	Direct transport to hospital	Minimal to no medical care
Basic life support (BLS) ambulance	Direct transport to hospital	Limited medical care, limited pediatric experience and expertise
Advanced life support (ALS) ambulance	Direct transport to hospital, emergency resuscitative care, some interventions available	Variable pediatric experience, limited diagnostic and interventional capabilities
Critical care ambulance	Direct transport to hospital, sophisticated medical care	Possibly limited or no pediatric expertise
Pediatric speciality interfacility transport	Direct transport to hospital, pediatric expertise	Limited resource, may not be immediately available

which the speed advantage may be diminished. Size and space limitations may limit patient assessment or intervention. Altitude physiology, including pressure changes and hypoxia, is important but may not be a major issue for low-altitude helicopter transport. Other flight issues include stress on equipment, humidity, fatigue, gravitational forces, weight restriction, and emergency survival, as well as increased noise and vibration. Helicopter travel may be limited by weather; many systems are regulated by visual flight rules rather than instrument flight rules (the capability for instrument flight rules enables safe flight in weather with diminished visibility). Helicopter transport is expensive.

Federal Aviation Administration (FAA) Helicopter Safety Initiative: An ongoing, proactive, approach to helicopter safety, 2004-2011. Available at www.nemspa.org.

7. What are the advantages and disadvantages of transport with fixed-wing aircraft?

Fixed-wing (jet, airplane) transport offers the advantage of speed and is appropriate for transports over 100 to 150 miles. An airplane often can fly over or circumvent bad weather. The cabin can be pressurized to diminish problems with altitude physiology and often offers more room than a helicopter for patient assessment and intervention. A disadvantage is that the airport location may not be convenient to the hospital. Required additional ground or helicopter transport increases the transport time and complexity as well as the risk to the patient.

8. Can a minor be transported without parental consent?

If a parent or legal guardian is not available for consent, the treating physician may decide and document that the benefits of transport exceed the risks of waiting for parents and may provide emergency consent. Consent would be provided for the treatment and transport for that emergent issue only. Seek and obtain consent from the legal guardians as soon as possible.

Woodward GA: Legal issues in pediatric interfacility transport. Clin Pediatr Emerg Med 2003;4:256-264.

9. Is it necessary for children to consent to transport?

The parent/legal guardian is responsible for consent for treatment. Ideally, assent (agreement) regarding the process is also obtained from the child, but it is not mandatory unless the child is emancipated. Rules for emancipation and age of majority vary by state. Review these prior to transport.

10. What are the key elements of a pediatric transport system?

Adequate preparation for transport involves designing and developing a transport process before it is needed. Educating all levels of providers, appropriate patient care during transport, and reviewing and addressing systems and quality issues during and after transport are imperative. Other key elements include medical supervision and involvement during transport, experienced on-site personnel, cooperation between referring and receiving hospitals and personnel, and adequate quality assurance and improvement.

Woodward GA (AAP, Editor in Chief): Guidelines for Air and Ground Transport of Neonatal and Pediatric Patients, 3rd ed. Elk Grove Village, IL, American Academy of Pediatrics, 2006.

11. In what areas is medical oversight important?

Qualified and experienced medical oversight may be the most important element of the transport system. Knowledgeable physicians must be involved at all levels:

- **Organizational physicians** help design the transport system and evaluate operations.
- **Referring physicians** should be experts in recognition of illness/injury, provision of initial care, and stabilization. They should recognize limitations in care opportunities (personnel, logistic, current or projected) and request assistance when needed. They should know how and when to refer patients to an appropriate transport service.
- **Medical control ("command" or "supervising") physicians** should be immediately available to the transport system for questions or issues that arise during transport.
- **Receiving physicians** must be aware of the patient's arrival and capable of streamlining the transition to care at the receiving hospital.

