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EMERGENCY MEDICINE SECRETS

FIFTH EDITION

QUESTIONS YOU WILL BE ASKED
TOP 100 SECRETS ■ KEY POINTS ■ WEB SITES

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DEDICATION

To my wife, Leslie, and daughters, Nicole, Tasha, and Nadia—the four greatest ladies in my world. I wish to thank them for their lifelong support of all my endeavors and, in particular, for understanding the time that the editing of this manuscript has taken away from my time with them. I would also like to acknowledge all the medical students, residents, and attending staff physicians with whom I have had the pleasure of working at the Denver Health Emergency Department over the past 33 years. Their enthusiasm and intellectual curiosity have stimulated many of the questions in this book.

VJM

To my wife, Kathy, whose love, support, and remarkable patience make every day worthwhile.

PTP

To Vince, for being a true mentor. To my parents, Phil and Ursula Awad, for believing in me more than I ever have myself. To my husband and eternal soulmate, Peter, for allowing me the freedom and support to follow my dreams. And to our children, Sam, Jessie, and Avery, because coming home to you is my favorite part of the day—I love you…plus one.

KMB

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PREFACE

This book is designed to be read by all students and practitioners of emergency medicine, both novice and experienced. As emergency medicine continues to evolve as a specialty, we have added several new chapters to our fifth edition and enhanced the format and referencing of questions. With difficulty, we have also selected the 100 Top Secrets from more than 220 submitted by authors. We hope this continues to be a valuable and enjoyable method of providing information and knowledge. Knowing some of the important questions about a particular presentation or problem is the first step to obtaining the answers needed at the patient's bedside. However, medicine is nothing if not humbling, and knowledge alone does not treat all that ails. Listen to your patients and make them feel heard. Getting the right diagnosis can be invigorating, but impacting a life confirms our calling.

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TOP 100 SECRETS

These secrets are 100 of the top board alerts. They summarize the concepts, principles, and most salient details of emergency medicine.

1. **When formulating the differential diagnosis, ask "What is the most serious possible cause of this patient's presenting signs and symptoms?"**

2. **When uncertain of the diagnosis, communicate this truthfully to the patient and indicate it in the final ED diagnosis.**

3. **Before discharging a patient from the ED, ask, "Why did the patient come, and have I made the patient feel better?"**

4. **Familiarity with the indications and limitations of rescue airway devices is essential.**

5. **Preoxygenation is a critical component of rapid sequence intubation because it will prevent significant hypoxia despite several minutes of apnea during the intubation process.**

6. **When evaluating results of a research paper, the smaller the number needed to treat, the more effective the intervention or treatment.**

7. **When in doubt, remember that a *p* value less than 0.05 is generally considered significant, the difference found by chance alone being 1 in 20.**

8. **Consider HIV/AIDS in patients at risk who present with an illness or infection, particularly those with opportunistic infections or extreme presentations of common diseases.**

9. **In febrile patients, a white blood cell or band count is rarely useful in differentiating between bacterial and viral illnesses.**

10. **A foreign body in the airway should be suspected in a child with sudden onset of respiratory symptoms and lack of response to appropriate treatment.**

11. **The diagnosis of gastroenteritis cannot be made without the presence of *both* vomiting and diarrhea.**

12. **Spinal epidural abscess should be suspected as the cause of back pain in immunocompromised patients and IV drug users who present with localized spinal tenderness and fever.**

13. **An afferent pupillary defect points to a defect of the retina or optic nerve.**

14. **Perilimbic flush suggests iritis or glaucoma, not conjunctivitis.**

15. **When a mandible fracture is suspected, a panoramic radiograph of the mandible is the least expensive and most accurate film to assess the patient.**

16. Documenting adherence to evidence-based guidelines is helpful in defending against a malpractice claim.

17. In patients with a high suspicion for bacterial meningitis, administer antibiotics promptly before the lumber puncture is performed and after blood cultures are obtained.

18. Consider Kawasaki's disease in children presenting with 5 days of fever.

19. The initial objectives in treating an asthma or chronic obstructive pulmonary disease (COPD) exacerbation are to relieve significant hypoxemia (oxygen), reverse airflow obstruction (β-agonists + ipratropium), and to reduce of the likelihood of recurrence (corticosteroids).

20. There is no increased risk for a serious bacterial illness in a child with a simple febrile seizure.

21. Continuous positive airway pressure (CPAP) or bi-level positive airway pressure (BiPAP) may well reduce the need for endotracheal intubation in both the ED and prehospital arenas.

22. The most important tool in assessing patients in whom you suspect ischemic heart disease is the history. The second most important tool is the history. The third most important tool is, well, you get the picture.

23. Serum lactate is a useful marker to assess the extent of systemic hypoperfusion and response to resuscitation.

24. It is not always necessary to identify a dysrhythmia prior to treating it. Assume all wide complex tachycardia with hemodynamic instability is ventricular tachycardia (VT).

25. An external pacemaker can be used if a permanent pacemaker malfunctions.

26. The diagnosis of atrial fibrillation (AF) can be made clinically by palpating a peripheral pulse and simultaneously auscultating the heart or visualizing the cardiac rhythm; AF is the only dysrhythmia that results in a pulse deficit (fewer beats palpated than observed or auscultated).

27. Every ED should have an interdisciplinary evidence-based guideline for the management of acute stroke.

28. Do not acutely lower the mean arterial pressure (MAP) more than 20% to 25% in patients with hypertensive encephalopathy.

29. It is not necessary to gradually empty the bladder when treating an episode of acute urinary retention.

30. The indications for emergency dialysis are acute pulmonary edema, life-threatening hyperkalemia, and life-threatening intoxication or overdose by agents normally excreted by the kidneys.

31. When a patient with end-stage renal disease presents with shortness of breath, volume overload is by far the most common cause, even when physical examination and chest X-ray are not diagnostic.

32. In a young woman presenting with rash, fever, and diarrhea, consider toxic shock syndrome and examine for a retained tampon.

33. Doxycycline is the drug of choice for most severe tick-borne infections, and it should be used empirically and early in the febrile, severely ill patient with a possible tick exposure.

34. A febrile patient returning from the tropics has malaria until proved otherwise.

35. Consider syncope as a cause of fall in a geriatric patient.

36. Patients with myocardial infarction may get symptomatic relief from antacids, and patients with esophageal disease may get symptomatic relief from nitroglycerin. Antacids and nitroglycerin are therapeutic medications, not diagnostic tests.

37. Any complaint of abdominal pain in an elderly patient should be taken seriously even if they have "normal" vital signs and no guarding or rebound tenderness on abdominal examination.

38. A neutropenic fever is a single temperature greater than 38.3°C and an absolute neutrophil count less than 1,000/mm^2.

39. Intussusception occurs between 3 months and 3 years of age.

40. Bilateral retinal hemorrhages in an infant strongly suggest child abuse (shaken baby syndrome).

41. Ketamine provides sedation, analgesia, and amnesia while protecting the cardiovascular status and airway reflexes, making it an ideal agent for procedural sedation in children.

42. Because of the fast helical computed tomography (CT) scanners of today, many infants and children can undergo this diagnostic procedure without sedation.

43. Mesenteric ischemia should be considered in any patient who has severe abdominal pain out of proportion to the physical examination, often requiring large doses of narcotics.

44. Dermal exposure or ingestion of hydrofluoric acid can result in profound hypocalcemia, hypomagnesemia, and hyperkalemia.

45. The most important action to take in the event of an attack of weapons of mass destruction is simply self-protection by donning appropriate personal protective equipment.

46. Sodium bicarbonate (1–2 mEq/kg) should be considered for all poisoned patients with ventricular dysrhythmias or a wide QRS.

47. Secure the airway early in the management of patients with significant soft-tissue neck injuries.

48. Consider a computed tomography angiography (CTA) of the neck in patients with facial or cervical spine fractures.

49. Hypotensive, tachycardic patients with penetrating chest trauma should be immediately evaluated for tension pneumothorax and pericardial tamponade because emergent treatment can be life-saving.

50. A suicide attempt should be considered in patients with illogical explanations for serious accidents.

51. Analyzing a mass gathering will allow informed decisions about the needed levels of staffing and equipment necessary to provide on-site care.

52. A CT scan for appendicitis is "negative" only if the entire appendix has been visualized and is normal.

53. Abdominal aortic aneurysm (AAA) can mimic renal colic.

54. Helical CT is the radiologic modality of choice for diagnosing ureteral calculus.

55. Consider testicular torsion in any male with lower abdominal pain.

56. Early goal-directed therapy in patients with severe sepsis reduces short-term mortality by 10% to 20% compared with an unstructured treatment regimen.

57. Culture and antibiotics are not indicated in non-immunocompromised patients with a cutaneous abscess.

58. Necrotizing fasciitis should be considered in any patient with a soft-tissue infection who has pain and tenderness out of proportion to the visible degree of cellulitis.

59. If using antibiotics to treat abscesses, assume Methicillin-resistant *Staphylococcus aureus* (MRSA) as the causative agent.

60. Amphetamine and cocaine toxicity should be treated with IV benzodiazepine in incremental doses titrated to adequate control of heart rate, blood pressure (BP), and temperature.

61. β-blockers are contraindicated in the treatment of stimulant toxicity because they may potentiate alpha effects and cause coronary artery vasoconstriction and hypertension.

62. No diagnostic studies are indicated in an asymptomatic patient exposed to smoke in a nonenclosed space.

63. In the presence of carbon monoxide (CO), pulse oximetry will yield a falsely elevated reading.

64. A BP greater than 140/90 in a pregnant woman is suspicious for preeclampsia.

65. A pregnant woman with hypertension and seizures should be treated with IV magnesium sulfate and consideration of emergent delivery of the fetus.

66. The most deceptive of serious hand injuries is the high pressure injection injury from a hydraulic paint or oil gun because despite a seemingly innocuous appearance on initial presentation, these injuries require aggressive, surgical management.

67. When allowing a patient to leave against medical advice, consideration of the patient's ability to pay is not part of the equation. Only the risks, benefits, and patient's ability to understand the risks and benefits are important.

68. Be aware of the long-term cancer risk of patients exposed to diagnostic radiation, particularly those who are young or have had multiple studies.

69. With few exceptions, procedures performed in the ED can be done with fewer complications and greater success using ultrasound guidance.

70. Any elderly patient with flank, back, abdominal pain, hypotension, syncope, or pulseless electrical activity (PEA) should have an emergency ultrasound examination to evaluate for an AAA.

71. IV bolus administration of epinephrine to a patient with an obtainable BP and pulse can result in ischemic cardiac pain, hypertension, supraventricular tachycardia (SVT), and VT.

72. Examine every patient with urticaria for mucosal edema, stridor, wheezing, and hypotension to rule out life threats associated with anaphylaxis.

73. A contaminated wound is one with a high degree of bacterial inoculum at the time of injury and not synonymous with a dirty wound.

74. Determination of pretest probability for venous thromboembolism (VTE) is critical in knowing when to initiate a diagnostic work-up and how to interpret your test results.

75. A D-dimer assay is only useful to exclude thromboembolic disease in patients with a low pretest probability.

76. The problem of "error" in medicine, and the adverse events that sometimes follow, are problems of psychology and engineering, not of medicine.

77. Emergency medicine, by its nature, has more failure-producing conditions than any other specialty in medicine.

78. CT of the head will identify 95% of patients with subarachnoid hemorrhage. Lumbar puncture (LP) is recommended for patients with a strong clinical suspicion, despite a negative CT of the head.

79. The patient with a posterior nasal packing in place must be monitored in the hospital for recognition of hypoxia or apnea secondary to stimulation of the nasopulmonary reflex.

80. In almost all cases, trauma patients with unstable vital signs and a positive ED-focused abdominal sonography for trauma (FAST) examination for free fluid should go directly to laparotomy.

81. In patients with hyponatremia, to avoid central pontine myelinolysis, serum sodium should never be raised by more than 0.5 mEq/hr or 12 mEq in 24 hours.

82. Consider a retropharyngeal space infection in a young child presenting with a history of fever, refusal to drink, sore throat, and reluctance to move their neck.

83. The concomitant ingestion of ethanol (ETOH) with methanol or ethylene glycol protects against toxic metabolites.

84. Myocarditis should be considered in a patient with a sustained, unexplained tachycardia.

85. Suspect ectopic pregnancy when there is no evidence of intrauterine pregnancy (IUP) by transvaginal ultrasound and the quantitative human chorionic gonadotropin (HCG) concentration is greater than 2000 IU/L.

86. In a lucid patient with blunt abdominal trauma, the clinical examination is the best guide for selection of diagnostic tests.

87. Obtain a CT scan of the head on any patient on warfarin (Coumadin) with even a minor head trauma.

88. A single negative abdominal ultrasound alone does not reliably exclude significant intraperitoneal injury.

89. Children manifest shock later than adults with the same percentage of blood loss, yet decompensate more quickly once this critical volume is lost.

90. In the case of vascular and/or skin compromise of a deformed limb, urgent realignment and splinting of the involved extremity should precede radiography.

91. Always exclude associated fractures of the spine and lower extremities in patients with calcaneal fractures.

92. Never restrain a patient in the prone position; restrain on their side to minimize risks of aspiration and sudden death.

93. Consider domestic violence in women with depression, suicidal ideations, chronic pain, psychosomatic complaints, or multiple ED visits.

94. As little as 2 weeks of chronic steroid use (prednisone $>$ 20 mg/day) will cause adrenal suppression, making a patient more prone to adrenal crisis.

95. Lightning strike is the one exception to the usual multicasualty incident (MCI) triage rules: The first priority should go to those who are not breathing and not moving because only those who present in cardiac arrest are at high risk of dying.

96. The NEXUS criteria are 99.6% sensitive and 12.9% specific for significant cervical spine injuries in adults.

97. Follow potassium closely when treating patients with insulin.

98. Glucose should not be withheld due to the unfounded fear of precipitating Wernicke Korsakoff's syndrome.

99. Zoos usually keep antivenin on hand for the exotic venomous animals in their collections.

100. Transient ischemic attack (TIA) is a harbinger of early acute stroke (up to 10% in first 48 hours).

DECISION MAKING IN EMERGENCY MEDICINE

Vincent J. Markovchick, MD, FAAEM, FACEP

1. Is there anything unique about emergency medicine?
Although there is significant crossover between emergency medicine and all other clinical specialties, emergency medicine has unique aspects that make it different, such as the approach to patient care and the decision-making process.

2. Describe the conventional method of evaluating a patient.
A comprehensive history, physical examination, *routine* laboratory diagnostic studies, special diagnostic procedures, and the formulation of a problem-oriented medical record and rational course of therapy constitute the *ideal* approach to patient care because it is so comprehensive.

3. Why is the conventional methodology not ideal for use in the ED?
Even though in retrospect only 10% to 20% of patients presenting to an ED truly have emergent problems, it must be presumed that every patient who comes to an ED has an emergent condition. Therefore, the first and most important question that must be answered is: "What is the life threat?" The conventional approach does not ensure an expeditious answer to this question. Time constraints also impede the use of conventional methodology in the ED.

4. How do I identify the life-threatened patient?
Three components are necessary to quickly identify the life-threatened patient:
- A chief complaint and a brief, focused history relevant to the chief complaint.
- A complete and accurate set of vital signs in the field and in the ED that are accurately taken and critically interpreted.
- An opportunity to visualize, auscultate, and touch the patient.

5. What is so important about the chief complaint?
The chief complaint, which sometimes cannot be obtained directly from the patient but must be obtained from family members, observers, emergency medical technicians (EMTs), or others at the scene, will immediately help categorize the general type of problem (e.g., cardiac, traumatic, respiratory).

6. Why are vital signs important?
Vital signs are the most reliable, objective data that are immediately available to ED personnel provided they are accurately taken and critically interpreted. Vital signs and the chief complaint, when used as triage tools, will identify the majority of life-threatened patients. Familiarity with normal vital signs for all age groups is essential.

7. What are the determinants of (normal) vital signs?
Age, underlying physical condition, medical problems (e.g., hypertension), and current medications (e.g., β-blockers) are important considerations in determining normal vital signs for a given patient. For example, a well-conditioned, young athlete who has just sustained major trauma and arrives with a resting, supine pulse of 80 beats per minute must be presumed to have significant blood loss because the normal pulse is probably in the range of 40 to 50 beats per minute.

8. What is the most inaccurate vital sign taken in the field and ED?

In the field, the most common inaccurate vital sign is the respiratory rate because it is sometimes estimated rather than counted. In the ED, the temperature may be inaccurate if a tympanic membrane thermometer was used or if the patient was hyperventilating or mouth breathing when the oral temperature was taken.

9. Why do I need to compare field vital signs with ED vital signs?

Most prehospital care systems with a level of care beyond basic transport also provide therapy to patients. Because this therapy usually makes positive changes in the patient's condition, the patient may look deceptively well on arrival in the ED. For example, a 20-year-old woman with acute onset of left lower quadrant abdominal pain, who is found to be cool, clammy, and diaphoretic, with a pulse of 116 beats per minute, a blood pressure of 78 palpable, and who receives 1500 mL of intravenous (IV) fluid en route to the ED, may arrive with normal vital signs and no skin changes. If one does not read and pay attention to the EMT's description of the patient and the initial vital signs, the presumption may be made that this is a stable patient.

10. When are *normal* vital signs abnormal?

This is where the chief complaint comes in and correlating it for consistency with the patient's presentation. For example, a 20-year-old man who states he has asthma and has been wheezing for hours arrives in the ED with a respiratory rate of 14 breaths per minute. An asthmatic who is dyspneic and wheezing should have a respiratory rate of at least 20 to 30 breaths per minute. Thus, a *normal* respiratory rate of 14 breaths per minute in this setting indicates the patient is fatiguing and is in respiratory failure. This is a classic example of when "normal" is extremely abnormal.

11. Why do I need to visualize, auscultate, and touch the patient?

In many instances, these measures help to identify the life threat (e.g., is it the upper airway, lower airway, or circulation?). Touching the skin is important to determine whether shock is associated with vasoconstriction (i.e., hypovolemic or cardiogenic) or with vasodilatation (i.e., septic, neurogenic, or anaphylactic). Auscultation will identify life threats associated with the lower airway (e.g., bronchoconstriction, tension pneumothorax).

12. Once I have identified the life threat, what do I do?

Do not go on. Stop immediately and intervene to reverse the life threat. For example, if the initial encounter with the patient identifies upper airway obstruction, take whatever measures are necessary to alleviate upper airway obstruction such as suctioning, positioning, or intubating the patient. If the problem is hemorrhage, volume restoration and hemorrhage control (when possible) are indicated.

13. I have identified and stabilized or ruled out an immediate life threat in the patient. What else is unique about the approach?

The differential diagnosis formulated in the ED must begin with the most serious condition possible to explain the patient's presentation and proceed from there. An example is a 60-year-old man who presents with nausea, vomiting, and epigastric pain. Instead of assuming the condition is caused by a gastrointestinal disorder, one must consider that the presentation could represent an acute myocardial infarction (MI) and take the appropriate steps to stabilize the patient (i.e., start an IV line, place the patient on O_2, and a cardiac monitor) and rule out an MI by completing an adequate history, physical examination, and electrocardiogram ECG).

14. Why does formulating a differential diagnosis sometimes lead to problems?

The natural tendency in formulating a differential diagnosis is to think of the most common or statistically most probable condition to explain the patient's initial presentation to the ED. If one does this, one will be right most of the time but may overlook the most serious, albeit sometimes a very uncommon, problem. Therefore, the practice of emergency medicine involves some degree of healthy paranoia in that one must consider the most serious

condition possible and, through a logical process of elimination, rule it out and thereby arrive at the correct and generally more common diagnosis.

15. Is a diagnosis always possible or necessary with information I can obtain in the ED?

No. Sometimes it takes days, weeks, or months for the final diagnosis to be made. It is unreasonable to expect that every patient evaluated in the ED should or must have a diagnosis made in the ED. If you have an obsessive-compulsive personality with a need to be absolutely certain about what a patient has before you can act to stabilize or treat the patient, then the ED is an unhealthy work environment for you.

16. Suppose I cannot make the diagnosis, what do I do?

It is advisable to be intellectually honest and admit to the patient and document in the medical record the inability to make a diagnosis. As stated previously, it is the role of the ED physician to rule out and stabilize serious or life-threatening causes of a patient's presentation, not to arrive at the definitive diagnosis. For example, a patient who presents with acute abdominal pain, who has had an appropriate history taken, who has had a physical examination and diagnostic studies performed, and who in your best judgment does not have a life-threatening or acute surgical problem should be so informed. The discharge diagnosis would be abdominal pain of unknown etiology. This avoids the trap so often encountered of labeling the patient with a benign diagnosis such as gastroenteritis or gastritis that is not supported by the medical record. More importantly, it avoids giving the patient the impression that there is a totally benign process occurring and will help to avoid the medical (and legal) problem of the patient presenting 2 days later with something more serious, such as a ruptured appendix.

17. What is the most important question to ask a patient who presents to the ED with a chronic, persistent, or recurrent condition?

"What's different now?" This question should be asked of all patients who have a chronic condition that has resulted in their visit to the ED. The classic example is migraine headache. The patient with a chronic, recurrent migraine headache who is not asked this question may, on this presentation, have had an acute subarachnoid bleed. Such a patient may not volunteer that this headache is different from the pattern of chronic migraines unless asked.

18. How do I decide if the patient needs hospitalization?

Obviously, the medical condition is the first factor to consider. The question that must be answered is: "Is there a medical need that can be fulfilled only by hospitalization?" For example, does the patient need oxygen therapy or cardiac monitoring? Another factor to weigh in the decision regarding hospitalization is whether the patient can be safely observed in the outpatient setting. For example, a patient who has sustained head trauma and needs to follow head trauma precautions at home, and who is either homeless or lives alone, cannot be safely discharged. The patient's ability to pay for services should never enter into ED disposition decisions. A short-stay ED observation unit can be helpful in avoiding the need for some inpatient admissions.

19. If the patient does not need admission, how do I arrange a satisfactory disposition?

Every patient seen in the ED must be referred to a physician or referred back to the ED for follow-up care. Failure to do so constitutes patient abandonment. Appropriate and specific follow-up instructions should be given to all patients.

20. What is the most important thing to consider and document in the ED discharge instructions?

All follow-up instructions must include specific mention of the most serious potential complication of the patient's condition. For example, a patient who is being discharged home with the diagnosis of a probable herniated L4–L5 intervertebral disk should be instructed to

return immediately if any bowel or bladder dysfunction develops. This takes into account the most serious complication of a herniated lumbar disk, which is a central midline disk herniation (cauda equina syndrome) with bowel or bladder dysfunction and which constitutes an acute neurosurgical emergency.

21. What two questions should always be asked (and answered) before a patient is discharged from the ED?

- Why did the patient come to the ED?
- Have I made the patient feel better?

Generally, most patients present to the ED because of pain, somatic or psychological, and a reasonable expectation is that this pain will be acknowledged and appropriately treated. If such pain cannot be alleviated, a thorough explanation should be given to the patient regarding the reasons why analgesics cannot be provided. An example of this is a patient with abdominal pain of unknown etiology, which may evolve into appendicitis, to whom narcotics are not given because they may delay the recognition of worsening symptoms and localized abdominal pain. Reassurance is sometimes all that is needed to relieve anxiety about serious medical conditions such as cancer or heart attack. Other agents such as antiemetics or antianxiety medications should be administered in the ED to alleviate presenting symptoms.

22. Why is the previous question and answer one of the most important in this chapter?

Attention to treating and alleviating a patient's pain will dramatically reduce subsequent complaints concerning care in the ED and remove one of the significant risk factors for initiation of a malpractice suit. It is also how you would want to be treated.

23. What about the chart?

The chart must reflect the answers to the preceding questions in this chapter. It need not list the entire differential diagnosis, but one should be able to ascertain from reading the chart that the more serious diagnoses were indeed considered. It also must contain appropriate follow-up instructions.

KEY POINTS: DECISION MAKING IN EMERGENCY MEDICINE

1. Stabilize the patient before performing diagnostic procedures.
2. Always consider the most serious possible cause of every patient's presentation signs and symptoms.
3. Always inquire about a patient's social situation prior to ED discharge.
4. Remember to focus on alleviating the patient's somatic or psychological pain.

MANAGEMENT OF CARDIAC ARREST AND PRINCIPLES OF RESUSCITATION

CHAPTER 2

Jason S.Haukoos, MD, MSc

1. What are the ABCs of resuscitation?

Airway, breathing, and circulation. The ABCs should be used to guide the resuscitation of all critically ill patients, including all patients experiencing cardiac arrest.

2. How should cardiopulmonary resuscitation (CPR) be performed as described by the American Heart Association?

a. If the arrest is in the out-of-hospital setting, activate emergency medical services (EMS) by calling 911; if it occurs in the hospital, activate the hospital's cardiac arrest team.
b. Open the airway by performing a head tilt-chin lift or a head tilt-jaw thrust maneuver. These maneuvers cause anterior displacement of the mandible and lift the tongue and epiglottis away from the glottic opening. To improve airway patency, suction the mouth and oropharynx and insert an oropharyngeal or nasopharyngeal airway.
c. Assist breathing by performing mouth-to-mouth, mouth-to-mask, or bag-value-mask breathing. The recommended technique depends on the clinical setting, the equipment available, and the rescuer's skill and training. Although these techniques can sustain oxygenation and ventilation indefinitely in ideal situations, they can be suboptimal in the emergency setting. Air leaks around the facemask may result in inadequate ventilation, insufflation of the stomach, and emesis and aspiration. To reduce the probability of such problems, deliver slow, even breaths, pausing for full deflation between breaths to avoid excessive peak inspiratory pressures. Use the Sellick maneuver (using your fingers to apply continuous posterior pressure to the cricoid cartilage) to compress the esophagus to reduce the risk of vomiting and aspiration.
d. After opening the airway and initiating rescue breathing, check for spontaneous circulation by palpating for a carotid or femoral pulse. If the patient is pulseless, begin chest compressions. Compress the chest smoothly and forcefully 100 times per minute, allowing for complete chest recoil. Minimize interruptions in chest compressions, providing approximately 30 compressions followed by two breaths until a defibrillator arrives or the patient begins to move.

3. How important is ventilation during resuscitation efforts in the out-of-hospital setting?

Active assisted ventilation during cardiac arrest may not always be beneficial and is now thought to be less important than previously believed. If performing ventilation contributes to interrupted chest compressions or excessive intrathoracic pressures, it may be deleterious.

4. What is passive oxygen insufflation?

Passive oxygen insufflation is accomplished by placing an oropharyngeal airway and a nonrebreather facemask with high-flow oxygen on the patient. Preliminary data suggest this approach may be superior when compared to a traditional active ventilatory approach using a bag-valve in conjunction with other cardiocerebral resuscitation strategies.

5. What is the *squeeze, release, release* method of providing mechanical ventilation?

Squeeze, release, release was first described in 1997 as a bag-valve-mask technique to provide an appropriate level of ventilation to pediatric patients. Subsequently, this technique

has been extended to adult patients and consists of performing ventilation at a rate consistent with someone saying, "squeeze, release, release" to maintain an appropriate ventilation rate.

6. What are the exceptions to the rule of the ABCs?

a. **Monitored cardiac arrest.** When a patient in a monitored setting experiences sudden pulseless ventricular tachycardia (VT) or ventricular fibrillation (VF), immediate electrical defibrillation is the priority.
b. **Traumatic arrest.** In traumatic cardiac arrest, closed-chest CPR is usually ineffective. In trauma, the cause of the arrest may be a tension pneumothorax, cardiac tamponade, or exsanguinating hemorrhage from the thorax or abdomen. An immediate thoracotomy, not CPR, is indicated. When neck injury is suspected, a jaw thrust (never a head tilt) should be used to open the airway.

7. Explain the mechanism of blood flow during CPR?

Two basic models explain the mechanism of blood flow during CPR. In the *cardiac pump model*, the heart is squeezed between the sternum and the spine. Chest compressions result in systole, and the atrioventricular valves close normally, ensuring unidirectional, antegrade flow. During the relaxation phase (diastole), intracardiac pressures fall, the valves open, and blood is drawn into the heart from the lungs and vena cavae.

In the *thoracic pump model*, the heart is considered a passive conduit. Chest compressions result in uniformly increased pressures throughout the thorax. Forward blood flow is achieved selectively in the arterial system because the stiff-walled arteries resist collapse and because retrograde flow is prevented in the great veins by one-way valves. In addition, chest recoil results in increased negative intrathoracic pressures, which improve ventricular filling and coronary blood flow. These mechanisms have been substantiated in animal models and both likely contribute to blood flow during CPR.

8. Is blood flow to the brain and heart adequate during CPR?

Even when performed by experts, CPR provides only approximately 30% of normal blood flow to the brain and 10% to 20% of normal blood flow to the heart. Blood flow to the heart occurs during the relaxation phase of CPR, whereas blood flow to the brain occurs during the compression phase of CPR. This is the foundation for the American Heart Association's recommended CPR duty cycle of 50% (the ratio of time spent in compression to the time spent in relaxation).

9. What is coronary perfusion pressure (CPP)?

Coronary perfusion pressure is defined as the aortic pressure minus the right atrial pressure during diastole.

10. What is the association between CPR, CPP, and return of spontaneous circulation (ROSC)?

Better CPR produces better CPPs. Higher CPPs translate into higher rates of ROSC. This emphasizes the importance of performing good CPR and explains how vasopressors (e.g., epinephrine) impact rates of ROSC by increasing CPPs.

11. Describe hands off CPR?

Hands off CPR refers to lifting the hands off the chest wall during decompression to maximize chest recoil. Incomplete chest wall recoil during CPR has been shown to result in hemodynamic deterioration of forward blood flow in animal models. In addition, in an observational human study, incomplete chest recoil was common during CPR.

12. Discuss the role of pharmacologic therapy during CPR.

The immediate goal of pharmacologic therapy is to improve CPPs, and thus, myocardial blood flow, which correlates with ROSC. Adrenergic agonists (e.g., epinephrine) augment the aortic-to-right atrial diastolic gradient by increasing systemic vascular resistance. Reports

suggest that nonadrenergic agonists (e.g., vasopressin) may be more effective than adrenergic agonists in improving myocardial blood flow. Additional clinical studies suggest that amiodarone improves rates of successful defibrillation and prevents recurrent postarrest dysrhythmias. These antifibrillatory effects may be independent of myocardial blood flow.

KEY POINTS: STANDARD DOSES OF CARDIAC ARREST MEDICATIONS

1. Epinephrine: 1 mg IV/IO push
2. Vasopressin: 40 U IV/IO push
3. Atropine: 1 mg IV/IO push
4. Amiodarone: 300 mg IV/IO push
5. Lidocaine: 1.0-1.5 mg/kg IV/IO push

13. Under what circumstances should *CPR* be used *before defibrillation*?

A growing body of research suggests that patients with untreated prolonged VF may benefit from CPR for 2 to 3 minutes prior to defibrillation, and several communities have adopted this in the prehospital setting.

14. What are the indications for open chest cardiac massage?

The primary indication for open chest cardiac massage is traumatic arrest. However, several other nontrauma-related indications include: hypothermia, pulmonary embolism, cardiac tamponade, abdominal hemorrhage, third-trimester pregnancy, and patients with chest wall deformities that prevent adequate chest compressions.

15. What are the most common causes of cardiopulmonary arrest?

Although the incidence of VF appears to be declining, it still remains a common initial rhythm encountered in patients suffering from cardiac arrest. Underlying coronary artery disease accounts for the majority of VF arrests. Other etiologies of VF include: drug toxicity, electrolyte disturbances (e.g., hyperkalemia), and prolonged hypoxemia.

The second most common initial rhythm encountered is asystole. This commonly results from prolonged untreated VF and is due to severe hypoxia and acidemia. Other causes of asystole include: drug toxicity, electrolyte disturbances, and hypothermia.

Pulseless electrical activity (PEA) is the third most commonly encountered initial arrest rhythm. As with asystole, PEA commonly results from prolonged untreated VF or defibrillation of VF after a prolonged untreated period (usually $>$5 minutes). Other causes of PEA include: hypovolemia, hypoxia, cardiac tamponade, tension pneumothorax, hypothermia, massive pulmonary embolism, drug toxicity, electrolyte disturbances, acidemia, or myocardial infarction.

16. What are other reversible causes and immediate treatments of cardiopulmonary arrest?

- **Hyperkalemia.** Calcium chloride (preferred over calcium gluconate), sodium bicarbonate, insulin and glucose, and nebulized albuterol.
- **Anaphylaxis.** Intravascular volume expansion (using crystalloid) and epinephrine.
- **Cardiac tamponade.** Pericardiocentesis or pericardiotomy.
- **Tension pneumothorax.** Thoracic decompression.
- **Hypovolemia.** Intravascular volume expansion using crystalloid solutions. In the setting of trauma, blood products should be given judiciously and concomitantly with crystalloid. Always consider using a level I infuser when large volumes are required over a short period of time.

- **Torsades de pointes.** Defibrillation, magnesium sulfate, isoproterenol, or overdrive pacing.
- **Toxic cardiopulmonary arrest**
  - *Carbon monoxide* poisoning occurs after prolonged exposure to smoke and inhalation of exhaust from incomplete combustion. High-flow and hyperbaric oxygen and management of acidosis are the cornerstones of treatment.
  - *Cyanide* poisoning occurs after intentional ingestion or after exposure to fire involving synthetic materials. The antidote for this includes intravenous (IV) sodium nitrite and sodium thiosulfate.
  - *Tricyclic antidepressants* act as type Ia antidysrhythmic agents and cause cardiac conduction slowing, ventricular dysrhythmias, hypotension, and seizures. Vigorous serum alkalinization and seizure control are required.
- **Primary asphyxia.** In addition to anaphylaxis, obstructive asphyxia may occur after foreign body aspiration, inflammatory conditions of the hypopharynx (e.g., epiglottitis or retropharyngeal abscess), or neck trauma. The latter results in edema or hematoma formation, subcutaneous emphysema, or laryngeal or tracheal disruption. Treatment includes establishment of a patent airway via endotracheal intubation or by cricothyrotomy and assisted ventilation with 100% oxygen.

17. How should VF be treated?

Rapid treatment is essential as the prognosis worsens with each untreated minute. Standard treatment consists of immediate defibrillation. Recommended energy levels include beginning at maximal or near-maximal energy (e.g., 150 J–200 J biphasic). The antidysrhythmic agent of choice is amiodarone, which enhances the rate of successful defibrillation and reduces the rate of recurrent VF after successful conversion. Administration of epinephrine or vasopressin before defibrillation may improve defibrillation success; in addition, CPR before defibrillation (see question 13) may also improve defibrillation success in the setting of prolonged VF.

18. What's the difference between monophasic and biphasic defibrillation?

The terms *monophasic* and *biphasic* refer to the energy waveforms produced by the defibrillation device. Monophasic waveforms vary in speed in which the waveform returns to the zero voltage point, whereas biphasic waveforms deliver current that first flows in a positive direction for a specific duration, then reverses direction for a specific duration. Biphasic defibrillation achieves the same defibrillation success rates as monophasic defibrillation but at significantly lower energy levels, resulting in less postresuscitation cardiac dysfunction.

19. What if VF persists after initial treatment?

a. Perform endotracheal intubation and ensure adequate oxygenation and ventilation.
b. Continue CPR.
c. Administer epinephrine (1 mg IV push) or vasopressin (40 U IV push) to augment aortic diastolic blood pressure and to improve myocardial perfusion.
d. Administer amiodarone (300 mg IV push). Amiodarone may be repeated at 150 mg IV push after 3 to 5 minutes.
e. Consider administering magnesium sulfate (1 g–2 g IV push).
f. Although lidocaine or procainamide have not been shown to improve defibrillation success rates or restore perfusing rhythms in patients with VF, also consider their administration (lidocaine 1.0–1.5 mg/kg IV push or procainamide 17 mg/kg at a rate of 30 mg/min intravenously).

20. Describe the three-phase model of cardiac arrest?

- The first phase, called the *electrical phase*, suggests that immediate defibrillation is the most efficacious treatment within the first 4 minutes of VF.
- The second phase, called the *circulatory phase*, follows the first phase and suggests that successful ROSC and overall survival are maximized with a period of CPR before defibrillation.

- The third phase, called the *metabolic phase*, is reached after about 10 minutes, is associated with a profound systemic inflammatory response syndrome, and no current therapies offer survival benefit in this setting.

21. How should asystole be treated?

a. Confirm the absence of cardiac activity in more than one electrocardiogram (ECG) lead. Check for loose or disconnected cables and monitor leads. Finally, increase the amplitude to detect occult, fine VF.
b. Administer epinephrine (1 mg IV push) or vasopressin (40 U IV push).
c. Administer atropine (1 mg IV push) to counteract high vagal tone.

KEY POINTS: MANAGEMENT OF CARDIAC ARREST

CPR and defibrillation are the most important components to the initial management of the cardiac arrest patient.

1. Treat VF with immediate defibrillation (if the arrest is witnessed), CPR, then defibrillation (if the arrest is unwitnessed), or amiodarone.
2. If the arrest is due to PEA, remember its common reversible causes (i.e., hypovolemia, hypoxia, cardiac tamponade, tension pneumothorax, hypothermia, massive pulmonary embolism, drug toxicity, electrolyte disturbances, acidemia, or myocardial infarction) and treat them appropriately.
3. If the arrest is due to asystole, remember to exclude fine VF.

22. Is defibrillation or electrical pacing useful for asystole?

Defibrillation is reserved for cases in which differentiation between asystole and fine VF is difficult. In these ambiguous situations, defibrillation should be employed after administration of epinephrine. Electrical pacing is occasionally attempted for asystole but is rarely effective in restoring pulses.

23. What are the appropriate routes of drug administration?

IV administration is the preferred route of drug therapy during CPR. A central venous catheter is ideal, although placement should not supersede optimal resuscitation, including performance of chest compressions. Use of a peripheral venous catheter results in a slightly delayed medication onset of action, although the peak drug effect is similar to that for the central route. An intraosseous (IO) line may also be used and should take precedence over other approaches, including intramuscular or endotracheal routes. All drugs used for resuscitation can be given in conventional doses using IO access. Intracardiac administration should be reserved for cases of open cardiac massage. Endotracheal drug administration should be used as a last resort.

24. I thought IO cannulation was only used as a last resort and for pediatric patients. What's the deal?

IO cannulation provides a quick, effective, and safe means to access a noncollapsible venous plexus, either in the proximal tibia, proximal humerus, or sternum. (The sternum should be avoided as an IO site in cardiac arrest because it would interfere with chest compressions.) It can be used in all age groups and allows for effective fluid resuscitation, drug delivery, and blood sampling for laboratory evaluation. In fact, the IO functions similar to that of a central line.

25. When may prehospital resuscitation efforts be terminated?

According to the most recent American Heart Association's Advanced Cardiac Life Support (ACLS) guidelines, prehospital resuscitation can be discontinued by EMS authorities when a

valid no-CPR order is presented to the rescuers or when a patient is deemed nonresuscitable after an adequate trial of ACLS, including successful endotracheal intubation, achievement of IV access and administration of appropriate medications, determination of a persistent asystolic or agonal rhythm, and when no reversible cause for the arrest is identified.

26. Which vasopressor should I administer in the setting of cardiac arrest, epinephrine or vasopressin?

This remains controversial. Epinephrine has been evaluated in human trials in approximately 9,000 patients. The recommended 1-mg dose was extrapolated from animal research, and trials comparing this dose with high-dose regimens (i.e., 0.1 to 0.2 mg/kg) demonstrated increased rates of ROSC in patients who received high-dose epinephrine; however, these studies have not shown improvements in survival or survival with good neurologic outcomes. Vasopressin acts directly on V_1-receptors and, unlike epinephrine, is more effective in an acidemic environment. Vasopressin has been compared to epinephrine in three human trials, totaling approximately 1,500 patients without a significant difference in survival.

27. Should I use amiodarone in the setting of cardiac arrest?

Amiodarone is a class III antidysrhythmic agent used, in part, to treat VT or VF. Two randomized clinical trials have demonstrated a survival to hospital admission (but not to hospital discharge) benefit for amiodarone over placebo and lidocaine, respectively. In most settings amiodarone has become the first-line agent for treating VT or VF.

28. Should I routinely administer sodium bicarbonate during resuscitation?

Sodium bicarbonate is not recommended as routine therapy in the setting of cardiac arrest. A no- or low-flow state causes progressive respiratory and metabolic acidosis as a result of accumulation of CO_2 and lactate. Neither state can be corrected without adequate oxygenation, ventilation, and tissue perfusion. At present, no clinical data support its routine use except in cases of hyperkalemia, tricyclic antidepressant overdose, or preexisting metabolic acidosis.

29. Should I routinely administer calcium during resuscitation?

Calcium is not recommended as routine therapy in the setting of cardiac arrest. Although no data exist to support its routine use, it may be beneficial in the setting of hyperkalemia (most often seen in chronic renal failure/dialysis patients), hypocalcemia, or calcium channel blocker toxicity.

30. What should I do after ROSC?

Once ROSC is achieved, the vulnerable postresuscitation period begins. This period is marked by a profound systemic inflammatory response syndrome resulting from whole-body ischemia and reperfusion. Patients commonly develop hemodynamic instability, resulting in multiple organ dysfunction and subsequent death (hours to days later). Prompt recognition and treatment of the inciting event and meticulous intensive care unit support are required to provide patients with the best probability for survival. Use of hemodynamic and inotropic agents is important for supporting patients during this period, and recent description of a hemodynamic optimization protocol has been reported. In addition, mild therapeutic hypothermia should be performed to improve neurologic recovery.

31. What percentage of all cardiac arrest patients survive to hospital discharge?

5%.

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CHAPTER 3

AIRWAY MANAGEMENT: RESCUE AIRWAY DEVICES

Barry C. Simon, MD

1. Do I really need to know about airway management?
Yes. Expeditious airway management saves lives.

2. How is the adequacy of ventilation assessed?
First, look at the patient. Cyanosis suggests profound hypoxia. Diaphoresis and somnolence indicate hypercapnia and respiratory acidosis. Measuring the respiratory rate and assessing the tidal volume by placing your hand over the endotracheal tube or the patient's mouth and nose are useful bedside clinical clues. If you are at all concerned, use a pulse oximeter. Mild-to-moderate hypoxia can be monitored with pulse oximetry, which measures arterial oxygen saturation. End-tidal CO_2 monitoring should be used for assessing ventilation.

3. Why do patients need airway management?
Assisted ventilation can help to decrease intracranial pressure or correct hypercarbia and acidosis. **Oxygenation** may be needed in patients with severe lung disease or injury who are unable to maintain an acceptable PaO_2. **Overcoming or preventing airway obstruction** is imperative in patients with neck trauma, epiglottitis, or airway burns from smoke inhalation or ingestion of caustic substances. **Prevention of aspiration** in patients with altered mentation is best accomplished with endotracheal intubation. **Administration of intratracheal drugs** (e.g., **epinephrine, atropine, lidocaine)** through the endotracheal tube is indicated in resuscitation until an intravenous (IV) line can be established.

4. What is the most common cause of airway obstruction?
The tongue because it obstructs the airway far more commonly than do foreign bodies or edema. With decreasing levels of consciousness, the supporting muscles in the floor of the mouth lose tone, and the tongue falls posteriorly, obstructing the oropharynx. The fastest, least invasive treatment modality is repositioning via the head tilt-chin lift maneuver. A nasopharyngeal or oral airway should be inserted in a patient with ongoing upper airway obstruction unrelieved by repositioning. Care must be taken in patients with potential or suspected cervical spine injury.

5. What is a Combitube?
The Combitube is a dual-lumen, dual-cuffed airway. The two lumens allow ventilation whether the tube is placed into the esophagus or in the trachea. The tube differs from the esophageal obturator airway because it does not require an adequate mask seal to affect adequate ventilation. This device is placed blindly and is usually placed in the esophagus. Dual balloons are inflated to seal the device. Tube/lumen 1 is ventilated if the device ended up in the esophagus and lumen 2 is ventilated if it was placed in the trachea. End-tidal CO_2 detectors are used to confirm placement and ventilation.

6. What are the indications for the Combitube?
The Combitube is most useful in the prehospital setting when assisted ventilation is needed, the providers are not trained or authorized to perform endotracheal intubation, or an attempt at endotracheal intubation has been unsuccessful.

7. What is the King airway?

This is an alternative device to the Combitube that uses a single channel to inflate both the smaller distal (esophageal) and larger proximal (oropharyngeal) balloons. It has the additional advantage of allowing passage of an airway exchange catheter into the trachea via its main port to facilitate endotracheal intubation. There is less risk of overinflating the esophageal balloon and rupturing the esophagus as can occur with the Combitube.

8. What are the relative contraindications to blind nasotracheal intubation (BNTI)?

Apnea is the most important contraindication because the chance of esophageal intubation is unacceptably high. Because epistaxis complicates BNTI in one third of cases, the procedure is contraindicated in patients with coagulopathies. Other routes of intubation are advisable in patients with maxillary facial or severe nasal fractures because a false passage, severe epistaxis, or rarely, cranial placement may occur. Hematomas, epiglottitis, and infections of the upper neck are relative contraindications because of the risk of sudden airway obstruction or laryngospasm.

9. Name some complications of BNTI.

Hypoxia may occur during the intubation process. In addition to epistaxis and esophageal intubation, there are other uncommon acute complications, such as avulsion of the turbinates, avulsion of the vocal cords, and pharyngeal perforations with retropharyngeal dissection. Significant elevation in intracranial pressure with coughing may precipitate uncal herniation in patients with head injuries. Sinusitis may occur several days later from obstruction of the paranasal ostia.

10. What is the laryngeal mask airway (LMA)?

The LMA (Fig. 3-1) is an irregular ovoid-shaped silicone mask with an inflatable rim connected to a tube that allows ventilation. The device can be passed blindly with a high degree of success. The nose of the mask is seated in the esophagus. When the rim is inflated, it prevents air from going into the esophagus and forces air into the trachea. This is a good temporizing device until a definitive airway can be established. All practicing emergency physicians should be familiar with the use of the LMA.

11. What are the benefits of the LMA?

The LMA is relatively simple to place with a high degree of success even for those who are inexperienced. The LMA should be considered the alternative airway device of choice in cases in which traditional endotracheal intubation is not successful. The LMA and the newer

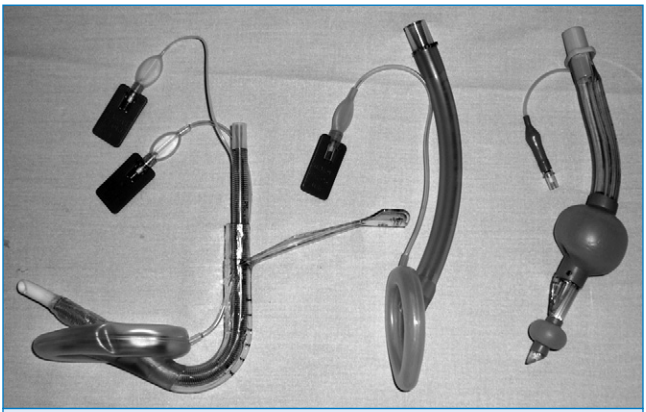

Figure 3-1. Airway rescue devices. *Left,* intubating laryngeal mask airway; *middle,* laryngeal mask airway; *right,* King airway.

intubating LMA (ILMA) have the added benefit of allowing practitioners to pass an endotracheal tube through the device into the trachea for definitive airway management.

12. What is rapid-sequence intubation (RSI)?

RSI is a method of facilitating endotracheal intubation by inducing short-term paralysis. Because all emergency patients are at risk for aspiration, the airway must be secured as quickly as possible. Paralysis with succinylcholine or rocuronium facilitates visualization and tube placement and reduces complications that occur with attempts to intubate an awake, struggling patient.

13. What are the predictors of a difficult intubation?

- Morbid obesity
- Abnormal facial shape
- Buck teeth
- Protruding/prominent tongue
- Prominent mandible
- Short neck/limited motion

The Mallampati score helps to predict the level of difficulty with intubation. A higher class score predicts a greater degree of difficulty (Fig. 3-2).

14. Don't you need to be an anesthesiologist to perform RSI? How is it done?

No. The basics can be remembered as the **five Ps:** preparation, preoxygenation, priming, pressure, and paralysis (Table 3-1).

a. **Prepare** equipment (e.g., suction, endotracheal tube, bag, mask, laryngoscope).
b. **Preoxygenate** with 100% oxygen (no positive pressure) ideally for 5 minutes.
c. Pretreat with a defasciculating dose of vecuronium or pancuronium (0.01 mg/kg).
d. **Prime** with thiopental, 3 to 4 mg/kg, or etomidate, 0.3 mg/kg rapid IV push.
e. Apply **pressure** with Sellick's maneuver (cricoid pressure) because consciousness is lost to prevent regurgitation and aspiration.
f. Follow thiopental or etomidate immediately with 1.5 mg/kg of succinylcholine or rocuronium 1.0 mg/kg IV push to **paralyze**.
g. Intubate the trachea and verify accurate placement with an end-tidal CO_2 detector.
h. Release cricoid pressure.

15. How do I preoxygenate a patient before intubation?

Bag-valve-mask ventilation is the only option in the apneic patient, even though this increases the risk of aspiration by raising gastric pressure. If a patient is making effective respiratory efforts, he or she should receive passive oxygenation via a nonrebreather mask on 100% oxygen for a full 5 minutes. In the apneic patient, eight vital capacity breaths using high-flow oxygen should be administered. Adequate preoxygenation will protect the patient against hypoxia for several minutes despite becoming apneic after induction and paralysis.

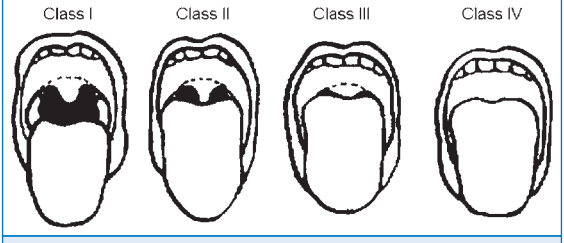

Figure 3-2. The Mallampati class score helps to predict the level of difficulty with intubation. The higher class score predicts a greater degree of difficulty.

TABLE 3-1. PARALYTIC DRUGS

	Succinylcholine (Anectine)	Rocuronium (Zemuron)	Pancuronium (Pavulon)	Vecuronium (Norcuron)
Uses	Skeletal muscle relaxation	Skeletal muscle relaxation	Skeletal muscle relaxation	Skeletal muscle relaxation
Supplied	200-mg vial, 20 mg/mL	100-mg vial, 10 mg/mL	10-mg vial, 1 mg/mL	10-mg powder, 1 mg/mL
Cost	*	***	*	**
Pregnancy class	C	B	C	C
Adult dose	1–1.5 mg/kg 3–4 mg/kg IM (max. 150 mg) 0.15 mg/kg priming dose	0.6–1.2 mg/kg	0.1 mg/kg 0.01 mg/kg priming dose	0.1–0.2 mg/kg 0.01 mg/kg priming dose
Pediatric dose	Succinylcholine 1 mg/kg Use atropine prior to succinylcholine in any child < 5 years AND any age repeat dosing Atropine 0.02 mg/kg IV (min dose 0.1 mg, max single dose 0.5 mg child, 1 mg adolescent)	0.6–1.2 mg/kg	0.04–0.1 mg/kg	Do not administer if less than 7 weeks of age
Onset	30–60 sec	45–90 sec	1–3 min	60-sec RSI
Duration	5–10 min	20–40 min	40–60 min	0.3–5 min standard 60–120 min RSI 20–40 min standard

Continued

TABLE 3-1. PARALYTIC DRUGS—cont'd

	Succinylcholine (Anectine)	Rocuronium (Zemuron)	Pancuronium (Pavulon)	Vecuronium (Norcuron)
Class	Ultrashort-acting depolarizing muscle relaxant	Rapid-acting nondepolarizing neuromuscular blocker	Long-acting nondepolarizing neuromuscular blocker	Intermediate-acting nondepolarizing neuromuscular blocker
Effects	Fasciculations, increased IOP, ICP, and IGP	Prolonged recovery with liver failure, vagolytic activity may increase HR, BP, and CO	Prolonged recovery with liver and renal failure	Prolonged recovery with liver and renal failure
	Caution: exaggerated hyperkalemic response with burns, spinal injury, stroke, paraplegia, neuromuscular disease (maximum at 7–10 days after injury) and acidosis, sepsis, crush muscle injury		Vagolytic activity may increase HR, BP, and CO	Onset and duration are dose dependent
		Onset and duration are dose dependent		Minimal histamine or CV effects
	Histamine release, cardiac arrhythmias, especially bradycardia, consider atropine 0.01 mg/kg			
Comments	Refrigerate or use in 14 days	Refrigerate or use in 30 days	Refrigerate or use in 6 months	Mix with 10 mL sterile H_2O

*, least expensive; ***most expensive.
Pregnancy class: B, presumed safety based on animal studies; C, uncertain safety; no human studies and animal studies show an adverse effect; D, Unsafe–evidence of risk that may in certain circumstances be justifiable.
BP, blood pressure; CO, cardiac output; CV, cardiovascular; HR, heart rate; ICP, intracranial pressure; IGP, intragastic pressure; IM, intramuscularly; IOP, intraocular pressure; RSI, rapid-sequence induction.

16. Which patients are likely to be difficult to ventilate with a bag-valve-mask?

Patients with:

- Excess facial hair
- Severe facial burns
- Morbid obesity
- Angioedema/facial fractures
- Unstable facial fractures

17. What is Sellick's maneuver?

Sellick described a method of applying pressure over the cricoid cartilage to help prevent aspiration. Pressure should equal the amount of force it takes to cause discomfort when pressing over the bridge of one's nose. Pressure is applied after loss of consciousness and is maintained until the endotracheal tube balloon is inflated and tube placement is confirmed. Yet the evidence supporting the widespread use of cricoid pressure to prevent aspiration is unconvincing. There is a risk that cricoid pressure may worsen the view of the larynx or can reduce airway patency. Therefore cricoid pressure should be released if there is any difficulty either intubating or ventilating the patient.

18. How do I remember the size of the endotracheal tube for children?

The easiest way is to carry a card in your wallet or refer to the Broselow tape. The following formula works for persons 2 to 20 years old:

$$\text{Tube size} = (\text{age in years} + 16) \div 4$$

19. Describe some of the induction drugs available for RSI.

- **Thiopental** is a short-acting barbiturate that has been used by anesthesiologists for decades. It is safe and effective with few serious complications but is a little longer acting than many of the newer agents (10–15 minutes). **Methohexital** is an ultrashort-acting barbiturate with a similar safety profile.
- **Midazolam,** a benzodiazepine, has the added benefit of being reversible.
- **Propofol** is a diisopropylphenol induction agent that has become popular among anesthesiologists and emergency physicians for short outpatient procedures such as reduction of dislocations. Its major disadvantage is a significant decrease in blood pressure.
- **Etomidate** has become the most popular induction drug in emergency settings for its rapid action, short duration, and absence of any effects on the cardiovascular system. Table 3-2 summarizes sedation and induction drugs.

20. Why is succinylcholine the most common paralyzing agent in RSI?

No other neuromuscular blocking agent has as rapid an onset of action (45–60 seconds) or as brief a duration of activity (4–7 minutes). This provides added safety with the return of spontaneous respiration within 7 minutes.

21. What are the theoretical risks of succinylcholine?

Despite its significant benefits, succinylcholine has many undesirable characteristics, some of which may be dangerous. It increases intragastric, intraocular, and intracranial pressure. Life-threatening hyperkalemia may occur in patients with neuromuscular disease or 3 to 4 days after major burns and trauma. Severe muscle contractions cause delayed pain and occasionally rhabdomyolysis. Rarely, it can precipitate malignant hyperthermia.

22. Are there any alternative paralytics?

- **Rocuronium** is gaining popularity, and many providers prefer rocuronium over succinylcholine. It has few complications and has an onset of action nearly as fast as succinylcholine. Its only significant drawback is a duration of action of 20 to 40 minutes.
- **Vecuronium** is another alternative, but its duration is even longer at 60 to 90 minutes. It is a poor choice for RSI because of its slow onset of action. Newer nondepolarizing drugs with properties similar to succinylcholine are on the horizon.

TABLE 3-2. SEDATION AND INDUCTION DRUGS

	Methohexital (Brevital)	Thiopental (Pentothal)	Propofol (Diprivan)	Etomidate (Amidate)	Ketamine (Ketalar)
Uses	Sedation, induction, anesthesia	Sedation, induction, anesthesia	Sedation, induction, anesthesia	Sedation, induction, anesthesia	Sedation, induction, analgesia, dissociative anesthesia
Supplied	500- mg powder 10 mg/mL	500-mg powder 25 mg/mL	200-mg ampule 10 mg/mL	20-mg ampule 2 mg/mL	500-mg vial 100 mg/mL
Cost	*	*	***	*	*
Pregnancy	B	C	B	C	?
Adult dose	1–1.5 mg/kg induction dose 0.25–1.0 mg/kg sedation	3–5 mg/kg induction dose 0.5–1.0 mg/kg sedation	1 mg/kg bolus then 0.5 mg/kg every 3-5 minutes as needed for procedural sedation	0.2–0.6 mg/kg induction dose Procedural sedation: 0.1 to 0.2 mg/kg IV over 30 to 60 seconds, followed by 0.05 mg/kg every 3 to 5 minutes as needed for procedural sedation	1.0–2.5 mg/kg induction dose 0.5–1.0 mg/kg IV sedation 5–10 mg/kg IM sedation
Pediatric dose	1–1.5 mg/kg 5–10 mg/kg IM	4–6 mg/kg	Loading dose: 1 mg/kg IV followed by 0.5 mg/kg every 3 to 5 min as needed for procedural sedation	only approved for children 10 years or older; same as adult dosing guidelines above.	1–2 mg/kg IV, 2–4 mg/kg IM

Onset	30–60 sec	30 sec	30 sec	30 sec	30 sec
Duration	5–10 min	3—5 min	3—5 min	3–10 min	5–15 min IV, 10–25 min IM
Class	Ultrashort-acting oxybarbiturate	Ultra-short thiobarbiturate	Diisopropylphenol hypnotic	Imidazole derivative nonbarbiturate hypnotic	Rapid-acting dissociative anesthetic
Effects	Rapid hypnosis: hypotension, apnea, no analgesia, excitatory phenomena (>children and elderly), seizure potential, bronchospasm, pain on injection, less fat uptake and faster onset/recovery than thiopental	Rapid hypnosis: hypotension, apnea, no analgesia, decreased ICP and CBF, maintains CPP, bronchospasm, decreased IOP, pain on injection, retrograde amnesia	Rapid hypnosis: hypotension, apnea, antiemetic and 10× more rapid recovery than thiopental, decreased IOP, ICP, MAP, and CPP, pain on injection, myoclonus, anaphylaxis with soy and egg allergy	Rapid hypnosis, relative CV and respiratory stability, no analgesia, decreased IOP, ICP, and CBF, maintain CPP, pain on injection, myoclonus (decreased with benzo/opioids), potential adrenal suppression	Rapid dissociative state, increased HR, BP, and maintenance of airway reflexes although apnea and hypotension possible in catechol-depleted patients, bronchodilation, analgesia, increased IOP and ICP, nystagmus, mydriasis
Comments	Mix with 50 mL NS	Mix with 20 mL NS	Slow IV increments, 20–40 mg every 10 sec		Push slowly over 2 min

*, least expensive; ***most expensive.
Pregnancy class: B, presumed safety based on animal studies; C, uncertain safety; no human studies and animal studies show an adverse effect; D, Unsafe–evidence of risk that may in certain circumstances be justifiable.
BP, blood pressure; CBF, cerebral blood flow; CPP, coronary perfusion pressure; CV, cardiovascular; HR, heart rate; ICP, intracranial pressure; IM, intramuscularly; IOP, intraocular pressure; IV, intravenously; MAP, mean arterial pressure; NS, normal saline.

23. Are there any contraindications to RSI?

Yes. Paralyze a patient only when you are sure he or she can be bag-mask ventilated if intubation is unsuccessful. Anticipation of a difficult airway based on anatomic features or traumatic anatomic distortion (e.g., patients with massive facial trauma or severe facial burns) is a relative contraindication. Inability to preoxygenate patients (e.g., patients with severe chronic obstructive pulmonary disease or asthma) is a relative contraindication to RSI. Patients with airway obstruction (e.g., foreign body, allergic reaction, airway infections, malignancies) who continue to make some respiratory effort should not be paralyzed.

24. How do I manage patients who have contraindications to RSI?

Nasotracheal intubation is a good alternative in patients with pulmonary disease. If unsuccessful, or if there is a contraindication to nasotracheal intubation, awake oral intubation with an induction agent, such as ketamine, allows the patient to maintain a certain degree of ventilation and airway protection during the procedure. Ketamine should not be used in patients with head injuries because it dramatically increases intracranial pressure. Benzodiazepines, such as midazolam, may be useful for induction because they can be reversed easily with flumazenil if the need arises. Continuous positive airway pressure (CPAP) or bi-level positive airway pressure (BiPAP) may be used in some patients to obviate the need for endotracheal intubation.

25. What alternatives do I have to standard RSI?

a. **Cricothyreotomy**, a surgical airway through the cricothyroid membrane, can be done rapidly, although it often is complicated by hemorrhage and is contraindicated in children younger than 8 years.
b. **Tracheotomy** is more time consuming but is the surgical airway of choice in children and patients with tracheal injury.
c. **Fiberoptic intubation** allows visualization of the cords and trachea but is technically difficult and time consuming.
d. In **tactile intubation**, the practitioner uses his or her index and middle fingers to palpate the epiglottis and guide the tube through the cords. The patient needs to be comatose or heavily sedated, and the success rate is lower than that of RSI.
e. **Retrograde intubation** involves placing a wire through the cricoid membrane and securing it through the mouth. The wire is used as a guide to pass the endotracheal tube.
f. **Percutaneous transtracheal ventilation** involves inserting a catheter into the trachea via the cricothyroid membrane and ventilating the patient with high-pressure oxygen. This is most useful in small children because cricothyreotomy is contraindicated.

The two last techniques are used rarely and require prior training or special equipment.

26. When the patient is intubated, how do I determine if the endotracheal tube is placed correctly?

Visualizing the tube pass through the cords is helpful but fallible. Monitoring oxygen saturation and the use of capnography or colorimetric end-tidal CO_2 devices are standard-of-care adjuncts. Other findings are helpful but are not definitive: The tube fogs and clears with ventilation, breath sounds are heard in both axillae but not over the stomach, and chest expansion is noted and symmetric.

27. Doesn't the chest radiograph confirm placement in the trachea?

No. Although the chest radiograph is helpful in ruling out bronchial intubation, the tube easily can be placed in the esophagus and appear to be in the trachea proximal to the carina.

KEY POINTS: AIRWAY MANAGEMENT

1. Never paralyze a patient unless you are certain that he or she can be ventilated using a bag-valve-mask or rescue airway device.
2. Assume that all ED patients who require active airway management have a full stomach and perform RSI to minimize the risk of aspiration.
3. Objective measures like a CO_2 detector or end-tidal CO_2 must be used to confirm endotracheal intubation in every patient.
4. The King airway laryngeal mask device is an invaluable rescue tool that should be in practicing emergency physicians' armamentarium.
5. Preoxygenation with 100% O_2 for 5 minutes is a critical component of RSI intubation preparation.

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CHAPTER 4

SHOCK

Jason S. Haukoos, MD, MSc

1. Define shock.

A clinical syndrome characterized by widespread *inadequate* oxygenation and supply of nutrients to tissues and organs resulting in cellular dysfunction.

2. How common is shock?

Although the prevalence is not precisely known, it is thought that shock constitutes approximately 1% of all ED visits.

3. What is the overall mortality rate of patients who develop shock?

The mortality rate exceeds 20% for patients across all categories of shock.

4. List the five categories of shock and provide examples of each.

- ***Hypovolemic:*** Examples: Trauma; gastrointestinal bleeding; ruptured ectopic pregnancy; ruptured abdominal aortic aneurysm; and diabetic ketoacidosis.
- ***Cardiogenic:*** Examples: acute myocardial infarction; cardiomyopathy; and valvular dysfunction.
- ***Distributive:*** Examples: sepsis; anaphylaxis; and spinal cord injury.
- ***Obstructive:*** Examples: pulmonary embolism; cardiac tamponade; and tension pneumothorax.
- ***Toxic/Metabolic:*** Examples: carbon monoxide; cyanide; β-blocker; calcium channel blocker; adrenal insufficiency; and thyroid storm.

5. How do you identify a patient in shock?

The successful treatment of an acutely ill patient with a high risk of death is predicated on early recognition and treatment. A patient in shock will generally appear ill. Shock is a clinical syndrome that reflects hypoperfusion. A brief focused history and targeted physical examination will help determine if shock is present and its underlying etiology. Examples of system-based symptoms and signs include:

- Central nervous system: altered mental status.
- Cardiovascular: decreased cardiac output; tachycardia; hypotension; and weak rapid pulses.
- Pulmonary: tachypnea; and hyperpnea.
- Renal: decreased urine output.
- Skin: delayed capillary refill; cool and mottled in the setting of hypovolemic or cardiogenic shock, and warm and moist in the setting of distributive shock.

6. How should urine output be used during resuscitation of a patient in shock?

Patients in shock should have a Foley catheter placed to accurately measure urine output. Urine output is an excellent indicator of organ perfusion, assuming the patient had normal renal function at baseline. A normal urine output is $>$1.0 mL/kg/hour, a *reduced* urine output ranges from 0.5 to 1.0 mL/kg/hour, and a *severely reduced* urine output is $<$0.5 mL/kg/hour. During resuscitation, targeted therapy should additionally focus on improving or normalizing urine output.

7. Describe compensated and decompensated shock.

Shock initiates a sequence of stress responses intended to preserve perfusion to vital organs. Compensated shock occurs soon after the onset of shock and is marked by the maintenance of tissue perfusion pressures. Such patients typically have evidence of a stress response (e.g., tachycardia and tachypnea) but also have a normal or high blood pressure and normal or mildly elevated serum lactate concentrations. If left untreated, compensated shock will progress to decompensated shock, which is characterized by profound global tissue hypoperfusion, elevated serum lactate concentration, and hypotension.

8. What is the initial management of a patient who presents in shock?

Management of patients in shock begins with the ABCs (i.e., airway, breathing, and circulation). Due to poor delivery and uptake of oxygen, all patients should be placed on either 15 L of oxygen by nonrebreather mask or intubated. Simultaneously, all patients should have large-bore intravenous access and be placed on a cardiac monitor.

9. Define oxygen delivery.

$$DO_2 = CaO_2 \times CO$$

DO_2, oxygen delivery
CaO_2, arterial oxygen concentration
CO, cardiac output

$$CaO_2 = (1.34 \times Hgb \times SaO_2) + (0.003 \times PaO_2)$$

Hgb, hemoglobin
SaO_2, arterial oxygen saturation
PaO_2, arterial oxygen partial pressure

Oxygen delivery is the product of the cardiac output and arterial oxygen concentration. Arterial oxygen concentration is defined by the hemoglobin level, the arterial oxygen saturation, and the arterial oxygen partial pressure. Maximizing cardiac output, hemoglobin, arterial oxygen saturation, and the arterial partial pressure of oxygen will maximize oxygen delivery.

10. How useful are vital signs in assessing and treating someone in shock?

Vital signs are vital. Heart rate, respiratory rate, blood pressure, and pulse oximetry should be monitored closely in patients in shock. Physiologic compensation and decompensation (see Question 7) are commonly reflected in a patient's vital signs. Additionally, normalization of abnormal vital signs is one indicator of a patient's response to resuscitation.

11. If a patient has normal vital signs, should I be reassured?

No. A patient's heart rate and blood pressure may be normal in the setting of severe illness. In the setting of shock, heart rate and blood pressure correlate poorly with cardiac output and often underestimate the severity of systemic hypoperfusion.

12. Are orthostatic vital signs a sensitive indicator of hypovolemia? What determines a positive orthostatic test?

To know what is abnormal, you first must know what is normal. Studies on healthy euvolemic people showed an average increase in pulse of 13 to 18 beats per minute with a large standard deviation. A pulse increase of 20 beats per minute as a determinant for hypovolemia is nonspecific because many normal individuals fall within this range. However, an increase of 30 beats per minute in heart rate is more specific. A 20% volume loss is required to produce this change in heart rate, making this an insensitive test at best. The development of symptoms (e.g., lightheadedness on standing) does not occur in healthy euvolemic individuals upon standing and should be considered abnormal. Patients in shock should not be allowed to stand to assess changes in vital signs.

13. Are there other signs that are helpful in assessing an acutely ill patient?
Yes. Besides vital signs, components of the physical examination (e.g., level of consciousness, capillary refill, and urinary output), you should pay close attention to the patient's serum lactate, central venous pressure (CVP), and central venous ($ScvO_2$) or mixed venous (SvO_2) oxygen saturation.

14. How should I use and interpret a serum lactate?
Serum lactate is a commonly used marker to assess the extent of systemic hypoperfusion and the degree to which a patient may be responding to resuscitation. In fact, it is an early marker of systemic hypoperfusion and is often elevated prior to overt changes in a patient's vital signs. Therefore, liberal use of this marker may help identify patients earlier in their disease processes. A serum lactate concentration >4 mEq/L is associated with the highest mortality rates.

15. What is the Lactate Clearance Index, and how can it be used during resuscitation of a patient in shock?
The Lactate Clearance Index refers to measuring serum lactate concentrations at two or more times during the course of the resuscitation. If after 1 hour of the beginning of resuscitation efforts, the serum lactate concentration has not decreased by 50%, additional steps should be undertaken to improve systemic perfusion.

16. What is a normal CVP and how is it measured?
A normal CVP ranges from 5 to 10 cm H_2O. Central venous pressure is measured by attaching an electronic pressure transducer or a water manometer to the end of an intravenous line placed into the central venous system. The zero reference point for measuring a CVP is at the point that bisects the fourth intercostals space and the midaxillary line in a supine patient, corresponding to the position of the right atrium.

17. How is CVP used during resuscitation of a patient in shock?
The guiding principal for using the CVP is to normalize or supranormalize its value. The target CVP should range from 10 to 15 cm H_2O to maximize cardiac preload. In many shock states, the heart becomes stiff and its function depressed. A supranormal CVP thus allows for improved cardiac filling.

18. What is venous oxygen saturation, and what is the difference between a $ScvO_2$ and SvO_2?
Venous oxygen saturation provides a measure of tissue oxygenation (i.e., the balance between oxygen supply [DO_2] and demand [VO_2]). SvO_2 is measured using a pulmonary artery catheter and includes deoxygenated blood returning to the heart from the body, as well as deoxygenated blood from the heart via the coronary sinus. It normally ranges between 65% and 75%. $ScvO_2$, on the other hand, is measured using a central venous catheter and consistently overestimates (albeit to a small degree) venous oxygen saturation because it does not include sampling of blood mixed with blood returning from the heart.

19. How do I use a $ScvO_2$ or a SvO_2 during resuscitation?
An $ScvO_2$ <65% suggests decreased oxygen supply or increased demand. In response, attempt to improve oxygen delivery by increasing arterial oxygen saturation (SaO_2) and/or arterial oxygen partial pressure (PaO_2) via oxygen supplementation; hemoglobin concentration via transfusion; and/or cardiac output via inotropic support.

20. What is early goal-directed therapy?
Goal-directed therapy refers to the practice of resuscitating patients to defined physiological endpoints (e.g., mean arterial pressure, CVP, urine output, serum lactate concentration, cardiac output, hemoglobin level, and SvO_2), indicating that systemic tissue perfusion and vital organ function have been restored. This has only been rigorously studied in patients with sepsis; however, it has recently been evaluated in postcardiac arrest. It is likely that early

goal-directed therapies will be evaluated in other forms of shock in the future, thus guiding emergency physicians' abilities to improve resuscitation endpoints and survival.

21. List the primary resuscitation goals in patients suffering from shock.

- Maximize oxygenation.
- Establish adequate ventilation.
- Improve hemodynamic dysfunction.
- Treat the underlying etiology.

22. What is the Trendelenburg position? What purpose(s) does it serve?

Trendelenburg refers to the patient placed in a supine, approximately 45-degree, head-down position. The purposes of this position have been reported to include improving blood pressure, redistributing circulating blood volume, placing central lines, and improving the sensitivity of abdominal ultrasound for intra-abdominal fluid. Although commonly used for the purpose of improving hemodynamic parameters, several studies have not demonstrated its utility in significantly improving blood pressure or redistribution of blood volume.

23. Define systemic inflammatory response syndrome (SIRS).

SIRS is defined by two or more of the following:

- Temperature $>$38°C or 36°C.
- Heart rate $>$90 beats per minute.
- Respiratory rate $>$20 breaths per minute or partial pressure of carbon dioxide ($PaCO_2$) $<$32 mm Hg.
- Serum white blood cell count $>$12,000 mm^3 or $<$4,000 mm^3 or 10% band forms.

It is important to note that this definition, although standardized, is not specific for defining serious illness. Although most commonly related to sepsis, SIRS may result from a variety of noninfectious insults, including trauma, burns, pancreatitis, and overdose.

24. Define sepsis, severe sepsis, and septic shock, and discuss their specific therapies.

See Chapter 47.

25. How do I treat cardiogenic shock?

The treatment of cardiogenic shock should focus on improving myocardial contractility and overall pump function. Provide oxygen and ventilatory support, including the judicious use of noninvasive positive-pressure ventilation when pulmonary edema is present. Initiate inotropic support using dobutamine or dopamine, and identify the etiology and administer specific treatment (e.g., thrombolysis or percutaneous coronary intervention in the setting of acute coronary syndrome). Consider intra-aortic balloon counterpulsation or cardiopulmonary bypass for patients with refractory shock.

26. Explain the mechanism of dobutamine.

Dobutamine is a synthetic catecholamine with primarily β_1-receptor (cardiac stimulation) and mild β_2-receptor (vasodilation) agonism.

27. Explain the mechanism of dopamine.

Dopamine is an endogenous catecholamine that when administered intravenously produces a dose-dependent activation of adrenergic and dopaminergic receptors. When given in low doses (e.g., 5 μg/kg/min), dopamine preferentially activates dopaminergic receptors, producing vasodilatation in renal, mesenteric, and cerebral circulations. When given in intermediate doses (e.g., 5 to 10 μg/kg/min), dopamine stimulates β-receptors, thus increasing cardiac output. When given in high doses (e.g., $>$10 μg/kg/min), dopamine activates α-receptors, producing a dose-dependent increase in systemic vascular resistance. It is important to note that dopamine has modest inotropic characteristics when compared to dobutamine and that tachyphylaxis may result from its use if used for a prolonged period of time.

28. How do I treat shock due to anaphylaxis?
See Chapter 18.

29. Explain the mechanism of epinephrine.
Similar to dopamine, epinephrine is a primary β-receptor agonist at low doses and an α-receptor agonist at high doses. However, epinephrine is significantly more potent than dopamine.

30. How do I treat shock due to pulmonary embolism (PE)?
Massive PE causes shock by reducing the cross-sectional area of the pulmonary outflow tract, thus increasing right-sided heart pressures, reducing blood flow to the left side of the heart, all of which results in a hemodynamic compromised state. Treatment centers on provision of oxygenation and ventilation, hemodynamic support using crystalloids and vasopressors, as necessary, and use of thrombolytics or surgical embolectomy in the setting of refractory shock.

31. How do I treat shock due to cardiac tamponade?
As always, ensure adequate oxygenation and ventilation. Similar to other forms of obstructive shock (e.g., PE), administration of intravenous fluids may help overcome increased cardiac filling pressures. However, the principal therapies for cardiac tamponade are pericardiocentesis or pericardiotomy.

32. What is neurogenic shock and how is it treated?
Neurogenic shock is a form of distributive shock resulting from spinal cord injury in which central or peripheral sympathetic tone is lost. Such patients are commonly hypotensive with either a normal or low heart rate. Administer intravenous fluids to normalize intravascular volume. If hypotension persists, several vasopressor options exist, although intravenous phenylephrine (0.15 to 0.75 μg/kg/min) is considered the classic first-line agent.

33. Explain the mechanism of phenylephrine.
Phenylephrine is a pure and potent α-agonist. Administration of this agent can induce a reflex bradycardia, resulting in decreased cardiac output.

KEY POINTS: SHOCK

1. Shock is defined as a clinical syndrome characterized by widespread *inadequate* oxygenation and supply of nutrients to tissues and organs, resulting in cellular dysfunction.
2. The five categories of shock are hypovolemic, cardiogenic, distributive, obstructive, and toxic/metabolic.
3. Serum lactate is a commonly used marker to assess the extent of systemic hypoperfusion and the response to resuscitation.
4. The primary resuscitation goals in patients suffering from shock are to maximize oxygenation, establish adequate ventilation, improve hemodynamic distribution, and treat the underlying cause.

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CHAPTER 5

EMERGENCY ULTRASOUND

John L. Kendall, MD, FACEP, and Catherine Erickson, MD

1. What is ED ultrasound all about?

An ultrasound probe in the hands of the clinician has historically been considered the stethoscope of the 21st century. Extending beyond this definition, the technology of ultrasound is increasingly considered to be an integral part of the evaluation and management of ED patients.

2. Why should ultrasound be performed in the ED?

Focused ultrasound examinations performed by ED physicians allow for more timely, less invasive, and safer evaluations of patients. Ectopic pregnancy and biliary colic may be evaluated rapidly, intra-abdominal traumatic hemorrhage may be diagnosed without the invasiveness of diagnostic peritoneal lavage or the delay of a computed tomography (CT) scan, and patients with major trauma or suspected abdominal aortic aneurysm (AAA) may be evaluated quickly in the safety of the ED.

3. How does emergency ultrasound differ from ultrasound performed by the radiology department?

Emergency ultrasound is meant to be a focused, goal-directed examination. Specific findings, such as the presence of intraperitoneal fluid in blunt abdominal trauma; intrauterine pregnancy (IUP) in suspected ectopic pregnancy; gallstones, wall thickness, or sonographic Murphy's sign in right upper quadrant pain; aortic dilation in suspected AAA; and pericardial fluid in patients with possible pericardial tamponade, are used to guide patient care. In contrast, a radiologist-performed ultrasound is more comprehensive in that all structures viewed are evaluated.

4. How about some basic ultrasonography physics?

Ultrasound images are generated as sound waves at various frequencies (MHz) that reflect off tissue interfaces. The higher the ultrasound frequency, the greater the resolution but at the expense of reduced tissue penetration. Dense tissues, such as bone or gallstones, appear bright because most of the ultrasound energy is absorbed or reflected. Solid organs, such as the liver or spleen, show a gray scale of tissue architecture. All of the ultrasound energy passes through fluid or blood, leaving a black (anechoic) area on the screen. Ultrasound energy does not propagate well through air. Thus, lung, hollow viscous structures, and air trapped within soft tissues are difficult to visualize. In general, abdominal and cardiac examinations utilize a 3.5- to 5-MHz probe, transvaginal ultrasound examinations a 7.5- to 10-MHz probe, and vascular studies a 10- to 12-MHz specialized probe.

5. Describe the basics of the trauma ultrasound examination.

The trauma ultrasound examination (also known as the focused assessment with sonography for trauma [FAST]) is done rapidly at the patient's bedside during the secondary survey. The primary goal is to detect free intraperitoneal fluid, which appears as anechoic areas within the peritoneal cavity. Sites in the abdomen that are evaluated are the potential spaces that occur at dependent sites within the peritoneal cavity. These include the hepatorenal recess or Morison's pouch (Fig. 5-1), splenorenal recess, retrovesicular recess (pouch of Douglas in females), and both pericolic gutters. Oblique

views of the right and left chest are obtained to search for hemothorax, and a subxiphoid or left parasternal cardiac image is obtained to locate pericardial effusion (Fig. 5-2).

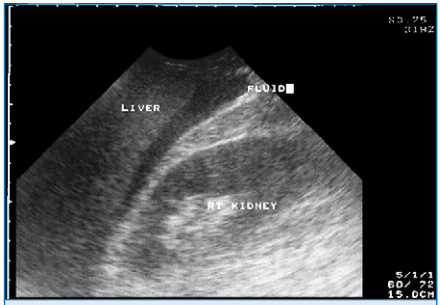

Figure 5-1. View of Morison's pouch showing intraperitoneal fluid.

6. Where is the best place to look for intraperitoneal fluid?

The sonographic examination should include all sites previously mentioned. The sensitivity increases from approximately 60% if one site is viewed to almost 90% if all are used.

7. How does ultrasound compare with traditional means of evaluating the traumatic abdomen?

Physical examination is only 50% to 60% sensitive for detecting abdominal injuries after blunt trauma. Diagnostic peritoneal lavage is 95% sensitive but is not specific, resulting in unnecessary laparotomies. CT is sensitive for detecting abdominal injuries (>95%) but is costly, is time consuming, and requires the patient to leave the ED. Prospective studies of ultrasound showed an 83% to 90% sensitivity for the detection of hemoperitoneum, with sensitivity approaching 100% in patients who were hypotensive from an abdominal source. The accuracy of ultrasound to detect the underlying parenchymal lesion varies widely.

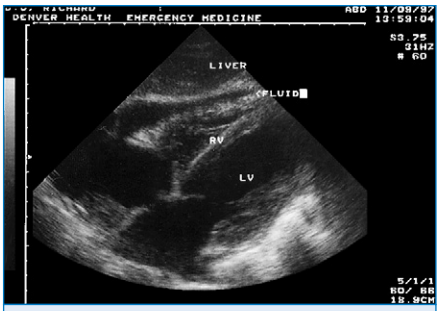

Figure 5-2. Subxiphoid cardiac view shows a pericardial effusion.

8. How should I use ultrasound in my evaluation of blunt trauma patients?

Consider patient scenarios based on vital signs and ultrasound findings:

a. Stable vital signs, negative ultrasound
b. Stable vital signs, positive ultrasound
c. Unstable vital signs, negative ultrasound
d. Unstable vital signs, positive ultrasound

KEY POINTS: PRIMARY CHARACTERISTICS OF THE EMERGENCY ULTRASOUND EXAMINATION

1. Performed for a defined indication
2. Focused, not complete
3. Easily learned and quickly performed
4. Directed toward one or two easily recognizable findings
5. Directly impacts clinical decision making
6. Performed at the bedside

Patients with stable vital signs and a negative ultrasound who have no other significant injuries, have normal mental status, and are not intoxicated can be managed with observation, serial physical examinations, and serial ultrasound studies. Patients with stable vital signs and a positive ultrasound warrant an abdominal CT scan. If the vital signs are unstable and ultrasound is negative or indeterminate, a bedside diagnostic peritoneal lavage can be done and other etiologies of hypotension considered. If the vital signs are unstable and the ultrasound is positive for free fluid, the patient should go directly to laparotomy.

9. Can I tell how much intraperitoneal fluid is present based on the ultrasound image?

No, conflicting data exist. No study has yet shown an accurate means of quantifying the amount of intraperitoneal fluid that is present based on its sonographic appearance.

10. What are some of the pitfalls I may encounter during a trauma ultrasound examination of the abdomen?

Although relatively rare, one of the more concerning aspects of emergency ultrasound is the false-negative study. In terms of abdominal trauma, clotted blood is the finding that mimics a negative study the closest. An example of clotted blood found in Morison's pouch is shown in Fig. 5-3. It initially was interpreted to be liver parenchyma because of a similar echogenic pattern. False-positive findings that simulate hemoperitoneum can occur in the setting of ascites, urine from a ruptured bladder, bowel contents from bowel perforation, perinephric fat, and fluid-filled stomach or bowel.

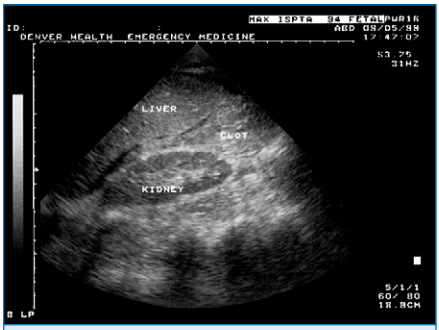

Figure 5-3. Clotted blood in Morison's pouch.

11. What is the sonographic appearance of the gallbladder and related structures?

The gallbladder is cystic, so the sonographic appearance is a pearlike structure that is anechoic. Surrounding this anechoic area is a ring of midechogenicity that corresponds to the gallbladder wall. Normally, it is less than 4 mm wide, but can be thicker immediately after eating or if in edematous states, such as liver failure, ascites, congestive heart failure, renal disease, or AIDS. Stones are typically circular in nature, can be of any size, and are bright, or hyperechoic, on their proximal side. Ultrasound does not penetrate stones well, so distal to the stone there is a shadow (Fig. 5-4). This also is called the *headlight sign*, signifying the presence of a calcified gallstone. Sludge is a collection of the precipitants of bile that layers within the gallbladder and appears sonographically as mildly echogenic material without any shadowing.

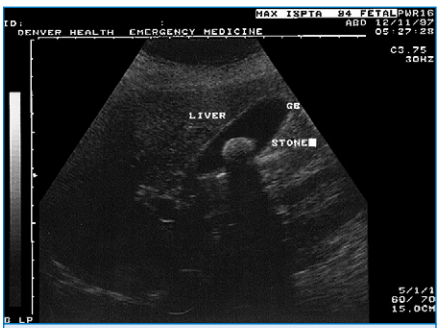

Figure 5-4. Long-axis view of the gallbladder shows a gallstone. The gallstone is represented by an echogenic proximal surface and distal attenuation shadow.

12. What findings are suggestive of acute cholecystitis?

The primary findings of the emergency gallbladder ultrasound are the presence of gallstones and a sonographic Murphy's sign (defined as maximal tenderness over an ultrasound-detected gallbladder). The presence of these primary findings has a 92% positive predictive value and a 95% negative predictive value for the presence of cholecystitis. Other findings, such as wall thickening (>4 mm), ductal dilation (>6 mm), pericholecystic fluid, sludge, and an emphysematous gallbladder, are considered to be secondary findings and are less reliably seen by emergency sonographers. Ultrasound is insensitive at detecting choledocholithiasis.

13. What are the indications for pelvic ultrasonography in the ED?

Ultrasonography is the imaging study of choice for evaluating abdominal pain or bleeding in pregnant patients in the first or second trimester. The goal of ED ultrasound is to establish the presence of an IUP, so as to effectively rule out an ectopic pregnancy. Ectopic pregnancy is the second leading cause overall of maternal mortality and the number one cause of maternal mortality during the first trimester.

14. How early can an IUP be detected using ultrasound? What value of β-human chorionic gonadotropin (HCG) does this correspond to?

An IUP may be detectable as early as 4.5 weeks by transvaginal ultrasound at a β-HCG level of 1,000 to 2,000 mIU/mL (6 weeks or greater with a β-HCG of 5,000 mIU/mL using transabdominal ultrasound). The **discriminatory zone**, or level of β-HCG at which one would expect to see evidence of an IUP, depends on the institution where the patient is being seen. A gestational sac is seen at approximately 4-5 weeks gestational age and cardiac activity can be measured as early as 6 weeks gestational age.

15. How sensitive is ultrasound for the evaluation of ectopic pregnancy?

Several studies have shown that 75% to 80% of patients have a diagnostic ultrasound (i.e., either an IUP or a demonstrable ectopic pregnancy). The problem is that in the remaining 20% of patients with nondiagnostic ultrasounds, nearly one fourth have ectopic pregnancies. This increase in ectopic pregnancy among patients with nondiagnostic ultrasound suggests that this group should have thorough evaluation, including an obstetric-gynecologic consultation in the ED.

16. Describe the pitfalls in pelvic ultrasonography.

For emergency physicians, the goal of pelvic ultrasonography is to determine whether an IUP is present. It is not clear how well emergency physicians evaluate the adnexa, pelvic free fluid, or ovaries. Cornual pregnancies may be mistaken for an IUP, with an attendant risk of rupture and hemorrhage. The question of heterotopic pregnancies (i.e., simultaneous IUP and ectopic pregnancy) must be considered. In populations without risk factors for ectopic pregnancy, the risk of a heterotopic gestation is approximately 1 in 30,000 pregnancies. The incidence increases markedly, however, in patients with preexisting pelvic inflammatory disease or scarring and is greatest for patients receiving medical fertility assistance, in whom the incidence is estimated to be 1 in 100 to 1 in 400 pregnancies. A pseudosac can be seen in 20% of ectopic pregnancies. It is formed in response to the β-HCG produced by the abnormal pregnancy. It consists of a single-ringed structure in the endometrial cavity, and it can be mistaken for a true gestational sac, which consists of **two** concentric rings.

17. What other abdominal structures can be evaluated by emergency ultrasound?

Evaluation of the abdominal aorta can be useful in elderly patients who present with a pulsatile abdominal mass, nontraumatic abdominal pain or flank pain, hypotension of unknown cause, or unexplained pulseless electrical activity. AAA is manifested by aortic diameter greater than 3 cm with most symptomatic aneurysms being greater than 5 cm (Fig. 5-5). Studies by emergency physicians showed sensitivity of 100% and a specificity of 98% for the detection of AAA. Studies showed a 90% correlation of ultrasound-determined aortic diameter to pathologic specimens.

18. What is the significance of increased aortic diameter?

Longitudinal studies have shown that patients with AAA have an increase in aortic diameter of approximately 0.5 cm/year. Patients with an aortic diameter of greater than 5 cm have a 25% chance of rupture within 5 years, with larger aneurysms having a greater chance of rupture. Aneurysms that rupture have a mortality of greater than 80%, so ultrasound is an important tool in the detection of AAA.

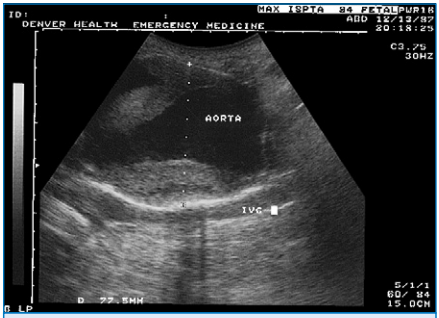

Figure 5-5. Long-axis view of a 7.75-cm diameter abdominal aortic aneurysm.

19. Describe the uses of cardiac ultrasonography in the ED.

These are primary indications for cardiac ultrasonography in the ED (see Table 5-1):

a. It may be used during the trauma examination to detect pericardial effusions in patients thought to have mechanisms of injury or clinical presentations consistent with pericardial tamponade or cardiac rupture.
b. It may be used for detection of nontraumatic pericardial effusions (i.e., malignancy, uremic, rheumatologic).
c. Another important indication includes the evaluation of patients presenting in cardiac arrest. Contractility can be assessed in patients presenting in cardiac arrest when there is a question of pulseless electrical activity. When there is no evidence of cardiac contractility and other reversible causes of pulseless electrical activity have been ruled out, strong consideration should be given to terminating the resuscitation.
d. Lastly, emergent echocardiography is starting to be used for detecting central venous volume status.

20. What is the role of ultrasound in the evaluation of patients with suspected renal colic?

By itself, ultrasound is only 64% to 75% sensitive for the identification of renal calculi and even less sensitive for the evaluation of acute hydronephrosis. Studies that combined kidney, ureter, and bladder radiographs and ultrasound in well-hydrated patients showed improved ability to identify kidney stones and hydronephrosis. In the end, a noncontrast CT is a far superior imaging tool for the patients presenting with suspected renal colic. If hydronephrosis without an etiology is seen on ED ultrasound, further imaging should be pursued.

21. How is lower extremity venous ultrasound performed in the ED to diagnose deep venous thrombosis (DVT)?

A linear transducer with a high frequency range is used. The examination should start proximally with the vein in a transverse plane just below the inguinal ligament where the common femoral vein can be visualized. Compression followed by no compression should occur in 1-cm increments until the femoral vein dives into the adductor canal. Next the popliteal region is visualized again in 1-cm increments. An examination is considered to be negative when complete compression occurs to the point that the anterior and posterior walls of the vein touch. In a positive study, the vessel walls will not touch; the clot echogenicity can vary greatly from echogenic to non-echogenic. Recent studies show the sensitivity and specificity of ED DVT studies to range from 70% to 95% and 89% to 95%, respectively. For accurate diagnosis of DVT, additional components, such as pretest probability and the D-dimer assay, may need to be considered.

TABLE 5-1. EMERGENCY ULTRASOUND CORE APPLICATION

Established Applications	Newer Applications
Abdominal	**Deep venous thrombosis evaluation**
Aortic	
Biliary	**Thoracic**
Urinary tract	Pleural effusion
	Pneumothorax
Pelvic	
Intrauterine pregnancy	**Musculoskeletal**
	Abscess incision and drainage
Trauma	Fracture evaluation
Focused abdominal sonography for trauma	
	Ocular
Cardiac	Retinal detachment
Emergent echocardiography	Vitreous hemorrhage
Pericardial effusion	
Tamponade	**Procedural**
Contractility	Pericardiocentesis
	Thoracentesis
Procedural	Foreign body and detection and removal
Central venous access	Arthocentesis
	Pacemaker placement

From American College of Emergency Physicians. ACEP emergency ultrasound guidelines—2008. *Ann Emerg Med* 53(4):550–570, 2009.

22. What are some future applications for emergency ultrasound?

Uses for emergency ultrasound continue to rapidly expand. This is clearly demonstrated in the 2008 American College of Emergency Physician's clinical practice guidelines for ultrasound that expands upon core applications. For instance, one of the fastest-growing applications is to guide invasive procedures. This is not confined with vascular access but also other procedures, such as localization and drainage of abscesses, nerve blocks, lumbar puncture, placement of an intravenous pacer wire, and suprapubic bladder aspiration, to name a few. Emergency ultrasound is tremendously useful in the evaluation of patients in cardiac arrest, with undifferentiated hypotension or shock, suspected DVT, testicular torsion, or an appendicitis diagnosis in the pediatric population.

23. Has the political environment changed with respect to emergency physicians using ultrasound?

Yes, there have been many changes in recent years. Ultrasound use by emergency physicians has gone from a novelty experience to something that is tested on emergency medicine specialty boards and the national inservice examination, is recommended by the American College of Emergency Physicians to be taught in all residency programs early in training, and is widely used in clinical practice. As such, the question is no longer whether or not ultrasound will be used by emergency physicians, but rather how it can be used for optimal care of ED patients.

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GERIATRIC EMERGENCY MEDICINE

Kenneth C. Jackimczyk, MD, FACEP

CHAPTER 6

1. Why dedicate a chapter to geriatric emergency medicine?

Elderly persons are a rapidly growing segment of the population. In 2004, approximately 36 million people in the United States were older than age 65. By the year 2030, this number is expected to double, and over the next 20 years the number of individuals older than age 85 will grow three times faster than the general population. Patients older than 65 years account for 15% of all ED visits and more than 40% of in-patient admissions. Elderly patients stay in the ED longer and require more diagnostic studies than younger patients.

Geriatric patients have unique medical and social characteristics. They often have multiple comorbid conditions that complicate evaluation of the presenting complaint. Diseases often present atypically in geriatric patients. These patients often have reduced functional reserves, which make a careful assessment of the patient's psychosocial environment essential prior to making decisions about disposition.

2. What important physiologic changes occur with aging?

- Musculoskeletal: Loss of muscle strength, impaired mobility, and decreased bone mass.
- Cardiovascular: Decreased cardiac output and increased systolic blood pressure.
- Pulmonary: Decreased vital capacity and decreased functional reserve.
- Head, eye, ear, nose, and throat (HEENT): Impaired hearing and vision.
- Renal: Decreased renal blood flow and glomerular filtration rate (GFR).
- Immune system: Decreased cellular immunity and decrease antibody titers.
- Dermatologic: Impaired thermoregulation and atrophy of skin.

KEY POINTS: PRINCIPLES OF GERIATRIC EMERGENCY MEDICINE ✓

1. Vital signs can be normal in the elderly patient with serious illness.
2. Adverse medication reactions are common in elderly patients.
3. Atypical presentations of serious diseases are more common.

3. How can prehospital personnel facilitate the care of elderly patients?

Elderly patients account for more than one third of emergency medical services (EMS) transports to the ED. Prehospital providers can obtain information from family or health care workers at the scene regarding the patient's social and physical environment, his or her baseline functional and mental status, and the reason for EMS activation. Ambulance personnel should obtain lists of medications the patient is using and any documentation regarding living wills or advance directives.

4. What is the significance of fever in elderly patients?

Elderly patients presenting with fever have a significant risk of serious bacterial infection. Conversely, because of their blunted fever response, bacteremic elderly patients may not be febrile. Don't be lulled into complacency by a lack of fever in an ill-appearing elderly patient because nearly half of patients with serious infections will not have a fever.

5. What are the four types of elder abuse?

a. **Physical abuse:** Nonaccidental force that results in bodily injury or pain (e.g., hitting, biting, slapping, sexual assault, burns, or unreasonable restraint [physical, chemical]).
b. **Psychological abuse:** Threats made with the intent of causing emotional pain or injury.
c. **Exploitation:** Caretaker use of the resources of an elder for monetary or personal profit.
d. **Neglect:** Failure of the caretaker to provide the services necessary to avoid physical harm, mental anguish, or mental illness. This neglect can be *intentional* or *unintentional.*

6. What red flags in the history should alert the physician to the possibility of elder abuse?

- Delay in presentation with injury.
- Vague or implausible explanation for injury.
- Repetitive injuries.
- Missed appointments and noncompliance with medications.
- No caregiver accompanying an impaired patient to the ED.

7. What red flags in the physical examination should alert the physician to the possibility of elder abuse?

- Subdued, oversedated, or withdrawn behavior.
- Unkempt appearance or poor nutrition.
- Multiple or unexplained bruises, abrasions, or lacerations.
- Burns, bites, or pressure sores.
- Occult fracture.

8. Why is it important to know the elderly patient's current medications?

Adverse drug-related events are a significant cause of morbidity in elderly patients and are the most common cause of iatrogenic illness in elderly patients. The average elderly person uses more than four prescription drugs and more than two over-the-counter medications daily. These numbers are even higher for institutionalized patients. Adverse reactions to medications are directly proportional to the number of medications being taken.

9. What presenting complaints should lead me to suspect that the patient is experiencing an adverse reaction to medications?

- Altered level of consciousness.
- Weakness.
- Dizziness.
- Syncope.

10. Don't elderly patients always have abnormal laboratory values?

No. Most laboratory values in geriatric patients do not require different reference ranges from traditional adult values, and the fact that the patient is elderly should not be used to justify abnormal laboratory values. There are, however, some exceptions in patients older than age 65:

- Elevated serum alkaline phosphatase (may be 2.5 times greater than the normal).
- Elevated fasting blood glucose (135 to 150 mg/dL).
- Elevated erythrocyte sedimentation rate (40 mm/hour).
- Decreased hemoglobin (11.0 g/dL in women or 11.5 g/dL in men).
- Elevated blood urea nitrogen (28 to 35 mg/dL).

11. Should I worry if a geriatric trauma victim has normal vital signs with apparently minor injuries?

Yes. Vital signs in the elderly may remain normal until acute deterioration occurs. Geriatric patients have a blunted tachycardic response to injury. A "normal" blood pressure of 120/80 mm Hg may represent relative hypotension in the elderly hypertensive patient. The elderly patient's diminished cardiovascular reserve, increased susceptibility to fractures, and the presence of comorbid conditions such as coronary artery disease can result in significant morbidity, even

with injuries that appear to be minor. Elderly patients have the highest trauma mortality rate of any age group, and normal vital signs or a low injury severity score should never put the physician at ease.

12. Which presentations in geriatric trauma are associated with an extremely high mortality rate?

- Automobile-pedestrian accidents (>50% mortality).
- Presenting systolic blood pressure less than 130 mm Hg.
- Acidosis (pH <7.35).
- Multiple fractures.
- Head injury (67% of unconscious elderly trauma patients die).
- Pelvic fractures.

13. Aren't falls a fact of life in elderly patients?

No. Any fall is a serious threat to the independence of the elderly patient. A fall should be considered a *significant symptom* that warrants a full ED evaluation because 10% to 15% of geriatric falls result in serious injury, and 50% of patients who require hospitalization die within 1 year of their fall.

14. What is different about evaluating the elderly patient who falls?

It is essential to assess the *cause* of the fall as well as the *injuries* that have occurred. Falls may result from either physiologic or environmental factors. Physiologic factors include muscle weakness, gait and balance disorders, visual impairment, postural hypotension, and syncope. Environmental disorders include dark hallways, loose rugs, and low-lying tables. The evaluation for syncope should focus on acute cardiovascular causes (e.g., dysrhythmias, myocardial infarction), neurologic events, hypovolemia, and adverse reactions to medications. Use of psychotropic medications, such as benzodiazepines and other sedatives, are associated with an increased risk of falls in the elderly.

15. Do emergency physicians have a role in prevention of recurrent falls in the elderly?

Yes. Several studies have shown that risk factors can be identified in the ED (e.g., muscle weakness, arthritis, cognitive impairment) and reported to the patient's primary physician so that interventions can be preformed. Psychotropic drugs may be discontinued or prescribed in reduced doses. Educating the patient on simple changes that can be made in the home to reduce falls has also been shown to be helpful.

KEY POINTS: FALLS IN GERIATRIC PATIENTS

1. Falls in elderly patients are a serious problem.
2. Check for both physiologic and environmental causes of the fall.
3. Consider syncope as the cause of the fall.

16. Can procedural sedation be performed safely in the geriatric patient?

Yes, but the physician must be aware of the altered pharmacokinetics and pharmacodynamics in elderly patients. As the body ages, there is a reduction in lean body mass and total body water and an increase in total body fat. There also is a decrease in renal and hepatic blood flow. This has an effect on the metabolism and the distribution of medications administered to an elderly patient. Elderly patients have increased central nervous system sensitivity to analgesic and sedative medications. Remember: *start low and go slow.*

17. Should I be concerned about atypical presentations of acute myocardial infarction (AMI) in elderly patients?

Yes. AMI is the leading cause of death in elderly patients and *atypical* presentations are actually *typical* for AMI in the elderly. Nearly 40% of elderly patients diagnosed with AMI did not complain of chest pain on presentation, and similarly, 50% had no evidence of ischemia or infarct on their presenting electrocardiogram (ECG). For these reasons, it is imperative that the ED physician know the atypical presentations of AMI in elderly patients. The mnemonic **GRANDFATHERS** refers to atypical presentations of AMI in elderly patients:

General malaise
Refers to a gastrointestinal complaint
Altered mental status
Neurologic deficits
Dyspnea
Falls or **F**lu symptoms
Atypical chest pain
Trouble walking
Hypotension
Exhaustion
Reverse in functional status
Syncope or presyncope

18. Should I resuscitate the elderly patient in cardiac arrest?

Yes. Resuscitation studies document no difference in the percentage of successful outcomes across the age spectrum, and elderly patients who survive are no more likely to sustain irreversible brain injury than younger patients. Unless there is a well-defined advance directive, there should be no discrimination based on age in resuscitating elderly patients in cardiac arrest.

19. How does my approach to acute abdominal pain change in elderly patients?

Any complaint of abdominal pain in the elderly should be taken seriously. Elderly patients have decreased pain perception and are more likely to have normal vital signs in the face of significant intra-abdominal pathology. Older patients are also less likely to demonstrate peritoneal findings because they lack well-developed abdominal musculature. These factors cause delays in diagnosis, higher perforation rates, and higher mortality rates in abdominal diseases in the elderly. Keep a broad differential diagnosis and consider the common disorders such as appendicitis and cholecystitis but also remember diseases specific to older patients, such as diverticulitis, volvulus, mesenteric ischemia, abdominal aortic aneurysm, and carcinomas. CT scanning should be used liberally in older patients suspected of having a surgical process, but do not delay surgical consultation waiting for laboratory results or imaging studies.

20. Which is more serious, dementia or delirium?

Delirium. Delirium is considered a medical emergency. The elderly patient may already have dementia, but a sudden change in mental status may represent an acute organic process, such as infection or an adverse reaction to a medication. To attribute a change in mental status to worsening dementia, without searching for an organic cause, is a serious error.

21. How do I differentiate between delirium and dementia?

See Table 6-1.

22. What special concerns are there in discharging elderly patients?

- **Cognitive function:** Does the patient understand the discharge instructions? Can the patient still live independently and self-administer medications?
- **Physical function:** Can the patient perform the activities of daily living (bathing, dressing, feeding)? Does the patient require an assistance device such as a walker or wheelchair?
- **Physical environment:** Can the patient safely return with his or her current cognitive or functional status? Did the current environment contribute to the ED presentation?

TABLE 6-1. DIFFERENTIATION BETWEEN DELIRIUM AND DEMENTIA

Delirium	Dementia
Acute in onset	Insidious in onset
Decreased level of consciousness	Clear consciousness
Waxes and wanes	Progressive decline
Reversible cause	Usually irreversible cause
Irregular sleep-wake pattern	Regular sleep-wake pattern

- **Social environment:** Will the caregiver or spouse be able to care for the patient? Is health care supervision available?
- **Resources:** Is a telephone available? Is money available for medicine or follow-up appointments? Is there transportation to get to a follow-up appointment?

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CHAPTER 7

SAFETY IN EMERGENCY MEDICINE

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1. The ED has been described as a *natural laboratory* for the study of safety. What makes it so?

The conditions of work in EDs typically detract from optimal human performance. These might be divided into two types: Failure-producing conditions (FPCs) and violation-producing conditions (VPCs). The former lead to what in folk wisdom are called *human errors*, and the latter are conditions that lead people to deviate from formal procedures.

2. Define some basic safety terms.

- An *error* is a term of historical interest only. It was originally defined as the failure of a planned action to be completed as intended (i.e., error of execution) or the use of a wrong plan to achieve an aim (i.e., error of planning). The concept of error has not proven useful in creating safer systems of care, and in fact may be harmful, because of its slipperiness and the pejorative baggage that it carries.
- *Active* faults are those whose effects are seen immediately. They are most often associated with those who perform on the front line (the ED is as front line as it gets).
- A *latent* fault is one whose adverse consequences may lie dormant for some time, only becoming evident when it combines with other factors to breach the system's defenses. Responsibility for latent faults can often be laid at the feet of those who designed or manage the system.
- *Slips* describe attentional or perceptual failures in the execution of an observable action sequence. Covert internal events (generally associated with memory failures) leading to a failure of execution are referred to as *lapses*. Both slips and lapses are actions that deviate from an intended plan.
- A *mistake* is a deficiency or failure in either the judgment or inferential process involved in the selection of an objective, or in the specification of the means to achieve it, irrespective of whether or not the actions directed by this decision-scheme run according to plan.
- An *incident* or *near miss* is a failure in some aspect of care that is either caught in time, mitigated, or fortunately has no effect on the patient.
- An *accident* or *adverse event* is a failure in some aspect of care that leads to injury.
- Both incidents and accidents are typically judged according to their preventability, but this is a fraught concept.
- *Negligent adverse events* represent a subset of preventable adverse events that satisfy legal criteria used in determining negligence (i.e., whether or not the care provided failed to meet the standard of care reasonably expected of an average physician qualified to take care of the patients in question).

3. Why don't we use the term *error* anymore?

Error is a folk model for explaining performance. Errors are mental constructs that are developed after-the-fact to explain outcomes. They are like optical illusions, simultaneously convincing and misleading. If we could make health care safer but still committed errors, we would be pleased, and if we eliminated errors but still had the same burden of adverse events, we would not. The problem is not error; the problem is harm.

4. I've heard the term *iatrogenic*. Isn't that when physicians make mistakes?

The term *iatrogenic* was originally used to describe "disorders induced in the patient by autosuggestion based on the physician's examination, manner, or discussion" but later gained a broader definition as "the creation of additional problems or complications resulting from treatment by a physician or surgeon" (Dorland's *Medical Dictionary,* 25th edition, 1974). Recently, it has come to be used in a more general sense to describe adverse outcomes that result from a patient's treatment within the health care system. A more appropriate term to describe such error is *comiogenic*, proposed by Sharpe and Faden (1998). It has its origin in the Greek root *komein* (to take care of), familiar to us in the term *nosocomial*, which describes disease (Gr. *nosos*) that originates in the hospital. This new term has the advantage of embracing the diverse sources of harm that can occur to patients in health care systems.

5. What's the breakdown of safety problems in the ED?

We don't really know because there haven't been any systematic studies to date. Most of what is known comes from incidental observations made in the major studies on hospitalized patients who came through the ED, a few ED studies, and anecdotal observations. It appears that the incident rate, especially of slips and lapses, is probably quite high, but that the majority of these are corrected before they result in an adverse outcome. One thing appears clear: the most costly and deadly problems generally result from mistakes associated with delayed or missed diagnoses.

6. Am I likely to survive a career in emergency medicine without being involved in a serious adverse event?

No. When you work in the jungle, you get bitten by snakes.

7. What's the ratio of detected to undetected failures?

About 1:50

8. What proportion of adverse events is preventable?

About 70%

9. What are FPCs? Give examples in the ED.

An FPC is any factor or condition that increases the probability of failure in a given system. There is no other area of medicine where this combination of FPCs exists so intensively. If you hadn't already realized it, your chosen career of emergency medicine is one of the most difficult areas of medicine. Examples of FPCs are:

- Diagnostic uncertainty.
- High decision density.
- High cognitive load.
- Novel or infrequently occurring situations.
- Time limitations for detection and correction of error.
- Low signal-to-noise ratio.
- Overcrowding/channel capacity overload (RACQITO).
- Mismatch between real and perceived risk.
- Poor feedback.
- Poor quality of person-to-person information transfer.
- Experience, training, and education limitations.
- Disruption of circadian rhythms by shift-work.
- Compromised task-pacing through interruptions or interventions.
- High physical and emotional stress levels.

10. Most of these look self-evident, but what is meant by the *low signal-to-noise ratio* FPC?

Signals are critical pieces of information that must not be missed. No signal is received in isolation. All signals are accompanied by noise, which consists of distracting stimuli or pieces of information that reduce the likelihood of detecting the signal. A low signal-to-noise ratio

occurs when the base-rate or incidence of the serious condition or diagnosis is low (e.g., subarachnoid hemorrhage) and well exceeded by the more common, and usually benign, diagnoses (tension and migraine headaches). The major problem in detection is that the signs and symptoms of both the signal and the noise can often be very similar to each other. Unfortunately, low signal-to-noise ratios exist for almost all serious conditions that present in the ED (e.g., abdominal aortic aneurysm as a cause of abdominal pain, pulmonary embolus as a cause of dyspnea, ectopic pregnancy as a cause of syncope, spinal column infection as a cause of low back pain, aortic dissection as a cause of chest pain).

11. What is the significance of the high cognitive load FPC?

Cognitive load refers to the amount of thinking activity that an emergency physician must deal with at a given moment in time. It requires varying degrees of memory, concentration, processing, and problem solving. Not infrequently, physicians are responsible for a variety of patients with a variety of illnesses, with a variety of acuities. It is akin to a juggler maintaining a number of objects in the air at the same time. In no other branch of medicine is cognitive load so high, and the burden of switching cognitive frames so great.

12. How can we reduce cognitive load?

In an ideal world, we should not be put in situations where cognitive loading is excessive because, inevitably, this will lead to failure. But the pace of the ED is sometimes difficult to predict and there will be times when volume and acuity reach dangerous levels no matter how well one has prepared. Under these conditions, any strategy or device that reduces the amount of cognitive work and cognitive time will reduce cognitive load. Appropriate designation and delegation of tasks within the caregiver team distributes the cognitive load and reduces the individual burden. Other examples are:

- Mnemonics
- Putting information in the world, rather than in one's head; for example:
  - Notes
  - *To-do* lists
  - The whiteboard
- Handheld computers
- Algorithms
- Decision rules
- Clinical practice guidelines and pathways
- Broselow-Luten pediatric resuscitation color-coding system

13. Don't all these aids lead to *medicine by numbers* and reduce my autonomy?

The practice of medicine is more complex than ever, and we need all the cognitive help we can get. There is ample room left for autonomy and clinical judgment. In addition, these aids can never be made specific enough to fit all clinical circumstances, so judgment in their application is still required.

14. How does the *poor feedback* FPC cause failures?

The efficient performance of any system depends on timely and reliable feedback. Good feedback results in good calibration and physicians are no exception. In the absence of feedback, emergency physicians will assume their diagnoses and management are acceptable and there is no need to change behavior. The reliability and timeliness of feedback in the ED is generally poor. Whether we admit patients to the hospital, or discharge them to outpatient care, we rarely know the consequences of our actions.

15. What is RACQITO?

It refers to conditions under which the vital signs of the ED become unstable. It is an acronym for **R**esource **A**vailability **C**ontinuous **Q**uality **I**mprovement **T**rade-**O**ff. It is analogous to the well-known speed-accuracy tradeoff first discovered in military domains. It is a tipping point

at which a trade-off begins between the resources available to the ED and the ability of the people working there to maintain continuous quality improvement (CQI) of care. Under conditions of RACQITO, the failure rate goes up, and the quality of patient care declines.

16. What are VPCs? Give examples in the ED.

Violation producing conditions are factors that lead clinicians to deviate from formal procedures or from other customs of good practice. They are associated with individual performance characteristics, having their origins in gender, cultural (local and general), and personality traits. Some examples are:

- Excessively burdensome procedures.
- Underconfidence.
- Overconfidence.
- Perceived requirement to follow authority gradient.
- Safety procedure compliance seen as an inconvenience.
- Maladaptive group pressures.
- Maladaptive copying behavior.
- Risk-taking behavior.
- Individual or group normalization of deviance.
- Production pressure.

In addition, some sorts of violations *(necessary violations)* are present in a complex system like the ED. These are violations that are required to get the work done or meet production goals. For example, *working to rule* (i.e., refusing to engage in necessary violations) is a common job action strategy that can bring production to a halt.

17. What is normalization of deviance?

Firstly, the *deviance* refers to the presence of individual or combinations of FPCs and VPCs. By definition, their very presence is a deviation from a safe environment. Usually, they are identified and the appropriate corrections made to restore safety. In some EDs, however, insufficient resources or other limiting factors lead to persistence of these conditions. Eventually, people simply get used to working under these conditions (i.e., the deviance becomes normalized and a chronic state of RACQITO is established).

18. What's the difference between safety management and CQI?

Safety seems to be a special element of quality. There is a great deal of overlap, but also significant differences.

19. What are the components of safety management?

The four main components of safety management are *reduction, containment, mitigation,* and *resilience.* Ideally, we would like to reduce the total number of failures; barring that, we would like to contain the failures that still occur so they do not affect patients (or staff); barring that, we would like to mitigate the effects of those failures that do affect patients (or staff). Finally, we need to maintain the capacity to respond effectively to unexpected events or threats.

20. Give examples of strategies for safety management in the ED.

- Designing good work environments using human factors engineering (HFE) principles.
- Improved detection and assessment of latent faults.
- Improved detection and reporting systems for active failures.
- Discovery, assessment, and elimination of specific FPCs.
- Cultural and individual awareness training to reduce VPCs.
- Recognizing RACQITO and the conditions that produce it.
- Training in teamwork behaviors in the ED.
- Improved response and support for individuals when adverse outcomes occur.
- Maintaining sufficient discretionary resources (i.e., staff, energy, space, and equipment) to enable effective response to unexpected events or threats.

21. What does the expression *geography is destiny* mean in the ED?

It refers to the triage process in the ED, and the tendency to be treated according to where, or in whose territory, the patient happens to be. Firstly, the triage system of EDs operates by trying to place the right patient in the right room. Thus, eye complaints go to the eye room, cardiac complaints into the cardiac room, and so on. Physicians and nurses tend to *anchor* on where the patient is initially placed, which can be problematic and lead to error when the presenting symptoms are misleading (e.g., a complaint of constipation might be a dissecting abdominal aortic aneurysm). Thus, we need to maintain a state of willingness to undo geographical cues. Secondly, it refers to the natural tendency of experts to see particular problems within their own frame of reference. Often, the process of perception depends less on what is before our eyes and more on what we expect to see. If one walks around with a hammer, everything begins to look like a nail. Right-sided abdominal pain in a female may look like appendicitis to the surgeon, renal colic to the urologist, pelvic inflammatory disease to the gynecologist, and somatization to the psychiatrist. Thus, when we send patients down particular paths, we may be committing them to particular destinies. Experts are best engaged at the point at which the problem has become fairly well defined, and until it is, the ED physician remains the best source of expertise. We should remember, too, that a consult is a consult and not a transfer of care.

22. What proportion of failures in the ED are due to negligence?

Relatively low, probably less than 5%. It is virtually useless to label bad outcomes as being the result of "bad apples." Human activity characterizes virtually all aspects of ED function, and whenever we see failure and its consequences it will usually have been mediated by humans. Inevitably, physicians, nurses, technicians, and others will be the human vector by which the failure makes its appearance. This association of humans with failure leads to a natural tendency to blame people when failures occur. This tendency is referred to as *fundamental attribution error.*

23. What is fundamental attribution error?

It's a term used by psychologists to describe our tendency to attribute blame to people when things go wrong. For example, if we see someone fall over we might characterize them as careless, clumsy, or accident-prone (i.e., we attribute the witnessed event to a failing, to dispositional qualities in that person). However, it might be the case that the person fell over because the floor was slippery and they were on their way to urgently assist someone. In this case, less visible, situational factors might have been more responsible for the outcome. This doesn't mean there are not people out there who are careless and clumsy, but rather we should be more willing to consider situational factors when seeking explanations for why things go wrong. Taking this to an extreme, some believe there should be no such term as *error* because, ultimately, we might explain all outcomes by situational factors. This takes us close to causal determinism and the so-called *illusion of free will.* Do we, in fact, enjoy any real control over what we do? On a less philosophical note, it is not uncommon to hear some emergency care-providers abnegating responsibility for poor quality of care by virtue of the system and conditions under which they are obliged to work, and over which they have limited control.

24. Are psychiatric patients especially vulnerable to failure in the ED?

Yes, in fact, the earliest reports of failures in the ED related to the management of psychiatric patients. Historically, we have failed to provide them with adequate medical clearance, we have underestimated their concurrent physical illness, and we have made attribution errors. Some studies have suggested the attitudes of ED personnel can actually increase the risk of suicide in vulnerable patients. Part of the problem is that the psychiatric patient in the ED does not fit the type of *model* patient that we like to see (see Table 7-1).

25. Do we make attribution errors in our perception of ourselves?

Yes. There is probably no one harder on physicians than physicians themselves. When we perceive ourselves as having committed an error, our reaction is often inappropriate, being

TABLE 7-1 CONTRASTING FEATURES OF PSYCHIATRIC AND NON-PSYCHIATRIC PATIENTS

Feature	Nonpsychiatric Patient	Psychiatric Patient
Physical illness	Present	Absent
Behavior	Passive, compliant	Passive/aggressive, non-compliant
Attitude of patient	Grateful or appreciative	Neutral, ungrateful or resentful
Diagnosis	Mostly objective	Mostly subjective
Work-up	Relatively fast	Usually slow
Lab or imaging studies	Contributory	Non-contributory
Management	Relatively clear	Difficult or deferred
End point	Often definitive	Poor, revolving
Compliance	Usually good	Usually poor
Attitude of staff	Good, supportive	Often unsupportive

overly harsh and punitive. However, by increasing our awareness and understanding of the contextual nature of these failures, we can develop a more appropriate response to it when it occurs.

26. This begins to sound like a psychology course. I thought I was in emergency medicine.

It's true that many of the terms that have come into usage in the new science of *Safety in Health Care* have their origin in the discipline of psychology. This is no accident! Much of the groundwork in this area was done by psychologists, ergonomists, sociologists, engineers, and others with a special interest in the area of human performance. One of the earliest commentators on error was James Sully, a professor of Mind and Logic at University College, London in the late 19th century. More recently, another professor at University College, the psychologist Charles Vincent, has made significant contributions to our understanding of accidents and incidents in medicine. The fathers of modern approaches to *Human Error* are James Reason, now Professor Emeritus in Psychology at the University of Manchester in England, and Jens Rasmussen, an industrial psychologist who worked for many years at Risø National Laboratories in Denmark.

27. What are the three major categories of safety problems in the ED?

Procedural, affective, and cognitive.

28. Give examples.

Procedural failures are those that occur during the performance of a procedure. They involve some sort of psychomotor failure through a breakdown in or between motor function and visual and touch sensory modalities. They are often highly visible, their immediate consequences are apparent, and they are usually improvable by training and practice. Some examples are: esophageal intubation, causing a pneumothorax putting in a central line, getting a venous sample while attempting an arterial blood gas, improper application of a cast, poor suturing technique, causing further injury while reducing a dislocation, injuring internal organs putting in a chest-tube, and so on. High-fidelity simulation techniques offer much promise in reducing procedural failures.

Affective problems occur when the physician's affective state influences the quality and validity of clinical decision making. This is usually occult, and physicians themselves may be

unaware of the influence of their own affective state on decision making. The affective state can be independent of or related to patients in their care. An example of an independent instance would be if the physician was experiencing a temporary mood disruption or even a depressed or hypomanic state. This might result in the quality of decisions for all patients being compromised. Related instances, in contrast, occur when the physician develops feelings, either positive or negative, toward a specific patient, or specific groups of patients. This is referred to as countertransference. *Negative countertransference* occurs when the physician develops negative feelings toward a patient, often on the basis of significant exemplars in the physician's past (i.e. the patient reminds the physician of a previous patient, class of patient, or some other figure with whom the physician has had a bad experience). As a result, the quality of decision making and care may be compromised. Patients with borderline personality disorder have an unusual capacity for generating negative countertransference in their caregivers. *Positive countertransference* can also compromise decision making and management. An example would be overinvestigating a trivial complaint in a patient (through concern about not missing something significant) toward whom the physician has strong positive feelings. The *chagrin factor* is another example when physicians modify their investigations so that they do not expose themselves, or the patient, to the chagrin which might result from turning up an undesirable finding.

Cognition is involved in all human behavior, from the simple *skill-based* levels, through the higher order, *rule-based* behaviors, to the most complex level of cognition, which is involved in *knowledge-based* behavior (see Table 7-2). The execution of a well-rehearsed, automatic motor skill (e.g., intubation) requires little cognitive input other than simple visual and touch monitoring. An increased level of cognitive input is clearly needed for rule-based behaviors, but even complex medical acts, such as those directed by advanced cardiac life support (ACLS) algorithms, can be performed with minimum cognitive involvement. However, knowledge-based cognitive behavior involves interpreting and understanding novel situations and problems within the context of specific domain knowledge (e.g., integrating the presenting complaint, past medical history, physical examination, and laboratory findings in a patient with syncope). There is clearly some overlap in cognitive complexity between different levels, and in some sense, they operate simultaneously. As experience accumulates, more and more behaviors can be relegated to lower levels of cognitive involvement. Thus, paradoxically, novices operate at the knowledge-based level almost all the time, whereas experts operate at the skill-based level most of the time.

Cognitive failures can occur at any level in this hierarchy of thinking processes. The incidence of cognitive failure increases under conditions of uncertainty, especially when

TABLE 7-2 THE COMPLEXITY OF COGNITIVE BEHAVIOR IN THE ED

Level	Activity
Skill-Based	Wound repair
	Dislocation reduction
	Intubation
Rule-Based	Radiographic decision rules
	Clinical practice guidelines
	Algorithms
Knowledge-Based	Clinical decision making
	Management decisions
	Diagnostic reasoning

thinking is hurried or pressured, and when heuristics are used. Interestingly, cognitive failures, when viewed in retrospect, are almost always judged preventable. This undoubtedly says more about the person making the judgment than it does about the actor(s) involved in the failure.

29. What are *heuristics*?

Heuristics are strategies for thinking. The term usually refers to strategies that build economy and abbreviation into the thinking process. Essentially, a well-established heuristic is a disposition or cognitive bias to respond in a particular way to a particular situation. For the most part, they are useful to us in the ED, where we are often looking for short cuts. Occasionally, however, they can get us into trouble.

30. Give some examples of cognitive biases.

There are probably over 30 discrete phenotypes of cognitive bias. Many of them derive from five archetypal heuristics: *representativeness, availability, anchoring, confirmation,* and *satisficing.*

31. What is representativeness?

Representativeness is a subjective assessment or judgment of how similar a particular example is to its parent population. For example, patients who are experiencing angina will classically present with gradual onset of a visceral quality of retrosternal pain, which may radiate to the arm, shoulder, neck or jaw, lasts 5 to 15 minutes, and may be associated with nausea, diaphoresis, and dyspnea. These symptoms and signs are generally held to be representative of the class of patients with angina. However, some patients (i.e., geriatric, diabetic, or female) are more likely to present with atypical symptoms. The more unrepresentative the patient's presentation is, the greater the chances of the diagnosis being delayed or missed altogether. Because of unrepresentativeness, young patients are also more likely to experience a failed diagnosis. Representativeness error accounts for a significant proportion of patients with chest pain caused by acute myocardial infarction (AMI) being sent home from the ED. Insufficient experience or training increases the likelihood of making the representativeness error. Unfortunately, most medical textbooks tend to describe prototypical disease and, therefore, students are unwittingly trained to look for representativeness or prototypical manifestations of disease.

32. What is availability?

In the normal course of thinking, some memories will be more available to us than others. For example, if an emergency physician saw a patient a week ago who presented with a headache that turned out to be a subarachnoid hemorrhage, the image of that patient and the association of headache with subarachnoid hemorrhage is more available or recent than, say, a headache that was seen a year ago. Thus, the physician may have a greater tendency to look for a subarachnoid than would otherwise be dictated by the presentation of a particular patient. Availability might similarly be increased by a colleague's description of a clinical encounter, a recent presentation of a case at rounds, or if the physician had recently read a review of a particular disease. Availability would be *decreased* by long intervals since encountering, or never having previously seen, a particular disease (out of sight, out of mind). Availability is not solely determined by recency of experience. It also depends on the salience and emotional valence of previous encounters. For example, if the physician had a particularly vivid experience 10 years ago, missing an AMI in a young person, thenceforth the physician might be overcautious in managing all patients with chest pain, which might result in a bias toward overconsultation, and poor utilization of resources. Thus, availability influences decision making and can lead to both overdiagnosing and underdiagnosing. The latter, when physicians are in the *out of sight, out of mind mode*, can result in serious misses.

33. What is anchoring?

Anchoring can give rise to particularly difficult failures in the ED. These occur when paramedics, nurses, or physicians attach, commit, or anchor to a particular diagnosis early on in the presentation. This usually occurs because certain sign and symptom patterns may

strongly suggest a particular diagnosis, which is adopted without giving sufficient consideration to other possibilities on the differential. For example, consider a 60-year-old male with a history of renal stones presenting with flank pain, nausea and vomiting, and hematuria. The obvious diagnosis is ureteral colic, and inexperienced nurses and physicians will anchor on this. For the vast majority of cases, the anchor will serve them well, but occasionally an aortic dissection will be missed, sometimes with fatal consequences. The order in which information is obtained strongly influences anchoring, with initial information being given greater importance than that gathered later. Anchoring is difficult to recognize in oneself; perhaps the only sure way out of it is to have a new set of eyes look at the problem (such as often occurs at change of shift).

34. What is confirmation bias?

It is the tendency to look for evidence or information that can be used to bolster a hypothesis that has already been adopted (i.e., to look for things that rule in a diagnosis); it also includes the tendency to fail to perceive evidence that might be disconfirming to the current worldview. Consider a patient who presents to the ED with a headache and fever and the physician hypothesizes that the headache has a benign origin associated with a flulike syndrome. In the course of physical examination, the physician finds neck stiffness, which he attributes to myalgia and tension of the neck muscles. This is confirmation bias; the physician is fitting a significant finding (in this context of headache and fever) into the preformed diagnosis of a flulike illness. Instead, a far more powerful strategy would be to look for disconfirming evidence that rejects a working hypothesis. In this case a lumbar puncture would quickly settle the issue and rule out meningitis. If anchoring occurs early on in a presentation, and the clinician tends to work with a strong confirmation bias, the boat may be missed completely.

35. What is search satisficing?

The term *satisficing* is derived from satisfy and suffice. This is an example of another cognitive bias, one that probably has its origin in both the representativeness and anchoring heuristics. Essentially, it refers to the tendency to call off a search once something has been found. It is illustrated by the question: "What is the most commonly missed fracture in the ED?" The answer is not C7, the scaphoid, or Lisfranc (all occasionally missed), but the *second fracture,* because we have a tendency to satisfy ourselves when we find the first fracture and call off the search for others. Search satisficing errors similarly arise when we call off the search for additional foreign bodies, concurrent diagnoses, or co-ingestants in a poisoning.

KEY POINTS: SAFETY IN EMERGENCY MEDICINE

- *Error* is a term of historical interest only; it is an illusion created by hindsight bias.
- Performance failures have their roots in the context of work and how it interacts with the human worker; the only malleable element is the context of work.
- Special skills in psychology and engineering are required to understand and successfully manage safety problems.

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CHAPTER 8

HOW TO CRITICALLY REVIEW EMERGENCY MEDICINE LITERATURE

Debra Houry, MD, MPH

1. **Can I skip this chapter if I don't plan to do research?**
   No! Reading the medical literature carefully and incorporating it into clinical practice are important for all physicians.

2. **Why should I read medical journals?**
   - To learn the clinical features and management of diseases seen in practice.
   - To determine whether a new or existing diagnostic test or treatment would be beneficial for your patients.
   - To stay abreast of recent medical developments and issues.

3. **Which study design is the best?**
   Randomized controlled trials are considered the strongest studies. Patients are randomly assigned to treatment groups, limiting selection bias. These studies are uncommon in the emergency medicine literature and often require large study populations. Other study designs may be more appropriate, such as in instances when performing a randomized trial would be unethical (withholding a life-saving treatment or exposing patients deliberately to harm).

4. **Are there any other types of study designs I should be familiar with?**
   - **Cohort studies** divide groups by exposure status and prospectively follow the groups over time to determine who develops the disease. These studies are used to calculate the relative risks of various exposures.
   - **Case-control studies** retrospectively compare cases (individuals with the disease) with controls (individuals without the disease) to determine the frequency of exposures. These research studies are subject to recall bias but can be used to determine odds ratios.
   - **Case series** report characteristics of patients with a particular disease and can be valuable when looking at rare diseases or outcomes (HIV first was reported as a case series of *Pneumocystis carinii* pneumonia in homosexual populations).

5. **What is blinding? Why is it important?**
   A technique in which patients, physicians, researchers, and anyone else involved in the research study are unaware of whether patients are in the experimental or control group. This helps eliminate potential bias, unequal distribution of groups, differential administration of interventions, and distorted results and outcome assessments.

6. **Do sample size and power matter?**
   Power is the probability that the study will detect a treatment effect between the two experimental groups. The smaller the size of the treatment effect being studied, the larger the sample size should be. Many studies do not have a large enough sample size to detect a statistically significant difference and may report negative results when a significant difference may have been detected in an appropriate sample size. Without adequate power, the study results may be inconclusive.

7. What does number needed to treat mean?

This is the number of patients who would have to receive the treatment for just 1 patient to benefit from the treatment. For example, if the number needed to treat is 100, then 100 patients would need to have the treatment for 1 person to benefit from it. A lower number needed to treat is obviously better, but if the benefit is preventing mortality, a larger value may be acceptable. You can calculate the number needed to treat by dividing 1 by the absolute risk reduction proportion.

8. What should I look for when evaluating a chart review study?

1. Trained chart abstractors.
2. Explicit criteria for case selection and exclusion.
3. Defined study variables.
4. Standardized abstraction forms for data collection.
5. Periodic meetings among researchers to resolve abstraction disputes.
6. Monitored performance of abstractors.
7. Blinded chart reviewers.
8. Measures of inter-rater agreement.

9. What does a *p* value refer to?

The probability that the results of a study or the differences between study subsets occurred by chance. The most commonly used value, $p < 0.05$, means that there is less than a 5% probability that the study results occurred by chance. This is statistically significant but not necessarily clinically significant. A decrease by 1 minute in overall ED length of stay may be statistically significant ($p < 0.05$), but a 1-minute reduction in overall length of stay likely has no clinical relevance for physicians or patients.

10. How do I interpret confidence intervals?

A confidence interval is the expected range of results in the study population. A 95% confidence interval means that you would expect 95% of your results to fall within the specified range. A smaller range of values or less variance usually is found with larger sample sizes. A wide confidence interval could mean that some of the study results may not be clinically significant. Look at the upper and lower boundaries of the confidence interval and determine if both values still would hold clinical significance for you. If only the upper boundary value would have significance, there may not be an overall clinical benefit.

11. Does it matter who sponsors a study?

Yes. Any direct involvement in a study by a sponsor, particularly one with a financial interest in the outcomes of the research (e.g., pharmaceutical industry), has the potential to influence the study. Sponsors should not have any input into study design, data collection, or method of reporting the results. Unfortunately, many research studies do not adhere to these standards. Disclosure of financial support is important and should alert the reader that there is the potential for introduction of bias into the study. Industry-sponsored studies may provide valuable information but must be reviewed carefully.

KEY POINTS: CRITICAL REVIEW OF EMERGENCY MEDICINE LITERATURE

1. Randomized controlled trials are the best studies, but other studies may also be valid.
2. A $p < 0.05$ is statistically significant.
3. A smaller confidence interval is better.
4. Sponsorship may influence how results are presented.

12. Should I read reviews on clinical topics?

This depends on many factors:

- Are you looking for basic knowledge or understanding of a disease process? If so, a clinical review may be sufficient and can provide the foundation for you to continue your reading on the topic.
- Are you looking for the latest information? Clinical reviews may be outdated by the time of publication because the literature on which they are based was written before the review.
- Is it a narrative or systematic review? In narrative reviews, the author selects the articles to include in the review and summarizes the topic based in part on his or her experience. In a systematic review, the author identifies articles through a search and includes or excludes the articles based on predefined criteria and summarizes the topic based on strength of the evidence from the included articles.

13. How do I practice evidence-based medicine?

Critically reviewing the medical literature and applying the best evidence to your practice is evidence-based medicine. After reading this chapter, you should be able to read research studies and determine the strength of the studies and their findings.

14. What are some of the statistical terms I should be familiar with?

- **Relative risk:** The risk of developing a disease after an exposure compared with individuals without an exposure: $A/(A + B) \div C/(C + D)$
- **Odds ratio:** The odds of developing a disease after an exposure compared with those without an exposure: $(AD)/(BC)$
- **Sensitivity:** The proportion of people with a positive test result who truly have the disease: $A/(A + C)$
- **Specificity:** The proportion of people with a negative test result who do not have the disease: $D/(B + D)$
- **Positive predictive value:** The likelihood that a person with a positive test result actually has the disease: $A/(A + B)$
- **Negative predictive value:** The likelihood that a person with a negative test result does not have the disease: $D/(C + D)$

See Figure 8-1 for reference.

		Disease	
		Present	Absent
Exposure/Test Results	Positive	A	B
	Negative	C	D

Figure 8-1. Disease versus exposure grid.

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CHAPTER 9

EVIDENCE-BASED RATIONAL USE OF DIAGNOSTIC IMAGING

Ronald R. Townsend, MD, MA, and Stephen V. Cantrill, MD

1. What does evidence-based rational use of imaging mean?

Evidence-based imaging is the application of evidence-based medicine methodology to decisions regarding the use of diagnostic imaging or interventional image-guided procedures. A rational decision is made regarding use of imaging in a particular clinical situation based upon knowledge of the results of published research on the use of imaging for the problem at hand, the clinical expertise of the provider(s), and the patient's values and preferences. Such an analysis may lead to a decision to perform a specific imaging study or no study at all. Use of evidence-based imaging is motivated by desires to provide optimal quality patient care and avoid costs and radiation exposure associated with examinations that will not benefit the patient.

2. Describe the evidence-based approach.

The evidence-based medicine approach incorporates five steps in the determination of a specific patient scenario:

- Ask an answerable question.
- Search the literature for current best evidence.
- Appraise the retrieved evidence.
- Apply the findings.
- Evaluate your success with the process.

Crucial to the process are narrow definitions of the question, complete retrieval of current literature, and critical analysis of the validity and relevance of the available research.

3. How is the evidence used by the clinician?

The clinician must decide what, if any, imaging is appropriate based on integration of the details of the patient's history, symptoms, and signs with the available evidence. The unique nature of a given patient's presentation may make an examination inappropriate, even if its use is generally supported by evidence.

4. When should I consult a radiologist prior to ordering an imaging study?

For many clinical problems, the appropriate evidence-based imaging may be well known to the clinician. Especially for complex questions or for patients with recurrent visits to the ED, consultation with a radiologist prior to any imaging may help optimize the patient's care.

5. How can I apply evidence-based imaging in my clinical practice?

Although education regarding use of evidence-based medicine (and its application to imaging) in medical schools, residencies, and in postgraduate settings is increasing, most practitioners are overwhelmed by the concept of doing a complete analysis themselves. Fortunately, there are many resources available to aid the physician in determining what the evidence suggests will be useful imaging for some common clinical problems.

Many specialty societies have developed *guidelines* or *appropriateness criteria* that include analyses of application of imaging in many emergency situations (e.g., ACR

Appropriateness Criteria). These range from opinion papers (not evidence-based) to true attempts at rigorous evidence-based analysis. Review of the materials accompanying these guidelines can help clarify their nature, but they may be a useful conduit to the literature in any case.

6. Are clinical prediction rules helpful?

Evidence-based clinical prediction rules are widely available validated tools to guide emergency imaging for many scenarios. They typically define specific history, physical findings, and/or laboratory parameters that accurately predict the utility or lack of utility of specific imaging.

7. Is radiation exposure from X-rays and CT used in ED patients dangerous?

When evidence-based imaging is performed, the benefit of the diagnostic information obtained will generally far outweigh any small risk associated with radiation exposure. For example, a victim of major trauma should not be denied a CT and the potential of image-directed life-saving treatment, even if she may be pregnant.

However, as of 2007, medical radiation is the largest source of exposure to the U.S. population, surpassing background radiation. The medically related radiation exposure of the U.S. population has increased substantially in recent years, related primarily to increased use of newer diagnostic studies (especially CT, interventional procedures, and nuclear medicine). Diagnostic radiographs (X-rays or *plain films*) have relatively low associated radiation, so less risk. Some examples of average adult effective dose for some imaging procedures are given below. Newer CT scanning technology may use lower radiation doses for many examinations, which helps minimize risk (see Table 9-1).

8. Which patients are at highest risk from imaging-related radiation exposure?

Young patients and those who receive many CT scans. The primary concern related to significant patient radiation exposure is risk of development of neoplasm. The lifetime risk is highest for children. The patients placed at highest risk as a result of medical imaging are those who receive multiple high-dose examinations (e.g., CT of multiple body parts), especially when done repeatedly over months or years. Again, the benefit may substantially

TABLE 9-1. ADULT EFFECTIVE DOSES FOR IMAGING PROCEDURES

Examination	Average Effective Dose (mSv)	Range of Values Reported (mSv)
PA chest radiograph	0.02	0.007–0.050
Pelvis radiograph	0.6	0.2–1.2
Head CT	2	0.9–4.0
Chest CT for pulmonary embolism	15	13–40
Abdomen CT	8	3.5–25
Pelvis CT	6	3.3–10
IR-pelvic vein embolization	60	44–78
Background (annual)	3	Geographic variation

mSv, milli-Sieverts (1 mSv = 100 mrem).
From Mettler FA, Huda W, Yoshizumi TT, et al: Effective doses in radiology and diagnostic nuclear medicine: a catalog. *Radiology* 248:254–263, 2008.

outweigh the risk for patients with clearly indicated examinations. Nevertheless, before ordering any examinations, it is prudent to carefully review the imaging history of a patient who frequently presents to the ED with recurrent problems such as chest pain, abdominal pain, or renal colic. For example, does a young woman with recurrent kidney stones need another CT to evaluate her stones or can she be effectively managed with ultrasound?

KEY POINTS: RISKS OF IMAGE-RELATED RADIATION

1. Risk benefit analysis clearly favors the performance of evidence-based imaging in the ED.
2. Patients who receive multiple high-dose imaging examinations (especially CT) are at highest risk of long-term consequences.
3. Young patients are at higher risk than older patients.

9. What question should be asked when ordering diagnostic imaging studies in young patients?

For young patients with chronic problems, an important question to consider: Is there an evidence-based imaging approach that can address the patient's need without use of ionizing radiation? This often involves use of ultrasound or magnetic resonance imaging (MRI). Consultation with a radiologist should be considered in planning the approach to such patients.

10. What else should be considered when ordering diagnostic imaging?

Unnecessary imaging contributes to the high cost of health care that ultimately is a burden to society. A CT of the chest, abdomen, and pelvis, for example, may result in charges of more than $5,000. For the individual patient, an incidental finding at imaging may result in wasted time and money to work up the finding, which most often proves not significant. There is the potential for substantial morbidity or even mortality if this leads to biopsy or surgery. This may be viewed as unavoidable if the initial imaging was clearly indicated but is even more tragic if it was not.

11. Should a cervical spine CT be obtained in all trauma patients?

No. Many trauma victims have virtually no likelihood of clinically significant cervical spine injury. An evidence-based approach would be to utilize one of the published clinical prediction rules to determine who should be imaged. The NEXUS criteria and Canadian C-spine rule define patients in whom no imaging is necessary (see Chapter 82). There is no clear evidence to favor one of these rules over the other and not enough data to confirm validity in children. In patients who are at high risk of cervical spine injury, CT is more sensitive and specific than plain radiography.

12. Which patients should get a cervical spine CT without cervical spine X-ray?

A validated rule (referred to as the Harborview high-risk cervical spine criteria) defines a subgroup of patients who meet NEXUS or Canadian C-Spine rule criteria who may be effectively managed with CT as the initial cervical spine imaging. This includes adults with any one of these parameters (who would typically be getting head CT contemporaneously):

Injury mechanism parameters:

- High-speed (>35 mph combined impact) motor vehicle accident (MVA)
- Crash with death at scene of MVA
- Fall from height >10 ft.

Clinical parameters:

- Significant closed head injury (or intracranial hemorrhage seen on CT)
- Neurologic symptoms or signs referred to the cervical spine
- Pelvic or multiple extremity fractures

13. Should all chest pain patients get a CT to exclude pulmonary embolism?

No. Such an approach would be expensive, subject many patients to unnecessary radiation, and potentially contribute to missed diagnoses of pathology not evident on CT. A clinical prediction rule can be used to distinguish patients who may benefit from imaging for possible pulmonary embolism from those unlikely to have embolism (See Chapter 27).

14. When should patients with clinical suspicion of kidney stones get a noncontrast CT of the abdomen and pelvis (CT-KUB)?

Evidence does support CT as the most accurate examination in the diagnosis of urinary stone disease. It clearly has the highest sensitivity and specificity of all imaging modalities for ureterolithiasis. It can facilitate management decisions by accurately assessing stone size and number and the degree of collecting system dilatation. However, many patients with prior CT documentation of urinary stone disease present to the ED on multiple occasions, and it may not be necessary or prudent to perform another CT-KUB at each visit.

15. What imaging other than CT-KUB should be considered for patients who frequently present to the ED with symptomatic urinary stone disease?

Many patients with recurrent urinary calculi may be managed with symptomatic treatment. If any imaging is necessary to facilitate management, ultrasound may provide the necessary information. Ultrasound may detect hydronephrosis as a sign of obstruction. The low sensitivity of ultrasound for ureteral calculi limits its utility in the initial evaluation of patients with possible stone disease.

16. Is CT or MRI ever appropriate to evaluate extremity trauma?

In the vast majority of clinical situations, the presence or absence of fracture in an extremity is accurately determined by physical examination with or without plain radiography. Evidence-based rules defining which trauma patients need and which do not need radiography are well validated for some body parts (e.g., Ottawa ankle, foot, and knee rules).

Some patients may have persistent symptoms, but no radiographic confirmation of fracture. The appropriate imaging approach to these patients depends on the anatomic site involved and specific symptoms and signs. In some situations, additional radiographic views (e.g., obliques) may define an injury. Many of these situations are uncommon enough that strong evidence to guide practice is limited. For many non–weight-bearing bones, persistent clinical suspicion of nondisplaced fracture can be addressed with 10-day follow-up radiography, at which time a healing fracture may become evident.

Evaluation of possible occult, lower extremity fracture in a patient who is unable to ambulate, especially with symptoms related to the hip, may require additional urgent imaging. CT, MRI, and bone scan all have been utilized to diagnose radiographically occult hip fracture. CT with multiplanar reconstructions is most useful to diagnose subtle cortical disruption, but MRI has the advantage of better assessing soft tissue (e.g., cartilage).

There is strong evidence to support the use of MRI in assessing soft-tissue injuries in the knee, but this is rarely required during an ED visit. Emergent CT to further define some fractures may be needed to plan treatment. This is most common for fractures of the hind and mid foot and intra-articular fractures about the knee, ankle, or elbow. When clinical findings lead to suspicion of vascular injury associated with extremity fracture or fracture-dislocation, further evaluation with catheter angiography or CT angiography may be appropriate. Strong evidence to support the use of CT angiography for this purpose is lacking for some vessels, however.

17. Does the evidence support use of CT or plain films for facial fracture imaging?

CT (especially thin section multidetector CT with multiplanar reconstruction) has higher sensitivity and specificity than plain radiography in diagnosis of many types of facial fractures. Complex facial fractures are almost all managed based on CT findings. In general practice,

most practitioners use CT as the initial and only examination in evaluating patients with definite fractures clinically and those felt to have high probability of fractures. (The exception is nasal bone fractures, which usually require no imaging for diagnosis or treatment.) However, there is a lack of strong evidence to support specific imaging algorithms for specific patient groups.

18. What are the indications for emergent MRI for ED patients?

MRI is usually the best examination for patients with acute atraumatic myelopathy, who may be at risk for progressive neurologic deficit related to spinal cord compression by tumor, abscess, or hematoma. The urgency of the examination cannot be completely defined by evidence but requires clinical judgment.

Acute focal neurologic deficits referable to intracranial pathology often require emergent imaging. Either CT or MRI (either examination often requiring contrast) may be supported by evidence in some circumstances. Patient-specific factors (i.e., history, details of deficit, time course) and local imaging equipment capability/availability may be important factors in deciding on CT or MRI. Consultation with the local radiologist should be considered.

Evidence supports use of contrast-enhanced CT in patients with clinical suspicion of aortic dissection. However, intravenous (IV) contrast administration may be contraindicated in patients with severe allergy or acute renal failure. MRI with contrast (contraindicated with renal failure) or without contrast may be appropriate in some patients. Transesophageal ultrasound may be an alternative in institutions where that is available.

19. What imaging should be done when appendicitis is suspected clinically?

No imaging should be done if management will not be changed (e.g., the surgeon is clinically convinced the patient has appendicitis and will operate no matter what is found at imaging). CT of the abdomen and pelvis has the best accuracy in diagnosis of appendicitis and differentiating it from other causes of right lower quadrant pain. Use of oral and/or rectal contrast for the examination is largely a matter of institutional experience or preference. IV contrast has been used in most studies evaluating CT for appendicitis, but accuracy is similar in other studies without it. Use of IV contrast may improve definition of associated abscess or other pathology causing right lower quadrant pain.

Compression ultrasound is less sensitive than CT for appendicitis but may be most useful in the effort to avoid radiation exposure in pregnant or other high-risk patients. MRI has been used to diagnose appendicitis in pregnant patients, but there is little evidence regarding its accuracy. Consultation with a radiologist should be considered regarding local experience with ultrasound or MRI if use of those examinations for diagnosis of appendicitis is considered.

20. What imaging should be performed for a clinical diagnosis of acute pancreatitis?

With a patient's first diagnosis of acute pancreatitis, ultrasound is appropriate to evaluate for gallstones as a possible cause of the pancreatitis. If biliary dilatation is identified on that examination, further evaluation may be required. CT with IV contrast is most useful to evaluate complications of pancreatitis (e.g., necrosis, pseudocyst) but usually not appropriate at the time of initial diagnosis in the ED.

21. What imaging should be performed to evaluate a palpable abdominal or pelvic mass?

The patient's demographics (e.g., gender, age) and location of the palpable mass affect imaging choice.

A palpable pelvic mass in a woman, most often related to uterine or ovarian pathology, is best evaluated with pelvic ultrasound (transabdominal and transvaginal), including Doppler.

A pulsatile midline abdominal mass in an older adult may be well evaluated with ultrasound of the abdominal aorta to identify any aneurysm and determine its size and extent. If ultrasound is technically limited (e.g., obese patient), CT can be used to evaluate for

aneurysm or another cause of the mass. In a patient with acute symptoms suspicious for aneurysm rupture, the patient's condition should determine whether imaging is advisable before intervention, but ultrasound cannot accurately determine presence or absence of blood leaking from an aneurysm. CT with IV contrast is best for that assessment.

In an adult, a palpable abdominal mass not clearly related to any organ by examination is best evaluated by CT. There is a paucity of data comparing imaging approaches for abdominal masses, however. When a palpable *mass* may be an enlarged organ (e.g., liver or spleen), ultrasound may confirm that diagnosis without requiring use of ionizing radiation.

In an infant, palpable masses often relate to kidneys or the biliary tree, with best initial evaluation with ultrasound.

22. What is appropriate evidence-based imaging for right upper quadrant pain?

Abdominal ultrasound is highly accurate in the diagnosis of cholelithiasis and should be the first imaging study when that is the primary question. Ultrasound and clinical and/or laboratory parameters together allow accurate diagnosis of acute cholecystitis in most patients without additional imaging. In problematic cases (especially possible acalculous cholecystitis), cholescintigraphy (nuclear medicine examination of the gallbladder) may be useful to diagnose acute cholecystitis, but it is not often required for management of patients in the ED. Cholescintigraphy does have a higher sensitivity than ultrasound in the diagnosis of acute cholecystitis. One advantage of ultrasound is its ability to identify nonbiliary causes of right upper quadrant pain in these patients (e.g., disease in the liver or right kidney).

KEY POINTS: EVIDENCE-BASED IMAGING

1. Base imaging choices on patient symptoms and signs. Avoid *shotgun imaging*.
2. Only perform imaging studies that will affect patient management. Abdominal radiographs are generally wasteful if CT or ultrasound will be performed, regardless of findings on the radiograph.

23. What imaging should be done for suspected small bowel obstruction?

Abdominal radiographs have limited sensitivity for detection of small bowel obstruction and limited ability to determine etiology of any obstruction present. If management decisions are not to be made based on results of the radiographs, they should not be obtained (e.g., if the patient will get and be managed based on results of CT whether the radiographs are positive or negative). CT of the abdomen and pelvis with IV (but not oral or rectal) contrast may best define presence of obstruction, its cause, and any evidence of secondary compromise of bowel. Ultrasound can also identify findings of small bowel obstruction, but it is probably not as sensitive as CT (little evidence).

24. What is appropriate evidence-based imaging for left lower quadrant pain?

When diverticulitis is the primary clinical concern, CT of the abdomen and pelvis with IV and oral (with or without rectal) contrast best defines the presence and extent of diverticulitis. It defines presence or absence of complications, such as perforation or abscess formation, which are important in patient management. Other conditions that can mimic diverticulitis clinically (e.g., epiploic appendigitis) can be diagnosed with CT. Compression ultrasound can be used for diagnosis of diverticulitis, but it appears less accurate than CT (limited data).

25. What imaging is appropriate for suspected abdominal abscess?

CT of the abdomen and pelvis with IV and enteric (oral and/or rectal) contrast can effectively evaluate for abdominal abscess in patients with abdominal pain and fever or other history, symptoms, and signs causing suspicion of abscess. If there are localizing symptoms and

signs, a targeted ultrasound may be effective (e.g., clinical question of pericholecystic abscess or question of abdominal wall abscess along a surgical wound), but there is little data comparing alternative imaging approaches in this context. For possible pelvic abscess related to infections of gynecologic origin, transabdominal and transvaginal pelvic ultrasound with Doppler should be considered.

26. When is imaging appropriate for patients with scrotal pain?

When the cause of acute scrotal pain is not evident clinically, scrotal ultrasound with Doppler evaluation of testicular blood flow is the most accurate examination in diagnosis of testicular torsion and distinguishing torsion from other pathologies. It should be performed emergently to optimize the chance of testicular salvage if torsion is present. Radionuclide perfusion studies can be utilized for diagnosis of torsion but may have limited specificity and are usually less available than ultrasound.

27. Should a head CT be performed in all trauma patients?

No. Many patients will not benefit from a head CT. History, symptoms, and signs can be used to identify patients at significant risk of intracranial injury post trauma (see Chapter 83). The New Orleans Criteria for patients with a minor head injury and a Glasgow Coma scale score of 15 limits CT to patients with one of seven findings: headache, vomiting, age older than 60 years, drug or alcohol intoxication, deficits in short-term memory, physical evidence of trauma above the clavicles, or seizure. In other patient populations (patients with a coagulopathy) other rules or guidelines may be useful.

28. How about a head CT on anticoagulated trauma patients?

There are data that anticoagulated trauma patients are at greater risk to develop a traumatic brain injury, and when it occurs, the injury will be more severe with a higher fatality rate. For this reason, the threshold for obtaining a head CT on an anticoagulated trauma patient should be very low. These patients may also require closer monitoring and potential repeat head CT because of the possible development of a delayed acute subdural hematoma.

29. Should patients with closed head injury routinely receive a CT of the abdomen and pelvis at time of head CT?

The clinical threshold for obtaining a CT of the abdomen and pelvis in trauma patients with head injuries is reduced at many centers. It is clear that occult injuries may be identified in such patients. There is a lack of strong evidence, however, regarding which patients benefit from this approach.

WEBSITES

ACR Appropriateness Criteria: www.acr.org/SecondaryMainMenuCategories/quality_safety/app_criteria.aspx
Canadian C-spine Rule: www.aafp.org/afp/20040615/tips/17.html
Harborview High-risk Cervical Spine Criteria: www.ajronline.org/cgi/reprint/174/3/713.pdf
NEXUS Criteria: www.aafp.org/afp/20060515/poc.html

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ALTERED MENTAL STATUS AND COMA

Kenneth C. Jackimczyk, MD, FACEP

1. What is coma? What terms should be used to describe altered sensorium?

A depressed mental state in which verbal and physical stimuli cannot elicit useful responses. Other terms, such as *lethargic, stuporous*, or *obtunded*, mean different things to different observers and should be avoided. You may be *alert but confused* as you read this chapter. It is best to describe the mental functions the patient can perform (e.g., the patient is oriented to person, place, and time; and can count backward from 10).

2. What causes coma?

Mental alertness is maintained by the cerebral hemispheres in conjunction with the reticular activating system. Coma can be produced by diffuse disease of both cerebral hemispheres (usually a metabolic problem), disease in the brain stem that damages the reticular activating system, or a structural central nervous system (CNS) lesion that compresses the reticular activating system. Less than 20% of patients have a structural cause for their coma.

3. How can I remember the causes of coma and altered mental status?

TIPS and vowels, that is, TIPS and AEIOU.

TIPS

T, **T**rauma, **t**emperature
I, **I**nfection (CNS and systemic)
P, **P**sychiatric
S, **S**pace-occupying lesions, **s**troke, **s**ubarachnoid hemorrhage, **s**hock

VOWELS

A, **A**lcohol and other drugs
E, **E**pilepsy, **e**lectrolytes, **e**ncephalopathy
I, **I**nsulin (diabetes)
O, **O**xygen (lack of), **o**piates
U, **U**remia

4. What important historical facts should be obtained from the patient with altered mental status or coma?

This seems like a stupid question because the patient with altered consciousness cannot give you a reliable history, and the comatose patient cannot give any history at all! You should carefully question prehospital personnel and attempt to contact the patient's friends or family. Ask about the onset of symptoms (acute or gradual), recent neurologic symptoms (e.g., headache, seizure, or focal neurologic abnormalities), drug or alcohol abuse, recent trauma, prior psychiatric problems, and past medical history (e.g., neurologic disorders, diabetes, renal failure, cancer, or liver failure). If you are having trouble getting historical information, search the patient's belongings for pill bottles, check the patient's wallet for telephone numbers or names of friends, and review previous medical records.

5. How can I perform a brief, directed physical examination on a patient with altered consciousness?

The goal of the physical examination is to differentiate structural focal CNS problems from diffuse metabolic processes. Pay special attention to vital signs, general appearance, mental status, eye findings, and the motor examination. Vital signs and eye findings are discussed elsewhere in this chapter.

The **general appearance** should be noted before examining the patient. Are there signs of trauma? Is there symmetry of spontaneous movements?

Motor examination is done to determine the symmetry of motor tone or strength and response of deep tendon reflexes.

6. How do I evaluate the patient's mental status?

Mental status can be assessed quickly. Ask four sets of progressively more difficult questions:

a. Orientation to person, place, and time.
b. Count backward from 10 (if done correctly, ask for serial 3s or 7s).
c. Recent recall of three unrelated objects.

7. What is the Glasgow Coma Scale?

A simple scoring system used in trauma patients to define the level of consciousness. It is useful for standardizing assessments among multiple observers and for monitoring changes in the degree of coma. The score is determined by eliciting the best response obtained from the patient in three categories (see Table 10-1). It is not sensitive enough to detect subtle alterations of consciousness in the noncomatose patient.

TABLE 10-1. GLASGOW COMA SCALE

Observation		Points
Eye opening	Spontaneous	4
	To verbal command	3
	To pain	2
	No response	1
Best verbal response	Oriented or converses	5
	Confused conversation	4
	Inappropriate words	3
	Incomprehensible sounds	2
	No response	1
Best motor response	Obeys	6
	Localizes pain	5
	Flexion withdrawal	4
	Decorticate posture	3
	Decerebrate posture	2
	No response	1
Total points		3–15

8. How important is measuring the temperature of the comatose patient?

Vital signs often provide clues to the cause of coma. A core temperature must be obtained. An elevated temperature should lead you to investigate the possibility of meningitis, sepsis, heat stroke, or hyperthyroidism. Hypothermia can result from environmental exposure, hypoglycemia, or, rarely, addisonian crisis. Do not assume that an abnormal temperature has a neurogenic cause until you eliminate other causes.

9. What is the significance of other vital signs?

- Check the **cardiac** monitor. Bradycardia or arrhythmias can alter cerebral perfusion and cause altered sensorium.
- Carefully count **respirations**. Tachypnea may indicate the presence of hypoxemia or a metabolic acidosis, and diminished respiratory efforts may require assisted ventilation.
- Check the **blood pressure**. Do not assume that hypotension has a CNS cause. Look for hypovolemia or sepsis as a cause for hypotension. Hypertension may be a result of increased intracranial pressure, but uncontrolled hypertension also may cause encephalopathy and coma.
- Do not forget to obtain the fifth vital sign—measurement of **oxygen saturation.**

10. What is Cushing's reflex?

An alteration of vital signs—increased blood pressure and decreased pulse—secondary to increased intracranial pressure.

11. Define decorticate and decerebrate posturing.

Posturing may be seen with noxious stimulation in a comatose patient with severe brain injury.

- **Decorticate posturing** is hyperextension of the legs with flexion of the arms at the elbows. Decorticate posturing results from damage to the descending motor pathways above the central midbrain.
- **Decerebrate posturing** is hyperextension of the upper and lower extremities; this is a graver sign. Decerebrate posturing reflects damage to the midbrain and upper pons. If you have trouble remembering which position is which, think of the upper extremities in flexion with the hands over the heart (*cor*) in de-*cor*-ticate posturing.

12. What information can be obtained from the eye examination of the comatose patient?

The eyes should be examined for **position**, **reactivity**, and **reflexes**. When the eyelids are opened, note the **position** of the eyes. If the eyes flutter upward, exposing only the sclera, suspect psychogenic coma. If the eyes exhibit bilateral roving movements that cross the midline, you know that the brain stem is intact. Pupil **reactivity** is the best test to differentiate metabolic coma from coma caused by a structural lesion because it is relatively resistant to metabolic insult and usually is preserved in a metabolic coma. Pupil reactivity may be subtle, necessitating use of a bright light in a dark room.

13. How do I test the eye reflexes?

Testing of the eye **reflexes** is the best method for determining the status of the brain stem. Two methods can be used:

a. Oculocephalic (doll's eyes)

Oculocephalic testing requires rapid twisting of the neck, which is a bad idea in the unconscious patient because occult cervical spine trauma may be present.

b. Oculovestibular (cold calorics)

Oculovestibular testing is easy to do and can be done without manipulating the neck. The ear canal is irrigated with 50 mL of ice water. A normal awake patient has two competing eye movements: rapid nystagmus away from the irrigated ear and slow tonic deviation toward the cold stimulus. Remember the mnemonic **COWS** (**C**old **O**pposite, **W**arm **S**ame), which refers to the direction of the fast component. (See Fig. 10-1.)

14. How do I interpret the eye reflexes?

A patient with psychogenic coma has normal reflexes and exhibits rapid nystagmus. A comatose patient with an intact brain stem lacks the nystagmus phase, and the eyes deviate slowly toward the irrigated ear. If the eyes do anything else (usually not a good sign), refer to a neurology text to determine the exact location of the lesion.

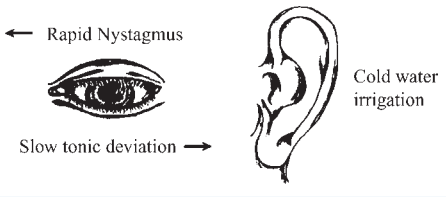

Figure 10-1. Testing of eye reflexes.

15. I want to impress the attending physicians. Do you have any tips on physical examination that will let me assume my rightful position as star student?

- If a confused patient is suspected of being postictal, look in the mouth. A tongue laceration supports the diagnosis of a seizure.
- Put on gloves and inspect the scalp. Occult trauma is often overlooked, and you may find a laceration or dried blood. An old scar on the scalp may tip you off to a posttraumatic seizure disorder.
- Do not be fooled by a *positive blink test* in a patient with suspected psychogenic coma. When you rapidly flick your hand at a comatose patient who has open eyes, air movement may stimulate a corneal reflex in a patient who is truly comatose.
- Do not be misled by the odor of alcohol. Alcohol has almost no detectable odor, which is why alcoholics drink vodka at work. Other spirited liquors such as brandy have a strong odor. The comatose executive who *smells drunk* may have had a sudden subarachnoid hemorrhage and spilled brandy on his or her shirt.

16. Which plain radiographs should be obtained in the comatose patient?

A cervical spine series (or cervical spine computed tomography [CT]) should be obtained in any comatose patient with suspected trauma because physical examination is unreliable. A chest radiograph may be helpful if hypoxemia, pulmonary infection, or aspiration is suspected.

17. Which diagnostic tests should be obtained in the patient with a significantly altered level of consciousness?

Obtain a rapid blood glucose, and correct hypoglycemia if it is found. Pulse oximetry should be obtained on all patients. If alcohol intoxication is suspected, determine the alcohol level with either a Breathalyzer or serum blood alcohol. If the pupils are constricted or if narcotic ingestion is suspected, intravenous naloxone should be given. If hypoglycemia or alcohol intoxication is not found to be the cause of the patient's confusion, further tests are warranted. A complete blood count, electrolytes, creatinine, blood urea nitrogen, and glucose should be obtained. Toxicologic screens may be done in a patient with a suspected ingestion, but they are expensive and do not detect routinely every possible ingested substance. Liver function tests, ammonia level, calcium level, carboxyhemoglobin level, and thyroid function studies may be helpful in selected patients.

18. When should I order a CT scan?

Although CT scans have revolutionized the practice of medicine, they are not indicated in every comatose patient. A good history, physical examination, and a few simple laboratory tests are adequate in many cases seen in the ED because drug and alcohol abuse are common. If a structural lesion is suspected (e.g., focal neurologic finding, head trauma, history of cancer), a noncontrast-enhanced CT scan should be ordered immediately. If the condition of a patient with a suspected metabolic coma worsens or does not improve after a brief period of observation, a CT scan should be obtained.

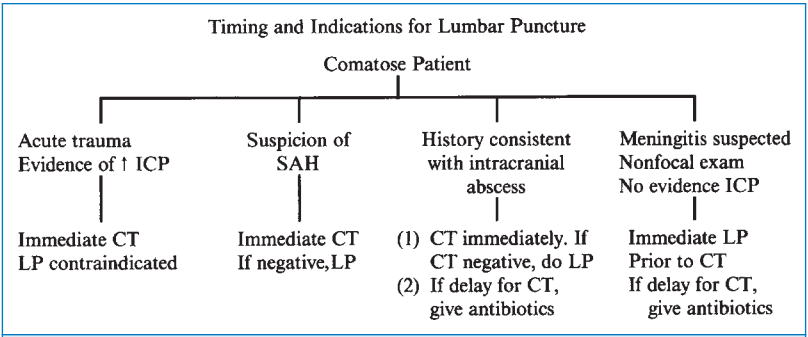

Figure 10-2. Timing and indications for lumbar puncture. CT, computed tomography; ICP, intracranial pressure; LP, lumbar puncture; SAH, subarachnoid hemorrhage.

19. When should a lumbar puncture (LP) be done?

The indications and timing of LP depend on two questions:

a. Is CNS infection suspected?

b. Is there a suspicion of a structural lesion causing increased intracranial pressure? (See Fig. 10-2.)

20. Okay. I have made the diagnosis of coma. What are my initial treatment priorities?

Emergency medicine requires simultaneous assessment and treatment. A brilliant diagnosis is useless in a dead patient. Start with the **ABCs**: airway, breathing, circulation and cervical spine. Intubate patients with apnea or labored respirations, patients who are likely to aspirate, and any patient who is thought to have increased intracranial pressure. Maintain cervical spine precautions until the possibility of trauma has been excluded. Hypotension should be corrected so that cerebral perfusion pressure is maintained.

KEY POINTS: ALTERED MENTAL STATUS AND COMA

1. Goal of physical examination is to differentiate structural from metabolic cause.
2. Focus on vital signs, mental status, and motor examination.
3. Check a rapid blood glucose on every comatose patient.
4. Immediately intubate the comatose patient if increased intracranial pressure is suspected.

21. I've addressed the ABCs. What do I do next?

Check a rapid blood glucose; if the glucose is low, treat hypoglycemia with D50W. It is better to do a rapid blood glucose determination rather than to give glucose empirically. Next, give 100 mg of thiamine, and if opioid use is suspected, give 2 mg of naloxone intravenously. Empirical administration of flumazenil (benzodiazepine antagonist) or physostigmine (reverses anticholinergic agents) is not routinely indicated in comatose patients. Antibiotic administration is considered in all febrile patients with coma of unknown origin. A comatose patient with increased intracranial pressure should be intubated and mechanically ventilated. Mannitol, 0.5 to 1 gm/kg intravenously, should be considered.

22. I think my patient is faking it. How can I tell if this is psychogenic coma?

First, be grateful. A patient in psychogenic coma is better than one who is angry and combative. Approach the patient incorrectly, and you can awaken the patient to a hostile alert state.

- Do a careful neurologic examination. Open the eyelids. If the eyes deviate upward and only the sclera show (Bell's phenomenon), you should suspect psychogenic coma. When the eyelids are opened in a patient with true coma, the lids close slowly and incompletely. It is difficult to fake this movement.
- Lift the arm and drop it toward the face; if the face is avoided, this is most likely psychogenic coma. If this does not work, you may want to check some simple laboratory tests, including a Dextrostix.
- If the patient remains comatose, irritating but nonpainful stimuli, such as tickling the feet with a cotton swab, may elicit a response. Remember that this is not a test of wills between you and the patient. There is no indication for repetitive painful stimulation because it can make the patient angry and ruin attempts at therapeutic intervention.
- If all else fails, perform cold caloric testing. The presence of nystagmus confirms the diagnosis of psychogenic coma. What do you do then? It is time to pick up a copy of *Psychiatry Secrets*.

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FEVER

Diane M. Birnbaumer, MD, FACEP, and Sarah M. Battistich, MD

1. What temperature constitutes a fever?

A temperature of 38°C (100.4°F) in infants or 38.3°C (100.9°F) in adults defines a fever.

However, immunocompromised or functionally immunocompromised patients may not be able to mount a temperature high enough to constitute a fever by this definition. In these patients *low-grade* temperature elevations should be addressed cautiously. Examples of patients in which the clinician should maintain a high index of suspicion for masked fever include the elderly, diabetics, intravenous drug users, chronic alcoholics, people with HIV/AIDS, people on chronic steroids or immune-modulating drugs, and neutropenic patients.

2. Are all methods of measuring temperature equivalent?

Rectal temperatures are the most accurate representation of core body temperature and are, therefore, considered the gold standard. Oral, axillary, and tympanic temperature measurements lack sensitivity, and thus a lack of fever when measured by these methods does not rule out a fever. In addition, there is no reliable correction factor for these alternate modalities. When an accurate temperature measurement is crucial to the patient's care, a rectal temperature measurement is necessary.

3. How does the body create fever?

Core body temperature is controlled by the anterior hypothalamus. A fever is caused by elevation of the hypothalamic set point. The body responds by attempting to generate heat (e.g., by shivering or by increasing the basal metabolic rate) to elevate core temperature.

4. What is the difference between a fever and hyperthermia?

In contrast to fever, hyperthermia results in an elevated temperature without alteration of the hypothalamic set point. In cases of hyperthermia, the body attempts to cool itself to achieve a normal temperature, primarily by increasing sweating. A temperature of 41.5°C (106.7°F) or greater usually represents hyperthermia and not a true fever, especially in adults. Some examples of hyperthermia include heat stroke, thyroid storm, burns, and toxidromes, such as neuroleptic malignant syndrome, serotonin syndrome, and malignant hyperthermia.

5. How do I address a patient with a subjective fever at home who is afebrile in the ED?

This situation is mostly commonly encountered in pediatrics. Mothers are accurate in assessing the presence or absence of a fever 50% to 80% of the time, and they seem to be more accurate at detecting when the child is febrile than they are at determining that the child is afebrile. Most experts feel that palpable fevers reported by mothers are probably real and need to be taken seriously. Additionally, the practice of attributing fevers to bundling has been disproved; bundling does not alter core body temperatures in infants.

6. Does the degree of fever indicate the severity of the illness?

In general, no. There is no degree of fever that has been clearly associated with a specific risk of serious infection in patients. The exception to this may be in nonimmunized children; prior to the widespread use of the *Haemophilus influenzae*

vaccine, temperatures over 41.1°C (105.98°F) were associated with a higher incidence of serious bacterial illness in children. Prior to the approval of the pneumococcal conjugate vaccine in 2000, occult pneumococcal bacteremia was observed to be three times (10% vs. 3%, respectively) more likely in children with a fever of 39.5°C (103.1°F) or greater versus a fever of 39.0°C (102.2°F) or greater.

7. What is the best way to reduce a fever?

Most physicians use antipyretics for patients who are uncomfortable because of fever. Within the range of 40°C to 42°C, there is no evidence that fever is injurious to tissue. Use of antipyretics should be considered in pregnant women and patients with preexisting cardiac compromise who would not tolerate the increased metabolic demands of a fever. Acetaminophen is the antipyretic of choice in most hospitals. Ibuprofen, other nonsteroidal anti-inflammatory drugs (NSAIDS), and aspirin are also effective. However, due to the association with Reye's syndrome, aspirin is usually not recommended for children. Response to these agents is seen with both serious and benign causes of fever. Recurrence of fever after antipyretics wear off is often concerning for parents, but it does not distinguish between serious and benign causes of fever, and parents should be encouraged to base their concerns on the child's behavior rather than the height of the fever or its response to antipyretics. Complementary methods, such as cool bathing and undressing the patient, are generally not felt to be effective at significantly lowering core body temperature and should be reserved as adjuncts for higher temperatures. If the temperature is above 41.5°C (106.7°F), the diagnosis of hyperthermia should be considered and rapid cooling measures used if any concern about this condition exists. (See Chapter 58.)

8. What are the causes of fever?

First and foremost, at the top of the list is infection (both bacterial and viral). Infection causes the vast majority of fevers, but other causes must also be included in the differential diagnosis:

- Neoplastic diseases (e.g., leukemia, lymphoma, or solid tumors)
- Collagen vascular diseases (e.g., giant cell arteritis, polyarteritis nodosa, systemic lupus erythematosus, or rheumatoid arthritis)
- Central nervous system lesions (e.g., stroke, intracranial bleed, or trauma)
- Illicit drug use (cocaine, ecstasy [MDMA], or methamphetamines)
- Withdrawal syndromes (delirium tremens or benzodiazepine withdrawal)
- Factitious fever
- Medications

9. Which medications can cause fevers?

Any drug is capable of producing a *drug fever*; however, the most common culprits are penicillin and penicillin analogs (see Table 11-1). The fever usually begins 7 to 10 days after initiation of drug therapy. There is an associated rash or eosinophilia in about 20% of cases. Drug fever should always be a diagnosis of exclusion.

10. What are some key elements of the history and physical in patients with fever?

Pay particular attention to associated symptoms (e.g., cough, dysuria, diarrhea, or headache), duration of fever, ill contacts, history or risk of immunocompromise, and past medical history, particularly comorbid illnesses. In the physical examination, note the general appearance of the patient, paying particular attention to subtleties, such as mild mental status changes or rashes that might be indicative of more serious systemic diseases. In addition to a thorough routine physical examination, in appropriate cases a more detailed examination of the patient should be done to look for occult sites of infection, such as the nose/sinuses, rectum (i.e., prostatitis, perirectal abscess), and pelvic examination (i.e., pelvic inflammatory disease, tubo-ovarian abscess).

TABLE 11-1. DRUGS COMMONLY ASSOCIATED WITH DRUG FEVERS

Antibiotics	Phenytoin
Isoniazid (INH)	Procainamide
Nitrofurantoin	Quinidine
Penicillins, cephalosporins	**Anticonvulsants**
Rifampin	Phenytoin
Sulfonamides	Carbamazepine
Anticancer drugs	**Nonsteroidal anti-inflammatory drugs**
Bleomycin	Ibuprofen
Streptozocin	Salicylates
Cardiac drugs	**Others**
Hydralazine	Barbiturates
Methyldopa	Cimetidine
Nifedipine	Iodides

11. What is the relationship between fever and tachycardia?

The pulse should increase about 10 beats per minute for each 0.6°C (1°F) increase in temperature. A *pulse-temperature dissociation* occurs when the patient has a fever but a heart rate that is lower than would be expected for the degree of fever. This dissociation occurs in typhoid, malaria, Legionnaires' disease, and mycoplasma. In early septic shock, tachycardia that is inappropriate for the degree of fever is often seen. Tachypnea out of proportion to fever is characteristic of pneumonia and gram-negative bacteremia. Hypotension, particularly paired with tachycardia, raises the concern of sepsis.

12. Do all septic patients have a fever?

No, in fact, remember that within the definition of systemic inflammatory response syndrome (SIRS) is temperature greater than 38°C (100.4°F) or less than 36°C (96.8°F). Not all fevers are caused by infection, and not all infected patients have a fever.

13. Should everyone with a fever get antibiotics?

Absolutely not. Antibiotic use should be based on the patient's specific presentation and diagnosis after an appropriate history and physical examination and directed laboratory and ancillary tests. Most clinicians advocate giving antibiotics immediately to any patient who appears toxic or has suspected bacterial meningitis, without delaying for results of ancillary test or culture results. Other patients who should be considered for early antibiotics are immunocompromised patients and elderly patients.

14. What is a *neutropenic fever*, and what is the appropriate workup?

In patients with neutropenia (an absolute neutrophil count below 1,000 per square mm), a single temperature above 38.3°C (100.9°F) is considered a fever, and fever in these patients is secondary to infection until proven otherwise. The risk of severe sepsis and septicemia is higher in these patients, and this initial workup should include screening for all sources of infection with history and physical. Initial studies should include, at a minimum, a cell count and differential, metabolic panel, blood cultures, chest radiograph, and urinalysis; other studies should be ordered as indicated. All these patients should receive antibiotics, and most are admitted to the hospital.

15. What is a fever of unknown origin (FUO)?

The classic 1961 definition by Petersdorf, et al., defines FUO as a fever greater than 38.3°C (100.9°F) documented on several occasions during a period longer than 3 weeks, with an uncertain diagnosis after 1 week of evaluation in the hospital. The most common cause of FUO is occult infection (particularly tuberculosis) and malignancy, each accounting for approximately 30% of cases.

16. For how long do typical febrile illnesses last?

In most cases, the fever resolves within 3 to 7 days.

17. How do I approach a child younger than 3 years with a fever?

See Chapter 60.