12. What degree or level of education should pediatric transport personnel possess?

No singular optimal degree or level has been established. The most important qualifications are experience in pediatric critical care or neonatal care and an appropriate skill level, as determined by routine competency assessments. Personnel must have specific training and experience in the care of critically ill children. They may include physicians, registered nurses, nurse practitioners, paramedics, and respiratory therapists. All who are involved must be familiar with the transport environment and capable of recognizing and stabilizing any problems that arise in that transport population.

13. What types of skills should be considered imperative for transport personnel?

The level of skill required for transport personnel depends on the patient population. For a pediatric/neonatal critical care transport service, the personnel must be skilled in managing all routine and emergent pediatric and neonatal issues. Examples include airway stabilization and management with bag/mask ventilation, intubation, rescue airways, surgical airway placement, and ventilatory support. Ability to understand and use critical adjuncts, such as rapid-sequence intubation; expertise in establishing vascular access, including intravenous (IV) and intraosseous routes; understanding of fluid resuscitation; and ability to use pharmacologic adjuncts for cardiovascular support are imperative. Knowledge of infection control and ability to assess patients adequately and effectively at a referral center are also important.

King BR, Foster RL, Woodward GA, McCans K: Procedures performed by pediatric transport nurses: How "advanced" is the practice? Pediatr Emerg Care 2001;17:410-413.

King BR, Woodward GA: Procedural training for pediatric and neonatal transport nurses. Part I: Training methods and airway training. Pediatr Emerg Care 2001;17:461-464.

King BR, Woodward GA: Procedural training for pediatric and neonatal transport nurses. Part II: Procedures, skills assessment and retention. Pediatr Emerg Care 2002;18:438-441.

14. What factor most increases the risk for transported patients?

Whenever the patient is moved from one location to another (e.g., bed to stretcher, incubator), the opportunity for line or tube displacement increases. Movement can cause appropriately immobilized patients to become malpositioned, perhaps decreasing desired protection, impeding respiratory efforts, or even worsening an existing injury. Multiple modes

of transport increase movement needs and potentially increase risk to the patient. If a patient deteriorates during or shortly after movement, consider sequelae of the move as a potential cause.

15. What equipment is important in a critical care ambulance environment?

The answer depends on the patient population. Minimal required equipment includes appropriate pediatric interventional supplies, medication, monitors, and communication equipment (radios, phones and other modalities). Monitoring capability should include pulse oximetry, respiratory rate, blood pressure, temperature, end-tidal carbon dioxide, and electrocardiography. Strive for point-of-care laboratory testing, which allows rapid analysis of blood gases, chemistries, hemoglobin, and glucose.

American Academy of Pediatrics, American College of Emergency Physicians, American College of Surgeons Committee on Trauma, et al. Joint Policy Statement: Equipment for ambulances. Prehospital Emergency Care 2014;18:92-97.

Stroud MH, Prodhan P, Moss M, et al: Enhanced monitoring improves pediatric transport outcomes: A randomized controlled trial. Pediatrics 2011;127:42-48.

Woodward GA, King BR, Garrett AL, Baker MD: Prehospital care and transport medicine. In Fleisher GR, Ludwig S (eds): Textbook of Pediatric Emergency Medicine, 6th ed. Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 85-124.

16. How can referral for transport be streamlined?

Development of a designated and advertised access number or specialized communication center with a centralized number is important. The ability to triage incoming calls efficiently to appropriate command physicians and transport personnel is vital. Simultaneous logistic planning is more easily accomplished with the use of a dedicated communication center than with direct referral to a receiving physician, when operational communications and arrangements will happen in sequence rather than in parallel. Transfer agreements and up-to-date knowledge of bed availability can markedly decrease time necessary for patient acceptance.