CONTROVERSY

18. Is a fever a friend or foe?

This question has been controversial for centuries. Although fever per se is self-limiting and rarely serious, it is often considered by patients and doctors to be a major and harmful sign of illness, and parents and medical practitioners may develop what has been termed *fever phobia*, treating the fever almost as an illness in itself rather than a symptom. More and more research is proving, however, that fever may be beneficial in fighting some infections. Higher temperatures increase the activity of neutrophils and lymphocytes and decrease the levels of serum iron, a substrate that many bacteria need to reproduce. It enhances immunological processes, including the activity of IL-1, T helper cells and cytolytic T cells, and B cell and immunoglobulin synthesis. A study of children with meningitis observed those with a fever had mortality benefit over those who were afebrile or hypothermic. A Finnish study of children with salmonella gastroenteritis demonstrated a significant negative correlation between the degree of fever and the duration of excretion of organisms.

19. Many physicians recommend alternating acetaminophen and ibuprofen for fevers. Is this effective?

This is not an evidence-based practice. There is presently no scientific evidence that this combination is safe or achieves faster antipyresis than an adequate dose of either agent alone. The observed fever reduction of 0.5°C when combining antipyretics, compared with a single antipyretic, is insufficient to warrant routine use. Additionally, alternating antipyretics can be confusing for caregivers, potentially leading to incorrect dosing of either product. The practice can also increase parents' fever phobia because it increases parental preoccupation with the height of the fever.

KEY POINTS: FEVER

1. Increased temperature may be indicative of either a fever or hyperthermia due to another cause, such as heat illness, particularly in times of increased ambient temperatures.
2. Response to antipyretics does not differentiate serious from more benign causes of fever.
3. The degree of temperature elevation is not predictive of serious illness.

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CHAPTER 12

CHEST PAIN

Eric A. Wong, MD, and Shamai A. Grossman, MD, MS

1. Why is the cause of chest pain often difficult to determine in the ED?

- Numerous disease processes in a variety of organs may result in chest pain.
- The severity of the pain is often unrelated to its life-threatening potential.
- The location of the pain as perceived by the patient frequently does not correspond with its source.
- Reproducible chest pain can have a cardiac etiology.
- Physical findings, laboratory assays, and radiologic studies are often non-diagnostic in the ED.
- More than one disease process may be present.
- The causes of acute chest pain often can be dynamic processes.

2. Why is the location of chest pain not diagnostic of its cause?

Somatic fibers from the dermis are numerous and enter the spinal cord at a single level, resulting in sharp, localized pain. Visceral afferent fibers from the thorax and upper abdomen are less numerous. They enter the spinal cord at multiple levels, resulting in a pain that is dull, aching, and poorly localized. Connections between the visceral and somatic fibers may result in the visceral pain being perceived as originating from somatic locations, such as the shoulder, arm, neck, or jaw.

3. List life-threatening causes of acute chest pain that must be considered first when evaluating a patient in the ED.

- Myocardial infarction
- Aortic dissection
- Unstable angina
- Pulmonary embolism (PE)
- Pneumothorax
- Acute pericarditis
- Esophageal rupture
- Myocarditis

4. List some other conditions that may present with chest pain.

- Stable angina
- Valvular heart disease
- Pneumonia
- Gastroesophageal reflux disease (GERD)
- Esophageal spasm
- Thoracic outlet syndrome
- Mediastinitis
- Musculoskeletal pain
- Peptic ulcer disease
- Cholecystitis
- Pancreatitis
- Herpes zoster
- Sickle cell anemia
- Anxiety

5. What is the best initial approach to patients presenting with chest pain?

All patients with acute chest pain should be approached with the assumption that a life-threatening cause is present. Once patient stability is established, with few exceptions, supplemental oxygen, intravenous access, and cardiac monitoring should be initiated (Fig. 12-1) before any diagnostic studies are started.

6. How do I evaluate the patient with chest pain?

An accurate history is the most important component of the evaluation. Factors to be considered include onset, character and quality, severity, location, pattern of radiation, duration of pain, and associated symptoms. Precipitating factors, such as exertion, movement, or inspiration, and relieving factors, such as rest or body position, may provide clues to the origin of the pain (Table 12-1). Relief of chest pain with nitroglycerin is not useful in distinguishing between cardiac and noncardiac causes of chest pain.

7. What are the major risk factors associated with ischemic heart disease, PE, and aortic dissection?

See Chapters 27, 29, and 31.

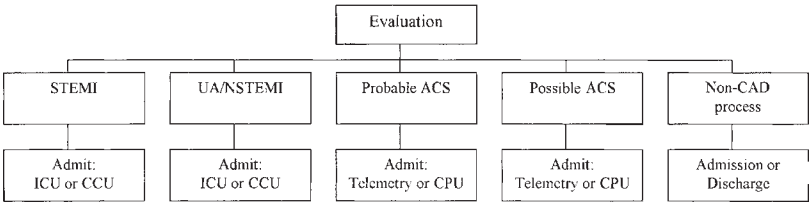

Figure. 12-1. Evaluation of chest pain. ACS, acute coronary syndrome; CAD, coronary artery disease; CCU, coronary care unit; CPU, chest pain observation unit; ICU, intensive care unit; NSTEMI, non-ST segment elevation myocardial infarction; STEMI, ST segment elevation myocardial infarction, UA, unstable angina.

8. Are risk factors for cardiac ischemia useful in the ED?

Although the classic risk factors for cardiac ischemia are useful in determining long-term risk of a patient developing coronary artery disease, risk factors have very limited utility in the ED setting when trying to determine the acute risk of acute coronary syndrome (ACS). Recent American College of Cardiology/American Heart Association (ACC/AHA) guidelines suggest that the most important factor in predicting ACS in a patient presenting with chest pain is the history of present illness, rather than cardiac risk factors.

9. Are there any useful clinical prediction rules for risk stratifying patients with suspected PE?

Yes. See Chapter 27.

10. Is radiation of chest pain significant?

Radiating chest pain is suggestive but not diagnostic of cardiac ischemia. Visceral pain, including aortic, esophageal, gastric, and pulmonary processes, may present with radiation of pain to the neck, shoulder, or arm. Chest pain that radiates to the arms increases the likelihood of acute myocardial infarction, with radiation only to the right arm having a higher likelihood than radiation to both arms, which interestingly has a higher likelihood than radiation to only the left arm.

11. How does the patient's appearance correlate with the origin of chest pain?

Catastrophic illnesses often result in anxiety, diaphoresis, and an ill appearance. Splinting may be caused by PE, pleurisy, pneumothorax, pneumonia, or musculoskeletal chest pain. Levine's sign, which consists of a patient placing a clenched fist over the sternum to describe the pain, is frequently associated with ischemic heart disease. Kussmaul's sign is a paradoxical filling of

TABLE 12–1. CLASSIC PATTERNS OF CHEST PAIN

Etiology	Quality	Location	Radiation	Duration	Associated Symptoms	Onset
Myocardial infarction	Visceral	Retrosternal	Neck, jaw, shoulder, arm	>15 min	Nausea, vomiting, diaphoresis, dyspnea	Variable
Angina	Visceral	Retrosternal	Neck, jaw, shoulder, arm	5–15 min	Nausea, diaphoresis, dyspnea	Gradual
Aortic dissection	Severe, tearing	Retrosternal	Interscapular	Constant	Nausea, dyspnea, diaphoresis	Sudden
Pulmonary embolism	Pleuritic	Lateral		Constant	Dyspnea, apprehension	Sudden
Pneumothorax	Pleuritic	Lateral	Neck, back	Constant	Dyspnea	Sudden
Pericarditis	Sharp, stabbing	Retrosternal	Neck, back shoulder, arm	Constant	Dyspnea, dysphagia	Variable
Esophageal rupture	Boring	Retrosternal, epigastric	Posterior thorax	Constant	Diaphoresis, dyspnea (late)	Sudden
Esophagitis	Aching, boring	Retrosternal	Interscapular	Minutes to hours	Dysphagia	Variable
Esophageal spasm	Visceral	Retrosternal	Interscapular	Minutes to hours	Dysphagia	Variable
Musculoskeletal	Sharp, aching, superficial	Localized		Variable	Dyspnea	Variable

the neck veins during inspiration, suggesting a right ventricular infarction, PE, or pericarditis with associated cardiac tamponade.

12. How are vital signs helpful?

A blood pressure difference of more than 20 mm Hg between the upper extremities or a loss or reduction of lower extremity pulses is suggestive of an aortic dissection. The presence of tachycardia should raise the suspicion of serious pathology with severe pain or anxiety as diagnoses of exclusion. Tachypnea may be caused by a PE, pneumonia, or pneumothorax, or may be secondary to pain. An elevated temperature usually indicates an inflammatory or infectious process, such as pericarditis or pneumonia. However, fever can also be associated with an acute myocardial infarction or PE.

13. Which physical examination findings may help differentiate the causes of acute chest pain?

Isolated physical findings are rarely diagnostic of the origin of chest pain, but when used in context with the history, they may be extremely valuable. Palpation may reveal localized tenderness and reproduce musculoskeletal pain, but 5% to 10% of patients with ACS have chest pain and associated palpable chest tenderness. Cardiac auscultation may reveal a new murmur of aortic insufficiency suggestive of aortic dissection or a new murmur of mitral regurgitation secondary to papillary muscle dysfunction from ACS. A third or fourth heart sound increases the likelihood of ACS. A pericardial friction rub is associated with pericarditis. Mediastinal air from an esophageal or bronchial rupture results in a crunching sound called *Hamman's sign*. Decreased breath sounds, localized subcutaneous emphysema, or hyperresonance may indicate a pneumothorax. Localized rales suggest pulmonary pathology as the cause of the chest pain. Patients with unilateral leg swelling, pitting edema of one leg, tenderness over the deep venous system, or calf swelling may have PE as the cause of their chest pain.

14. How is the electrocardiogram (ECG) helpful in the evaluation of chest pain?

The ECG findings most often associated with ACS are ST segment elevation, ST segment depression, inverted T waves, and new bundle branch blocks. However, the initial ECG may be normal in 20% to 50% of ED patients who are later diagnosed as having had an acute myocardial infarction. Furthermore, there is no difference in the frequency of ACS between patients with chest pain at the time of acquisition of a normal ECG and those without chest pain during acquisition of a normal ECG. Comparison with previous ECGs is critical when possible. In pericarditis, the initial ECG changes may consist of diffuse ST elevation with depression of the PR segment. The ECG associated with a PE most often demonstrates a normal sinus rhythm. Common ECG findings associated with acute PE are sinus tachycardia or nonspecific ST-T wave abnormalities in the right precordial leads. Right heart strain secondary to a PE will result in a peaked P wave, right axis deviation, or a prominent S wave in lead I; a Q wave in lead III; and a new T-wave inversion in lead III (S1 Q3 T3 pattern); however, the S1 Q3 T3 pattern associated with PE occurs infrequently.

15. What abnormalities may appear on the chest radiograph in diseases causing chest pain?

The chest X-ray films of patients presenting with chest pain are frequently normal but may be diagnostic, as in a pneumothorax. The mediastinum will be shifted away from the side of a tension pneumothorax. Aortic dissection may show a widened mediastinum, depression of the left main stem bronchus, loss of the paratracheal stripe or a 4- to 5-mm or greater separation between the calcified intima and the lateral edge of the aortic knob. A PE usually will show nonspecific signs such as atelectasis or an elevated hemidiaphragm. Rare PE signs include Hampton's hump, a wedge-shaped, pleural-based infiltrate representing an area of infarction, and Westermark's sign, which is an absence of pulmonary shadows distal to a central embolism. Pneumonia typically produces one or more areas of pulmonary consolidation, a pleural effusion, or cavitation. Esophageal rupture is classically associated

with subcutaneous emphysema, pneumomediastinum, a left-sided pleural effusion, or a left-sided pneumothorax.

16. Are cardiac enzymes useful in the evaluation of chest pain in the ED?
Yes. See Chapter 29.

17. Are there any bedside tests or medications that may help to identify the origin of acute chest pain?
Several bedside tests may be helpful, but they are rarely diagnostic in themselves. Relief with nitroglycerin occurs in both angina and esophageal spasm, whereas acute myocardial infarction and unstable angina (ACSs) may remain unrelieved. The use of nitroglycerin as a diagnostic test should be avoided. Antacids or *gastrointestinal cocktails*, consisting of viscous lidocaine and an antacid, frequently resolve esophageal pain but also relieve pain in 7% of patients with angina. The use of antacids as a diagnostic test should be avoided. Pain from pericarditis is frequently worse in the supine position and relieved when leaning forward. Pain from esophageal disease is worsened with changes in position such as leaning forward or lying down. Musculoskeletal pain is worsened with movement.

18. Are there any other useful diagnostic imaging studies to help determine the cause of chest pain?
Select low-to-moderate risk patients may be risk stratified for ACS with coronary computed tomography (CT) angiography. Aortic dissection may be diagnosed by a thoracic arteriogram, a rapid-sequence CT with intravenous contrast scan, magnetic resonance imaging (MRI), or by a transesophageal echocardiogram. A suspected PE may be confirmed by a ventilation-perfusion scan, spiral CT scan of the thorax, or pulmonary angiography. Esophageal rupture may be diagnosed by an esophagogram with a water-soluble contrast material.

19. What special considerations must be taken into account when evaluating chest pain in geriatric, diabetic, or female patients?
Although the sources of chest pain in the elderly do not differ significantly from the general population, their presenting symptoms are often atypical. Instead of chest pain, ischemic heart disease may manifest as sudden progressive dyspnea, abdominal or epigastric fullness, extreme fatigue, confusion, or syncope. Patients with diabetes mellitus may have altered pain perception, resulting in an atypical presentation similar to that of the elderly. The risk of coronary heart disease in women increases with menopause. Women with ischemic heart disease present in atypical patterns more frequently than men. This is because of the higher prevalence of less common causes of ischemia such as vasospastic and microvascular angina.

KEY POINTS: CHEST PAIN

1. The primary goal of the evaluation of acute chest pain is the inclusion or exclusion of a life-threatening disease process.
2. A normal ECG on initial presentation does not exclude an ACS.
3. Of patients with ACS, 25% do not have a primary complaint of chest pain.
4. Chest pain relieved by nitroglycerin or antacids is not diagnostic for cardiac or noncardiac disease.
5. Chest pain in elderly and diabetic patients is caused by common diseases but often presents in atypical fashion.

20. Because approximately 2% to 4% of patients with chest pain caused by acute myocardial infarction are discharged to home, what factors have been associated with failure to make the diagnosis?

- Young age group
- Failure to obtain an accurate history
- Incorrect interpretation of the ECG
- Failure to recognize atypical presentations
- Hesitance to admit patients with vague symptoms
- Reliance on laboratory assays, such as cardiac enzymes
- Insufficient experience or training

WEBSITES

American Heart Association: www.americanheart.org

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ABDOMINAL PAIN

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CHAPTER 13

1. What is the difference between visceral and somatic pain? How is this of practical importance?

Evolving patterns of pain frequently reveal the source and give an idea of the extent to which the process has advanced. Early, the patient may describe a deep-seated, dull pain **(visceral pain)** emanating from hollow viscera or the capsule of solid organs. This pain is poorly localized but generally falls somewhere along the midline of the abdomen. Later, as inflammation progresses to the parietal peritoneum, the pain becomes better localized, lateralized over the involved organ, sharper in intensity **(somatic** or **parietal pain)**, and constant. Visceral pain that is superseded by somatic pain frequently signals the need for surgical intervention. A clear understanding of the process enables the clinician to identify more precisely the cause and rate of progression of pathology.

2. What is the difference between localized and generalized peritonitis?

As the peritoneum adjacent to a diseased organ becomes inflamed, palpation or any abdominal movement causes stretching of the sensitized peritoneum and, consequently, pain localized at that site (localized peritonitis). If irritating material (e.g., pus, blood, or gastric contents) spills into the peritoneal cavity, the entire peritoneal surface may become sensitive to stretch or motion, and any movement or palpation may provoke pain at any or all points within the abdominal cavity (generalized peritonitis).

KEY POINTS: MESENTERIC ISCHEMIA

1. Abdominal pain out of proportion to physical findings.
2. Diffuse abdominal tenderness, rebound, and rigidity are ominous signs.
3. Definitive diagnosis by mesenteric arteriography or surgical exploration.

3. Which tests for peritoneal irritation are best?

Rebound tenderness is the traditional physical examination finding for peritonitis. In a patient with likely generalized peritonitis (e.g., obvious distress, excruciating pain every time the ambulance hits a bump), the standard tests for rebound tenderness are unnecessarily harsh. Asking the patient to cough generally supplies adequate peritoneal motion to give a positive test. When in every respect the examination is normal, one highly sensitive test for peritoneal irritation is the heel-drop jarring (Markle) test. The patient is asked to stand, rise up on tiptoe with knees straight, and forcibly drop down on both heels with an audible thump. Among patients with appendicitis, this test was found to be 74% sensitive, compared with 64% for the standard rebound test.

4. Why is it important to establish the temporal relationship of pain to vomiting?

Generally, pain preceding vomiting is suggestive of a surgical process, whereas vomiting before onset of pain is more typical of a nonsurgical condition. Epigastric pain that is relieved by vomiting suggests intragastric pathology or gastric outlet obstruction.

5. **What is the relationship of peritoneal inflammation to loss of appetite?**
Anorexia, nausea, and vomiting are directly proportional to the severity and extent of peritoneal irritation. The presence of appetite does not rule out a surgically significant inflammatory process, such as appendicitis. A retrocecal appendicitis with limited peritoneal irritation may be associated with minimal gastrointestinal (GI) upset, and one third of all patients with acute appendicitis do not report anorexia on initial presentation.

6. **Discuss the pitfalls in evaluating elderly patients with acute abdominal pain.**
Advanced age may and often does blunt the manifestations of acute abdominal disease. Pain may be less severe; fever often is less pronounced, and signs of peritoneal inflammation, such as muscular guarding and rebound tenderness, may be diminished or absent. Elevation of the white blood cell (WBC) count is also less sensitive. Cholecystitis, intestinal obstruction, and appendicitis are the most common causes for acute surgical abdomen in the elderly. Because of atypical clinical presentations, additional screening tests, such as amylase, liver function studies, and alkaline phosphatase, and the liberal use of ultrasound or computed tomography (CT) scan may be useful in this age group.

7. **What other factors should be sought in the history that may alter significantly the presentation of patients with abdominal pain?**
Symptoms and physical findings in patients with schizophrenia and diabetes may be muted significantly. The prior use of steroids or antibiotics may alter signs and laboratory results substantially.

KEY POINTS: APPENDICITIS

1. The most sensitive findings are right lower quadrant tenderness, nausea, and anorexia.
2. Clinical scoring systems are useful for risk stratification but not for excluding the diagnosis.
3. Ultrasound, CT, and magnetic resonance imaging (MRI) are most useful in equivocal cases or patient groups with multiple possible alternative diagnoses.

8. **What is the significance of obstipation?**
Obstipation is the inability to pass either stool or flatus for more than 8 hours despite a perceived need, and is highly suggestive of intestinal obstruction.

9. **What vital sign is associated most closely with the degree of peritonitis?**
Tachycardia is virtually universal with advancing peritonitis. The initial pulse is less important than serial observations. An unexplained rise in pulse may be an early clue that early surgical exploration is indicated. However, this response may be blunted or absent in elderly patients.

10. **Does the duration of abdominal pain help in categorizing cause?**
Severe abdominal pain persisting for 6 or more hours is likely to be caused by surgically correctable problems. Patients with pain of more than 48 hours' duration have a significantly lower incidence of surgical disease than do patients with pain of shorter duration.

11. **Name the two most commonly missed surgical causes of abdominal pain.**
Appendicitis and acute intestinal obstruction.

12. **Is there a place for narcotic analgesics in the management of acute abdominal pain of uncertain cause?**
For fear of masking vital symptoms or physical findings, old, conventional surgical wisdom proscribes the use of narcotic analgesics until a firm diagnosis is established. Increasingly,

however, studies have demonstrated that pain medication may be given to selected patients with stable vital signs because the analgesic effect may be reversed readily at any time by the administration of naloxone. In a review article, Ranji et al found that pain control with opiates may alter the physical examination findings, but these changes result in no significant increase in management errors. Although inconclusive, a growing body of data suggests that evaluation of acute abdominal disease may be facilitated when severe pain has been controlled and the patient can cooperate more fully.

13. Which are the most useful preliminary laboratory tests to order?

A complete blood count with differential and urinalysis are generally recommended. The initial hematocrit helps to define antecedent anemia. An elevated WBC count suggests significant pathology but is nonspecific. Elevated urinary specific gravity reflects dehydration, and an increased urinary bilirubin in the absence of urobilinogen points toward total obstruction of the common bile duct. Pyuria, hematuria, and a positive dipstick for glucose and ketones may reveal nonsurgical causes for abdominal pain. For patients with epigastric or right upper quadrant pain, lipase and liver function studies are advised. Any woman with childbearing capability should receive a pregnancy test. Serum electrolytes, glucose, blood urea nitrogen, and creatinine are indicated if there is clinical dehydration or other reason to suspect abnormality such as renal failure, diabetes, or a metabolic acidosis.

14. Are plain radiographs always indicated?

No. Plain films of the abdomen have the highest yield when used in the evaluation of patients with suspected bowel obstruction, intussusception, ileus, and free air secondary to a perforated viscus. They have much less utility in detecting intra-abdominal mass, renal calculi, diverticulitis, gallbladder disease, and abdominal aortic aneurysms. If these disorders are suspected, other studies such as ultrasound or abdominal CT are more appropriate. Conversely, among patients with uncomplicated peptic ulcer disease or massive hematemesis, pain present for more than 1 week, strangulated abdominal wall hernias, or other obvious clinical indications for laparotomy, plain radiographs add little.

15. Which plain films are most useful?

Traditional teaching holds that plain abdominal films should include a supine view, plus either an upright view or a left lateral decubitus view (if unable to stand).

The **supine view of the abdomen** is the most informative and worthwhile abdominal film. The upright film is superior for visualizing air-fluid levels associated with ileus, obstruction, or biliary air. The **erect chest radiograph** is most sensitive for detection of free intraperitoneal air and may show basal pneumonia, ruptured esophagus, elevated hemidiaphragm, air-fluid levels associated with subdiaphragmatic or hepatic abscess, pleural effusion, and pneumothorax. In the evaluation of patients with abdominal pain, the upright chest film, taken alone, has been shown to be more useful than films of the abdomen itself.

16. Are air-fluid levels within the intestine always abnormal?

No. It is commonly taught that air-fluid levels when seen on an upright abdominal film are *pathognomonic* for small bowel obstruction. A study of 300 normal patients by Gammill and Nice showed, however, that the average number of air-fluid levels was four per patient, with some films showing 20. Although typically less than 2.5 cm in length, some were 10 cm. Most of the air-fluid levels were found in the large bowel; only 14 of 300 normal patients studied showed air-fluid levels in the small bowel. The authors suggested that before air-fluid levels are used as the sole criterion for the diagnosis of paralytic ileus or mechanical obstruction, one should see more than two air-fluid levels within dilated loops of the small bowel.

17. A 7-year-old child presents with acute abdominal pain with a history of several similar bouts over the past 5 months. Physical examination is unremarkable. What is the most likely cause?

In children older than age 5, abdominal pain that is intermittent and of more than 3 months' duration is **functional** in greater than 95% of cases, especially in the absence of objective findings such as fever, delayed growth patterns, anemia, GI bleeding, or lateralizing pain and tenderness.

KEY POINTS: COMMON DISEASES THAT CAN SIMULATE ACUTE ABDOMEN

1. DKA
2. Food poisoning
3. Pneumonia
4. Pelvic inflammatory disease

18. A patient with severe abdominal pain is found to be in diabetic ketoacidosis (DKA). How do I decide whether the abdominal pain is a manifestation of the DKA or whether a surgical condition has precipitated DKA?

Patients with established DKA often present to the ED with severe abdominal pain. Although the precise mechanism of abdominal pain and ileus in patients with DKA is not well understood, hypovolemia, hypotension, and a total body potassium deficit probably contribute. An acute surgical lesion may initiate DKA; nevertheless, most patients in DKA have no such pathology. Abdominal symptoms characteristically resolve as medical treatment restores the patient to biochemical homeostasis. Treatment of the DKA must precede any surgical intervention because of the extremely high intraoperative mortality among patients not so stabilized. If symptoms persist despite adequate correction of DKA, then an underlying surgical pathology becomes more likely.

19. Is a rectal examination necessary in the patient with suspected acute appendicitis?

The literature is inconsistent as to its usefulness in aiding the diagnosis; however, failure to perform a rectal examination is cited frequently in successful malpractice claims. Some other diseases may be effectively diagnosed only by rectal examination (e.g., prostatitis or occult GI bleed).

20. Is there a reliable diagnostic test that will either rule in or rule out appendicitis?

No, not yet anyway. Kentsis et al, have shown that high-accuracy mass spectrometry urine proteome profiling allowed identification of diagnostic markers of acute appendicitis. These biomarkers may add significantly to the diagnostic accuracy of appendicitis in the near future.

WEBSITES

www.emedicine.medscape.com
Acute appendicitis: www.emedicine.medscape.com/article/773895-overview

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CHAPTER 14

NAUSEA AND VOMITING

Juliana Karp, MD

1. Vomiting? Do I really need to read this chapter when there are so many more interesting chapters in this book?

Yes! One of the most common and harmful mistakes made in the ED is assuming that nausea and vomiting are the result of gastroenteritis without thinking of and ruling out more serious causes. In addition, vomiting is one of the most common presenting complaints in the ED.

2. What causes vomiting?

The act of vomiting is a highly complex act involving a vomiting center in the medulla. This center may be excited in four ways:

- Via vagal and sympathetic afferents from the peritoneum; gastrointestinal, biliary, and genitourinary tracts; pelvic organs; heart; pharynx; head; and vestibular apparatus.
- By impulses converging at the nucleus tractus solitarius in the medulla.
- Via the chemoreceptor trigger zone located in the floor of the fourth ventricle.
- Via the vestibular or vestibulocerebellar system (motion sickness and some medication-induced emesis).

3. Can vomiting itself lead to potential complications?

Yes. Some of these are life-threatening:

- Esophageal perforation or Mallory-Weiss tear
- Severe dehydration
- Metabolic alkalosis
- Severe electrolyte depletion (particularly sodium, potassium, and chloride ions)
- Pulmonary aspiration
- Esophageal or gastric bleeding

4. List the common gastrointestinal disorders that cause vomiting.

- Gastroenteritis
- Gastric outlet obstruction
- Gastric retention
- Alcoholic gastritis
- Pancreatitis
- Hepatitis
- Small bowel obstruction
- Appendicitis
- Cholecystitis
- Diabetic gastroparesis

KEY POINTS: CHARACTERISTICS OF GASTROENTERITIS

1. True abdominal or pelvic tenderness is not usually present in gastroenteritis.
2. Gastroenteritis usually consists of both vomiting and diarrhea.
3. Gastroenteritis is usually a self-limited disorder, but IV rehydration and electrolyte replacement may be necessary.

5. Are there different gastrointestinal causes of vomiting in children?

Yes, particularly during the first year of life. These include gastrointestinal atresia, malrotation, volvulus, Hirschsprung's disease, gastroesophageal reflux, pyloric stenosis, intussusception, and inguinal hernia. (Vomiting in children presents considerations not covered in this chapter. See Chapter 63.)

6. List the common causes of vomiting other than gastrointestinal disorders.

- Infections
- Pneumonia
- Meningitis
- Sepsis
- Metabolic disturbances (e.g., diabetic ketoacidosis, uremia, or hypercalcemia)
- Toxicologic (e.g., digoxin, theophylline, aspirin, or iron)
- Cancer chemotherapy
- Neurologic (e.g., hydrocephalus, cerebral edema, or migraine headache)
- Renal calculi
- Ovarian or testicular torsion
- Pregnancy
- Ruptured ectopic pregnancy
- Labyrinthine disorders
- Myocardial ischemia
- Psychiatric

7. Can the character of the vomit help you to make a diagnosis?

Sometimes, especially with gastrointestinal disorders. In acute gastritis, vomit is usually stomach contents mixed with a little bile. In biliary or ureteral colic, the vomit is usually bilious. In sympathetic shock (acute torsion of abdominal or pelvic organ), it is common for the patient to retch frequently but vomit only a little. In intestinal obstruction, the character of vomit varies—first gastric contents, then bilious material, with progression to brown feculent material that is pathognomonic of distal small or large bowel obstruction. Vomiting of blood is a whole different story (see Chapter 33).

8. What else do I need to ask the patient?

- **Associated signs** and **symptoms**, such as pain, fever, jaundice, and bowel habits. Think of hepatitis or biliary obstruction with jaundice. Always remember that gastroenteritis is uncommon without diarrhea.
- **Relationship of vomiting to meals.** Vomiting that occurs soon after a meal is common with gastric outlet obstruction from peptic ulcer disease. Vomiting after a fatty meal is common with cholecystitis. Vomiting of food eaten more than 6 hours earlier is seen with gastric retention.
- **Do not always focus on the gastrointestinal system**. Ask about medications and possible drug use, headache and other neurologic symptoms, and last menstrual period and possibility of pregnancy. Inquire about cardiac risk factors, especially in older patients.

9. What do I look for on the physical examination?

Physical examination is helpful but can be unreliable. Look for signs of dehydration, particularly in children. Check for bowel sounds, which are increased in gastroenteritis and absent with obstruction or serious abdominal infections. Abdominal tenderness may be present in a variety of disorders, but a rigid abdomen points to peritonitis, a surgical emergency. Women of childbearing age with vomiting and abdominal or pelvic pain require a pelvic examination and pregnancy test. Always remember the neurologic examination if there are any associated neurologic symptoms, such as headache or vertigo.

10. Are laboratory tests indicated?

This question must be answered on an individual basis. In general, tests should be ordered based on the history and physical examination. Diabetics and elderly patients can *hide* serious infections and metabolic disturbances. Be careful with these patients.

11. When should I order radiographs?

This must be judged on an individual basis. Abdominal radiography is usually nonspecific but may show free air with perforation of an abdominal viscus or dilated bowel with obstruction. A chest film can be useful in cases of protracted vomiting to rule out aspiration or pneumomediastinum. Lobar pneumonia with diaphragmatic irritation may cause vomiting with abdominal pain and few respiratory symptoms.

KEY POINTS: DIAGNOSIS OF THE VOMITING PATIENT

1. Always consider etiologies other than gastrointestinal disorders.
2. Take a thorough history, especially in the young and elderly.
3. Always consider accidental ingestions in children and medication side effects or toxicities in adults.
4. Laboratory testing and radiographs are seldom useful in gastroenteritis but may be helpful to identify other causes of vomiting.

12. How should I treat the vomiting patient?

a. Always remember to **protect the airway**. Patients with altered mental status should be placed on their side to prevent aspiration. Intubate early when necessary.
b. **Intravenous (IV) fluids** usually are indicated for rehydration; normal saline or lactated Ringer solution is preferred.
c. **Nasogastric suction** can be therapeutic and diagnostic and is always indicated when there is a suspicion of a gastrointestinal bleed or small bowel obstruction.
d. **Medications** to relieve nausea and vomiting must be used judiciously, especially in patients with altered mental status, hypotension, or uncertain diagnosis.
e. Determine and, if possible, treat the underlying cause.

13. What medications should I use?

See Table 14-1.

WEBSITES

www.fda.gov/safety/medwatch

TABLE 14-1. ANTIEMETIC MEDICATIONS

Generic Name	Trade Name	Indication	Dose
Palonosetron	Aloxi	Vomiting with chemotherapy	0.25 mg IV prior to chemotherapy
Meclizine	Antivert	Vertigo and motion sickness	25 mg PO qid
Dolasetron mesylate	Anzemet	Vomiting associated with anesthesia or chemotherapy	12.5–100 mg PO or IV single dose
Hydroxyzine	Atarax	Nausea, vomiting, anxiety	25–100 mg PO or IM tid or qid
Diphenhydramine Nabilone	Benadryl Cesamet	Motion sickness Nausea and vomiting with chemotherapy	25–50 mg PO or IV qid 1–2 mg PO bid
Prochlorperazine Dimenhydrinate	Compazine Dramamine	Nausea, vomiting, anxiety Nausea, motion sickness	10 mg PO, IM, or IV qid, 25 mg PR bid 50–100mg PO, IM, or IV qid
Aprepitant	Emend	Nausea and vomiting, with chemotherapy	125 mg PO on day 1, 80 mg PO on days 2 and 3
Phosphorated carbohydrate	Emetrol	Nausea and vomiting,	1–2 tbs PO every 15 min (not to exceed 5 doses)
Droperidol	Inapsine	Nausea and vomiting	0.625–2.5 mg IV or 2.5 IM (black box warning: QT prolongation)
Granisetron	Kytril	Nausea and vomiting with chemotherapy	10 μg/kg IV or 1 mg PO bid (only on day of chemotherapy)
Dronabinol	Marinol	Refractory nausea and vomiting with chemotherapy	5 mg PO tid or qid
Promethazine	Phenergan	Nausea, vomiting, motion sickness, anxiety	12.5–50 mg PO, PR, or IV qid (black box warning: children younger than 2 years old)
Metoclopramide	Reglan	Nausea, vomiting, gastro-esophageal reflux, gastroparesis	5–10 mg PO or IV dosage varies (box warning, tardive dyskinesia)

Continued

TABLE 14-1. ANTIEMETIC MEDICATIONS—cont'd

Generic Name	Trade Name	Indication	Dose
Chlorpromazine	Thorazine	Nausea, vomiting, anxiety	10–25 mg PO qid, 25 mg IM, or qid 100 mg PR qid
Trimethobenzamide Thiethylperazine	Torecan Tigan	Nausea and vomiting Nausea and vomiting	250 mg PO tid or qid , 200 mg PR tid or qid , 200 mg IM tid or qid Dosage varies
Scopolamine	Transderm Scop	Nausea, vomiting, motion sickness	1 patch every 3 days
Hydroxyzine pamoate	Vistaril	Nausea, vomiting, anxiety	25–100 mg PO or IM tid or qid
Ondansetron	Zofran	Nausea and vomiting	4 mg IV or IM

bid, twice a day; IM, intramuscularly; IV, intravenously; PO, per os (by mouth); PR, per rectum; qid, four times a day; tid, three times a day.

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HEADACHE

Jonathan A. Edlow, MD, FACEP

CHAPTER 15

1. How common are headaches and what percentage of ED patients have headache as a chief complaint?

Nearly everyone has a headache at some point in their lives; migraine headache is found in about 12% of the general population. Most patients do not seek medical care, certainly not in the ED. Overall, approximately 2% of all ED visits are for headaches. Of those who come to the ED with headache, only about 5% will have a serious cause.

2. When someone has a headache, what exactly is it that hurts?

The brain, the pia and arachnoid mater, the skull, and the choroid plexus are not the source of headache pain. The structures in the head that are pain sensitive include the scalp; skin; vessels; scalp muscles; parts of the dura mater; dural arteries; intracerebral arteries; cranial nerves V, VI, and VII; and the cervical nerves. Irritation, inflammation, distention, or traction of any of these may result in a headache.

3. Name the most common headaches for which patients seek treatment.

Muscle contraction (tension) and vascular (migraine) headaches are by far the most common, even in an acuity-skewed ED population. These are often referred to as *primary headache disorders*. Although painful, these disorders do not have life-threatening sequelae. There are a number of *cannot miss* causes of headache that, although less common, are crucial for emergency physicians to diagnose correctly.

4. What causes of headache are *cannot miss*?

True emergencies, or *cannot miss* causes of headaches, are conditions that threaten life, limb, brain, or eye and treatable (see Table 15-1). Headaches that are true emergencies include:

- Intracranial bleeding (subarachnoid hemorrhage; SAH)
- Subdural or epidural hematoma
- Intraparenchymal hemorrhage
- Ischemic cerebrovascular accident (CVA)
- Dissection of a carotid or vertebral artery
- Hypertensive encephalopathy
- Brain tumor
- Giant cell arteritis (temporal arteritis) and other vasculitides
- Central nervous system infections (meningitis and abscess)
- Pseudotumor cerebri
- Cerebral venous sinus thrombosis
- Narrow angle glaucoma
- Spontaneous intracranial hypotension

5. What are some clinical clues to distinguish primary headaches from *cannot miss* headaches?

By definition, tension and migraine headaches are recurrent episodes; these episodes are usually similar to one another in any one individual patient. Therefore, any first severe headache can never be definitively diagnosed as tension or migraine. A headache that is described as a *first* or *worst* headache, or even substantially different from prior

TABLE 15–1. RED FLAGS IN PATIENTS WITH HEADACHES

Headache Characteristics	Differential Diagnosis	Possible Work-Up (beyond history and physical examination)
Headache begins after age 50	Mass lesion, temporal arteritis, stroke	ESR, neuroimaging
Sudden onset of headache	SAH, pituitary apoplexy, hemorrhage into a mass lesion or vascular malformation, mass lesion (especially posterior fossa), vascular dissection and CVST	Neuroimaging, LP if CT is negative
Headaches increasing in frequency and severity	Mass lesion, subdural hematoma, medication overuse	Neuroimaging, drug screen
New-onset headache in patient who has risk factors for HIV, cancer	Meningitis (chronic or carcinomatous), brain abscess (including toxoplasmosis), metastasis	Neuroimaging, LP if neuroimaging is negative
Headache with fever, meningismus, rash, or altered mentation	Meningitis, encephalitis, Lyme disease, systemic infection, collagen vascular disease	Neuroimaging, LP, serology
Focal neurologic symptoms or signs of disease (other than typical aura)	Mass lesion, vascular malformation, stroke,	Neuroimaging
Papilledema	Mass lesion, pseudotumor, meningitis	Neuroimaging, LP
Headache that worsens with standing up	Spontaneous intracranial hypotension, postdural puncture headache (if following an LP)	For the former—LP with opening pressure, MRI
Headache with ocular or visual symptoms	Pseudotumor cerebri, acute narrow angle glaucoma, temporal arteritis	LP for pseudotumor, tonometry for glaucoma, ESR and biopsy for arteritis
Headache after head trauma	Intracranial hemorrhage, subdural hematoma, epidural hematomas, post-traumatic headache	Neuroimaging of brain, and possibly cervical spine

CT, computed tomography; CVST, cerebral venous sinus thrombosis; ESR, erythrocyte sedimentation rate; LP, lumbar puncture; SAH, subarachnoid hemorrhage.

headaches, requires close evaluation. A sudden, severe onset, commonly described as "the worst headache I have ever had," is classic for a SAH. Associated fever requires evaluation for infection, tumor, or drug use. A careful history is usually the diagnostic element that helps decide which headaches to evaluate beyond history and physical examination. Any headache associated with new focal neurologic signs should be investigated.

6. Why are age and context important in the history of a patient with a headache?

Migraines most commonly begin before age 30. Tension-type headaches usually begin before age 50. Headaches that begin after age 55 are much more likely to have a serious cause, such as a mass lesion, giant cell arteritis, or cerebrovascular disease. Headaches occurring in the peripartum period may be caused by cortical vein or cerebral venous sinus thrombosis. In general, if a patient has a long history of previous similar attacks, a serious cause is less likely. If a patient reports numerous identical attacks treated at home, it is important to understand why this particular episode led to an ED visit.

7. What questions in the history are most important to ask in evaluating a patient with a headache?

a. Do you get headaches frequently? Have you ever needed to go to an ED for one? Is this current headache the same as prior ones that you have had? If not, how does it differ? These questions are aimed at assessing the *quality* of pain.
b. How bad is this headache? Have you had headaches this severe in the past? These questions assess the *severity*.
c. Did the headache start suddenly or gradually? If sudden, what were you doing at the time it began? These questions go after the *onset*.
d. What symptoms accompany the headache? Did you vomit? Was there any fainting, seizure, photophobia, or double vision? Did you have these same associated symptoms with prior episodes or not? (for patients with a prior history of headache). These *associated symptoms* can suggest secondary causes. For example, a patient with migraines, who has never had photophobia or vomiting with prior episodes, and now does, ought to undergo further evaluation. On the other hand, if this headache is similar to prior episodes, it is most likely due to that same etiology.
e. Have you had any recent head trauma? Note that this includes even minor trauma for elderly patients, who are more susceptible to chronic or delayed presentation subdural hematomas.
f. What treatment have you used at home and was it helpful? This can also help if a headache has responded in the past the same as it does for the current visit. But be careful; see Question 13.

8. Does the physical examination add any information?

The history often leads to the correct diagnosis or at least a short list of possible diagnoses. The physical findings may support or refute those diagnoses or change the likelihood of various possibilities. Fever may reflect infection. Hypertension may cause headache, be a sign that there is increased intracranial pressure, or simply be caused by the headache or anxiety of an ED visit. Abnormal pulse or respiration may be due to infection or toxins.

- Palpate the temporal arteries, sinuses (see Question 21), temporo-mandibular joints, and the scalp for tenderness.
- Examine the fundi for papilledema and spontaneous venous pulsations.
- Check for nuchal rigidity and photophobia.
- Perform a neurologic examination as indicated by the patient's history and general physical examination.

9. What is the sensitivity of a noncontrast, head computed tomography (CT) for detection of a SAH?

Even with advances in imaging technology, only approximately 90% to 95% of SAHs are detected on CT scans of the head. This number is higher in patients scanned within the first 12 to 24 hours but declines rapidly with time from the onset. This is due to cerebrospinal fluid

(CSF) circulation. A lumbar puncture (LP) and CSF analysis will rule out SAH in a patient with a normal CT scan with close to 100% sensitivity.

10. What are the CSF findings in a SAH?

As with CT, the findings on LP evolve with time. Even in the first hours after SAH, the large numbers of red blood cells are found in the lumbar theca. Over days, these numbers fall due to the circulation of CSF and the breakdown of the red blood cells and hemoglobin. Thus, red cells are nearly always present early, and xanthochromia (the yellow color due to hemoglobin catabolism) is almost always found later (until about 2 weeks post-SAH, depending on the method one uses for detection). Measuring the opening pressure can help too because it is often elevated in SAH.

11. How do I differentiate between a traumatic tap and a SAH?

There are many tests, but none is perfect. Clearing of blood from earlier- to later-collected tubes is commonly used and is helpful, but unless the last tube contains zero cells, SAH is still a possibility. Wasting a few milliliters of CSF between the first and last tubes facilitates this. An elevated opening pressure suggests a SAH and not a traumatic tap. Xanthochromia is almost always present if blood has been in the CSF for 12 hours or longer and confirms an intracranial bleed. As with CT, one must factor in the timing of the LP (with respect to the onset of the headache) in interpreting the LP results.

12. If the CT and LP are both normal, do I need to pursue the diagnosis of SAH with some form of angiography?

The data strongly support, and American College of Emergency Physicians (ACEP) clinical policy recommends, stopping the work-up if both tests are negative. However, this assumes that SAH is the major consideration. Rarely, an unruptured aneurysm that is acutely expanding, dissecting, or thrombosing can cause an acute headache. Furthermore, there are other causes of acute, severe, sudden-onset headache associated with a normal CT and LP. These include (see Table 15-2):

- Pituitary apoplexy.
- Cervical artery dissections.
- Cerebral venous sinus thrombosis.
- Posterior reversible encephalopathy syndrome (related to eclampsia).
- Acute stroke (especially posterior fossa).

13. What are migraine headaches?

Although people may refer to any severe headache as a migraine, a migraine is a specific type of headache. Migraines tend to be familial and affect women twice as often as men. The underlying pathophysiology is thought to be *vasogenic inflammation*. The first headache usually occurs in an individual in the teens or twenties. Headaches typically are described as unilateral, severe, throbbing, and are commonly associated with photophobia and nausea. The headache may also be non-throbbing. Variations on all of the symptoms occur, but each patient tends to experience a similar constellation of symptoms with each headache. Patients who experience an aura will often have *positive* symptoms (e.g., flashing lights or zig-zag patterns in vision, tingling of the face or arm or shaking of a limb) as opposed to *negative* symptoms (e.g., absence of vision, anesthesia or absence of movement of a limb), which are more common with brain ischemia or infarction. Occasional migraine patients will have weakness, however. Patients will often use the word *migraine* to describe any severe headache, so if a patient says they have a history of migraines, get more details about their duration, frequency, and what work-up has been done. Make sure that their headaches are truly migraines.

14. If a headache patient improves or the pain completely resolves with sumatriptan or ketorolac, does that mean that the diagnosis is migraine (or some other primary headache cause)?

The answer to this question is an emphatic "*no*." Because the final common pathway for most pain in the head is limited, and vasogenic inflammation probably plays a role, the response to any analgesic or antimigraine medication is of no etiologic significance. This includes triptans,

which have been documented to improve the headaches of patients with SAH and cervical artery dissections.

15. What specific entities must be considered in patients with a headache and a history of cancer or immunosuppression?

In a patient with a history of cancer, consider brain metastases, or infections related to immunosuppression. In patients who are HIV positive, especially if they have low CD4 counts or high viral loads, opportunistic infections, such as cryptococcal meningitis or

TABLE 15-2. DIFFERENTIAL DIAGNOSIS AND WORK-UP FOR ACUTE, SEVERE HEADACHE

Pathologic Process	Clinical Characteristics	Work-Up
Subarachnoid hemorrhage	Headache worst of life Headache abrupt, effort related Normal neurologic examination to focal deficit or coma	CT; if normal, do LP
Cervical artery dissections	History of trauma, Marfan syndrome, collagen disorders Headache is ipsilateral Carotid: neck or head pain, Horner syndrome, stroke Vertebral: occipital-nuchal pain and posterior circulation stroke	Magnetic resonance or CT angiography Vascular ultrasound and conventional angiography
Intracerebral hemorrhage	History of hypertension History of brain tumor Severe headache with signs of elevated intracranial pressure and depressed mental status	CT
Cerebral venous thrombosis (superior sagittal sinus or transverse sinus)	Postpartum, hypercoagulable states, and abrupt, dull, constant headache Sixth nerve palsy, seizures Signs of raised intracranial pressure	MRI, magnetic resonance venography, or conventional angiography; CT angiography shows promise
Pituitary apoplexy	Abrupt severe headache, progressive visual loss with subsequent signs of pituitary insufficiency	CT or MRI with coronal views of the pituitary

CT, computed tomography; CVA, cerebrovascular accident; LP, lumbar puncture; MRI, magnetic resonance imaging.

toxoplasmosis, brain abscess, and primary lymphoma of the central nervous system, should be considered.

16. What specific diagnosis should be considered in older patients with a new onset headache and general malaise or other systemic symptoms?

Temporal arteritis is a systemic arterial vasculitis that is rare before age 50 and dramatically increases in incidence afterward. Also known as *giant cell arteritis*, temporal arteritis should be suspected in any patient older than age 50 who has a new onset headache or a change in an established pattern of headache. It is associated with localized scalp tenderness (anywhere in the scalp), malaise, myalgias, arthralgias, polymyalgia rheumatica, low-grade fevers, or other constitutional symptoms. Untreated, temporal arteritis can result in blindness or stroke. Jaw claudication, if present, is strongly suggestive of the disorder. Erythrocyte sedimentation rate (ESR) is usually greater than 50 mm/hour, and biopsy is required to establish the diagnosis. Treatment should be initiated in the ED, based on the clinical presumption and results of the ESR and not delayed by biopsy. The initial doses of prednisone range from 60 to 80 mg daily. Finally, because primary headaches start less commonly after the age of 50, many of the other serious etiologies become more common in this age group, conditions such as stroke and tumors.

17. What is a sentinel bleed?

Up to 50% of patients with aneurysmal SAH will have experienced a warning or sentinel hemorrhage before their catastrophic bleed. These small hemorrhages occur days to months before the major event. These events are still characterized by abrupt onset of severe, unusual headache and, if worked up with CT and LP, should be diagnosable in the vast majority of cases. Note that these headaches resolve over days to weeks because of the circulation of CSF mentioned previously. Unfortunately, these episodes are often not worked up and are misdiagnosed as migraine, sinusitis, or tension-type headache, however, and the patients are discharged from medical care.

18. How do I treat a migraine headache?

Patients who are unable to control their headache at home often present to the ED for better pain control or supportive therapy. The choice of treatment is based on case presentation, prior medications used, time elapsed since onset, patient's prior response to therapy, existence of comorbid conditions, and severity of the current attack. Narcotics should be used as a last resort (Table 15-3).

19. How are cluster headaches different from migraines? How are they treated?

These are nonfamilial headaches predominantly affecting men. Excruciating, unilateral pain lasting 30 to 90 minutes occurs multiple times a day for weeks, followed by a pain-free interval. During the attacks, autonomic signs of rhinorrhea and lacrimation frequently occur ipsilateral to the headache. Attacks may be induced by smoking or alcohol. Oxygen sometimes relieves 90% of cluster headaches within 15 minutes. Other treatments include corticosteroids, calcium channel blockers, lithium, intranasal lidocaine, and methysergide.

20. How do I treat tension headaches?

If the diagnosis is secure, treatment starts with reassurance and education. Because these headaches are usually chronic, they should be treated with nonaddictive analgesics. Biofeedback and acupuncture may be beneficial. All patients with this diagnosis should be screened for mood disorders because depression is a common cause of tension headaches.

21. Which toxin may bring in entire families complaining of headache?

Carbon monoxide poisoning. See Chapter 72.

22. Does sinusitis commonly cause headache? If a CT scan shows *sinusitis,* is that the likely cause of a patient's headache?

Acute bacterial sinusitis can certainly cause headache, but headache from sinusitis is not nearly as common as some patients and doctors think. Patients will often use the term *sinus headache* just as inaccurately as they use the term *migraine*. When sinusitis causes headache,

TABLE 15-3. SELECTED MEDICATIONS FOR ACUTE MIGRAINE ATTACKS

Medication	Dose and Route*	Comments
Mild to moderate		
Acetaminophen	500–1000 mg	Avoid in patients with liver disease
Aspirin	650–1000 mg	GI upset
Ibuprofen	600–800 mg	GI upset
Naproxen sodium	275–550 mg	GI upset
Indomethacin	50 mg rectal suppository	
Moderate to severe		
Dihydroergotamine	1 mg IV or IM	May be repeated in 1 hour but not if triptans used already Contraindicated in HTN, PVD, CAD, and pregnancy
Sumatriptan	6 mg SQ	May be repeated in 1 hour but not if ergots used already Contraindicated in HTN, PVD, CAD, and pregnancy
Metoclopramide	10 mg IV or IM	Sedation and dystonic reaction
Prochlorperazine	10 mg IV or IM	Sedation and dystonic reaction
Ketorolac	30–60 mg IM or 15–30 mg IV	GI upset; caution in elderly and patients at risk for renal failure
Morphine sulfate	0.1 mg/kg	Opioids should be used as last resort
Hydromorphone	0.5–2 mg IV (note: 1 mg hydromorphone = 8–10 mg of morphine)	Opioids should be used as last resort
Butorphanol	2 mg IV	Opioids less efficacious than other medications
Refractory attack, status migrainosus		
Dihydroergotamine	1 mg IV	Use in conjunction with antiemetic
Steroids	Various regimens	Controversial; based on anecdotal evidence

CAD, coronary artery disease; GI, gastrointestinal; HTN, hypertension; IM, intramuscularly; IV, intravenously; PVD, peripheral vascular disease; SQ, subcutaneously.

*Assumes average-size adult patient.

there are generally other symptoms and signs of sinusitis (e.g., nasal congestion, fever, boggy nasal mucosae), and the pain is generally unilateral. Tenderness over a sinus is non-specific and may be a function of how hard one is pressing. Finally and very importantly, CT findings of chronic sinusitis, such as mucosal thickening, retention cysts, or ostial narrowing, should never be considered the cause of a patient's acute headache.

23. What special diagnostic considerations must be given to a patient with AIDS and headache?

Headache is a frequent complaint among AIDS patients, occurring in 11% to 55% of patients, and may occur in many AIDS-related conditions. Acute lymphocytic meningitis can be seen in patients at the time of acute HIV infections, sometimes associated with fever, lymphadenopathy, sore throat, and myalgias. *Toxoplasma gondii* produces multiple brain abscesses and bilateral, persistent headaches. The diagnosis of toxoplasmosis is made by CT, magnetic resonance imaging (MRI), or brain biopsy. Other central nervous system lesions include B-cell lymphoma and progressive multifocal leukoencephalopathy. Cryptococcal meningitis is a common cause of headache in AIDS patients, occurring in 10% of patients. Meningitis is characterized by fever, headache, and nausea. The presence of meningismus, or mental status changes, is uncommon. Patients who have HIV and who present to the ED with persistent headache usually require neuroimaging and, if imaging is normal, LP should be done.

24. What rapidly progressive infectious entity presents with headache, fever, and altered mental status?

Herpes simplex encephalitis, the most common form of sporadic encephalitis, is a necrotizing, hemorrhagic infection that results in brain destruction that mandates early aggressive treatment with antiviral therapy. LP with polymerase chain reaction of the CSF and gadolinium-enhanced MRI are the diagnostic methods of choice. On imaging, there is a predilection for temporal lobe involvement. Note that there are other viral encephalitides (e.g., West Nile, Eastern Equine) but there is currently no specific treatment for them.

25. What is idiopathic intracranial hypertension, and what is the complication if not treated appropriately?

Also known as *benign intracranial hypertension* or *pseudotumor cerebri*, this entity presents classically in obese young women with recurrent headaches that are constant or intermittent and that may present with bilateral papilledema and loss of spontaneous venous pulsations. Transient pulsatile tinnitus and visual symptoms are common. Occasionally, sixth nerve palsy is found. Note that a sixth nerve palsy has no localizing value; it is the cranial nerve with the longest intracranial course and is thus sensitive to pressure and inflammation. Brain imaging should be done to rule out a mass lesion and, if negative, LP is done; this not only is diagnostic but also commonly therapeutic. High opening pressure (25 to 40 cm H_2O) and a suggestive clinical scenario are diagnostic. It is important to consider the diagnosis of cerebral venous sinus thrombosis because these two entities can mimic one another. Without treatment, there is a risk of visual loss. Treatment is with serial LPs, acetazolamide, and diuretics such as furosemide. Optic nerve fenestration is indicated in refractory cases.

26. Which cranial nerves pass through the cavernous sinus?

Cranial nerves III, IV, V1, and $V_{1\text{-}2}$. Cavernous sinus disease may present as only a retro-orbital headache. Any combination of involvement of the nerves passing through the cavernous sinus is suggestive of the diagnosis, however, and warrants further evaluation. Invasion by tumor, vascular disease such as aneurysm or carotid cavernous sinus fistula, and clot (either bland or infection related) are the more common causes. Patients with other cerebral venous sinus thromboses will often present with isolated headache, seizure, and elevated intracranial pressure.

27. How common are headaches in children?

As with adults, headaches are also common in children. The history and physical examination are paramount in sorting out who needs a work-up and who does not. Treatment can start

with acetaminophen or ibuprofen. Ruling out significant pathology is crucial in children. SAH, primary cerebral tumors, stroke, metabolic conditions, and toxicologic causes should be considered in the appropriate setting.

28. What is a blood patch?

One third of patients experience headaches within hours of a diagnostic LP. This is due to a persistent CSF leak from the dural rent that results in low CSF pressure, dilation of intracranial vessels, and traction on intracranial contents. This postdural puncture headache is worse when the patient sits or stands up and improves with bed rest. Treatment includes bed rest, fluids, and analgesia. Some practitioners use intravenous (IV) caffeine. If conservative methods fail, autologous blood clot is used, the so-called blood patch. Blood is drawn from the patient and injected into the soft tissue at the site of the LP. In most institutions, this is performed by an anesthesiologist. Data suggest that using a small caliber LP needle and a noncutting tip can decrease the incidence of postdural puncture headache.

29. Are there other forms of low pressure spinal headache?

In the absence of a prior LP, patients can still get a low pressure headache, a condition known as *spontaneous intracranial hypotension*. The headache is positional (worse with standing, better with lying down), similar to a post-dural puncture headache. Occasionally, patients will develop neurological deficits. Diagnosis is by imaging and LP. This is another diagnosis, albeit an uncommon one, that supports measuring the opening pressure when doing an LP.

30. In the pregnant (or recently postpartum) woman, are there particular causes of headache that I should worry about?

Pregnant women can get any kind of headache that nonpregnant women can get; however, some headache disorders that occur more commonly, or exclusively, in this situation are cerebral venous sinus thrombosis, eclampsia, pituitary apoplexy, SAH, and posterior reversible leukoencephalopathy syndrome (PRES). Added to this list in the postpartum patient who has had an epidural anesthetic are postdural puncture headache and the additional complication of a postpuncture subdural hematoma. As for imaging, MRI has certain obvious advantages during pregnancy, but one should get the tests that are needed to make the diagnosis, trying to balance radiation exposure with accurate diagnosis.

31. Is high blood pressure causing my patient's headache?

One potential mistake is to diagnose hypertensive *urgency* in headache patients who have a high blood pressure. Coexistent headache and hypertension can occur for several reasons. Probably the most common is that pain and anxiety are elevating the blood pressure.
A second cause is that the problem causing the headache is also causing some degree of raised intracranial pressure, and the body is raising the arterial blood pressure to preserve cerebral perfusion pressure. If hypertension is primarily causing the symptoms (the third possibility), then end organ dysfunction is occurring (in this case, the end organ is the brain). Lowering the blood pressure about 25% below the peak in this situation, using rapidly acting, titratable agents, will both treat and help establish the diagnosis because the headache ought to improve dramatically. In patients with acute ischemic stroke and headache, one should be cautious about pharmacologically treating high blood pressure because it is likely that the high pressure is simply due to the brain autoregulating.

32. When should I be concerned about a brain tumor?

Isolated headache is rarely caused by a brain tumor. Only about half of all patients with brain tumors have headache and the pain characteristics are not specific. The classic early morning headache is uncommon. Localization (other than neck pain with posterior fossa tumors) does not usually occur. One risk for patients with brain tumors is to have a headache that is different from their previous headaches. Therefore, a careful history and physical examination are most important in deciding which patients need a work-up for a tumor as they are for any other secondary cause of headache.

KEY POINTS: HEADACHE

1. A response to analgesics does *not* exclude life-threatening causes of headache.
2. Even with modern CT, scanners will miss 5% to 10% of SAHs. LP is needed if SAH is a major diagnostic concern.
3. HIV-positive patients presenting with headache should have a CT head scan with contrast to exclude opportunistic infections, including toxoplasmosis.
4. A careful history and physical examination, including neurologic examination, will identify most patients who need further evaluation.

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SYNCOPE, VERTIGO, AND DIZZINESS

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CHAPTER 16

1. **Do I need to be concerned by a complaint of dizziness?**
   Yes. There are over 25 million ED visits a year for dizziness; it is the most common complaint in patients older than 75 years and may herald a stroke.

2. **How do I approach this vague and ill-defined complaint?**
   Start your history with an open-ended question that allows the patient to describe the symptoms. *Dizzy* can describe a sensation of vertigo (the illusion of motion); lightheadedness (presyncope or frank syncope); or disequilibrium (imbalance). Those with disequilibrium often describe imbalance while walking, especially in the dark.

3. **How does one know his or her position in space?**
   A combination of visual clues and vestibular input determine our spatial orientation. When they don't agree or are asymmetric, we feel dizzy.

4. **How does the vestibular system work?**
   The semicircular canals use the principle of liquid inertia to determine angular acceleration. The canals, oriented in three planes to encompass all aspects of space, are filled with a fluid, endolymph. When the head turns, the fluid stimulates hair cells in response to this relative movement, sending impulses to the brain via cranial nerve VIII.

5. **How do you define central versus peripheral vertigo?**
   It is an anatomical definition. Peripheral vertigo is caused by a dysfunction of the inner ear or vestibular nerve, whereas central vertigo is from etiologies of the brain and a brain stem. Benign paroxysmal positional vertigo (BPPV), vestibular neuritis, and Ménière disease are common etiologies of peripheral vertigo, whereas vertebrobasilar ischemia, multiple sclerosis, cerebellar infarction/hemorrhage, and basilar migraine are central causes.

6. **What are the characteristics of peripheral vertigo?**
   DR FLIP. This mnemonic reminds you that the Epley maneuver, which *flips* the patient, helps BPPV.
   Deafness (unilateral hearing loss)
   Ringing in the ears (tinnitus)
   Fatigable on repeated testing
   Latency after Dix-Hallpike maneuver
   Intense symptoms
   Positional in nature

7. **What are the characteristics of central vertigo?**
   Cranial nerve deficits
   Vertical nystagmus (not seen in peripheral vertigo)
   Ataxia (with gait impairment)

8. **What are the key points for the main causes of peripheral vertigo?**
   See Table 16-1.

TABLE 16-1. KEY POINTS FOR THE MAIN CAUSES OF PERIPHERAL VERTIGO

Benign paroxysmal positional vertigo
Most common cause (50%)
Recurrent <1 minute paroxysms elicited with head turning
Normal between brief attacks
Due to otolith dislodgement into a semicircular canal
Responds to Epley maneuver
Vestibular Neuritis
Less common cause (20%)
Probable viral etiology
Positive asymmetric head thrust test
May respond to steroids
Ménière Disease
Less common cause (10%)
Less acute onset (hours)
Associated with hearing loss, tinnitus
Due to high endolymph volume
Responds to diuretics or fluid restriction

9. What targeted bedside tests aid in the diagnosis of vertigo?

Examine the eyes for ocular palsies and nystagmus. Examine the ears for infection, perforation, and hearing function (using the Weber and Rinne tests). Perform a full neurologic examination including cranial nerves, gait, stance, and cerebellar function. The Dix-Hallpike and Epley for BPPV and head thrust maneuvers to diagnose labyrinthitis or neuronitis may be beneficial if these specific etiologies are suspected.

10. How is nystagmus evaluated in the work-up of vertigo?

With peripheral etiologies, asymmetric impulses from the semicircular canals cause the eyes to drift toward the diseased side and *correct* with the fast component of nystagmus to the opposite normal side. By convention, the direction of the fast component determines the *direction*. Vertical, changing direction, and nonsuppressible nystagmus are characteristics of central etiologies.

11. What is the head thrust maneuver? What does it mean?

The examiner stands in front of the patient and holds the patient's head in both hands instructing the patient to look at the examiner's nose. The head is rapidly turned 5 to 10 degrees to one side and the response of the eyes is noted. Normally, the eyes continue to fix on the examiner's nose (intact vestibulo-ocular reflex), but if one side has a unilateral lesion such as vestibular neuritis, the eyes don't stay fixed on the target and need to correct to refocus on the target after head movement. Turning the head in the opposite direction serves as control. A patient with unidirectional horizontal nystagmus, a positive head thrust test opposite the fast phase direction of the nystagmus and no other neurological features, is likely to have vestibular neuritis or labyrinthitis.

12. What is the Dix-Hallpike maneuver?

This diagnostic maneuver tests for BPPV and involves moving the patient rapidly from a sitting position with the head turned 45 degrees to one side with the head hanging down in a

supine position. Nystagmus, often toward the dependent eye or forehead, is characteristically associated with a delay of a few seconds and fatigable symptoms of vertigo on repetition.

13. What is the Epley maneuver?

The Epley maneuver treats BPPV and is utilized to physically move otoliths, most commonly in the posterior semicircular canal into the utricle. It is successful about 75% of the time on first attempt and up to 98% on two attempts. The technique involves performing the Dix-Hallpike maneuver, then turning the head to the opposite side while still supine and then turning over in the same direction prior to rising. (See Fig. 16-1)

14. How do I treat peripheral (and central) vertigo?

Vestibular neuritis benefits from steroids in a 22-day taper. BPPV is treated by the Epley maneuver. Nonspecific vestibular suppressants include:

- Anticholinergics (e.g., scopolamine transdermal)
- Antihistamines (meclizine 25–50 mg PO every 6 hours, dimenhydrinate 50 mg PO every 4–6 hours, and diphenhydramine 25–50 mg PO every 4–6 hours)
- Benzodiazepines (diazepam 5–10 mg PO every 6 hours)

Central vertigo requires evaluation by neurology or neurosurgery. If the patient cannot walk or has intractable symptoms, magnetic resonance imaging (MRI) to evaluate the posterior fossa and brainstem and admission is recommended.

15. What is syncope?

A sudden temporary loss of consciousness with the inability to maintain postural tone. It is a symptom, not a disease, with a wide variety of benign and life-threatening causes. Coma, head trauma, shock, and seizures may mimic syncope.

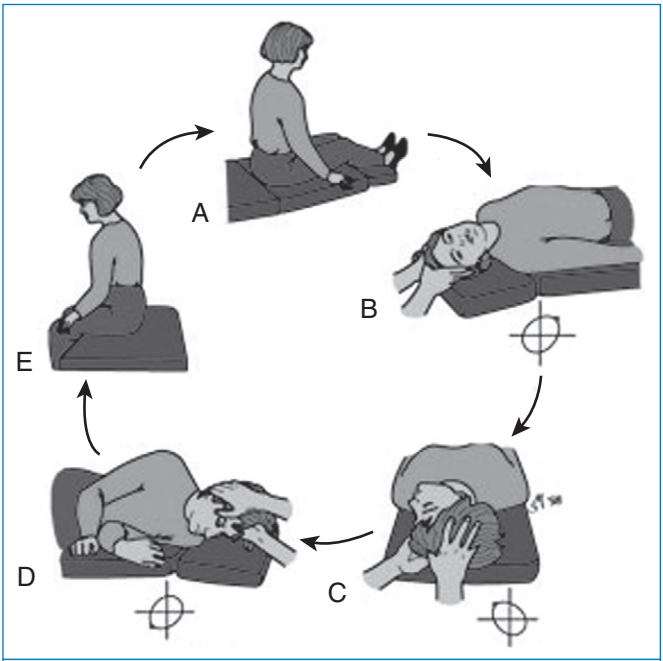

Figure 16–1. Epley maneuver.

16. What are the odds of determining the cause of a syncopal episode?
Despite extensive and expensive work-ups, no cause is found in about 50% of cases. This should be discussed with the patient so that there are no unrealistic expectations.

KEY POINTS: CAUSES OF SYNCOPE

1. HEAD (**h**ypoxemia, **e**pilepsy, **a**nxiety, **d**ysfunctional brain)
2. HEART (**h**eart attack, **e**mbolism of pulmonary artery, **a**ortic obstruction, **r**hythm disturbance, **t**achydysrhythmia)
3. VESSELS (**v**asovagal, **e**ctopic, **s**ituational, **s**ubclavian steal, **E**NT, **l**ow **s**ystemic vascular resistance, **s**ensitive carotid sinus)

17. Discuss the causes of syncope as related to the head.
Diffuse cerebral malfunction from lack of vital nutrients, such as oxygen (hypoxemia) or sugar (hypoglycemia), are often correctable, but easily overlooked. Seizures don't cause but can mimic syncope. Vertebrobasilar insufficiency and SAH represent a *dysfunctional brain.*

18. Discuss the cardiovascular causes of syncope.
Cardiac causes of syncope comprise the riskiest group of patients and include acute coronary syndrome (ACS), pulmonary embolism, physical aortic outflow obstructions (from hypertrophic obstructive cardiomyopathy, aortic stenosis, and atrial myxoma), slow rhythms such as sick sinus syndrome, and tachyarrhythmias. Brugada syndrome, pre-excitation, and long QT syndrome can precipitate lethal dysrhythmias.

19. What about the vascular causes of syncope?
Vascular causes include:
- The *common faint* (vasovagal)
- Hypovolemia
- Situational faints (e.g., micturition, defecation, cough, or Valsalva maneuver)
- Subclavian steal
- Ear, nose, and throat (ENT) causes (e.g., glossopharyngeal and trigeminal neuralgia)
- Low systemic vascular resistance (from medications and autonomic insufficiency)
- Carotid sinus sensitivity (only accounting for 4% of syncope cases)

20. Summarize the initial concerns when treating a patient with syncope.
Most patients with syncope rapidly return to a normal mental status and have stable vital signs. There are treatment priorities, however.
a. Obtain vital signs and evaluate and treat for immediate life threats.
b. Oxygen, intravenous access, and cardiac and blood pressure monitoring should be initiated on patients who have abnormal vital signs, a persistent altered level of consciousness, chest pain, dyspnea, abdominal pain, or a significant history of cardiac disease.
c. Assess for any trauma secondary to fall. Elderly patients are more likely to suffer head trauma secondary to syncope, and this may be a greater life threat initially than the cause of the syncope.

21. Okay, I've ruled out the immediate life threats. Now what do I do?
Obtain a detailed history, do a directed physical examination, and obtain an electrocardiogram (ECG). Then do a risk assessment to determine whether further testing or admission is indicated.

22. What components of the history are most important?

The most important historical clue is the patient's recollection of the events just before the syncope. An abrupt onset of loss of consciousness with a brief (<5 seconds) prodrome is indicative of a cardiac etiology. Similarly, syncope associated with exercise, or while reclining or recumbent, is associated with cardiac obstructive causes or arrhythmias. Patients who have vasovagal syncope often have premonitory symptoms of dizziness, yawning, nausea, and diaphoresis, and the event is during a period of some psychosocial stress. Clues to hypovolemia include thirst, postural dizziness, decreased oral intake, melena, or unusually heavy vaginal bleeding. Syncope after micturition, cough, head turning, defecation, swallowing, or meals suggests situational syncope. Note previous episodes of syncope, upper extremity exertion (e.g., subclavian steal syndrome), and the presence of cardiac risk factors. A family history of sudden death may suggest Brugada, pre-excitation, or long QT syndromes. Many medications and medication interactions can cause syncope, so determine all of the patient's current medications, especially when treating the elderly.

23. How do I know it was not a seizure?

Victims of arrhythmias and vasovagal faints often exhibit myoclonic jerks that may mimic a seizure. Recovery from syncope is usually rapid, whereas a generalized seizure patient awakens slowly with prolonged confusion or postictal state. Both may have trauma. The absence of an anion gap on blood drawn within 30 minutes of the event or no postictal state argues against a generalized seizure. Lateral tongue biting has been shown to be specific but insensitive for a seizure.

24. What is a directed physical examination?

Be a detective, using head, heart, and vessels as a guide. The patient with abrupt effort or exercise syncope may have aortic stenosis or hypertrophic cardiomyopathy; look for narrow pulse pressure, systolic murmur, or change in murmur with Valsalva. The presence of physical signs of congestive heart failure (CHF) places the patient at high risk. Examine the head carefully for trauma, bruits, and focal neurologic signs. Check blood pressure in both arms looking for subclavian steal. Search for occult blood loss or autonomic insufficiency.

25. What tests are needed to assist in diagnosis?

Other than a urine pregnancy test in females, a detailed history, physical examination, and ECG are often sufficient. The addition of a specific confirmatory test (e.g., echocardiography) is recommended for suspected cardiomyopathy.

26. Who needs an ECG? What am I looking for?

Almost all patients with syncope should have an ECG because it is not invasive, may be diagnostic of a problem such as Brugada syndrome or long QT, and helps in risk stratification for ACS. Check for markers of cardiac disease, such as ischemia, infarction, arrhythmias, pre-excitation, long QT intervals, and conduction abnormalities. Left ventricular hypertrophy may be a clue to aortic stenosis, hypertension, or cardiomyopathy.

27. If the basic evaluation is not diagnostic, who should receive further testing?

Patients with CHF, older age, abnormal ECG and unexplained syncope who have suspected heart disease should be admitted and evaluated for an acute coronary syndrome. Echocardiography, exercise treadmill testing, Holter ECG monitoring, and electrophysiologic studies also may be helpful.

28. What factors help to assign a patient to a high-risk or low-risk group?

Physician gestalt plays a large role. Studies attempting to determine highly sensitive risk factors have had mixed results (see Table 16-2). For example, the San Francisco Syncope Rule was found to have only 75% sensitivity on external validation.

TABLE 16-2. SYNCOPE AT HIGH RISK FOR CARDIAC ETIOLOGY

Historical	ED evaluation
Age >65 years	Abnormal vital signs
Cardiovascular disease history	Systolic blood pressure <90
Lack of prodrome	Evidence of congestive heart failure
Exertional	Abnormal electrocardiogram
Chest pain with event	
Palpitations preceding the event	
Family history of sudden death	

29. My consultant wants orthostatic vital signs; are they helpful?

Yes and no. Orthostatic hypotension (defined as a blood pressure drop of 20 mm Hg on standing for 3–4 minutes) is associated with volume loss or autonomic insufficiency and an increased risk of fall and syncope. However, parameters for orthostatic hypotension are neither sensitive nor specific. A blood pressure drop of 20 mm Hg has only 29% sensitivity and 81% specificity for 5% or greater fluid deficit. Of normal euvolemic patients older than 65 years, more than 25% generate false-positive results. Reproducible symptoms are a better predictor than any number change.

30. Are there guidelines for the evaluation of syncope?

Yes.

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SEIZURES

Kent N. Hall, MD

CHAPTER 17

1. What is a seizure?

A seizure is an episode of abnormal brain function caused by excessive and aberrant electrical discharge in the brain. This electrical discharge may, or may not, result in characteristic muscle activity that is recognized as *seizure activity*. In addition to tonic-clonic muscle activity, generalized seizures may also manifest as staring episodes, lip smacking or other minor motor activity, or complete disruption of muscle tone (*drop attacks*). Generalized seizures are often followed by a *postictal phase* characterized by confusion and/or lethargy. This phase usually lasts for 5 to 15 minutes, although it may last longer.

The recognition and appropriate management of seizures are critically important to the patient and the emergency physician because they are a common ED presenting problem. Prolonged excessive electrical activity in the brain causes neuronal destruction. This destruction is not due to build-up of metabolic by-products but is actually directly related to the electrical activity itself. For an unknown reason, the hippocampus is particularly sensitive to the damage from this electrical activity.

2. How are seizures classified?

Seizures are classified according to the amount of brain involved in the abnormal electrical activity, its resulting physical manifestation, and its underlying cause. In general, seizures are divided into two groups, generalized and focal (Table 17-1). Generalized seizures affect a large volume of brain tissue in the abnormal electrical activity, whereas focal seizures involve a specific brain area. Because of this, the manifestations of focal seizures, whether simple or complex, may lead to bizarre manifestations including hallucinations, memory disturbance, visceral symptoms (abdominal symptoms), and perceptual distortions. This has often resulted in the patient with partial seizures being misdiagnosed as having a psychiatric problem.

Often the emergency physician is presented with a patient who is not actively seizing but has *had a seizure*. In this case, it is important to evaluate for secondary signs of seizure activity. These include bowel or bladder incontinence, biting of the tongue or buccal mucosa, and postictal confusion.

3. What are the causes of seizures?

Seizures are abnormal electrical activity in the brain. **Primary seizures** are recurrent episodes without an underlying cause. This is classically referred to as epilepsy. **Secondary seizures** (also called **reactive seizures**) have a (usually non-neurologic) underlying condition. Table 17-2 lists the most common etiologies for secondary seizures.

4. What is included in the differential diagnosis of seizure?

Anything that can cause a sudden disturbance of neurologic function may be mistaken for a seizure. Common processes that fall into this category include syncope, hyperventilation syndrome, migraines, movement disorders, and narcolepsy. Pseudoseizure is a special category and is discussed later.

TABLE 17-1. CLASSIFICATION OF SEIZURES

Type	Manifestations
Generalized	
Tonic-clonic (grand mal)	Loss of consciousness followed immediately by tonic contractions of muscles, then clonic contractions of muscles (jerking) that may last for several minutes. A period of disorientation (postictal period) occurs after the tonic-clonic activity.
Absence (petit mal)	Sudden loss of awareness with cessation of activity or body position control. The period usually lasts for seconds to minutes and is followed by a relatively short postictal phase.
Atonic (drop attacks)	Complete loss of postural control with falling to the ground, sometimes causing injury. It usually occurs in children.
Myoclonic	Brief, vigorous, spasmodic muscle contractions. These may affect the entire body or only specific areas.
Tonic	Prolonged muscle contraction. It occurs usually with associated deviation of the head and eyes in a particular direction.
Clonic	Repetitive jerking motions occur without any associated tonic muscle contraction.
Partial or focal	
Simple partial	Multiple patterns are possible depending on the area of the brain affected. If the motor cortex is involved, the patient will have contraction of the corresponding body area. If nonmotor areas of the brain are involved, the sensation may be paresthesias, hallucinations, or déjà vu.
Complex partial	Usually there is loss of ongoing volitional major motor activity with repetitive minor motor activity, such as lip smacking and walking aimlessly.
Partial with secondary generalization	Initial manifestations are the same as partial. However, the activity progresses to involve the entire body, with loss of postural control and possibly tonic-clonic muscle activity.

5. What should my priorities be in managing a patient who is actively seizing?

Clinical priorities are the **ABCs**. Attention is always directed to the airway first. It is rare that a seizure patient needs to be intubated. Supplemental oxygen, usually via nasal cannula, should be given because of the increased oxygen demand caused by the generalized muscle activity. Supplemental ventilation (bag-valve-mask) is rarely needed. Evaluation of the circulatory status can be readily accomplished by noting blood pressure, pulse, and capillary refill. Check the temperature and administer antipyretics in appropriate doses when needed.

Airway protection can usually be accomplished by positioning the patient on his or her side. Suctioning of the patient's oral secretions will also help decrease the chance of aspiration. However, nothing should be put into the patient's mouth that might be bitten off

TABLE 17-2. ETIOLOGIES OF SECONDARY SEIZURES
Metabolic
Hypoglycemia, hyperglycemia
Hyponatremia, hypernatremia
Hypocalcemia
Hypomagnesemia
Uremia
Hypothyroidism
Hepatic encephalopathy
High anion gap acidosis
Fever (“febrile seizures”)
Infectious diseases
Meningitis
Encephalitis
Cerebral abscess
Cerebral parasitosis
HIV
Drugs/toxins (multiple)
Subtherapeutic antiepileptic drug levels
Cocaine, lidocaine
Antidepressants
Theophylline
Alcohol withdrawal
Drug withdrawal
Structural
Trauma (recent and remote)
Intracranial hemorrhage
Vascular lesions
Mass lesions
Eclampsia
Hypertensive encephalopathy

(including fingers) and thus become an obstructing foreign body. *Gently* restraining the patient so he or she is not harmed is also important.

Diagnostic priorities should focus on likely secondary causes of seizures that are reversible. Hypoglycemia is a common cause of secondary seizures that is treatable with rapid results. Similarly, immediate evaluation of significant electrolyte abnormalities (i.e., sodium, calcium, magnesium) in a patient with prolonged seizure activity is warranted.

TABLE 17-3. ANTICONVULSANTS

Drug	Adult dose
Phenytoin	15–20 mg/kg IV at <50 mg/min
Fosphenytoin	15–20 mg PE/kg at 100–150 mg PE/min; may be given IM
Phenobarbital	20 mg/kg IV at 60–100 mg/min. May be given as IM loading dose
Valproate	15–30 mg/kg IV should be given over one hour
Pentobarbital	5 mg/kg IV at 25 mg/min, then titrate to EEG. Intubation required
Isoflurane	Via general endotracheal anesthesia

EEG, electroencephalogram; IM, intramuscularly; IV, intravenously; PE, phenytoin sodium equivalents; PR, per rectum.

6. What do I do if the patient doesn't stop seizing?

Most seizures last for less than 2 minutes. When a seizure lasts longer, pharmacologic intervention is generally indicated. Benzodiazepines are the accepted first-line therapy. The intravenous (IV) route is the preferred route of administration. Lorazepam (2–4 mg intravenously) is the conventional first choice because of theoretical issues related to duration of action. However, diazepam (5–10 mg intravenously) may also be used. Diazepam may be administered intravenously, rectally, or intraosseously but is not recommended for intramuscular use because of uneven uptake.

Once the seizure has ceased, anticonvulsants are used to keep it from recurring. Phenytoin is considered the first-line therapy. Table 17-3 shows the medications in this class along with their dosage and route of administration.

7. What is status epilepticus? How is it managed?

When seizures last longer than 5 minutes despite acute pharmacologic intervention or recur so frequently that normal mentation does not resume between the seizures, it is called *status epilepticus*. In this case, immediate pharmacologic intervention is indicated. Table 17-4 gives an algorithm that can be used to manage the patient with status epilepticus.

8. Is the history important?

The history if vitally important! The mnemonic **COLD** can be used to ensure you have covered the aspects of the seizure activity itself.

- **C**haracter: What type of seizure activity occurred?
- **O**nset: When did it start? What was the patient doing?
- **L**ocation: Where did the activity start?
- **D**uration: How long did it last?

In general, seizures start abruptly, are stereotyped (similar seizure activity recurs from attack to attack in an individual patient), are not provoked by environmental stimuli, are manifested by purposeless or inappropriate motor activity, and except for petit mal seizures, are followed by a period of confusion or lethargy (the postictal phase). Other important historical aspects include the patient's past medical history (especially previous seizure history), alcohol use,

TABLE 17-4. PROPOSED GUIDELINES FOR MANAGEMENT OF THE PATIENT WITH STATUS EPILEPTICUS

Time Frame	Measures
	Establish/maintain airway
	IV/oxygen/monitor
0–5 min	Dextrose, 0.5 gm/kg IV, if indicated
	Consider thiamine, 100 mg IV, and magnesium 1–2 gm IV for alcoholic or malnourished patients
	Lorazepam, 1 mg per min IV up to 0.1 mg/kg (or diazepam, 5 mg IV every 5 min up to 20 mg)
10–20 min	Phenytoin, 20 mg/kg IV at 50 mg/min, or fosphenytoin, 20 mg/kg PE IV at 150 mg/min
	Phenobarbital up to 20 mg/kg IV at 50–75 mg/min IV
	Valproate up to 30 mg/kg IV over 1 hour
30 min	And/or
	General anesthesia with midazolam, 0.2 mg/kg slow IVP, then 0.75–10 μg/kg/min
	Or propofol, 1–2 mg/kg IV, then 1–15 mg/kg/hr
	Or pentobarbital, 10–15 mg/kg IV over 1 hour, then 0.5–1.0 mg/kg/hr

IV, intravenous; IVP, intravenous push; PE, phenytoin sodium equivalents. Adapted from Lowenstein DH, Aldredge BK: Status epilepticus. *N Engl J Med* 338:970, 1998.

toxic ingestions, current medications, any history of central nervous system (CNS) neoplasms, and history of recent or remote trauma.

9. In addition to the neurologic examination, what other parts of the physical examination are important?

A complete head-to-toe examination is important. In addition to looking for causes of the seizure, the physician should look for trauma caused by the seizure. The examination is often normal but occasionally may give clues to an underlying problem. Specifically, examination of the skin might reveal lesions from meningococcemia, other infectious problems, or stigmata of liver failure. Examine the head for trauma. If nuchal rigidity is found, meningitis or subarachnoid hemorrhage should be suspected.

The neurologic examination is most important. Focal neurologic findings, such as focal paresis after the seizure (Todd's paralysis), may indicate a focal cerebral lesion (i.e., tumor, abscess, or cerebral contusion) as the cause of the seizure. Evaluation of the cranial nerves and the fundi can point to increased intracranial pressure.

10. What ancillary testing should I do in the patient with a history of seizures?

Extensive ancillary testing is reserved for the patient with new onset seizure. For patients with a prior history of seizures who have an unprovoked attack, measurement of appropriate serum anticonvulsant levels is all that is required. The decision to proceed with further testing depends on the patient's history and physical findings at the time of presentation. If there is a question whether the patient had a major motor seizure, then measurement of the anion gap within 1 hour of the seizure might be of benefit.

A transiently (<1 hour) raised anion gap is good evidence that a grand mal seizure has occurred. This is determined by blood samples drawn as close to the time of seizure as possible. Field blood samples are ideal for this study. If there is no anion gap acidosis, one may presume that the patient did not have a major motor seizure.

KEY POINTS: ANCILLARY LABORATORY TESTING IN PATIENTS WITH SEIZURES

1. Usually not indicated for patients with recurrent seizures unchanged from prior episodes.
2. Should be limited to those tests designed to find underlying causes of seizures.
3. Include blood chemistries (i.e., sodium, calcium, magnesium), blood sugar, appropriate drug screen, kidney function, and liver function.

11. And if the patient does not have a history of seizures?

Routine screening laboratory tests in a patient with new onset seizure who has returned to baseline have low yield. In general, it is recommended that these patients have a blood sugar and blood sodium determination. If the patient is a woman of child-bearing years, a pregnancy test can be useful because it may affect choice of antiepileptic therapy and/or disposition. The routine use of lumbar puncture in new onset seizure patients is not supported by the literature. Additional testing (i.e., calcium, magnesium, drug screening, and kidney and liver function testing) should be obtained if clinically indicated.

12. What about imaging studies?

In the patient with a first-time seizure, emergent noncontrast head CT is recommended for patients in whom a structural lesion is suspected. This includes patients with new focal deficits, persistent altered mental status, fever, recent head trauma, persistent headache, history of cancer, or presence of a coagulopathy or platelet disorder; patients who are on anticoagulation therapy; and patients who are HIV positive or otherwise immunosuppressed. Emergent neuroimaging should also be considered in the patient with first-time seizure who is older than 40 years or who has a partial seizure.

13. What should be the disposition of the patient who presents with a seizure?

Patients who present with any of the following should be considered for emergent admission to the hospital for inpatient evaluation and therapy: persistent altered mental status, CNS infection, new focal abnormality, new intracranial lesion, underlying correctable medical problem (e.g., significant hypoxia, hypoglycemia, hyponatremia, dysrhythmia, and significant alcohol withdrawal), acute head trauma, status epilepticus, and eclampsia. If the patient has a history of seizure and has a simple seizure and a subtherapeutic anticonvulsant level, then this should be addressed prior to discharge.

Patients with new onset seizures who have normal work-ups in the ED and are medically stable may be considered for discharge. In this case, follow up with the patient's primary care physician or a consulting neurologist must be arranged. The patient should be informed via ED discharge instructions on the possibility of another seizure and be advised to avoid working with hazardous machines, driving an automobile, and doing any other activities that can result in serious injury if the patient has another seizure. Also, most states have laws that require reporting a patient with seizures, if the patient has a driver's license.

KEY POINTS: EMERGENT NEUROIMAGING RECOMMENDATIONS FOR PATIENTS WITH SEIZURES

1. Seizure with focal neurological deficits, persistent altered mental status, fever, recent trauma, persistent headache, history of cancer, history of anticoagulation, HIV positive (AIDS), and when timely follow-up cannot be ensured.
2. Urgent imaging for patients who have completely recovered from their seizure and for whom no clear-cut cause has been identified to help identify a possible structural cause.
3. Patients with first-time seizures who are older than 40 years or have partial onset seizure.
4. Patients with prior history of seizures who have a new or different seizure pattern.

14. Should I start the patient with a new seizure on antiepileptic medication prior to discharge?

This decision is best made in consultation with the patient's primary care physician or neurologist. Most patients with a single new onset seizure who can be discharged do not need to be started on anticonvulsants until seen in follow-up and further testing (i.e., electroencephalogram [EEG]) is completed.

15. What is a pseudoseizure?

Pseudoseizures are functional events that may mimic seizures in their motor activity or behavior. They are not caused by abnormal electrical discharges in the brain. In general, patients with pseudoseizures have underlying anxiety or hysterical/histrionic personality disorders. Pseudoseizures are sometimes difficult to diagnose in the ED. Some maneuvers that may be of benefit include suggesting to the patient that the seizure will stop soon or attempting to distract the patient with loud noises or bright lights during the *seizure* activity. Patients who show asynchronous extremity movements, forward thrusting movement of the pelvis, and eyes deviated toward the ground no matter what the head position are more likely to be having pseudoseizures. Simultaneous video and EEG monitoring can help to differentiate true seizures from pseudoseizures. In addition, a serum prolactin level drawn within 20 minutes of seizure activity should be elevated in the patient with true seizure. A normal anion gap on serum electrolytes drawn immediately after the *grand mal seizure* supports the diagnosis of a pseudoseizure.

WEBSITES

American College of Emergency Physicians: http://www.acep.org

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CHAPTER 18

ANAPHYLAXIS

Vincent J. Markovchick, MD, FAAEM, FACEP, and Nadia S. Markovchick, MD

1. What is anaphylaxis?

A serious allergic reaction that is immunoglobulin E (IgE) mediated, rapid in onset, and may cause death after exposure to an allergen in a previously sensitized individual within minutes to hours of allergen exposure. The three diagnostic criteria include skin and mucosal edema, respiratory compromise, and hypotension and/or gastrointestinal symptoms.

2. What is an anaphylactoid reaction?

A potentially fatal syndrome clinically similar to anaphylaxis, which is not an IgE-mediated response and may follow a single first-time exposure to certain agents, such as radiopaque contrast media, salicylates, and opioids.

3. Name the most common causes of anaphylaxis.

Ingestion, inhalation, or parenteral injection of antigens that sensitize predisposed individuals. Common antigens include:

- Drugs (e.g., penicillin)
- Foods (e.g., shellfish, nuts, or egg whites)
- Insect stings (hymenoptera) and bites (snakes)
- Diagnostic agents (ionic contrast media)
- Physical and environmental agents (e.g., latex, exercise, and cold)

Idiopathic anaphylaxis is a diagnosis of exclusion that is made when no identifiable cause can be determined.

4. How do I make the diagnosis clinically?

Involvement of at least two of the following must be present:

- Cutaneous manifestations (e.g., urticaria or rash)
- Mucous membranes (angioedema)
- Upper respiratory tract (e.g., edema and hypersecretions)
- Lower respiratory tract (bronchoconstriction)
- Gastrointestinal symptoms (e.g., nausea, vomiting, or abdominal cramping)
- Cardiovascular system (vasodilation and cardiovascular collapse)

5. What are the most common signs and symptoms?

The clinical presentation ranges from mild to life-threatening. Mild manifestations that occur in most people include urticaria and dermal angioedema. Life-threatening manifestations involve the respiratory and cardiovascular systems. Respiratory signs and symptoms include acute upper airway obstruction presenting with stridor or lower airway manifestations of bronchospasm with diffuse wheezing. Cardiovascular collapse presents in the form of syncope, hypotension, tachycardia, and dysrhythmias.

6. What is the role of diagnostic studies?

There is no immediate role for diagnostic studies in the ED because diagnosis and treatment are based solely on presenting clinical signs and symptoms. However, if there is a question about the diagnosis, serum tryptase and plasma and urine histamine levels

are elevated for up to 6 hours after an allergic reaction and can be measured in the ED if there is a question about the diagnosis. There is a role for skin testing either before administration of an antigen or in follow-up referral to determine the exact allergens involved.

7. What is the differential diagnosis?

Hereditary angioedema, septic and cardiogenic shock, asthma, croup and epiglottitis, vasovagal syncope, and any acute cardiovascular or respiratory collapse of unclear origin.

8. What is the most common form of anaphylaxis, and how is it treated?

Urticaria, either simple or confluent, is the most benign and the most common clinical manifestation. This is thought to be due to a capillary leak mediated by histamine release. It may be treated by the administration of antihistamines (i.e., orally, intramuscularly, or intravenously) or epinephrine (i.e., subcutaneously or intramuscularly).

9. What is hereditary angioedema? How is it related to anaphylaxis?

Angioedema is edema of subcutaneous tissue, most often involving the face, tongue, lips larynx, gastrointestinal tract and, in men, the genitals. When angioedema occurs with urticaria, it is likely an allergic reaction. If angioedema occurs without urticaria, it may be hereditary angioedema.

10. How does the treatment of hereditary angioedema differ from that of anaphylaxis?

Hereditary angioedema is a genetic condition, usually presenting first in adolescence, involving a deficiency or absence of C1 esterase inhibitors. In adults, the condition can present as an acquired C1 esterase deficiency; angiotensin-converting enzyme (ACE) inhibitors have been implicated as a trigger. Regardless of the cause, it is not IgE mediated; antihistamines and steroids are not as effective as in anaphylaxis. Because the initial diagnosis of C1 esterase deficiency is often unknown at the time of ED presentation, treat as an allergic reaction. If there is minimal or no response to therapy, consider intravenous (IV) fresh frozen plasma (which contains C1 esterase inhibitor) or C1 esterase inhibitor concentrate.

11. Summarize the initial treatment for life-threatening forms of anaphylaxis.

a. Upper airway obstruction with stridor and edema is treated with high-flow nebulized oxygen, racemic epinephrine, and IV epinephrine. If airway obstruction is severe or increases, perform endotracheal intubation or cricothyroidotomy.
b. Acute bronchospasm is treated with epinephrine. Mild-to-moderate wheezing in patients with normal blood pressure may be treated with 0.01 mg/kg of 1:1000 epinephrine administered ***intramuscularly***. If the patient is in severe respiratory distress or has a *silent* chest, administer IV epinephrine via a drip infusion: 1 mg of epinephrine in 250 mL of D_5W at an initial rate of 1 μg/min with titration to desired effect. Bronchospasm refractory to epinephrine may respond to a nebulized β-agonist, such as albuterol sulfate or metaproterenol.
c. Cardiovascular collapse presenting with hypotension is treated with a constant infusion of epinephrine, titrating the rate to attain a systolic blood pressure of 100 mm Hg or mean arterial pressure of 80 mm Hg.
d. For patients in full cardiac arrest, administer 1:10,000 epinephrine, 1 mg slow IV push or via endotracheal tube. Immediate endotracheal intubation or cricothyroidotomy should be performed.

KEY POINTS: ANAPHYLAXIS

1. Life-threatening target organs are the upper airway mucosa, bronchiole smooth muscle, and the cardiovascular system.
2. Hypotension is the indication for IV epinephrine.
3. Administer IV epinephrine as a drip, not as a bolus, in the noncardiac arrest situation.

12. What are the adjuncts to initial epinephrine and airway management?

If intubation is unsuccessful and cricothyroidotomy is contraindicated, percutaneous transtracheal jet ventilation via needle cricothyroidotomy should be considered, especially in small children. IV diphenhydramine (1 mg/kg up to 50 mg) should be given to all patients. Simultaneous administration of an H_2 blocker, such as cimetidine, 300 mg intravenously, may be helpful. Aerosolized bronchodilators, such as metaproterenol, are useful if bronchospasm is present. For refractory hypotension, pressors, such as norepinephrine or dopamine, may be administered. Glucagon, 1 mg intravenously every 5 minutes, may be helpful in epinephrine-resistant patients who are on long-term β-adrenergic blocking agents, such as propranolol. Corticosteroids have limited benefit because of the delayed (4–6 hours) onset of action, but may be beneficial in patients with prolonged bronchospasm or hypotension.

13. What are the complications of bolus IV epinephrine administration?

When epinephrine 1:10,000 is administered via IV push in patients who have an obtainable blood pressure or pulse, there is significant potential for overtreatment and the potentiation of hypertension, tachycardia, ischemic chest pain, acute myocardial infarction, and ventricular dysrhythmias. Extreme care must be exercised in elderly patients and in patients with underlying coronary artery disease. It is much safer to give IV epinephrine by a controlled titratable drip infusion with continuous monitoring of cardiac rhythm and blood pressure.

14. What is biphasic anaphylaxis? How common is it?

A recurrence of the symptoms of anaphylaxis after the initial symptoms resolve. This may occur anywhere from several hours to as long as 72 hours. This may be caused by persistence of the allergen or immune mediators relative to the duration of the therapy. The reported incidence is between 1% and 23% of all anaphylactic reactions. Some risk factors that may make biphasic anaphylaxis more likely are:

- A history of biphasic anaphylaxis
- Delays in onset of initial symptoms, in initial treatment, or in resolution of symptoms with proper therapy
- Severe reactions involving hypotension or laryngeal edema
- Patients taking β-blockers

15. Is there a role for prophylactic treatment in anaphylaxis? How is this performed?

When the potential benefits of treatment or diagnosis outweigh the risks (e.g., administration of an antivenom for life-threatening or limb-threatening snake bites), informed consent should be obtained if the patient is competent. Pretreat with IV diphenhydramine (Benadryl) and corticosteroids and prepare an IV epinephrine infusion drip. The patient should be in an intensive care unit (ICU) setting with continuous monitoring of blood pressure, cardiac rhythm, and oxygen saturation; have full intubation and cricothyroidotomy equipment at the bedside. Under the supervision of a physician capable of immediately administering IV epinephrine and managing the airway, administration of the antigen (e.g., the antivenom) should be started. Nonionic contrast medium for diagnostic imaging studies should be given to patients with a history of anaphylaxis to ionic contrast material.

16. What about steroids?

Because corticosteroids have an onset of action of approximately 4 to 6 hours after administration, they have limited to no benefit in the initial acute treatment of anaphylaxis. The administration of hydrocortisone (250–1,000 mg intravenously) or methylprednisolone (125–250 mg intravenously), followed by a tapering dose over 7 to 10 days, is an acceptable regimen after the resolution of the initial anaphylactic episode.

17. What is the disposition of a patient who initially responds to aggressive treatment?

Although most patients become asymptomatic after early, aggressive treatment, all patients with true anaphylactic reactions should be admitted to either an ED or hospital observation unit for 2 to 4 hours minimum. Patients who rebound or continue to have life-threatening symptoms (e.g., bronchospasm, hypotension, or upper airway obstruction) should be admitted.

18. What follow-up instructions are given to patients treated for anaphylaxis?

Patients who have had a moderate-to-severe anaphylactic reaction (anything other than isolated urticaria) should be prescribed and educated in the self-administration of epinephrine into the muscles of the thigh with an autoinjector at the first sign of anaphylactic symptoms. Self-administration of oral diphenhydramine is indicated to treat mild reactions, such as urticaria or concomitant with the administration of epinephrine.

19. Is there an advantage of intramuscular (IM) over a subcutaneous epinephrine injection?

Yes, if injected into the thigh. A recent study has demonstrated higher peak plasma levels when epinephrine is injected into the muscles of the lateral thigh over subcutaneous or deltoid muscle injections.

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CHAPTER 19

LOW BACK PAIN

Kevin Andruss, MD, and Robert Hockberger, MD

1. Can I skip this chapter?

Not if you anticipate a career that involves caring for adults. Low back pain (LBP) is the second most common cause of physician visits, following upper respiratory symptoms. Approximately 70% to 85% of all people experience LBP during their lives. It is the most common cause of activity limitation in people younger than 45 years and the third most common cause in people older than 45 (after heart disease and arthritis). The cost of diagnosis, treatment, disability, lost productivity, and litigation due to LBP exceeds $50 billion annually, making it the third most expensive medical disorder in the United States, after heart disease and cancer.

2. What are the causes of LBP?

Roughly 97% of LBP cases are caused by mechanical spine disorders. Only 3% of cases are caused by nonmechanical spine disorders (especially spinal malignancy and infection) or visceral disease (particularly abdominal aortic aneurysm); however, these are medically significant causes of LBP that should not be missed. (See Table 19-1.)

3. What should I ask when taking the patient's history?

The goal of the history is to distinguish medically significant causes of LBP from the much more common mechanical spine disorders.

- **Spinal infection** should be suspected in children, immunocompromised patients, and intravenous drug users who present with localized spinal tenderness and fever (although only 50% of patients will have fever at the time of presentation).
- **Spinal malignancy** should be suspected in patients with a history of cancer or recent weight loss and in patients older than 50 years with progressive LBP lasting more than 1 month.
- **Occult spinal fracture** should be suspected in the elderly and in patients with known malignancy or osteoporosis (from steroid use or inactivity) who present with LBP of unclear cause.
- **Visceral causes** of LBP are usually distinguished by associated signs and symptoms.

4. How should I focus my physical examination?

All patients with LBP should get a complete neurologic examination, focusing on lower extremity strength, sensation, and reflexes (see Table 19-2). Mechanical spine disorders, with the exception of herniated lumbar disks or severe spondylolisthesis, should not compromise neurologic function.

- Rectal tone and sensation should be assessed if there is any concern for cord compression or sacral lesions. Localized spinal tenderness is suggestive of fracture, infection, or malignancy.
- A straight leg raise (SLR) test should be performed in patients with leg symptoms (see Question 6).
- An abdominal examination is important to assess for visceral disease including an abdominal aortic aneurysm.

TABLE 19-1. DIFFERENTIAL DIAGNOSIS OF LOW BACK PAIN

Mechanical Spine Disorders	Nonmechanical Spine Disorders	Visceral Disease
Lumbar strain	Malignancy	Abdominal aortic
Degenerative disk/facet disease	Multiple myeloma	Aneurysm
	Metastatic cancer	Pelvic organs
Herniated disk	Spinal column or cord cancer	PID
Spinal stenosis	Lymphoma	Prostatitis
Spondylolysis	Infection	Renal disease
Spondylolisthesis	Septic discitis	Pyelonephritis
Congenital spinal disease	Osteomyelitis	Nephrolithiasis
Traumatic fracture	Epidural abscess	Gastrointestinal disorders
Osteoporotic compression fracture	Shingles	Pancreatitis
	Inflammatory arthritis	Penetrating ulcer
		Cholecystitis

PID, pelvic inflammatory disease.

TABLE 19-2. CLINICAL FEATURES OF LUMBAR DISK HERNIATION

Disk	L4	L5	S1–2
Pain	Front of leg	Side of leg	Back of leg
Weakness	Knee extension	Great toe dorsiflexion	Foot plantar flexion
Sensory loss	Knee and medial foot	Side of calf, web of great toe	Back of calf and lateral foot
Reflex loss	Knee jerk	None	Ankle jerk

5. What does it mean when a patient with LBP also has leg pain?

Patients with LBP and leg pain (termed *sciatica*) may have one of two syndromes.

- *Referred pain* is caused by inflammation of the sciatic nerve. It is usually dull and poorly localized, does not radiate distal to the knee, and is not associated with a positive SLR test or neurologic impairment.
- *Radicular pain* is usually caused by nerve root impingement from a herniated lumbar disk or the narrowing of a vertebral foramen from spinal stenosis, but it may also occur with epidural metastases or abscesses in high-risk patients. It is sharp and well localized, frequently (but not always) radiates distal to the knee, invariably is associated with a positive SLR test, and may be associated with neurologic impairment.

6. How do I perform an SLR test? How do I interpret the results?

To perform an SLR test, have the patient lie supine while you slowly raise the involved leg (flexing the hip while keeping the knee extended) until the patient complains of discomfort. A positive SLR test occurs when leg elevation between 30 and 70 degrees results in pain that radiates down the involved leg; merely evoking pain confined to the low back or hamstrings does not count as a positive test. The SLR test is 91% sensitive but only 26% specific for a herniated disk; a crossed-SLR test, in which raising the uninvolved leg evokes pain radiating down the involved leg, is only 29% sensitive but 88% specific for disk herniation.

7. What *Red Flag* signs and symptoms should prompt further work-up?

Table 19-3 lists the high-risk signs and symptoms (and associated causes of LBP) that should prompt spinal imaging: lumbosacral radiographs, computed tomography (CT), or magnetic resonance imaging (MRI).

8. In addition to imaging, what other tests should I consider?

When spinal infection or malignancy is suspected, an erythrocyte sedimentation rate (ESR) should be obtained. An elevated ESR (usually greater than 60–80 mm/hr) should lead to further investigation, usually with a spinal CT or MRI. These tests should be obtained emergently in patients whenever there is evidence of acute neurologic compromise (e.g., loss of bowel or bladder function, motor weakness, or sensory changes).

9. What should I know about children who present with back pain?

Back pain is rare in children. LBP that interferes with activities previously enjoyed by a child may be indicative of serious underlying pathology. Spondylolysis and spondylolisthesis due to sports are the most common causes of LBP in children (see question 10). Scoliosis does not usually cause back pain, but conditions that cause scoliosis (e.g., cancer, fracture, limb length discrepancy, infection, or tumors) may cause pain. Although every attempt should be made to limit gonadal radiation in pediatric patients, children with LBP that is not clearly mechanical in

TABLE 19-3. RED FLAG FEATURES OF LOW BACK PAIN

Red Flag Features	Possible Cause	Imaging
Age >50 years	Fracture, malignancy	LS-spine X-ray
Trauma	Fracture	LS-spine X-ray
Fever, intravenous drug use, recent infection	Infection	MRI or CT
Unexplained weight loss, history of cancer	Metastases	LS-spine X-ray
Urinary retention, motor deficits at multiple levels, fecal incontinence, saddle anesthesia	Cauda equina syndrome	MRI
Progressive motor weakness	Myelopathy	MRI
Failure to improve after 1 month	Fracture, malignancy	LS-spine X-ray
Immunosuppression or steroid use	Fracture, infection	LS-spine X-ray, MRI, or CT
Midline spinal tenderness	Fracture, infection, malignancy	LS-spine X-ray

CT, computed tomography; LS, lumbosacral; MRI, magnetic resonance imaging.

nature should be imaged. An ESR may prove helpful when infection or malignancy is suspected.

10. Is there a difference between spondylosis, spondylolysis, and spondylolisthesis?

Yes. The terminology is confusing. The prefix *spondylo-* means vertebrae.

- **Spondylosis** is a nonspecific term for degenerative spine disease.
- **Spondylolysis** implies severe degeneration with a resulting fracture of the pars interarticularis, which is the portion of the lateral mass of the vertebrae between the superior and inferior articular processes.
- When spondylolysis occurs bilaterally, anterior slippage of one vertebral body on another can occur, termed ***spondylolisthesis***. Severe spondylolisthesis can cause neurologic impairment.

KEY POINTS: MEDICALLY SIGNIFICANT CAUSES OF LBP

1. Abdominal aortic aneurysm
2. Cauda equina syndrome
3. Lumbar disk herniation with severe neurologic compromise
4. Spinal malignancy
5. Spinal infection

11. Who should be hospitalized for treatment?

With the exception of previously discussed patients who require emergent CT or MRI, there are no standard indications for hospitalization. Patients with suspected disk herniation who are in significant physical distress or exhibit evidence of severe motor impairment of the lower extremities are often admitted for pain control and strict bed rest because failure to respond to aggressive conservative management may necessitate surgical intervention.

12. How should patients be treated in the ED?

Quickly. There is no need to await definitive diagnosis before providing pain relief. Oral or parenteral nonsteroidal anti-inflammatory drugs (NSAIDs) and application of superficial heat are first-line agents. Parenteral narcotics may be necessary to provide adequate analgesia.

13. How should patients with musculoskeletal LBP be treated as outpatients?

Bed rest is not recommended in patients with acute LBP. Patients who remain active have less pain and are better able to perform everyday activities than those who rest in bed. Most patients benefit from oral NSAIDs, but some require opioids to produce adequate analgesia during the first few days. Sedatives and muscle relaxants may be effective in treating LBP, but given the side effects (i.e., drowsiness and dizziness) and risk of long-term dependence, these should *not* be used as first-line agents.

14. What aftercare instructions should I give my patients?

Patients with suspected disk disease and patients with symptoms that don't improve within 1 to 2 weeks should be seen by a physician for follow-up evaluation. All patients should be instructed to return immediately if they develop worsening symptoms, particularly bowel or bladder dysfunction or progressive weakness.

15. What happens to patients with LBP when they leave the ED?

The prognosis for patients having a first episode of mechanical LBP is good: 70% are better by 1 week, 80% by 2 weeks, and 90% by 1 month. Most studies comparing medical management, chiropractic manipulation, and other treatment modalities rarely find significant differences in long-term outcome because almost everyone gets better no matter what you do. Patients who do not improve with conservative management may have significant medical disorders (e.g., inflammatory disorders, malignancy, infections, or disk disease) that were not apparent at the time of initial evaluation or, alternatively, may suffer from psychiatric disorders, drug dependence, or job dissatisfaction.

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NONTRAUMATIC OCULAR EMERGENCIES

Martin R. Huecker, MD, and Daniel F. Danzl, MD

1. What are some tricks to evaluate the red eye?

Always document near or far visual acuity in each eye independently. Topical application of anesthetic drops should decrease or eradicate pain secondary to an abrasion or conjunctivitis (not so with iritis or glaucoma). Redness at the corneal-scleral junction (perilimbic flush) suggests iritis or glaucoma. Shining a light into the normal eye should make the opposite eye hurt if the patient has iritis (because of consensual movement of the inflamed affected contralateral iris). In addition to the consensual pupillary reflex test, the accommodative test is suggestive, which is simply pain precipitated by accommodation. Pain with either maneuver suggests ciliary spasm.

2. What typical findings help with the differential diagnosis of the red eye?

See Table 20-1.

3. What is conjunctivitis?

Inflammation of the bulbar and palpebral conjunctivae or mucous membranes. Viral conjunctivitis is usually bilateral with clear epiphora or tearing and may be associated with an upper respiratory infection (URI). A preauricular node suggests epidemic keratoconjunctivitis (adenovirus). Two common viral pathogens are herpes simplex, with dendritic ulcers, and herpes zoster, with involvement of the fifth cranial nerve. Ocular zoster is suggested by involvement of the nasociliary branch of V_1, manifested by lesions on the tip of the nose (Hutchinson's sign).

Bacterial conjunctivitis initially may be unilateral with purulent crusty drainage. Always consider an undiagnosed foreign body with unilateral conjunctivitis. *Chlamydia* or *Gonococcus* should be considered in neonates or adults with sexually transmitted diseases. Allergies may cause papillae under the lids, chemosis, and itching.

4. How is conjunctivitis treated?

Common agents include aminoglycoside drops and sulfacetamide; the latter stings, which can decrease compliance. Erythromycin 0.5% is available only in ointment form. Reserve the topical fluoroquinolones for more severe infections and for contact lens wearers who are at risk for *Pseudomonas*. Avoid neomycin because hypersensitivity reactions are common.

5. What is endophthalmitis?

Infection or inflammation within the globe. It usually is seen as a collection of pus in the anterior chamber (hypopyon) that resembles a dependent meniscus similar to the blood collection in a hyphema. Antecedent causes include corneal ulcers, direct inoculation or hematogenous spread, and conjunctivitis with organisms capable of penetrating the cornea (e.g., *Neisseria gonorrhoeae, Corynebacterium, Listeria,* or *Haemophilus aegyptius*).

6. What is the difference between periorbital and orbital cellulitis?

Periorbital (preseptal) cellulitis is soft-tissue infection of eye structures anterior to the tarsal plate, usually localized to the eyelids and conjunctivae. **Orbital cellulitis** is a more serious infection (behind the septum) that involves posterior eye structures. Both tend to be unilateral and may be preceded by trauma and upper respiratory, sinus, or dental infections. Orbital

TABLE 20–1. DIFFERENTIAL DIAGNOSIS OF THE RED EYE

	Conjunctivitis	Acute Iritis	Angle-Closure Glaucoma
Incidence	Extremely common	Common	Uncommon
Discharge	Moderate to copious	Reflex epiphora	None
Vision	Normal	Slightly blurred	Very blurred (halos)
Pain	Gritty	Moderate	Severe
Conjunctival injection	Diffuse	Perilimbic	Perilimbic
Cornea	Clear	Keratotic precipitates	Steamy or hazy
Pupil size	Normal	Constricted or dilated	Dilated
Pupillary light response	Normal	Poor and painful	Poor
Intraocular pressure	Normal	Normal	Elevated

cellulitis is most often the result of direct spread from ethmoid sinusitis, whereas periorbital cellulitis usually is caused by hematogenous spread of bacteria.

7. How do I differentiate clinically between periorbital and orbital cellulitis?

The two may be difficult to distinguish clinically, especially in children. **Periorbital cellulitis** tends to cause local eyelid symptoms and occasionally ocular discharge and may be associated with fever or leukocytosis. Visual acuity and pupillary reflexes are normal.

Orbital cellulitis may present with all of the previous symptoms plus exophthalmos, fever, and pain with extraocular movements. Decreased visual acuity, loss of sensation over the ophthalmic and maxillary branches of the trigeminal nerve in V_1 and V_2 (division of cranial nerve V), and increased intraocular pressure are uncommon findings. Contrast computed tomography (CT) scanning of the orbit is liberally indicated with periorbital swelling when there is a possibility of postseptal infection.

8. What is the common clinical presentation of cavernous sinus thrombosis?

Patients often progress from fever, headache, and chemosis to ophthalmoplegia, exophthalmos, and altered level of consciousness. Paralysis of cranial nerves III, IV, and VI is usually noted. In exophthalmos, the sclera is visible above and below the cornea. Magnetic resonance imaging (MRI) is indicated.

9. Describe the clinical presentation of iritis.

Patients often present with perilimbic injection, ciliary spasm, and a constricted miotic pupil. Iritis can be bilateral and misdiagnosed as conjunctivitis. Perform a slit lamp examination of the anterior chamber for cells, flare, and for keratotic precipitates (white cells) on the back of the cornea.

10. How is iritis treated?

Iritis is treated with systemic analgesics and a topical cycloplegic, not simply a mydriatic, to paralyze accommodation and dilate the iris. This prevents adhesions between the iris and the lens (posterior synechiae). Consider steroids in consultation with an ophthalmologist.

11. What is acute angle-closure glaucoma?

Glaucoma is optic nerve damage from increased intraocular pressure. In a patient with a narrow anterior chamber angle, reduced illumination causes mydriasis; folds of the peripheral

iris can block the angle, which prevents aqueous humor outflow. The rapid elevation of intraocular pressure causes a hazy cornea, ciliary flush, firm globe, and optic nerve damage if not treated promptly. The diagnosis may be delayed by the misleading systemic complaints of nausea, vomiting, and abdominal pain.

12. How is acute angle-closure glaucoma treated?

Acute glaucoma is treated with intravenous mannitol or glycerol to decrease intraocular pressure by osmotic diuresis, topical miotics (i.e., 2% pilocarpine or 0.5% timolol) if not contraindicated to decrease pupil size and increase aqueous outflow, and acetazolamide intravenously to decrease aqueous production. Topical sympathomimetics such as apraclonidine also reduce aqueous humor production. Emergent ophthalmologic consultation is indicated.

13. What is a subconjunctival hemorrhage?

Subconjunctival hemorrhage occurs when a blood vessel ruptures under the conjunctiva. Without trauma, it often results from a Valsalva maneuver associated with coughing or vomiting. Reassure the patient that vision will not be affected and that the blood will be absorbed over 10 to 14 days. Patients on anticoagulants should have their international normalized ratio (INR) measured.

14. What are some common diseases of the cornea?

Ulcerations are often surrounded by a cloudy white cornea. Emergent ophthalmologic recommendations often include a topical fluoroquinolone, such as moxifloxacin.

A pterygium is a wedge of conjunctival fibrovascular tissue that extends over the cornea, unlike a pinguecula. Both are benign and can be electively excised.

15. What are some of the unique issues regarding ophthalmologic pharmacology?

Topical agents may have systemic effects, so exercise caution when prescribing β-blockers, vasoconstrictors, and anticholinergics. Ointments have a longer duration of action, but blur vision. Generally wait 10 minutes before instilling different drops.

Diagnostic medications include stains, such as fluorescein, that help identify corneal and conjunctival abnormalities, and topical anesthetics, which should never be dispensed. Nonsteroidal anti-inflammatory drugs, such as ketorolac or diclofenac, are useful for pain relief. Topical corticosteroids should generally be used after consultation with an ophthalmologist.

Miotic eye drop bottles have green tops, and mydriatic/cycloplegic agents have red tops. Never allow Hemoccult® drops (yellow or blue top) in an *eye room* because severe alkali burns can occur.

Some patients will present with a pupil dilated from a medication. If 1% pilocarpine fails to constrict the pupil, it is pharmacologically blocked, most commonly by phenylephrine, handling a scopolamine patch, or aerosolized anticholinergics/β-agonists. Other causes of a unilateral dilated pupil include post-traumatic mydriasis, third nerve palsy, or a normal variant.

16. Name some of the considerations involving pupillary dilation.

Phenylephrine (2.5%) is a direct sympathomimetic and mydriatic. Dilation may last 4 hours, and patients with a shallow anterior chamber may develop acute glaucoma after leaving the ED. Pupils generally do not require dilation in the ED. A panoptic ophthalmoscope provides a five times larger view of the undilated fundus. For short-term cycloplegia, consider tropicamide (1–6 hours) or 2% to 5% homatropine (1–2 days); never use atropine (1–3 weeks).

17. What does the presence of an afferent pupillary defect (APD), also known as a Marcus Gunn pupil, indicate?

If the patient has an APD, it confirms damage in the retina or optic nerve. To perform the swinging flashlight test, swing the light after several seconds from the normal eye to the other eye. After a brief pupillary constriction in the abnormal eye, the redilation in response to light reflects afferent deprivation; response may only be appreciated in a dark room.

KEY POINTS: COMMON CAUSES OF AN APD

1. Central retinal artery occlusion
2. Central retinal vein occlusion
3. Optic neuritis
4. Retrobulbar neuritis

18. In a patient with anisocoria, how does one determine which pupil is abnormal?

Begin the examination in a darkened room; if there is more anisocoria in the light, the large pupil is failing to constrict and is abnormal. More anisocoria that develops going into the dark indicates that the miotic pupil is failing to dilate. Never just assume that the larger pupil is abnormal.

KEY POINTS: COMMON CAUSES OF ANISOCORIA

1. Horner's syndrome
2. Argyll-Robertson pupil
3. Adie's pupil
4. Post-traumatic or medication-induced mydriasis
5. Third nerve palsy

19. What are common causes of a miotic pupil?

The two most common are Horner's syndrome and an Argyll-Robertson pupil. The clinical manifestations of Horner's syndrome include ptosis, miosis, and anhydrosis (in a cold ED, check for dilated conjunctival vessels). Bronchogenic carcinoma, stroke, and brachial plexus pathology may present with Horner's syndrome.

The Argyll-Robertson pupil is miotic and irregular, and displays light-near dissociation. The pupil constricts to accommodation but not to light. This finding is common with diabetes and syphilis. A common testing error is to hold and shine a penlight directly in front of the eye, which can cause the pupil to constrict from accommodation, not light.

20. Is there another cause of light-near dissociation?

The only other cause is Adie's pupil, which results from idiopathic parasympathetic denervation in the ciliary ganglion in the eye. The patient is often a young female with a mydriatic pupil that accommodates but does not react to light. Herpes zoster is another cause of Adie's pupil. There are no diseases that cause a pupil to react to light but fail to accommodate.

21. What are some common causes of nontraumatic loss of vision?

See Table 20-2.

22. Describe the presentation and treatment of central retinal artery and central retinal vein occlusion.

Both occur in middle-aged atherosclerotic patients or elderly hypertensive patients and present as sudden painless loss of vision. Embolic occlusion of the retinal artery or its branches results in a dilated nonreactive pupil with an APD on the affected side. The retina is

TABLE 20-2. COMMON CAUSES OF NONTRAUMATIC LOSS OF VISION

Transient monocular	**Acute binocular**
Amaurosis fugax	Migraine
Temporal arteritis	Vertebral basilar insufficiency
Migraine	Cerebrovascular disease
Persistent monocular or binocular	Toxins (e.g., methanol, salicylates, quinine)
Central retinal artery occlusion	Hysteria
Central retinal vein occlusion	Malingering
Retinal detachment or hemorrhage	
Vitreous or macular hemorrhage	
Optic or retrobulbar neuritis	
Macular degeneration	

pale with a cherry-red spot at the macula (macular blood supply is from the choroidal circulation). Occasionally, amaurosis fugax precedes central retinal artery occlusion. The funduscopic examination of an ischemic central retinal vein occlusion is described as a *blood and thunder fundus* because of the presence of multiple large hemorrhages. Efforts to decrease intraocular pressure and dilate retinal vessels by increasing the pCO_2 (e.g., paper bag or carbogen), and globe massage are rarely useful acutely for arterial occlusions. Prognosis for both entities is poor.

23. What are other causes of sudden painless monocular loss of vision?

Suspect vitreous hemorrhage in diabetics with an obscured red reflex and retinal details. Nontraumatic retinal detachments are more common in patients with significant myopia. Patients often see flashing lights or a falling curtain. Most commonly, patients report dark floating spots or floaters, which reflect vitreous separations and not a retinal detachment.

24. How do optic neuritis and papilledema differ?

Although these two processes appear similar on funduscopic examination, **optic neuritis** involves focal demyelination of the optic nerve, resulting in a hyperemic nerve head developing over hours to days. The average age of onset is in the thirties, and there is a 40% association with multiple sclerosis.

Papilledema is swelling of the optic disc caused by increased intracranial pressure. It is usually bilateral but may be asymmetric and may be the result of brain abscess or tumor, intracranial bleeding, meningitis or encephalitis, hydrocephalus, severe hypertension, or pseudotumor cerebri. The earliest sign of papilledema is the loss of spontaneous venous pulsations normally present in 75% of patients. When difficult to appreciate, they can be elicited with ipsilateral jugular compression. (See Table 20-3.) Bedside ocular ultrasonography can facilitate the diagnosis of vitreous hemorrhage, a detached retina, and increased intracranial pressure (nerve sheath diameter).

25. What are a couple of tricks to prove that a patient can see?

Induce nystagmus by spinning an opticokinetic drum, or simply hold a mirror in front of the eyes and slowly move it—tracking requires vision.

TABLE 20–3. OPTIC NEURITIS VERSUS PAPILLEDEMA

	Optic Neuritis	Papilledema
Pupil reactivity	Slow	Normal
Visual acuity	Decreased	Normal
Ocular pain	Present	Absent
Usual localization	Unilateral	Bilateral
Fundus	Blurred disc margins	Blurred disc margins

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NONTRAUMATIC ENT EMERGENCIES

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CHAPTER 21

EPISTAXIS

1. What are the most common causes of epistaxis?

Nosebleeds usually occur spontaneously, often secondary to dry nasal mucosa or infection. Infectious causes are most commonly viral or bacterial rhinitis. Local trauma from nose picking and direct blows to the nose are also frequent causes. Less commonly seen causes include foreign bodies, tumors, coagulopathies, use of anticoagulant drugs such as aspirin, Plavix, or warfarin, and exposure to toxic or caustic materials, such as cocaine. Approximately 60% of people experience at least one nosebleed in their lifetime, and 6% of those seek medical attention for it.

2. Doesn't hypertension cause epistaxis?

Probably not acutely. The hypertensive patient who presents with a nosebleed typically has hypertension as a chronic condition and has developed atherosclerosis, which makes the blood vessels relatively fragile and more prone to bleeding. Recent studies suggest an association between hypertension and epistaxis, but proof of a causal relationship has not been established.

3. Does bleeding originate from any one particular source?

Approximately 90% of nosebleeds originate from the anterior portion of the nose, a rich vascular network on the anterior-inferior portion of the septum known as *Kiesselbach's plexus* or *Little's area*. The blood supply for most of this region is derived from the external carotid system. From a practical standpoint, a nosebleed with a source that can be seen directly or is controlled after proper placement of an anterior nasal pack is considered anterior. Posterior bleeds arise from a branch of the *sphenopalatine* artery and tend to be more difficult to control. Posterior bleeds usually occur in patients older than 50. The hemorrhage tends to be more severe with patients often swallowing large amounts of blood.

4. List the key questions to ask the patient.

a. Is there a prior history of nosebleeds?
b. A history of excessive alcohol use or bleeding dyscrasias?
c. Was trauma involved? Nose picking?
d. On which side did the bleeding start?
e. Any recent sinus infections or surgeries?
f. Warfarin, Plavix or aspirin use?

5. Summarize the key points to successful management of nosebleeds.

There are two key considerations. The first is **preparation**. Because epistaxis rarely presents as a life-threatening condition, there is time to assemble the necessary equipment and supplies for treatment (Table 21-1). While obtaining the history and quickly assessing the ABCs (airway, breathing, and circulation), have the patient pinch the nose firmly (bilateral nasal ala compressing the septum) or place a nasal clamp on the patient with firm pressure on the septum. The examiner should wear disposable gloves, mask, and eye protection. The second key is to **identify the source** of the hemorrhage.

TABLE 21-1. SUPPLIES FOR THE TREATMENT OF NOSEBLEEDS

Examination	Stabilization	Treatment
Protective garb	Bayonet forceps	Silver nitrate cautery sticks
Head lamp or light	Cotton pledgets	Electrocautery (if available)
Nasal speculum	Lidocaine 4%	Gelfoam (or similar material)
Cotton swabs	Epinephrine 1:1000	Merocel sponge or nasal tampon
Fraser tip suction	Tetracaine 0.5%	1/2-inch petroleum-impregnated gauze
Emesis basin	Oxymetazoline hydrochloride (Afrin)	Antibiotic ointment
4 × 4 gauze	0.25% phenylephrine (Neo-Synephrine)	Foley catheter or commercial balloon
		Rolled 4 × 4 gauze with silk suture

From Lucente F, Har-El G, editors: *Essentials of otolaryngology*, ed 4, New York, 1999, Lippincott, Williams & Wilkins; Kucik CJ, Clenney T: Management of epistaxis. *Am Fam Physician* 7(12):305–311, 2005.

6. How do I treat epistaxis?

Using a nasal speculum, suction, and water-moistened cotton swabs, remove the existing clots in an effort to identify the bleeding site. Alternatively you can ask the patient to blow the nose, which helps in the removal of clot. Insert a pledget soaked with topical anesthetic plus a vasoconstrictor (e.g., lidocaine 4% and phenylephrine) for 5 to 10 minutes. Remove the pledget and attempt to identify the bleeding site. If the source is in Kiesselbach's plexus and is less than 1 cm^2, use silver nitrate or electrocautery. Alternatively, a small piece of absorbable gelatin sponge (Gelfoam), absorbable cellulose (Surgicel), or similar substance may be moistened with a vasoconstrictor and applied to the bleeding site.

If these methods are unsuccessful, an anterior nasal packing should be inserted. A dry Merocel sponge or nasal tampon should be placed by coating the outside with antibiotic ointment with insertion into the nostril. Once in place, moisten this with saline or phenylephrine until it expands to tamponade the nasal cavity. If inspection of the posterior pharynx reveals no continued bleeding after the vasoconstrictor wears off (about 30 minutes), the patient may be discharged.

7. Any other pearls about treatment with silver nitrate?

- Silver nitrate is only helpful when the bleeding is slow or minimal. It won't work in the presence of brisk bleeding.
- Only hold the silver nitrate to the septum for 5 to 10 seconds and only use electrocautery or chemical cauterization (silver nitrate) on *one* side of the septum. Cauterizing for too long or to both sides of the septum can lead to perforation or permanent damage to the blood supply of the region.

8. What are the important discharge instructions?

a. The pack (any type) should be left in place for 2 to 3 days.
b. Treat each patient who has packing with prophylactic antistaphylococcal antibiotics to prevent sinusitis or toxic shock syndrome. Sinusitis may occur because the paranasal sinuses cannot drain properly with a pack in place. Either cephalexin or trimethoprim-sulfamethoxazole is a typical choice.
c. Any recurrent epistaxis that fails to respond to direct firm pressure for 10 minutes should be seen in the ED.
d. Regular application of petroleum jelly or antibiotic ointment and use of room humidifiers may prevent bleeding from desiccated nasal mucosa.

KEY POINTS: INSTRUCTIONS FOR PATIENTS WITH AN ANTERIOR NASAL PACKING

1. The pack should be left in place for 2 to 3 days.
2. Treat each patient who has packing with prophylactic antistaphylococcal antibiotics.
3. If recurrences fail to respond to direct firm pressure for 10 minutes, the patient should seek medical attention.
4. Regular application of petroleum jelly or antibiotic ointment and use of room humidifiers may prevent bleeding from desiccated nasal mucosa.

9. How do I diagnose posterior epistaxis?

If a properly placed anterior pack fails, the patient may have a posterior bleed and more aggressive treatment is required. Posterior packs are accomplished with rolled 4 × 4 inch gauze, a Foley catheter (French 16 or 18), or other commercially available balloon products. Take a Foley and place in the nose until you can see it in the oropharynx. Fill the balloon with 10 to 15 mL of saline and pull gently but firmly until the balloon is wedged in the far posterior nasal cavity. Clamp the Foley in this position with an umbilical clamp placed just outside the nose. Because the Foley will be stretched a bit, place gauze between the nose and clamp so as not to cause pressure necrosis of the nose.

10. Do I discharge a patient to home with a posterior pack?

No. All patients who require posterior packing require an admission and otolaryngology (ENT) consultation. Although the mechanism is unclear, posterior packing stimulates the *nasopulmonary reflex*, which can lead to hypoxia and apnea. The patient should be on supplemental oxygen and continuous pulse oximetry. It should be noted that 10% of posterior bleeds are not controlled by posterior packing.

KEY POINTS: DIAGNOSIS AND MANAGEMENT OF POSTERIOR EPISTAXIS

1. When an anterior packing fails to control epistaxis, a posterior bleed originating from sphenopalatine artery should be suspected.
2. Treatment consists of an ENT consult, posterior nasal packing and hospital admission to monitor for hypoxia and apnea secondary to the nasopulmonary reflex.

11. When should I consult an ENT specialist?

ENT referral is needed if you cannot control the anterior bleed with adequate **bilateral** nasal packing, raising suspicion of a posterior bleed. The patient may need endoscopic cauterization, ligation of the sphenopalatine artery, embolization, or septal surgery. An outpatient referral can be made for those patients with recurring anterior epistaxis.

12. What is the role of interventional radiology (IR)?

Severe epistaxis may be refractory to more traditional packing methods. Surgical ligation or arterial embolization may be required. IR-based techniques were developed in response to the near 15% failure rate for surgical ligation and are typically targeted at embolizing the sphenopalatine artery. However, the decision-making process when choosing between these two techniques remains controversial. Severe epistaxis from the ethmoidal system may be better treated with surgical ligation because of the subsequent risk of blindness and stroke associated with embolization of the internal carotid system. In contrast, critical patients may not be stable enough for general anesthesia. In a recent study of nearly 10,000 inpatients with an admitting diagnosis of epistaxis, no difference was found between transfusion rates or length of stay in those patients treated with packing, ligation, or embolization. Embolization, however, was associated with a significantly higher cost.

13. Didn't you forget to mention laboratory studies?

No. Most patients don't need them. The exceptions are patients taking warfarin or those patients who are hemodynamically unstable. In this case, a complete blood count, coagulation studies, and a type and screen are most often adequate.

FOREIGN BODIES

14. How should I remove a foreign body from the ear?

The following instruments can assist in extraction: alligator forceps, right-angle probe, tissue forceps, cyanoacrylate glue, Fraser tip suction, irrigation syringe, Adson forceps, Fogarty biliary catheter, ear curette, water-pik, skin hook, and day hook.

If a live insect is in the external auditory canal (EAC), it should first be killed by instilling 2% lidocaine (which is quicker and less messy than mineral oil) before removal. If the tympanic membrane is intact and space exists between the EAC and the object, a stream of liquid can be directed behind the foreign body to force it out. A mixture of water and isopropyl alcohol as an irrigation solution tends to cause less swelling of organic matter and is evaporated more quickly. Direct instrumentation or suction removes most other objects. Cyanoacrylate glue at the end of a Q-tip or small balloon-tipped catheter can do also do the trick. Using an aural speculum when guiding the Q-tip will prevent adherence of glue to the external auditory structures.

15. How do patients with nasal foreign bodies present?

Unless the patient or witness reports the insertion of a foreign body, the chief complaint is that of unilateral, malodorous nasal discharge. The discharge may be mucoid or serosanguineous but is classically purulent.

16. Is there any special trick to removing foreign bodies from the nose?

A small Foley catheter (or commercially available Katz extractor) can be passed into the superior affected nasal cavity. Once past the foreign body, the balloon is insufflated and the device pulled out, taking the foreign body with it. Alternatively, the provider can prepare a 50/50 mixture of a topical vasoconstrictor and 4% topical lidocaine, and spray it into the involved nostril with an atomizer or spray bottle. Nebulized epinephrine has also been used with good results. This anesthetizes nasal mucosa and reduces congestion, facilitating removal. When this is done, the patient can occlude the unaffected nostril and blow forcefully, often expelling the object.

If the patient is unable or unwilling to attempt this maneuver, positive-pressure insufflation can be attempted. The unaffected nostril is occluded, and a quick breath is delivered through a facemask connected to an Ambu-bag. Alternatively, a parent or caregiver can do this in direct, mouth-to-mouth fashion. If insufflation maneuvers are unsuccessful, an attempt should be made to remove the foreign body with suction or forceps. The techniques listed for ear foreign body removal can be applied to the nose.

17. "I think I've got something stuck in my throat." How is the patient with this complaint managed?

The fact that the patient can talk is a good sign. Airway compromise must be immediately addressed. The patient should be asked about the nature of the foreign body, duration of the sensation, the ability to swallow liquids or solids, and the perceived location of the object. Patient estimates of location are often surprisingly accurate.

Direct visualization can identify sharp objects, such as fish bones, that may become impaled in the posterior pharynx or the base of the tongue. Indirect or fiberoptic laryngoscopy, in conjunction with local anesthesia (e.g., nebulized lidocaine), may help localize objects stuck in the vallecula, epiglottis, or pyriform sinus.

It is important to note that the pain of myocardial ischemia can present as a feeling of something stuck in the throat. If the history and physical are at all suspicious for acute coronary syndrome, consider an electrocardiogram (ECG) and troponins.

18. If the physical examination does not reveal the foreign body, what should be done next?

Soft-tissue density lateral radiographs of the neck or chest radiographs should be obtained. Large, sharp, angulated objects tend to lodge in the esophagus. If radiographs do not localize the foreign body, a water-soluble radiographic contrast agent like Gastrografin can be used as part of an esophagram done under fluoroscopy (by radiology). Barium should be avoided initially because it interferes with visualization during endoscopy. Esophagoscopy should be considered in patients with persistent symptoms or when the diagnosis is unclear.

19. If I can see a foreign body, how do I remove it?

Apply a topical spray anesthetic, such as topical benzocaine or nebulized 4% Xylocaine. Objects that are visualized may be removed with bayonet forceps or a Kelly clamp. Smooth objects such as coins in the esophagus for less than 24 hours can be removed by placing the patient in Trendelenburg position (head down), passing a Foley catheter beyond the object, expanding the balloon, and withdrawing the catheter. Due to potential complications, this procedure is typically performed under fluoroscopy by experienced radiologists.

Pharmacologic treatments for passage of esophageal foreign bodies are variably effective. Sublingual nitroglycerin relaxes the lower esophageal sphincter and is occasionally successful at relieving a distal obstruction, such as a food bolus. Intravenous glucagon (0.5–2.0 mg) also relaxes the lower esophageal sphincter, allowing a distal obstruction to pass. However, because glucagon commonly elicits vomiting, it has been associated with esophageal perforation in this setting. Benzodiazepines may also be effective. *Never* use papain-containing agents; they dissolve meat and, due to gas formation, are associated with esophageal perforation. Sharp objects should be removed endoscopically.

KEY POINTS: ESOPHAGEAL FOREIGN BODIES

1. In the patient presenting with an esophageal foreign body sensation, esophagoscopy should be considered with persistent symptoms or an uncertain diagnosis.
2. Because glucagon commonly elicits vomiting, it may cause esophageal perforation.

20. Any other pearls?

Of esophageal foreign bodies that pass through the gastrointestinal (GI) tract, 80% to 90% pass without significant problems. The remainder requires surgical removal. These latter objects tend to be sharp or long (>6.5 cm) and are among the 1% that cause perforation. A special case should be made for disk or button batteries. Because most are prone to leakage, every effort should be made to remove them immediately if localized to the esophagus. Otherwise, their location in the GI system should be followed with serial X-rays until elimination is confirmed.

KEY POINTS: NATURAL HISTORY OF GI FOREIGN BODIES

1. Of foreign bodies, 80% to 90% pass through the gastrointestinal tract without significant problems.
2. The following often require surgical removal: sharp or long (>6.5 cm) objects, disk or button batteries, and those that have not migrated on serial radiographs.

SINUSITIS

21. What is sinusitis? What are the common causes?

Sinusitis is an inflammation of the paranasal sinuses, which include the maxillary, ethmoid, frontal, and sphenoid sinuses. It is the consequence of ostia occlusion, most commonly caused by local mucosal swelling secondary to a viral upper respiratory infection. Allergies, trauma, mechanical obstruction from tumors, foreign bodies, or abnormal anatomy may also cause occlusion that leads to bacterial overgrowth and excess mucus production. Of all viral upper respiratory infections, 0.5% to 5% are complicated by bacterial rhinosinusitis. When symptoms are present less than 3 weeks, the process is characterized as acute.

22. How do I make the diagnosis?

The four most helpful signs and symptoms when diagnosing bacterial rhinosinusitis are purulent nasal discharge, upper tooth or facial pain (especially unilateral), maxillary sinus tenderness (unilateral), and a worsening of symptoms after initial improvement. The physical examination is often unrewarding. Anterior rhinoscopy with a headlamp and nasal speculum may reveal the presence of pus, foreign bodies, masses, or anatomic abnormalities.

23. Which other diagnostic studies should I pursue?

Plain films and computed tomography (CT) are **not** recommended for initial diagnosis but may be used for recurrent or chronic conditions. A single Water's view is as sensitive as a full sinus series. Findings may include mucosal thickening (>6 mm), air-fluid levels, and opacification. For uncomplicated sinusitis, CT is not specific because 40% of asymptomatic patients and 87% of patients with a recent upper respiratory infection have abnormal findings on CT scan. However, CT can be used to diagnose intrafacial or intracranial involvement. Nasal endoscopy is an excellent modality for identifying disease but is done only by an otolaryngologist and rarely on an emergent basis.

24. How is sinusitis treated?

Approximately 65% of cases of acute rhinosinusitis in adults and children will resolve spontaneously. Most patients with a viral upper respiratory infection improve within 7 days. Thus, antibiotics should be reserved for patients who meet the clinical criteria described previously, and those whose symptoms have persisted for more than 7 days. The most likely organisms are *Streptococcus pneumoniae*, nontypable *Haemophilus influenzae*, *Moraxella catarrhalis*, other *Streptococcus* species, and anaerobes.

Initial antibiotic therapy options include amoxicillin, trimethoprim-sulfamethoxazole, amoxicillin-clavulanate, doxycycline, or azithromycin. In children, consider amoxicillin, amoxicillin/clavulanate, cefpodoxime, or cefuroxime. Appropriate treatment duration remains unclear; a 10-day course is most often used. The use of vasoconstrictor sprays such as phenylephrine (Neo-Synephrine) or oxymetazoline (Afrin) offer symptomatic relief but should not be used longer than 3 days because of the propensity for rebound edema. Antihistamines should be avoided because they are implicated in mucosal crusting and blockage of the ostia. Ultimately, daily nasal saline irrigation and nasal topical steroids should be encouraged before antibiotics are prescribed.

25. Which patients need referral and admission? What are the complications?

If there is no improvement after two complete courses of antibiotics, the patient should be referred to an otolaryngologist. Complications arising during therapy can be classified as local, orbital, and intracranial. Patients with sinusitis who show evidence of orbital or central nervous system involvement should be treated as medical emergencies.

Locally, mucoceles and osteomyelitis can develop. Orbital complications are the most frequent, especially in children, and range from cellulitis to abscess formation. Cavernous sinus thrombosis, resulting from the direct spread of infection through valveless veins, is truly life-threatening. It is heralded by a toxic appearance, high fever, cranial nerve palsies, retinal engorgement, and bilateral chemosis and proptosis. Other intracranial complications demanding aggressive intensive therapy include meningitis, subdural empyema, and brain abscess. The majority of these complications can be diagnosed by CT.

26. Any other pearls?

Yes. Check a fingerstick glucose in a sick patient with sinusitis. *Mucor* in diabetic patients and *Aspergillus* in immunocompromised patients can be life-threatening. These patients require hospital admission and specialist consultation.

KEY POINTS: SINUSITIS

1. The four most helpful physical examination signs and symptoms when diagnosing bacterial rhinosinusitis are purulent nasal discharge, upper tooth or facial pain (especially unilateral), and maxillary sinus tenderness.
2. A single Water's view is as sensitive as a full sinus series. Findings may include mucosal thickening (>6 mm), air-fluid levels, and opacification.
3. For uncomplicated sinusitis, CT is not specific because 40% of asymptomatic patients and 87% of patients with a recent upper respiratory infection have abnormal findings on CT scan.
4. Cavernous sinus thrombosis resulting from sinusitis is heralded by a toxic appearance, high fever, cranial nerve palsies, retinal engorgement, and bilateral chemosis and proptosis.
5. Mucor in diabetic patients and *Aspergillus* in immunocompromised patients can be life-threatening.

EPIGLOTTITIS

27. How did George Washington die?

George Washington is believed to have died from epiglottitis. It is recorded that on December 14, 1799, the morning of his death, he had a severe sore throat, developed stridor and hoarseness, and was unable to lie supine.

28. List the signs and symptoms of epiglottitis in adults.

Symptoms

- Sore throat (100%)
- Odynophagia/dysphagia (76%)
- Fever (88%)
- Shortness of breath (78%)
- Anterior neck tenderness
- Hoarseness or muffled (*hot potato*) voice

Signs

- Lymphadenopathy
- Drooling
- Respiratory distress
- Extreme pain with palpation of the larynx

29. What is the thumbprint sign?

A finding on lateral neck radiographs caused by the presence of an edematous epiglottis. Lateral neck films are of limited use because they are only 38% sensitive and 76% specific.

30. Name the most common organisms identified in adult epiglottitis.

The two most common organisms found are *H. influenzae* and beta-hemolytic streptococci. In most cases, no organism is found pointing to a viral cause. With the introduction of the Hib vaccine in children, the reservoir for *H. influenzae* has decreased dramatically so that epiglottitis is now seen more frequently in adults.

31. How do I manage epiglottitis? What signs and symptoms indicate the need for airway intervention?

Antibiotics should be started immediately. Use a second- or third-generation cephalosporin active against *H. influenzae* and beta-hemolytic streptococci such as cefotetan or cefoxitin. Steroids are often used but remain controversial and have not been shown to provide any benefit. It is traditionally taught that racemic epinephrine should be avoided because of the potential for rebound edema, but there is little data to support this. Patients with symptomatic respiratory distress, stridor, drooling, shorter duration of symptoms, and *H. influenzae* bacteremia are at increased risk for airway obstruction. Patients with a respiratory rate of less than 20 breaths per minute and no respiratory distress should be observed closely in an intensive care unit (ICU). In patients with a respiratory rate greater than 30 breaths per minute, moderate-to-severe respiratory distress, PCO_2 of greater than 45 mm Hg, or cyanosis, consider immediate active airway intervention.

32. How is the definitive diagnosis of epiglottitis made?

The gold standard for definitive diagnosis of epiglottitis in **adults** is direct laryngoscopy and visualization of the inflamed or edematous epiglottis. In **children,** the appropriateness of direct visualization is more controversial. Some believe that any attempt at visualizing the inflamed epiglottis should take place in a controlled setting, such as the operating room. Others believe it is appropriate to use a tongue depressor or laryngoscope blade to depress the tongue and visualize the epiglottis of a small child sitting in his or her parent's lap. In either case, visualization should take place only by someone experienced in the management of pediatric airways.

OTITIS EXTERNA

33. How does otitis externa present?

The classic finding is pain with manipulation of the external ear. Cardinal symptoms are itching, pain, and tenderness to palpation. Common signs are erythema and edema of the auditory canal, with crusting, pus, or weeping secretions. Predisposing factors for otitis

externa, also called swimmer's ear, are excessive moisture in the ear canal and trauma (typically from overzealous cleaning).

34. What bacteria are usually responsible?

Pseudomonas aeruginosa and *Staphylococcus aureus*.

35. How is it treated?

The goals for treatment are twofold: to avoid precipitants and to eradicate infection. To treat infection, 2% acetic acid (for drying) combined with hydrocortisone (for inflammation) should be placed on a wick in the ear canal. Alternatively, topical antibiotic drops can be used. Cortisporin otic suspension works well because it has antibacterial, anti-inflammatory, and drying properties, as well as a nontoxic pH.

Additionally, unlike Cortisporin solution, **Cortisporin suspension** can be used in the presence of a perforated tympanic membrane. If the external ear canal is extremely inflamed and narrowed, a wick can be placed to ensure drainage and instillation of medication. If otitis media coexists, be sure to add systemic antibiotics.

36. What is malignant otitis externa?

Malignant otitis externa is a potentially lethal extension of infection of the external ear canal into the mastoid or temporal bone. It is caused most commonly by *P. aeruginosa* and occurs in patients with diabetes or other immunocompromised states. The mortality rate approaches 50%. Malignant otitis externa should be considered when, despite adequate treatment, headache and otalgia persist. CT or magnetic resonance imaging (MRI) confirms the diagnosis. Treatment includes admission, intravenous antipseudomonal antibiotics, and potentially surgical debridement.

PERITONSILLAR ABSCESS

37. State the typical signs and symptoms seen with peritonsillar abscess (quinsy).

- **Symptoms**: Fever, unilateral sore throat, odynophagia, trismus, and occasionally referred otalgia. Patients typically have had pharyngitis for some time with recent antibiotic treatment. Smokers, males, and those with periodontal disease are at increased risk.
- **Signs**: Limited opening of the mouth (usually cannot open more than 2.5 cm), drooling, speaking in a muffled *hot potato* voice, and rancid breath. Examining the oropharynx shows erythema with a deeper redness over the affected area. There is tense swelling of the anterior pillar and soft palate. Subsequently, the tonsil is pushed downward and toward the midline. The uvula may be in an abnormal position, either shifted away from or lying flat against the affected side.

38. What are the treatment options for a peritonsillar abscess?

Needle aspiration followed by antibiotics is the treatment of choice, successful in 85% to 95% of patients. The patient should be seated with his or her head resting against the bed or dental chair headrest. Visualize the tonsils with the aid of a tongue depressor or laryngoscope (a laryngoscope neatly provides its own light source). Topical anesthetic should be applied using lidocaine or Cetacaine. A needle cover should be cut to provide a needle guard for an 18-gauge needle, exposing no more than 1.0 cm of the needle. The guarded needle is inserted at the most fluctuant portion of the abscess. If available, ultrasound utilizing an endocavitary probe can help identify location of the abscess during drainage. The physician should not penetrate deeper than 1 cm and stay **medial** to avoid the more lateral positioned carotid artery. A positive aspiration is achieved if 1 mL or more of pus is obtained. If needle aspiration fails, referral to an ENT physician is necessary for surgical incision and drainage versus tonsillectomy.

39. Describe the presentation of a retropharyngeal abscess.
Common presenting symptoms of retropharyngeal abscess include fever, odynophagia, and neck pain out of proportion to oropharyngeal findings. Patients are ill appearing and may hold the neck in slight extension. Patients may also resist neck movement, mimicking meningitis.

40. Why is this diagnosis so concerning?
The retropharyngeal space of the neck involves three fascial layers between the paraspinal muscles and the pharynx. Infections and abscesses located here have the potential to cause airway compromise and offer a path of direct extension into the mediastinum.

KEY POINTS: OTHER HEAD AND NECK SOFT-TISSUE INFECTIONS

1. In the patient with respiratory compromise and suspected epiglottitis, evaluation is best performed in a controlled environment, with someone skilled at performing an emergent nonsurgical and surgical airway.
2. Malignant otitis externa is caused most commonly by *P. aeruginosa* and occurs in patients with diabetes and immunocompromised states. The mortality rate can be greater than 50%.
3. Infections and abscesses in the retropharyngeal space can lead to airway compromise and direct extension into the mediastinum.

41. What organisms are found in retropharyngeal and peritonsillar abscesses?
Retropharyngeal and peritonsillar abscesses have similar microbial flora: anaerobes, group A streptococci (*Streptococcus pyogenes*), *S. aureus*, and *H. influenzae*.

42. How is a retropharyngeal abscess diagnosed and treated?
It is sometimes visible on a soft-tissue lateral neck radiograph as an increase in soft-tissue density best seen with the neck in slight extension. Definitive diagnosis is made by CT scan. Advanced airway management equipment should be at the bedside while an emergent consultation with an ENT physician is obtained. Intravenous antibiotics should be started, but as with pus formation anywhere in the body, definitive treatment is incision and drainage. The patient should be admitted to the ICU or taken directly to the operating room by the appropriate service. Mediastinal involvement mandates the involvement of a cardiothoracic surgeon.

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CHAPTER 22

DENTAL AND ORAL SURGICAL EMERGENCIES

Richard D. Zallen, DDS, MD, and Valerie N. Byrnside, DDS

1. When should an emergent dental or oral surgical consultation be obtained?

- Facial swelling associated with tooth pain
- Avulsed tooth
- Alveolar housing fractures
- Refractory bleeding from tooth extraction
- Facial fractures

2. How are teeth numbered?

In adults, teeth are numbered starting from the upper right third molar (number 1) and continuing around the teeth to the upper left third molar (number 16). From here the numbering continues when you drop down to the lower left third molar (number 17) and continues around to the lower right third molar (number 32). (See Table 22-1.)

In children, the 20 deciduous teeth (baby teeth) are lettered starting on the upper right second molar (A) and continuing around the teeth to the upper left second molar (J). From here the lettering continues on the lower left second molar (K) and continues around to the lower right second molar (T). (See Table 22-2.)

Children between the ages of 6 and 13 are in a mixed dentition stage with some adult and some deciduous teeth. Their teeth are numbered and lettered as the previous descriptions.

3. Describe the different types of tooth fractures. Which require treatment?

The two basic types are fractures of the crown and fractures of the root. Ellis classified tooth fractures for anterior teeth as I, II, III, and IV (Fig. 22-1). An Ellis class I fracture involves the enamel only and does not require emergent treatment. An Ellis class II fracture involves the enamel and dentin. An Ellis class III fracture involves the enamel, dentin, and pulp. Ellis class II and III fractures require placement of calcium hydroxide (Dycal). An Ellis class IV fracture involves the root of the tooth and may require extraction, root canal therapy, splinting with dental resin, ligature wire, or Erich arch bars depending on the level of fracture.

4. How should an avulsed tooth be transported?

The best transport medium is the socket from which the tooth came if the tooth can be gently rinsed off and replaced. If this is not possible, Hank's balanced salt solution (EMT Tooth Saver) is the next best transport medium. If no transport solution is available, milk can be used or a wet handkerchief. The patient's (or caregiver) saliva or saline may also be used, but these can be damaging to the periodontal ligament that is adherent to the root surface.

TABLE 22-1. UNIVERSAL NUMBERING SYSTEM FOR PERMANENT TEETH

Maxillary right quadrant								Maxillary left quadrant							
1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
Third molar	Second molar	First molar	Second premolar	First premolar	Cuspid	Lateral incisor	Central incisor	Central incisor	Lateral incisor	Cuspid	First premolar	Second premolar	First molar	Second molar	Third molar
32	31	30	29	28	27	26	25	24	23	22	21	20	19	18	17
Third molar	Second molar	First molar	Second premolar	First premolar	Cuspid	Lateral incisor	Central incisor	Central incisor	Lateral incisor	Cuspid	First premolar	Second premolar	First molar	Second molar	Third molar
Mandibular right quadrant								Mandibular left quadrant							

TABLE 22-2. UNIVERSAL LETTERING SYSTEM FOR PRIMARY TEETH

Maxillary right quadrant					Maxillary left quadrant				
A	B	C	D	E	F	G	H	I	J
Second molar	First molar	Cuspid	Lateral incisor	Central incisor	Central incisor	Lateral incisor	Cuspid	First molar	Second molar
T	S	R	Q	P	O	N	M	L	k
Second molar	First molar	Cuspid	Lateral incisor	Central incisor	Central incisor	Lateral incisor	Cuspid	First molar	Second molar
Mandibular right quadrant					Mandibular left quadrant				

KEY POINTS: PROCESS USED TO PREPARE AND APPLY CALCIUM HYDROXIDE PASTE (DYCAL)

1. Isolate and dry tooth with gauze.
2. Dispense equal volume of base and catalyst paste onto a mixing pad.
3. Stir immediately using an applicator (the wooden end of a cotton-tipped applicator will suffice) for 10 seconds until a uniform mixture occurs.
4. Quickly apply the mixed material onto the tooth and cover any exposed dentin or pulp.
5. Wait. The mixed material will set in about 2 minutes.
6. Remove excess paste with a sharp instrument. Be sure to check bite.

5. When should an avulsed tooth be replanted? How is it stabilized?
An avulsed tooth should be replanted within 1 to 2 hours of injury. Teeth should not be replanted if more than 2 hours have passed since avulsion. Stabilization of replanted teeth is indicated provided that there is not extensive caries, periodontal disease, or large alveolar housing fractures. Deciduous teeth should not be replanted. Teeth are stabilized by physiologic splinting with dental composite and ligature wire for 7 to 10 days.

6. What are alveolar housing fractures? How are they treated?
Fractures of the alveolar ridge encompassing the dentition of the maxilla or mandible. The usual treatment involves the manual reduction of the fracture and rigid splint fixation with an Erich arch bar for 4 to 6 weeks.

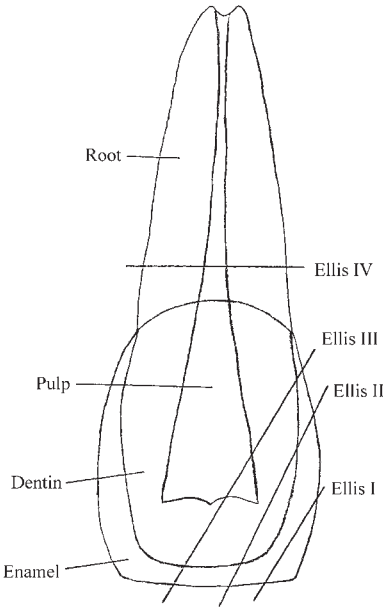

Figure 22-1. Ellis classification of tooth fractures.

7. Should antibiotics be prescribed for an alveolar housing fracture or reimplanted tooth?
Yes. A 5-day course of penicillin is recommended. The oral dose of penicillin V is 500 mg four times a day for adults and 25 to 50 mg/kg/day in four divided doses for children. In penicillin-allergic patients, clindamycin is preferred. The oral dose of clindamycin is 300 mg four times a day for adults and 10 to 20 mg/kg/day in four divided doses for children.

8. What are the concerns with electrical and thermal burns to the mouth?
- **Electrical burns** are deceptive. The ultimate extent of tissue damage is greater than is present on initial examination, and the full extent of the injury may not be appreciated for 4 to 7 days. Wound contracture may produce microstomia. Close observation is warranted due to the possibility of delayed arterial hemorrhage.

- **Thermal burns** are typically treated with antibiotic ointment and topical steroids. Both electrical and thermal burn contractures may need to be treated with skin grafts and splinting.

9. How should a tongue laceration with profuse bleeding be treated?

Initially, packing with gauze and applying pressure should allow visualization of the source of bleeding. Securing the airway may be necessary with some tongue lacerations due to expanding hematoma and possible compromise of the airway. A silk traction suture placed into the tip of the tongue can aid in visualization of the laceration. Injection of a local anesthetic with 1:100,000 epinephrine aids with vasoconstriction. Clamping, ligating, or electrocautery helps to control the larger bleeders when the site can be identified clearly. If minor bleeding persists, the laceration should be closed in a layered fashion using resorbable sutures for deep approximation and Vicryl or Dexon for surface approximation.

10. How should a through-and-through lip laceration be closed?

Initial debridement of the wound may require surgical and saline debridement. Wound exploration and possible soft-tissue radiographs to rule out foreign body (e.g., tooth fragments, metal, etc.) should be considered. Mucosal preparation with hexachlorophene (pHisoHex) is recommended. The mucosa is closed with 3-0 plain gut suture. Skin preparation with chlorhexidine following mucosal closure is recommended. A layered closure of the lip, using 4-0 Vicryl suture for deep tissue/muscle approximation and 5-0 or 6-0 nylon for skin, should be used. If the laceration involves the vermillion border, this should be approximated first because any misalignment is extremely noticeable. Care should also be taken to align the orbicularis oris muscle to avoid any deformity. Prophylactic antibiotic coverage with penicillin for 5 days is recommended.

11. How should human or animal bites to the mouth be treated?

Human bites are managed best with copious irrigation, surgical debridement, tetanus prophylaxis, and prophylactic antibiotics. The drug of choice is amoxicillin with clavulanic acid. Wounds should be closed primarily if possible, although delayed primary closure is an option in some cases. Animal bites are handled in a similar way. Antibiotics are recommended and may vary depending on species. Patients bitten by animals with suspected rabies must be treated aggressively, including irrigation with chlorhexidine solution followed by copious saline irrigation and rabies postexposure prophylaxis.

12. When should antibiotics be used in management of dental infections?

An acute dentoalveolar abscess usually requires antibiotic therapy, with penicillin being the drug of choice. Adjunctive therapy should include root canal treatment or extraction of the offending tooth with incision and drainage. The patient should be followed closely, usually within 24 hours.

13. List some nonodontogenic sources of orofacial pain.

- Temporomandibular joint
- Muscles of mastication
- Salivary glands
- Nose and paranasal sinuses
- Blood vessels (arteritis)
- Nerves
- Oral ulcers
- Cardiac ischemia can occasionally present with jaw pain

14. When should a patient with a dental abscess be admitted to the hospital?

Admission criteria should be based on history and physical findings: size and location of swelling, rapidity of onset, dysphagia, dyspnea, fever, malaise, trismus, age, state of hydration, laboratory evaluation, and immune status of the patient. Urgent admission for

airway concerns, as well as intravenous (IV) antibiotics and fluid resuscitation, may be necessary.

15. Name the risks of dental local anesthesia.

Local anesthetic toxicity including seizures, allergy, syncope, trismus, needle tract infection, intra-arterial or IV injection, paresthesia, hematoma, and transient Bell's palsy from accidental injection into the area of the parotid gland affecting cranial nerve VII. Broken needles rarely occur.

16. What is the best way to perform local dental anesthesia?

Prior to all dental injections, the injection site should be cleansed with gauze and topical anesthetic applied if desired. The most predictable way to provide anesthesia to the maxilla is to infiltrate the buccal and palatal (painful injection) mucosa above the offending tooth with a 27-gauge short or long needle.

For the mandible, an inferior alveolar nerve block is the best way to provide anesthesia for lower teeth on the affected side along with infiltration. To perform an inferior alveolar nerve block, a 25- or 27-gauge long needle with an aspirating syringe is needed. Using the nondominant hand, grasp the anterior mandible with your thumb intraorally near the ascending ramus at the level of the teeth. Aim the needle at the external auditory canal while inserting the needle in mucosa about 0.5 to 1 cm above the plane of teeth (bisecting your thumbnail) while approaching from the opposite mandibular premolars. The needle tip should enter the mucosa at the fold between the pharynx and buccal mucosa (pterygomandibular raphe). The needle tip should be advanced approximately 1.5 to 2 cm until the medial side of the mandible is felt, and then the needle is withdrawn a few millimeters. After aspirating, approximately 1.8 mL of local anesthetic should have a therapeutic effect.

17. What is acute necrotizing ulcerative gingivitis (ANUG)? How is it treated?

ANUG is an acute infection of the gingiva that can be precipitated by psychological stress, smoking, and poor oral hygiene. ANUG typically presents with blunted interdental papilla, which represents areas of necrosis, gingival bleeding, pain, fetor oris, gingival swelling, and lymphadenopathy. ANUG responds well to local debridement and irrigation. Oral rinses with chlorhexidine are necessary. Antibiotics should be used only in refractory cases, and penicillin is the drug of choice.

18. Why is a lateral pharyngeal abscess of great concern?

This infection is potentially life threatening because of airway obstruction and requires urgent incision and drainage. This abscess occurs between the pharyngeal mucosa and the superior constrictor muscle. Presenting symptoms usually include dysphagia, pain, trismus, and fever. Medial bulging of the lateral pharyngeal wall frequently occurs, causing displacement of the uvula to the opposite side. This complication is usually secondary to mandibular third molar extractions and/or needle tract infections.

19. What is Ludwig angina?

An emergent infection of the submandibular, sublingual, and submental spaces bilaterally; if untreated, airway compromise is inevitable. A dental cause is present in 90% of cases. Treatment consists of maintaining the airway, removal of the offending tooth with incision and drainage, antibiotics and IV hydration.

20. How are aphthous ulcers and herpetic lesions differentiated in the oral cavity?

Recurrent aphthous ulcers, also known as *canker sores*, occur as a single circular ulcer and are usually less than 1 cm in diameter. The lesion has a central yellow area surrounded by a prominent band of erythema. Herpetic lesions usually present as clusters of small vesicles that eventually coalesce. Recurrent aphthous ulcers may occur anywhere in the oral cavity except the lips, hard palate, and attached gingiva. Recurrent herpes occurs exclusively in the lips, hard palate, and attached gingiva. Both of these types of lesions can be quite painful.

21. How are oral cavity ulcers treated?

Recurrent aphthous ulcers are treated many different ways, including topical corticosteroids, antibiotics, and anesthetic mouth rinses. An attapulgite (Kaopectate), diphenhydramine (Benadryl), and lidocaine (Xylocaine)—**KBX**—suspension has been shown to provide relief in cases of multiple recurrent aphthous ulcers. The treatment of herpes simplex virus is aimed at palliation of pain. Topical acyclovir, when used during the prodromal stage, has been shown to decrease size of lesions and duration of symptoms. Children may become dehydrated and require admission.

22. How is postextraction hemorrhage evaluated and treated?

The patient's past medical history and current medications should be thoroughly reviewed. Clinical inspection must include good lighting and suction to evaluate the alveolus for a bleeding source. Application of a gauze dressing maintained with firm digital pressure over the extraction site stops most bleeding episodes.

Some hemostatic agents such as gelatin sponges (Gelfoam), absorbable knitted fabric (Surgicel), and topical thrombin may also be useful. Injecting the area with a local anesthetic containing a vasoconstrictor can also be effective. A carefully placed suture aids hemostasis. Refractory bleeding should be evaluated further with appropriate laboratory studies.

23. What is the classification of mandibular fractures?

The best clinical classification is by anatomic region: symphysis, parasymphysis, body, angle, ramus, condyle, and alveolar housing. These fractures may be further described by the specific type of fracture: simple, compound, comminuted, multiple, greenstick, or pathologic. (See Fig. 22-2.)

24. List different ways to radiologically examine a patient for a mandible fracture.

Panoramic radiograph, computed tomography, mandible series (i.e., Towne's views, posteroanterior, and lateral oblique right and left), dental periapical radiographs.

25. How do you clinically examine a patient for a mandibular fracture?

The main diagnostic criteria are a history of trauma, abnormal mandibular movements elicited by bimanual palpation, step deformities or changes in the occlusion, loose teeth, and soft-tissue trauma including laceration or hematoma.

26. Describe a lasso ligature.

A 24-, 25-, or 26-gauge wire that is placed around one or two teeth adjacent to a fracture to approximate fragments of a mandible fracture. The wire is tightened as the patient's occlusion is maintained to bring the fracture into closer alignment. This helps to relieve pain, stop bleeding, and prevent the continued contamination of saliva into the fracture site.

27. Are antibiotics indicated for a mandibular fracture?

Antibiotics are indicated in all open mandibular fractures and all fractures involving teeth are considered open fractures. Fractures of the subcondylar/condylar region that do not communicate with the external auditory canal are closed and are not treated with antibiotics.

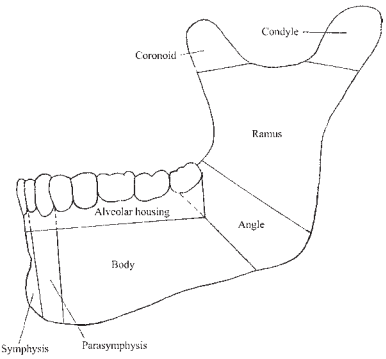

Figure 22-2. Clinical classification of mandibular fractures by anatomic region.

28. List the immediate clinical problems associated with a fractured mandible.

- Airway compromise
- Bleeding
- Pain
- Fracture displacement
- Displaced or aspirated teeth
- Lacerations
- Trismus
- Subcutaneous emphysema

29. What is a mandible contrecoup fracture?

A fracture distant from the site of trauma. A classic example is trauma to the symphysis or parasymphysis area with unilateral or bilateral subcondylar fractures.

30. Describe the treatment of dry socket (alveolar osteitis).

Dry socket is treated by gently irrigating the extraction site and placing a sedative dressing (BIPS and gauze or Alvogyl) into the socket. Local anesthetic may be necessary for pain relief during application of the dressing. Follow-up is needed to ensure pain relief and absence of infection. Multiple treatments are sometimes necessary.

KEY POINTS: COMMON CHARACTERISTICS OF DRY SOCKET (ALVEOLAR OSTEITIS)

1. Extraction 2 to 3 days prior to onset of pain
2. No purulence
3. No fever
4. No clot in extraction site (exposed bone)
5. No trismus

31. What oral complication is being seen with the use of bisphosphonates in the treatment of malignancy?

Bisphosphonate-related osteonecrosis of the jaw (BRONJ) is associated with IV bisphosphonate or oral bisphosphonate usage, usually following dentoalveolar surgery or in areas of bony protuberances and presents as an area of necrotic bone with or without pain or infection. It is treated with chlorhexidine 0.12% mouth rinses, analgesics, antibiotics, hyperbaric oxygen therapy, and surgery, depending on the severity.

KEY POINTS: BRONJ

1. Area of necrotic bone present for more than 8 weeks.
2. May or may not have pain or infection.
3. Occurs in the mandible 2:1 over maxilla.
4. Use of bisphosphonates for treatment of cancer-related conditions or osteoporosis.

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TRANSIENT ISCHEMIC ATTACK AND CEREBROVASCULAR ACCIDENT

Michael M. Liao, MD

1. Define cerebrovascular accident (stroke)?

Stroke is any vascular injury that impairs or blocks cerebral blood flow to a specific region of the brain, resulting in ischemic injury to brain cells. Stroke is the third most common cause of mortality in the United States and the leading cause of adult disability.

2. What are the major types of acute stroke?

The two major types are ischemic and hemorrhagic. The critical diagnostic study to differentiate between the two is a noncontrast head computed tomography (CT).

3. What are the causes of ischemic stroke?

Ischemic stroke causes 80% of all strokes.

- **Thrombotic**: atherosclerosis, vasculitis, lacunar
- **Embolic**: atrial fibrillation, mechanical heart valve, low cardiac ejection fraction, endocarditis, atrial septal defects, cervical artery dissection (i.e., carotid or vertebral arteries)

4. What are the causes of hemorrhagic stroke?

Hemorrhagic stroke causes 20% of all strokes. These can be either intracerebral or subarachnoid hemorrhage.

5. What are the potential mimics of acute stroke?

Postictal Todd's palsy, hypoglycemia, complex migraine, conversion disorder, Bell's palsy, acute spinal cord compression, brain tumor, systemic infection, and multiple sclerosis.

6. Define transient ischemic attack (TIA)?

The classic definition of TIA has been time-based (i.e., $<$24 hours of symptoms), yet most TIAs will resolve within 1 hour. However, up to 67% of classic TIAs will have evidence of acute ischemic lesions on diffusion-weighted magnetic resonance imaging (MRI). Because no time cutoff can reliably determine if underlying ischemic infarction has occurred, in 2009 the American Heart Association and American Stroke Association (AHA/ASA) transitioned to a tissue-based definition of TIA (i.e., transient symptoms with lack of tissue injury confirmed by neuroimaging).

7. Why be concerned about a TIA?

Just as acute coronary syndrome can be a harbinger of myocardial infarction, TIAs are associated with a high risk of early acute stroke (up to 10% within the first 2 days). The $ABCD^2$ score may be useful in predicting 2-day stroke risk (Table 23-1). Evidence suggests that rapid evaluation and initiation of preventative measures within 24 hours may significantly reduce the risk of recurrent stroke.

8. How do I differentiate between TIA and stroke?

If a TIA or stroke presents acutely, it may be impossible to differentiate between them without MRI. Both should be emergently managed as a possible acute stroke.

TABLE 23–1. ABCD² SCORE

• **Age** ≥60	1 point
• **BP**: Initial SBP ≥140 or DBP ≥90	1 point
• **Clinical features**:	
Unilateral weakness	2 points, or
Speech impairment without weakness	1 point, or
Other	0 points
• **Duration of TIA**:	
≥60 min	2 points, or
10–59 min	1 point, or
<10 min	0 points
• **Diabetes**	1 point
2-day stroke risk:	
• High: total 6–7 pts (8.1% risk)	
• Mod: total 4–5 pts (4.1%)	
• Low: total 0–3 pts (1.0%)	

BP, blood pressure; DBP, diastolic blood pressure; SBP, systolic blood pressure; TIA, transient ischemic attack.

9. How do I approach a patient with acute stroke symptoms?

Initial triage should be emergent. As with all acute patients, airway, breathing, and circulation (ABCs), intravenous (IV) access, oxygen, and monitoring are the critical first steps. Early management must also include a fingerstick glucose test, noncontrast head CT, electrocardiogram (ECG), and immediate notification of a stroke team, if available. History is critical to stroke assessment and must include time of onset, evidence of preceding seizure, anticoagulation use, and potential associated trauma. A complete neurologic examination is essential. Abnormal blood pressures (BP) are important to recognize, but management will depend on if the stroke is hemorrhagic or ischemic.

10. How do I determine the onset time for an acute stroke?

Symptom onset is critical to determine eligibility for thrombolytic therapy and must be documented for every patient. Unless the symptom onset is clearly witnessed or known by the patient, the time when the patient was last seen to be normal is used. If the patient awakens from sleep with stroke-like symptoms, then the last time the patient was awake and normal is considered the time of onset.

11. When should I consider extracranial arterial dissection as a cause of acute stroke?

Dissection of the extracranial carotid and vertebral arteries (also called cervical arteries) is an important etiology of acute ischemic stroke. Injury to these vessels can cause stroke from either thrombus embolization or vessel occlusion. Consider this etiology in those with neck trauma (including even minor trauma); cervical spine fractures; young patients (<45 years old); and those with neck pain.

12. What is a primary stroke center?

In 2003, The Joint Commission launched a Primary Stroke Center Certification Program in collaboration with AHA/ASA. To obtain Primary Stroke Center Certification, a hospital must address 11 major aspects of acute stroke care, including acute stroke teams, written care protocols, and multidisciplinary integration.

13. Who is on the stroke team?

The optimal management of stroke requires a multidisciplinary collaboration among the emergency medical service (EMS), ED, neurology, neurosurgery, neuroradiology, interventional neuroradiology (if available), laboratory services, and critical care. In a primary stroke center, an acute stroke team, consisting of at least two healthcare providers experienced in diagnosing and treating acute stroke, is on call 24/7 to evaluate a patient within 15 minutes.

14. What role do emergency medical technicians (EMTs) play?

Besides acute stabilization, EMTs are tasked with early recognition of potential acute stroke and the rapid communication of these findings with the receiving hospital. This allows early activation of the acute stroke team and preparation of CT/MRI, which can save precious minutes in the early evaluation of acute stroke.

15. When do I activate the acute stroke team?

As soon as you suspect the patient of having an acute stroke within the window period for therapy, you should activate the acute stroke team immediately.

16. What is the window for systemic thrombolytics?

The publication of European Cooperative Acute Stroke Study (ECASS) III in 2008 has extended the potential window for thrombolytics from 3 hours to 4.5 hours.

17. What is the evidence for tissue plasminogen activator (tPA) in acute ischemic stroke?

Alteplase (also called tPA) is the only thrombolytic currently approved by the Food and Drug Administration (FDA) for acute stroke. In 1995, the National Institute of Neurological Disorders and Stroke **(NINDS) trial** showed that tPA improved functional outcome at 3 months (i.e., modified Rankin scale $\leqslant$1) if given within 3 hours of symptom onset, with a number needed to treat (NNT) 6 and number needed to harm (NNH) 17. In 2008, **ECASS III** showed similarly improved functional outcome within the 3- to 4.5-hour timeframe (NNT 14, NNH 47; NNH 23 with NINDS definition). NNH represents symptomatic intracerebral hemorrhage, and NNT represents modified Rankin scale $\leqslant$1 at 3 months.

18. What is the risk of tPA?

The primary risk of tPA is systemic bleeding, particularly intracerebral hemorrhage (ICH). Angioedema may occur in 1% to 5% of patients. For the **NINDS trial**, ICH with tPA was 6.4% versus 0.6% for the nontreatment group. For **ECASS III**, ICH with tPA was 2.4% versus 0.2% (7.9% versus 3.5% if original NINDS definitions were used). The factors that appear associated with increased risk of hemorrhage are older age, brain edema or mass effect on CT, and higher baseline stroke severity.

19. What is the importance of the National Institutes of Health stroke scale (NIHSS)?

The NIHSS is the most commonly used objective measure of acute stroke severity. It ranges from 0 to 42, involves 13 questions, and requires the use of standardized pictures, sentences, and words. A booklet and standardized template are free online at *www.strokecenter.org/trials/scales/nihss.html*.

20. What are the indications and contraindications for tPA?

Note that the criteria for ECASS III are more restrictive than for NINDS (Table 23-2). In addition, AHA/ASA guidelines have been modified slightly from original trial criteria, thus established institutional protocols should be developed.

KEY POINTS: SUCCESSFUL ADMINISTRATION OF TPA

1. Adhere to stroke alert protocol.
2. Be exact about the time of onset; document it.
3. Immediately consult with neurologist.
4. Expedite the head CT and final radiology reading.
5. Send labs early (i.e., fingerstick glucose, complete blood count [CBC], PT/PTT, chemistry, and troponin).
6. Mix the tPA early; calculate your dosage (0.9 mg/kg actual body weight; max 90 mg).
7. Double-check all inclusion and exclusion criteria.
8. Obtain informed consent for tPA.

21. Why is there controversy with tPA for acute ischemic stroke?

There remains controversy partly stemming from the potential for serious harm (i.e., ICH) that can occur with tPA therapy. Critics argue that the original NINDS trial inappropriately distorted the measured benefit by recruiting half of subjects within 0 to 90 minutes from symptom onset because, in typical ED practice, the majority of patients will not fall into this time window. Additionally, there is concern that widespread community usage will have different results, although part of this difference likely results from protocol violations, an argument supporting rigorous adherence to stroke protocol.

22. What are the key points in getting informed consent?

Informed consent requirements will vary depending on institutional protocols. At minimum, the risks, benefits, and alternatives should be explained and confirmed to be understood by the patient or the patient's surrogate decision maker. The alternative is not giving tPA. In community observational studies, the reported range of symptomatic ICH within 3 hours is 3.3% to 7.3%. There are no community data yet for the 3- to 4.5-hour window. Furthermore, improved functional outcome can occur spontaneously (NINDS 26% in placebo arm, ECASS III 45%).

23. What must I do after giving tPA?

Current guidelines recommend admission to the intensive care unit (ICU) for at least 24 hours with frequent neurologic checks. Avoid other antithrombotic agents for 24 hours (i.e., heparin, warfarin, aspirin, ticlopidine, and clopidogrel). Maintain BP below 180/105 for the first 24 hours. Invasive procedures (i.e., venipuncture, catheter placement, and nasogastric tube) should be avoided for 24 hours.

24. How should I manage ICH?

In the setting of tPA, consider new ICH if a sudden neurological decline, new headache, nausea or vomiting, and sudden BP rise within 24 hours. If suspected, immediately stop the tPA, perform stat head CT, and send labs (i.e., type and cross-match, prothrombin time [PT], partial thromboplastin time [PTT], platelets, fibrinogen). If ICH is confirmed, consider giving:

- 10 units cryoprecipitate
- 6 to 8 units of platelets (or one single donor unit)

- Neurosurgical consult for possible evacuation
- Additionally, for all ICH consider the following general steps using the mnemonic **RAMP It** up:
  - **R**everse any anticoagulants or antiplatelet agents.
  - **A**ntiepileptics
  - **M**ean arterial pressure (MAP) reduced to 110 to 130 or 15%
  - **P**osition head of bed to 30 degrees.
  - **I**ntracranial pressure (ICP) control (aggressive measures)
    a. Mannitol IV 1 g/kg (then 0.25–0.5 g/kg every 6 hours)
    b. Barbiturate coma
    c. Hyperventilate ($PaCO_2$ 30–35 mm Hg)
    d. Neuromuscular blockage
    e. Ventriculostomy

TABLE 23-2. INCLUSION AND EXCLUSION CRITERIA FOR TISSUE PLASMINOGEN ACTIVATOR

NINDS inclusion criteria:	NINDS exclusion criteria:	ECASS III additional exclusion criteria (3- to 4.5-hour time window):
• No age cutoff • Objective evidence of neurologic deficit on NIHSS (scale 0–42) • Symptom onset <3 hours (if unknown timing, last seen normal time used; if still unclear, exclude)	• Stroke or serious head trauma <3 months • Major surgery <14 days • Any current or history of ICH • SBP >185 or DBP >110 (see Question 28) • Rapidly improving or minor symptoms of stroke • Symptoms suggestive of SAH • GI or GU hemorrhage <21 days • Arterial puncture at noncompressible site <7 days • Seizure at onset of stroke • If on anticoagulation prior 48 hours, PT >15 seconds (or INR >1.7) • If on heparin prior 48 hours, PTT above normal range • Platelets <100,000 mm^3 • Blood glucose <50 mg/dL and >400 mg/dL	(Note MUST meet NINDS criteria as well) • Age <18 or >80 years old • Severe stroke, defined as NIHSS >25 or imaging >⅓ of MCA territory • Combination of previous stroke and diabetes • Oral anticoagulation therapy (warfarin) • Major surgery or severe trauma <3 months • Other major disorders associated with an increased risk of bleeding

DBP, diastolic blood pressure; GI, gastrointestinal; GU, genitourinary; ICH, intracranial hemorrhage; INR, international normalized ratio; MCA, middle cerebral artery; NIHSS, National Institutes of Health stroke scale; NINDS, National Institute of Neurological Disorders and Stroke; PT, prothrombin time; PTT, partial thromboplastin time; SAH, subarachnoid hemorrhage; SBP, systolic blood pressure.