Key Points: Methods of Increasing Efficiency of Transport

1. Centralized phone number
2. Central communication center
3. Transport-literate personnel capable of triaging incoming calls
4. Predetermined transfer agreements
5. Current awareness of bed availability

17. What information should be gathered and documented at the time of patient referral?

- Referring physician's name, location (hospital and unit), and phone number
- Patient data, including name, age, weight, acute medical history, working diagnosis, pertinent past medical history, current vital signs, clinical parameters, and laboratory and radiographic results
- Brief summary of interventions performed and patient's response
- Any confounders or projected issues with expected care
- Verification of consent to transfer

18. What are the responsibilities of the referring physician during the transport process?

The referring physician must assess and stabilize the patient to the best of his or her ability. He or she must ensure that the chosen transport service has the appropriate skills and equipment to avoid a decrease in the level of care. Discuss plans for stabilization and intervention with the receiving hospital or transport service, and, if at all possible, follow these recommendations. Discuss disagreements about patient stability or inability to perform a task for other reasons with the command physician. If the patient's condition changes, notify the transport service. The family must consent to the particular type of transport (air, ground) and to the receiving hospital. Document the consent in writing. Place a written order for transport services. The referring physician and care team should be available for discussion in person or by telephone when the transport service arrives.

In addition, the referring physician and team must ensure the following:

- IV access has been secured.
- All tubes and lines are taped securely to help avoid dislodgement during transport.
- A copy of medical records, radiographs (hard copy, disk, or electronic transmission), and laboratory results accompanies the patient or is sent as soon as possible after the acute transfer.
- A medical summary is completed and included with transfer materials.
- Blood products or medications are ordered and available, if their use is considered for the transport.

Bolte RG: Responsibilities of the referring physician and referring hospital. In McCloskey K, Orr R (eds): Pediatric Transport Medicine. St. Louis, Mosby, 1995, pp 33-40.

Woodward GA (AAP, Editor in Chief): Guidelines for Air and Ground Transport of Neonatal and Pediatric Patients, 3rd ed. Elk Grove Village, IL, American Academy of Pediatrics, 2006.

19. Can a general rather than a pediatric transport service adequately transport children?

The optimal decisions on mode and staffing for pediatric transport depend on patient acuity and resource availability. If a pediatric critical transport service is available, it potentially offers the highest level of care for pediatric patients. If a pediatric transport service is not available, a general transport service may adequately transport the child. What one gains in availability and efficiency of response, however, may be affected by limited abilities to assess and appreciate progression of disease or clinical deterioration and to intervene for the pediatric patient. General services may not transport a significant number of critically ill pediatric patients; therefore, their pediatric expertise may be diminished by lack of experience. Referring and receiving physicians should take the opportunity to increase the pediatric expertise of general transport services to ensure adequate care.

20. How can a transport service develop pediatric expertise?

Many options are available. One is to visit and emulate the best practices of other services. The American Academy of Pediatrics section on Transport Medicine is a great resource for collegial pediatric transport assistance and expertise, publishes the *Guidelines for Air and Ground Transport of Neonatal and Pediatric Patients*, and provides a course in pediatric education for prehospital professionals. Introduction to pediatric advanced life support (ALS) is provided by the American Heart Association. The Committee on Accreditation of Medical Transport Systems offers standards and an accreditation process for transport systems.

Woodward GA (AAP, Editor in Chief): Guidelines for Air and Ground Transport of Neonatal and Pediatric Patients, 3rd ed. Elk Grove Village, IL, American Academy of Pediatrics, 2006.

21. How can care during transport be reviewed and critiqued?

A review of patient care with the care providers and involved facilities is important. Accomplish this review in a constructive and nonaccusatory fashion, at a time when all involved may be most receptive to review. The goal is to professionally review delivered care and identify plans (communication, outreach, etc.) to ensure optimal care for the next patient.

22. What time factors are involved in patient transport?

- Time from presentation of illness or injury to initial care
- Initial assessment, stabilization, and management
- Decision to transport and contact with the transport service
- Acceptance of patient by the receiving center or transport service
- Arrival of the transport team
- Further pretransport stabilization at the scene or referring hospital
- Arrival at the receiving hospital
- Transition to in-hospital services

23. What major issue should be considered in determining the specifics of a critical care transport?

Determine whether the immediate goal is to transport the patient to an intensive care setting or to bring intensive care capabilities to the patient. The answer may help to determine the type of transport and level of sophistication necessary for the transport personnel and service.