KEY POINTS: MANAGEMENT OF ICH

1. Reverse any anticoagulants or antiplatelet agents.
2. Antiepileptics.
3. MAP control.
4. Position head of bed.
5. ICP control.

25. What about recombinant factor VIIa (rF7a)?

rF7a has shown promise for rapid hemostasis in ICH; however, it is associated with an increased risk of arterial thromboembolic events (i.e., myocardial infarction [MI] or ischemic stroke), especially at high dosages. Furthermore, a recent phase 3 clinical trial did not show improvement in 3-month death or severe disability. As such, it remains experimental at this time.

26. What medications should be started in the ED for acute stroke?

This depends on what type of stroke you're dealing with and if the patient is a candidate for tPA. TPA protocol requires no anticoagulation or antiplatelet therapy within 24 hours of tPA administration. For hemorrhagic stroke, **see Question 24.** For ischemic stroke not receiving tPA or anticoagulation, early aspirin therapy (325 mg PO) is recommended in the ED, unless contraindicated. Certain patients with acute ischemic stroke may instead benefit from anticoagulation.

27. What are the indications for heparin?

There is currently no evidence evaluating early initiation of heparin or low-molecular-weight heparin in the ED for acute ischemic stroke. Early initiation must be carefully weighed against the risk of bleeding at the stroke site. However, it may be beneficial to start early in patients with cardioembolism from intracardiac thrombus, large artery stenosis with intraluminal thrombus, or cervical or intracranial artery dissection. This decision should ideally be made in collaboration with a neurologist.

28. How do I approach the stroke patient with hypertension?

Before deciding on management, it is critical to distinguish ischemic from hemorrhagic stroke.

- **Ischemic stroke**: Permissive systemic hypertension may be critical to maintaining cerebral perfusion, and aggressive lowering may result in clinical deterioration. Most experts recommend lowering slowly by 15% only if systolic blood pressure (SBP) $\geq$220 mm Hg or diastolic blood pressure (DBP0 $\geq$120 mm Hg. The important exception is when **thrombolytic therapy** is being considered. In this setting, one to two dosages of labetalol intravenously 10 to 20 mg over 1 to 2 minutes is allowed if SBP $>$185 mm Hg or DBP $>$110 mm Hg.
- **Hemorrhagic stroke**: The benefits of lowering BP are reduced bleeding and vascular damage, but again higher than normal BPs may be necessary to optimize cerebral perfusion. Without ICP monitoring, one should defer to consult recommendations, but a modest reduction of elevated BPs is reasonable to consider in this setting.

29. What is the best way to minimize risk of litigation for tPA?

Patient care and safety should always be your primary concern. However, one must recognize that acute ischemic stroke remains a medicolegal target both for failure to treat with tPA and adverse events associated with tPA therapy. Good medicine is good law. Have a current multidisciplinary evidence-based stroke protocol and strictly adhere to it. Involve stroke team consultants early. When possible, obtain written informed consent.

30. What if I accidentally give tPA to a stroke mimic?

Evidence is limited, so true safety in this setting is unclear. However, some evidence suggests this situation may be both safe and rare.

31. Are there alternatives to tPA for acute ischemic stroke?

Intra-arterial thrombolysis and mechanical clot disruption are promising new alternatives for acute ischemic stroke and may extend the therapeutic window beyond 4.5 hours; however, these therapies remain experimental and are often limited to academic institutions. Intra-arterial thrombolysis after systemic tPA is also a promising alternative for community hospitals in close proximity to academic centers. These alternatives should only be considered in collaboration with or within a primary stroke center with these capabilities.

32. Which ED patients are at high risk for stroke?

In the ED, three groups of patients are potentially identifiable before a stroke occurs.

a. **Acute MI:** Most emboli associated with MI occur soon after MI. Patients with anterior wall MIs are at highest risk. Appropriate intervention with anticoagulation or antiplatelet therapy in the ED may prevent a stroke a few hours later.

b. **Multiple traumatic injuries:** These patients are notorious for having a stroke in the hospital after their "life has been saved." The highest risk of trauma-related stroke is in patients with direct trauma to the carotid or vertebral arteries. The vertebral artery is particularly prone to injury from rapid head motions. Facial fractures have been associated with a higher risk of carotid injury, leading to carotid occlusion, dissection, or artery-to-artery embolism. Recognition of high-risk patients and early angiography allow preventive measures, such as anticoagulation therapy, to be instituted if there are no contraindications.

c. **TIA or recent ischemic stroke:** Recall that TIA has a high risk of acute stroke within 2 days. Prompt institution of antiplatelet therapy is worthwhile in the ED and should not be deferred until the patient is admitted to the hospital floor.

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MENINGITIS

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CHAPTER 24

1. What is meningitis? Why is it important?

Meningitis is an inflammatory disease of the tissues surrounding the brain and spinal cord. Mortality from bacterial and fungal meningitis is 10% to 30%. Prompt recognition and treatment of bacterial meningitis can lessen morbidity and mortality.

2. What are the causes of meningitis?

See Table 24-1.

3. Which organisms are most commonly involved in each age group?

See Table 24-2.

4. Who is at risk for meningitis?

Those older than 60 and younger than 5 are at highest risk. Medical conditions that put patients at risk include: diabetes, alcoholism, cirrhosis, sickle cell disease, immunosuppressed states, history of splenectomy, thalassemia major, bacterial endocarditis, malignancy, history of ventriculoperitoneal shunt, and intravenous drug abuse. Other risks include recent exposure to others with meningitis, crowding, contiguous infection (e.g., sinusitis), and dural defect (e.g., traumatic, surgical, congenital).

5. List the common presenting symptoms of meningitis.

- Fever (most sensitive sign)
- Change in mental status
- Headache
- Photophobia
- Stiff neck
- Lethargy
- Irritability
- Malaise
- Confusion
- Seizures

TABLE 24-1. CAUSES OF MENINGITIS

Infectious causes	Noninfectious causes
Bacteria	Neoplastic
Viruses	Collagen vascular
Fungi	Drugs (i.e., antibiotics and anti-inflammatory medications)
Parasites	
Tuberculosis	

TABLE 24-2. ORGANISMS MOST COMMONLY INVOLVED BY PATIENT GROUP

Age or Condition	Most Commonly Encountered Organisms
Newborns	Group B or D streptococci, nongroup B streptococci, *Escherichia coli*
Infants and children	*Streptococcus pneumoniae, Neisseria meningitides, Haemophilus influenzae*
Adults	*S. pneumoniae, H. influenzae, N. meningitides,* staphylococci, streptococci, *Listeria* spp.
Patients with impaired cellular immunity	*Listeria monocytogenes,* gram-negative bacilli, *S. pneumoniae, N. meningitides*
Head trauma, neurosurgery, or CSF shunt	Staphylococci, gram-negative bacilli, *S. pneumoniae*

CSF, cerebrospinal fluid.

KEY POINTS: CLASSIC CLINICAL TRIAD FOR MENINGITIS

1. The classic clinical triad of fever, neck stiffness, and altered mental status is present in less than two thirds of patients with meningitis.
2. The absence of all three signs of the classic triad virtually eliminates a diagnosis of meningitis.

6. What clinical signs are characteristic of meningeal irritation?

- Nuchal rigidity
- Brudzinski's sign: flexion of the neck results in flexion of the knees and hips
- Kernig's sign: pain or resistance of the hamstrings when the knees are extended with the hips flexed at 90 degrees
- Jolt accentuation: baseline headache increases when the patient turns the head horizontally two to three rotations per second. (This physical finding is found more reliably in meningitis than are the previously mentioned physical findings.)

These findings are often absent in the very young and older patients.

7. List the presenting signs of meningitis in infants.

- Bulging fontanelle (may not be present if patient is dehydrated)
- Paradoxic irritability (quiet when stationary, cries when held)
- High-pitched cry
- Hypotonia
- Skin or spine may have dimples, sinuses, nevi, or tufts of hair indicating a congenital anomaly communicating with the subarachnoid space.

8. If the symptoms are not specific and physical findings are absent, what are the indications for lumbar puncture (LP)?

LP should be done whenever meningitis is suspected because analyzing spinal fluid is the only way to diagnose meningitis.

9. What tests should be done before doing an LP?

a. Funduscopic examination—check for papilledema and spontaneous venous pulsations.
b. Computed tomography (CT) scan—order only if following are present: papilledema, absence of spontaneous venous pulsations, altered mental status, focal neurologic examination, new-onset seizure, or clinical suspicion for recent trauma or subarachnoid bleed.
c. Coagulation studies and platelet count—if suspicion for bleeding disorder.

10. What is the most common error in ED management of meningitis?

Delaying administration of antibiotics until the LP is done. If there is a clinical suspicion of bacterial meningitis, antibiotics should be administered promptly. Intravenous antibiotics given 2 hours or less before the LP (and ideally after blood and urine cultures are obtained) will not affect the results of the cerebrospinal fluid (CSF) analysis.

11. Discuss the risks of LP.

- Paralysis: unlikely (needle inserted below level of spinal cord at L2 or below)
- Transient leg paresthesias during LP: due to irritation of nerve roots by the needle
- Cauda equina syndrome: from hematoma in patients with coagulopathy (rare)
- Headache: most common sequela, seen in 5% to 30% of patients
- Tonsillar herniation: from increased intracranial pressure (no risk with normal CT)

12. What is the secret to performing LP successfully?

Proper positioning of the patient is crucial. If the LP is done with the patient lying down, be sure the shoulders and hips are in a straight plane perpendicular to the floor. The patient should be in the tightest fetal position possible. If the LP is done with the patient sitting up, have the upper body rest on a bedside table and have the patient push his or her back toward you as if he or she is an angry cat.

13. When is it essential to perform the LP with the patient lying down?

This is important when you want to obtain an opening pressure. If you are unable to perform the LP with the patient lying down, you can place the needle with the patient sitting up and then have him or her lay down to obtain the opening pressure.

14. What can cause a falsely elevated intracranial pressure?

A tense patient, the head being elevated above the plane of the needle, marked obesity, or muscle contraction.

15. Which laboratory studies should be ordered on the CSF?

Four tubes are usually collected, each containing 1 to 1.5 mL. More CSF is needed if special tests are required.

- Tube 1: Cell count and differential
- Tube 2: Gram stain, culture, and sensitivities (special tests that may be ordered include viral cultures, tuberculosis cultures and acid-fast stain, fungal antigen studies and India ink stain, and serologic tests for neurosyphilis. Countercurrent immunoelectrophoresis is used occasionally to detect specific bacterial antigens in the CSF.)
- Tube 3: glucose and protein
- Tube 4: cell count and differential

In pediatric patients, three tubes are collected: tube 1 for microbiology, tube 2 for glucose and protein, and tube 3 for cell count and differential.

16. What findings on LP are consistent with bacterial meningitis?

See Table 24-3.

TABLE 24-3. FINDINGS CONSISTENT WITH BACTERIAL MENINGITIS

Parameter	Finding
Opening pressure	In range of 20–50 cmH_2O
Appearance	Cloudy
White blood cell count	1,000–5,000 cells/mm^3
Cells	Neutrophil predominance
Glucose	<40 mg/dL
Ratio of CSF to serum glucose	<0.4
CSF protein	Elevated (often >100 mg/dL)
CSF lactate	>3.5 mmol/L (more useful in postoperative patients than in community-acquired meningitis)

CSF, cerebrospinal fluid.

KEY POINTS: CORRECTIONS FOR TRAUMATIC TAPS

1. CSF from a traumatic LP should contain 1 white blood cell (WBC) per 700 (red blood cells (RBCs).
2. When traumatic LP has occurred, correct CSF protein for the presence of blood by subtracting 1 mg/dL of protein for each 1,000 RBCs.
3. A high CSF protein level associated with a benign clinical presentation should suggest fungal disease.

17. Which antibiotics should be prescribed when the causative organism is unknown?

See Table 24-4.

18. What about steroids?

The rationale behind the use of steroids is that attenuation of the inflammatory response in bacterial meningitis may be effective in decreasing pathophysiologic consequences such as cerebral edema, increased intracranial pressure, and altered cerebral blood flow. The current recommendations are listed here:

- The Infectious Disease Society of America includes dexamethasone in its algorithm for treatment of meningitis both in adults and in infants.
- Use dexamethasone (0.15 mg/kg) in adults with suspected or proven pneumococcal meningitis. Then only continue if CSF Gram stain shows gram-positive diplococci.
- Use dexamethasone (0.15 mg/kg) in children with suspected or proven *Haemophilus influenzae* meningitis.
- Do not give dexamethasone to adult patients who have already received antimicrobial therapy.

TABLE 24-4. RECOMMENDATIONS FOR KNOWN ORGANISMS AND GENERALIZED RECOMMENDATIONS

Organism	Antibiotic Treatment
Neisseria meningitides	Penicillin G, 3–4 million IU IV every 4 hours, or ampicillin, 2 g IV every 4 hours, or third-generation cephalosporin
Streptococcus pneumoniae	Vancomycin plus a third-generation cephalosporin
Haemophilus influenzae	Cefotaxime, 2 g IV every 6 hours or Ceftriaxone 2 g IV every 12 hours or Chloramphenicol 50–100 mg/kg/day in four divided doses
Staphylococcus aureus	Nafcillin 2 g IV every 4 hours
Escherichia coli and other gram-negative enterics except *Pseudomonas aeruginosa*	Cefotaxime, 2 g IV every 4 hours
P. aeruginosa	Ceftazidime, 2 g IV every 8 hours, plus gentamicin, 3–5 mg/kg/day IV in three divided doses
Listeria monocytogenes	Ampicillin, 2 g IV every 4 hours, plus gentamicin (as for *P. aeruginosa*)
Group B streptococci	Penicillin G 4 million units IV every 4 hours or Ampicillin 2 g IV every 4 hours
Generalized (empiric Rx) recommendations	
Age or condition	Antibiotic treatment
Age <3 months	Ampicillin + broad-spectrum cephalosporin
Age 3 months to 50 yr	Vancomycin + broad-spectrum cephalosporin
Age >50 yr	Ampicillin + broad-spectrum cephalosporin + vancomycin
Impaired cellular immunity	Ampicillin + ceftazidime + vancomycin
Head trauma, neurosurgery, CSF shunt	Vancomycin + ceftazidime

CSF, cerebrospinal fluid; IV, intravenously.

19. Do people exposed to a patient with meningitis need antibiotics?

Individuals who have had close contact with someone who has, or is suspected to have, meningococcal meningitis should take rifampin, 600 mg twice a day for 2 days. Other accepted prophylaxis regimens for *Neisseria meningitides* include the following: ciprofloxacin, 500-mg single dose; ceftriaxone, 250-mg intramuscular (IM) dose (used in pregnancy); or a single oral dose of azithromycin, 500 mg. A 4-day course of rifampin is recommended for most individuals who have been in close contact with someone with *H. influenzae* type B meningitis. Individuals exposed to someone with another type of meningitis, especially viral, do not need prophylactic antibiotics.

WEBSITE

Infectious Diseases Society of America: *www.idsociety.org*.

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BREATHING AND VENTILATION

John L. Kendall, MD, and Ryan Paterson, MD

1. How useful is the respiratory rate in the evaluation of a patient?

The respiratory rate is invaluable as a vital sign. Normal respiratory rate in children varies with age, whereas adults typically breathe 12 to 16 times per minute. As a testament to its usefulness, the respiratory rate can be helpful in the diagnosis of many conditions other than those with primary pulmonary pathology. For example, it is elevated in patients with anemia, arteriovenous fistula, pregnancy, cyanotic heart disease, metabolic acidosis, febrile illness, central nervous system pathology, anxiety, and those at high altitude. It is important that the respiratory rate be counted carefully for at least 30 seconds. The respiratory rate is often incorrectly estimated from a short period of observation.

2. Which breathing patterns are associated with pathologic conditions?

- **Kussmaul** respirations are deep, rapid breaths that are associated with metabolic acidosis.
- **Cheyne-Stokes** breathing comprises respirations that wax and wane cyclically so that periods of deep breathing alternate with periods of apnea. Causes include congestive heart failure (CHF), hypertensive crisis, hyponatremia, high altitude illness, and head injury.
- **Ataxic** breathing is characterized by unpredictable irregularity. Breaths may be shallow or deep and may stop for short periods. Causes include respiratory depression and brain stem injury at the level of the medulla.

3. Which pulmonary function tests are commonly used in the ED?

Other than the respiratory rate, the most useful pulmonary function test for ED patients is the peak expiratory flow rate. It is measured by having a patient exhale at a maximum rate through a peak flowmeter. Normal values range from 350 to 600 L/min in adults. Lower levels are characteristic of increased airway resistance as commonly seen in asthma and chronic obstructive pulmonary disease (COPD) exacerbations. Patients who present with values of less than 100 L/min have severe airflow obstruction. Comparing a patient's current peak expiratory flow rate to his or her personal best can provide good insight into the severity of respiratory distress and necessary treatment. Serial measurements are helpful for objectively quantifying response to treatment. A less commonly used test is the forced end-expiratory volume at 1 second, which helps quantify the severity of obstructive and restrictive lung disease.

4. How does pulse oximetry work?

Pulse oximetry is based on a combination of spectrophotometry and plethysmography.

- **Spectrophotometry** is based on the Beer-Lambert law, which holds that optical absorbance is proportional to the concentration of a substance and the thickness of the medium. Using this principle, the absorbance of light within a pulsatile vascular bed is used to distinguish between oxyhemoglobin (O_2Hb) and reduced hemoglobin (Hb).
- **Plethysmography** measures the tissue displacement caused by an arterial pulse. This allows for assessment of the increase in light absorption caused by local arterial flow compared with the background of composite tissues and venous blood. Plethysmography also allows determination of the heart rate.

Pulse oximeters function by placing a pulsatile vascular bed between a light-emitting diode (LED) and a detector. Light is transmitted through the tissue at two wavelengths, 660 nm (primarily absorbed by O_2Hb) and 940 nm (primarily absorbed by Hb), allowing differentiation of O_2Hb from Hb. The detector compares the concentration of O_2Hb and Hb and displays the result as a percent saturation.

5. How can pulse oximetry be useful? In which situations can it yield false readings?

Pulse oximetry is useful when monitoring arterial O_2Hb saturation in cardiopulmonary disorders; monitoring oxygen saturation during conscious sedation, airway management, or in patients with a decreased level of consciousness; or quantifying the arterial O_2Hb saturation response to therapeutic interventions. Situations in which the usefulness of pulse oximetry is limited include vasoconstriction, excessive movement, low O_2Hb saturations ($<$83%), intravascular dyes, exposure of the measuring sensor to ambient light sources, and when nail polish is present. Oxygen saturation measurements may be falsely elevated in the presence of carboxyhemoglobin and falsely decreased in the presence of methemoglobin.

KEY POINTS: PULSE OXIMETRY

1. Pulse oximetry measures oxygenation, not ventilation.
2. Poor peripheral perfusion is a frequent reason pulse oximeters provide unreliable readings.

6. Why can a good pulse oximetry reading be falsely reassuring?

Clinicians often rely on the pulse oximeter as part of monitoring a patient's respiratory status, particularly when using procedural sedation. The pulse oximeter only measures oxygenation and provides no information regarding CO_2 exchange and thus does not assess for adequate ventilation. A preoxygenated patient can be apneic for several minutes without an appreciable decrease in oxygen saturation, while significant hypercarbia is developing. Although the pulse oximeter has become indispensable, the clinician must always remember that it only assesses one part of a patient's respiratory status.

7. What is end-tidal CO_2 ($EtCO_2$) and when is its monitoring useful?

$EtCO_2$ is another monitoring device used to evaluate ventilation, and when combined with the pulse oximeter, it provides a more complete evaluation of the patient's respiratory status. $EtCO_2$ monitors exhaled CO_2, displaying its concentration in both numerical and graphical format. The CO_2 concentration in the breath correlates directly with the concentration of CO_2 in the alveoli. The CO_2 in the alveoli is dependent on the ventilation/perfusion (V/Q) relationship, which is influenced by a number of physiologic and pathologic states. A CO_2 increase or decrease may represent the earliest change in a patient's ventilation and perfusion states. $EtCO_2$ is currently being used in a number of ways:

- During conscious sedation
- In patients with sepsis to monitor perfusion status
- During cardiopulmonary resuscitation (CPR) to monitor effectiveness of compressions
- For monitoring airway response to treatment in patients with COPD and asthma
- To monitor for tube placement or dislodgement by emergency medical services (EMS) during intubation and transport

8. What percentage of fraction of inspired oxygen (FiO_2) corresponds with the various types of oxygen delivery systems?

The three primary means of oxygen delivery are nasal cannula, simple facemask, and facemask with an oxygen reservoir. A nasal cannula can be used to deliver oxygen at rates of

1 to 6 L/min. With a nasal cannula, every 1 L/min of flow increase causes the FiO_2 to rise by 4%. As a result, a nasal cannula can deliver a FiO_2 between 25% and 45%. A simple facemask relies on an oxygen flow of 5 to 10 L/min with a resulting FiO_2 ranging from 35% to 50%. A facemask with an oxygen reservoir has a constant flow of oxygen so that higher concentrations of oxygen can be achieved. A properly fitted facemask with an oxygen reservoir with a 15 L/min flow rate can deliver up to 85% FiO_2.

9. What is noninvasive ventilation?

It is a means of delivering positive-pressure ventilation without placing a nasotracheal or endotracheal tube. As such, ventilatory assistance is possible without the risks of intubation and mechanical ventilation. Careful selection of patients can make noninvasive ventilation a useful tool to the emergency physician. Any patient who does not have an appropriate mental status, is unable to protect the airway, or is unable to develop an adequate respiratory rate would not be a good candidate for noninvasive ventilation.

10. What forms of noninvasive ventilation are available to emergency physicians?

The two most useful forms are mask continuous positive airway pressure (CPAP) ventilation and bilevel positive airway pressure (BiPAP). Each method involves placing a tight-fitting mask over the patient's face and delivering breaths by positive pressure.

- **CPAP** delivers a continuous amount of positive airway pressure during and after inspiration and expiration.
- **BiPAP** not only provides a set positive pressure during exhalation but also delivers a set inspiratory pressure when the patient initiates a breath. The inspiratory pressure is always set higher than the expiratory pressure, can be sustained for various periods, and stops when the patient ceases to inhale or begins to exhale.

11. In what circumstances would noninvasive ventilation be preferred over standard invasive ventilation?

Noninvasive ventilation has been shown to be useful in many conditions, including pulmonary edema, pneumonia, asthma, COPD, and nocturnal hypoventilation. In properly selected patients, CPAP is particularly useful in the treatment of pulmonary edema and BiPAP in respiratory distress due to COPD. Patients with COPD are notoriously difficult to wean from mechanical ventilators, and noninvasive ventilation can frequently be used to turn around COPD patients in moderate respiratory distress who would otherwise have required standard invasive ventilation. Lastly, some patients with advance directives forbidding mechanical ventilation can benefit from the respiratory support provided by noninvasive ventilation.

12. How do I determine the initial ventilator settings in someone who has just been intubated?

Ventilator settings must take into account the patient's oxygenation status and his or her ventilation or acid-base status. The primary method for affecting the oxygenation of a patient is to alter the FiO_2 and positive end-expiratory pressure (PEEP). Initially, intubated patients should be given 100% oxygen or an FiO_2 of 1.00. Subsequently, if arterial blood gas analysis reveals that the PaO_2 is high, the FiO_2 and PEEP may be lowered incrementally.

The main factors determining a patient's ventilatory status are tidal volume and respiratory rate. Changes in each are reflected by the CO_2 from arterial blood gas analysis. High respiratory rates and large tidal volumes decrease the carbon dioxide level, whereas the converse elevates the carbon dioxide level. Initially, the tidal volume can be estimated to be 8 to 10 mL/kg; for a 70-kg patient, that is 560 to 700 mL.

The initial respiratory rate varies depending on the clinical situation. On average, the rate should be set between 10 and 16 breaths per minute.

13. Are ventilator settings always the same?

No. When you intubate a patient you must remember that you have now placed a bet that you can do a better job directing that patient's ventilation than his or her brain. Keep in mind that

your patient's respiratory center has millions of years of evolution backing it up compared with your measly few years of experience. Imagining how your patient's respiratory center would respond to the clinical situation and where the failure has occurred will help you to determine the best ventilator settings for your patient. For example, a patient with an obstructive condition such as asthma does best with small tidal volumes, high respiratory rates, and low levels of PEEP. In contrast, a patient with a COPD exacerbation requires lower respiratory rates, higher tidal volumes, no PEEP, and a prolonged expiratory time. Following $EtCO_2$ and pulse oximetry can provide real-time feedback of the adequacy of your settings. Other common ventilator settings for patients with closed head injury, CHF, metabolic acidosis, and sepsis are shown in Table 25-1.

14. What are the different ventilator modes?

The main modes of ventilation are controlled mechanical ventilation (CMV), assist control (AC), intermittent mandatory ventilation (IMV), and synchronized intermittent mandatory ventilation (SIMV). In the CMV mode, the ventilator delivers a certain volume or pressure at a preset rate, regardless of any ventilatory effort by the patient. AC is similar to CMV in that the tidal volume or inspiratory pressure and minimum respiratory rate are set. It differs from CMV by allowing patients to trigger the ventilator over a set minimum respiratory rate. IMV allows the patient to breathe spontaneously without having a preset tidal volume or pressure. A set rate similar to the CMV mode is in place. This allows the patient to breathe spontaneously, while ensuring a set respiratory rate and tidal volume. SIMV differs from IMV in that the ventilator senses the patient's spontaneous respirations and does not deliver a breath if the patient has already triggered the ventilator. This prevents stacking of respirations, which can be a component of the IMV mode.

15. What is PEEP?

PEEP is pressure applied during expiration. PEEP prevents collapse of alveoli at end-expiration leading to an increase in functional residual capacity. The end result is improved V/Q matching in the pulmonary circulation, improving oxygenation. On the flip-side, PEEP can induce barotrauma, diminish venous return to the heart, and elevate intracranial pressure. PEEP is usually set at 2.5 or 5.0 cm H_2O.

TABLE 25-1. VENTILATOR SETTINGS ACCORDING TO CONDITION

Condition	Tidal Volume (mL/kg)	Respiratory Rate (breaths per minute)	FiO_2	PEEP (cm H_2O)
Asthma	5–10	10–16	100%	2.5–10
COPD	8–12	6–8	100%	None
Head injury	12–15	14–20	100%	None
CHF	8–12	8–12	100%	5–10
Metabolic acidosis	8–12	14–20	100%	2.5–5
Sepsis	6	10–16	100%	2.5–5

CHF, congestive heart failure; COPD, chronic obstructive pulmonary disease; FiO_2, fraction of inspired oxygen; PEEP, positive end-expiratory pressure.

American Heart Association: *Pediatric Advanced Life Support Provider Manual*, Dallas, 2002, American Heart Association, p 109.

KEY POINTS: VENTILATOR MANAGEMENT

1. Each clinical situation calls for a different approach to ventilator management.
2. Tidal volume and respiratory rate affect the patient's ventilation and pCO_2.
3. FiO_2 and PEEP affect the patient's oxygenation and pO_2.
4. Oxygenation and ventilation problems in patients on mechanical ventilators can be managed by removing them from the ventilator and following the DOPE mnemonic.

16. What is auto-PEEP?

Auto-PEEP develops when a positive-pressure breath is delivered before complete exhalation of the previous breath. As a result, air becomes trapped and pressure within the lungs increases. This leads to increased airway pressures, diminished venous return to the right heart, and consequently hypotension. The increased airway pressures can lead to barotrauma, pneumothorax, and inaccurate pulmonary artery catheter measurements. Auto-PEEP can be a particular problem in the mechanical ventilation of COPD and asthmatic patients. The immediate solution is to disconnect the ventilator circuit and allow a full exhalation followed by appropriate changes to the ventilator settings.

17. What are the most common complications of mechanical ventilation?

The most common direct complication seen in the ED is **barotrauma**. High pressure can lead to rupture of the alveolar wall, which can lead to pneumomediastinum, pneumothorax, tension pneumothorax, pneumoperitoneum, and subcutaneous emphysema. Pneumonia leads the list of ventilator complications overall, followed by sinusitis, tracheal necrosis, local trauma to the nares and mouth, increased intracranial pressure, renal failure, hyponatremia, and fluid retention.

18. How do I approach a patient on a ventilator with acutely worsening oxygenation or ventilation?

A systematic approach to this situation will serve you well. The **DOPE** mnemonic taught in pediatric life support can be helpful in remembering the approach:

- First remove the patient from the ventilator and have an assistant hand ventilate the patient. Many problems involving a $30,000 ventilator can be solved with a $15 resuscitation bag.
- **D**isplacement: Confirm the endotracheal tube is in the proper place by using some combination of auscultation, CO_2 exchange, radiography, and direct visualization.
- **O**bstruction: Confirm that the endotracheal tube is patent by passing suction catheter. Sometimes an endotracheal tube can become kinked simply due to patient positioning.
- **P**neumothorax: Confirm that there is no evidence of barotrauma, usually by a combination of physical examination and a chest radiograph.
- **E**quipment: Confirm that the ventilator circuit and ventilator itself are functioning properly.

CHAPTER 26

ASTHMA, CHRONIC OBSTRUCTIVE PULMONARY DISEASE, AND PNEUMONIA

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ASTHMA

1. What is asthma and what are the presenting symptoms of asthma exacerbation?

Asthma is a chronic inflammatory disorder of the airways, resulting in recurrent episodes of wheezing, breathlessness, chest tightness, and coughing. The airway inflammation contributes to airway hyperreactivity, airflow obstruction, and chronic disease.

2. In addition to asthma, what should be included in the differential diagnosis of wheezing?

Chronic obstructive pulmonary disease (COPD), congestive heart failure (CHF), foreign body aspiration, anaphylaxis, epiglottitis, tracheobronchitis, reactive airway disease, viral respiratory infections, and vocal cord dysfunction.

3. Which aspects of the asthmatic's history are important to the current exacerbation?

Ask questions regarding exposure to common precipitants, such as viral upper respiratory tract infections, allergens, cold, exercise, and possible aspirin or nonsteroidal anti-inflammatory drug use. Also important are duration and severity of symptoms, past history and frequency of sudden exacerbations, prior hospitalizations and intubations, number of recent ED visits, current medications, worsening of symptoms while on or if weaning off corticosteroids, and other comorbidities. Non-Caucasian race and lower socioeconomic standing are also risk factors for severity requiring hospitalization.

4. Are there any helpful ancillary diagnostic tests?

Bedside spirometry provides a rapid, objective assessment of patients and serves as a guide to the effectiveness of therapy. The forced expiratory volume in 1 second (FEV_1) and the peak expiratory flow rate (PEFR) directly measure the degree of large airway obstruction.

- FEV_1 or PEFR $\geqslant$70% of predicted (or personal best) indicates **mild** obstruction
- FEV_1 or PEFR 40%–69% of predicted (or personal best) indicates **moderate** obstruction
- FEV_1 or PEFR $<$40% of predicted (or personal best) indicates **severe** obstruction

Pulse oximetry is a useful and convenient method for assessing oxygenation and monitoring oxygen saturation during treatment. Most other tests, including arterial blood gases, complete blood counts, and electrocardiograms, are not useful in the management of asthma except in cases of active or impending respiratory failure. Chest X-ray may be helpful if the patient does not respond to initial treatment or if a pulmonary complication, such as foreign body obstruction, pneumonia, pneumomediastinum, pneumothorax, or CHF, is suspected.

5. What are the key objectives when treating an asthma exacerbation? How are they achieved?

The key objectives include correction of significant hypoxemia, rapid reversal of airflow obstruction, and reduction of the likelihood of recurrence of severe airflow obstruction.

First-line treatment includes β_2-agonists and corticosteroids in moderate exacerbations, and oxygen if needed. Ipratropium should be added when treating severe exacerbations. Relief of airflow obstruction (bronchoconstriction) is usually accomplished by administration of either intermittent or continuous doses of aerosolized β_2-agonists. Studies contain mixed conclusions as to whether there is any added clinical benefit to levalbuterol in comparison to racemic formulations. Current evidence does not suggest an improved benefit from intravenous β_2-agonists compared to aerosol. Early administration of systemic corticosteroids addresses the inflammatory component of acute asthma and has been demonstrated to prevent some hospitalization, although beneficial effects of corticosteroids are often not noted until several hours after administration. High-dose inhaled corticosteroids may have some benefit in the acute setting and can be continued safely by patients already on inhaled steroids. Aerosolized ipratropium should be added if FEV_1 or PEFR is $<$40% of predicted because studies reveal that they increase pulmonary function modestly and decrease need for hospitalization in these patients. Hypoxemia is usually corrected by administration of supplemental oxygen with a goal of oxygen saturation of 90% to 95%. (See Table 26-1.) Epinephrine or terbutaline may be administered subcutaneously to patients unable to coordinate aerosolized treatments. Theophylline is not recommended in the acute setting.

6. How can I determine if my patients are improving?

Ask them how they feel, re-examine them, and obtain objective measures of pulmonary function. Either FEV_1 or PEFR (the best of three attempts) should be obtained on presentation and after treatment and compared with the patient's percent predicted (or personal best) FEV_1 or PEFR, if known, to determine the need for more aggressive therapy or hospitalization.

7. What measures are available if my patient isn't responding as expected?

Magnesium, heliox, ketamine, and continuous positive-pressure ventilation may offer some benefits when all other treatment modalities have failed and patients remain in severe status after conventional therapy. Magnesium sulfate has been noted to help reverse bronchospasm in conjunction with standard therapy if PEFR is 25% or less of predicted but is not useful in patients with mild or moderate obstruction. Although widely discussed in the literature, the data for ketamine, heliox, and continuous positive-pressure ventilation are less compelling.

There are no absolute indications for intubation except for respiratory arrest and coma. Possible indication for intubation includes exhaustion, worsening respiratory distress, persistent or increasing hypercarbia, and changes in mental status. Intubate semielectively, before the crisis of respiratory arrest, because intubation is difficult in patients, who have asthma.

8. How should I decide whether a patient can be discharged or requires hospitalization?

Disposition of patients is usually determined by clinical response after three doses of aerosolized β_2-agonist therapy; ipratropium (if used); and corticosteroids. If patients have clear breath sounds, are no longer dyspneic or are back to baseline, and have an FEV_1 or PEFR 70% of predicted, they may be discharged home. Patients with an incomplete response to treatment, that is, FEV_1 between 50% and 70% of predicted and mild dyspnea, can be considered for discharge after assessing their individual circumstances. Patients with a poor response to bronchodilators, that is, FEV_1 $<$50% of predicted and who have moderate to severe symptoms after treatment, require hospitalization. If an ED observation capability exists, observation for 4 to 6 hours poststeroid administration will decrease the number of inpatient admissions.

TABLE 26-1. MEDICATIONS USED TO TREAT ASTHMA AND COPD EXACERBATIONS

Medications	Dosage and Route
Inhaled short-acting β_2–agonists	
Albuterol Nebulizer solution (5 mg/mL)	2.5–5 mg every 20 minutes for three doses, then 2.5–10 mg every 1–4 hours as needed, or 10–15 mg/hour continuously or 7.5 mg bolus
MDI (90 μg/puff): *must be used with spacer device*	Four to eight puffs every 20 minutes up to 4 hours, then every 1–4 hours as needed
Systemic (injected) β_2-agonists*	
Epinephrine 1:1000 (1 mg/mL)	0.3–0.5 mg every 20 minutes for three doses subcutaneously
Terbutaline (1 mg/mL)	0.25 mg every 20 minutes for three doses subcutaneously
Inhaled anticholinergics	
Ipratropium bromide nebulizer solution (0.25 mg/mL)	0.5 mg every 30 minutes for three doses then every 2–4 hours as needed
MDI (18 μg/puff): *must be used with spacer device*	Four to eight puffs as needed
Systemic corticosteroids	
Prednisone or prednisolone Methylprednisolone	40–60 mg by mouth 125 mg intravenously

COPD, chronic obstructive pulmonary disease.
*Exercise extreme caution in patients with known coronary artery disease.

9. What should be considered at time of discharge?

Patients who received corticosteroids acutely should continue oral steroid therapy at home for 3 to 10 days. For courses of less than 1 week, there is no taper required. For a 10-day course, there remains no need to taper if the patients are concurrently taking inhaled formulations. Dosing parameters are controversial, so choose a moderate regimen (about 40–50 mg prednisone per day); Patients not already on controller medications who have mild persistent asthma should be started on low-dose inhaled corticosteroids or oral leukotriene modifiers, such as zafirlukast or montelukast. Long-acting β-agonists, such as salmeterol, should be added to the regimen of patients with moderate persistent asthma who are inadequately controlled on inhaled corticosteroids. All patients should be advised to use their short-acting β-agonists on a regularly scheduled basis for a few days and then as needed. Patient education should be provided at discharge, as well as an appointment for a follow-up visit within several weeks.

10. Does pregnancy change the management of acute asthma?

No. It is important to treat pregnant asthmatics aggressively to prevent maternal hypoxia and subsequent fetal morbidity and mortality. Patients should not be undertreated because of fear of teratogenicity; the risks from respiratory failure and severe acute asthma are greater than from therapy with standard medications. The standard therapy and dosages are the same.

KEY POINTS: EMERGENCY TREATMENT OF ASTHMA

1. Relieve significant hypoxemia: oxygen.
2. Reverse airflow obstruction: β-agonists + ipratropium.
3. Reduce the likelihood of recurrence: corticosteroids.
4. Provide objective measure of improvement: PEFR or FEV1.
5. Adequate discharge planning includes education, medications, and follow-up.

CHRONIC OBSTRUCTIVE PULMONARY DISEASE

11. What is COPD and what are the presenting symptoms of a COPD exacerbation?

COPD is a disease characterized by chronic airflow limitation that is not fully reversible, is progressive, and is associated with an abnormal inflammatory response to noxious particles or gases. It is a combination of small airway disease and parenchymal destruction. It includes emphysema and chronic bronchitis and can coexist with asthma. The characteristic symptoms of COPD are cough, sputum production, and dyspnea on exertion. Exacerbations are characterized by increased dyspnea, often accompanied by wheezing and chest tightness, increased cough and sputum, change in color or thickness of sputum, and fever. Smoking, exposure to occupational dusts and chemicals, and air pollution are the most common causes of COPD.

12. In addition to COPD, what should be included in the differential diagnosis?

- In patients who present with **wheezing**, the differential diagnosis includes asthma, CHF, pneumonia, cardiogenic pulmonary edema, and bronchitis.
- In those who present with **dyspnea**, the differential diagnosis includes myocardial ischemia, pericardial effusion, pneumothorax, pulmonary embolism, pneumonia, asthma, acute respiratory distress syndrome (ARDS), bronchiectasis, pulmonary fibrosis, pleural effusion, tuberculosis, and metabolic disturbances, acidosis and shock.

13. Which diagnostic tests are helpful in the management of COPD?

Pulse oximetry should be used in every patient with COPD. Oxygen saturation less than 90% indicates severe hypoxia. Arterial blood gas measurements often can identify patients with increased and continuing hypoxia, hypercarbia, and respiratory acidosis, especially if compared with the patient's baseline values. Check theophylline levels if indicated. Chest radiographs are appropriate in COPD exacerbations to help manage complications and concomitant disease. In patients with cor pulmonale, continuous cardiac monitoring may identify any associated arrhythmias. The use of B-type natriuretic peptide (BNP) does not substitute for clinical judgment when trying to differentiate COPD from CHF as a numeric cut-off value that differentiates between the two diseases remains elusive. In contrast to asthma, acute pulmonary function tests are less helpful in the emergency setting because of the difficulty that sick patients with COPD have in performing these tests properly. Differentiation of mild, moderate, and severe COPD relies on the FEV_1 and the ratio of FEV_1/forced vital capacity (FVC) $<70\%$. Making this calculation without formal pulmonary function tests (PFTs) is generally not possible in the ED.

14. What are the key objectives when treating a COPD exacerbation and how are they achieved?

The key objectives are to relieve hypoxemia, alleviate reversible bronchospasm, and treat the underlying etiology of the exacerbation. The cornerstone of initial management is treating the hypoxia with supplemental oxygen with a goal of oxygen saturation of 90% or greater. Despite

adequate oxygen saturation, CO_2 retention due to the obstructive nature of the disease can occur insidiously with little change in symptoms. Thus, oxygen administration should be carefully monitored by frequent clinical assessment, continuous pulse oximetry, and arterial blood gases, when needed. Excessive supplemental oxygen in this small subset of patients can cause respiratory arrest secondary to loss of their hypoxemia-induced ventilatory drive. Relief of airflow obstruction (bronchoconstriction) is usually accomplished by administration of either intermittent doses of aerosolized β_2-agonists or anticholinergics, such as ipratropium. Systemic corticosteroids are indicated in severe exacerbations of COPD. The use of methylxanthines (theophylline or aminophylline) remains controversial and should be used when only when there is inadequate response to short-acting bronchodilators.

15. What about antibiotics?

Routine antibiotic coverage is controversial, but the Global Initiative for Chronic Obstructive Lung Disease (GOLD) guidelines recommend antibiotic therapy for patients with three cardinal symptoms: increased dyspnea, increased sputum volume, and increased sputum purulence or patients that require invasive or noninvasive mechanical ventilation. The antibiotic choices should reflect local antibiotic sensitivity to *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Moraxella catarrhalis*. Guidelines for treatment of pneumonia, if present, should be considered.

16. How can I determine if my patient is improving?

Ask the patient how he or she feels, re-examine, and monitor the oxygen saturation. If the patient was able to perform objective measures of pulmonary function, compare FEV_1 or PEFR (the best of three attempts) obtained on presentation with that obtained after treatment.

17. When should a patient with COPD be intubated?

Noninvasive modalities such as continuous positive airway pressure (CPAP) and bi-level positive airway pressure (BiPAP) often can obviate the need for intubation by improving gas exchange, decreasing hypoxia, and reducing work of breathing. Noninvasive intermittent ventilation (NIV) has been studied in several randomized controlled trials consistently providing positive results with success rates of 80% to 85%. Any patient unable to tolerate NIV or not responding to NIV with changes in mental status, increased respiratory distress with cyanosis, acute deterioration, respiratory arrest, shock, severe acidosis (pH of 7.25), or hypercapnia ($PaCO_2$ >60 mm Hg) should be intubated and mechanically ventilated immediately.

18. How can I decide whether a patient can be discharged or requires hospitalization?

Relapse rates remain high because patients with COPD have less respiratory reserve and function that is not quickly reversible. These patients often take longer than an ED visit to recover and require hospitalization. Failure to improve while in the ED, failed outpatient management, and concerning pulmonary infections are reasons for hospitalization. Patients who return to near baseline with improvement from ED treatment and have good social support systems in place may be discharged home with close follow-up.

19. What should be considered at time of discharge?

Patients who received corticosteroids acutely should continue oral steroid therapy at home for up to 10 days. Dosing parameters are controversial, so choose a moderate regimen (about 40–50 mg of prednisone per day); no tapering is required. Patients should continue to use their short-acting rescue medications. Adding inhaled anticholinergics plus longer-acting sympathomimetic bronchodilators may improve lung function and help improve effectiveness of pulmonary rehabilitation. The use of chronic inhaled corticosteroids is most beneficial for patients with an FEV_1 between 1 and 2 L. Antibiotics should be prescribed to patients deemed well enough for discharge who have experienced increase in sputum production, thickness, or change in sputum color. Patients with a $PaCO_2$ lower than 60 mm Hg at baseline should be

evaluated for home oxygen therapy. Patient education should be provided at discharge, as well as an appointment for a follow-up visit within several days.

20. When is ipratropium bromide contraindicated in the management of patients with asthma or COPD?

Ipratropium bromide contains derivatives of soy lecithin and related food products. Patients with soybean or peanut allergies may develop anaphylaxis if exposed to this medication in either metered dose inhaler (MDI) or nebulized forms.

KEY POINTS: EMERGENCY TREATMENT OF COPD

1. Relieve significant hypoxemia: oxygen.
2. Reverse airflow obstruction: β-agonists + ipratropium.
3. Consider antibiotics if there are changes in sputum production.
4. Patients with COPD have less respiratory reserve and require admission more frequently than patients with asthma.
5. CPAP or BiPAP may obviate the need for endotracheal intubation.
6. Adequate discharge planning includes education, medications, and careful follow-up.

PNEUMONIA

21. Why do I need to know about pneumonia?

Pneumonia is the seventh leading cause of death overall and the leading cause of death from infectious disease in the United States. There are approximately 4 to 5 million identified cases of community-acquired pneumonia (CAP) each year, resulting in 1.3 million hospital admissions. The ED serves as the portal of entry for 75% of these admissions. When properly identified and treated as an outpatient, the mortality of CAP decreased from 30% to about 1%. Overall mortality has decreased by 3% since 1990. The role of the emergency physician is to diagnose pneumonia accurately, initiate timely antibiotic therapy, and make an appropriate disposition.

22. How does a pulmonary infection develop? What predisposes people to it?

Pneumonia is an infection of the alveolar spaces of the lung. It commonly develops via inhalation of infectious particles or aspiration of oropharyngeal or gastric contents and less commonly through hematogenous spread of infection, direct invasion from contiguous structures, direct inoculation, and reactivation of prior disease. Table 26-2 lists predisposing factors.

23. What are differences in presentation of *typical* pneumonia and *atypical* pneumonia?

- **Typical** pneumonia presents with the abrupt onset of high fever, cough productive of purulent sputum, shortness of breath, and pleuritic chest pain. Infants may present with fever associated with irritability, tachypnea, intercostal retractions, nasal flaring, and grunting. Cough may be absent in infants. Elderly or debilitated patients may present with nonspecific complaints and findings such as confusion or deterioration of baseline function, rather than classic symptoms. The most common organism is *Streptococcus pneumoniae*.

TABLE 26-2. FACTORS PREDISPOSING TO DEVELOPMENT OF PNEUMONIA

Factor	Likely Populations
Impaired swallowing/airway protection	Patients with history of alcohol abuse, CVA, ET and NT intubation, head injury, impaired gag reflex, seizures
Extremes of age	Very young and very old
Underlying pulmonary disease	Pulmonary embolism, COPD, pulmonary foreign body or tumor, pulmonary contusion, atelectasis
Chest wall disorders Prevent good cough and clearing of secretions	Rib fracture, surgical wounds, myopathies affecting chest muscles
Impaired mucociliary clearance mechanisms	Smokers, smog, alcohol, underlying viral infection, chronic lung disease
Impaired immune function	HIV, cancer, chemotherapy, malnutrition, sickle-cell disease, chronic steroid use
Other predisposing risks—these may lead to more severe infections with more virulent organisms	Diabetes, alcoholism, recent antibiotic use, recent hospitalization

COPD, chronic obstructive pulmonary disease; CVA, cerebrovascular accident; ET, endotracheal; NT, nasotra-
Adapted from www.clevelandclinicmeded.com\diseasemanagement\infectiousdisease\communitypneumonia.htm.

- **Atypical** pneumonia has a more insidious onset and includes a prominent cough often with the absence of sputum production. Patients may have only a mild fever and are more likely to have extrapulmonary manifestations such as sore throat, dermatitis, headache, cardiac complications (i.e., pericarditis, myocarditis), hepatitis, and renal disease. There are no consistent clinical or radiographic criteria available to distinguish typical from atypical pneumonia. The most common organism is *Mycoplasma pneumoniae*.

24. What are the most common causative agents in CAP and nosocomial pneumonia?

The causative organism is unknown in 30% to 50% of patients with CAP. In those patients for whom the causative organism is known, *S. pneumoniae* is the most common agent (see Table 26-3). During hospitalization, exposure to more virulent organisms changes the pattern of infection. Gram-negative bacilli, particularly *Klebsiella*, *Pseudomonas aeruginosa*, and *Escherichia coli*, are responsible for more than 50% of cases. *Staphylococcus aureus* accounts for another 10% to 20% of hospital-acquired pneumonias and tends to be associated with more severe cases. The remainder of cases is usually due to anaerobic oral flora, *S. pneumoniae*, *Legionella*, and *Moraxella catarrhalis* (each accounting for $<$10% of cases). Nosocomial acquired pneumonias are rising rapidly and in some cases may account for 17% of pneumonias when patients return to the ED. Patients who develop a hospital-acquired pneumonia have an attributable mortality of 27% to 50%.

25. What are the presenting signs and symptoms in a patient with pneumonia?

Patients with pneumonia usually present with cough, dyspnea, sputum production, fever, and pleuritic chest pain. The physical examination may show evidence of alveolar fluid (inspiratory

TABLE 26-3. IDENTIFIED PATHOGENS IN COMMUNITY-ACQUIRED PNEUMONIA

Pathogen	Percentage of Cases	Usual Pattern Caused
Streptococcus pneumoniae	20–60	Typical
Haemophilus influenzae	3–10	Typical
Mycoplasma pneumoniae	1–6	Atypical
Staphylococcus aureus	3–5	Typical
Viral (various incl. influenza)*	2–16	Atypical
Legionella species	2–8	Typical
Chlamydia pneumoniae	4–6	Atypical
Aspiration	6–10	Variable
Gram-negative bacilli (*Klebsiella, Pseudomonas,* etc.)	3–10	Typical
Others	3–5	Variable

*Percentage of viruses is highly variable and was as high as 36% in one study and may be higher in infants and young children than in adults.
Adapted from www.clevelandclinicmeded.com\diseasemanagement\infectiousdisease\communitypneumonia.htm.

rales); consolidation (bronchial breath sounds); pleural effusion (dullness and decreased breath sounds); or bronchial congestion (rhonchi and wheezing). Findings consistent with pneumonia include fever, tachypnea, tachycardia, decreased oxygen saturation, and altered mental status associated with severe illness.

26. What diagnostic studies are useful in the evaluation of pneumonia?

Although some providers will treat healthy, low-risk patients with suspected pneumonia empirically, others feel a chest X-ray is mandatory in every patient with a history and symptoms suggestive of pneumonia. It is difficult to identify a set of specific criteria for ordering a chest X-ray but all patients who present with a cough do not need chest radiography. Clinical judgment must be used in the presence of clinical indicators. The American Thoracic Society (ATS) and the Infectious Disease Society of America (IDSA) include radiographic findings as part of their definitions of pneumonia. The IDSA 2007 pneumonia guidelines state in their executive summary that, "In addition to a constellation of suggestive clinical features, a demonstrable infiltrate by chest radiograph or other imaging technique, with or without supporting microbiological data, is required for the diagnosis of pneumonia."

An arterial blood gas may augment the information obtained through pulse oximetry to assess the need for respiratory support. In addition, the following laboratory tests may be used to aid in risk stratification of patients: complete blood count and serum electrolytes. The use of sputum Gram stain and blood cultures is controversial.

27. What radiographic findings are helpful in making a microbiologic differential diagnosis?

X-ray findings may suggest the underlying microbial etiology, but the overlapping variations in radiographic signs between different organisms may lead to misclassification. An X-ray is not diagnostic of a specific pathogen. A chest X-ray is helpful in defining the extent and location of the infiltrate (e.g., perihilar or multilobar involvement). In addition, dehydration and the radiographic manifestations of chronic diseases may obscure the infiltrates of pneumonia (see Table 26-4).

TABLE 24-4. RADIOGRAPHIC APPEARANCES OF COMMUNITY-ACQUIRED PNEUMONIA*

Radiographic Pattern	Suggested Organisms
Lobar	*Streptococcus pneumoniae, Klebsiella* species, pneumonia due to bronchial obstruction
Diffuse patchy infiltrate involving multiple lobes	*Staphylococcus aureus, Haemophilus influenzae* or gram-negative organisms
Interstitial pattern	*Mycoplasma pneumoniae, Legionella,* viral, *Pneumocystis* (patients with HIV or HIV risks) *Chlamydia psittaci*
Cavitary lesions with air-fluid levels	*S. aureus, Klebsiella, Pseudomonas aeruginosa, Mycobacterium tuberculosis*†

*The development and resolution of X-ray findings may lag clinical findings by hours to days.
†Tuberculosis may take on almost any radiographic appearance with some predilection for the upper lobes.
Adapted from www.clevelandclinicmeded.com\diseasemangement\infectiousdisease\communitypneumonia.htm.

28. How do I determine the disposition of a patient with pneumonia?

Once a diagnosis of pneumonia is strongly suspected by history, physical, and X-ray results, the next decision is whether the patient is appropriate for discharge or requires hospital admission. Severity-of-illness scores, such as the CURB-65 criteria (confusion, uremia, respiratory rate, low blood pressure, age 65 years or greater), or prognostic models, such as the pneumonia specific severity of illness (PSI), are useful disposition aids. The PSI uses a combination of 20 parameters to evaluate patients, assign disease severity and mortality risk, and guide disposition. (See Tables 26-5 to 26-7.) Because of its prognostic accuracy, effectiveness, and safety as a decision aid, the PSI has become the reference standard for risk stratification. Although there are no clear guidelines for intensive care unit (ICU) admission, several rules have been published. Patients requiring ventilatory assistance or pressors and those who have altered mental status, multilobar or bilateral infiltrates, pleural effusion, age >65, comorbid conditions, respiratory rate >30, heart rate >125, Sat <90%, white blood cell count <3 or >20, blood urea nitrogen (BUN) >11, pH <7.35, and sodium <130 are among the patients who should be considered for an ICU setting.

29. What treatment should be started in the ED?

Supportive care, including oxygen and ventilatory support, should be given as required. Rehydration, antipyretics, and pain control should also be started as indicated. Antibiotic therapy should begin, based on the most likely pathogens, as soon as the diagnosis of pneumonia is made or strongly suspected. Studies have shown a decreased mortality and length of stay in a group of patients admitted for CAP when antibiotics were administered within a range 4 to 8 hours of arrival. All patients being admitted for pneumonia from the ED should have their first dose of antibiotics begun prior to transfer to the floor or ICU.

30. Which antibiotic should I use?

The choice of which antibiotic to begin is based on the site of treatment and suspected pathogens. The suggestions in Table 26-8 should be used in consideration with the clinical picture, recent literature, local preference, and resistance patterns. Increasing evidence has strengthened the recommendation for combination empirical therapy for severe CAP. Presence of comorbidities, such as chronic heart, lung, liver, or renal disease; diabetes mellitus; alcoholism; malignancies; asplenia; and immunosuppressing conditions all influence the empiric choice of antimicrobials.

TABLE 26-5. FACTORS IN PNEUMONIA DISPOSITION DECISION

PORT PSI Scoring	
PORT Characteristics	***Points Given for Presence of Characteristic***
Demographics	
Age—male patient	Age in years (one point per year)
Age—female patient	Age in years: −10
Lives in nursing home	+10
Coexisting illnesses	
Neoplastic disease	+30
Liver disease	+20
CHF	+10
Cerebrovascular disease (TIA or CVA)	+10
Renal disease	+10
Physical examination findings	
Acute disorientation, stupor or coma	+20
Respiratory rate 30 per minute	+20
Systolic blood pressure 90 mm Hg	+20
Temperature <35°C or 40°C	+15
Heart rate 125 beats per minute	+10
Laboratory and x-ray findings (if study performed)	
Arterial pH <7.35	+30
Blood urea nitrogen 30 mg/dL	+20
Sodium <130 mmol/L	+20
Glucose 250 mg/dL	+10
Hematocrit <30%	+10
Partial pressure of arterial oxygen	+10
<60 mm Hg or oxygen saturation <90%	
Pleural effusion	+10
Total points = age + (-10 if female) + sum of above comorbidities, examination findings, and testing	

CHF, congestive heart failure; CVA, cerebrovascular accident; PORT, Patient Outcome Research Team; PSI, pneumonia-specific severity of illness; TIA, transient ischemic attack.

TABLE 26-6. PSI CLASS BASED ON POINT TOTALS AND MORTALITY*

Points Calculated from PSI	Class	Mortality
<51 points	I	0.1%
51–70	II	0.6%
71–90	III	0.9%
91–130	IV	9.5%
>130	V	26.7%

PSI, pneumonia-specific severity of illness.
*Those patients who are younger than 50 years and without any comorbid illnesses or vital sign abnormalities fall into class I and may be safely treated as outpatients. Patients not falling into risk class I require additional laboratory testing so that they may be assigned to risk classes II to V. Patients in classes II and III may be appropriate for outpatient management or a brief observation stay. Patients in class IV or V require hospital admission with a subset requiring intensive care unit admission.

TABLE 26-7. OTHER FACTORS (NOT PART OF PORT PSI) THAT IMPACT DISPOSITION DECISION

- Patient's clinical appearance
- Patient's ability to tolerate oral intake
- Patient reliability
- Social factors such as home support
- Clinical judgment of the physician (most important)

PORT PSI, Patient Outcome Research Team pneumonia-specific severity of illness score.

31. Has the epidemiology of pneumonia changed in recent years?

The epidemiology of CAP continues to change due to a number of constantly changing factors, such as the discovery of new pathogens, changing antibiotic resistance, an aging population, and new tools for fighting infection. Pneumonia due to *S. pneumoniae* continues to be the most common single agent and is continually evolving resistance to a wider array of antibiotics. Viral and atypical agents are the most rapidly growing causes. *Pneumocystis carinii* pneumonia and tuberculosis (TB) are significant pathogens, particularly in the developing world. The severe acute respiratory syndrome (SARS) was first described in 2002 in China and subsequently spread worldwide. Influenza virus is predicted to be the next global pandemic. Diagnostic and treatment guidelines are available on the Centers for Disease Control and Prevention (CDC) website at www.cdc.gov.

32. What is the role of sputum Gram stain and culture?

The value of the Gram stain for expectorated sputum is controversial because it is uncertain how accurately expectorated sputum reflects lower respiratory tract secretions and pathology. Gram stain is frequently negative for specific organisms and the results rarely change therapy.

TABLE 26–8. EMPIRICAL ANTIMICROBIAL THERAPY FOR COMMUNITY-ACQUIRED PNEUMONIA IN IMMUNOCOMPETENT ADULTS

Patient/Setting	Common Pathogens	IDSA/ATS Consensus 2007 Empiric Therapy
Outpatient <60 years old No comorbid diseases	*Streptococcus pneumoniae* *Mycoplasma pneumoniae* Chlamydia pneumoniae *Haemophilus influenzae* Viruses	A macrolide or doxycycline
Outpatient >65 years old or having comorbid disease or antibiotic therapy within past 3 months	*S. pneumoniae* (drug resistant)	Fluoroquinolone alone* or a macrolide plus a beta lactam
	M. pneumoniae *C. pneumoniae* *H. influenzae* Viruses	
	Gram-negative bacilli*†	
Inpatient Not severely Ill	*Staphylococcus aureus**† *S. pneumoniae*	
		A macrolide and beta lactam or a fluoroquinolone‡ alone
Inpatient Not severely ill	*H. influenzae* Polymicrobial Anaerobes *S. aureus* *C. pneumoniae* Viruses	
Inpatient Severely ill	*S. pneumoniae*	Beta lactam/beta lactamase inhibitor and azithromycin, or a fluoroquinolone‡ *Pseudomonas aeruginosa* possible: (intravenous macrolide or fluoroquinolone and aminoglycoside intravenously) or (antipseudomonal quinolone) and antipseudomonal beta lactam For methicillin-resistant *S. aureus* Add vancomycin or linezolid

Continued

TABLE 26–8. EMPIRICAL ANTIMICROBIAL THERAPY FOR COMMUNITY-ACQUIRED PNEUMONIA IN IMMUNOCOMPETENT ADULTS—cont'd

Patient/Setting	Common Pathogens	IDSA/ATS Consensus 2007 Empiric Therapy
	Legionella *Gram-negative bacilli* *M. pneumoniae* Viruses *S. aureus*	

ATS, American Thoracic Society; IDSA, Infectious Disease Society of America.
*In the outpatient setting, many authorities prefer to reserve fluoroquinolones (levofloxacin, gatifloxacin, moxifloxacin, gemifloxacin) for patients with comorbid diseases/risk factors.
†In most cases, patients with pneumonias due to these organisms should be hospitalized.
‡Levofloxacin, gatifloxacin, moxifloxacin, or gemifloxacin
§Critically ill patients in areas with significant rates of high-level pneumococcal resistance and a suggestive sputum gram-stain should receive vancomycin or a newer quinolone pending microbiologic diagnosis.

Gram stain may be more useful in high-risk or hospitalized patients and should be considered in this group. The use of sputum with other stains (such as acid-fast for TB) and techniques such as direct fluorescent antibody staining have a continuing and developing role but are probably not helpful in ED management of these patients.

33. Are routine blood cultures helpful in the management of CAP?

The utility of blood cultures to determine causative agents in unselected patients with CAP is only 5% to 14% and rarely alters therapy for patients presenting to the ED with pneumonia. More discriminatory use may potentially reduce resource utilization. However, in patients with severe symptoms or significant risk factors, blood cultures may demonstrate uncommon causative organisms or unexpected antibiotic resistance. Currently, guidelines suggest that blood cultures be obtained in the ED prior to initiating antibiotics on hospitalized patients, requiring ICU admission and those with cavitary lesions, leukopenia, active alcohol abuse, severe liver disease, asplenia or pleural effusion.

KEY POINTS: EMERGENCY TREATMENT OF PNEUMONIA

1. Begin empiric treatment early based on suspected pathogens.
2. Calculation of the PSI or CURB-65 score is a reliable predictor of mortality and a tool to assist with disposition decisions.
3. Support oxygenation, ventilation, and circulation as indicated by the patient's condition.
4. Recently hospitalized and nursing home patients will be infected with different organisms and require additional antibiotic coverage.
5. Consider the presentation typical versus atypical when making therapy decisions.

WEBSITES

http://pda.ahrq.gov/clinic/psi/psi.htm
www.clevelandclinicmeded.com\diseasemanagement\infectiousdisease\communitypneumonia.htm
www.emedicine.com/emerg/topic465.htm
www.nhlbi.nih.gov/guidelines/asthma/asthgdln.htm

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CHAPTER 27

VENOUS THROMBOEMBOLISM

Stephen J. Wolf, MD

1. **What is Virchow triad of thromboembolism?**
   Venous stasis, vascular trauma, and hypercoagulable state.

2. **What two diseases represent the continuum of venous thromboembolism (VTE)?**
   Deep venous thrombosis (DVT) and pulmonary embolism (PE).

3. **What percentage of patients diagnosed with DVTs have concomitant PE when studied?**
   Fifty percent. Additionally, a similar percentage of patients with a diagnosed PE will have a concomitant DVT when studied.

KEY POINTS: MAJOR RISK FACTORS CARRYING A RELATIVE RISK OF 5 to 20 FOR VTE

1. Recent surgery ($\leq$ 4 weeks)
2. Immobilization (equivalent to bed rest $\geq$ 3 days)
3. Pregnancy (third $>$ second $>$ first trimester)
4. Postpartum (for up to 42 days)
5. Malignancy (treatment active, within 6 months, or palliative)
6. History of VTE

4. **List the minor risk factors for VTE.**
   - Cardiovascular disease (i.e., heart failure, hypertension, or congenital heart disease)
   - Indwelling vascular access
   - Estrogen use (hormone replacement or oral contraceptives)
   - Obesity
   - Neurologic disease (i.e., cerebrovascular accident [CVA], paresis)
   - Inflammatory bowel disease (i.e., Crohn's disease, ulcerative colitis)
   - Tobacco
   - Advanced age
   - Hypercoagulable states (i.e., factor V Leyden thrombophilia, prothrombin mutation, circulating lupus anticoagulant, antithrombin III deficiency, and protein C or S deficiency)

5. **What is the relative risk for VTE in a patient with a minor risk factor?**
   Two to four times that of a patient without a minor risk factor.

6. Are there any signs or symptoms of PE that are diagnostic?

No. Although the common clinical signs and symptoms of shortness of breath, chest pain, tachypnea, and tachycardia occur in upward of 97% of patients diagnosed with PE, they are nonspecific. Patient presentations can range from mild shortness of breath to cardiovascular collapse.

7. Why is a clinician's pretest probability for VTE so important?

Because no diagnostic test available for the evaluation of VTE is absolute (with a perfect sensitivity and specificity), the results of any given test must be considered in combination with the pretest probability to yield a posttest likelihood of disease. Thus, the pretest probability should be used to determine when to initiate a patient work-up and how to interpret the results of any test. See Fig. 27-1.

8. When determining pretest probability for DVT, what are the Wells Criteria?

- Malignancy (+1.0 point)
- Paralysis/cast (+1.0 point)
- Recent immobilization or surgery (+1.0 point)
- Tenderness along deep veins (+1.0 point)
- Swelling of entire leg (+1.0 point)
- 3-cm difference in calf circumference (+1.0 point)
- Pitting edema (+1.0 point)
- Collateral superficial veins (+1.0 point)
- Alternative diagnosis more likely than DVT (-2.0 points)

9. Once I have calculated the total Wells score for DVT, how do I interpret it?

The incidences of DVT based on the Wells Criteria for DVT follow:

- Low pretest probability (<2 points): 3% incidence
- Moderate pretest probability (2–6 points): 17% incidence
- High pretest probability (>6 points): 75% incidence

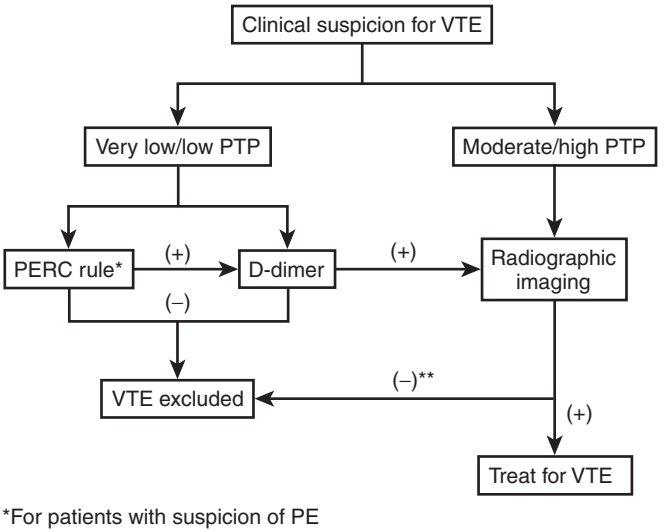

Figure 27-1. Clinical suspicion for VTE.

10. What is the pulmonary embolism rule-out criteria (PERC) rule for pulmonary embolism?

PERC is a clinical decision rule that can be used to identify patients who do not require a laboratory or radiograph work-up to exclude the diagnosis of pulmonary embolism. The criteria include:

- Age <50 years
- Heart rate <100 beats per minute
- Initial room air SaO_2 >94% at sea level
- No clinical signs suggesting DVT/PE
- No hemoptysis
- No recent surgery or trauma
- No history of VTE
- No oral hormone use

11. How do I use the PERC rule?

In patients with a low gestalt clinical suspicion for PE, if all criteria are met, the patient has a less than 2% risk of PE, and further work-up is not indicated. Patients with clinical suspicion who do not meet all criteria may require further evaluation.

12. When determining pretest probability for PE, what are the Wells Criteria?

- Signs/symptoms of DVT (+3 points)
- No alterative diagnosis more likely than PE (+3 points)
- Heart rate >100 beats per minute (+1.5 points)
- Recent immobilization or surgery (+1.5 points)
- History of previous VTE (+1.5 points)
- Hemoptysis (+1.0 point)
- Malignancy (+1.0 point)

13. Once I have calculated the total Wells score for PE, how do I interpret it?

The incidences of PE based on the Wells Criteria for PE follow:

- Low pretest probability (<2 points): 4% incidence
- Moderate pretest probability (2–6 points): 21% incidence
- High pretest probability (>6 points): 67% incidence

14. What is a D-dimer test? How is it used?

D-Dimer, a degradation product of cross-linked fibronectin, is found in increased levels of the circulation of patients with acute VTE. The enzyme-linked immunosorbent assay (ELISA), rapid ELISA, turbidimetric, and whole-blood agglutination D-dimer assay are useful to exclude thromboembolic disease in patients with low pretest probability for VTE. Traditional latex agglutination tests cannot be used in these algorithms because of poor negative predictive values. Although useful in ruling out venothromboembolic disease in select populations, owing to a lack of specificity, D-dimer has not proved useful at ruling in the diagnosis.

15. Which patients can have VTE excluded, based on a negative D-dimer?

Low to low-moderate pretest probability patients only. You would miss the diagnosis anywhere from 5% to 20% (depending on the type of assay) of the time if you used a negative D-dimer to rule out VTE in a patient with a moderate to high pretest probability (>40%).

16. What are some clinical situations that cause a false-positive D-dimer, lending to a decreased specificity?

Sepsis, disseminated intravascular coagulation (DIC), aortic dissection, pregnancy, recent surgery, and severe trauma.

17. What are some clinical situations that might cause a false-negative D-dimer?

- Subacute thrombosis (>7 days)
- Recent anticoagulation

18. What noninvasive imaging methods are available for the diagnosis of DVT?

- **Duplex ultrasound**: The sensitivity and specificity are operator dependent and related to patient symptomatology, but this test can detect more than 95% of acute symptomatic proximal DVTs. It should be noted that its specificity for acute thrombosis decreases in the settings of chronic or recurrent VTE.
- **Radio fibrinogen leg scanning**: Good for detecting distal clots, including clots in the calf, popliteal ligament, and distal thigh vein, but relatively poor for more proximal clots.
- **Impedance plethysmography**: The diagnostic sensitivity and specificity depend on the technical expertise of the person doing the study, but in many centers this test detects more than 95% of acute proximal lower extremity DVT.
- **Spiral computed tomography (CT) venography.** Although rarely used and not extensively studied, reports show promise for this modality, with a sensitivity and specificity comparable to ultrasound.
- **Magnetic resonance imaging (MRI) venography.** Can be useful, particularly for patients with inconclusive ultrasound studies or a contraindication to radiation or contrast dye (i.e., pregnant patients). It has proved accurate for both lower extremity and pelvic DVT.

19. Can a single duplex ultrasound exclude DVT in isolation?

No. In patients with a moderate to high pretest probability for DVT, a negative D-dimer or a repeat duplex ultrasound in 5 to 7 days is indicated to definitively exclude the diagnosis.

20. Are there classic chest X-ray (CXR) findings in patients with PE?

No. The chest radiograph may be normal in up to 30%. Subtle abnormalities such as focal atelectasis, slight elevation of a hemidiaphragm, or focal hyperlucency of the lung parenchyma, may be present. Specifically, local oligemia of vascular markings (Westermark's sign) or a plural-based wedge-shaped infiltrate suggestive of pulmonary infarct (Hampton's hump) is relatively uncommon.

21. Are there classic electrocardiogram (ECG) findings in patients with PE?

No. Normal or near-normal ECGs with sinus tachycardia or nonspecific ST-T wave changes may be seen up to 30%. The findings classically associated with PE (i.e., S1, Q3, T3 pattern or a new right bundle-branch block) occur in less than 15% of patients and occur with the same frequency in patient work-up whether or not they are diagnosed with PE.

22. What imaging studies can be used to evaluate PE?

- **Computed tomography angiography (CTA) scan.** CTA is rapid and generally the test of choice. It is highly sensitive in diagnosing central or segmental emboli and other intrathoracic pathology, however not as sensitive in ruling out subsegmental clots. Outcomes data using newer generation multirow detector CTAs are showing higher sensitivities. Diagnostic accuracy can be improved by performing CTA with a CT venography (CTV), if indicated.
- **Ventilation/perfusion (V/Q) scan.** Traditionally, a normal V/Q scan has been used to essentially rule out a diagnosis of PE with a posttest probability of disease of $<4\%$. Likewise, a high-probability scan is considered to rule in the diagnosis. Unfortunately, upward of 60% of V/Q scans are read as nondiagnostic (low or intermediate probability), particularly if the chest radiograph is abnormal or the patient has underlying cardiopulmonary disease. A nondiagnostic scan should be followed up with further diagnostic work-up. Limitations of the V/Q scan include tech support, availability, and interpretation variability.
- **Pulmonary angiogram.** This test has been the traditional gold standard for the diagnosis, even though its inter-rater agreement on interpretation has been reported to be as low as 65%. Limitations include contraindications to contrast dye injection, interventional radiology support, interpretation variability, and the need for expertise.
- **Magnetic resonance angiogram (MRA).** Limited studies have shown MRA has sensitivities and specificities comparable to standard pulmonary angiogram. Although often not

immediately available, MRA is a useful modality when contraindications to conventional studies, such as contrast allergies or pregnancy, exist.

23. What are the relative contraindications to CTA for PE?

- Contrast dye allergy
- Renal insufficiency
- Inability to lie flat
- Severe claustrophobia
- Morbid obesity exceeding the CT scanner's weight limit

24. What are the diagnostic test options for PE with the pregnant patient?

Although less specific in pregnancy, a D-dimer test can still be sensitive for excluding the diagnosis in low pretest probability patients. For patients requiring diagnostic imaging, it is important to recognize that both CTA and the V/Q scan carry a radiation risk to the fetus that should be discussed with the patient. Recently, CTA (without concomitant CTV) has been shown to confer lower fetal radiation doses than the V/Q scan. CTV should be avoided due to the high pelvic radiation doses. When available, MRI is a diagnostic option that carries minimal radiation risk; however, it requires cardiac and respiratory gating in addition to significantly more time. Finally, the ultrasonographic identification of DVT in pregnant patients with respiratory complaint can obliviate the need for thoracic testing.

25. What happens if the diagnosis of PE is missed?

PE is listed as one of the most common causes of death in the United States, and yet only about 25% of cases are diagnosed. Of the undiagnosed 75%, a small number die within 1 hour of presentation, so it is unlikely that diagnosis and intervention could improve outcome in that group. In the rest, however, the mortality from untreated PE is approximately 30%.

26. What is a massive PE?

A massive PE can be either anatomically defined as the occlusion of greater than 50% of the pulmonary vasculature or physiologically defined as an embolus that is complicated by severe cardiopulmonary distress. These two definitions are not synonymous because a normal individual can lose 50% of pulmonary circulation without significant hemodynamic compromise, whereas a patient with significant underlying cardiopulmonary disease could suffer major hemodynamic compromise with a much smaller clot.

27. What is the treatment for VTE?

Anticoagulation should be started in the ED. Studies suggest that patients with proximal DVTs and temporary risk factors can be anticoagulated with heparin (80 mg/kg loading dose followed by 18 mg/kg/hour infusion) followed by Coumadin for 3 months, whereas patients with calf DVTs need to be treated for only 6 weeks. Patients with permanent risk factors potentially need lifelong treatment but should be anticoagulated for at least 6 months.

28. What is the role of low-molecular-weight heparin (LMWH) in the treatment of VTE?

LMWH is at least as effective as heparin for the treatment of DVT and probably should be considered the treatment of choice based on efficacy, low side effect profile, and cost-effectiveness. Outpatient management of DVT with LMWH is commonplace and has proved safe and effective. Subgroup analysis indicates that LMWH probably will be adopted for the treatment of PE as well, although further conclusive studies are needed.

29. Under what conditions can an inferior vena caval filter be considered in the treatment of VTE?

- Contraindication to anticoagulation
- Recurrent VTE despite adequate anticoagulation

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CONGESTIVE HEART FAILURE AND ACUTE PULMONARY EDEMA

Jeffrey Sankoff, MD, FACEP, FRCP(C)

1. What is congestive heart failure (CHF)?

Cardiac dysfunction that leads to an inability of the heart to work as a pump to meet the circulatory demands of the patient. As a result, pulmonary congestion occurs, and when the problem is severe enough, pulmonary edema results.

2. What causes CHF?

CHF results from any of four types of processes:

- Restrictive (hemochromatosis, pericardial disease)
- Ischemic (myocardial infarction)
- Congestive (volume overload of the ventricle from valvular insufficiencies)
- Hypertrophic (longstanding hypertension or valvular stenoses)

3. Describe the symptoms of CHF.

Common symptoms are **dyspnea** (the subjective feeling of difficulty with breathing) and **fatigue**. Early in the course of CHF, the patient reports exertional dyspnea; the heart is able to supply enough cardiac output for sedentary activities but does not have the reserve to increase cardiac output during exercise. As heart failure worsens, even minimal activity may be difficult. Patients also report orthopnea (dyspnea relieved by assuming an erect posture), paroxysmal nocturnal dyspnea (sudden onset of dyspnea at night), and nocturia.

KEY POINTS: CARDINAL SYMPTOMS OF CHF

1. Exertional dyspnea
2. Fatigue
3. Paroxysmal nocturnal dyspnea
4. Orthopnea

4. What causes these symptoms?

When the patient with CHF assumes the supine posture, venous return from the abdomen and lower extremities is improved, increasing right ventricular cardiac output to the pulmonary vasculature. Because of limitations on the ability of the left ventricle to increase output increased pulmonary hydrostatic pressure results. The patient has difficulty lying flat and sleeps with several pillows or sits in a chair to relieve these symptoms. Redistribution of fluid may also lead to increased urine output and nocturia. In severe CHF, volume redistribution may be sufficient to lead to acute pulmonary edema.

5. Name the four main determinants of cardiac function in CHF.

Cardiac output (CO) = stroke volume (SV) × heart rate (HR)

SV is determined by:
- Preload
- Afterload
- Myocardial contractility

6. What is preload?

Within limits, the amount of work cardiac muscle can do is related to the length of the muscle at the beginning of its contraction. This relationship is shown graphically by the **Frank-Starling curve**, in which ventricular end-diastolic volume (VEDV) represents muscle length and the SV represents cardiac work. (It is easier to measure pressure than volume, so we graph ventricular end-diastolic pressure [VEDP] versus SV.) Thus, preload is measured as VEDP. As VEDP increases, SV increases. At higher VEDP, the increase in SV is less for a given increase in VEDP.

7. What are the effects of decreased contractility?

From Figure 28-1, it can be seen that the heart can function on different Frank-Starling curves, depending on the contractility. A heart with better contractility will produce more CO than a heart with poorer contractility for the same preload. CHF results when the right and left ventricles begin to respond differently to similar preloads (i.e., operate on different Frank-Starling curves). If the right ventricular output for a given preload is better than the left for the same preload, the left ventricle cannot keep pace with the right and the difference remains in the pulmonary vasculature increasing hydrostatic forces and eventually leading to pulmonary edema.

8. What about afterload?

Afterload refers to the pressure work the ventricle must do. The important components here are ventricular wall tension and systemic vascular resistance (SVR). As SVR increases (hypertension), the left ventricle must generate more force to push blood forward against this resistance. The result is an increase in ventricular wall tension that may compromise blood flow to myocytes. The response of the myocardium to chronic hypertension is to increase in size. This hypertrophy eventually compromises the ability of the heart to produce adequate CO.

9. What about HR?

Low HR may cause low CO even in normal hearts because CO = SV × HR. But at excessively high HRs, there may be insufficient time to fill the ventricle during diastole, leading to decreased left ventricular end-diastolic pressure (LVEDP) and SV, so CO may become compromised despite the tachycardia.

10. How does this physiology relate to treatment?

The goal of treatment of CHF is to improve CO. This can be accomplished by modifying each of these parameters. Diuretics, dietary salt, and water restriction decrease preload and

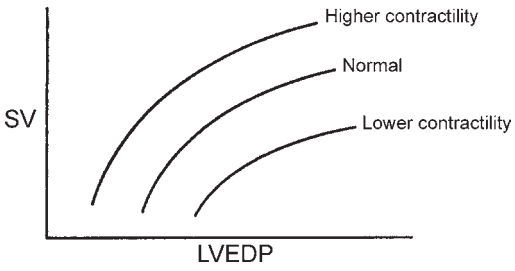

Figure 28-1. The Frank-Starling curve.

improve volume work. Inotropic agents such as digoxin improve contractility. Vasodilators are helpful in reducing afterload and the pressure work required of the heart.

11. Describe the role of B-type natriuretic peptides (BNP) in CHF.

Natriuretic peptides are hormones produced by the heart in response to increased wall stress and are secreted into the circulation as a marker of failure. BNP is an independent predictor of increased LVEDP, and levels correlate with symptoms and severity of disease. It has been suggested as a screening tool for the diagnosis of CHF in the ED, although its utility is limited.

12. How do I interpret BNP levels?

- <100 pg/mL unlikely to be CHF
- 100–500 pg/mL may be CHF
- 500 pg/mL most consistent with CHF (There is difficulty interpreting elevated BNP when patient has known severe CHF.)

13. How do patients with CHF present to the ED?

Patients with CHF present to the ED in one of two ways:

- As a subacute gradual worsening with slow progression of symptoms and signs
- Or as an acute, dramatic change from baseline with acute *flash* pulmonary edema

With respect to the first presentation, these patients tend to have evidence of worsening fluid overload, with elevated jugular venous distension and peripheral edema. They may have associated pulmonary edema as well, but it is usually mild to moderate with no respiratory distress. The second presentation is seen in patients who are generally euvolemic but have profound pulmonary edema as their main symptom.

KEY POINTS: ED PRESENTATIONS OF CHF

1. Subacute, fluid overloaded
2. Acute flash pulmonary edema, euvolemic

14. Discuss acute pulmonary edema.

The most dramatic presentation of CHF is acute pulmonary edema. To understand pulmonary edema, we must return to the physiologist Starling, who described the interaction of forces at the capillary membrane that lead to flow of fluid from capillaries to the interstitium. Simply put, there is a balance between hydrostatic pressure and osmotic pressure. Under normal circumstances, this leads to a small net movement of fluid from the capillaries into the lung interstitium. This fluid is carried away by lymphatics. In CHF, the left ventricular CO changes suddenly while the right ventricle remains unchanged. As a result there is a sudden increase in pulmonary vascular volume and the capillary hydrostatic pressure increases to the point that the lymphatics no longer can handle the fluid. This then leads to interstitial edema, and subsequently to alveolar edema.

15. How do patients with acute pulmonary edema usually present?

Patients develop acute shortness of breath and generally are fighting for air. These patients sit upright to decrease venous return (preload); they cough up frothy, red-tinged sputum. Auscultation of the lungs reveals wet rales throughout and sometimes wheezes (owing to bronchospasm, or cardiogenic asthma). This presentation is a true emergency and requires immediate aggressive therapy. Because of the stress response that this causes, patients have a large catecholamine release and are almost always very hypertensive. In these patients, hypertension is the response to and not the cause of the acute CHF, although left unchecked, the hypertension will result in an overall worsening.

16. What is the treatment of acute pulmonary edema?

First, follow the ABCs (airway, breathing, and circulation). In severe hypoxia, airway and breathing may be compromised and the patient needs to be intubated. The use of noninvasive mask continuous or bilevel positive airway pressure (BiAP) has decreased the need for intubation of patients with pulmonary edema; however, this has not significantly affected in-hospital mortality. Intubation may also be avoided with aggressive medical treatment. It is important to continuously re-evaluate the patient with an eye on intervening with more aggressive measures. For example, you might decide, "I will intubate if the patient is not better in 15 minutes or worsens during that time." Administer oxygen to maintain sufficient oxygen saturation (>90%), either by nasal cannula or nonrebreather mask, continuous positive airway pressure, or BiPAP. Continuously monitor oxygen saturation with pulse oximetry.

17. What about drug therapy?

Drug therapy is aimed at decreasing preload. Nitrates are first-line drugs and are useful in the form of sublingual nitroglycerin (NTG), topical NTG paste, or intravenous NTG drip. NTG is predominantly a venodilator, reducing preload, but also dilates coronary arteries, so it may be especially helpful in the setting of coronary artery disease. Diuretics should only be administered in patients with signs of obvious fluid overload (i.e., peripheral edema, elevated jugular venous distension). In most cases of acute CHF, patients are actually euvolemic and the administration of diuretics is associated with worse outcomes. When indicated, furosemide is given as a 40-mg intravenous bolus (larger amounts if the patient is already on diuretics although high-dose diuretics are associated with worse outcomes). Initially, within 5 to 15 minutes of the injection, venodilation occurs, although this is of limited clinical benefit in the setting of nitrates. This action is followed within 30 minutes by diuresis. In addition to furosemide, morphine may be given, 5 to 10 mg, intravenously, to decrease anxiety and the work of breathing. It also is a mild venodilator, further decreasing preload. With decreased anxiety, there is decreased sympathetic response and decreased afterload.

KEY POINTS: ED MANAGEMENT OF CHF

1. Noninvasive mask ventilation may prevent the need for intubation
2. Frequent re-evaluation of the patient with an eye toward increasingly aggressive measures is important
3. Nitrates are first-line pharmacotherapy
4. Diuretics should be reserved for patients with fluid overload

18. Are there other drugs that are useful in the treatment of acute pulmonary edema?

Yes. For the patient who is hypertensive, it is often helpful to lower the blood pressure (afterload). Hypertension and tachycardia generally result from reflex mechanisms because of the acute decompensation and often correct themselves with the initial treatment outlined previously. With severe hypertension, nitroprusside is the treatment of choice. It is a venodilator and arterial dilator, reducing preload and afterload. Start the infusion at 10 mcg/min and titrate upward every 5 minutes. It is important to monitor the blood pressure closely. If the patient becomes hypotensive, stopping the infusion causes a prompt increase in blood pressure because nitroprusside has such a short half-life. Generally, doses of 0.5 to 2 mcg/kg/min are sufficient.

19. What about giving positive inotropic drugs?

Digoxin, which is used in the treatment of chronic CHF, has little role in the treatment of acute pulmonary edema. Inotropic agents that are helpful include dobutamine, dopamine, and

milrinone. Dobutamine and dopamine are positive inotropic agents. Dopamine has more alpha effect, especially at higher doses, and should be reserved for hypotensive patients. In cardiogenic shock that is refractory to these agents, milrinone infusion may be given. The ideal situation for administering these agents is in an intensive care unit (ICU) with pulmonary artery monitoring to measure filling pressures, CO, and other hemodynamic parameters.

20. When the initial treatment has begun, what else needs to be done?

After the patient is stabilized, routine tests are done, the most important being chest radiograph and electrocardiogram (ECG). Cardiac monitoring is begun; pulse oximetry is monitored continuously, and vital signs are recorded frequently. It is generally necessary to insert a Foley catheter for close monitoring of urine output. The search is on to try to discover the underlying reason for acute decompensation (almost always ischemic in nature, although occasionally related to arrhythmias).

21. Do all patients with CHF need to be admitted to the hospital?

Patients with a new diagnosis of CHF need an inpatient work-up that includes serial cardiac enzymes and an assessment of the global function of the heart. Patients with known CHF who have mild symptoms or signs may be managed on an outpatient basis, assuming that they are compliant with medications, have an appropriate social network, and follow up with their primary care physician. All patients with acute CHF require admission.

22. What are the usual precipitating causes of CHF exacerbations?

The most common cause of the subacute CHF exacerbation is undermedication, either as a result of patient noncompliance with medication orders, dietary salt restrictions, or as a result of a change in medication under a physician's supervision. The patient gradually retains more and more fluid, resulting in eventual overload and presentation to the ED. Causes of acute CHF exacerbations are principally cardiac and include acute myocardial infarction, arrhythmias, and rarely severe hypertension (as previously noted, hypertension is usually the result and not the cause of the acute exacerbation). Noncardiac causes include infection and anemia. When precipitating factors are identified, specific therapy should be initiated.

23. What is the outpatient treatment of CHF?

Angiotensin-converting enzyme (ACE) inhibitors are the mainstay of long-term treatment of CHF, leading to a decrease in mortality and an increase in functional capacity. Other drugs that act on the renin-angiotensin system (angiotensin receptor antagonists and spironolactone) also are effective. β-blockers are useful in that they block the cardiac effects of long-term adrenergic stimulation. Diuretics also are beneficial, especially in patients with volume overload. Digoxin causes an improvement in symptoms but no overall decrease in mortality. Combined therapy with hydralazine and isosorbide dinitrate has shown a decrease in mortality and is particularly useful in patients who have contraindications to other classes of drugs.

24. How do ACE inhibitors work in CHF?

In response to cardiac decompensation, the renin angiotensin system is activated. Angiotensin is a potent vasoconstrictor, leading to increased afterload. Stimulation of aldosterone leads to sodium retention and extracellular fluid volume expansion and increased preload. ACE inhibitors help to decrease afterload by decreasing angiotensin II-mediated vasoconstriction and decrease preload by blocking sodium retention and volume expansion.

25. What is the long-term prognosis for patients with CHF?

Prognosis depends on the cause and severity of the heart failure. The prognosis is good when the underlying cause can be corrected, as in valvular heart disease. Patients with mild disease that can be controlled with ACE inhibitors with or without low doses of diuretics generally do well. Overall, however, patients with CHF have a 10% to 20% yearly death rate, and fewer than half survive 5 years.

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ISCHEMIC HEART DISEASE

Edward P. Havranek, MD

CHAPTER 29

1. How is ischemic heart disease classified?

a. Chronic stable ischemic heart disease
- Asymptomatic
- With angina
- Postmyocardial infarction (MI), with or without angina

b. Acute coronary syndromes
- Unstable angina
- Acute non-ST segment elevation myocardial infarction (NSTEMI)
- Acute ST segment elevation MI (STEMI)

2. How do patients with acute ischemic heart disease present?

The most common presenting symptom is **central chest discomfort**. The discomfort also may be felt (in descending order of frequency) in the right or left chest, the right or left shoulder or arm, the throat or jaw, the epigastrium, and rarely, the ear. Less commonly, patients may present with episodic dyspnea. Some patients may have ischemia and even acute MI with no symptoms.

3. To define the discomfort better, what information should be obtained?

- Chronology
- Frequency
- Location
- Precipitating factors
- Radiation
- Alleviating factors
- Duration
- Quality
- Intensity
- Associated symptoms

4. Describe the typical features of chest discomfort in ischemic heart disease.

Typically, patients with **stable angina** have discomfort during exertion relieved within minutes by rest. The degree of exertion bringing on the discomfort can vary from day to day. The discomfort is often vague, is described most often as a tightness, pressure, or heaviness, and is substernal in location, with radiation to the jaw, shoulder, or upper extremity common.

Patients with **unstable angina** usually have similar pain, but it occurs with progressively less exertion (crescendo angina) or at rest. Patients with unstable angina who have pain at rest also should have pain with exertion. **Prinzmetal's angina**, which is caused by coronary artery spasm, is an exception to this rule. Patients with Prinzmetal's angina typically have pain at rest, usually in the early morning hours, and often do not have exertional discomfort. True vasospastic angina is uncommon.

Patients with **acute MI** typically have pain that is more severe than any preceding angina. It may be described as crushing but usually not ripping or tearing.

5. What other symptoms are associated with the chest discomfort of ischemic heart disease?

Shortness of breath commonly accompanies angina. Many conditions other than angina that cause chest discomfort, such as pulmonary disease and anxiety disorder, also are accompanied by shortness of breath. Diaphoresis occurs less frequently with angina but should raise one's level of suspicion of angina because it does not occur often with other disorders that cause chest pain. Nausea and vomiting can occur with acute MI—the larger the infarction, the more common are nausea and vomiting. Thus, patients with anterior MI are more likely to have nausea and vomiting than are patients with inferior MI.

KEY POINTS: TYPICAL FEATURES OF ANGINA

1. Substernal location
2. Radiation to the arm, neck, or jaw
3. Precipitation by exertion, relief with rest
4. Sensation of tightness, pressure or heaviness
5. Accompanied by shortness of breath

6. Is there anything different about evaluating elderly patients?

The symptoms associated with ischemic heart disease in patients older than 75 years are more likely to be atypical. The older the patient, the more atypical the symptoms become.

7. What about diabetics?

Although it is often taught that patients with diabetes are more likely to present with atypical symptoms or no symptoms at all, the literature suggests that diabetic patients present just like everyone else except perhaps for being a little more likely to have shortness of breath.

8. Should demographic features and the presence or absence of coronary risk factors change your mind about the diagnosis?

They are not particularly useful for making a diagnosis except in patients who have objective evidence of prior ischemic heart disease in whom the likelihood of another ischemic event occurring is rather high. A young woman with no risk factors but with typical symptoms and electrocardiogram (ECG) changes should be suspected of having ischemic disease. Conversely, a middle-aged man with diabetes and hypertension whose chest pain has no typical features should still be treated as if he may have the disease but may be less likely to have the disease.

9. List the key elements of the initial evaluation of a patient with a suspected acute coronary syndrome.

- The patient should be placed on O_2 and a cardiac monitor and have intravenous (IV) access established.
- The patient should have an **ECG** within 5 minutes of arrival.
- **Vital signs** are vital. The presence of hypertension, hypotension, or tachycardia must be dealt with early.
- A **history** directed at the key elements described previously and a **cardiovascular examination** come next; the examination is useful for ruling out other diagnoses, such as pericarditis and aortic dissection.
- Administer aspirin (325 mg).

10. What is the significance of abnormal ST segment changes on an ECG?

Abnormal ST segment changes may or may not represent ischemic cardiac injury. The *current of injury* that accompanies ST segment elevation MI is typically a convex-downward elevation of the ST segment. It may be confused with pericarditis or early repolarization. Other causes of ST elevation are as follows:

- Left ventricular hypertrophy
- Acute pericarditis
- Acute cor pulmonale
- Hyperkalemia
- Hypercalcemia
- Hypothermia
- Left ventricular aneurysm

ST depression may be caused not only by cardiac ischemia but also by such things as ventricular hypertrophy, drugs (e.g., digoxin), atrioventricular junctional rhythm with a retrograde P wave, and electrolyte abnormalities.

11. What is the typical time course of ECG changes in ischemic cardiac injury?

The initial changes are T-wave prolongation and increased T-wave magnitude, either upright or inverted. Next, the ST segment displays elevation or depression. A Q wave may be seen in the initial ECG or may not develop for hours to days. As the ST segment returns to baseline, symmetrically inverted T waves evolve. This classic evolution is documented in approximately 65% of patients with acute MI.

12. Can the ECG be normal while a patient is having cardiac ischemia or an acute MI?

Although serial ECGs showing evolving changes are diagnostic for acute MI in greater than 90% of patients, 20% to 50% of initial ECGs are normal or show only nonspecific abnormalities. The initial ECG may be diagnostic for acute MI in only half of patients. Therefore, an early repeat ECG may be helpful.

13. Are cardiac markers useful in the ED?

Maybe. Troponin I, the most commonly used cardiac marker, doesn't rise definitively until 4 to 6 hours after the onset of ischemia, but borderline elevations in high-sensitivity assays may begin to rise before then. Single determinations are generally not sufficiently specific to make a diagnosis. One should not wait to see an elevated troponin before making a decision to treat for acute coronary syndrome.

14. How can echocardiography be useful in ED patients with suspected acute coronary syndrome?

It may be useful when the ECG is nondiagnostic, such as when there is a left bundle-branch block or minimal ST elevation. The presence of a wall motion abnormality is evidence that supports the diagnosis of ischemia, although it may be the result of an old rather than acute infarction. Echocardiography also may provide information about complications, such as mitral regurgitation and pericardial effusion.

The sensitivity of echocardiography is limited. A negative echocardiogram in the setting of a typical history and ECG does *not* rule out the diagnosis of an acute coronary syndrome. Waiting for the results of an echocardiogram may unnecessarily delay treatment.

15. What other diagnoses should be considered in a patient with chest pain?

The patient's history is paramount:

- A life-threatening condition, **dissecting aortic aneurysm**, also may cause sustained chest pain or typical angina.
- Pleuritic chest pain may be caused by pleuritis, pericarditis, pneumothorax, or pulmonary embolism.

- Nonanginal pain may be found with the onset of herpes zoster or with cervical or thoracic nerve root compression.
- Esophagitis, esophageal spasm, and esophageal rupture may mimic pain of cardiac origin.
- Patients with anxiety or depression syndromes often complain of chest pain.

16. What are the indications for reperfusion therapy in acute MI?

- ST elevation greater than 1 mm in two leads
- Pain and ST elevation not immediately responsive to nitroglycerin
- Pain lasting less than 6 hours (In many patients, reperfusion therapy still may be of benefit 12 hours after the onset of pain.)

17. What if persistent ST elevation is not present?

There is no proven benefit to thrombolytics *or* immediate angiography and coronary intervention.

18. What is the preferred method of reperfusion therapy in acute MI with ST elevation (STEMI), thrombolytic therapy, or primary coronary intervention?

Angioplasty (usually with coronary stenting) in the setting of acute MI if done within approximately 90 minutes of arrival reduces mortality to a greater degree than does thrombolytic therapy. Primary coronary intervention has the added advantages of lower rates of reocclusion in the subsequent few days and lower risks of intracranial bleeding. The problem is that studies that include data from nontertiary hospitals show that optimally delivered angioplasty is not often accomplished. Unless a skilled catheterization laboratory team can intervene within 90 minutes after presentation, thrombolytic therapy is preferable.

19. What if cardiac intervention is not available on site?

Transfer to an intervention-capable facility is beneficial if the time to reperfusion will be less than 90 minutes. Although some studies have suggested that transfer is beneficial, even if time-to-reperfusion might be longer, current guidelines favor thrombolytic therapy under these circumstances.

20. How do you choose which thrombolytic agent to use?

Streptokinase (and its modified form, anistreplase) is currently the least popular agent in acute MI because it is thought to be slightly less effective than the other available agents. Reteplase and tenecteplase have become more popular than alteplase because they can be administered in bolus form rather than an infusion with a varying dose schedule. No matter which agent is chosen, it should be given within 30 minutes of arrival.

21. What is the preferred therapy for cardiogenic shock?

The only therapy shown to decrease the historically high mortality associated with this syndrome is primary coronary intervention (angioplasty, generally with stent implantation). This should be performed without delay. The added benefit of immediate transfer to a catheterization laboratory is that this facilitates insertion of an intra-aortic balloon pump (IABP). An IABP is thought to be superior to pharmacologic therapy for decreasing afterload and augmenting cardiac output.

An exception to this is cardiogenic shock caused by **right ventricular infarction**. This problem should be suspected whenever shock accompanies an acute inferior MI. The presence of jugular venous distention is an important clue to the diagnosis of this syndrome, and volume expansion usually reverses the hypotension.

22. List the contraindications to thrombolytic therapy.

Absolute contraindications

- Active bleeding
- Major surgery or trauma in the past 3 weeks
- Neurosurgery or stroke in the past 3 months

- Prolonged (>10 minutes) or traumatic cardiopulmonary resuscitation (CPR)
- Hypertension (systolic >180 mm Hg, diastolic >110 mm Hg)

Relative contraindications

- Major trauma or surgery more than 3 weeks ago
- Neurosurgery or stroke more than 3 months ago
- Active peptic ulcer
- Hemorrhagic ophthalmic condition, especially diabetic retinopathy

Patients with a known allergy to streptokinase or anistreplase (also known as APSAC [anisoylated plasminogen-streptokinase activator complex]) should be treated with another agent. Exposure to streptokinase or anistreplase in the previous 6 months or streptococcal infection in the previous 6 months are reasons to use another agent.

23. What other diagnoses should be considered before giving thrombolytic therapy?

Aortic dissection and **acute pericarditis** can mimic acute MI. Both have had fatal outcomes when thrombolytics were given. Dissection can be excluded with a careful history, examination of peripheral pulses, and chest radiograph or chest CT scan. Pericarditis can be excluded by carefully listening for a rub and examining the ECG for widespread, concave-upward ST elevation.

24. What is the risk for fatal complications of thrombolytic therapy for acute MI?

Mortality, which almost invariably results from intracranial hemorrhage, occurs in about 0.5% of treated patients.

25. What other medications are useful adjuvants to reperfusion therapy?

- **Morphine.** MI can cause excruciating pain and severe fear and anxiety. IV morphine sulfate should be administered in increments of 2 to 4 mg to alleviate these symptoms.
- **Aspirin and other anti-platelet agents.** Unless the patient has had an aspirin, it should be given immediately because it reduces mortality independent of other therapy. In patients who will undergo immediate intervention, it may be beneficial to start abciximab and a thienopyridine, such as clopidogrel, but this decision should be a collaborative one involving the emergency physician and the cardiologist.
- **β-blockers.** Metoprolol, atenolol, or esmolol given intravenously reduce mortality and infarct size. They are better tolerated than one may think and are underused. Contraindications include heart failure, bradycardia (heart rate <55 beats per minute), and bronchospasm.
- **Heparin and other anticoagulants.** With tissue plasminogen activator (tPA), initiation of heparin is imperative at least 1 hour before the completion of the thrombolytic infusion. With streptokinase or APSAC, administration of heparin should be delayed 4 to 6 hours. Anticoagulants should be continued for at least 48 hours. In addition to unfractionated heparin, fondaparinux and the low-molecular-weight heparin, enoxaparin, have been shown to be acceptable alternatives.
- **Nitroglycerin.** If control of blood pressure is an issue, nitroglycerin is the preferred agent.

KEY POINTS: MOST IMPORTANT MEDICATIONS FOR ALL PATIENTS WITH AN MI IN THE ED

1. Aspirin
2. IV β-blockers

26. What other arrhythmias occur with acute MI?

Ventricular irritability, with frequent premature ventricular contractions (PVCs), nonsustained ventricular tachycardia, and ventricular fibrillation, may occur. Secondary causes such as drugs and electrolyte imbalance should be looked for and treated. Isolated PVCs usually respond to β-blockade. Higher grades of ventricular arrhythmias should be treated with lidocaine or amiodarone. Sustained ventricular tachycardia (lasting >30 seconds) is uncommon in acute MI. Accelerated idioventricular rhythm (heart rate, 60–100 beats per minute) should not be treated.

Bradyarrhythmias also may occur. Second-degree or third-degree heart block that accompanies inferior MI is usually transient, and a temporary pacemaker generally is not required. When heart block accompanies an anterior MI, a temporary pacer *is* required. A prophylactic temporary pacer should be considered when severe conductive system disease (bifascicular block or left bundle-branch block plus first-degree block) accompanies an anterior acute MI.

27. Which patients with unstable angina are at highest risk for MI and benefit from more aggressive treatment?

- ECG changes: Transient or fixed ST segment depression or T-wave inversion, especially when these changes are in leads V_1 through V_3
- Elevated troponin levels
- Age greater than 65
- Known coronary artery disease
- Presence of three or more coronary risk factors (i.e., smoking, hypertension, diabetes, elevated cholesterol, family history)
- Severe angina within the prior 24 hours

These patients are thought to benefit more from more aggressive medical treatment (see question 24) and early catheterization.

28. What medications are useful in the acute coronary syndromes: unstable angina and acute MI without ST elevation (NSTEMI)?

- Always administer **aspirin.** If the patient is intolerant of aspirin or if management without angiography is planned, a thienopyridine, such as clopidogrel, should be added. If angiography is planned, a thienopyridine, such as clopidogrel (including a loading dose), tirofiban, eptifibatide, or abciximab should be added to aspirin. The emergency physician and the cardiologist should coordinate management closely here. For instance, abciximab may be the best choice if there will not be a delay before angiography and if it is highly likely angioplasty and stenting will occur. Otherwise, one of the other agents may be a better choice.
- **Heparin** should be started. Current thinking is that unfractionated heparin and the low-molecular-weight heparin preparation, enoxaparin, are equally effective. Less evidence is available for bivalirudin and fondaparinux, but both appear to be effective. Fondaparinux is thought to be preferable when the patient is at high risk for a bleeding complication. Here again, plans for an invasive strategy may make one agent preferable over another, and collaboration between emergency physician and cardiologist is important.
  - Virtually all patients with unstable angina should be treated with **β-blockers.** It may be desirable to start this therapy in the ED.
- For patients with ongoing pain, always treat with **nitroglycerin**. Start with the sublingual route of administration, and move to IV nitroglycerin if that does not work. Nitroglycerin is the preferred agent when the patient has concurrent hypertension.
- Add **calcium channel blockers** when symptoms recur despite aspirin, nitrates, and β-blockers. *Never* use short-acting dihydropyridines, such as nifedipine, without β-blockers. In the setting of unstable angina, use of calcium channel blockers without concurrent β-blockade increases the risk of MI.

29. Which is better, low-molecular-weight heparin or unfractionated heparin?

There is no consensus about which type of heparin is clinically more effective, so practical considerations are important. The advantage of low-molecular-weight heparin is that it can be given in the ED as a single bolus and doesn't require an infusion pump. The major disadvantage of low-molecular-weight heparin is that it may be difficult to communicate to other providers that the patient has received it (as a result some patients have gotten double doses) and that the effects last longer, making invasive procedures more problematic.

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CHAPTER 30

CARDIAC DYSRHYTHMIAS, PACEMAKERS, AND IMPLANTABLE DEFIBRILLATORS

Christopher B. Colwell, MD, and Ashley C. Mull, MS, MD

1. What is a sinus beat?

At the end of each heartbeat, all myocardial cells are depolarized and experience a refractory period. At this point, certain cardiac cells (sinoatrial and atrioventricular [AV] nodes and some ventricular cells) float back up toward *threshold potential*. It is like a race, and typically the sinoatrial node cells win this race, achieve threshold, fire, and assume the pacemaker *sinus beat* function of the heart.

2. What is the AV node?

The AV node is not simply a passive connection between the atria and ventricles. It is *smart*. Normally, all atrial impulses are conducted to the ventricles. When the ventricular rate becomes sufficiently rapid that cardiac output is compromised, conduction velocity begins to slow in the AV node. This progressive slowing filters the rapid atrial impulses so that serial atrial impulses are not conducted at all. This progressive AV nodal conduction block is a protective mechanism to prevent a dysfunctional rapid ventricular rate.

3. Is it necessary to identify a dysrhythmia before treating it?

If the patient is hemodynamically unstable, no. In the unstable patient, a general rule of thumb: if the heart rate is fast, shock (perform electric cardioversion); and if the heart rate is slow, pace.

4. What is hemodynamic compromise?

In an adult, hemodynamic compromise is hypotension (a systolic blood pressure $<$90 mm Hg) in combination with alteration in mental status, chest pain, or shortness of breath.

5. How do I know whether a patient's dysrhythmia is causing hemodynamic compromise?

Typically, if a patient's ventricular rate is between 60 and 100 beats per minute, any hemodynamic instability is caused by something else. It is unusual, although not impossible, for a tachydysrhythmia with a rate less than 150 beats per minute to be the primary cause of hemodynamic instability. It is extremely rare for a patient with a heart rate less than 150 beats per minute to require electrical cardioversion.

6. How do I treat bradyarrhythmias?

Do not treat bradycardia if the patient is hemodynamically stable and asymptomatic. Always treat the patient, not a number. If the patient has a heart rate less than 60 beats per minute and is hemodynamically unstable:

- Give 0.5 mg (0.01 mg/kg in a child) intravenous (IV) atropine (may be repeated).
- Initiate pacemaker therapy, starting with external pacing. Placement of a transvenous pacemaker (especially without fluoroscopy) always takes much longer than you think it will.

7. How do I treat tachyarrhythmias?

Any unstable patient with a tachyarrhythmia that either is or may be the cause of the hemodynamic instability requires electric cardioversion. Supraventricular tachycardia

(SVT) and atrial flutter often respond to low voltages (50 J), whereas most other tachyarrhythmias typically require at least 100 J to convert to a sinus rhythm. If the patient is hemodynamically stable, the next step is to identify whether the tachyarrhythmia is *narrow-complex* or *wide-complex*.

8. What is a narrow-complex tachycardia?

The AV node conducts impulses directly to the Purkinje system, which courses over the endocardial surface of the ventricles. An electrical impulse travels along the Purkinje fibers quickly: 2 to 3 m/sec. If an impulse enters the ventricles from the AV node, it can rapidly activate the entire ventricular muscle mass—in 0.12 sec, 120 msec, or three little boxes on electrocardiogram (ECG) paper. We see this as a *narrow-complex* QRS on the ECG: a QRS complex with a width of less than 120 msec. A narrow-complex tachycardia must originate above the AV node. Sinus tachycardia, SVT, atrial fibrillation (AF) with rapid ventricular response, and atrial flutter are examples of narrow-complex tachycardias.

9. How do I make the diagnosis of AF when the ventricular rate is fast?

AF is by definition an irregular rhythm, but very rapid AF may appear regular and be impossible to differentiate from SVT on a cardiac rhythm strip. The diagnosis of AF is made by palpating a peripheral pulse and simultaneously auscultating the heart or visualizing the cardiac rhythm. AF is the only arrhythmia that results in a pulse deficit (fewer beats palpable than observed or auscultated) and that has an irregular pulse with varying intensity of the pulse.

10. How do I treat narrow-complex tachycardia in a hemodynamically stable patient?

A narrow-complex tachycardia must originate above the AV node. To control the ventricular rate, you need to block the AV node pharmacologically. If the patient has a rapid narrow-complex tachycardia that cannot be definitively identified, the best initial agent is adenosine, 6 mg IV rapid bolus followed by 12 mg, if needed (which also may be repeated). For SVT, adenosine has a response rate of 85% to 90%, few serious side effects, and a very short half-life. Alternatively, verapamil, 5 to 10 mg, or diltiazem, 20 mg, intravenously over to 1–2 minutes, terminates or controls the ventricular response rate in 80% to 90% of cases. If the patient clearly has AF, rate control, rather than conversion to a sinus rhythm, is the primary goal. β-blockers (metoprolol, 5–10 mg over 2 minutes) and calcium channel blockers (diltiazem, 20 mg over 2 minutes) are effective AV nodal blocking agents and can achieve adequate rate control in most patients with AF. Patients may experience chest tightness, nausea, and shortness of breath with adenosine and should be warned about these temporary unpleasant effects. Rarely, calcium channel blockers can cause hypotension, and there are case reports of life-threatening events after administration of adenosine, so it is important to have good IV access and an advanced cardiac life support (ACLS) cart nearby when giving any of these agents. Adenosine exhibits little effect on infranodal conduction, which has led some authors to recommend its use as a diagnostic agent in wide-complex tachycardias.

11. Is there a time when I shouldn't use adenosine or a calcium channel blocker for a narrow-complex tachycardia?

The one situation where it would be potentially dangerous to use these agents is AF in the setting of Wolff-Parkinson-White syndrome (WPW). In this disorder, there is an accessory pathway between the atria and the ventricles that bypasses the AV node. If an AV nodal blocking agent is given, conduction through the accessory pathway could speed up, making the tachycardia worse and potentially precipitating hemodynamic collapse. AF in WPW can present as narrow- or wide-complex tachycardia. It is difficult to tell on the ECG if someone has WPW if the rhythm is very fast, but if the patient has a known history of WPW, do not give adenosine or a calcium channel blocker. Procainamide or electricity should be used instead.

12. Define premature ventricular contraction.

A premature ventricular contraction occurs when a ventricular site wins the *race* among myocardial cells and ventricular depolarization originates from an ectopic ventricular site.

13. What is a wide-complex tachycardia?

When an impulse originates from damaged or ischemic ventricular muscle instead of the sinoatrial or atrioventricular node, it does not use the Purkinje *superhighway* of conduction and therefore takes longer to activate the ventricular mass: >0.12 second, 120 msec, or three little boxes on the ECG paper. We see this as a *wide complex* QRS complex.

14. What is the most common cause of wide-complex tachycardia?

Ventricular tachycardia (VT). Of awake patients presenting to the ED with a wide complex tachycardia, 70% to 90% have VT, and only 10% to 30% have supraventricular tachycardias with aberrancy (see question 16). VT is even more likely if the patient has a history of a prior myocardial infarction or congestive heart failure. Other causes of wide-complex tachycardia include ventricular fibrillation (VF), a wide-complex, irregular, nonperfusing rhythm that requires electrical defibrillation; and torsades de pointes, a wide-complex rhythm associated with prolonged QT interval.

15. Is VT always hemodynamically unstable?

No. Hemodynamic status should not be used to determine the nature of a wide QRS tachycardia: Do not assume a wide-complex tachycardia is not VT if the patient is hemodynamically stable.

16. What is a supraventricular rhythm with aberrancy?

Usually a supraventricular rhythm traverses the AV node and courses through the large endoventricular conduction fibers, activating the ventricles rapidly and resulting in a narrow QRS complex (<0.12 sec). A wide-complex tachycardia typically represents a tachycardia of ventricular origin. Although less frequent, a supraventricular origin impulse that travels through the ventricle in an aberrant fashion also can be wide and is called supraventricular rhythm with aberrancy. One example, as discussed in Question 11, is AF in the setting of WPW; this is a supraventricular arrhythmia that can present as a narrow- or wide-complex tachycardia depending on the direction of conduction through the accessory pathway.

17. Differentiate VT from SVT with aberrancy based on findings on the 12-lead ECG.

In general, assume VT and treat accordingly whenever there is any question. These findings on the 12-lead all strongly suggest VT:

- AV dissociation
- Fusion or capture beats
- Left or right axis deviation
- QRS width of greater than 140 msec
- Concordance of QRS complexes
- Monophasic or biphasic QRS in lead V1
- RS or QS in lead V6
- History of coronary artery disease or congestive heart failure
- Evidence of AV dissociation on physical examination (Cannon A waves)

Heart rate is *not* an accurate way to differentiate VT from SVT with aberrancy. Again, if there is any doubt, assume VT. Treating SVT with aberrancy as if it were VT is less problematic than treating VT as if it were SVT with aberrancy.

18. How do I treat wide-complex tachycardia?

See Table 30-1.

TABLE 30-1. TREATMENT OF WIDE-COMPLEX TACHYCARDIA

Clinical Situation	Treatment
Unstable patient	**Cardioversion**
Wide-complex tachycardia known to be SVT with aberrancy	**Adenosine** (6 mg intravenous push followed by 12 mg intravenous push if ineffective)
Wide-complex tachycardia of unknown type with preserved cardiac function (no clinical signs of congestive heart failure)	**Amiodarone** (150 mg IV given over 10–15 minutes) or **procainamide** (17 mg/kg IV at a rate of 20 mg/min, to be stopped if the dysrhythmia is suppressed, hypotension occurs, or the QRS complex widens by 50% of its original width)
Wide-complex tachycardia of unknown type in a patient with clinical evidence of congestive heart failure	**Amiodarone**
Rhythm known to be ventricular in origin	**Amiodarone**, **procainamide**, or **lidocaine** (1.0 to 1.5 mg/kg IV, repeated every 5 minutes to a maximum of 3 mg/kg; consider **magnesium** (2 g IV) if torsades de pointes suspected

IV, intravenously; SVT, supraventricular tachycardia.
Adapted from Shah CP, Thakur RK, Xie B, et al: Clinical approach to wide QRS complex tachycardias. *Emerg Med Clin North Am* 16:331–360, 1998.

19. What does amiodarone do?

Amiodarone is a class III antiarrhythmic that, among other effects, prolongs the action potential duration and refractory period, slows automaticity in pacemaker cells, and slows conduction in the AV node. It is approved for the treatment of ventricular and supraventricular arrhythmias, including AF, atrial flutter, and accessory pathway syndromes. Current ACLS guidelines suggest amiodarone be used as a first-line agent for stable VT, and it is also a good option to consider in a hemodynamically stable patient with a wide-complex tachycardia of unknown mechanism. Primary side effects are hypotension and bradycardia. The loading dose for adults is 150 mg intravenously given over 10 to 15 minutes. Amiodarone exhibits a slow onset of action and an even slower clearance.

20. What drug is contraindicated in the treatment of any wide-complex tachycardia?

Verapamil. Because all wide-complex tachycardias must be considered to be of ventricular origin, verapamil carries a high risk of causing hypotension and may cause degeneration of the rhythm to VF or asystole.

21. What is synchronized cardioversion?

Synchronization of delivered energy to match the QRS complex. This reduces the chance that a shock will induce VF, which can occur when electrical energy impinges on the relative refractory portion of the cardiac electrical activity (down slope of the T wave).

22. How do I perform synchronized cardioversion?

- Apply the defibrillation pads to the patient: one attaches to the anterior chest and the other is placed on the patient's back.
- Turn on the defibrillator.
- Select a lead on the monitor that clearly reveals an R wave of greater amplitude than the T wave.
- Engage the synchronization mode by pressing the sync control button, and look for markers on the R waves indicating the sync mode is functioning and capturing the QRS complex and not the T wave.
- You may need to adjust the R wave again until the sync markers occur with each QRS complex. Then select the appropriate energy level.
- Always remember to use adequate sedation in an awake patient. (If you are using defibrillation paddles, coat both paddles with conductive gel and apply 25 lbs. of downward pressure.)

23. Does it make sense to cardiovert asystole?

Strictly speaking, no. Theoretically, electrical cardioversion synchronously depolarizes all myocardial cells simultaneously. All cells then should repolarize synchronously and spontaneously reinitiate sinus rhythm. With asystole, there is nothing to depolarize and nothing to cardiovert. Although the American Heart Association currently does not recommend the routine shocking of asystole, there are two scenarios when cardioversion of apparent asystole may be helpful.

- Conceivably, if the major QRS vector is perpendicular to the axis of the ECG lead, VF may appear as asystole.
- It is also possible to have a fine (very low voltage) VF, which is difficult to distinguish from asystole on the monitor.

If available, a bedside ultrasound of the heart is useful in these circumstances.

24. When is it necessary to anticoagulate a patient with AF prior to cardioversion?

Anticoagulation in patients who have AF for less than 48 hours is unnecessary because the risk of thromboembolism is lower. If the duration of AF has been greater than 48 hours and the patient is stable, cardioversion may be delayed until the patient is fully anticoagulated.

25. Should we be using monophasic or biphasic waveform defibrillation in the ED?

Theoretical advantages to biphasic waveforms include less energy required to achieve effective defibrillation and less postshock myocardial damage and dysfunction at equivalent energy levels. A study published in 2003 showed that biphasic waveforms were more likely to achieve a return to an organized rhythm with one shock than monophasic waveforms but did not see any statistically significant difference in overall survival. A more recent study saw trends toward the requirement for fewer shocks, faster return of spontaneous circulation, and improved survival rates with biphasic waveforms. Despite these promising results, more research is needed to establish a clear, clinically significant benefit to biphasic waveform defibrillation.

26. What is a pacemaker?

An external source of energy used to stimulate the heart. It consists of a pulse generator (i.e., power source), an output circuit, a sensing circuit, a timing circuit, and pacing leads. In the ED, pacing is performed via a temporary external or transvenous pacemaker. Longer-term therapy requires the placement of a surgically implanted device. It is often possible to palpate these devices on physical examination; they are also visible as radiopaque foreign bodies on a chest radiograph.

27. What are the indications for temporary pacemakers?

Temporary emergency pacing is indicated for therapy of significant and hemodynamically unstable bradyarrhythmias and prevention of bradycardia-dependent malignant arrhythmias. In symptomatic or unstable patients who don't respond to atropine or other

pharmacotherapies, emergency pacing should be initiated immediately for any of the following rhythms:

Sinus node dysfunction

- Sinus bradycardia
- Sinus pauses >3 seconds

AV nodal block

- Second-degree AV block (Mobitz I)
- Complete heart block

Infranodal block

- New bifascicular block associated with AMI
- Alternating bundle-branch block with changing PR interval
- Complete heart block

Pacemakers also can be used for overdrive pacing in an attempt to terminate VT by placing a ventricular extrasystole during the vulnerable period of the cardiac cycle. Prophylactic temporary pacing is indicated for insertion of a pulmonary artery catheter in a patient with an underlying left bundle-branch block or use of medications that may cause or exacerbate hemodynamically significant bradycardia.

28. Where are external/transcutaneous pacemakers placed? How are they operated?

Pacing pads and monitor leads are placed preferably in the midanterior chest and just below the left scapula. The desired heart rate is chosen, and the current is set to 0 mA. The external pacemaker is turned on, and the current is increased as tolerated until capture is achieved.

29. State the limiting factors in the use of external pacemakers.

Skeletal muscle contraction can be uncomfortable and often limits use of external pacemakers. Placing electrodes over areas of least skeletal muscle may minimize the discomfort. The physician should use the lowest effective current. Sedation should be strongly considered if these measures are inadequate.

30. Can an external pacemaker be used if a permanent pacemaker malfunctions?

Yes, but be careful to place the external pacer on a *pace only* (fixed-rate) mode and not the sensing mode; otherwise, it may sense spikes from the permanent pacemaker and not fire.

31. What are the advantages of transvenous versus transcutaneous pacemakers?

Transcutaneous leads are the easiest to use for rapid initiation of temporary pacing. Transvenous leads are more reliable and more comfortable because external pacing requires 30 to 100 times the current needed for internal transvenous pacing.

32. How are transvenous and transthoracic pacemakers placed?

Semifloating or flexible balloon-tipped catheters can be placed with central venous access into the subclavian or internal jugular veins. In the ED, using ECG guidance, an alligator clip is connected to a precordial lead such as V_1 with another clip attached to the pacing wire. When a current of injury (ST elevation) is seen on the monitor, the wire should be withdrawn slightly, leaving it in pacing position. If available, fluoroscopy is preferred to ensure proper placement.

33. Can cardiopulmonary resuscitation (CPR) be performed with a pacemaker?

CPR can be performed safely with the external pacing pads in place. Turning the external pacemaker off during CPR is advisable, in particular when defibrillating or cardioverting a patient. If using separate defibrillator paddles, they should be placed at least 2 to 3 cm away from pacing pads to prevent arching of current.

34. List the indications for a permanent pacemaker.

Indications for permanent pacing are constantly evolving. Currently, permanent pacing is indicated for:

- Sick sinus syndrome
- Symptomatic sinus bradycardia
- Tachycardia-bradycardia syndrome
- AF with a slow ventricular response
- Complete heart block
- Chronotropic incompetence (inability to increase the heart rate to match a level of exercise)
- Long QT syndrome

More controversial applications include:

- Cardiomyopathies (hypertrophic or dilated)
- Congestive heart failure (cardiac resynchronization therapy [CRT])
- Severe refractory neurocardiogenic syncope
- Paroxysmal AF (atrial pacing)

35. Describe the complications of permanent pacemaker implantation.

Routine placement of a pacemaker generator into a subcutaneous or submuscular pocket carries the risk of pocket hematoma, which if large enough to palpate often needs surgical drainage. Pocket infection can also occur and manifests as local inflammation, fluctuance, and abscess formation or local cellulitis. Rarely, the pocket itself may erode with extrusion of the generator secondary to infection, trauma, or local tissue ischemia. Infection usually is caused by *Staphylococcus aureus* acutely and *Staphylococcus epidermidis* in chronic infections. Treatment is empiric antibiotics and ultimately removal of the device and reimplantation at a remote site. Wound dehiscence may require admission for debridement and reapproximation of wound edges.

36. What does a pacer setting of *DDD* mean?

The letters represent a pacing code. The code consists of five letters that describe the different types of pacer function; the first three letters are the most relevant to the emergency physician (see Table 30-2). The first letter indicates the chamber paced; the second indicates the chamber in which electrical activity is sensed, and the third indicates the response to a sensed event. A fourth and fifth letter may be added to describe whether the pacemaker is programmable and whether special functions to protect against tachycardia are available. A DDD pacer is able to pace and sense atria and ventricles ([D]ual chambers) and has a (D)ual response to the sensed ventricular and atrial activity (i.e., can pace either the atrium or the ventricle). Spontaneous atrial and ventricular activity inhibits atrial and ventricular pacing; atrial activity without ventricular activity triggers only ventricular pacing.

TABLE 30-2. MODIFIED PACING CODE

First Letter: Chamber Paced to a Sensed Event	Second Letter: Chamber Sensed	Third Letter: Response to Sensed Event
A (atrium)	A (atrium)	I (inhibition)
V (ventricle)	V (ventricle)	T (triggering)
D (dual chamber)	D (dual chamber)	D (dual response)
O (none)	O (none)	O (no response)

37. How can the type of permanent pacemaker be identified in the ED?

Patients should carry a card with them providing information about their particular model. Most pacemaker generators have an X-ray code that can be seen on a standard chest radiograph. The markings, along with the shape of the generator, may assist with determining the manufacturer of the generator and pacemaker battery.

38. What is the most common cause of permanent pacemaker malfunction?

Lead dislodgement. Today, most pacemaker failures are the result of problems with the electrodes or the wires, not the battery or the pulse generator. Because of greater technologic sophistication, patients with pacemaker problems present to the ED much less commonly now than in the past.

39. What is the most reliable indicator of pacer malfunction?

Rates that are usually inappropriate for paced hearts. A nonpaced ventricular rate less than 60 beats per minute or a paced rate greater than 100 beats per minute is probably secondary to pacemaker malfunction.

40. What does a magnet do?

Placing a pacemaker magnet over the pulse generator stops the pacemaker from sensing or responding to a sensed event. The pacemaker reverts to one of three fixed rate modes:

- AOO (atrium paced)
- VOO (ventricle paced)
- DOO (atrium and ventricle paced)

The purpose is to check the pacing rate, which should be done quickly because the pulse generator is no longer prevented from firing during the T wave or from inhibiting serious arrhythmias. Magnets can also be used to turn off some automatic implantable cardioverter defibrillators (AICDs; see Question 49).

41. How do I assess a patient with potential pacemaker malfunction?

- Take a focused history on symptoms related to pacemaker malfunction, including palpitations, weakness, fatigue, shortness of breath, hiccups, syncope, fever, or pain or erythema at the generator site.
- The physical examination should focus on vital signs, mental status, cardiovascular system, and inspection of the generator site.
- An ECG should be obtained to evaluate pacemaker function, and anteroposterior and lateral chest radiographs should be obtained to check pacemaker lead placement and lead and connector integrity.
- Evaluate the ECG. Are there pacing spikes present?

If pacing spikes are not present, apply a circular magnet over the pacemaker site. If the application of the magnet does not result in pacing spikes being produced, there is some mechanical failure present.

If pacing spikes are present, look for capture (a P wave in response to an atrial spike or a QRS complex in response to a ventricle spike, or both, depending on the type of pacemaker). If there is failure to capture, it usually indicates mechanical failure such as lead fracture or dislodgement, but ischemia, metabolic derangements, and certain drugs have also been implicated. If pacing is occurring at an inappropriately short interval between atrial or ventricular contractions, it may be because the pacer is oversensing. If a pacer spike is seen immediately following a native QRS complex, it may be because the pacer is undersensing. See Table 30-3 for a description of common pacemaker malfunctions.

42. What is pacemaker syndrome?

A clinical spectrum of lightheadedness, fatigue, palpitations, syncope, dyspnea on exertion, and hypotension that usually is attributed to asynchronous AV contraction and loss of atrial functional support.

TABLE 30–3. MALFUNCTIONS OF PERMANENT PACEMAKERS

Complication	Description
Oversensing	Occurs when a pacer incorrectly senses electrical activity and is inhibited from correctly pacing. This may be due to muscular activity, electromagnetic interference, or lead insulation breakage.
Undersensing	Occurs when a pacer incorrectly misses intrinsic depolarization and paces despite intrinsic activity. This can be due to poor lead positioning, lead dislodgement, magnet application, low battery states, or myocardial infarction.
Operative failures	This includes malfunction resulting from mechanical factors (such as a pneumothorax, pericarditis, infection, hematoma, lead dislodgement, or venous thrombosis).
Failure to capture	Occurs when a pacing spike is not followed by either an atrial or ventricular complex. This may be due to lead fracture, lead dislodgement, a break in lead insulation, an elevated pacing threshold, myocardial infarction at the lead tip, drugs, metabolic abnormalities, cardiac perforation, poor lead connection, and improper amplitude or pulse width settings.

43. What is twiddler's syndrome?

The most common cause of late lead dislodgement. It occurs when the patient twists or *twiddles* the pulse generator in its pouch to the point of twisting leads around the generator box, shortening and dislodging them from their proper position. The pulse generator may erode through the skin.

44. What is pacemaker-mediated tachycardia?

A normally functioning pacemaker may initiate a tachyarrhythmia. Retrograde conduction of a ventricular beat may cause the atrium to trigger a second ventricular contraction that falls during the pacemaker's refractory period. Because this contraction is not sensed by the pacemaker, the pulse generator fires, initiating a reentrant tachycardia. Treatment consists of lengthening the AV time by any of the following methods:

- Programming an increase in the atrial refractory time
- Administering adenosine or verapamil
- Increasing atrial sensory threshold
- Applying a magnet to stop atrial sensing by the pacemaker

45. What is a runaway pacemaker?

Malfunction of the pacemaker that is manifested by tachycardias secondary to rapid ventricular pacing. The problem is recognized when rates are greater than the upper rate limit settings of the pacemaker and may require drastic measures, such as cutting the pacer leads.

46. What happens as pacemakers lose battery power?

Pacemakers usually show a decline in the rate of magnet-mediated pacing, usually to a predetermined manufacturer's rate. Pacer response varies with manufacturer; some models may also change pacer mode (e.g., DDD to VVI).

KEY POINTS: CARDIAC DYSRHYTHMIAS

1. An unstable patient with any tachydysrhythmia, regardless of the mechanism, requires electrical cardioversion.
2. When trying to decide if the rhythm your patient is in is VT or SVT with aberrancy, assume VT and treat accordingly.
3. The most common reason for early pacemaker malfunction is lead dislodgement.
4. Temporary transcutaneous or transvenous pacing should be used for hemodynamically unstable bradycardias, as well as for overdrive pacing in an attempt to terminate VT.
5. Calcium channel blockers should not be used to treat wide-complex tachycardias.

47. Can a patient with a permanent pacemaker be defibrillated?

Yes, but it is important to place the pads or paddles away from the pulse generator, preferably in the anteroposterior position. Defibrillation can damage the pulse generator. Temporary and even permanent loss of ventricular or atrial capture may occur secondary to elevation of the capture threshold of the pacer leads.

48. What is an AICD?

An **automatic implantable cardioverter defibrillator (AICD)** is a specialized device designed to treat a cardiac tachyarrhythmia. If the device senses a ventricular rate that exceeds the programmed cutoff rate of the implantable cardioverter defibrillator, the device performs cardioversion/defibrillation. Alternatively, the device may attempt to pace rapidly for a number of pulses, usually around 10, to attempt pace termination of the VT. Newer AICDs are a combination of implantable cardioverter defibrillator and pacemaker in one unit.

49. Discuss malfunctions associated with an AICD.

See Table 30-4.

TABLE 30-4. MALFUNCTIONS ASSOCIATED WITH AN AICD

Complication	Description
Operative failure	Similar to operative failures in pacemakers
Sensing failure	Oversensing and undersensing occur, for similar reasons as with pacemakers
Inappropriate cardioversion	May occur if a patient presents in atrial fibrillation or has received multiple shocks in rapid succession
Ineffective cardioversion	Can be seen because of T wave oversensing, lead fracture, lead insulation breakage, electrocautery, MRI, or electromagnetic interference. Can also be caused by inadequate energy output, a rise in the defibrillation threshold because of antidysrhythmic medications, myocardial infarction at the lead site, lead fracture, insulation breakage, or dislodgement of the leads of the cardioversion patches
Failure to deliver cardioversion	Can be caused by failure to sense, lead fracture, electromagnetic interference, and inadvertent AICD deactivation

AICD, automatic implantable cardioverter defibrillator; MRI, magnetic resonance imaging.
Adapted from Higgins GL III: The automatic implantable cardioverter-defibrillator: Management issues relevant to the emergency care provider. *Am J Emerg Med* 8:342–347, 1990.

50. Name the most frequent type of AICD malfunction.
Inappropriate cardioversion.

51. What will a magnet do when placed over an AICD?
Use of a magnet over the AICD inhibits further shocks, but it does not inhibit bradycardic pacing should the patient require it. In older devices, application of a magnet produces a beep for each QRS complex. If the magnet is left on for 30 seconds, the AICD is disabled, and a continuous tone is produced. To reactivate the device, the magnet is removed and replaced. After 30 seconds, a beep returns for every QRS complex.

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HYPERTENSION, HYPERTENSIVE CRISIS, AORTIC DISSECTION, AND AORTIC ANEURYSMS

CHAPTER 31

Björk Ólafsdóttir , MD, and Bo E. Madsen, MD

1. Describe the blood pressure (BP) classification system used by the latest Joint National Committee (JNC 7) report.

Hypertension class is defined as:

- Normal BP <120/80 mm Hg
- Prehypertension systolic blood pressure (SBP) 120–139 mm Hg or diastolic blood pressure (DBP) 80–89 mm Hg
- Stage 1 hypertension: SBP 140–159 mm Hg or DBP 90–99 mm Hg
- Stage 2 SBP >160 mm Hg or DBP >100

2. Describe the importance of anti-hypertensive treatment from a public health perspective.

Hypertension (HTN) is common. A 55-year-old normotensive patient has a 90% lifetime risk of developing hypertension. Treating hypertension has a significant impact on mortality and morbidity and has been associated with a 35% reduction in stroke incidence, a 20% reduction in myocardial infarction (MI), and a 50% reduction in heart failure. It is estimated that by achieving a SBP reduction of 12 mm Hg for 10 years in patients with stage I hypertension and additional cardiovascular risk factors, it will prevent one death for every 11 patients treated.

3. Should treatment be initiated in asymptomatic patients with elevated BP?

Generally not. It has been found that a significant number of patients even with BP >160 in the ED will not have hypertension on follow-up visits. However, if no follow-up can be arranged and the physician feels compelled to initiate treatment, it is recommended to start a thiazide diuretic in the absence of renal or cardiac disease. For patients with SBP >180 mm Hg or DBP >110 mm Hg, consideration should be given to starting an antihypertensive agent. Patients with SBP >200 mm Hg or DBP >120 mm Hg should be started on an antihypertensive agent at discharge.

4. Is ED diagnostic testing necessary in a patient with elevated BP and no symptoms?

Testing is generally not necessary because these patients should receive urgent follow-up with a primary care physician who can confirm the diagnosis and perform diagnostic studies. However, if outpatient treatment is to be started by the emergency physician, a basic metabolic profile is recommended.

5. What is the difference between primary and secondary HTN?

Essential or **primary HTN** accounts for more than 90% of patients with true HTN, and the cause is unknown. Its etiology is likely multifactorial, a combination of both genetics and environment.

Secondary HTN has an identifiable cause. It can result from:

- Primary neurologic disorders (increased intracranial pressure [ICP])
- Renal disorders (glomerulonephritis, polycystic kidney disease, chronic pyelonephritis)

- Vascular disorders (coarctation of the aorta, renal artery stenosis)
- Endocrine disorders (Cushing's syndrome [increased cortisol], Conn syndrome [increased aldosterone], pheochromocytoma [increased catecholamines], thyroid disorders
- Pregnancy-induced HTN, that is, preeclampsia and eclampsia
- Sleep apnea

6. List other causes of transient HTN.

Anxiety, pain, illicit drug use (i.e., cocaine, amphetamines, phencyclidine [PCP], or lysergic acid diethylamide [LSD]), over-the-counter medications containing sympathomimetics, certain toxidromes, alcoholism, and alcohol withdrawal. In addition, certain foods containing large amounts of tyramine can cause transient hypertension. The combination of tyramine-containing foods and monoamine oxidase inhibitors (MAOIs) can cause prolonged severe HTN. MAOIs, in combination with certain drugs (i.e., meperidine, tricyclic antidepressants [TCAs], ephedrine, and amphetamines), can also cause severe hypertension.

7. Define hypertensive emergency/crisis and list some examples.

It is defined as severely elevated BP with acute end-organ damage. Examples include hypertensive encephalopathy; ischemic and hemorrhagic stroke; subarachnoid hemorrhage (SAH); cerebrovascular accident (CVA); acute myocardial infarction (AMI); congestive heart failure (CHF); aortic dissection; acute renal failure (ARF); and preeclampsia/eclampsia.

8. How does hypertensive urgency differ from hypertensive emergency?

With hypertensive urgency, a patient has very high BP but no evidence of acute end-organ damage. There may be a history of chronic HTN and chronic end-organ damage, but if there is no acute worsening, it is classified as an urgency.

9. What symptoms might be present in a patient with hypertensive emergency?

The signs and symptoms of hypertensive crisis are manifestations from the organ systems involved. Central nervous system involvement may cause headache, lethargy, dizziness, confusion, focal neurological deficits, paresthesias, or vision changes and, if left untreated, this can progress to seizures, blindness, and coma. Chest pain, back pain, shortness of breath, and lower extremity swelling may reveal cardiovascular compromise. Decreased urine output, nausea, and generalized malaise and weakness may suggest ARF.

10. What signs support the diagnosis of hypertensive crisis?

Confusion, altered level of consciousness, and focal neurologic findings concurrent with arteriovenous (AV) nicking, copper-wiring, flame-shaped hemorrhages, exudates, and papilledema on funduscopy examination. Crackles, hepatomegaly, and lower extremity edema may be present, as well as a gallop, jugular venous distension, and a displaced point of maximal impulse.

11. What studies should be considered in a patient with a hypertensive emergency?

- If neurologic symptoms or examination findings are present, order a computed tomography (CT) of the head to evaluate for hemorrhagic, ischemic stroke, hypertensive encephalopathy, or SAH.
- Obtain an electrocardiogram (ECG) to screen for hypertrophy, ischemia, or infarction and a chest X-ray (CXR) to look for CHF and aortic dissection.
- A troponin should be ordered in a patient with chest pain, back pain, shortness of breath, confusion, or altered level of consciousness.
- If there is concern for dissection, a stat CT angiogram should be obtained.
- A chemistry panel will screen for renal failure, and a urine sample can be obtained to check for protein, blood, and glucose.

12. How do I diagnose hypertensive encephalopathy?

The classic triad associated with hypertensive encephalopathy is altered mental status (AMS), HTN, and papilledema. Symptoms are reversible with appropriate BP reduction but, if left untreated, coma and death occur within hours.

13. Describe the pathophysiology of hypertensive encephalopathy.

With abrupt, severe elevations in BP, cerebral autoregulation fails. When this occurs, blood flow to the brain is no longer controlled, causing overperfusion, vasospasm, cerebral ischemia, and increased vascular permeability. This leads to cerebral edema and elevated ICP.

14. Why is it important to understand cerebral autoregulation?

Cerebral autoregulation works only within a certain range of mean arterial pressure (MAP), above or below this pressure range cerebral blood flow (CBF) is significantly affected. CBF depends on cerebral perfusion pressure (CPP) and cerebrovascular resistance (CVR); CBF = CPP/CVR. The CPP is defined as MAP-venous pressure. Under normal conditions venous pressure is governed by ICP. MAP = (2 × DBP + SBP) ÷ 3. To maintain CBF and CPP at relatively constant levels, cerebral arteries vasoconstrict when MAP increases and vasodilate when MAP decreases. In normotensive individuals, cerebral autoregulation maintains constant CBF between a MAP of 60 and 120 mm Hg. In hypertensive patients, the lower limit of autoregulation is higher than in normotensive patients and, for both hypertensive and normotensive patients, the lower limit of autoregulation has been found to be approximately 25% below the resting MAP.

15. In a patient with a hypertensive encephalopathy, how much should I decrease the BP?

When treating hypertensive encephalopathy, it is recommended to carefully decrease the MAP 25% over the first hour.

16. How do I treat a patient with hypertensive encephalopathy?

One of several medications may be chosen. They work by different mechanisms but should have three important properties in common:

- An intravenous (IV) drip, enabling easy titration
- Rapid onset
- Short duration of action

IV nicardipine, labetalol, or esmolol are the current recommended medications.

17. What is the treatment threshold for hypertension in ischemic stroke?

Currently there is an absence of conclusive data regarding treatment of HTN in the setting of ischemic stroke. The Stroke Council for the American Heart Association recommends cautiously lowering the BP in ischemic stroke if the SBP is >220 or DBP is >120 mm Hg. If the patient is a thrombolysis candidate, it is recommended to lower the BP to <185/110 mm Hg. The decision making regarding BP management should be done in close consultation with a neurologist or neurosurgeon.

18. What are the recommendations regarding treatment of hypertension in hemorrhagic stroke?

This is currently unclear. There are several ongoing trials aiming at clarifying this. The importance is striking a balance between reducing BP to reduce hemorrhagic volume but avoiding ischemia in the surrounding tissue for hypotension. The decision making regarding BP management should be done in close consultation with a neurologist or neurosurgeon.

19. How do I treat HTN if associated with SAH?

There are no definitive data on what blood pressure is beneficial to the patient. However, most try to achieve a SBP <160. The medications used are labetalol, esmolol, and nicardipine. Other modalities, such as analgesics for pain, should also be used.

KEY POINTS: SPECIAL CONSIDERATIONS WITH HYPERTENSIVE EMERGENCIES

1. Avoid precipitous or excessive drop in BP with cerebrovascular emergencies.
2. Start esmolol before nitroprusside with aortic dissections, or use labetalol.
3. Do not give pure β-blockers for catecholamine-induced hypertensive emergencies.

20. How do I treat a patient with severe HTN and evidence of pulmonary edema?
Start with standard treatment for pulmonary edema: oxygen, diuretics, and afterload reduction with nitroglycerine, which will treat both the hypertension and pulmonary edema.

21. How do I treat a patient with severe HTN and chest pain due to ischemia?
Angina is often accompanied by severe HTN. Reduction of BP is crucial to decrease the work of the myocardium and prevent ongoing ischemia. First-line treatment is IV nitroglycerin combined with a β-blocker and morphine. If this fails to control BP, nicardipine or fenoldopam can be added. Avoid nitroprusside because it can cause a coronary-steal phenomenon in patients with coronary artery disease, causing increased mortality in the presence of an AMI.

22. What are the agents of choice in a patient with severe HTN and ARF?
IV fenoldopam is a dopamine-1-receptor agonist, which is short acting and has advantages over traditional nitroprusside therapy. It increases renal blood flow, creatinine clearance, sodium excretion, and diuresis, and there is no issue with potential cyanide toxicity. It is as effective as nitroprusside with no reported adverse effects. It is, however, more costly. Other reasonable alternatives include nicardipine and labetalol. Angiotensin-converting enzyme (ACE) inhibitors should be avoided if bilateral renal artery stenosis has not yet been ruled out.

23. What should you always think about in a pregnant or postpartum woman with HTN?
Preeclampsia! See Chapter 79.

24. What are the two types of antihypertensive medications that, if stopped abruptly, can cause rebound HTN?
Short-acting sympathetic blockers, such as clonidine, and β-blockers.

25. What antihypertensive medications are contraindicated in a catecholamine-induced hypertensive emergency?
β-blockers. β-receptor-induced vasodilation results in unopposed alpha-adrenergic vasoconstriction and elevates BP further. In patients with concomitant cocaine ingestions, β-blockers enhance coronary artery vasoconstriction, increase BP, fail to decrease heart rate, decrease the seizure threshold, and increase mortality. Labetalol, an α- and β-blocker, theoretically avoids the problem of unopposed alpha, but some sources say it may still cause harm in patients with cocaine ingestion or pheochromocytoma. Antihypertensive agents that can be used for treatment of a catecholamine-induced hypertensive emergency include nicardipine, fenoldopam, phentolamine, and nitroprusside. However, most cocaine or amphetamine-induced HTN responds to appropriate doses of benzodiazepines.

26. What about using oral agents to treat hypertensive emergencies?
They should not be used. The response is unpredictable, cannot be discontinued immediately, or is rapidly titrated. Oral agents are preferable for use in patients with hypertensive urgencies if it is needed to start an antihypertensive medication.

AORTIC DISSECTION

27. Is ischemia the only cause of chest pain to worry about in a hypertensive patient?

No. Always think about the possibility of acute thoracic aortic dissection because this can be a rapidly fatal cause of chest pain, and HTN is the most common risk factor. Other risk factors include congenital heart disease, Ehlers-Danlos, Marfan syndrome, intra-aortic balloon pump use, age, male gender, pregnancy, smoking, family history, stimulant use, or major trauma (due to deceleration injury [not to be discussed here]).

28. What symptoms or signs may be present in a patient with thoracic aortic dissection?

Classic symptoms include:

- Sudden onset of severe chest pain and pain in the jaw/neck/interscapular region that is most severe at onset and is often described as sharp, ripping, or tearing in quality.
- Additionally the patient can experience propagating pain as the dissection progresses.
- The patient often has high blood pressure on arrival.
- Nausea, vomiting, diaphoresis, lightheadedness, and apprehension may be present.
- Syncope can also be the presenting complaint, in some cases even the only symptom. Proximal dissections may cause aortic regurgitation and tamponade.
- Occlusion of aortic branches may cause AMI (coronary artery involvement); stroke (carotid involvement); or paresthesias and arm pain (subclavian artery involvement), which may be suggested by unequal bilateral arm pressures and unequal pulses.
- Spinal artery occlusion will cause neurologic compromise, and hoarseness may result from recurrent laryngeal nerve compression.
- Chest pain unrelieved by large doses of narcotic analgesics should raise the concern for this diagnosis.
- Peak age: Proximal dissection: 50 to 55 years
- Distal dissection: 60 to 70 years

29. What diagnostic studies should be done when thoracic aortic dissection is suspected?

Order a CXR followed by a CT, magnetic resonance imaging (MRI), or transesophageal echocardiography (TEE). CXRs are abnormal in more than 80% of the cases but the abnormalities are non-specific. Spiral contrast enhanced CT is the most practical modality in the ED because it is quick, accurate, and readily available. MRI is sensitive and specific, but scan times are long and access to the patient during the study is limited. TEE is great for determining involvement of the aortic valve and coronary arteries and can detect the presence of pericardial effusion or tamponade, but emergent access to this study is likely limited. Aortography, once considered the gold standard, is rarely used any longer as the initial diagnostic study. If the patient is hypotensive, a bedside echocardiogram can rule out pericardial effusion with tamponade. An ECG should be done to evaluate for AMI or myocardial ischemia.

30. What findings on a CXR suggest thoracic aortic dissection?

Widened mediastinum, loss of the aortic knob, left pleural effusion, deviation of the trachea or nasogastric tube to the right, apical pleural capping, and the *calcium sign* (displacement of the intimal calcium layer in the aorta). The CXR can be normal in up to 20% of cases.

31. Describe the difference between type A and type B thoracic aortic dissections.

Type A dissections (60%) involve the proximal/ascending aorta and require emergent surgical repair, whereas type B dissections (40%) affect the descending aorta (distal to the great vessels) and are managed medically.

32. How do I treat a patient with suspected aortic dissection?

Provide opioids in adequate doses for pain control. Start IV antihypertensive medications immediately if hypertensive and call a cardiothoracic surgeon before the patient heads for the scanner. ED therapy for both types of dissections aims to lessen the pulsatile load and shear forces on the aorta. Rapid reduction of SBP to a range of 100 to 110 mm Hg is indicated. Traditional therapy includes an IV β-blocker such as esmolol in combination with IV nitroprusside. Esmolol is started before nitroprusside to prevent reflex tachycardia and increased shear forces. Alternative treatment regimens include IV labetalol used as a single agent and IV nicardipine or fenoldopam in place of nitroprusside. If the patient is hypotensive, perform a bedside ultrasound to evaluate for pericardial tamponade. Pericardiocentesis can be a life-saving, temporizing intervention until the patient reaches the operating room.

33. Describe the two different types of aortic aneurysms.

- **A true aneurysm,** as seen with most abdominal aortic aneurysms (AAAs), involves dilation of all three layers of the arterial wall: the intima, media, and adventitia.
- In a **pseudoaneurysm** as is seen with acute thoracic aortic dissections, blood communicates with the arterial lumen but is contained solely within the adventitia or surrounding soft tissue. This is much less common.

34. Are aortic dissections and aortic aneurysms somehow related?

No. These two disease processes are totally unrelated, have different symptoms, require different work-ups, and are managed differently. Aortic dissection is caused by a weakness/tear of the intima leading to the formation of a false lumen within the media. Blood enters here and dissects proximally, distally or both.

35. What risk factors are associated with aortic aneurysms?

Aneurysms result from a degenerative process that affects the aortic wall. Risk factors include: tobacco use, hypercholesterolemia, HTN, male gender, family history, and advanced age. Other rare causes include infection, such as tertiary syphilis (which leads to aneurysmal dilation in the aortic root/ascending aorta), after blunt chest trauma (usually resulting in pseudoaneurysms), patients with connective tissue diseases (such as Marfan syndrome and Ehlers-Danlos), and in arteritis. Although true aneurysms can develop anywhere along the aorta, 75% are AAAs.

ABDOMINAL AORTIC ANEURYSM

36. What are common presenting signs and symptoms of an abdominal aortic aneurysm (AAA)?

Most patients with AAAs are asymptomatic, and their aneurysm is found incidentally on physical examination or on diagnostic studies done for other reasons. It is estimated that 2% to 3% of men older than 50 years of age have an occult AAA. Approximately 75% of aneurysms $>$5 cm can be palpated, but only 5% to 10% of patients with an AAA have an abdominal bruit. The classical triad of ruptured AAA is pain, hypotension, and a pulsatile abdominal mass. Oftentimes the patients only have one or two of these symptoms and sometimes none. A patient with an acutely expanding or ruptured aneurysm will likely complain of constant abdominal pain, radiating to the back, flank, chest, thigh, inguinal area, or scrotum. It is often described as dull, throbbing, or colicky. Hypotension, syncope, or low hematocrit may signify significant blood loss.

37. What should I do when I suspect a ruptured AAA?

Place two large-bore IVs, type, and cross-match for at least six units of packed red blood cells, and call a vascular surgeon. The goal should be to get the patient to the operating room (OR) as soon as possible, and transport should not be delayed for definitive studies or to attempt full resuscitation in the ED. A bedside ultrasound can be done quickly to screen for an AAA. The ultrasound can confirm AAA but rarely detects rupture because most AAAs rupture into the retroperitoneum. CT scans are only appropriate in hemodynamically stable patients, but have a

100% sensitivity for detecting AAA and 77% to 100% sensitivity for picking up retroperitoneal bleeding. The CT can be performed without contrast if there is concern over the patient's kidney function. The mortality for elective repair of an unruptured AAA is approximately 5% as opposed to a greater than 50% mortality associated with acute repair of an already ruptured AAA.

38. Discuss the dilemmas behind aggressive fluid resuscitation in a hypertensive patient with ruptured AAA.

Unfortunately, no prospective studies exist to guide optimal fluid resuscitation, and the appropriate amount of volume to give is controversial. Allowing some degree of hypotension may slow bleeding, allow some clot formation and temporarily tamponade the bleeding; too much fluid may have the opposite effect and may also cause an increased blood pressure and a dilutional coagulopathy, further increasing bleeding. On the other hand, these patients likely have other comorbid conditions that could be potentially fatal in the presence of prolonged hypotension. The goal should be to maintain adequate perfusion to the vital organs, using warm saline/blood without intentionally raising the BP above 90 to 100 mm Hg systolic.

39. What common diseases may mimic ruptured AAA?

Everything! Renal colic, syncope attributed to other causes, pancreatitis, perforated peptic ulcer, AMI, gallbladder pathology, diverticulitis, appendicitis, perforated viscus, bowel obstruction, musculoskeletal back pain, and intestinal ischemia. Thus, always consider this diagnosis in the patients older than 50 years of age with any one of the symptoms in the classic triad: pain, hypotension, and pulsatile mass.

40. When do you need to worry about an AAA?

The risk of rupture is minimal for an AAA measuring <4 cm, but the risk increases dramatically at diameters larger than >6 cm. The annual rupture risk for AAA 6 to 7 cm is 10% to 20%, from 7 to 8 cm 20% to 40% and, if >80 cm, the annual rupture risk is increased to 30% to 50%. Rapid expansion is the greatest predictor of impending rupture, and routine screening with known AAAs is important because it significantly affects mortality. All patients with an AAA 5 cm or greater in diameter should have vascular surgery follow-up.

41. When should a symptomatic unruptured AAA be repaired?

This represents a dilemma. It has been found that the mortality associated with emergency repair of symptomatic AAA is associated with about a 25% mortality as opposed to a 5% mortality associated with semielective repair. The question is the risk of rupture in the interim.

42. List some atypical symptoms that may be related to the presence of an AAA.

- Gastrointestinal bleeding in a patient with previous aortic repair may signify fistula formation between the wall of the aorta and the small or large bowel.
- A large aneurysm may cause mass effect on surrounding structures resulting in a bowel or ureteral obstruction.
- Radicular pain may occur if the bleeding is retroperitoneal. Leg ischemia may occur due to peripheral embolization of mural plaques.

43. How are AAAs surgically repaired?

Traditional repair involves laparotomy and cross-clamping the aorta. A newer, less invasive approach involves placement of a self-expanding stent graft via the femoral artery under fluoroscopic guidance. This approach allows repair of AAA in a group of patients that would not be candidates for AAA repair.

44. What are the complications of endovascular aortic repair (EVAR)?

Endovascular stent graft repair is an alternative to surgical repair. The short-term outcomes seem equal or favorable to open repair. However, the longer-term mortality of patients who have undergone EVAR seem to be equal to the open approach. Additionally, there is a higher

reintervention rate and AAA rupture rate in the EVAR group. Many of the complications of EVAR are similar to the complications of open surgical repair. Certain complications have decreased in occurrence with the development of the technique and advance in materials used. Complications include:

- Graft infection, that can lead to aorto-enteric fistula (AEF) formation, commonly presenting with upper gastrointestinal bleed
- Limb occlusion
- Device migration
- Continued sac expansion (endotension)
- Endoleak is the most frequent complication and occurs in up to one fourth of all patients who have undergone EVAR. Some types of endoleaks place the patient at a higher risk of AAA rupture.

KEY POINTS: ABDOMINAL AORTIC ANEURYSMS

1. The triad is abdominal pain, pulsatile mass, and hypotension.
2. Do not wait for a definitive study before calling surgery.
3. Bedside ultrasound is an excellent screening tool for AAA.
4. CT is the gold standard for making the diagnosis of a ruptured AAA.

45. Summarize the common parenteral antihypertensive medications and their indications and contraindications.

See Table 31-1.

TABLE 31–1. PARENTERAL ANTIHYPERTENSIVE MEDICATIONS

Drug	Dose	Onset	Duration	Indications	Contraindications
Nitroprusside	0.3–10 μg/kg/min IV	1–2 min	1–2 min	CHF, aortic dissection, catecholamine excess, hypertensive encephalopathy	Pregnancy, AMI, hepatic or renal insufficiency, caution with increased ICP
Nitroglycerin	10–100 μg/min IV	2–5 min	3–5 min	AMI, CHF	CVA, ARF
Nicardipine	5–15 mg/hr IV	15 min	6 hours	AMI, ARF, eclampsia, hypertensive encephalopathy, catecholamine excess	CHF, second- or third-degree AVB

Continued

TABLE 31–1. PARENTERAL ANTIHYPERTENSIVE MEDICATIONS—cont'd

Drug	Dose	Onset	Duration	Indications	Contraindications
Fenoldopam	0.1–1.7 μg/kg/min IV	5–15 min	1–4 hours	AMI, CHF, ARF, aortic dissection, hypertensive encephalopathy, catecholamine excess	Glaucoma (can cause increased IOP)
Hydralazine	10–20 mg IV bolus; repeat every 4–6 hours prn (max 40 mg)	10–20 min	3–8 hours	Eclampsia	AMI, CVA, aortic dissection
Esmolol	500 μg/kg IV bolus over 1 min, then 50–300 μg/kg/min	1–2 min	10–20 min	CAD, aortic dissection	CHF, second- or third-degree AVB
Labetalol	20 mg IV bolus, then 40–80 mg every 10 min up to 300 mg or 2 mg/min IV	2–10 min	2–4 hours	CAD, aortic dissection, hypertensive encephalopathy, eclampsia	CHF, second- or third-degree AVB, asthma
Phentolamine	5 mg IV, repeat prn (max 20 mg)	1–2 min	10–30 min	Catecholamine excess	AMI

AMI, acute myocardial infarction; ARF, acute renal failure; AVB, atrioventricular block; CAD, coronary artery disease; CHF, congestive heart failure; CVA, cerebrovascular accident; ICP, intracranial pressure; IOP, intraocular pressure; IV, intravenously; prn, as needed.

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PERICARDITIS AND MYOCARDITIS

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CHAPTER 32

PERICARDITIS

1. Describe a normal pericardium.
The pericardium is 1 to 2 mm thick and envelops the heart. It has two layers. Between the two layers is the pericardial space, which normally contains 25 to 50 mL of fluid.

2. What is pericarditis?
Inflammation of the pericardium.

3. What causes pericarditis?
Infectious agents, such as viruses and bacteria, can cause pericarditis as a result of direct spread of infection to the pericardium. Pericarditis also may be caused by the antibody-mediated autoimmune reaction that occurs 2 to 4 weeks after a viral illness (see Table 32-1). This postviral pericarditis, termed *idiopathic* because a viral source has

TABLE 32–1. CAUSES OF PERICARDITIS

Infectious	Immunologic Mediated Diseases	Trauma	Drugs	Other
Viral: Coxsackie B Echovirus HIV	Autoimmune disorders	Blunt	Procainamide	Sarcoidosis
Bacterial:	Acute rheumatic fever	Penetrating	Cromolyn sodium	Amyloidosis
Staphylococcus				
Tuberculosis				
Fungal	Rheumatoid arthritis	Postcardiac injury syndrome	Hydralazine	Uremia
Parasitic	Connective tissue diseases	Postpericardiotomy		Radiation
Rickettsia	Lupus erythematosus			Neoplasm
	Postinfarction			Aortic dissection

not been isolated, is probably the most common form of pericarditis. An autoimmune reaction to cardiac antigens may occur after cardiac instrumentation or acute myocardial infarction (MI). The likelihood of postinfarction pericarditis is reduced by half (from approximately 12% to 6%) when a thrombolytic agent is used.

4. Who is most susceptible to infectious pericarditis?

Viral and idiopathic pericarditis occur most commonly in healthy persons between 20 and 40 years old. Bacterial pericarditis occurs in patients with a bacterial infection of the lungs, endocardium, or blood. Patients with HIV are susceptible to pericarditis caused by opportunistic infections.

5. Describe the clinical presentation of pericarditis.

The most common symptom is chest pain, often described as midline and sharp. The pain is generally worse with movement and breathing, and relief is obtained from sitting up and leaning forward. It may radiate to the neck, back, or left shoulder. Dyspnea, malaise, and fever may occur. The pathognomonic clinical finding is a friction rub, which is a scratchy noise, similar to creaking leather. The optimal patient position for a rub to be auscultated is sitting up, leaning forward, and in full expiration. The diaphragm of the stethoscope should be pressed firmly to the chest at the lower left sternal border. A little luck may be needed to detect a rub because it occurs intermittently.

6. How does the electrocardiogram (ECG) appear in pericarditis?

The ECG typically evolves through four stages:

- **In stage 1,** The first hours to days of illness may show ST segment elevation and PR segment depression in all leads except aVR and V_1, in which reciprocal changes occur.
- **In stage 2,** the ST and PR segments normalize, and the T waves flatten.
- **In stage 3,** deep T-wave inversion occurs.
- **In stage 4**, the ECG reverts to normal. Occasionally, stage 4 does not occur, which results in permanent generalized or focal T-wave inversions and flattenings. The ST segment displacement seen in stage 1 is attributed to the associated subepicardial myocarditis, whereas the PR segment depression is attributed to subepicardial atrial injury.

7. How can acute pericarditis be distinguished from acute MI?

ST segment elevations in stage 1 of acute pericarditis tend to be upwardly concave rather than convex, and simultaneous T-wave inversions are not typically seen. The progression to T-wave inversions in stage 2 tends to occur after the ST segments have returned to baseline, whereas in acute MI, the T-wave inversion is more likely to accompany ST segment elevation. The ST segment elevations in acute pericarditis typically are diffuse as opposed to an anatomic distribution, which is more likely to be seen in the setting of an acute MI.

Patients with acute pericarditis are more likely to be younger, to be otherwise healthy, and to have a history of a preceding viral illness and pleuritic-type chest pain. Patients with acute MI are more likely to be older with risk factors for coronary artery disease. Ventricular arrhythmias are not associated with isolated pericardial disease and suggest the presence of underlying cardiac disease.

8. How can acute pericarditis be distinguished from musculoskeletal chest pain?

Musculoskeletal chest pain generally is not relieved by sitting up, and the characteristic friction rub and ECG abnormalities of pericarditis are not present.

9. Is pericardial effusion a concern in patients with pericarditis?

Yes. Pericardial effusion occurs most commonly in patients with acute viral or idiopathic, neoplastic, postradiation, or posttraumatic pericarditis. Its effects range from insignificant to life-threatening if tamponade occurs.

10. How much pericardial effusion is significant?

The answer depends entirely on the clinical situation. A patient with a stab wound to the heart may be able to accommodate only 80 to 200 mL of pericardial fluid before tamponade develops. Patients with long-standing pericardial fluid collections may tolerate 2,000 mL or more without hemodynamic compromise.

11. How can a pericardial effusion be diagnosed?

The physical examination is unreliable in detecting or excluding a pericardial effusion. Similarly, the cardiac silhouette is not enlarged on chest radiograph until at least 250 mL of fluid has accumulated. **Echocardiography** has excellent sensitivity and specificity; it can detect as little as 15 mL of pericardial fluid.

12. What is cardiac tamponade?

Cardiac tamponade exists when accumulating pericardial fluid leads to increased pericardial pressure to the point that it prevents the atria and ventricles from filling adequately during diastole, decreasing the volume of blood available to be pumped during systole and causing hemodynamic compromise. Although any form of pericarditis may lead to cardiac tamponade, acute tamponade usually is caused by trauma. Subacute tamponade occurs most commonly in neoplastic pericarditis.

13. How is cardiac tamponade diagnosed?

The first step is to confirm the presence of a pericardial effusion by echocardiography. Absence of a pericardial effusion rules out cardiac tamponade. If an effusion is present, a combination of physical examination and echocardiographic findings can confirm the diagnosis of tamponade. Physical examination findings suggestive of tamponade include tachycardia, hypotension, cyanosis, dyspnea, jugular venous distention, pulsus paradoxus, and elevated central venous pressure (>15 mm Hg). Echocardiographic findings are more specific and develop sequentially as pericardial pressure increases: right atrial collapse, right ventricular collapse, and bowing of the interventricular septum. Another helpful finding is to perform the *sniff test*. Instruct the patient to inhale quickly through the nose while the ultrasonographer visualizes the inferior vena cava. Incomplete collapse of the inferior vena cava correlates well with elevated central venous pressure measurements.

14. What is pulsus paradoxus?

An abnormally large (>10 mm Hg) drop in the systolic blood pressure with inspiration. Kussmaul termed this phenomenon *paradoxical* because of the disappearance of the pulse during inspiration when the heart was obviously beating. Pulsus paradoxus is a pulse, not a pressure, change and is an exaggeration of the normal inspiratory fall in arterial flow and systolic pressure. Inspiration favors right-sided heart filling by decreasing pericardial pressure, whereas expiration favors left-sided heart filling. Pulsus paradoxus usually signals large reductions in ventricular volumes and equilibration of mean pericardial and all cardiac diastolic pressures. The detection of pulsus paradoxus on physical examination suggests (and may be one of the earliest clues to) the existence of cardiac tamponade.

15. What is the appropriate ED management of pericarditis?

Anti-inflammatory agents, such as indomethacin (Indocin), 25 to 75 mg four times a day; aspirin, 650 mg every 3 to 4 hours; or ibuprofen, 600 mg four times a day, should be administered. The use of corticosteroids is controversial. Although corticosteroids are effective anti-inflammatory agents, 10% to 20% of patients develop recurrent pericarditis as tapering occurs. Echocardiography is indicated to rule out pericardial effusion. If cardiac tamponade is present, percutaneous pericardiocentesis should be performed to relieve intracardiac pressure. Intravenous fluids should be infused rapidly to increase arterial pressure and cardiac output.

16. What is the prognosis for patients with pericarditis?

Most patients recover fully, although 15% to 20% have a recurrence, probably because of an autoimmune mechanism. Nonsteroidal anti-inflammatory drugs are used for recurrences. If these agents are ineffective, corticosteroid therapy is initiated. Colchicine holds promise as an adjunctive therapy in recurrent pericarditis. If medical interventions fail, pericardiectomy usually is performed.

MYOCARDITIS

17. What is myocarditis?

An inflammation of the myocardium in the absence of ischemia.

18. What causes myocarditis?

In the **United States**, myocarditis is caused most commonly by viruses. Enteroviruses, especially the Coxsackie B virus, predominate as causative agents. Infectious agents cause myocardial damage by three basic mechanisms:

a. Direct invasion of the myocardium
b. Production of a myocardial toxin (e.g., diphtheria)
c. Immunologically mediated myocardial damage. The immunologically mediated destruction of cardiac tissue from infiltration of host cellular immune components is probably the more common mechanism in adults, whereas in neonates, damage from direct viral invasion is more likely.

Worldwide, Chagas disease is the leading cause of myocarditis. Other organisms that are known to infiltrate the myocardium include influenza A and B, adenovirus, hepatitis A and B, tuberculosis, *Chlamydia pneumoniae*, *Borrelia burgdorferi* (Lyme disease), *Legionella pneumophila*, cytomegalovirus, *Toxoplasma gondii*, *Trichinella spiralis*, and *Corynebacterium diphtheriae*.

19. When should a diagnosis of myocarditis be considered in the ED?

Diagnosing myocarditis in the ED can be a challenge, and because the presenting symptoms and signs are typically nonspecific, this is often a diagnosis of exclusion. Nonspecific symptoms include fatigue, myalgias, nausea, vomiting, fever, dyspnea, palpitations, and precordial discomfort. Chest pain may reflect associated pericarditis. Patients may present with a dilated cardiomyopathy without evidence of ischemia or valvular disease. Myocarditis probably should be considered in any previously healthy person who develops dyspnea, orthopnea, decreased exercise tolerance, palpitations, or syncope when no other obvious cause is found. Patients should be asked about concomitant or recent upper respiratory or gastrointestinal illness.

20. What clinical findings may be present?

Tachycardia is common and can be disproportionate to the temperature or apparent toxicity. This may be the only clue that something more serious than a simple viral illness exists. Clinical evidence of congestive heart failure occurs only in more severe cases. A pericardial friction rub may be auscultated if myopericarditis is present. Complications of myocarditis include ventricular arrhythmias and left ventricular aneurysms.

21. Are there any chest radiograph or ECG abnormalities?

- The **chest radiograph** may be normal or abnormal, depending on the extent of disease. The cardiac silhouette may be enlarged, which can be due to a dilated cardiomyopathy or a pericardial effusion.
- The **ECG** commonly shows a sinus tachycardia and may show low electrical activity. Nonspecific ST segment and T-wave abnormalities, a prolonged corrected QT interval, atrioventricular block, or acute MI pattern also may occur. Atrial arrhythmias have been described.

22. How is myocarditis diagnosed?

Making the diagnosis clinically can be difficult. Endocardial biopsy is considered the gold standard, although it has highly variable sensitivity and specificity. In contrast to patients with pericarditis, cardiac enzymes frequently are elevated in patients with myocarditis. White blood cell count and erythrocyte sedimentation rate may be elevated but are nonspecific. Indium-111 antimyosin antibodies show myocardial necrosis by binding to exposed myosin in damaged myocardial cells. When myocarditis is suspected clinically, indium-111 antimyosin imaging may be helpful. Viral titers have been suggested but have a low yield. Echocardiography often shows global dysfunction that does not correspond to a specific coronary artery distribution.

23. How can acute myocarditis be distinguished from acute MI?

Myocarditis occurs primarily in young healthy patients without significant cardiac history or risk factors for coronary artery disease. Chest pain, dyspnea, ECG abnormalities, and cardiac enzyme elevation may occur in both conditions. In the ED, it may be impossible to distinguish between these two entities, in which case treatment for acute MI should be initiated.

24. Is myocarditis a concern in AIDS?

Yes. The incidence of myocarditis found at autopsy of AIDS patients has been reported as high as 52%, compared with less than 10% in the population as a whole. The increased risk of myocarditis in patients with AIDS may be due to an abnormal autoimmune reaction, opportunistic infections, or HIV itself.

25. In what other clinical situations should myocarditis be considered?

Myocarditis and dilated cardiomyopathy have been associated with cocaine use. Myocarditis is a common autopsy finding in patients who have died from cocaine abuse.

26. Describe the appropriate ED management of a patient with myocarditis.

The current recommended treatment consists of supportive therapy. The only uniformly accepted beneficial therapy is bed rest. All patients with suspected myocarditis should be admitted to a monitored bed in the hospital. Antibiotics are appropriate when a bacterial cause is suspected. Dilated cardiomyopathy is treated with diuresis, afterload reduction, and digoxin. In severe cases, temporary pacing and external circulatory support may be needed. Patients with a fulminant clinical course may require cardiac transplantation. Immunosuppressive therapy has been studied, but controlled studies have not established efficacy. High-dose gamma globulin has been studied and may be associated with improved left ventricular function and better survival during the first year after initial presentation.

27. What is the prognosis for patients with acute myocarditis?

Mortality for patients with myocarditis has been reported to be 20% at 1 year and 56% at 4 years, although many patients do recover completely.

KEY POINTS: PERICARDITIS AND MYOCARDITIS

1. The physical examination or chest radiography is neither sensitive nor specific for pericardial effusion; echocardiography is the gold standard.
2. Myocarditis should be considered in patients with significant tachycardia that cannot otherwise be explained or in any patient with the combination of viral symptoms and evidence of cardiac disease.
3. Viruses are the most common causes of pericarditis and myocarditis, and a history of preceding or concurrent viral illness is quite common.
4. Myocarditis is very common in AIDS patients, with rates at autopsy as high as 52%.

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ESOPHAGUS AND STOMACH DISORDERS

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1. How are gastrointestinal (GI) problems differentiated from acute myocardial infarction?

Esophageal or gastric pain can present with visceral-type chest pain (e.g., ache, pressure) or upper abdominal pain and nausea that are difficult to differentiate from pain and nausea related to myocardial ischemia or infarction. Description of the pain, determination of cardiac risk factors, and appropriate use of an electrocardiogram (ECG) in adult patients with visceral-type pain or cardiac risk factors will minimize clinical errors. Nitroglycerin, antacids, and GI cocktails are therapeutic interventions, not diagnostic tests. Patients with esophageal spasm may respond to nitroglycerin and antacids, or GI cocktails may provide a placebo-like benefit to patients with cardiac ischemia. The response to these interventions can mislead the unwary physician.

2. What is a GI cocktail?

The two most commonly used GI cocktails contain antacids (30 mL), viscous lidocaine (10 mL), and either Donnatal (10 mL) or dicyclomine (Bentyl; 20 mg). These cocktails may provide temporary symptomatic relief of minor esophageal and gastric irritation. Of note, it has been concluded that these cocktails are no more effective than antacids alone.

3. What is heartburn?

Retrosternal burning discomfort that may radiate to the sides of the chest, neck, or jaw. The description of the pain may be similar to the pain of cardiac ischemia. Heartburn is characteristic of reflux esophagitis and often is made worse by bending forward or lying recumbent after meals. It may be relieved by upright posture, liquids (including saliva or water), or more reliably, antacids. Heartburn is probably due to heightened mucosal sensitivity to acid and can be reproduced by infusion of dilute hydrochloric acid into the esophagus (Bernstein test).

4. How is reflux esophagitis treated?

In addition to antacids, general measures include elevation of the head of the bed (e.g., 4 inches), weight reduction, and elimination of factors that increase abdominal pressure. Patients should avoid alcohol, chocolate, coffee, fatty foods, mint, orange juice, smoking, ingestion of large quantities of food and drink, and certain medications (e.g., anticholinergics or calcium channel blockers). Antacids after meals, H_2-blockers (e.g., cimetidine) before bedtime or daily proton pump inhibitors (e.g., omeprazole) are often helpful. Treatment is usually for 1 to 2 months, and the disease may recur.

5. What are the esophageal causes of odynophagia?

Odynophagia, or painful swallowing, is a characteristic of nonreflux esophagitis. Infectious esophagitis is a common cause and usually occurs in immunocompromised patients, and it can be due to fungal (e.g., monilial), viral (e.g., herpes, cytomegalovirus), bacterial (e.g., *Lactobacillus*, β-hemolytic streptococci), or parasitic organisms. Other types of nonreflux esophagitis include radiation, corrosive, pill-induced, and certain systemic diseases (e.g., Behçet's, Crohn's, pemphigus vulgaris, Stevens-Johnson

syndrome). Odynophagia is unusual in reflux esophagitis but may occur with a peptic ulcer of the esophagus (Barrett's ulcer).

6. How does esophageal obstruction present?

Except in infants, there is usually a history of eating or swallowing something that is followed by the onset of chest pain, odynophagia, or inability to swallow. Foreign bodies usually lodge at one of four locations: cervical esophagus, upper esophageal sphincter, aortic arch, and lower esophageal sphincter. Obstruction by food may occur wherever there is narrowing of the lumen because of stricture, carcinoma, or a lower esophageal ring. Foreign bodies, especially those that are sharp, or impacted food are best removed endoscopically. Round, blunt objects may be removed using a Foley catheter, a procedure most often done under fluoroscopy. Meat tenderizer should not be used to facilitate passage of obstructed meat. Glucagon, 0.5 to 2 mg intravenously, may relieve distal esophageal food obstruction in a minority of patients.

7. What is Mallory-Weiss syndrome?

Mallory-Weiss syndrome is a mucosal tear that usually involves the gastric mucosa near the squamocolumnar mucosal junction; it also may involve the esophageal mucosa. It usually is caused by vomiting and retching. Patients with a Mallory-Weiss tear may present with upper GI bleeding.

8. What causes esophageal perforation, and how is it diagnosed and treated?

Esophageal perforation, a true emergency, can be caused by iatrogenic damage during instrumentation, trauma (most often penetrating), increased intraesophageal pressure associated with forceful vomiting (Boerhaave's syndrome), or diseases of the esophagus (e.g., corrosive esophagitis, ulceration, neoplasm). Esophageal perforation causes chest pain that is often severe and may be worsened by swallowing and breathing. Chest radiograph may reveal air within the mediastinum, pericardium, pleural space (pneumothorax), or subcutaneous tissue, pleural effusion, or may appear normal. Esophageal perforation may lead to leakage of gastric contents into the mediastinum and secondary infection (i.e., mediastinitis). The diagnosis is confirmed radiographically by swallow and leakage of radiopaque contrast material. Treatment includes broad-spectrum antibiotics, gastric suction, and surgical repair and drainage as soon as possible.

9. What are causes of abdominal pain that are gastric or duodenal in origin?

An estimated 10% of cases of abdominal pain seen in the ED are due to gastric or duodenal disease. Gastritis and peptic ulcer disease (PUD; ulcer of the stomach or duodenum resulting from gastric acid) account for most patients with abdominal pain secondary to gastric or duodenal disease. Perforated PUD and gastric volvulus are the two most serious conditions requiring immediate diagnosis and treatment.

10. What are the common causes of gastritis and PUD?

Gastritis is associated with alcohol, salicylates, nonsteroidal anti-inflammatory drugs (NSAIDs), and hiatal hernia. PUD is related to family history, associated diseases (e.g., chronic obstructive pulmonary disease [COPD], cirrhosis, or chronic renal failure), male gender, advanced age, and smoking. The use of certain drugs, such as aspirin or NSAIDs, may be related to PUD, but diet (e.g., caffeine and spicy foods) and alcohol are not. *Helicobacter pylori* has been shown to be a frequent cause of duodenal ulcers. First-line treatment for patients with *H. pylori* is the combination of a proton pump inhibitor (PPI), clarithromycin, and amoxicillin.

11. How does perforated PUD present?

Perforated PUD (and gastric volvulus) presents with sudden onset of abdominal pain that may or may not be related to eating. It often radiates to the back but also may radiate to the chest or upper abdomen. The pain is usually steady and refractory to antacids. Vomiting is present in approximately 50%.

On **physical examination**, patients appear in acute distress and often have tachycardia. Blood pressure may be elevated secondary to pain or decreased secondary to extensive fluid loss from generalized peritonitis. Patients usually lie still and avoid movement. Involuntary guarding, rebound tenderness, and abdominal rigidity are common. Bowel sounds are usually absent or significantly decreased.

Laboratory work may reveal nonspecific leukocytosis; 40% have a white blood cell count >14,000 white blood cells/mm^3. If vomiting has been protracted, hypochloremic, hypokalemic, or metabolic alkalosis may be seen. A small percentage of patients have mild elevation in amylase or lipase. Free air is present on upright chest radiograph or abdominal left lateral decubitus view in more than 70% of patients.