24. How does EMTALA affect the transport of pediatric patients?

EMTALA places clear duties on both referring and receiving hospitals. The referring clinicians must do everything possible to stabilize the patient's medical condition before transport unless the facility cannot provide an adequate level of care. Patient or parent consent for transport must be obtained. EMTALA requires receiving hospitals to accept a patient for transport if space and appropriate level of care are, or can be, available. The patient's ability to pay should not be considered by the referring or receiving hospital. When considering critical care transport for an acute medical or surgical need, do not discuss financial ramifications of the decision to transport with the patient or family.

Woodward GA: Legal issues in pediatric interfacility transport. Clin Pediatr Emerg Med 2003;4:256-264.

25. What are two major concerns about altitude physiology?

- According to **Boyle's law**, an increase in altitude brings a decrease in barometric pressure. Decreased barometric pressure (P) results in an increase in volume (V) of gas (Boyle's law: $P_1V_1 = P_2V_2$). Boyle's law has ramifications for air in enclosed spaces, such as ear canals, endotracheal tube and blood pressure cuffs, intestinal gas, pneumothorax, and pneumocranium. There is approximately a 20% increase in gas volume between sea level and 5000 feet and a 100% increase between sea level and 18,000 feet.
- **Dalton's law**, or the law of partial pressure ($P_{total} = P_1 + P_2 + P_3 \ldots$), states that the total pressure of a gas is a sum of its components. Although the concentration of oxygen in air is always 21%, air is less dense at higher altitudes; therefore, an increase in altitude results in a decrease in ambient oxygen available to the patient.

Wilson GD, Sittig SE, Schears GJ: The laryngeal mask at altitude. J Emerg Med 2008;34:171-174.

Woodward GA, Vernon DD: Aviation physiology in pediatric transport. In Jaimovich DG, Vidyasagar D (eds): Handbook of Pediatric and Neonatal Transport Medicine, 2nd ed. Philadelphia, Hanley & Belfus, 2002, pp 43-54.

26. How serious is the concern about problems related to altitude?

Most patients do not have problems related to air physiology at the relatively low altitudes at which helicopters usually fly or with pressurized fixed-wing transport. However, if the patient has an unrecognized air collection, decompression sickness, or diving illness, or if the air transport involves significant altitude changes, associated problems may develop. The decrease in ambient oxygen is usually not a factor because of the availability of supplemental oxygen and positive-pressure support. For patients already receiving maximal oxygen and pressure support at sea level, however, an increase in altitude may be of significant concern.

Woodward GA, Vernon DD: Aviation physiology in pediatric transport. In Jaimovich DG, Vidyasagar D (eds): Handbook of Pediatric and Neonatal Transport Medicine, 2nd ed. Philadelphia, Hanley & Belfus, 2002, pp 43-54.

27. What is on-line medical command?

With on-line medical command, an off-site physician or other advanced provider offers medical advice to the on-site team in real time during the transport. Off-line medical support involves the development of protocols or guidelines, without interaction at the immediate time of transport.

28. What is the value of exposing pediatric residents to interfacility transport?

The transport environment provides an opportunity for senior pediatric residents to apply skills developed during the first part of a residency program. Although the patient population and disease processes are similar to those in the hospital, the environment in which care is given is markedly different. Significant preparation is required for optimal participation in the transport process, including development of skills for communicating with transport team personnel and receiving and referring physicians and families. Residents will also learn about transport vehicles (ambulance and aircraft) and equipment (medications, monitors, interventional equipment). Residents on transport will also learn to anticipate and respond to issues that do not occur within the hospital, including delays in transport, traffic, mechanical factors, and differences in skill levels and personalities at referring and receiving hospitals. These are often enlightening and rewarding growth experiences.

Giardino AP, Tran XG, King J, et al: A longitudinal view of resident education in pediatric emergency interhospital transport. Pediatr Emerg Care 2010;26(9):653-658.