12. How should a patient suspected of having a perforated ulcer be managed?

Patients with severe abdominal pain should be undressed, placed on cardiac and SaO_2 monitors, and have a large-bore intravenous catheter placed for fluid resuscitation with crystalloid (e.g., normal saline). Patients with oxygen saturation <93% should be given supplemental oxygen. A prompt but thorough physical examination should be done, including pelvic and rectal examinations. Blood should be drawn for complete blood count, electrolytes, blood urea nitrogen (BUN), creatinine, lipase, and type and screen. An ECG should be obtained on patients older than 40 years. Urine should be obtained for urinalysis and a pregnancy test should be performed for all menstruating females. Patients should have nothing by mouth (NPO). A portable upright chest radiograph or abdominal left lateral decubitus view often helps to show free intraperitoneal air. A nasogastric (NG) tube should be placed after anesthetizing the nasopharynx and prompt surgical consultation obtained. Broad-spectrum antibiotics should be given and the patient prepared for emergency laparotomy. Intravenous analgesics (opiates) should be given for patient comfort.

13. What differentiates upper from lower GI hemorrhage?

Upper GI hemorrhage is bleeding that is proximal to the ligament of Treitz, and lower GI bleeding is distal. In the ED, this is evaluated by placement of a nasogastric (NG) or orogastric tube and aspiration of gastric and proximal duodenal contents. Physical appearance of the aspirate (coffee grounds, red-tinged fluid, or fresh blood) is the best way of determining the presence of significant upper GI bleeding. While generally reliable, false-negative and false-positive results may occur when using Gastroccult cards.

14. Do all patients presenting with only lower GI bleeding require NG tube placement?

The routine use of NG aspiration (NGA) has been advocated to rule out occult upper GI bleeding. However, recent studies have added some controversy to this practice. NG tube placement is a painful procedure that is not without possible complications. Reports state that 10% of patients presenting with only hematochezia will have positive NGA and occult upper GI bleeding. Some conclude that in low-risk patients, NGA has a low yield and is almost as likely to yield false-positive as true-positive results.

15. How is a patient classified as low risk for having occult upper GI bleeding?

It has been demonstrated that male sex, history of GI tract bleeding, black stool, hematocrit <30%, BUN/creatinine ratio = 30, and age <50 years were all independently associated with upper GI bleeding. Three factors, BUN/creatinine ratio, black stool, and age, were shown to be strong predictors and correlated well with NG aspiration. Patients with no risk factors are considered low risk. Nasogastric aspiration is mandatory in all patients presenting with any two of these three factors and those who are hemodynamically unstable.

16. List the causes of upper GI bleeding.

- PUD (45%)
- Gastric erosions (23%)
- Varices (10%)
- Mallory-Weiss tear (7%)
- Esophagitis (6%)
- Duodenitis (6%)

17. Discuss the emergency management of upper GI bleeding.

Management begins with a rapid assessment and management of the patient's airway, breathing, and cardiovascular status. Patients should be undressed, placed on cardiac and SaO_2 monitors, and if SaO_2 <93% (at sea level), given supplemental oxygen. The history of GI bleeding (i.e., vomiting blood or passing black or bloody stool) is sufficient to lead to the placement of a large-bore, peripheral intravenous catheter with infusion of normal saline. A focused physical examination should be done, checking for signs of shock (e.g., altered mental status, tachycardia, hypotension, cool extremities, and delayed capillary fill). Patients who have abnormal vital signs or signs of shock should have two or more intravenous lines placed and be given rapid infusion of crystalloid. The evaluation should include testing of stool for blood. During the initial examination and resuscitation, a history should be obtained. Blood should be drawn for type and cross-matching, hematocrit, platelet count, prothrombin time, electrolytes, BUN, and creatinine. Elderly patients, patients with a history of cardiovascular disease or chest pain, and patients who are severely anemic should have an ECG to evaluate for signs of cardiac ischemia (i.e., ST depression). An upright chest radiograph should be obtained to rule out subdiaphragmatic air or pulmonary aspiration. A nasogastric or orogastric tube should be placed to determine the presence of blood in the stomach and then removed.

18. What happens to patients with GI hemorrhage?

GI bleeding usually stops spontaneously, and no further ED management is necessary other than admission and perhaps transfusion if there is significant anemia (i.e., hematocrit <25%). In 20% or less of patients, continued GI hemorrhage requires further management and treatment.

19. How do I facilitate the placement of an NG tube?

Applying an anesthetic spray to the nose and posterior pharynx or having the patient breathe nebulized 4% lidocaine decreases the discomfort of placing an NG tube.

20. How should a patient with continued GI hemorrhage be managed?

Blood replacement should begin in patients who continue to show signs of shock or cardiovascular instability. Surgery and gastroenterology consultation should be initiated quickly. Patients who do not respond promptly (i.e., remain hypotensive) to a 30 mL/kg infusion of crystalloid should be given O-negative blood if type-specific blood is not yet available. Cross-matched blood usually takes approximately 45 to 60 minutes to become available. Upper GI bleeding can often be stopped endoscopically, but emergency operative repair may be required in patients with persistent GI bleeding.

21. Is placement of a nasogastric or orogastric tube contraindicated in someone with esophageal varices?

There is no evidence that a properly placed NG or orogastric tube results in a significantly increased risk of tearing varices or increased size of a Mallory-Weiss tear. NG or orogastric tubes can perforate the esophagus or posterior pharynx if they are placed too aggressively. Diagnostic NG or orogastric tubes are unnecessary if the patient vomits gastric contents in the ED because this may be inspected for the presence of blood.

22. When should gastric lavage be used in patients with upper GI bleeding?

Gastric lavage is necessary only in patients who have no aspirate after the NG tube is placed. Regular tap water is used for lavage; the fluid need not be saline or sterile.

23. Should all patients with upper GI bleeding undergo endoscopy?

Endoscopy is the most accurate diagnostic tool available for the evaluation of patients with upper GI bleeding. Endoscopy will identify a lesion in 78% to 95% of patients if it is done within 12 to 24 hours of hemorrhage. Accurate identification of the bleeding site allows risk stratification with respect to predicting rebleeding and mortality. Risk stratification facilitates a proper disposition decision.

24. What are the low-risk criteria that allow a patient who is complaining of GI bleeding to be sent home?

- No comorbid diseases
- Normal vital signs
- Normal or trace positive stool guaiac
- Negative gastric aspirate, if done
- Normal or near-normal hemoglobin and hematocrit
- Proper understanding of signs and symptoms of significant bleeding
- Good home support
- Follow-up arranged within 24 hours
- Immediate access to emergent care, if needed

KEY POINTS: ESOPHAGUS AND STOMACH DISORDERS

1. Epigastric pain may be due to myocardial ischemia, so an ECG should be obtained in adult patients with epigastric discomfort, visceral-type pain, or cardiac risk factors.
2. Antacids often provide symptomatic relief of abdominal discomfort related to gastroesophageal disease.
3. *H. pylori* is a common, treatable cause of peptic ulcer disease.
4. Patients with upper GI bleeding who are hemodynamically unstable should receive rapid intravenous crystalloid infusion, urgent surgery, and gastroenterology consultation.

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CHAPTER 34

BOWEL DISORDERS

Vikhyat S. Bebarta, MD

1. When do you consider evaluating a patient for appendicitis?

Consider appendicitis in anyone presenting with abdominal pain. It can occur at any age, but is most prevalent in the teens and twenties. With the high prevalence of appendicitis in the population, atypical presentations are common. Appendicitis is one of the most commonly missed diagnoses in the ED, and it is the most common nonobstetrical emergency during pregnancy.

2. What is the pathogenesis of acute appendicitis?

The appendiceal lumen becomes obstructed, most commonly by a fecalith, leading to bacterial overgrowth and dilation of the appendix. Early on, the distended lumen causes dull, diffuse abdominal pain. As the inflammation progresses, a localized peritonitis develops, producing the classic right lower quadrant (RLQ) pain with involuntary guarding and rebound on physical examination.

3. How does appendicitis present clinically?

The classic presentation is nonspecific, umbilical abdominal pain that migrates over several hours to the RLQ of the abdomen. Associated symptoms include nausea, anorexia, and fever. However, variation of the appendix location leads to varied clinical presentations. For example, a retrocecal appendix may cause back or flank pain that can be mistakenly diagnosed as pyelonephritis or symptomatic nephrolithiasis. An extra long appendix with an inflamed tip may produce left lower quadrant pain. In pregnancy, the appendix is displaced into the right upper quadrant and, when inflamed, may be mistaken for symptomatic gallbladder disease. Other diagnoses of RLQ pain should also be considered (see Table 34-1).

4. Is the physical examination reliable in appendicitis?

Unfortunately, the classic physical examination findings of appendicitis—RLQ guarding and rebound, and positive psoas, obturator, or Rovsing's signs—are neither specific nor sensitive enough to accurately diagnose appendicitis. Standard laboratory test results may raise or lower your clinical suspicion, but only an abdominal computed tomography (CT) scan or direct visualization with surgery can reliably diagnose an inflamed appendix. Frequently, nonspecific RLQ pain and tenderness are the only clinical findings of appendicitis. Parenteral analgesia, such as morphine, may improve the physical examination for appendicitis.

5. What laboratory tests are helpful in evaluating RLQ pain?

Although no laboratory test is diagnostic of appendicitis, tests can aid in the evaluation of the patient and exclude other diagnoses:

- **White blood cell count**: >10,000 per mm^3 in approximately 90% of cases
- **Urinalysis**: To exclude urinary tract infection (However, mild pyuria or hematuria may be present when an inflamed appendix lies near the bladder or ureter.)
- **β-human chorionic gonadotropin:** To help exclude ectopic pregnancy

TABLE 34–1. DIFFERENTIAL DIAGNOSIS FOR RIGHT LOWER QUADRANT ABDOMINAL PAIN	
Acute ileitis	Inflammatory bowel disease
Diverticulitis	Acute cholecystitis
Perforated gastric or duodenal ulcer	Volvulus
Intussusception	Small bowel obstruction
Inflammation of Meckel's diverticulum	Uterine or tubo-ovarian pathology (e.g., tubo-ovarian abscess, ovarian torsion, ovarian cysts)
Incarcerated inguinal hernia	Ectopic pregnancy
Testicular torsion or epididymitis	Mittelschmerz
Mesenteric adenitis	Pyelonephritis, symptomatic nephrolithiasis

6. What radiologic study is best at imaging the appendix?

Abdominal CT is the imaging modality of choice for appendicitis. The scan is routinely done with intravenous and oral or rectal contrast enhancement. It has a reported accuracy of 93% to 98% in ruling in or out the diagnosis of appendicitis and is more sensitive and specific than any combination of physical examination and laboratory findings. Additionally, the CT scan may show other diseases responsible for the patient's symptoms.

Unenhanced CT (CT without contrast) has a sensitivity of 88% to 96% but is dependent on body habitus. Additional intraperitoneal fat improves sensitivity. However, CT scan has the risk of radiation and iodinated contrast. Ultrasound may be useful in children, pregnancy, and thin patients. It has a sensitivity of 88% to 94%, but the sensitivity is variable and is dependent on the patient's body habitus and the sonographer's and radiologist's experience. Ultrasound is useful to confirm a suspicion of appendicitis, but it is not useful to exclude it.

7. What is the treatment for appendicitis?

Appendectomy is the definitive treatment. Once appendicitis has been diagnosed, or is highly suspected, a surgical consult should be obtained. In a suspected case, start fluid resuscitation, pain control, and broad-spectrum antibiotics while waiting for surgery. A delay in diagnosis and treatment increases the perforation risk.

8. What is mesenteric ischemia?

Mesenteric ischemia is caused by insufficient blood supply to the intestines leading to tissue ischemia and infarction. The common causes are arterial emboli (most common) or thrombus, venous thrombosis, or nonocclusive hypoperfusion states. Patients should be assessed for risk factors of mesenteric ischemia (see Table 34-2).

TABLE 34–2. RISK FACTORS FOR MESENTERIC ISCHEMIA	
Age greater than 50 years	Recent myocardial infarction
Valvular or atherosclerotic heart disease	Dysrhythmias (e.g., atrial fibrillation)
Peripheral vascular disease	Critical illness with hypotension or sepsis
Congestive heart failure	Diuretics or vasoconstrictive drugs

9. How do patients with mesenteric ischemia present?

Patients complain of a diffusely painful abdomen. In the early stage, patients complain of severe pain but have minimal physical findings—the characteristic *pain out of proportion to the examination*. As infarction of bowel develops, peritoneal signs occur. Vomiting, hematochezia, hematemesis, abdominal distention, fever, and shock are late signs that often indicate infarcted bowel.

10. How do I diagnose mesenteric ischemia?

Diagnosing mesenteric ischemia can be difficult. The combination of clinical suspicion, radiographic imaging, and laboratory findings can help lead to the correct diagnosis. Direct surgical visualization of the bowel remains the gold standard. The abdominal CT with intravenous and oral contrast can show the location of the vascular occlusion and secondary findings consistent with ischemia, such as air within the bowel wall, intestinal wall thickening, and local inflammation. Laboratory findings may include leukocytosis; hemoconcentration; metabolic acidosis; and elevated phosphate, lactate, or lactate dehydrogenase. These lab findings may indicate ischemic bowel but lack sensitivity and specificity.

11. How is mesenteric ischemia treated?

Initial treatment includes vigorous resuscitation, parenteral antibiotics, correction of predisposing factors, and early surgical consultation. Definitive management involves selective vasodilator infusion, anticoagulation in venous occlusion, or embolectomy. Laparotomy is necessary for resection of necrotic bowel.

12. What is intussusception?

Intussusception occurs when an intestinal segment invaginates and telescopes into an adjacent segment. This is a disease predominately seen in children (see Chapter 63), but it can occur in adults. Typical pathologic lesions include tumors, Meckel's diverticulum, and inflammatory lesions. The high frequency of mass lesions in adults mandates surgical exploration.

13. What is IBD?

Inflammatory bowel disease (IBD) is an idiopathic, chronic inflammatory disease of the intestine. It encompasses two main groups:

- **Crohn's disease (CD).** CD is also known as *regional enteritis* or *granulomatous ileocolitis*.
- **Ulcerative colitis (UC).**

CD and UC are rising in incidence. Common clinical features are summarized in Table 34-3.

14. How do CD and UC present?

Although they are pathologically distinct diseases, CD and UC can present in a similar fashion and affect all age groups (see Table 34-3). Both diseases may present with diarrhea, abdominal pain, fever, anorexia, weight loss, and bloody diarrhea; however, UC is more likely to present with bloody diarrhea. In nonfulminating colitis, the diagnosis can be confirmed by endoscopy or barium enema.

15. What is the ED management for IBD?

Patients with mild disease and no signs of life-threatening complications can be treated as outpatients with close follow-up. Treatment usually consists of sulfasalazine, steroids (oral or rectal), steroid-sparing agents such as 6-mercaptopurine, antidiarrheal agents (e.g., loperamide, Lomotil, and cholestyramine), and analgesia. Antidiarrheal agents should be used with caution because they can predispose to toxic megacolon. Metronidazole may help treat the chronic perirectal complications of CD. Patients should be admitted if they have severe disease or any life-threatening complications. Extraintestinal manifestations of IBD can also occur (see Table 34-4).

TABLE 34–3. COMMON FEATURES FOR INFLAMMATORY BOWEL DISEASE

Clinical Feature	Crohn's Disease	Ulcerative Colitis
Weight loss	Common	Fairly common
Fever	Common	Fairly common
Diarrhea	Fairly common	Very common
Rectal bleeding	Fairly common	Very common
Perianal disease	Common	None
Site		
Colon	⅔ of patients	Exclusively
Ileum	⅔ of patients	None
Jejunum, stomach, or esophagus	Uncommon	None
Intestinal complications		
Stricture	Common	Unknown
Fistulas	Fairly common	None
Toxic megacolon	None	Unknown
Perforation	Uncommon	Unknown
Cancer	Fairly common	Common
Endoscopic findings		
Friability	Fairly common	Very common
Aphthous and linear ulcers	Common	None
Cobblestone appearance	Common	None
Rectal involvement	Fairly common	Very common
Radiologic findings		
Distribution	Discontinuous, segmental	Continuous
Ulceration	Deep	Superficial
Fissures	Common	None
Strictures for fistulas	Common	Rare
Ileal involvement	Narrowed, nodular	Dilated

Adapted from Podolsky DK: Inflammatory bowel disease. *N Engl J Med* 347:417–429, 2002.

16. Describe what happens during intestinal obstruction.

When the large and small bowels become obstructed, loss of the normal forward flow of digested food and secretions occurs. Proximal to the obstruction, a buildup of bowel gas, gastric secretions, and food develops. The bowel then becomes distended, causing pain, vomiting, and decreased oral intake. The cause of the obstruction can be mechanical or

TABLE 34–4. COMMON EXTRAINTESTINAL MANIFESTATIONS OF INFLAMMATORY BOWEL DISEASE

Clinical Category	Disorder
Ocular	Uveitis, episcleritis
Dermatologic	Erythema nodosum, pyoderma gangrenosum
Musculoskeletal	Ankylosing spondylitis, peripheral arthritis, sacroiliitis
Hepatobiliary	Cholelithiasis, pericholangitis, hepatitis, fatty liver, primary sclerosing cholangitis, cholangiocarcinoma, pancreatitis
Hematologic	Thromboembolic disease, chronic anemia
Renal	Nephrolithiasis, amyloidosis leading to renal failure

adynamic. Mechanical obstruction from adhesions or tumors frequently requires surgical intervention, whereas an adynamic ileus usually resolves spontaneously within a few days.

17. What are the common causes of mechanical small bowel obstruction (SBO)?

Overall, adhesions, hernias, and cancer account for more than 90% of mechanical SBO cases. **Postoperative adhesions** are the most common cause of an SBO (56%), followed by **incarcerated hernia** (25%) and **cancer** (10%). Other less common causes include IBD, gallstones, volvulus, intussusception, radiation enteritis, abscesses, congenital lesions, and bezoars.

18. What are the clinical features of SBO?

Patients present with diffuse abdominal pain, distention, and occasionally, vomiting. Early on, the pain is mild, crampy, and colicky in nature. An early SBO can be difficult to diagnose. The patient has pain but continues to have flatus and passage of some stool. As the obstruction progresses, the intestinal contents build up proximally, leading to nausea and vomiting. The intestine distal to the obstruction empties of stool and has decreased peristaltic motion leading to obstipation (inability to pass feces or flatus). Auscultation may reveal high-pitched, hyperactive *tinkling* or *rushing* sounds. Rectal examination may reveal impacted stool.

19. Describe the radiographic findings in SBO.

The classic finding on abdominal plain films is multiple air-fluid levels and distended loops of small bowel. When the obstructed intestine contains more fluid than gas, small round pockets of air may line up to form the *string of pearls* sign. A paucity of stool and gas is noted distal to the obstruction. Plain films have a sensitivity of 41% to 86% and a specificity of 25% to 88%; therefore, an early SBO may be missed by using only radiographs. Abdominal CT scan has a higher sensitivity (100%) and specificity (83%). Additionally, CT scan can show the location of the obstruction and help identify the cause (e.g., mass or infection such as appendicitis or diverticulitis).

20. What is the treatment for SBO?

The initial emergency management includes cardiopulmonary support, electrolyte replacement, decompression with a nasogastric tube, and intravenous fluids. Patients lose a large amount of fluid into the obstructed bowel and can be significantly intravascularly depleted. SBOs can often be managed nonoperatively with observation, intravenous fluid resuscitation, and bowel rest. However, some complete or mechanical obstructions require surgery. A surgical consultation is indicated while the patient is in the ED.

21. What are the characteristics of an ileus?

The terms *ileus* and *adynamic ileus* are synonymous for a paralyzed intestine. The bowel is unable to perform peristalsis. This is the most common cause of SBO. Causes of an ileus include infection (e.g., peritonitis), drugs (e.g., narcotics, anticholinergics), electrolyte imbalance (e.g., hypokalemia), spinal cord injuries, and recent bowel surgery. Patients present with abdominal distention, nausea and vomiting, and obstipation. Abdominal examination reveals hypoactive bowel sounds, mild tenderness, and absence of peritoneal signs. Radiographs usually show minimally distended bowel throughout the entire gastrointestinal (GI) tract, with diffuse air-fluid levels in the small bowel.

22. How is an ileus treated?

Management is similar to SBO. Limit oral intake, resuscitate with intravenous fluids, and correct electrolyte abnormalities, particularly hypokalemia. If abdominal distention is present, place a nasogastric or orogastric tube to decompress the stomach. Identify and limit the administration of medications, such as opioids, that slow intestinal motility. If the ileus is prolonged (>3–5 days), obtain additional imaging to search for an underlying cause.

23. What are the causes of large bowel obstruction (LBO)?

LBO is caused most commonly by colon cancer (60%), volvulus (20%), and diverticular disease (10%). Primary adenocarcinoma accounts for most cancerous lesions. Other less likely causes include metastatic carcinoma, gynecologic tumors, IBD, intussusception, and fecal impaction. In infants, consider congenital disorders, such as Hirschsprung's disease or an imperforate anus. Hernias and adhesions are uncommon causes of LBO.

24. What are diverticula and what are common complications?

Diverticula are sac-like outpouchings of the colon that occur through weakened areas of the muscularis of the colon wall. They commonly occur in persons of industrialized nations and increase in frequency with age. It is estimated that one third of the U.S. population will develop diverticula by age 50, and two thirds by 85 years. Complications from diverticula include bleeding and diverticulitis, a localized infection. Diverticulitis is caused by obstruction of the opening of diverticula, usually by stool, leading to infection from the proliferation of colonic bacteria and build-up of bowel secretions within the diverticula.

25. How does diverticulitis clinically present?

The most common symptom of diverticulitis is abdominal pain. The pain usually evolves over 1 to 2 days from dull, diffuse abdominal pain to more intense, localized left lower quadrant pain. Patients may complain of fever, nausea, vomiting, and decreased appetite. Diverticulitis occurs most frequently in the descending and sigmoid regions of the colon but can occur throughout the colon. The abdominal CT scan is the diagnostic procedure of choice and can show evidence for abscesses, bowel perforation, and severity of disease.

26. How do you manage diverticulitis?

Management consists of intravenous fluids, electrolyte replacement, parenteral analgesics, bowel rest, and broad-spectrum antibiotics. Patients with mild symptoms who are able to eat and have close follow-up can be managed as outpatients with oral antibiotics and close follow-up. Patients who have systemic or severe symptoms, older age, comorbidities, abscess, or bowel perforations require hospitalization, intravenous antibiotics, and serial examinations. Surgery may be required for repeat episodes or for bowel perforation. Abscess requires surgical or interventional radiology catheter drainage.

27. What are common causes of lower GI bleeding?

Patients frequently present to the ED with complaints of rectal bleeding. Lower GI bleeds occur from many causes, and a thorough history and examination are vital to diagnose the bleeding source. Investigating anatomically from the rectum proximally, evaluate for

hemorrhoids and rectal fissures, then, based on history and examination, consider diverticulosis, polyps, cancer, arteriovenous (AV) malformation, IBD, ischemic colitis, infectious diarrhea, and finally an upper GI source.

28. How do you perform anoscopy?

Anoscopy can provide a direct view of the anus and distal rectum. A lubricated anoscope with the obturator in place is advanced gently through the anal orifice. The obturator is removed to view the distal rectal mucosa; a light source is shined into the barrel of the anoscope, and the anoscope is withdrawn slowly while searching for internal hemorrhoids, fissures, abscess, masses, or bleeding proximal to the rectum.

29. What are hemorrhoids?

Hemorrhoids are engorged vascular cushions comprised of internal or external hemorrhoidal veins and present most often with bleeding, pain, or rectal itching. They are associated with prolonged increase in resting pressure in the anal canal, most often from constipation but also seen in pregnancy, excessive straining, and in certain occupations (e.g., truck driver).

30. How do internal and external hemorrhoids differ?

- **Internal hemorrhoids** arise above the dentate line, are covered by mucosa, and are not usually palpable or painful. They are seen during anoscopy and typically present as bright red blood in the toilet bowl or on toilet paper.
- **External hemorrhoids** are covered by skin and are easily visible and palpable at the anal orifice. A common complication of external hemorrhoids is thrombosis, which is painful and requires excision of the thrombus.

31. How are hemorrhoids treated?

Treat mildly symptomatic hemorrhoids with irrigation during the shower or bath, stool softeners, high-fiber diet, bulk laxatives (e.g., psyllium or methylcellulose), increased fluid consumption, proper anal hygiene, and analgesics if necessary. Nonthrombosed prolapsed hemorrhoids should be gently reduced. Thrombosed hemorrhoids should be excised. Patients with intractable symptoms need surgical referral.

32. What is an anal fissure?

An anal fissure is a linear crack or ulcer in the epithelium in the distal anal canal. Anal fissures are the most common cause of rectal pain. Most are idiopathic, but any anal canal trauma can cause a fissure. Most benign anal fissures occur in the posterior midline, followed by the anterior midline. Fissures in other locations are associated with CD, infection, malignancy, or immunodeficiency.

33. How do I treat an anal fissure?

Most anal fissures can be managed conservatively with sitz baths, stool softeners, high-fiber diet, bulk laxatives (e.g., psyllium or methylcellulose), additional fluid consumption, proper anal hygiene, and analgesics. Recent studies have shown good success with the use of topical 0.2% nitroglycerin ointment applied twice daily for 6 weeks or a single botulinum injection. Fissures that do not improve with conservative therapies should be referred to a surgeon for consideration of a lateral internal sphincterotomy.

34. Can I drain anorectal abscesses in the ED?

Small isolated perianal abscesses can be drained successfully in the ED. These abscesses can be painful, requiring both local anesthetic and oral or parenteral sedation. For complicated or deep rectal abscesses, consult surgery for operative drainage.

KEY POINTS: BOWEL DISORDERS

1. Appendicitis is common, and unusual presentations are frequent; therefore, always consider appendicitis in a patient with abdominal pain.
2. A patient with atrial fibrillation and abdominal pain has mesenteric ischemia until proven otherwise.
3. Surgical adhesions are the most frequent cause of SBO.
4. Patients with SBO should be aggressively resuscitated with intravenous fluids in the ED due to the extensive depletion of intravascular fluid.
5. Although diverticulitis is most commonly seen in the older patient population, younger patients (20–40 years) also develop it.
6. IBD can cause complicated rectal abscesses or fissures that require surgical consultation.

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CHAPTER 35

LIVER AND BILIARY TRACT DISEASE

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1. What are the common manifestations of biliary disease?

Cholelithiasis is the presence of gallstones in the gallbladder without evidence of infection. Among adults, 8% of men and 17% of women have gallstones, and the incidence increases with age with an incidence as high as 27% in the elderly.

- **Biliary colic** is right upper quadrant or epigastric pain sometimes radiating to the right shoulder or scapula. It usually lasts less than 6 hours, is persistent, not colicky, occurs after a fatty meal, and is thought to be due to transient obstruction of the cystic duct by a gallstone.
- Of patients with colic, 30% progress to **cholecystitis**, a bacterial overgrowth and inflammation of the gallbladder caused by obstruction of the cystic duct by a stone. The pain with cholecystitis is similar to biliary colic but persists beyond 6 hours, is accompanied by a Murphy's sign, and can be present with or without fever and chills
- **Choledocholithiasis** occurs when a gallstone lodges in the common bile duct (CBD) and can cause cholecystitis or pancreatitis (if the ampulla of Vater is obstructed) or both.
- **Ascending cholangitis** is a severe infection of the biliary tract from complete biliary obstruction (most commonly the CBD) in the presence of a bacterial infection. It presents as right upper quadrant pain, fever and chills, and jaundice (Charcot's triad), although only 25% of patients have all three. It may include shock and mental status changes (Reynold's pentad), more commonly seen with gangrenous or emphysematous cholecystitis
- **Emphysematous cholecystitis** is caused by complete cystic duct obstruction with subsequent abscess formation in the gallbladder wall by gas-forming bacteria. It is seen with vascular insufficiency, severe burns, and trauma. It is more frequent in men and diabetic patients and often is accompanied by sepsis.

2. Do all gallstones produce pain? Does a lack of stones preclude cholecystitis?

Of patients with gallstones, 80% are asymptomatic. Of asymptomatic patients, 15% to 30% develop symptoms within 15 years. Although 90% to 95% of cholecystitis cases are in the setting of gallstones, 5% to 10% are not secondary to cholelithiasis and are termed *acalculous cholecystitis*. It is a difficult diagnosis because it is often a complication of another process such as diabetes, burns, multisystem trauma, AIDS, or sepsis.

3. What is Murphy's sign?

The sign is named after a prominent Chicago surgeon, John B. Murphy (1857–1916). The patient is asked to take a deep breath while the examiner applies pressure over the area of the gallbladder. If the gallbladder is inflamed, the descending diaphragm forces it against the examiner's fingertips, causing pain and often a sudden halt to the inspiration. A sonographic Murphy's sign uses the ultrasound probe instead of the examiner's fingers and is positive when the site of maximal tenderness localizes to the gallbladder. The finding is 97% sensitive for acute cholecystitis.

4. Can a plain radiograph of the abdomen aid diagnosis?

Maybe. However, ultrasound is the preferred first-line diagnostic test. Only 10% to 20% of gallstones contain sufficient calcium to be radiopaque. Air can be seen in the biliary tree or the gallbladder wall when infection is due to gas-forming bacteria or there is a biliary-intestinal fistula.

5. What is the gold standard for diagnosing cholecystitis?

Although ultrasound is the test of choice in the ED, a **hepatobiliary iminodiacetic acid (HIDA) scan** is the gold standard with 95% accuracy if the gallbladder does not fill with radioisotope within 4 hours after injection.

6. Is an elevated temperature or white blood cell count necessary for diagnosis?

No, they are not helpful for diagnosis, as is seen in one study in which 71% of patients with acute nongangrenous cholecystitis were afebrile, and 32% had normal white blood cell count. Past the age of 60, the typical signs and symptoms may not be present. The sensitivity of the Murphy's sign decreases to 48% with age older than 60.

7. Describe the ultrasound findings in cholecystitis.

Gallstones as small as 2 mm can be detected directly, or sometimes their presence can be inferred by interference with transmission of ultrasound waves (*acoustic shadowing*) (Fig. 35-1). Other helpful findings include a thickened gallbladder wall (>3 mm), fluid collections around the gallbladder (*pericholecystic fluid*), and common ductal dilation (>6 mm). The ultrasound is 94% sensitive and 78% specific for identifying cholecystitis

KEY POINTS: ULTRASOUND FINDINGS OF CHOLECYSTITIS

1. Presence of gallstones
2. Gallbladder wall thickening >3 mm
3. Pericholecystic fluid
4. CBD dilatation >6 mm

8. When should elective surgery be considered in patients with asymptomatic cholelithiasis?

Cholecystectomy should be considered in diabetics, patients with a porcelain gallbladder, and patients with a history of biliary pancreatitis.

- Diabetics have increased morbidity and mortality when urgent cholecystectomy is done in the setting of cholecystitis.
- Calcified or porcelain gallbladders have a 22% association with carcinoma.
- The risks of pancreatitis may outweigh the risks of elective cholecystectomy.

9. What are Courvoisier's law, Klatskin's tumor, and Fitz-Hugh-Curtis syndrome?

- **Courvoisier's law** states that a palpable gallbladder in the setting of painless jaundice is likely to represent obstruction of the CBD by a malignancy, usually carcinoma of the pancreatic head.
- **Klatskin's tumor** is a malignant tumor located where the hepatic ducts form the common duct.
- **Fitz-Hugh-Curtis syndrome** is caused by pelvic inflammatory disease extending up the right paracolic gutter, causing inflammation of the capsule of the liver (perihepatitis), and can lead to adhesions between the liver and abdominal wall.

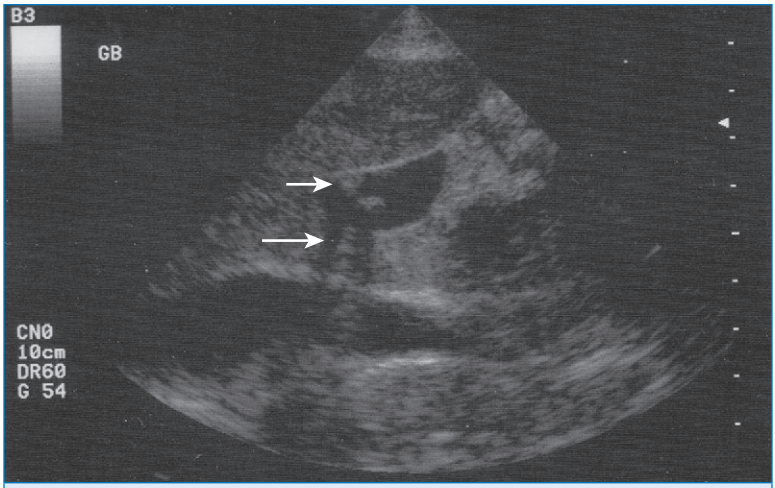

Figure 35-1. Ultrasound image reveals an anechoic gallbladder containing two echogenic stones, which are creating acoustic shadowing inferiorly. The short arrow is pointed to the gallstones within the gallbladder, and the long arrow points to the shadowing effect of the stones.

10. What is a porcelain gallbladder?

A gallbladder with calcified walls. This is an important finding because 22% are associated with carcinoma, and it is an indication for cholecystectomy in asymptomatic patients. There is a higher incidence in women and American Indians, especially members of the Pima tribe.

11. Are all gallstones created equal?

No. The most common are cholesterol stones and usually are found in the stereotypical, **female, fat, forty, fertile** patient. Patients of Asian descent, those with parasitic infections (*Ascaris lumbricoides*), chronic liver/biliary disease, or chronic hemolysis states (i.e., sickle cell, spherocytosis) are more likely to have pigment stones.

12. What is endoscopic retrograde cholangiopancreatography (ERCP)? What is the most common complication that presents to the ED after an ERCP procedure?

ERCP is a procedure that examines the pancreatic and bile ducts for disease or irregularities with the ability of removing lodged stones and opening narrowed ducts with stents. The most common serious complication is pancreatitis, which occurs in approximately 1% of cases.

13. What are liver function tests?

Aspartate aminotransferase (AST) and alanine aminotransferase (ALT) are markers of acute liver injury and have no correlation with liver function. Liver function is analyzed best by measuring factors affected by hepatic protein synthesis. Acute liver failure results in a decrease in vitamin K-dependent coagulation factors (except factor VIII), leading to a prolonged prothrombin time. The liver also synthesizes albumin, although its longer half-life makes it a better marker of subacute or chronic liver disease.

14. What is the difference between conjugated and unconjugated bilirubinemia?

Bilirubin is a breakdown product of hemoglobin and heme-related proteins. In its **unconjugated**, hydrophobic form, it is unable to be excreted into bile, although it can traverse the blood-brain barrier and placenta. Bilirubin is **conjugated** in the liver with glucuronic acid, making it more water soluble for excretion into the bile. A predominance of unconjugated bilirubin occurs when

there is overproduction (hemolysis) or decreased conjugation (decreased intrinsic metabolic activity of the liver by acute or chronic injury). A primarily conjugated bilirubinemia results from reflux into the plasma from impaired excretion, secondary to biliary obstruction from cholestasis, gallstones, tumors, or strictures.

15. State the major causes of acute hepatitis.

Viruses such as hepatitis A through E viruses, Epstein-Barr virus, herpes simplex virus (HSV), Coxsackie, and cytomegalovirus. It also can result from exposure to toxins such as ethanol, *Amanita phalloides* mushrooms, carbon tetrachloride, acetaminophen, halothane, and chlorpromazine.

16. What are the risk factors for viral hepatitis? Which can result in a carrier state?

Hepatitis B and C are transmitted via blood and body fluid exposures: sexual intercourse, intravenous drug abuse, blood transfusions, tattoos or body piercings, hemodialysis, and needle sticks. Hepatitis A and E are transmitted via fecal/oral exposure (i.e., foreign travel, raw seafood ingestion, poor hygiene or sewage management, and close contact with a person infected with hepatitis). Hepatitis A and E are often self-limited, whereas hepatitis B and C can result in a carrier state and progress to chronic hepatitis.

17. What is the most common form of liver disease in the United States?

Alcoholic hepatitis. It is most often diagnosed by history, but the following are highly suggestive associated findings: spider angiomas, gynecomastia, palmar erythema, ascites, and an elevated AST and ALT in a ratio of greater than 2:1.

18. Which patients with hepatitis should be admitted?

KEY POINTS: CRITERIA FOR ADMISSION IN PATIENT WITH HEPATITIS

1. Coagulopathy, international normalized ratio (INR) >3
2. Active bleeding
3. Encephalopathy
4. Unable to tolerate intake by mouth
5. Social issues that make follow up care and compliance problematic

19. What is the initial treatment of hepatic encephalopathy? What is asterixis?

- **Hepatic encephalopathy** is the accumulation of nitrogenous waste products normally metabolized by the liver. It comprises a spectrum of clinical presentations ranging from lethargy to coma. In addition to supportive care, lactulose, neomycin, and a low-protein diet are the mainstays of treatment. Lactulose reduces ammonia absorption by increasing gastrointestinal (GI) motility and by trapping ammonia as ammonium in the stool via fecal acidification in the form of lactic acid; neomycin is an aminoglycoside that reduces the bacteria that produces ammonia.
- **Asterixis** is a clinical manifestation of moderate hepatic encephalopathy in which the hands *flap* (low-amplitude alternating flexion and extension) when the arms are held straight and the wrists are held in extension.

KEY POINTS: TREATMENT OF HEPATIC ENCEPHALOPATHY

1. Supportive care
2. Lactulose, 15–30 mL PO every 6–8 hours
3. Neomycin, 0.5 gm PO every 4–6 hours
4. Low-protein diet

20. What are complications of chronic liver disease to watch for in the ED?

The most common complication of cirrhotic ascites is **spontaneous bacterial peritonitis (SBP)**, which can present with any of the following: fever, abdominal pain, or mental status changes. Paracentesis is diagnostic if it shows white blood cell count greater than 1000 cells/mm^3, neutrophils greater than 250 cells/mm^3, or a positive Gram stain or culture. Portal hypertension causes the development of **esophageal varices**, which can lead to massive GI bleeding. Management should focus on resuscitation, local control (balloon tamponade or endoscopic ligation/sclerotherapy), and reduction of portal pressure (vasopressin plus nitroglycerin, somatostatin/octreotide, and if necessary, emergent transjugular intrahepatic portosystemic shunt). Patients with chronic liver disease are at greatly **increased risk of bleeding** because of deficits of the coagulation cascade proteins, platelet abnormalities, and increased fibrinolysis. Renal failure in cirrhotic patients with structurally normal kidneys represents the **hepatorenal syndrome.** One study showed 38% 1-year survival in patients with the hepatorenal syndrome.

KEY POINTS: PERITONEAL FLUID CRITERIA FOR SBP

1. White blood cell count $>$ 1000 cells/mm^3
2. Neutrophil count $>$ 250 cells/mm^3
3. Positive Gram stain
4. Positive culture result (gold standard)

21. Are there any special issues to watch for in the postliver-transplant patient?

Transplant rejection is common and manifests as fever, pain, and elevated transaminases and bilirubin. This can be treated with high-dose steroids and increased immunosuppressive medication. Other causes of transplant dysfunction include biliary strictures, recurrence of viral hepatitis, and vascular thrombosis. Immunosuppressive therapy can cause nephrotoxicity, neurotoxicity, and hypertension. As with other immunosuppressed patients, opportunistic infections, such as cytomegalovirus, Epstein-Barr virus, mycobacteria, and *Pneumocystis,* and fungal infection should be considered.

WEBSITES

Hepatitis B: www.hepb.org/
Hepatitis C: http://hepatitis-central.com/

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RENAL COLIC AND SCROTAL PAIN

Christopher M.B. Fernandes, MD

1. What are the most common forms of renal stones?

Calcium stones account for 80% of all renal stones: Two thirds are calcium oxalate, and the remainder are calcium phosphate. Struvite (magnesium ammonium phosphate), uric acid, and cystine account for 20% of renal stones.

2. List factors that predispose to stone formation.

Calcium stones

- Chronic dehydration
- Antacid use
- Hypercalciuria
- Hyperoxaluria
- Acid urine
- Ingestion of vitamins A, C, and D

Struvite stones: chronic infection by urea-splitting organisms

Cystine stones: cystinuria

3. What lethal conditions are sometimes misdiagnosed as renal colic?

Aortic and iliac aneurysms. A careful search for bruits and pulsatile masses is mandatory when renal colic is suspected.

4. What clinical features help distinguish renal colic from other causes of abdominal pain?

Renal colic usually begins abruptly, causing terrible pain in the flank, costovertebral angle, lateral abdomen, and genitals. Patients often are profoundly distressed, more so than patients with other abdominal pathologies. Pallor, diaphoresis, restlessness, and nausea are prominent. Renal colic causes flank tenderness, but in contrast to other causes of lateralized abdominal pain (e.g., appendicitis, diverticulitis, cholelithiasis, and ectopic pregnancy), it produces little or no abdominal tenderness.

5. In which patients would imaging be absolutely indicated to confirm the diagnosis of renal colic?

- Patients with a first episode of renal colic
- Patients in whom the diagnosis is unclear
- Patients in whom a proximal urinary tract infection, in addition to a calculus, is suspected
- Elderly patients

6. What is the role of the abdominal flat plate in diagnosing renal colic?

The abdominal flat plate, or *kidneys-ureter-bladder (KUB)*, is less sensitive and less specific than the clinical examination and, by itself, has no role in the work-up of suspected renal colic. If a stone is diagnosed on ultrasound, it may be appropriate to view the stone on a plain film. Subsequent radiographs may be helpful to document stone progression.

7. Has helical computed tomography (CT) supplanted the intravenous pyelogram (IVP) as the diagnostic test of choice? Why or why not?
Helical noncontrast CT has replaced IVP as the preferred diagnostic test. The IVP pinpoints stone size and location, clarifies the degree of obstruction, and shows ongoing renal function. Helical CT has been shown to be 97% sensitive and 96% specific in diagnosing renal stones. Used for this purpose, helical CT does not require intravenous contrast material and is faster than IVP—requiring only 1 to 2 minutes of scanner time to complete a study. Even though helical CT provides no information about renal function, this can be ascertained by a urinalysis and serum creatinine. The marginal cost is less, and it can identify other important causes of flank pain.

8. Is pregnancy a contraindication to IVP?
Ultrasound is the investigation of choice in pregnant patients, but if ultrasound is nondiagnostic, a limited IVP (scout film and 20-minutes postinjection film, preferably coned to the area of concern) is appropriate because the risk of radiation from CT KUB is greater than the limited exposure of plain film radiography with this limited IVP.

9. What IVP findings suggest a renal stone?
Typical findings include a delayed, intense, and often prolonged nephrogram on the involved side, delayed filling and dilation of the affected collecting system (hydroureter and hydronephrosis), and an uninterrupted column of dye extending from the kidney to the calculus. An unobstructed ureter, because it is peristaltic, does not normally appear opacified with contrast in its entirety.

10. Why is the postvoid film important? What other special views are helpful?
Contrast in the bladder obscures the distal ureter. The postvoid film provides optimal visualization of the distal ureter and the ureterovesical junction. The postvoid film also shows whether the bladder is emptying completely. Oblique views help to confirm that a visualized stone is in, rather than overlying, the ureter. Prone films often provide a better view of the ureter than do standard supine films.

KEY POINTS: MOST COMMON FORMS OF RENAL STONES

1. Calcium stones (80%)
   - Calcium oxalate: two thirds
   - Calcium phosphate: one third
2. Struvite, uric acid, and cystine (20%)

11. What if the ureter is not visualized on the standard IVP?
In high-grade ureteral obstruction, contrast material may not reach the distal ureter for many hours. If the ureter cannot be visualized at 1 hour, take a 2-hour film. If this fails, take a 4-hour film. The interval between films should be doubled until adequate visualization is achieved. It is important not to abandon the IVP until contrast material reaches the calculus.

12. Name the most common sites of ureteral stone impaction.
The ureteropelvic junction, the pelvic brim (where the ureter crosses the iliac vessels), and the ureterovesical junction (the most narrow point in the ureter).

13. Can the likelihood of spontaneous passage be predicted based on the size and location of the stone?
Stones reaching the distal ureter are more likely to pass than those impacting proximally. Stones 2 to 4 mm pass 95% of the time; stones 4 to 6 mm pass 50% of the time, and stones

greater than 6 mm pass 10% of the time. When estimating stone size, remember that the X-ray image is magnified; the actual size is 80% of what is measured on the films.

14. What if the imaging study is normal, but the patient still appears to have renal colic?

Re-examine the patient carefully to ensure that you have not missed another cause of abdominal pain and that the patient is not developing a condition requiring surgery. If the physical examination is still compatible with renal colic, treat the patient, not the test result. Occasional false-negative results occur with all tests, and imaging modalities may miss small stones, but this may not be clinically relevant because small stones are unlikely to require specific therapy. Persistent severe flank pain can be caused by a leaking abdominal aortic aneurysm (AAA).

15. Isn't an ultrasound just as accurate as helical CT or an IVP?

Ultrasound is safe and noninvasive but is more prone to false-negative results than the other studies. Ultrasound is sensitive for stones in the bladder and renal pelvis but often fails to visualize those in the mid and distal ureter—the most common sites for stone impaction. When ultrasound fails to identify a stone, however, it may show dilation of the renal collecting system, providing evidence of ureteral obstruction.

16. List secondary signs of ureteral obstruction shown on helical CT.

- Unilateral obstruction
- Stranding of perinephric fat
- Hydronephrosis
- Nephromegaly

17. What is the soft tissue rim sign on helical CT? How is it useful?

This sign shows soft-tissue attenuation around a ureteral calculus and helps differentiate a calculus from a phlebolith.

18. What other tests are useful in the ED in patients with renal calculi?

Urine dipsticks are sensitive for microscopic hematuria, which is present in 80% of patients with renal colic. Urinalysis is recommended to rule out pyuria and bacteriuria. Urine culture is indicated if symptoms, signs, or urinalysis findings suggest infection. Determination of blood urea nitrogen (BUN), creatinine, and electrolyte levels is helpful if the patient has been vomiting or if presence of an underlying renal disease is suspected. There is usually no need for a more extensive metabolic work-up in the ED.

19. Why is coexistent infection a major problem?

Bacteria in an obstructed collecting system can cause abscess formation, renal destruction, bacteremia and sepsis. The presence of infection in an obstructed ureter mandates immediate consultation with a urologist and high-dose intravenous antibiotics.

20. Has lithotripsy supplanted percutaneous and open surgical methods of stone removal?

Not always. Optimal therapy depends on the size, type, and location of the stone. Ureteroscopic techniques probably are still preferable for lower ureteral stones. Extracorporeal shock wave lithotripsy (ESWL) is optimal for stones 2 cm in size, particularly those in the renal pelvis. Percutaneous stone removal techniques are indicated for larger stones, when there is obstructive uropathy, and when less invasive techniques have failed. For some stones, a combination of ESWL followed by percutaneous instrumentation is optimal. Some large stones still require open surgery. The method of removal is best determined by a urologist. Of note, newer technologies for treatment have led to an increased frequency of procedural interventions, with an overall cost increase attributable to stones compared to the pre-ESWL era.

21. What are the basics of ED treatment of renal colic?

Hydration, analgesia, and antiemetics. Patients who have clinical dehydration secondary to vomiting and decreased oral intake, as well as if radiocontrast media study is planned, should receive intravenous fluid hydration. Various analgesics and antiemetics are available for rapid control of symptoms (see Table 36-1). Intravenous pain control is the mainstay of ED treatment. Analgesic treatment should not be delayed waiting for test results. Opiate analgesics have long been the standard medication. Rectal or intravenous nonsteroidal

TABLE 36–1 ANALGESICS AND ANTIEMETICS FOR RENAL COLIC

Opioid analgesics			
Anileridine (Leritine)	PO 50 mg	q 4 h	prn
Hydromorphone	IV 1–2 mg	q 2–4 h	
	IM 1–2 mg/kg	q 2 h	prn*
Meperidine (Demerol)	IV 25–50 mg	q 5–10 min	prn
Morphine sulphate	IV 3–5 mg	q 5–10 min	prn
	IM 0.1–0.2 mg/kg	q 3 h	prn*
Oxycodone and acetaminophen (Percocet)	PO 2 tabs	q 4 h	prn
Oxycodone and acetylsalicylic acid (Percodan)	PO 2 tabs	q 4 h	prn
Antiemetics			
Metoclopramide (Reglan)	IV 10–20 mg	q 15 min	prn
Perphenazine (Trilafon)	IM 5 mg	q 6 h	prn*
	PO 4 mg	q 6 h	prn
Prochlorperazine (Compazine)	IV 5–10 mg	q 4 h	prn
	IM 5–10 mg	q 6 h	prn*
	PO 5–10 mg	q 4 h	prn
Nonsteroidal analgesics			
Diclofenac (Voltaren)	50- or 100-mg suppositories, 150 mg/day		
Indomethacin	50- or 100-mg suppositories, 200 mg/day		
Ketorolac (Toradol)	IV 30 mg	q 6 h	
	IM 30 mg	q 6 h	

IM, intramuscularly; IV, intravenously; PO, per os (by mouth); prn, as needed; q, every.
*Intramuscular route not recommended for ED management of acute, severe pain.

anti-inflammatory drugs (NSAIDs), which inhibit renal prostaglandin synthesis, are effective and may be given concurrently with opioids. A recent systematic review suggested that for the management of acute renal colic, NSAIDs achieve slightly better pain relief, reduce need for rescue analgesia, and produce much less vomiting than do opioids. Optimal ED pain control involves the combined administration of NSAIDs and opioids (balanced analgesia).

22. Who requires hospitalization? Urology consultation?

Patients with high-grade obstruction, intractable pain or vomiting, associated urinary tract infection, a solitary or transplanted kidney, and in whom the diagnosis is uncertain. Obtain urologic consultation for patients with stones larger than 5 mm in diameter, urinary extravasation, and renal insufficiency regardless of symptoms.

23. What advice should I give to patients being discharged from the ED?

Patients should be advised to drink plenty of fluids, strain their urine, and return to the ED if they develop symptoms of infection or recurrent severe pain. Follow-up with a urologist within a week should be recommended.

24. Which analgesics are recommended for outpatient pain control?

Gastrointestinal irritation limits the usefulness of oral NSAIDs in patients with renal colic; however, rectal NSAIDs (diclofenac, indomethacin) may provide adequate analgesia. If necessary, oral opioids can be combined with NSAIDs in patients with documented ureteral calculi.

KEY POINTS: INDICATIONS FOR HOSPITALIZATION

1. Patients with high-grade obstruction
2. Intractable pain or vomiting
3. Associated urinary tract infection
4. Solitary or transplanted kidney
5. Patients in whom the diagnosis is uncertain
6. Stones larger than 5 mm in diameter
7. Urinary extravasation
8. Renal insufficiency regardless of symptoms

25. Why should patients be given a urine strainer on discharge?

If the stone can be analyzed, the patient can then receive follow-up counseling on dietary modification or medications that may reduce the risk of recurrence.

26. When should patients return to the ED?

Patients should be instructed to seek medical care immediately if they have continued or increasing pain, nausea and vomiting, fever or chills, or any other new symptoms.

27. What medical alternatives to active stone removal are available?

In patients with ureteral stones $<$10 mm and whose symptoms are controlled with medications, observation with periodic evaluation is an option. For such patients, appropriate medical therapy can be offered. In such patients, calcium channel blocker or α-blocker therapy can be used. With some data suggesting faster passage with tamsulosin, treatment can be initiated for four weeks.

28. What is the differential diagnosis in a patient presenting with an acutely painful scrotum?

The differential diagnosis of acute scrotal pain includes testicular torsion, torsion of the testicular or epididymal appendages, epididymitis, orchitis, scrotal hernia, testicular tumor, renal colic, Henoch-Schönlein purpura, and Fournier's gangrene. Although not life-threatening, testicular torsion is a significant cause of morbidity and sterility in the male. Thus, any case of an acute scrotum should be considered testicular torsion until proven otherwise.

29. What is testicular torsion?

Testicular torsion results from maldevelopment of the normal fixation that occurs between the enveloping tunica vaginalis and the posterior scrotal wall. This maldevelopment then allows the testis and the epididymis to hang freely in the scrotum (the so-called bell-clapper deformity), allowing the testis to rotate on the spermatic cord. The degree of testicular ischemia is dependent on the number of rotations of the cord.

30. When is testicular torsion most likely to occur?

The annual incidence of testicular torsion is estimated to be 1 in 400 for males younger than the age of 25. Testicular torsion has a bimodal distribution, with peak incidence in the neonate within the first few days of life and in preadolescence.

KEY POINTS: SIX DIFFERENTIAL DIAGNOSES OF ACUTE SCROTUM

1. Testicular torsion
2. Torsion of the testicular or epididymal appendages
3. Epididymo-orchitis
4. Scrotal hernia
5. Testicular tumor
6. Fournier's gangrene

31. What history is suggestive of testicular torsion?

Usually, there is a history of trauma or strenuous event before the onset of scrotal pain in testicular torsion. One study reported sudden onset of scrotal pain to be present in 90% of patients with testicular torsion, compared with 58% of patients with epididymitis and 78% of patients with normal scrotum. Fever was present in 10% of patients with testicular torsion compared with 32% of patients with epididymitis.

32. What clinical features are suggestive of testicular torsion?

In testicular torsion, the affected testis usually is firm, tender, and aligned in a horizontal rather than a vertical axis. The presence of the cremasteric reflex appears to be one of the most helpful signs in ruling out testicular torsion with 96% negative predictive value. It is elicited by gently stroking the inner aspect of the involved thigh and observing more than 0.5 cm of elevation in the affected testis.

33. What is the proper management of testicular torsion?

The proper management of a suspected testicular torsion is immediate urologic consultation and surgical exploration. If surgical consultation is not immediately available, manual detorsion should be attempted.

KEY POINTS: PROPER MANAGEMENT OF TESTICULAR TORSION

1. Emergent urologic consultation
2. Attempt at manual detorsion

34. How is manual detorsion performed?

This procedure is best done by standing at the foot or right side of the patient's bed. The torsed testis is detorsed in fashion similar to opening a book. The patient's right testis is rotated counterclockwise, and the left testis is rotated clockwise. A testis viability rate of 100%, 70%, and 20% for 6, 6–12, and 12–24 hours of symptoms, respectively, has been reported.

35. Is imaging testing helpful to confirm the diagnosis of testicular torsion?

Testicular torsion is mainly a clinical diagnosis. If it is suspected, immediate urologic evaluation is mandatory and should precede any further testing because time is critical. However, imaging tests could be helpful adjuncts to the work-up of the acute scrotum when the diagnosis is unclear.

36. What are the diagnostic imaging tests that can be used to evaluate the acute scrotum?

Doppler ultrasound and radionucleotide scintigraphy are the two imaging tests that can be used to evaluate the acute scrotum. Both measure the blood flow to the testis; Doppler ultrasound carries a sensitivity of 86% and 97% accuracy, whereas radionucleotide scintigraphy has 80% sensitivity and 97% specificity.

37. How is testicular torsion treated surgically?

The involved testis must be detorsed and then checked for viability. If it is viable, it is fixed (orchiopexy). Because approximately 40% of patients have a bell-clapper deformity of the contralateral testis, the unaffected testis should be fixed to prevent recurrence.

38. What are testis and epididymal appendix?

The appendix testis is a Müllerian duct remnant that is attached to the superior pole of the testicle and rests in the groove between the testis and epididymis. The appendix epididymis is a Wolffian duct remnant that is attached to the head of the epididymis.

39. What are clinical features of torsion of testis and epididymal appendix?

Both torsion of testis and epididymal appendix result in unilateral pain. The pain of epididymal appendix torsion typically is more gradual in onset and is usually not quite as severe as that associated with true testicular torsion. The most important aspect of the physical examination is pain and tenderness localized to the involved appendix. However, late in its course, generalized scrotal swelling and tenderness may be encountered, making it difficult to differentiate from testicular torsion. The classic blue dot sign (visualization of the ischemic or necrotic appendix testis through the scrotal wall on the superior aspect of the testicle) is pathognomonic for appendix testis torsion, but it is also relatively uncommon.

40. How is torsion of testis or epididymal appendix treated?

Torsion of epididymal and testicular appendix are self-resolving, benign processes. Rest, scrotal elevation, and analgesia are the mainstays of treatment. Resolution of the swelling and pain should be expected within 1 week.

41. What is epididymitis?

Epididymitis arises from swelling and pain of the epididymis. It usually occurs secondary to infection or inflammation from the urethra or bladder. Patients with epididymitis present with

increasing, dull, unilateral scrotal pain during a period of hours to days. Possible associated symptoms include fever, urethral discharge, hydrocele, erythema of the scrotum, and palpable swelling of the epididymis. Involvement of the ipsilateral testis is common, producing epididymitis-orchitis.

42. List the most common causes of epididymitis.

The most common causes of epididymitis in males older than 35 years are gram-negative organisms such as *Escherichia coli*, *Klebsiella*, and *Pseudomonas* species. Among sexually active men younger than 35 years, epididymitis is often caused by *Chlamydia trachomatis* or *Neisseria gonorrhoeae*. *E. coli* infection also may occur in men who are insertive partners during anal intercourse.

43. What is the treatment for epididymitis?

Admission should be considered for any febrile, toxic-appearing patient with epididymitis or when testicular or epididymal abscess should be excluded. In-patient therapy includes bed rest, analgesia, scrotal elevation (performed by taping a towel under the scrotum and over the proximal anterior thighs in the supine position), NSAIDs, and parenteral antibiotics based on presumed etiology.

When sexually transmitted disease is suspected to be the cause of epididymitis, or in males younger than 35 years, urethral culture should be taken for *Chlamydia* and gonorrhea, followed by empirical treatment with ceftriaxone 250 mg intramuscularly once, plus doxycycline 100 mg orally twice a day for 10 days *or* ofloxacin 300 mg orally twice a day for 10 days. When gram-negative bacilli are suspected to be the cause for epididymitis, or in males older than 35 years, treatment includes ciprofloxacin 500 mg orally twice a day or levofloxacin 750 mg once a day for 10 to 14 days.

Treatment in all patients should also include bed rest, analgesia, and scrotal elevation. Follow up with a urologist within 5 to 7 days is recommended.

44. What is Fournier's gangrene?

Fournier's gangrene, a surgical emergency, is a life-threatening disease characterized by necrotizing fasciitis of the perineal and genital region. It is generally the result of a polymicrobial infection from bacteria that are normally present in the perianal area. The diagnosis and treatment of Fournier's gangrene are similar to those of necrotizing fasciitis. Diabetes mellitus, alcohol abuse, and local trauma are known risk factors. Empirical broad-spectrum antibiotics with early aggressive surgical debridement are the mainstays of therapy. Reexploration is commonly needed, and some patients require diverting colostomies or orchiectomies.

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CHAPTER 37

ACUTE URINARY RETENTION

John P. Marshall, MD

1. What is acute urinary retention (AUR)?

A painful inability to urinate. AUR is most commonly the result of bladder outlet obstruction, but it also may result from neurogenic, pharmacologic, or other causes of detrusor muscle dysfunction. Urine is produced normally but is retained in the bladder, which then becomes distended and uncomfortable.

2. Is there chronic urinary retention?

Yes. It generally represents prolonged retention. The hallmarks of chronic urinary retention are (a) the absence of pain and (b) overflow incontinence. It most frequently occurs in mentally debilitated or neurologically compromised patients.

3. What is the most common cause of AUR? Who gets it?

Obstruction of the lower urinary tract (bladder and urethra) is the most common cause encountered in the ED. In general, AUR is a disease of older men, although it is occasionally encountered in women. The usual site of obstruction is the prostate gland, but lesions of the urethra or penis also may cause retention. Patients with indwelling catheters (suprapubic or Foley) are at risk for episodes of retention because of obstruction or dysfunction of these drainage systems.

4. How does benign prostatic hypertrophy (BPH) cause AUR?

BPH with bladder neck obstruction is the most common cause of AUR. Of men older than age 60, 50% have histologic evidence of BPH. As the prostate hypertrophies, urine outflow is obstructed by enlargement of the median lobe of the gland impinging on the internal urethral lumen. The typical patient with BPH gives a progressive history suggestive of urinary outlet obstruction. Symptoms such as hesitancy, diminished stream quality, dribbling, nocturia, and the sensation of incomplete bladder emptying may precede the episode of acute retention. New medications or increased fluid loads may precipitate an acute episode of retention in these patients.

5. List the other causes of AUR.

- **Obstructive**: BPH, prostate carcinoma, prostatitis, urethral stricture, posterior urethral valves, phimosis, paraphimosis, balanitis, meatal stenosis, calculi, blood clots, circumcision, urethral foreign body, constricting penile ring, and clogged or crimped Foley catheter
- **Neurogenic**: Spinal cord injuries, herniated lumbosacral disks (cauda equina syndrome), central nervous system (CNS) tumors, stroke, diabetes, multiple sclerosis, encephalitis, tabes dorsalis, syringomyelia, herpes simplex, herpes zoster, and alcohol withdrawal
- **Pharmacologic**: Anticholinergics, antihistamines, antidepressants, antispasmodics, narcotics, sympathomimetics, antipsychotics, and antiparkinsonian agents (see Question 14)
- **Psychogenic**: Diagnosis of exclusion

6. What are the important features in the history and physical examination?

When taking the history, any previous prostate or urethral conditions should be elicited. Patients often have a history of chronic voiding hesitancy, a decreased force to the urinary stream, a feeling of incomplete bladder emptying, or nocturia. Information about neurologic symptoms, trauma, previous instrumentation, back pain, and current medication is essential. On physical examination, the distended bladder often is palpable above the pubic rim and indicates at least 150 mL of urine in the bladder. The penis or vulva, and particularly the urethra should be examined carefully for any signs of stricture, which may be evident on palpation. A rectal examination is essential and often provides clues to the diagnosis of BPH, prostate carcinoma, or prostatitis. A careful neurologic examination, including rectal tone and perineal sensation, is vital in any patient suspected of having a neurologic lesion.

7. Are there any red flags in the history and physical examination that might indicate a more serious, potentially surgical, cause?

Yes. New urinary symptoms, particularly obstruction, in patients with a history of trauma or back pain should alert the examiner to the possibility of spinal cord compression resulting from disk herniation, fracture, epidural hematoma, epidural abscess, or tumor. Be especially suspicious if there is no prior history of bladder, prostate, or urethral disorders.

8. How do I treat AUR?

Catheterization and bladder decompression using a Foley catheter.

9. What if I can't pass a Foley catheter?

Occasionally, simple passage of a 16- or 18-French Foley catheter cannot be accomplished. One trick that often helps is to fill a 30-mL syringe with lidocaine (Xylocaine) jelly and inject it into the urethral meatus. Still no luck? Try an 18- or 20-French coudé catheter. The coudé-tipped catheter has a gentle upward curve in the distal 3 cm that may be helpful in pointing the catheter up and over the enlarged prostatic lobe. Never force a catheter through an area of significant resistance because this can cause urethral perforation, false lumens, and subsequent stricture formation.

10. Is bigger better?

A loaded question. If you are unable to pass a 16-French (standard adult) catheter, it is generally recommended to move up in size to an 18- or 20-French Foley catheter. Usually, the stiffness and larger bulk of the bigger catheter are more successful in passing through the bladder neck than a smaller, more flexible catheter. Remember, never force a catheter through significant resistance.

11. What if nothing is working?

If you still cannot pass a catheter, the obstruction may be more severe than anticipated, or a stricture may be present. One clue to the presence of a stricture in adult males is that the obstruction occurs less than 16 cm from the external meatus of the urethra. If this is the case, an attempt may be made using a pediatric-sized urinary catheter. If this fails, more sophisticated instrumentation may be required, such as filiforms and followers or catheter guides. These techniques should be done only by a urologist or practitioner with extensive training in their use. If AUR cannot be relieved by transurethral bladder catheterization, placement of a suprapubic catheter may be necessary.

12. What is suprapubic catheterization? How is it done?

A procedure used to pass a urinary catheter directly into the bladder through the lower anterior abdominal wall (see Fig. 37-1). It is indicated when bladder drainage is necessary and other methods have failed or when urethral damage from trauma is suspected. The procedure is done under sterile conditions with local anesthesia. The presence of a distended bladder is confirmed by ultrasound or percussion. A small midline incision is made 2 cm above the symphysis pubis. Depending on the technique, either a needle or a trocar is used to penetrate the bladder through the incision. When urine is aspirated, a catheter is advanced over the cannula.

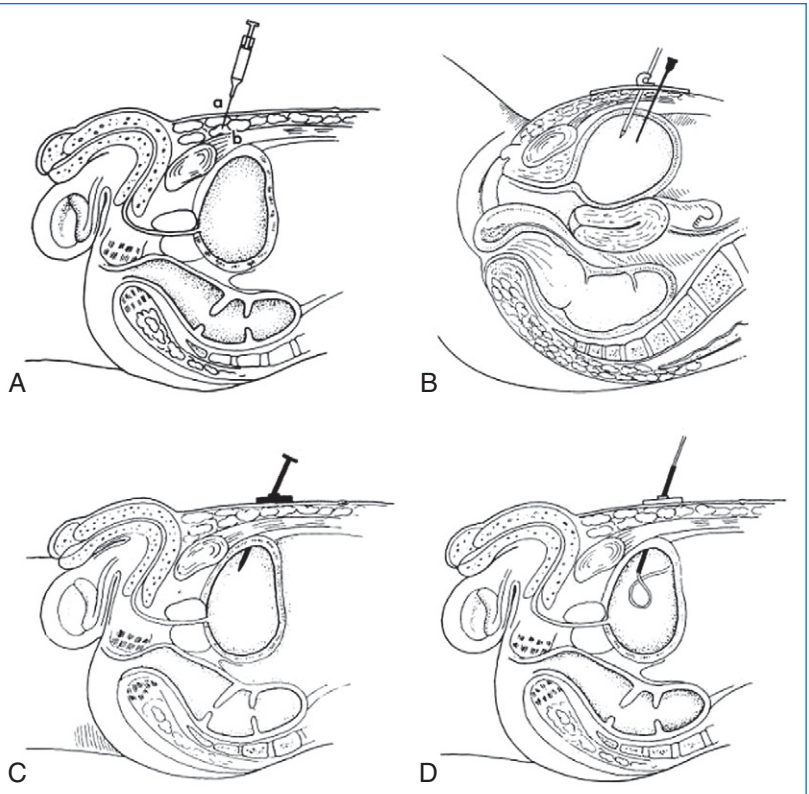

Figure 37-1. Suprapubic catheterization. (From Roberts J, Hedges J: *Clinical procedures in emergency medicine*, Philadelphia, 2000, W. B. Saunders.)

KEY POINTS: TREATMENT OPTIONS FOR AUR

1. Foley catheter placement
2. Coudé catheter placement
3. Filiforms and followers
4. Suprapubic catheterization

13. What diagnostic studies are useful in the evaluation of AUR?

Bedside ultrasonography can be helpful during the initial evaluation, and, if needed, can facilitate suprapubic aspiration. Always check a urinalysis with microscopic examination and urine culture. It is generally recommended to check blood urea nitrogen and creatinine levels to evaluate renal function, especially in cases of suspected chronic retention.

14. Which medications may cause AUR?

Table 37-1 presents the broad categories, as well as some specific medications that can cause AUR.

TABLE 37–1. MEDICATIONS THAT CAN CAUSE ACUTE URINARY RETENTION

Sympathomimetics (Alpha-Adrenergic)	**Antipsychotics**
Ephedrine	Haloperidol
Pseudoephedrine (Sudafed, Actifed)	Chlorpromazine (Thorazine)
Phenylephrine hydrochloride (Neo-Synephrine)	Prochlorperazine (Compazine)
Phenylpropanolamine hydrochloride (Contac)	Risperidone (Risperdal)
Amphetamine	Clozapine (Clozaril)
Cocaine	Quetiapine (Seroquel)
Sympathomimetics (Beta-Adrenergic)	**Antihypertensives**
Isoproterenol	Nifedipine (Procardia)
Terbutaline	Hydralazine
Antidepressants	Nicardipine
Tricyclic	**Muscle Relaxants**
Fluoxetine (Prozac)	Diazepam (Valium)
Antidysrhythmics	Cyclobenzaprine (Flexeril)
Quinidine	**Narcotics**
Disopyramide (Norpace)	Morphine sulfate
Procainamide	Codeine
Anticholinergics	Meperidine (Demerol)
Antihistamines	Hydromorphone hydrochloride (Dilaudid)
Antiparkinsonian Agents	**Miscellaneous**
Benztropine (Cogentin)	Indomethacin
Amantadine (Symmetrel)	Metoclopramide (Reglan)
Levodopa (Sinemet)	Carbamazepine (Tegretol)
Trihexyphenidyl (Artane)	Mercurial diuretics
Hormonal Agents	Dopamine
Progesterone	Vincristine
Estrogen	MDMA
Testosterone	Cannabis

MDMA, 3,4-methylenedioxymethamphetamine.

15. Summarize the different neurogenic causes of AUR.

- **Upper motor neuron lesions**: Lesions located in the spinal cord above the sacral micturition center (L2 vertebral level, S2–S4 spinal segments) result in a spastic or reflex bladder. Common causes are spinal cord trauma, tumor, and multiple sclerosis. Lesions of the cerebral cortex (e.g., acute stroke, bleed) usually cause chronic loss of bladder control and incontinence, except in the acute phase, when the lesions typically produce AUR.
- **Lower motor neuron lesions**: Lesions at the micturition center in the cauda equina interrupt the sacral reflex arc and produce vesical dysfunction. There is loss of sensation of bladder fullness leading to overstretch, muscle atony, and poor contraction. Large residuals are common. The most common causes include spinal trauma, tumor, herniated intervertebral disks, and multiple sclerosis.
- **Bladder afferent and efferent nerve dysfunction**: Dysfunction in this pathway disrupts the micturition reflex arc that is necessary for proper urination, causing AUR. Common causes include diabetes mellitus, herpes simplex infection, and the postoperative state.

16. Name the most common complications of AUR.

Infection, hemorrhage, and postobstruction diuresis. All three are more common in patients with chronic urinary retention.

17. What is autonomic dysreflexia/hyperreflexia, and what does it have to do with AUR?

An abnormality of the autonomic nervous system seen in patients with long-standing cervical or high thoracic spinal cord lesions (i.e., quadriplegics and high paraplegics). It is caused primarily by unchecked reflex sympathetic discharge secondary to visceral or somatic stimuli below the level of the spinal injury. This potentially life-threatening syndrome includes severe paroxysmal hypertension, diaphoresis, tachycardia or bradycardia, anxiety, headache, flushing, seizures, and coma. Morbidity has resulted from cerebrovascular accident, subarachnoid hemorrhage, and respiratory arrest. One of the most common precipitating stimuli is overdistention of the bladder (AUR) from a plugged or kinked catheter. Therefore, it is always important to evaluate these types of patients for potential Foley catheter problems.

18. What is postobstruction diuresis? How is it managed?

The inappropriate excretion of salt and water after relief of urinary obstruction. Patients with abnormal renal function or chronic urinary retention are most susceptible. A physiologic diuresis is normal because the kidneys excrete the overload of solute and volume retained while obstructed. If urine output persists at high levels, significant fluid and electrolyte abnormalities may develop. Any patient who exhibits a continuous diuresis after clinical euvolemia is reached requires hospitalization for hemodynamic monitoring and fluid and electrolyte repletion.

19. Who can I send home? Who needs admission? Can I remove that catheter?

Most patients with AUR caused by an obstruction require Foley catheterization with continuous drainage. Reliable patients in good health and without signs of serious systemic infection are candidates for careful outpatient management with a leg bag and timely urologic follow-up. The use of prophylactic antibiotics in these patients is controversial. Patients with new neurogenic causes, severe infection, systemic toxicity, or any lesion that may need surgical intervention require hospital admission. Some younger patients with pharmacologic urinary retention may have the catheter removed after decompression. The causative medication should be discontinued, and the patient should be discharged with instructions to return if symptoms recur. If the catheter is removed, it is prudent to be sure that patients can void on their own prior to discharge from the ED.

CONTROVERSY

20. I have heard that gradual emptying of the distended bladder best helps to prevent complications. Is this true?

Traditionally, the medical literature has recommended gradual emptying of the obstructed, distended bladder to decrease the risk of hematuria, hypotension, and postobstructive diuresis. The validity of this practice has long been questioned and inadequately studied. Recently, however, one study reviewed all of the available literature for each of these complications and compared quick, complete decompression with gradual emptying. Their review revealed that, although hematuria, transient hypotension, and postobstructive diuresis occasionally do occur after rapid emptying of the bladder, they are rarely of any clinical significance and do not require any treatment. The recommendation is that gradual, incremental bladder decompression is unnecessary.

WEBSITE

Urinary obstruction: www.emedicine.com

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CHAPTER 38

URINARY TRACT INFECTION: CYSTITIS, PYELONEPHRITIS, AND PROSTATITIS

Sara M. Krzyzaniak, MD

1. Define the terms relevant to the spectrum of urinary tract infection (UTI).

- **Bacteriuria**: The presence of bacteria anywhere in the urinary tract. *Significant bacteriuria* is defined as $>10^5$ colony-forming units (CFU) per mL of urine.
- **Cystitis**: Significant bacteriuria with bladder mucosal invasion, characterized by dysuria, urgency, frequency, and sometimes suprapubic discomfort. This term is often used interchangeably with *UTI* in clinical practice.
- **Pyelonephritis**: Infection of the renal parenchyma and collecting system, characterized by flank pain, fever, and significant bacteriuria
- **Urethritis or acute urethral syndrome**: The clinical syndrome of dysuria, frequency, and urgency in the absence of significant bacteriuria
- **Prostatitis**: A chronic or acute syndrome of prostatic inflammation that presents with a wide range of symptoms

KEY POINTS: CHARACTERISTICS OF CYSTITIS

1. Dysuria
2. Frequency
3. Urgency
4. Suprapubic discomfort
5. Significant bacteriuria

2. What are the most common causes of UTI?

Escherichia coli is the most common pathogen. *Staphylococcus saprophyticus* is the second most common. In immunocompromised or chronically ill patients and in complicated UTI, *Pseudomonas* and many members of the Enterobacteriaceae family, such as *Klebsiella*, *Proteus*, and *Enterobacter*, are commonly involved. *Proteus* and *Klebsiella* predispose to stone formation and grow more frequently in patients with calculi. Remember these organisms by the mnemonic SEEK PP.

3. What is asymptomatic bacteriuria?

The presence of greater than or equal to 10^5 uropathogens/mL on voided midstream urine from an asymptomatic patient. When two consecutive specimens contain the same organism at this concentration, the probability of true bacteriuria rises to 80% to 95%.

4. When should asymptomatic bacteriuria be treated?

- **Pregnancy**: associated with 20% to 40% risk of developing symptomatic UTI, including pyelonephritis. Pyelonephritis during pregnancy is associated with increased risk of prematurity and low birth weight.
- **Preoperatively**: treating urologic surgical patients prior to surgery decreases the risk of postoperative complications, including bacteremia.

- There is no evidence to support the treatment of asymptomatic bacteriuria in catheterized patients or in the elderly; this results in development of resistant pathogens.

5. List the differential diagnoses of dysuria.

- **Infectious**: Cystitis, urethritis (gonococcal versus nongonococcal), pyelonephritis, epididymitis, prostatitis, vulvovaginitis
- **Structural**: Calculi, neoplastic lesions
- **Traumatic**: Blunt trauma, sexual intercourse or assault, chemical irritants, allergy

6. When should a pelvic examination be done in a female patient with dysuria?

Whenever there is a suspicion that the cause is not a classic UTI. Clinical situations include external dysuria suggestive of vulvovaginitis, low abdominal pain or bilateral flank pain to rule out pelvic inflammatory disease, any history of trauma or chemical irritant, and any patient at high risk for a sexually transmitted disease or sexual abuse. Any patient who fails to respond to empirical antibiotic therapy for cystitis or who has a negative urinalysis or cultures with a suspected UTI should have a pelvic examination.

7. What is a routine urinalysis?

There is no standardization of what constitutes a *routine urinalysis* in the literature. The definition of *significant bacteriuria* depends on the relatively costly and slow results of urine culture. Many screening tests have been evaluated to try to detect UTI earlier and to predict negative cultures, reducing the number of full urine cultures ordered. These screening tests include the following:

- **Pyuria**: The measurement of white blood cells (WBC) in the urine, commonly by microscopic examination of centrifuged urine sediment and quantification of WBCs per high-power field (HPF). More than five WBCs/HPF is abnormal.
- **Microscopic evaluation for bacteria**: Variable techniques include examination of unstained and Gram-stained specimens of centrifuged and uncentrifuged urine. Standardization of this technique is poor, and it is an insensitive test because pathogens in quantities less than 10^4 CFUs/mL are difficult to find by this technique.
- **Epithelial cells**: Estimates of epithelial cells per HPF are used mainly to estimate perineal contamination of midstream specimens. Although epithelial cells can be derived from anywhere in the urinary tract, their presence on urinalysis is usually from vaginal epithelial cells and suggests contamination.
- **Leukocyte esterase**: An enzyme found in neutrophils. This test depends on the ability of any leukocytes present to convert indoxyl carboxylic acid to an indoxyl moiety. When positive, it is suggestive of but not confirmatory for pyuria.
- **Nitrite**: Produced from nitrate by nitrate reductase, an enzyme present in gram-negative bacteria. To be positive, the bacteria must act on the urine for 6 hours, making a first-voided morning specimen necessary for optimal testing. It is a specific but not sensitive test for UTI.

8. What is the utility of urinalysis and urine dipsticks in the diagnosis of UTI?

The reported sensitivities, specificities, and likelihood ratios for the previously mentioned screening tests vary widely in the literature. The pretest probability of cystitis in a population of patients presenting with any symptoms of dysuria, frequency, or urgency has been estimated to be approximately 70%. Estimates of screening test sensitivities and specificities vary so much that the predictive value of a positive test ranges from 75% to 99%, and the predictive value of a negative test ranges from 40% to 99%. Evidence suggests that urinalysis and dipstick testing done under practice conditions are not as reliable as when done under research protocol conditions. As a result, it may be sensible to continue to develop clinical guidelines involving the empiric treatment of uncomplicated UTI, limiting the use of screening tests to only patients with low-to-moderate pretest probability estimates.

9. When should I order a urine culture?

A urine culture generally is not required to treat presumptively uncomplicated cystitis in women. Most clinicians recommend a culture with sensitivities in suspected pyelonephritis

because of the potential for serious sequelae if an inappropriate antibiotic is used. All cases of potentially complicated UTI should also have a urine culture done.

10. What is the difference between complicated and uncomplicated UTI?

A complicated infection is associated with any neurologic, structural, or comorbid condition that increases the risk for acquiring infection and subsequently reduces the efficacy of standard treatment regimens. Factors that predispose to a complicated UTI include structural abnormalities (e.g., calculi, urinary catheters, stents, prostatic infection, and urinary diversion procedures), metabolic or hormonal abnormalities (e.g., diabetes or pregnancy), vesicoureteral reflux, immunocompromise, recent urinary tract instrumentation, male gender, extremes of age (the elderly, young children), unusual pathogens, recent antibiotic use or failed treatment for UTI, and presence of symptoms for longer than 7 days. It can be argued that true uncomplicated UTIs occur only in nonpregnant, healthy women with no neurologic or structural dysfunction, with 80% of UTIs falling into this group.

KEY POINTS: CHARACTERISTICS OF COMPLICATED UTIS

1. Structural or neurological abnormality
2. Immunocompromised host
3. Recent instrumentation
4. Failed treatment
5. Male gender or extremes of age
6. Stone or obstructive pathology

11. How should acute, uncomplicated cystitis be treated?

The causative agents are quite predictable, and treatment should be tailored to the specific bacterial sensitivities and patterns of resistance identified at each institution. The Infectious Diseases Society of America (IDSA) has recommended that a 3-day regimen of trimethoprim-sulfamethoxazole (TMP-SMX) be standard therapy for acute uncomplicated cystitis. Fluoroquinolones, such as ofloxacin, levofloxacin, or ciprofloxacin, for 3 days are considered to have similar effectiveness as TMP-SMX but are considerably more expensive. Also, in an effort to postpone emergence of resistance to these drugs, the IDSA does not recommend them as initial empirical therapy except in regions with known resistance to TMP-SMX of greater than 10% to 20% among uropathogens. Nitrofurantoin for 7 days or a single dose of fosfomycin may become more useful as resistance to TMP-SMX increases.

12. What is the ideal length of treatment for uncomplicated, symptomatic cystitis?

Multiple studies have examined ideal length of treatment for uncomplicated, symptomatic cystitis. Regimens from 1 to 14 days have been investigated. Most studies recommend a course of antibiotics ranging from 3 to 6 days. This includes elderly women and children.

13. Explain the role of adjunctive treatment with phenazopyridine.

Phenazopyridine (Pyridium) acts as a topical analgesic for the urinary tract and can help relieve the dysuria associated with symptomatic UTIs. Patients should be advised that it will turn body secretions/excretions orange, including their urine and tears. Dosing is 200 mg three times daily after meals for 48 hours.

14. How should acute, uncomplicated pyelonephritis be treated?

The IDSA recommends 14 days of antimicrobial therapy for young, nonpregnant women with normal urinary tracts. In mild cases, oral fluoroquinolones are considered the first choice for

outpatient treatment of pyelonephritis (e.g., ciprofloxacin 500 mg every 12 hours, levofloxacin 250 mg daily, or ofloxacin 200–300 mg every 12 hours). TMP-SMX for 14 days is an inexpensive option, but because of increasing antibiotic resistance to this agent in pyelonephritic strains, the IDSA recommends it be used only if the organism is known to be susceptible. In patients with a contraindication to fluoroquinolones, alternatives include cefixime 400 mg daily and cefpodoxime proxetil 200 mg every 12 hours for 14 days. A single-dose parenteral antibiotic (gentamicin or ceftriaxone) followed by any of the aforementioned regimens is an acceptable alternative.

KEY POINTS: CHARACTERISTICS OF PYELONEPHRITIS

1. Fever
2. Flank pain
3. Costovertebral angle tenderness
4. Significant bacteriuria

15. Which patients with pyelonephritis should be admitted?

Admission should be strongly considered for patients who are unable to maintain oral hydration or take oral medications and patients with uncertain social support or concern about compliance. Other indications include immunocompromise, severe illness with extreme pain, or marked debility. There is increasing evidence that some subsets of complicated pyelonephritis can be treated successfully on an outpatient basis. Pregnant patients at less than 24 weeks' gestation who are hemodynamically stable have been treated safely as outpatients if they can be reached easily by phone and are likely to be compliant with medication and follow-up. Another reasonable option is 24-hour observation for intravenous hydration and antibiotics with subsequent discharge home on a 10- to 14-day course of appropriate antibiotics with close follow-up.

16. When should emergency imaging of the urinary tract be obtained in acute pyelonephritis?

If the patient remains febrile for more than 72 hours on appropriate antibiotic therapy, computed tomography (CT) or ultrasound should be considered to rule out obstruction and renal or perinephric abscess. Contrast-enhanced CT scans provide the highest sensitivity for identifying abscess, obstruction, and acute inflammation.

KEY POINTS: FACTORS THAT MAY REQUIRE ADMISSION FOR PATIENTS WITH PYELONEPHRITIS

1. Pregnancy
2. Severe illness
3. Inability to maintain oral hydration
4. Uncertain social support/compliance
5. Complicated UTIs

17. How does treatment change for cases of complicated UTI?

- **Duration**: A longer course of 7 to 10 days should be prescribed.
- **Pregnancy**: Appropriate antibiotic choices include amoxicillin, cephalexin, or nitrofurantoin. TMP-SMX should not be used in the first two trimesters. Duration is 3 days for asymptomatic bacteria, otherwise treat as per recommendations for complicated UTIs.

18. What are the signs and symptoms of acute bacterial prostatitis?

The presentation can be dramatic and usually includes frequency, urgency, dysuria, and some obstructive voiding symptoms in greater than 80% of patients. Other common complaints include fever (60%), rigors, myalgias, and perineal discomfort (38%). Some patients also complain of low back pain or rectal pain. The prostate is warm, swollen, and extremely tender.

19. How is acute prostatitis managed?

The pathogen usually can be isolated from voided urine. Prostatic massage and urethral catheterization should be avoided because they are painful and may precipitate bacteremia. Severely ill patients should be admitted and treated with intravenous antibiotics. An aminoglycoside-penicillin derivative combination is often used. Less severely ill patients respond well to oral fluoroquinolones or TMP-SMX. In one study, more than 95% of cultured organisms were sensitive to aminoglycosides, cephalosporins, ciprofloxacin, and imipenem as opposed to 83% sensitivity to TMP-SMX. The duration of therapy should be at least 30 days to prevent chronic bacterial prostatitis. Supportive measures include hydration, nonsteroidal anti-inflammatory drugs, sitz baths, and stool softeners. If urinary retention is a problem, suprapubic aspiration or suprapubic catheter placement is recommended.

KEY POINTS: DURATION OF ANTIBIOTIC TREATMENT FOR UNCOMPLICATED DISEASE

1. Cystitis: 2-4 days
2. Pyelonephritis: 14 days
3. Prostatitis: At least 30 days

20. Name the most common cause of recurrent UTI in men.

Chronic bacterial prostatitis.

21. What are the signs and symptoms of chronic bacterial prostatitis?

This is a syndrome of relapsing subacute illness characterized by mild symptoms of frequency, urgency, and dysuria. Other symptoms may include back pain, scrotal or perineal pain, voiding dysfunction, hematospermia, and painful ejaculation. Fever and rigors should not occur. Symptoms must be present for more than 3 months. Examination is highly variable and may be normal. A premassage and postmassage of the prostate urinalysis generally should be done and show repeated postmassage bacteriuria with the same organism.

22. How is chronic bacterial prostatitis treated?

Treatment is difficult with poor cure rates and frequent relapse. Several studies report initial treatment success with TMP-SMX. However, in the event of failure, prolonged treatment (2–3 months) with fluoroquinolones is recommended. Recalcitrant infection may require long-term, low-dose therapy or resection of the prostate. Referral to a urologist generally is recommended, if possible, before treatment with antibiotics. α_1-blocking agents show some promise for relief of symptoms and prevention of recurrence.

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CHAPTER 39

CHRONIC RENAL FAILURE AND DIALYSIS

Allan B. Wolfson, MD

1. Isn't renal failure just another genitourinary disorder?

No. **Chronic kidney disease (CKD)** is a complex multisystem disorder. The absence of renal function has obvious consequences for the regulation of total body fluid and electrolyte balance, limiting the ability to handle fluid and electrolyte loads. CKD also results in subtle metabolic abnormalities, such as glucose intolerance and lipid disturbances. Renal failure is associated with numerous end-organ effects, ranging from pericarditis to renal osteodystrophy, that compromise comfort and normal function.

2. What are the special concerns in patients with renal failure?

Iatrogenic illness is one important consideration, whether arising from overadministration of fluids or from drug toxicity. Because the effects of renal failure on drug metabolism and disposition are often complex, it is always advisable to check recommended dosage adjustments for patients with CKD before administering or prescribing medications. Even apparently innocuous drugs, such as antacids and cathartics, may cause morbidity and mortality if used improperly. Patients with CKD have complications both from the underlying disease that caused renal failure and from complications of dialysis therapy. They have a limited capacity to respond to infection, trauma, or other intercurrent illnesses.

3. How is hemodialysis performed?

In hemodialysis, the patient's blood is brought into contact with a semipermeable artificial membrane, on the other side of which is a chemically balanced aqueous dialysis solution. Metabolic waste and electrolytes flow from the patient's blood into the dialysate, and other substances (e.g., calcium) may flow from the dialysate into the blood, acting to normalize blood chemistries. To achieve adequate total body clearances over the time available for hemodialysis, a high blood-flow rate is necessary. This requires the cannulation of large vessels or, for long-term dialysis, the creation of an artificial vascular access site that can be used repeatedly. Hemodialysis typically is performed for 3 to 5 hours, three times per week.

4. How is peritoneal dialysis (PD) performed?

The patient's peritoneal membrane serves as the semipermeable barrier between the blood (in the peritoneal capillaries) and a balanced dialysate solution. The latter is introduced into the patient's peritoneal cavity and allowed to dwell for a period of hours before being drained and replaced. An osmotic gradient is created by using a dialysate with high concentrations of glucose that, through osmosis, pulls water from the intravascular space into the dialysate, acting to correct volume overload. For patients with CKD on **chronic ambulatory peritoneal dialysis (CAPD)**, about 2 L of dialysate dwells continuously within the peritoneal cavity. It is exchanged for fresh dialysate in a sterile fashion by the patient four times a day. In another type of peritoneal dialysis, termed **automated peritoneal dialysis (APD**) or **continuous cycling peritoneal dialysis (CCPD)**, exchanges are performed by an automatic cycling machine. For both types, special peritoneal access is required in the form of a surgically implanted Teflon catheter (Tenckhoff catheter), through which dialysate is infused and drained.