29. What training and capabilities are required of a resident physician participing in transports?

Any resident involved in the transport of critically ill children should have the experience of critical care rotations, including pediatric intensive care, emergency and neonatal care, and general pediatric experience. Capabilities for airway, breathing, and circulatory intervention and ability to access more sophisticated knowledge and expertise are imperative. Transport-specific training is also essential for all who care for patients during the process. The use of simulation in the training environment is an extremely valuable adjunct for the logistic and cognitive aspects of care.

LeFlore JL, Anderson M: Effectiveness of 2 methods to teach and evaluate new content to neonatal transport personnel using high-fidelity simulation. J Perinat Neonatal Nurs 2008;22:319-328.

Key Points: Critical Care Transport

1. It is an integral component of the care continuum for ill and injured pediatric and neonatal patients.
2. It requires specific equipment and training and expertise with pediatric critical care skills.
3. It demands understanding of transport personnel and modes of transport for all who participate in transport, whether as a referring, receiving or on-site care provider.
4. It offers significant educational opportunities for medical trainees.

30. Should a parent accompany their child on transport?

Whenever possible, allow parents to be part of the transport process. Their presence can be invaluable for information and for patient comfort. It is also important to maintain the family support unit as much as possible during these transfers. If, however, parental involvement puts the patient at risk (weight or space limitations of the vehicle; a parent who is abusive, inebriated, or belligerent; or a parent who may distract medical attention from the patient during the transport), reconsider their accompaniment.

Woodward GA, Fleegler EW: Should parents accompany pediatric interfacility ground ambulance transports? The parents' perspective. Pediatr Emerg Care 2000;16:383-390.

Woodward GA, Fleegler EW: Should parents accompany pediatric interfacility ground ambulance transports? Results of a national survey of pediatric transport team managers. Pediatr Emerg Care 2001;17:22-27.

31. Identify potential care personnel participants for pediatric transport.

Transport personnel can include physicians, nurses, nurse practitioners, respiratory therapists, and emergency medical services personnel who hold variable titles, including volunteer, first responder, emergency medical technician–basic (EMT/BLS), intermediate (EMT-I), and paramedic (EMT-P/ALS), and critical care emergency medical transport paramedic (CCEMTP).

32. True or false: Seat belts and restraint devices should be used during transport.

True. Safety of the transport patient and personnel is paramount. *Unless an acute patient change dictates that personnel are temporarily mobile*, personnel must be restrained at all times when in a moving transport vehicle. In addition, secure any objects that might move during a rapid acceleration or deceleration to help ensure a safe environment. Move any objects that are in an area where one's head might strike them during a motor vehicle accident.

King BR, Woodward GA: Pediatric critical care transport—The safety of the journey: A five-year review of vehicular collisions involving pediatric and neonatal transport teams. PreHosp Emerg Care 2002;6:449-454.

National Highway Traffic Safety Administration: Working Group Best Practice Recommendations for the Safe Transportation of Children in Emergency Ground Ambulances. NHTSA, U.S. Department of Transportation, September 2012. Available at www.nhtsa.gov.

Woodward GA: Legal issues in pediatric interfacility transport. Clin Pediatr Emerg Med 2003;4:256-264.

33. List strategies for follow-up to a stressful transport or patient care experience.

Critical incident stress management helps personnel to understand and cope with stressful events. These preparations and interventions include understanding stressful issues in the transport environment and education regarding coping skills prior to involvement, as well as defusing (discussing) and debriefing (formal incident review) processes after an incident has occurred.

Woodward GA (AAP, Editor in Chief): Guidelines for Air and Ground Transport of Neonatal and Pediatric Patients, 3rd ed. Elk Grove Village, IL, American Academy of Pediatrics, 2006.

34. Where can one find a guideline for recommended ambulance equipment?

A multidisciplinary effort, published in 2009 and revised in 2014, provides an updated list of recommended equipment for basic life support (BLS), ALS, and interfacility ambulances. Use these recommendations as a starting point for expected ambulance equipment and augment with state-required as well as specialty-appropriate materials. Updates to these recommendations will be available every few years.

American Academy of Pediatrics, American College of Emergency Physicians, American College of Surgeons Committee on Trauma, et al. Joint Policy Statement: Equipment for ambulances. Prehospital Emergency Care. 2014;18:92-97.

American College of Surgeons, Committee on Trauma, American College of Emergency Physicians: Equipment for ambulances. Bull Am Coll Surg 2009;94:23-29.

35. Can an ambulance circumvent traffic laws while transporting emergency ill patients?

In general, ambulances should adhere to standard traffic laws. Lights and sirens are ideally used to alert those in other vehicles and to help ensure adequate space for efficient and safe ambulance transport. Do not assume that the use of lights and sirens allows for disregard of speed limits or need to stop at lights and intersections. Many of the injuries and fatalities noted with ground transport are related to disregard for standard safety traffic expectations and are preventable with appropriate adherence to expected traffic norms.

Becker LR: Ambulance crashes: Protect yourself and your patients. J Emerg Med Serv 2003;28:24-26.

Becker LR, Zaloshnja E, Levick N, et al: Relative risk of injury and death in ambulances and other emergency vehicles. Accid Anal Prev 2003;35:941-948.

National Highway Traffic Safety Administration: Solutions to Safely Transporting Children in Emergency Vehicles. NHTSA, U.S. Department of Transportation, 2009. Available at www.nhtsa.gov.

National Highway Traffic Safety Administration: Working Group Best Practice Recommendations for the Safe Transportation of Children in Emergency Ground Ambulances. NHTSA, U.S. Department of Transportation, September 2012. Available at www.nhtsa.gov.

36. Do specialty pediatric transport teams impact outcome?

Yes. Compelling evidence suggests that the use of rapid initiation of goal-directed therapy and use of specialty transport teams that can continue therapy en route provide safer care with less unplanned care events and better outcomes for critically ill children. Although specialty teams are not always an option owing to their availability, always consider use of these teams for a critically ill infant.

Belway D, Henderson W, Keenan SP, et al: Do specialist transport personnel improve hospital outcome in critically ill patients transferred to higher centers? A systematic review. J Crit Care 2006;21:8-17; discussion 17-18.

Lim MT, Ratnavel N: A prospective review of adverse events during interhospital transfers of neonates by a dedicated neonatal transfer service. Pediatr Crit Care Med 2008;9:289-293.

Orr RA, Felmet KA, Han Y, et al: Pediatric specialized transport teams are associated with improved outcomes. Pediatrics 2009;124:40-48.

37. How can one evaluate the effectiveness of their transport system?

Surveying constituents and stakeholders is a good place to start and can occur after every transport or in a more general approach. Evaluate responsiveness, efficiency, medical care delivery, and outcomes. Having an outside reviewer review the system and providers can also be invaluable. Obtaining certification through an accreditation agency such as CAMTS (Commission on Accreditation of Medical Transport Systems) can help ensure that the team is aware of and functions to expected transport safety standards.

Frazer E: What is a medical transport program? Air Med J 2010;29(3):96.

McPherson ML, Jefferson LS, Graf JM: A validated pediatric transport survey: How is your team performing? Air Med J 2008;27:40-45.

38. True or false: Care during transport can be improved mainly by improving the transport team's knowledge and skills.

False. Although it is true that the transport team should be as skilled and up to date as possible, even bigger gains might be available from ensuring appropriate recognition of illness and treatment at the referring hospital. The transport team may develop insights into referral provider capabilities and opportunities and can use that information to provide appropriate

outreach training and support prior to acute need. If the referral teams have better knowledge, equipment, and skills, the transport team can validate that the initial stabilizing care is appropriate (rather than the referring team waiting for the transport system to provide initial stabilization and/or advanced care).

Cichon ME, Fuchs S, Lyons E, Leonard D: A statewide model program to improve emergency department readiness for pediatric care. Ann Emerg Med 2009;54(2):198-204.

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