5. What is the most common problem relating to the vascular access device in the ED?

Thrombosis should be suspected when patients report loss of a pulse or thrill in the vascular device. More often, they present to the ED when there has been a problem establishing adequate flow during a hemodialysis session. The only intervention necessary is a prompt call to a vascular surgeon. An angiogram defines the nature and extent of the obstruction and delineates anatomic lesions, allowing the surgeon to revise or replace the access.

KEY POINTS: PROBLEMS WITH VASCULAR ACCESS DEVICES

1. Thrombosis
2. Hemorrhage
3. Infection (often inapparent)

6. How do I diagnose and treat a vascular access infection?

Infection is obvious when the patient presents with signs of inflammation localized to the access site. The difficulty is that many patients present only with fever and without specific localizing signs. A useful rule of thumb in such instances is to assume that an endovascular access infection is present and to treat accordingly. After blood cultures are obtained, patients typically can be sent home after one dose of an appropriate antibiotic, provided that they look well and are reliable for follow-up. A single dose of vancomycin, 1 g intravenously, is the treatment of choice because most infections are staphylococcal and the drug's duration of action is 5 to 7 days in CKD. Vancomycin is not hemodialyzable, and its major toxicity is to the kidneys. If gram-negative infection is suspected, a third- or fourth-generation cephalosporin, aztreonam, or an aminoglycoside should be added to the regimen. Careful follow-up should be arranged with the patient's dialysis nurse or physician.

7. When can the vascular access device be used for giving intravenous (IV) infusions or for drawing blood?

Hemodialysis patients are instructed never to allow their blood pressure to be taken in the arm with the vascular access site or to allow their blood to be drawn or IV fluids to be infused through the vascular access. This is to protect the access device, which is truly the patient's lifeline. Occasionally, however, there is no reasonable alternative but to use the access device for blood drawing or IV lines. In these situations, cautious use of the vascular access device is permissible, provided that certain guidelines are followed.

When using the access site to draw blood, a tourniquet should not be used. At most, one finger can be used to tourniquet the vein lightly. The presence of a thrill should be documented before and after the procedure. The area should be cleaned thoroughly with a topical antiseptic, and sterile technique should be observed. Care should be taken not to puncture the back wall of the vessel, and after the puncture, firm but nonocclusive pressure should be applied to the site for several minutes to ensure that extravasation does not occur. Obvious aneurysms should not be punctured.

When using the vascular access for an IV line, similar precautions should be observed. Because the vessel is under arterial pressure, a pressure bag or, preferably, an automated infusion device is an absolute requirement (certainly when infusing medications).

8. How is PD-associated peritonitis diagnosed?

Peritonitis associated with PD occurs about once per year in even the most fastidious and well-motivated patients. In contrast to other types of peritonitis, it tends to be mild clinically, and most patients can be managed without hospital admission. PD-associated peritonitis is caused most commonly by gram-positive organisms, which are thought to be introduced

during the exchange procedure. The diagnosis is often suspected by the patient on the basis of the new appearance of cloudiness of the dialysis effluent. Patients are instructed to seek medical attention promptly when this occurs, and for this reason most episodes of peritonitis are relatively mild. If the patient delays seeking medical attention, however, the symptoms tend to become progressively more severe. Most patients have abdominal pain and tenderness, but only a few have fever, nausea, vomiting, or even (at least early on) an elevated peripheral white blood cell count. Localized peritoneal findings are suggestive of an acute surgical abdomen rather than PD-associated peritonitis.

9. How is PD-associated peritonitis treated?

When fluid has been obtained from the effluent bag and laboratory studies have confirmed the presence of a significant number of white cells (>100 cells/mm^3 with >50% polymorphonuclear leukocytes) or a positive Gram stain, antibiotic treatment is initiated. Commonly, vancomycin (30 mg/kg) is given intraperitoneally and may be repeated weekly. Gram-negative coverage, with a third-generation cephalosporin, aztreonam, or an aminoglycoside, can be added if thought appropriate. These may be given intraperitoneally as well and should be followed by daily intraperitoneal maintenance doses as an outpatient. Usually, each center has its own protocols for treatment, so the patient's nephrologist or dialysis nurse should be consulted. Follow-up should be in 48 hours, at which time cultures and clinical findings are rechecked and therapy adjusted as necessary. Admission criteria include severe pain, nausea and vomiting, a toxic appearance, or the inability of the patient to comply with outpatient therapy and follow-up.

KEY POINTS: PD-ASSOCIATED PERITONITIS

1. Diagnosis: Cloudy dialysate effluent, abdominal pain, fever
2. Treatment: Typically with intraperitoneal antibiotics

10. What are the indications for emergency dialysis?

Acute pulmonary edema, life-threatening hyperkalemia, or life-threatening intoxication or overdose secondary to dialyzable toxins that ordinarily are excreted by the kidneys.

11. What is unique about a dialysis patient with cardiac arrest?

Two potentially reversible entities always should be considered in a CKD patient with cardiac arrest:

- **Severe hyperkalemia** may cause severe rhythm disturbances and ultimately cardiac arrest without any other warning or clinical signs. When a patient suffers an arrest from whatever cause, respiratory and metabolic acidosis and the efflux of potassium from cells can be expected to produce hyperkalemia secondarily. In the patient who already may have a tendency toward hyperkalemia, this further increase could cause the patient to be refractory to standard advanced cardiac life support (ACLS) interventions. CKD patients in cardiac arrest always should be given IV calcium if they do not respond immediately to the first round of ACLS measures.
- **Acute pericardial tamponade** may result from the accumulation of pericardial fluid or spontaneous bleeding into the pericardial sac. Patients with tamponade tend to display refractory hypotension or pulseless electrical activity, or both. Although less likely than other entities to be the cause of refractoriness to resuscitation measures, the possibility of pericardial tamponade always should be considered in patients in whom other measures have failed. Bedside ultrasound may be diagnostic and emergency pericardiocentesis may be life saving.

KEY POINTS: INDICATIONS FOR EMERGENCY DIALYSIS

1. Acute pulmonary edema
2. Life-threatening hyperkalemia
3. Life-threatening intoxication or overdose with agents normally excreted by the kidneys

12. What are the treatment options for acute pulmonary edema in patients with CKD?

CKD patients with pulmonary edema do not have the ability to rid themselves of excess fluid through the kidneys and ultimately require dialysis to correct volume overload. Most of the interventions that are useful in patients with functioning kidneys also are useful in patients with CKD while awaiting the initiation of acute dialysis. The patient should be given oxygen and placed in a sitting position. Nitrates administered sublingually or intravenously are the mainstay of temporizing therapy. Sublingual nitroglycerin can be given every 3 minutes to decrease preload and afterload as blood pressure permits. IV nitroglycerin is a useful alternative. IV morphine, although less popular, also may be helpful in decreasing pulmonary venous hypertension, although patients may be more likely to require intubation and mechanical ventilation because of its sedative action.

Dialysis is the definitive therapy and should be instituted as early as possible. The PD patient with acute pulmonary edema presents a slightly different problem because intensified dialysis, even with 4.25% glucose solution, is a slow means of removing fluid and because the presence of 2 L of dialysate in the peritoneal cavity tends to have an adverse effect on diaphragmatic excursion and pulmonary mechanics. Intubation and mechanical ventilation may be necessary while continuing hourly exchanges of high-concentration dialysate.

13. How should I treat hyperkalemia in a dialysis patient?

The approach is similar to that taken with nondialysis patients. IV calcium gluconate (10 mL of a 10% solution) acts rapidly to antagonize the cardiotoxic effects of hyperkalemia (without affecting the serum potassium level), but its effects last for only a few minutes. It should be used only as a temporizing measure in patients with cardiovascular compromise or a widened QRS complex on the electrocardiogram (ECG).

Nebulized albuterol (10 to–20 mg by inhalation) acts within a few minutes to shift potassium into cells. It is easy to administer, generally has minimal side effects, and is effective for a few hours. The dose can be repeated as necessary.

Glucose and insulin (typically 50 g and 0.1 units/kg, respectively, as a slow IV infusion) also move potassium into cells but require close serial monitoring of blood glucose levels.

IV sodium bicarbonate (50 mEq over 5 minutes) has a similar action but can exacerbate volume overload and can acutely decrease the ionized calcium.

If dialysis is not immediately available to remove potassium from the body, sodium polystyrene sulfonate (Kayexalate), a sodium-potassium exchange resin typically given orally with sorbitol to enhance passage through the gut, can remove significant amounts of potassium from the body. It is slow-acting, however, and should be reserved for situations in which the patient is stable but requires continuing control of the serum potassium over hours, until dialysis can be performed.

In all cases of acute hyperkalemia, the serum potassium level should be checked frequently, and continuous ECG monitoring is mandatory until definitive treatment with dialysis can be initiated.

KEY POINTS: TREATMENT OF HYPERKALEMIA

1. IV calcium
2. Inhaled albuterol
3. IV glucose and insulin
4. IV sodium bicarbonate
5. Oral or rectal Kayexalate
6. Dialysis

14. What about air embolism?

Although air embolism has become rare with the advent of sophisticated monitoring and alarm systems on hemodialysis machines, when it does occur it is often a devastating event and one for which the patient almost surely will be brought to the nearest ED.

Air embolism should be suspected when a patient experiences a sudden acute decompensation during the course of a hemodialysis treatment. Several immediate measures are thought to be helpful. Any IV lines should be clamped. The patient should be given 100% oxygen and laid on the left side with the head down, in an attempt to cause the air to collect at the apex of the right ventricle. At this point, if the patient is reasonably stable, an interventional radiologist or cardiologist can be consulted for consideration of passage of a central venous catheter into the right ventricular apex, allowing the air to be aspirated directly out of the heart. For patients who are in close proximity to a hyperbaric chamber, treatment with 100% oxygen at several atmospheres can shrink the size of the bubbles and enhance resorption of the gas. One should be certain before embarking on this course, however, that the patient's symptoms are due to air embolism rather than, for example, a sudden spontaneous pneumothorax.

15. How should a patient with acute shortness of breath be evaluated?

The rule of thumb is to dialyze CKD patients who are short of breath because volume overload is the most common cause. It is sometimes difficult to make the diagnosis of volume overload. The patient's weight may be the best guide. Physical examination is not always helpful, and chest radiographs may be misleading.

16. What are the main differential diagnostic considerations for chest pain in CKD?

Always think first of either angina or pericarditis. Some patients with CKD, particularly those who are anemic, may have angina and cardiac ischemia even if a previous cardiac catheterization has shown a *noncritical* coronary obstruction. This is due to increased cardiac oxygen demands and decreased oxygen delivery to the heart. Although cardiac enzyme levels may be altered in CKD, renal failure does not obscure the usual ECG and enzyme changes of acute myocardial infarction.

17. What is the differential diagnosis of hypotension in a patient with end-stage renal disease (ESRD)?

The most common entities are hypovolemia after dialysis, sepsis, hemorrhage, and acute pericardial tamponade.

KEY POINTS: MOST COMMON CAUSES OF HYPOTENSION IN DIALYSIS PATIENTS

1. Hypovolemia after dialysis
2. Sepsis
3. Hemorrhage
4. Pericardial tamponade

18. What are the major causes of altered mental status in patients with ESRD?

Dysequilibrium syndrome, caused by rapid solute shifts during hemodialysis, is a consideration, but a major pitfall is to attribute every change in mental status to this entity. Drug effects are a major cause of altered mental status, as is spontaneous intracranial hemorrhage. Any patient with localizing signs should have a computed tomography (CT) scan of the head; patients without localizing signs should also undergo CT scanning, however, because subdural hematoma may not cause focal findings.

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HEMOSTASIS AND COAGULOPATHIES

Thomas B. Barry, MD and Kathryn Getzewich, MD

1. What is meant by hemostasis?

Hemostasis is a balance between excessive bleeding and thrombosis. It is the active process of clot formation and degradation in response to injury of a blood vessel. This response normally occurs through the coordinated efforts of the blood vessel endothelium, platelets, the clotting factor cascade, and fibrinolysis.

KEY POINTS: THREE PHASES OF HEMOSTASIS

1. Platelet plug formation
2. Propagation of the coagulation cascade and fibrin clot formation
3. Balanced fibrinolysis

2. Is hemophilia the main cause of hemostatic abnormality?

Most hemostatic abnormalities result from drugs such as heparin, warfarin, and aspirin or from associated disease such as liver or kidney failure. The hemophilias are important but less common.

3. Do I really need to know the whole clotting cascade to manage patients?

A working knowledge should include the basics of the three phases of hemostasis, some key clotting factors, and familiarity with basic testing and therapeutics.

- In **primary hemostasis**, after injury, platelets and von Willebrand factor (vWF) from the endothelium interact to form a plug (platelet adhesion). Platelet activation and aggregation occur along with vessel constriction. Disorders include problems with platelet quantity and function, as well as vWF problems and vascular abnormalities such as hereditary telangiectasia. Platelet count and bleeding time are used to assess this phase of hemostasis.
- In **secondary hemostasis**, the platelet plug is reinforced with cross-linked fibrin from the coagulation cascade (factor XIII causes covalent cross-links). Effective functioning of the cascade may be impaired by deficiencies of coagulation factor activity (hemophilia A and B) or by inadequate factor production such as with warfarin use.
- In **tertiary hemostasis**, the fibrin clot is enzymatically broken down by plasmin. Endothelial cells release plasminogen activator, which converts plasminogen to plasmin. The plasmin breaks down fibrin and fibrinogen into fibrin split products and D-dimers. Excessive fibrinolytic activity or deficiencies of fibrinolytic inhibitors can increase bleeding. Because protein C and protein S are involved in the regulation of blood clotting, deficiencies can result in excessive intravascular clotting.

4. What are the intrinsic and extrinsic coagulation pathways? How can I tell the difference?

Prothrombin time (PT) is affected by the extrinsic (and common) pathways of the coagulation cascade and partial thromboplastin time (PTT) by defects in the intrinsic (and common) paths.

The extrinsic pathway is activated by tissue factor exposed at the site of injury. The intrinsic pathway is initiated by blood exposure to a negatively charged surface. A patient with a prolonged PTT and a normal PT is considered to have a defect in the intrinsic coagulation pathway. The name indicates that all of the components of the PTT test (except kaolin) are *intrinsic* to the plasma. On the other hand, a patient with a prolonged PT and a normal PTT has a defect in the "extrinsic" coagulation pathway (tissue factor being extrinsic to the plasma). Prolongation of both the PT and the PTT implies that the defect is in a common pathway. Both pathways converge to activate factor X, which activates prothrombin to thrombin.

5. What parts of the history and physical can help me assess a suspected bleeding abnormality?

It is important to ask about medications, previous medical history (especially liver, kidney, and malignant disease), previous problems with bleeding (such as with surgeries and dental work), and family history of bleeding disorders. In patients with known bleeding disorders, ask about the nature of their disease and previous therapies. They are frequently knowledgeable about their individual disease. Platelet disorders frequently result in petechia, purpura, epistaxis, and gum and other mucosal bleeding. They are common in women and usually acquired as opposed to congenital. Problems with coagulation are more commonly congenital, found more often in men, and are likely to present as deep muscle or joint bleeding. Coagulopathy is rarely the cause of epistaxis, menorrhagia, or gastrointestinal (GI) bleed.

6. How do I interpret PT, PTT, and international normalized ratio (INR)?

PT tests the factors of the extrinsic and common pathways. It is prolonged by deficiencies of prothrombin, fibrinogen, and factors V, VII, and X. A PT 2 seconds more than the control is significant. PTT tests all the intrinsic and common pathways, including all factors except VII and XIII. INR reduces interlaboratory variation by indexing thromboplastin test lot activity to an international standard. Liver disease, warfarin use, and other abnormalities of the vitamin K sensitive factors (i.e., II, VII, IX, X) affect the PT and INR. INR of 1 is normal. An INR between 2 and 3 indicates a therapeutic level of warfarin.

7. What are the causes of thrombocytopenia?

- Decreased production: Marrow disease, chemotherapy, alcohol or thiazide effect
- Immune destruction: Idiopathic thrombocytopenic purpura (ITP), systemic lupus erythematosus (SLE), lymphoma, quinine, quinidine, and postinfectious disease
- Toxic destruction: Disseminated intravascular coagulation (DIC), thrombotic thrombocytopenic purpura (TTP), hemolytic uremic syndrome (HUS), **h**emolysis with **e**levated **l**iver enzymes and **l**ow **p**latelets (HELLP) syndrome
- Splenic sequestration (hypersplenism; rare): Hematologic malignancy, portal hypertension, autoimmune hemolytic anemia, hereditary spherocytosis
- Dilution: Massive transfusion
- Lab error: It happens. "I'm shocked, shocked. . . ." (Claude Rains, *Casablanca*, Warner Brothers, 1942)

8. What are the differences between idiopathic and chronic thrombocytopenic purpura?

- **ITP** should be a diagnosis of exclusion after considering SLE, antiphospholipid syndrome, HIV, and lymphoproliferative disorders. It is associated with antiplatelet antibody immunoglobulin G (IgG). The acute form is seen in children 4 to 6 years old several weeks after a viral prodrome. It is self-limited with a 90% rate of spontaneous remission. Morbidity and mortality rates are low, and steroid therapy does not seem to alter the course.
- **Chronic ITP** is found in adults. It is three times more common in women than men. Severity waxes and wanes with only a 1% mortality, but spontaneous remission is rare. It may respond to therapy with glucocorticoids, intravenous (IV) immunoglobulin, and splenectomy if recurrent. Other treatments include plasmapheresis, androgen therapy with danazol, cyclophosphamide, azathioprine, vincristine, thrombopoietin, antiCD40 ligand, rituximab, and anti-D immunoglobulin (WinRho) in Rh-positive presplenectomy patients.

Platelet transfusion is reserved for life-threatening bleeds because it may increase antiplatelet antibodies.

9. What are the five clinical signs of TTP?

Only 40% of patients have all five:

- Fluctuating change in mental status
- Thrombocytopenia
- Fever (in 90% of patients)
- Microangiopathic hemolytic anemia
- Renal impairment

10. What causes TTP? Is it worse than ITP?

TTP results from subendothelial and intraluminal deposits of fibrin and platelet aggregation in capillaries and arterioles. Prostacyclin and abnormal platelet aggregation are thought to contribute to its origins. It may affect patients of any age or gender, although most are 10 to 40 years old and 60% are female. When untreated, there is 80% mortality at 3 months as a result of microthrombi in the heart, brain, and kidneys. Recently plasmapheresis has reduced this to 17%. Other therapies include steroids, splenectomy, α-globulin, vincristine, and antiplatelet agents such as aspirin and dipyridamole (Persantine). Platelet transfusions may cause additional microcirculatory thrombi and should be avoided unless bleeding is life-threatening.

11. What is hemolytic uremic syndrome (HUS)?

HUS is similar to TTP in that they both present with hemolytic anemia, fever, neurologic abnormality, and renal dysfunction. However, HUS causes less change in mental status and more renal dysfunction. Patients with HUS tend to be younger (children are more common than adults), and onset is often associated with a bacterial gastroenteritis such as *Escherichia coli* O157:H7 and *Shigella* spp.

12. Should I worry about thrombocytopenia during a large-volume blood transfusion?

Stored banked blood is platelet poor because platelets have a life span of only 9 days. Follow platelet counts after 10 units of blood. Transfuse platelets when the count is down to 50,000/mL.

13. How does aspirin increase bleeding?

Aspirin blocks cyclooxygenase, which decreases thromboxane formation leading to decreased platelet aggregation and less vasoconstriction. Aspirin poisons this reaction for the life of the platelet. Nonsteroidal anti-inflammatory drugs, such as indomethacin, have this effect only while in the circulation. Uremia has a similar reversible effect.

14. What are the indications for platelet transfusions?

As previously noted, platelet transfusion should be delayed in ITP and TTP to avoid disease-specific complications and alloimmunization. It is more commonly indicated for primary bone marrow problems. In a patient with a platelet count greater than 50,000/mL, hemorrhage due to the deficiency is unlikely. From 10,000 to 50,000/mL there is variable risk with trauma, ulcer, and invasive procedures. Choosing when to transfuse at these levels is not an exact science. Platelet transfusion is indicated with counts less than 10,000/mL because there is a significant risk of spontaneous hemorrhage. Each bag of random donor platelets may be expected to raise the platelet count 5,000/mL. They are usually ordered six at a time.

15. What is the most common inherited bleeding disorder?

It is von Willebrand's disease (5–10 cases per million population). It is usually autosomal dominant. There is a deficiency or dysfunction of vWF and a mild factor VIII defect. Treatment is with desmopressin in the mild, most common, type I form of the disease.

In more severe types, therapy is with cryoprecipitate based on the patient's factor VIII level. Usual dose is one to two bags/10 kg. In von Willebrand's this is found to stimulate factor VIII activity and less is needed in redosing.

16. Do people with hemophilia A have low levels of factor VIII?

It is the activity of factor VIII that is impaired, technically not its level. Seventy percent of cases are transmitted by sex-linked recessive (X chromosome) inheritance; 30% of cases are due to spontaneous mutation. Severe disease has less than 1% activity, and spontaneous bleeding (joints, deep muscles, urinary tract, and central nervous system [CNS]) is a problem. Between 1% and 5% activity is classified as moderate disease with problems occurring mostly after trauma and surgery. Above 5% is mild disease, but some trauma and surgical risks persist. PTT is only prolonged with less than 35% activity.

Pearl: 1 unit of factor VIII per kilogram increases the activity level by 2% (unless adversely affected by anti-factor VIII antibodies (IgG), which develop in 7% to 20% of patients). Recombinant DNA factor VIII is the replacement of choice and lacks the hepatitis B, C, and HIV risks of fresh frozen plasma (FFP) and cryoprecipitate.

17. How is factor VIII dosed in hemophilia A?

Twenty-five units per kilogram for moderate bleeding, 50 units/kg for severe hemorrhage or life-threatening bleeding site (GI, neck, sublingual, retroperitoneal, intra-abdominal, head injury, CNS bleed, and necessary surgical procedures). Because the half-life is 8 to 12 hours, redose with half the loading dose after 8 to 12 hours. Recombinant factor VIII unit concentration is noted on the label. Cryoprecipitate (from FFP) is assumed to be 80 to 100 units of factor VIII per bag.

18. What is Christmas disease?

Hemophilia B, which involves decreased factor IX activity. The clinical presentation is the same as hemophilia A. The genetic pattern is the same, although less prevalent in the population with only ⅕ the number of cases. Treatment is with factor IX, 50 U/kg or FFP.

Pearl: There is no factor IX in cryoprecipitate.

19. What does desmopressin (DDAVP) do?

DDAVP is a synthetic analog of antidiuretic hormone. It works by causing release of vWF from endothelial storage sites and increases levels of factor VIII in hemophilia A and some cases of von Willebrand's. The dose is 0.3 μg/kg IV; it lasts 4 to 6 hours and is most effective in mild to moderately deficient patients.

20. What factors are affected by vitamin K deficiency? Warfarin? Liver disease? Banked blood?

- Vitamin K deficiency affects II, VII, IX, and X, the same ones affected by warfarin.
- Hepatic insufficiency affects all factors except VIII.
- Stored blood is low in V, VIII, and platelets.

KEY POINTS: HEMOSTATIC DEFICIENCIES

1. With hemophilia A and B, the bleeding time is normal (as is the PT and the PTT in mild and moderate cases).
2. Bleeding time is increased with von Willebrand's disease.
3. PT reflects extrinsic pathway abnormality through factor VII deficient activity.
4. Factor VII has the shortest half-life of the factors (3–5 hours) and causes the first manifestations of production deficiency.
5. An INR of 2–3 is recommended with most warfarin therapy.
6. Deficiency of factors VIII, IX, and XI account for 99% of inherited bleeding disorders. If congenital bleeding disorder is suspected, FFP at 15 mL/kg will support hemostasis while a definitive diagnosis is being made.

21. What happens in DIC?

Platelets and clotting factors (especially V, VIII, and XIII) are consumed. Thrombin formation overwhelms fibrinolysis and activates fibrinogen. Fibrin is deposited in small vessels of multiple organ systems. Fibrin degradation products are released, and platelet function, as well as fibrin polymerization, are decreased.

Treatment is with platelets and FFP. Heparin may be used if fibrin deposition and thrombosis dominate the clinical picture.

22. What are heparin-induced thrombocytopenia (HIT) and heparin-induced thrombocytopenia with thrombosis (HITT)?

HIT type I is a nonimmune-mediated thrombocytopenia that usually resolves without treatment or complication. The more serious HIT type II (the form usually referred to when discussing HIT) is caused by antibodies to heparin/platelet factor 4 complex. It results in platelet activation and clot formation. It usually occurs 5 to 10 days after exposure to heparin but may occur after as few as 10 hours. It occurs in 2% of patients on unfractionated heparin anticoagulation therapy and less commonly with low-molecular-weight heparin. Platelet counts drop to 50,000 to 100,000/mL. HITT develops in 50% of the patients with HIT. HIT and HITT require discontinuation of heparin (including heparin flushes). Prophylactic platelet transfusions should be avoided. A direct thrombin inhibitor is indicated in patients with thrombosis (i.e., lepirudin or argatroban). Doppler ultrasound of the legs is indicated in HIT because recent studies have found subclinical deep venous thrombosis (DVT) in up to 50% of HIT patients.

23. Need help with HELLP?

HELLP criteria:

- Microangiopathic hemolytic anemia
- Serum aspartate transaminase levels greater than 70 U/L
- Platelets less than 100,000/mL

The HELLP syndrome is a form of preeclampsia. Gestational thrombocytopenia (100,000–150,000/mL) is found in 5% to 10% of third trimester pregnancies. It is even more common in pregnancies complicated by preeclampsia (15%–20%) and eclampsia (40%–50%). Fetal and maternal mortality are increased. Treatment is primarily supportive, although platelet transfusion may be required prior to cesarean section. DIC may develop.

24. How do heparin and low-molecular-weight heparin (LMWH) work?

Heparin catalyzes the inactivation of thrombin and factor X by antithrombin. It also has some effect on factors II, IX, and XI. Factor VII is not affected. At usual doses, it will prolong the PTT (and thrombin time [TT]) but not the PT. Occult GI bleeding is a relative contraindication to its use, and clearance is prolonged in hepatic and renal dysfunction.

LMWH is derived from smaller pieces of the heparin molecule. Weight-based subcutaneous dosing of LMWH without anticoagulation monitoring has proven safe and effective in clinical trials. (This is fortunate, because LMWH inactivates factor X more than it does thrombin, so PTT is not significantly affected and cannot/need not be used to monitor clinical effect and therapeutic plasma concentrations.) Weight-based pharmacokinetic predictions for LMWH are not reliable in patients weighing more than 100 kg, pregnant patients, and those with decreased creatinine clearance. If LMWH is used in these patients, anti-X activity must be monitored. Unfractionated heparin often becomes the drug of choice in these patients.

25. How do I treat hemorrhage secondary to heparin therapy?

With major bleeding episodes, heparin can be 100% reversed with protamine sulfate at a dose of 1 mg/100 U of circulating heparin to a maximum dose of 250 mg. It is given slowly, intravenously, over 10 minutes. Rapid infusion increases the risk of anaphylaxis. Protamine is only 60% effective in reversing LMWH, so unfractionated heparin is usually preferred in cases when surgery or invasive procedures are likely.

KEY POINTS: DIAGNOSIS AND TREATMENT OF COAGULOPATHIES

1. Thrombocytopenia: Increased bleeding time, epistaxis, purpura, petechia, mucosal bleeding (6 bags random donor platelets yields 30,000/mL increase)
2. PT and INR: Extrinsic and common paths—warfarin (i.e., II, VII, IX, X)
3. PTT: Intrinsic and common paths (all factors except VII and XIII)—heparin
4. For severe bleeding with hemophilia A, 50 U/kg factor VIII
5. FFP, 15 mL/kg (will support hemostasis until definitive diagnosis)

26. How does warfarin work? How do I deal with elevated INR?

Warfarin (an oral anticoagulant therapy [OAT]) inhibits the reduction of vitamin K to its active form causing depletion of factors II, VII, IX, and X. Starting dose is 5 mg/day, with 4 to 5 days required for full anticoagulant effect. Heparin or LMWH is continued in the interim due to the early inactivation of protein C and S, which causes a temporary procoagulant effect. Target is usually an INR of 2 to 3. Significant bleeding occurs in 3% of patients on chronic OAT. Drug interactions are common, and INR must be monitored. Head computed tomography (CT) evaluation should be performed even in minor head trauma with therapeutic dosing. Minor bleeding with elevated INR less than 5 can be treated by withholding doses until INR returns to the desired range. The underlying need for anticoagulation should be considered. Asymptomatic patients with an elevated INR may receive oral vitamin K without significantly altering the ability to control anticoagulation. Serious bleeding is treated with FFP (10–15 mL/kg) and 10 mg IV vitamin K, given slowly (FFP for immediate effect; vitamin K effect takes several hours).

27. What's new in antithrombotics?

Activated protein C. Sepsis causes a cascade of inflammation and coagulation with impaired fibrinolysis. Microvascular hypoperfusion and organ dysfunction contribute to mortality. Among its effects, activated protein C inactivates factors V and VIII inhibiting thrombosis and promoting fibrinolysis. Early studies of activated drotrecogin alfa (Xigris), a recombinant human activated protein C, showed a reduction in 30-day mortality in adult sepsis patients with acute organ dysfunction who were at a high risk of death. However, its use remains highly controversial within the critical care community.

28. What's new in prothrombotics?

Recombinant activated factor VII (NovoSeven) is currently FDA approved for promoting hemostasis in hemophiliacs with antibody inhibitors to coagulation factors VIII or IX. Although it is also being used for a number of off-label uses in nonhemophilic patients, to date there are not enough consistent high-level data to support its formal recommendation in these situations.

29. Is it true they are close to developing safe and effective fake blood?

Development of a blood substitute has been a major goal of transfusion research groups for many years. Advantages could include improved storage requirements and shelf-life, and decreased antigen reactions and viral and bacterial contamination. Hemoglobin-based oxygen carriers (HBOCs) had been developed in the 1980s and seem to have taken the lead over perfluorocarbon blood substitutes. A number of agents have proceeded to clinical trials, many of which have hit roadblocks due to adverse cardiovascular effects. Newer generations of these substitutes are being developed to try to combat these issues. Despite more than a decade of significant advances, much remains to be done.

30. Is there anything we can do to help control massive hemorrhage from trauma?

Holding pressure is the best way to start; but this is temporizing at best and not always practical in situations of massive trauma and bleeding from noncompressible sites. The military has been using new clotting agents to stabilize patients with traumatic bleeding on the combat field. Quick Clot is a granulated mineral compound that can be poured directly into a wound. It works by removing liquid substances and concentrating the clotting factors, thus allowing a clot to form quickly. The HemCon bandage is made of chitosan, a substance with mucoadhesive properties. It becomes extremely sticky when in contact with blood, and seals the wound to control the bleeding. Although these two agents are already FDA approved for civilian use, additional human studies are ongoing with a very promising dry fibrin-sealant dressing. This dressing contains human fibrinogen, human thrombin, and calcium chloride, but does not transmit human viruses as did older fibrin sealants. We should be seeing these hemostatic agents more frequently in ambulances and hospitals in the near future.

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CHAPTER 41

SICKLE CELL DISEASE

Brad Talley, MD

1. What is sickle cell disease (SCD)?

SCD is a hereditary disorder that affects the structure and function of hemoglobin, the protein in red blood cells that is responsible for oxygen delivery to tissues in the body. The sickle hemoglobin (Hemoglobin S [HbS]) differs from the normal hemoglobin (Hemoglobin A [HbA]) by a single amino acid substitution—valine for glutamine—at the sixth position on the β-globulin chain of the hemoglobin molecule. This substitution causes abnormal polymerization of HbS when it is deoxygenated, resulting in sickle-shaped, nondeformable red blood cells that cannot traverse small capillaries. These sickle cells are responsible for the vaso-occlusive phenomena and hemolysis that are the hallmarks of the disease.

2. What is sickle cell trait?

SCD is an autosomal recessive disorder. Therefore, patients who are heterozygous for the sickle cell gene (HbSA) are said to have sickle cell trait, whereas patients who are homozygous for the sickle cell gene (HbSS) are said to have SCD. Unlike homozygous individuals, patients with sickle cell trait have a normal life span and are generally asymptomatic.

3. Are there sickle cell variants?

Hemoglobin C and β-thalassemia are abnormal hemoglobins that create hemolytic anemias of their own. Hemoglobin C (HbC) is an inherited abnormal β-chain, which when combined with the sickle cell gene produces a less severe form of SCD known as HbSC. β-thalassemia is also an inherited abnormal β-chain. When combined with a copy of the sickle cell gene, the clinical presentation of the disease can range from a mild to severe form of SCD depending on the quantity of hemoglobin A. Table 41-1 summarizes the spectrum of SCD.

4. What causes red blood cells to sickle?

The rate and extent of polymer formation in a circulating HbS red cell depend primarily on three independent variables: the cell's degree of deoxygenation, the intracellular hemoglobin concentration, and the presence or absence of hemoglobin F (fetal hemoglobin). Any factors that promote global or local hypoxia, such as circulatory stasis, cardiovascular disease, pulmonary disease, and high altitude, can cause sickling. Low temperature promotes sickling through vasoconstriction. Acidosis also promotes sickling, as does radiographic contrast dye. The presence of hemoglobin F in a red blood cell prevents sickling.

5. How common is SCD?

In the United States, 1 of every 600 people of African descent is born with SCD. Approximately 8% are heterozygous for the sickle cell gene. The prevalence is increasing in the United States, due to both population growth and improved longevity of patients with SCD. Mortality rates for children with SCD have declined with the advent of newborn screening programs, education initiatives for parents, antibiotic prophylaxis, and *Haemophilus influenzae* and *Streptococcus pneumoniae* vaccination. Despite these improvements in the care provided, the median age at death is 42 years for men and 48 years for women.

TABLE 41–1. THE SPECTRUM OF SCD

Disease	Genetics	Clinical Severity
SCD	Two copies of sickle cell gene	Most severe
Sickle cell trait	One copy of sickle cell gene, other Hb gene is normal	Usually asymptomatic with a normal life span
HbSC disease	One copy of sickle cell gene, one of Hb C gene	Less severe symptoms, life span is approximately 20 years longer than in SCD
Sickle cell-β-thalassemia	One copy of sickle cell gene, one of β-thalassemia gene	20% have symptoms as severe as those with SCD; 80% have less severe symptoms

Hb, hemoglobin; HbSC, hemoglobin S and hemoglobin C; SCD, sickle cell disease.

6. What are the typical baseline laboratory findings in patients with SCD?

Because sickle cells have a shorter life span than normal red blood cells (17 days vs. normal 120 days), most patients will have a mild to moderate anemia with a hematocrit of 20% to 30% and a reactive reticulocytosis of 3% to 15%. The white blood cell count, platelet count, and alkaline phosphatase levels are usually elevated. Mean bilirubin levels and lactate dehydrogenase levels are also elevated, whereas serum haptoglobin levels are low, secondary to early red cell death. Creatinine and electrolyte levels are generally normal.

7. What causes vaso-occlusion (pain) crisis?

Vaso-occlusion typically causes recurrent painful episodes (previously called sickle cell crisis) that can result in a variety of serious organ system complications. There is a high degree of clinical heterogeneity among patients who experience vaso-occlusion episodes. The pathophysiology is poorly understood, but it is thought to be a combination of poorly pliable sickled red blood cells, modified vascular endothelium, and an inflammatory response involving leukocytes, platelets, and plasma proteins that result in microvascular occlusion. The vaso-occlusion episode typically manifests as pain that can affect any part of the body and can last from days to weeks at time. Many episodes are accompanied by objective clinical signs such as low-grade fever, swelling, tenderness, tachypnea, hypertension, nausea, and vomiting. Standard laboratory testing is not helpful in identifying vaso-occlusion crisis.

8. How should vaso-occlusive (pain) crisis be managed?

Vaso-occlusive (pain) crises are the most frequent reason patients with SCD seek medical attention. These patients should be managed with oral (in mild cases) or intravenous rehydration and analgesics. Pain is often undertreated because many physicians are not familiar with the pharmacology of analgesia or are overly concerned with the potential for addiction. If pain is refractory to treatment, the patient should be admitted.

9. What causes an acute or worsening anemia in sickle cell patients?

- **Sequestration crisis** occurs when large numbers of red blood cells pool in the spleen due to their abnormal shape and stiffness. Patients present with splenic enlargement, abdominal pain, a falling hematocrit, pallor, tachycardia, and dyspnea. Massive sequestration can lead to hypovolemic shock and death. This usually occurs between the ages of 5 months and 2 years because by 2 years of age the spleen is typically

autoinfarcted and fibrosed due to multiple vaso-occlusive events. It is the most dangerous crisis for young children, with a mortality of 10% to 15%. Splenectomy is usually recommended after the first event.

- **Aplastic crisis** is caused by a transient arrest of erythropoiesis, characterized by a decreased hematocrit and depressed reticulocyte count resulting in fatigue, dyspnea, and pallor. It is frequently the result of an infection, with parvovirus B19 being the most common precipitant. Most patients require acute transfusion therapy and reticulocytes typically reappear within 2 to 14 days.
- **Hemolytic crisis** usually occurs in response to infection or drugs, resulting in a more rapid rate of hemolysis. A rapidly falling hematocrit, an elevated reticulocyte count, pallor, and jaundice are observed. Patients with glucose 6-phosphate dehydrogenase (G6PD) deficiency are even more prone to hemolysis and hypoxia. It can be precipitated by certain foods (e.g., fava beans) and medications (e.g., aspirin and sulfonamides).

10. What treatments are available for SCD?

Because a definitive cure is not currently available for most patients, treatment is aimed largely at symptom control during vaso-occlusive episodes. In the acute care setting, the goal is to stop the cycle of deoxygenated sickling and microvascular sludging. Therapies include rest, hydration, and analgesia. Transfusion and antibiotic therapy for infection are also indicated in some cases. Hydroxyurea is the only FDA-approved therapy and works by increasing fetal hemoglobin levels. Fetal hemoglobin alters the dynamics of HbS polymerization, thereby decreasing the rates of vaso-occlusive crisis, acute chest syndrome (ACS), and transfusion. However, long-term effects (mutagenic and carcinogenic properties) of prolonged hydroxyurea are unknown. Bone marrow transplantation has been successful in a limited number of children and may possibly provide a cure for SCD, but the experience is quite limited and long-term outcomes are unknown. Patient selection, along with the risks of the procedure, are also important factors still being researched. Gene therapy is still experimental but may have the potential to cure SCD. Other experimental medical therapies include nitric oxide, which directly inhibits polymerization of HbS; butyric acid, which modulates gene expression of Hemoglobin F; and clotrimazole, which modifies the cellular dehydration that worsens cell sickling.

11. Do cerebrovascular accidents (CVAs) occur in SCD?

Yes. Children with SCD carry a 300-fold increased risk for stroke, making it the most common cause of childhood stroke. By age 20, 10% to 20% of patients with SCD will have experienced a clinical stroke syndrome, and a further 17% to 22% will have subclinical evidence of cerebral infarction on brain magnetic resonance imaging (MRI). The mean age of onset of CVA is 10 years. Of patients with CVA, 67% will suffer another, usually within 36 months. The incidence of stroke in children has been decreasing by prophylactic transfusions and regular transcranial Doppler studies for children at risk.

KEY POINTS: INDICATIONS FOR ADMISSION OF PATIENTS WITH SCD

1. Pain not controlled with oral medications
2. Unable to hydrate orally
3. Neurologic findings
4. Pulmonary findings (e.g., hypoxia, infiltrate on chest X-ray, new rales)
5. Sequestration crisis
6. Aplastic crisis
7. Fever (If >40°C, toxic appearing, or poor follow-up)

12. Is there a role for blood transfusion in SCD?

Simple transfusion is appropriate for single transfusions to restore oxygen-carrying capacity or blood volume. Partial exchange transfusions are required in emergencies and in the setting of chronic transfusions. Transfusion therapy to prevent or treat pain is controversial because of the risk of iron overload and alloimmunization. Hemoglobin concentration should not be raised above 10g/dL because of increases in blood viscosity and the risk of vaso-occlusive episodes.

KEY POINTS: INDICATIONS FOR CONSIDERATION OF TRANSFUSION IN PATIENTS WITH SCD

1. Aplastic crisis
2. Sequestration crisis
3. ACS
4. CVA
5. Prior to major surgery
6. Acute multiorgan failure
7. Pregnancy
8. Priapism

13. What is ACS?

ACS (acute chest syndrome) is defined as a new pulmonary infiltrate on chest radiograph accompanied by either fever (>38.5°C), chest pain, cough, wheezing, or tachypnea. ACS often develops after a vaso-occlusive crisis and is a leading cause of hospitalization and death in SCD.

14. What causes ACS?

Infection, fat embolism, rib infarction, thromboemboli, reactive airway disease, fluid overload, and atelectasis due to splinting can cause ACS. Regardless of the root cause, the final common pathway in the pathogenesis of ACS is small-vessel vaso-occlusion, infarction, and inflammation with alveolar wall necrosis. In a vicious cycle, these factors lead to regional hypoxemia and acidosis, which in turn causes increased sickling and sludging.

15. How should ACS be managed?

If ACS is suspected after a thorough history and physical, a complete blood count, reticulocyte count, blood cultures, and chest X-ray should be ordered. Sputum cultures may also be of use in this population. If ACS is diagnosed, the patient should be admitted and treated with intravenous hydration, antibiotics, analgesics, and aggressive pulmonary toilet. Early treatment with broad-spectrum antibiotics, which should include a third-generation cephalosporin to cover *S. pneumoniae, H. influenza,* and *Klebsiella pneumonia* and a macrolide to cover *Mycoplasma pneumoniae* and *Chlamydia pneumoniae*, are recommended until culture results are available. In many cases, exchange transfusion is indicated to lower the concentration of HbS. Those who present with severe anemia, thrombocytopenia, or multilobar pneumonia should receive a transfusion before respiratory distress develops. Early aggressive ventilatory support is mandatory.

16. How should a patient with SCD be managed when he or she presents with fever?

Patients with SCD are functionally asplenic, so they are at increased risk for serious bacterial infection, especially with encapsulated organisms (*S. pneumonia* and *H. influenza*). Fever in children should be considered a medical emergency, requiring a thorough search for the source, blood cultures, and empiric antibiotics. The clinician should also have a low threshold for admitting adults and administering empiric antibiotics. One approach is to divide patients into two categories:

- **High risk:** Patients with SCD or sickle cell-β-thalassemia who appear toxic, have temperature >40°C, or are not receiving prophylactic penicillin. Admit for intravenous ceftriaxone.
- **Low risk:** Patients with SCD or sickle cell-β-thalassemia who appear to be well, have temperature <40°C, and are taking prophylactic penicillin; *or* patients with HbSC who have temperatures >38.5°C. Obtain blood cultures, observe for several hours in the ED, administer ceftriaxone, and arrange follow-up within 24 hours.

17. How is bone infarction differentiated from osteomyelitis in SCD?

It is extremely challenging. Both entities cause bone pain and fever. In attempting to make the diagnosis, fever greater than 38.4°C may indicate osteomyelitis, although this cutoff is imperfect. A presentation of multifocal, rather than unifocal, bone pain is more consistent with bone infarction. The absence of leukocytosis or elevated erythrocyte sedimentation rate (ESR) may also suggest infarction. Although positive cultures of bone can diagnose osteomyelitis, a period of at least 48 hours is required to process them, and sensitivity is not 100%. The leading cause of osteomyelitis is *Salmonella*, followed by *Staphylococcus aureus*. Plain radiographs, contrast-enhanced computed tomography (CT) scan, MRI, and radio nucleotide bone scans have all had disappointing results. Therefore, antibiotics are recommended for all patients with fever and bone pain until osteomyelitis can be ruled out.

18. What other important clinical complications can occur with SCD?

Vaso-occlusion in the skin can cause frequent leg ulcers and myofascial syndromes in patients with SCD. Hepatobiliary complications are common with patients often having chronic cholelithiasis secondary to pigmented gallstones. Iron overload can occur in patients requiring frequent transfusions. Males with SCD often present to the ED with priapism. Retinal complications can occur, including proliferative retinopathy, retinal artery occlusion, and retinal detachment and hemorrhage. Patients with SCD can experience renal insufficiency from vaso-occlusion of the vasa recta capillaries in the renal medulla. Patients with SCD are also at increased risk for developing pulmonary hypertension.

19. Are pregnant patients with SCD at an increased risk of complications?

Yes. Pregnancy is associated with both fetal and maternal complications related to compromised placental blood flow. The fetus is at risk for spontaneous abortion, preterm labor, intrauterine growth restriction, and low birth weight, whereas pregnant females are at risk for cerebral vein thrombosis, pneumonia, pyelonephritis, deep venous thrombosis, postpartum infection, and sepsis. They are also more likely to undergo cesarean delivery, to experience pregnancy-related complications (such as hypertension, pre-eclampsia/eclampsia, abruption, and antepartum bleeding) and to have cardiomyopathy or pulmonary hypertension. Close maternal-fetal surveillance is warranted.

KEY POINTS:

1. Patients with SCD have functional asplenia and therefore are at an increased risk for bacterial infections, in particular encapsulated organisms. Initiate broad-spectrum antibiotics for suspected bacterial infections.
2. Patients with vaso-occlusion crisis are often undertreated for pain.
3. Diagnosis of uncomplicated vaso-occlusive pain crisis is one of exclusion and can only be made after all other causes of pain and precipitating events have been ruled out first.

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CHAPTER 42

ONCOLOGIC EMERGENCIES

Nicholas J. Jouriles, MD

1. What is an oncologic emergency?

An oncologic emergency is a life- or limb-threatening problem in a patient with an underlying neoplasm. These problems may be caused by the cancer, its systemic effects, or by therapeutic interventions against the cancer. There may also be psychosocial issues.

2. Is this important in the ED?

Yes. Cancer is the second leading cause of death in the United States. It is also second only to trauma in years of potential life loss. In addition, as cancer treatments improve, we will see an ever-increasing number of ED patients with oncologic emergencies.

3. Name several oncologic emergencies.

See Table 42-1.

4. Which of the entities on this list are life- or limb-threatening?

The life-threatening diseases are those that can lead to shock or death. They can be divided into the standard categories of shock: volume loss (bleeding) or impaired vascular return (superior vena cava syndrome [SVCS]); pump impairment (cardiac tamponade); and derangement of systemic vascular resistance (sepsis). There are serious metabolic derangements (hypercalcemia) and disabling neurologic problems (spinal cord compression [SCC]).

5. Tell me about these.

- **SVCS**: Caused by obstruction of the superior vena cava. Although it may be caused by mediastinitis or aortic aneurysms, most cases are caused by a neoplastic process. Lung cancer is the most common, usually the small cell or squamous types. Adenocarcinoma of the breast and lymphoma are also common. SVCS may also occur secondary to metastatic lesions from distant primary sites. Diagnosis is clinical and is verified by imaging. Treatment usually involves radiation therapy, but chemotherapy and endovascular stenting are also options.
- **Cardiac tamponade**: Usually occurs secondary to metastatic disease of the pericardium. It has been found in 2% to 21% of patients dying of cancer (Fig. 42-1). Patients with cardiac tamponade usually have a large tumor burden and a poor 6-month survival. The diagnosis of a malignant cardiac effusion with tamponade is suspected clinically in the hypotensive patient with muffled heart sounds, elevated neck veins, and an enlarged cardiac silhouette on chest X-ray. It is most commonly seen in lung and breast carcinomas and lymphoma. Treatment involves pericardial drainage. ED bedside ultrasound is the best way to make the diagnosis and to guide drainage.
- **Infections**: Because all patients with tumors are by definition immunocompromised, the variety of potential infections is unlimited. Immune status may be further compromised by chemotherapeutic agents. When patients become neutropenic—total neutrophil count less than 500—any type of infection may occur. This includes bacterial, viral, or fungal

TABLE 42–1. EMERGENCIES IN PATIENTS WITH UNDERLYING NEOPLASTIC DISEASES (PARTIAL LIST)

Airway compromise	**Intestinal obstruction**
Head and neck mass	**Intestinal perforation**
Tracheal compression	**Malignant pericardial effusion with tamponade**
Adrenal crisis	**Metabolic abnormalities**
Primary tumor	Hypercalcemia
Metastatic lesion	Acute tumor lysis syndrome
Anemia	Hyponatremia/SIADH
Bone marrow replacement with tumor	Hyperuricemia
Chemotherapy effects	Hypoglycemia
Bleeding	**Obstructive jaundice**
Primary mass	**Obstructive uropathy**
Low platelet count	**Pain**
Abnormal clotting factors secondary to liver metastases	**Complications of radiotherapy**
Carcinoid syndrome	Dermatitis
Complications of chemotherapy	GI toxicity
Bone marrow suppression	**Emotional stress**
Cardiac toxicity	Death and dying
GI toxicity	DNR orders
Pulmonary toxicity	Family issues
Renal toxicity	**Seizures**
Graft vs. host disease	**Spinal cord compression**
Hemorrhagic cystitis	Motor/sensory loss
Chemotherapy induced	Incontinence
Radiotherapy induced	Back pain
Hyperviscosity syndrome	**Superior vena cava syndrome**
Infection	**Tinnitus**
With neutropenia	
Postobstructive pneumonia	

DNR, do not resuscitate; GI, gastrointestinal; SIADH, syndrome of inappropriate secretion of antidiuretic hormone.

infections that can lead to septic shock, adult respiratory distress syndrome, and death. The neutropenic febrile patient should be placed in isolation and treated with broad-spectrum antibiotics.

- **Hypercalcemia**: Occurs in up to 30% of patients with cancer. Neoplasms that lead to metastatic involvement of the skeletal system are commonly associated with hypercalcemia. Common presenting signs are lethargy, constipation, and altered mental status. Treatment involves hydration with normal saline and bisphosphates, such as pamidronate.

- **SCC**: Occurs in up to 5% of all patients with metastatic disease. The spinal cord or nerve root is directly compressed by an extradural mass, causing secondary neurologic dysfunction. The most common causes of SCC are lung, breast, and prostate cancers and multiple myeloma. The most common presenting symptom is back pain. Any patient with an underlying malignancy who presents with back pain, motor loss, paresthesias, or incontinence should be considered to have SCC. Prompt diagnosis with emergent magnetic resonance can save neurologic function. Up to 40% of patients with SCC may have normal plain radiographs. Steroids should be given in the ED. Treatment is emergent radiation therapy or surgical decompression individualized to the patient.

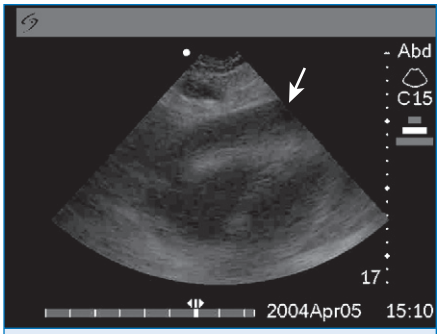

Figure 42-1. Ultrasound of pericardial effusion leading to cardiac tamponade.

KEY POINTS: SCC

1. Negative plain films do not rule out SCC.
2. Suspicion of SCC is an indication for emergent magnetic resonance image.
3. Steroids and analgesics are the initial ED management while arranging for appropriate treatment.

6. Are these common problems?

Of the life-threatening problems, SCC, infection, and hypercalcemia are relatively common.

7. What other problems are common in patients with an underlying malignancy?

The most common problems are complications of cancer treatment. Each chemotherapeutic agent has side effects. Nausea, vomiting, and diarrhea are common, whereas renal involvement, pulmonary toxicity, and cardiac toxicity occur often enough to be seen in the ED. Pain and death are universal concerns.

8. How is an oncologic emergency diagnosed?

The most important element is clinical suspicion. In any patient with a neoplasm, a complication should be suspected. This includes patients who have been "cured" of cancer, as well as those with risk factors but no diagnosis.

After concentrating on the airway, breathing, circulation, and vital signs, an extensive history should be taken, followed by a complete physical examination. A presumptive diagnosis should be made and appropriate data obtained.

9. What symptoms can be related to an underlying oncologic emergency?

Common ED complaints such as abdominal pain (colon cancer) or back pain (SCC) can be the initial presentation of an oncologic process. Unfortunately, any ED presenting symptom can be caused by a neoplasm. A neoplastic process should be considered in any patient who presents with pain, unexplained weight loss, weakness, dizziness, altered mental status, headache, and new-onset seizures, especially in an elder.

KEY POINTS: FEBRILE NEUTROPENIC PATIENT

1. Early antibiotics improve outcome.
2. ED antibiotics should be broad spectrum and reflect local infection and resistance patterns.
3. Protective isolation should be used.

10. When should the patient be admitted?

All patients with life- or limb-threatening disease should be admitted. Patients in whom the diagnosis of an oncologic process is first made in the ED are usually admitted. A special group of patients who need to be admitted are those who lack the resources at home to care for themselves. It is not uncommon for families to give so much of themselves that they need a break, and an admission for respite care is indicated.

For all other patients, it is probably best to discuss the matter with the patient, family, and primary physician. Most patients with cancer have a primary oncologist who knows the patient and his or her situation in detail. The emergency physician should balance the current medical problem with all the patient's needs. Many patients have already spent much time at the hospital and would like to be with their loved ones as much as possible at home.

11. Can cancer be cured?

Modern therapies offer excellent success with medical (e.g., testicular cancer, lymphoma, leukemia), surgical (e.g., lung, colon, and breast cancer), and combination (e.g., radiotherapy and chemotherapy for head and neck, anal cancers) treatments. Many patients today survive for long periods of time, giving them ample opportunity to access emergency care.

12. How is a patient with a terminal neoplastic disease treated?

Often the best treatment for a patient with a terminal malignancy is adequate analgesia, comfort measures, and supportive care. The emergency physician can also be challenged by issues related to "do not resuscitate" orders in the ED and the out-of-hospital arena. It is vital to communicate well with the patient to arrive at the very best individualized treatment plan.

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FLUIDS AND ELECTROLYTES

Corey M. Slovis, MD

1. Why is the study of fluid and electrolytes so difficult?

Most people who teach fluid and electrolytes are well educated and talk about things like "the negative log of the hydrogen ion concentration," "idiogenic osmols," and "pseudo-pseudo triple acid-base disturbances." Luckily, this chapter is not written by a person who believes in, or understands, logarithms.

2. What is the anion gap (AG)?

The AG measures the amount of negatively charged ions in the serum (unmeasured anions) that are not bicarbonate (HCO_3^-) or chloride (Cl^-). The AG is calculated by subtracting the sum of HCO_3^- and Cl^- values from the sodium (Na^+) value, the major positive charge in the serum. Potassium (K^+) values are not generally used in the calculation because of the huge amount of intracellular potassium (155 mEq) and the relatively low amount of potassium in the serum (only about 4 mEq). The formula for determining AG is as follows:

$$AG = Na^+ - (Cl^+ + HCO_3^-)$$

The normal AG is generally accepted as $8 - 12 \pm 2$.

3. Why must AG be calculated each time an electrolyte panel is evaluated?

An elevated AG means there is some unmeasured anion, toxin, or organic acid in the blood. If you do not calculate the gap, you could miss one of the only clues to a potentially life-ending disease or overdose. The AG also allows acidosis to be divided into two types: wide gap (AG $>$ 12–14) and normal gap (AG $<$ 12–14).

4. There are two types of acidosis: wide gap and normal gap. What is a hyperchloremic metabolic acidosis?

A hyperchloremic acidosis is just another name for a normal gap acidosis. Just think: If the AG is going to be normal, and the formula for $AG = Na^+ - (Cl^- + HCO_3^-)$, if HCO_3^- goes down Cl^- has to rise, or, more simply, you become hyperchloremic, hence the name *hyperchloremic metabolic acidosis*.

5. Is there an easy way to remember the differential diagnosis for wide gap metabolic acidosis?

My favorite is taken from Goldfrank and is called **MUDPILES**.

M = **M**ethanol
U = **U**remia
D = **D**iabetic ketoacidosis (DKA) and alcoholic ketoacidosis (AKA)
P = **P**henformin and Metformin (the original P, Paraldehyde, is no longer available)
I = **I**NH (Isoniazid) and **I**ron
L = **L**actic acidosis
E = **E**thylene glycol
S = **S**alicylates and **S**olvents

6. What are the clues to each of the entities in MUDPILES?

See Table 43-1.

TABLE 43–1. CLUES TO THE DIFFERENTIAL DIAGNOSIS OF WIDE GAP METABOLIC ACIDOSIS

Disease or Toxin	Clues
Methanol	Alcoholism, blindness or papilledema, profound acidosis
Uremia	Chronically ill-appearing, history of chronic renal failure, BUN > 100 mg/dL, and creatinine > 5 mg/dL
DKA	History of diabetes mellitus, polyuria, and polydipsia, glucose > 500 mg/dL
AKA	Ethyl alcohol, glucose < 250 mg/dL, nausea and vomiting
Phenformin/ Metformin	Diabetes, medication history, recent contrast study
INH	Tuberculosis, suicide risk, refractory status seizures
Iron	Pregnant or postpartum, hematemesis, radiopaque tablets on abdominal film (unreliable finding)
Lactic acidosis	Hypoxia, hypotension, sepsis
Ethylene glycol	Alcoholism, oxalate crystals in urine with or without renal failure, fluorescent mouth or urine (from drinking antifreeze—unreliable finding)
Salicylates	History of chronic disease requiring aspirin use (i.e., rheumatoid arthritis); mixed acid-base disturbance (primary metabolic acidosis plus primary respiratory alkalosis); aspirin level > 20–40 mg/dL
Solvents	History of exposure or huffing; spray paint on face

AKA, alcoholic ketoacidosis; BUN, blood urea nitrogen; DKA, diabetic ketoacidosis; INH, isoniazid.

7. What are the causes of narrow gap acidosis?

Memorize the mnemonic **HARDUPS.**

H = **H**yperventilation (chronic)
A = **A**cetazolamide, **A**cids (e.g., hydrochloric), **A**ddison's disease
R = **R**enal tubular acidosis
D = **D**iarrhea
U = **U**reterosigmoidostomy
P = **P**ancreatic fistulas and drainage
S = **S**aline (in large amounts)

If you do not want to memorize anything, it is important to know that diarrhea, especially in children, and renal tubular acidosis, especially in adults, are the two most common causes of a narrow gap acidosis.

8. Why should normal saline (NS) or lactated Ringer's (LR) solution rather than 0.5 NS or dextrose in 5% water (D_5W) be given to someone who needs volume replacement?

Fluid goes into three different body compartments: (a) inside blood vessels (intravascular), (b) into cells (intracellular), and (c) in between the two (interstitial). NS and LR solutions go into all three compartments, and only 25% to 33% stays in the intravascular compartment. A person who lost 2 U of blood (1000 mL) would need 3 to 4 L of crystalloid for volume

resuscitation. One-half NS (0.45 NS) provides only half of what NS or LR provide; of each liter of 0.45 NS provided, only 125 to 175 mL stays in blood vessels (vs. 250–333 mL for NS and LR). D_5W is the worst for trying to give intravascular volume; it puts only about 80 mL per 1000 mL of D_5W into the vasculature. The rest goes into cells and the interstitium.

9. Which solution is better, NS or LR?

Both fluids are excellent for early volume replacement.

- **NS** has a pH of 4.5 to 5.5 and has a sodium *and* chloride content of 155 mEq/L each. It is acidotic, has an osmolarity of 310, and has a little more sodium than serum and a lot more chloride than serum (155 mEq/L of Cl^- in NS vs. about 100 mEq/L of Cl^- in serum). Too much NS too quickly may cause a hyperchloremic metabolic acidosis.
- **LR** is considered more physiologic in that it is much closer to serum in its content. Its sodium content is lower than NS at 130 mEq/L, and its chloride is only 109 mEq/L (vs. 155 mEq/L of NS). The solution is called lactated because it has 28 mEq/L of bicarbonate in the form of lactate, which becomes bicarbonate when it is in the body. LR has 4 mEq of potassium (none in NS) and has 3 mEq/L of calcium. Critics of LR do not like all the bicarbonate in it and believe that potassium therapy should be individualized. The bottom line is that neither NS nor LR is better; both are equal in quantities of 2 to 3 L over 24 hours. Patients with protracted vomiting should be given NS, which is higher in chloride. Patients with severe diarrhea and the resultant hyperchloremic metabolic acidosis should be given LR, which has the equivalent of a half an ampule of bicarbonate per liter.

10. What is the most dangerous electrolyte abnormality? What are its five most common causes?

Hyperkalemia is the most dangerous electrolyte abnormality. It may result in sudden dysrhythmogenic death because of its effect on the cells' resting membrane potential. The most common cause of hyperkalemia is often referred to as "laboratory error." Actually, the laboratory does a perfect analysis, but the serum sample has hemolyzed after, or while, it is being drawn.

The number one cause of hyperkalemia is **spurious** elevation. The other common causes are as follows:

- **Chronic renal failure** (the true number one cause of hyperkalemia)
- **Acidosis** (potassium moves out of the cell as the pH falls)
- **Drug induced** (including nonsteroidal anti-inflammatory drugs, potassium-sparing diuretics, digoxin, angiotensin-converting enzyme inhibitors, and administration of intravenous potassium chloride)
- **Cell death** (when potassium comes out of injured muscle or red cells); including burns, crush injuries, rhabdomyolysis, tumor lysis syndrome, and intravascular hemolysis.

Much less common causes of hyperkalemia include adrenal insufficiency, hyperkalemic periodic paralysis, and hematologic malignancies.

11. What electrocardiogram (ECG) changes are associated with hyperkalemia?

The first ECG change seen in hyperkalemia is usually a tall, peaked T wave that may occur as potassium values rise to between 5.5 and 6.5 mEq/dL. Loss of the P wave may follow as potassium levels rise to between 6.5 and 7.5 mEq/dL. The most dangerous ECG finding (generally associated with levels of 8.0 mEq/dL) is widening of the QRS, which may merge with the abnormal T wave and create a sine-wave-appearing ventricular tachycardia.

12. Summarize the best treatment for hyperkalemia.

Treatment is based on (a) serum levels, (b) the presence or absence of ECG changes, and (c) underlying renal function. If the patient has life-threatening ECG changes of hyperkalemia (widening QRS or a sine-wave-like rhythm), 10% calcium chloride should be given in an initial dose of 5 to 10 mL to temporarily reverse potassium's deleterious electrical effects. Most patients, however, with hyperkalemia usually just require moving potassium intracellularly,

then removing potassium from the body, rather than receiving a potentially dangerous calcium infusion.

13. How can potassium be moved intracellularly?

The most effective way is by giving glucose and insulin. Glucose and insulin work by activating the glucose transport system to move glucose into the cell. As glucose is carried intracellularly, potassium is carried along. The usual dose of glucose is 2 ampules of $D_{50\%}$ (100 mL) and 10 U of insulin. Bicarbonate may be used to drive potassium into the cell, but it is effective *only* in acidotic patients. Usually 1 to 2 ampules of bicarbonate (44.6–50 mEq of bicarbonate per ampule) are given over 1 to 20 minutes, depending on how sick or acidotic the patient is. Another method of driving potassium into the cell is use of inhaled β-agonist bronchodilators. β-agonists may be helpful in a renal failure patient with fluid overload because they additionally treat the bronchospasm of pulmonary edema. Intravenous magnesium, which also drives potassium into the cell, is not used because most hyperkalemic patients are hypermagnesemic also.

KEY POINTS: HYPERKALEMIA

1. Hyperkalemia is asymptomatic; you must check the ECG.
2. The ECG changes seen as potassium rises are: (a) a tall peaked T wave; (b) loss of the P wave; and (c) widening of the QRS complex.
3. Administering glucose and insulin, supplemented by an inhaled beta agonist, is the most effective method to drive potassium in to the cell and acutely lower serum potassium.
4. Bicarbonate only works to lower serum potassium in acidotic patients.
5. Only give calcium in hyperkalemia for a wide QRS.

14. After potassium's electrical effects have been counteracted (if indicated) and potassium has been driven intracellularly, how do you remove it from the body?

Potassium can be removed from the body by diuresis, potassium-binding resins, and hemodialysis. Diuresis with saline, supplemented by furosemide, is an excellent way to lower total body potassium. Most hyperkalemic patients, however, have renal failure and cannot make much or any urine, which is how they became hyperkalemic in the first place. Sodium polystyrene sulfonate (Kayexalate) is a sodium-containing resin that exchanges its sodium content for the patient's potassium. Each 1 g of Kayexalate, which must be mixed with sorbitol, can remove about 1 mEq of potassium from the patient's body. The best method of lowering potassium is by hemodialysis, and it is the method of choice for any severely ill, acidotic, or profoundly hyperkalemic patient.

15. Discuss the most common causes of hyponatremia.

Hyponatremia is a serum sodium of less than 135 mEq/dL. Most patients with mild hyponatremia (levels $>$ 125–130 mEq/dL) are on diuretics or have some degree of fluid overload as a result of heart failure, renal failure, or liver disease. Diuretic-induced hyponatremia is the most common cause in the elderly. Patients with heart failure, liver failure, or renal failure develop hyponatremia as a result of secondary hyperaldosteronism. Aldosterone is released because of renal hypoperfusion, resulting in fluid retention, volume overload, and a dilutional hyponatremia (even in the face of total body sodium excess). Moderate-to-severe hyponatremia (levels $<$ 125 mEq/dL) are most commonly due to the syndrome of inappropriate secretion of antidiuretic hormone (SIADH), psychogenic polydipsia (compulsive water drinking), or intentional water ingestion (marathon runners and ecstasy users).

16. What is SIADH?

Abnormally high levels of hormone from the posterior pituitary gland, which blocks free water excretion. Normally, when sodium levels fall, levels of antidiuretic hormone (ADH) also decrease, resulting in urinary losses of water (diuresis). In this syndrome, ADH is released inappropriately, and serum sodium levels fall as more excess free water is retained (antidiuresis). The hallmark of this syndrome is relatively concentrated urine, rather than the maximally diluted urine one sees in a water-overloaded patient. Patients cannot be given this diagnosis if they are taking diuretics or have a reason to be water overloaded (i.e., congestive heart failure, chronic renal failure, or liver failure).

17. What are the classic neurologic signs of hyperkalemia? What are the classic ECG signs of hyponatremia?

No, not a misprint, just a trick to wake you up after antidiuresing. Potassium causes cardiovascular, not neurologic, symptoms via its effects on the ECG (see Question 11). Sodium causes no ECG changes but does affect the brain because of its effects on osmolality; symptoms include dizziness, confusion, coma, and seizures.

18. How fast should hyponatremia be corrected?

There has been much debate over how rapidly (about 2 mEq/h) or how slowly (about 0.5 mEq/h) sodium should be corrected. Patients should be corrected slowly and serum sodium should be allowed to rise by no more than 0.5 mEq/h. This approach avoids the possible development of central pontine myelinolysis (which is also called the *osmotic demyelinating syndrome* by some purists), a catastrophic neurologic illness of coma, flaccid paralysis, and usually death seen with too-rapid correction.

19. Should sodium levels *ever* be treated quickly?

There are some specific indications for raising a patient's sodium rapidly by infusing 3% saline. Patients who have serum sodium levels of significantly less than 120 mEq/L *and* who have acute alterations in mental status, seizures, or new focal findings should have their levels raised about 4 to 6 mEq/dL over a few hours. Hypertonic saline should be given very carefully in these acutely ill patients: 100 mL over 10 to 60 minutes. Other than these rare patients with severe, symptomatic hyponatremia, slow correction by water restriction, often with a slow infusion of saline, is all that is required.

20. What is osmolality? What is the osmolal gap?

Osmolality is calculated by multiplying the serum sodium by 2 and adding the glucose (GLU) divided by 18, plus the blood urea nitrogen (BUN) divided by 2.8. Normal is approximately 280 to 290 mOsm.

$$\text{Osmolarity} = 2 \infty \text{Na} + \text{GLU}/18 + \text{BUN}/2.8$$

The osmolal gap is determined by using this formula, then asking the laboratory to measure the osmolality. The difference in the lab's measured osmolarity and your calculated osmolarity should be only about 10; if it is more, something else is in the serum (e.g., an alcohol, intravenous contrast media, or mannitol).

$$\text{Osmolal gap} = \text{laboratory-determined osmolarity} - \text{calculated osmolarity}$$

21. How do you use the osmolal gap in figuring out if someone has ingested methanol or ethylene glycol?

If the osmolal gap is elevated, you should measure the patient's serum ethanol level immediately. Because of ethanol's molecular weight, every 4.2 mg/dL of alcohol *weighs* 1 mOsm. If the alcohol level is 100 mg/dL, the patient's osmolal gap should be about 30 to 35 (about 25 from alcohol, added to the normal osmolal gap, which is about 5–10).
If there is a higher gap, these unaccounted osmols may represent methanol, ethylene glycol, or isopropyl alcohol. Because isopropyl alcohol causes ketosis without acidosis, a wide gap

acidosis plus an unexplained osmolal gap often means a life-threatening overdose. Hints to methanol and ethylene glycol overdose appear in answer 6.

22. What are the most common causes of hypercalcemia? How do they present?

Mild hypercalcemia is usually due to dehydration, thiazide diuretics, or hyperparathyroidism. It is often asymptomatic, but mild fatigue, renal stones, or nonspecific gastrointestinal symptoms may be present. Severe hypercalcemia, with levels greater than 2 to 3 mg/dL above normal, usually presents as depressed mental status along with the signs and symptoms of profound dehydration.

23. Describe the emergency treatment of hypercalcemia.

Symptomatic hypercalcemia is treated by aggressive volume resuscitation with saline supplemented by furosemide after intravascular volume has been normalized. Once their volume status is normalized, patients should receive approximately 150 to 200 ml of NS per hour, plus enough furosemide to keep urine output at 1 mL/kg or higher. Saline blocks the proximal tubules from absorbing calcium, and furosemide, once thought to block distal tubular absorption, assists in maintaining a diuresis. Older patients and patients with impaired cardiac function must be closely followed as they are volume resuscitated and placed on the saline infusion; otherwise, turn to the chapter on congestive heart failure.

KEY POINTS: FLUIDS AND ELECTROLYTES

1. An elevated AG should alert you to a potentially serious disease or overdose.
2. Large quantities of normal saline may cause a hyperchloremic metabolic acidosis.
3. Don't raise serum sodium by more than 0.5 meq/L each hour or by more than 10 to 12 meq/L day.
4. Seizures, coma, and acute neurological findings in a previously normal patient are the only indications to give hypertonic saline in patients with profound hyponatremia.
5. The therapy of hypercalcemia centers on a saline-induced diuresis carefully supplemented by furosemide.

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ACID–BASE DISORDERS

Stephen L. Adams, MD, FACP, FACEP, and Morris S. Kharasch, MD, FACEP

CHAPTER 44

1. Name the four types of acid-base disorders seen in the ED, and give a common example of each.

Actually, there are five:

- Metabolic acidosis (e.g., cardiac arrest)
- Respiratory acidosis (e.g., chronic obstructive pulmonary disease with carbon dioxide [CO_2] retention)
- Metabolic alkalosis (e.g., protracted vomiting)
- Respiratory alkalosis (e.g., hyperventilation syndrome)
- Mixed acid-base disorder (e.g., respiratory alkalosis and metabolic acidosis, as seen in an adult with salicylate intoxication; metabolic acidosis with respiratory compensation)

2. What does pulse oximetry contribute to the understanding of the patient's acid-base status?

Nothing. Pulse oximetry measures oxygen saturation and does not provide a measurement of acid-base or ventilatory status. Arterial blood gas analysis is necessary to determine acid-base status.

3. What are the most commonly cited causes of an elevated anion gap?

An elevated anion gap, usually indicating a low bicarbonate level, should give the clinician cause to consider the presence of a metabolic acidosis. The differential diagnoses may be remembered by the mnemonic **DR. MAPLES**:

D = **D**iabetic ketoacidosis (DKA)
R = **R**enal failure
M = **M**ethanol
A = **A**lcoholic ketoacidosis
P = **P**araldehyde
L = **L**actic acidosis
E = **E**thylene glycol
S = **S**alicylate intoxication

These are only some of the causes of a metabolic acidosis.

4. Name some obscure causes of an elevated anion gap metabolic acidosis.

Sulfuric acidosis, short bowel syndrome (D-lactic acidosis), nalidixic acid, methenamine, mandelate, hippuric acid salt, rhubarb (oxalic acid) ingestion, and inborn errors of metabolism, such as the methylmalonic acidemias and isovaleric acidemia. Toluene intoxication (glue sniffing) can cause either an elevated anion gap metabolic acidosis or a hyperchloremic metabolic acidosis (no anion gap).

5. Is the size of the anion gap clinically useful?

In one study, an anion gap of greater than 30 mEq/L was usually the result of an identifiable organic acidosis (i.e., lactic acidosis or ketoacidosis). Almost 30% of patients with an anion gap of 20 to 29 mEq/L had neither a lactic acidosis nor a ketoacidosis.

6. What are some causes of lactic acidosis?

Shock, seizure, hypoxemia, isoniazid (INH) toxicity, metformin, cyanide poisoning, ritodrine, inhaled industrial acetylene, phenformin ingestion, iron intoxication, ethanol abuse, and carbon monoxide poisoning. Sodium nitroprusside, povidone-iodine ointment, sorbitol, xylitol, and streptozocin are other drugs that have been listed as causing increased lactic acid formation.

7. Name a vitamin deficiency associated with a fatal metabolic acidosis.

Thiamine deficiency (vitamin B_1 deficiency), which is associated with neurological deficits (e.g. Wernicke's encephalopathy, Korsakoff syndrome), high output cardiac failure (Beriberi), and lactic acidosis, has been cited as a cause of fatal metabolic acidosis. Thiamine deficiency should be considered in such high-risk populations as those who have a history of alcohol abuse or nutritionally deficient states. Wernicke's encephalopathy has also been associated with an anion-gap primary metabolic acidosis in conjunction with a primary respiratory alkalosis.

8. How severe is the acid-base disturbance that results from a grand mal seizure? How long does it take to resolve the acidosis?

A grand mal seizure can result in a profound lactic acidosis. The pH levels may plummet to 6.9 or lower. The acidosis in an uncomplicated seizure usually resolves spontaneously within 1 hour.

9. Can a patient have a metabolic acidosis without evidence of an elevated anion gap?

Yes. A patient with a hyperchloremic metabolic acidosis may have no evidence of an elevated anion gap. This condition is caused, in effect, by adding hydrogen chloride to the serum. The fall in serum bicarbonate is offset by the addition of Cl^-; consequently, there is no increased anion gap.

10. How can I remember some of the causes of a normal anion gap metabolic acidosis?

Use the mnemonic **USED CARP**:

U = **U**reteroenterostomy
S = **S**mall bowel fistula
E = **E**xtra chloride
D = **D**iarrhea
C = **C**arbonic anhydrase inhibitors
A = **A**drenal insufficiency
R = **R**enal tubular acidosis
P = **P**ancreatic fistula

11. In a patient with DKA who is improving with appropriate therapy, why might the measurement of serum ketones show an increase?

There are three ketone bodies: β-hydroxybutyrate (BHB), acetoacetate (AcAc), and acetone. BHB and AcAc are acids; acetone is not. The proportion of BHB to AcAc depends on the oxidation-reduction status of the patient. A patient who is in DKA on presentation often is severely dehydrated, and the preponderance of ketone bodies may be in the form of BHB. The test by which ketones are noted is the nitroprusside reaction test (Acetest, Ketostix), which measures AcAc and acetone but is not sensitive to BHB. In the patient with DKA, as fluids and insulin therapy are instituted, the amount of BHB converted to AcAc increases, and the nitroprusside reaction, which initially may have been weakly positive or even negative, becomes increasingly positive.

12. List nine disorders that can cause a hyperketonemic state.

- Isopropyl alcohol intoxication
- DKA
- Alcoholic ketoacidosis

- Starvation
- Paraldehyde intoxication (pseudoketosis)
- Cyanide intoxication
- Industrial acetylene inhalation
- Hyperemesis gravidarum
- Bovine ketosis
- Stress hormone excess

13. What may contribute to metabolic acidosis in an abuser of alcohol?

Ketoacidosis has been well documented in the chronic alcoholic who binges, then presents with nausea, vomiting, abdominal pain, and poor caloric intake. Lactic acid, acetic acid, and indirect loss of bicarbonate in the urine (nonanion gap metabolic acidosis) also may contribute to an alcoholic acidosis.

14. Which electrolyte is affected most commonly by a change in acid-base status?

Serum potassium. Patients with severe acidosis tend to have elevated serum K^+ levels, whereas patients with severe alkalosis tend to have low serum K^+ levels. A change of pH of 0.10 is consistent with a corresponding change in serum K^+ of about 0.5 mEq/L (range, 0.3–0.8 mEq/L). If the pH is elevated by 0.10, the serum K^+ falls by about 0.5 mEq/L. If the pH is diminished by 0.10, the serum K^+ rises by about 0.5 mEq/L. This concept is well known to clinicians who treat patients who present in DKA. Although the patient's total body K^+ may be severely depleted, initial serum K^+ levels may be elevated in the severely acidotic patient. As the patient is treated appropriately and acidosis resolves, K^+ supplementation is indicated because serum levels may fall precipitously.

15. What is a pseudometabolic acidosis?

Underfilling of Vacutainer tubes can cause a significant decline in bicarbonate and an increase in anion gap that may be mistaken for metabolic acidosis. It is theorized that because atmospheric pressure contains less than 5% CO_2, the lower partial pressure of CO_2 over the blood in an underfilled tube causes CO_2 to diffuse out of the venous solution, decreasing the bicarbonate with which it is in equilibrium. Tubes should be filled completely to prevent creating a pseudometabolic acidosis.

16. Are there any potential ill effects of using paper bag rebreathing in the treatment of hyperventilation syndrome?

Yes. When normal volunteers hyperventilated into a brown paper bag, inspired oxygen was decreased sufficiently so as to endanger hypoxic patients. Paper bag rebreathing therapy probably should not be used unless myocardial ischemia can be ruled out and arterial blood gas analysis or pulse oximetry excludes hypoxia.

17. How does core temperature affect arterial blood gases?

Uncorrected arterial blood gases yield a falsely elevated pH and a falsely decreased PO_2 and PCO_2 in hypothermia. For every 1°C decrease in body temperature, the pH is elevated 0.015, PCO_2 (mm Hg) decreases 4.4%, and PO_2 decreases 7.2% (37°C reference). Hyperthermia decreases the pH and increases the PCO_2 and PO_2 by an equivalent amount. The clinical use of corrected versus uncorrected pH determinations in hypothermia is controversial.

18. What acid-base alterations are seen commonly in heatstroke?

Metabolic acidosis (81% of patients in one study) and respiratory alkalosis (55% of patients). The prevalence of metabolic acidosis was associated significantly with the degree of hyperthermia. Of patients, 63% with a rectal temperature of 41°C, 95% with a temperature of 42°C, and 100% with a temperature of 43°C had a metabolic acidosis. This association was not true for respiratory alkalosis. Patients who had a metabolic acidosis had a large anion gap (24 ± 5).

19. What disease process can present with an anion gap *higher* than the serum glucose?

Alcoholic ketoacidosis, a well-known cause of an elevated anion gap metabolic acidosis, may present with hypoglycemia. One case report presented a patient with alcoholic ketoacidosis and a concomitant illness, pneumonia, with an anion gap of 36 and a serum glucose of less than 20 mg/dL. Severe hypoglycemia may cause a lactic acidosis and usually occurs in the setting of a defect in gluconeogenesis, which may be seen in a patient with chronic alcohol ingestion. A concomitant illness commonly is seen in the patient with alcoholic ketoacidosis.

KEY POINTS: ACID-BASE DISORDERS

1. Patients with a metabolic acidosis may have an elevated serum K+ even though they may have a low total body K+.
2. Patients with alcoholic ketoacidosis should appropriately be treated with crystalloids containing glucose.
3. Thiamine deficiency should be considered in the appropriate clinical setting and treated in those at risk for the consequences of same.

20. How can patients with HIV have an abnormality in the anion gap?

A patient with HIV may have a low anion gap. Hypergammaglobulinemia, resulting from an increased number of immunoglobulin-secreting β-cells because of failure in immunoregulation, has been reported in patients with HIV. Consequently, an elevation of immunoglobulin G (IgG) and immunglobulin A (IgA) may occur. The anion gap may be low because of the cationic charge of IgG. One case report described a patient with HIV with lactic acidosis, which should elevate the anion gap, who had a "deceptively" normal anion gap. A patient with hyperlactacidemia and a normal anion gap acidosis should prompt an evaluation of coexisting illnesses that may be responsible for the low anion gap.

21. What is the most common cause of metabolic acidosis in the pediatric population?

Significant diarrheal illnesses in this population may produce a starvation ketosis.

22. In addition to the toxic alcohols, name two entities causing a metabolic acidosis with an elevated anion gap that have been associated with an elevated osmolal gap.

Alcoholic ketoacidosis and lactic acidosis. It has been speculated that, in patients with lactic acidosis, organic substances of low molecular weight are released from ischemic tissues, accounting for unmeasured osmols. In alcoholic ketoacidosis, it has been speculated that an increased osmolal gap could be attributed to acetone, an uncharged ketone of low molecular weight that may be elevated if the ketoacidosis is severe and prolonged. The exact pathogenesis of the gap in these two entities is not certain, however. As can be seen, the elevated osmolal gap is not specific for a toxic alcohol ingestion.

23. Name a base and an outfielder.

Al Kaline (Detroit Tigers 1953–1974, Hall of Fame 1980).

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CHAPTER 45

DIABETES MELLITUS

C. Ryan Keay, MD, FACEP

1. Describe the physiologic and clinical differences between type I and type II diabetes mellitus.

- **Type I disease** (formerly known as insulin dependent) is characterized by pancreatic β-cell destruction, which causes an absolute insulin deficiency. Patients with type I disease have little or no endogenous production of insulin and can therefore develop diabetic ketoacidosis (DKA). Insulin repletion is necessary; dietary modifications and oral hypoglycemic agents are inadequate therapy.
- **Type II disease** (formerly known as adult-onset diabetes) is characterized by peripheral insulin resistance. There is also a spectrum of defective insulin production by pancreatic β-cells. Glucose levels often respond to oral dietary modification and oral hypoglycemic agents; however, insulin is sometimes necessary to control glucose levels.

2. What are the diagnostic criteria for diabetes mellitus?

Normal fasting plasma glucose (FPG) is <100 mg/dL. A random serum glucose >140 mg/dL is suggestive of diabetes and should prompt a fasting plasma glucose (FPG) measurement. Any FPG level of 100 to 125 mg/dL is considered impaired glucose tolerance, often referred to in lay terms as *pre-diabetes*. Two separate measurements of an FPG level >126 mg/dL is diagnostic for diabetes.

3. List the physiologic complications of hyperglycemia.

- Osmotic diuresis (polyuria)
- Dehydration
- Electrolyte abnormalities
- Coronary artery disease
- Cerebral vascular disease
- Peripheral vascular disease
- Nephropathy
- Retinopathy
- Neuropathy
- Infection secondary to impaired leukocyte function
- Cutaneous manifestations
- Ketoacidosis (in type I patients)

4. Describe the pertinent clinical and laboratory findings of DKA.

A patient with DKA presents with nausea, vomiting, or abdominal pain secondary to gastric distention or stretching of the liver capsule. Others may have some degree of altered mentation. Further clinical indicators include osmotic diuresis with resultant dehydration; polyuria, polydipsia, and polyphagia; weight loss; tachypnea; Kussmaul breathing; and fruity breath odor.

Laboratory findings include hyperglycemia; metabolic acidosis; and potassium, sodium, chloride, calcium, magnesium, and phosphorus depletion.

5. What causes DKA?

DKA is a state of insulin deficiency most commonly triggered by infection (30%–50% of cases), noncompliance with medications, new-onset diabetes, and other physiologic stressors. Insulin is

the primary anabolic hormone produced by the pancreas. Without insulin, cells cannot take up glucose, resulting in an increase in the body's catabolic hormones: glucagon, catecholamines, cortisol, and growth hormone. Catabolism stimulates lipolysis, breaking down fatty acids, which are then oxidized to acetoacetate and β-hydroxybutyrate, resulting in a metabolic acidosis. These breakdown products are the ketones measured in DKA. The overall shift in metabolism during DKA is from a state of carbohydrate metabolism to fat metabolism.

6. How does one make the diagnosis of DKA?

- Blood glucose >300 mg/dL
- Low bicarbonate (<15 mEq/L)
- Low pH (<7.3) with ketonemia and ketonuria

7. How should DKA be treated in the ED?

- **Fluid resuscitation.** Patients often have a fluid deficit of 5 to 10 L. Normal saline (NS) should be administered by giving 1 to 3 L in the first hour, the next 2 L over hours 2 to 6, and finally 2 more L over hours 6 to 12. Titrate fluid resuscitation to urine output, blood pressure, heart rate, and mental status. In children, give 10 to 20 mg/kg/h of fluid for the first hour, repeated once. Do not give more than 50 mL/kg of intravenous (IV) fluid in the first 4 hours. Fluids should be administered at approximately 1.5 times maintenance for 24 hours.
- **Insulin.** Initial dosage is 0.1 to 0.4 U/kg IV bolus followed by 0.1 U/kg/h via IV infusion. Frequent blood sugars should be checked with a goal of dropping the glucose level by 50 to 75 mg/dL/h. Do not start insulin before checking a potassium level.
- **Potassium replacement.** Serum potassium should be replaced because it will drop with insulin and fluids administration. Adding 20 to 40 mEq in each 1 L bag, after potassium is under 5.5 mEq/L, will help correct the deficit slowly. Goal levels are between 4 and 5 mEq/L.
- **Phosphorus and bicarbonate.** Therapy is controversial and should be reserved for profound disturbances (phosphorus <1 and pH <6.9).
- **Glucose.** When serum levels drop below 250 mg/dL, IV fluids should be switched to half normal saline with the addition of 5% dextrose. Insulin infusion is still required to treat persistent serum ketones.
- **Magnesium and calcium.** Levels should be followed and replaced accordingly.

8. Do all patients with DKA need to be admitted?

No. Patients with mild DKA who meet the following criteria may be discharged from the ED: good follow-up, normal vitals, stable lab values, normal mentation, no other underlying chronic or infectious sources, and ability to tolerate oral intake.

9. List the potential complications of therapy for DKA in the ED.

- Hypoglycemia
- Hypokalemia
- Hypophosphatemia
- Adult respiratory distress syndrome
- Cerebral edema

10. What is the hyperosmolar hyperglycemic state (HHS)?

HHS (formerly termed hyperosmolar hyperglycemic nonketotic coma) is a life-threatening emergency, defined as severe hyperglycemia (usually >600 mg/dL), elevated plasma osmolality (>320 mOsm/kg), serum bicarbonate >15 mEq/L, arterial pH >7.3, negative serum ketones (can be mildly positive), and altered mental status.

11. How is plasma osmolarity determined?

$$\text{Osmolarity (mOsm/kg water)} = 2(\text{serum sodium}) + (\text{serum glucose}/18 + \text{BUN}/2.8)$$

where BUN = blood urea nitrogen

12. What occurs pathophysiologically to cause HHS?

HHS usually occurs in older, Type II diabetics. The pathophysiology is the same as DKA, without the generation of ketones. In the absence of insulin, cellular receptors are unable to transport glucose intracellularly, creating an osmotic gradient. Extracellular volume expands at the expense of intracellular dehydration. Elevated glucose levels overcome renal filtration, and glucosuria results, causing osmotic diuresis and profound dehydration. Why these patients are not ketotic remains controversial. One likely factor is slightly more available levels of insulin in HHS than DKA, inhibiting lipolysis. A poorly understood aspect of HHS pathogenesis is the lower levels of catabolic hormones found in HHS patients compared to their DKA counterparts.

13. What are the precipitants of HHS?

Any illness leading to dehydration is a risk factor for HHS in the Type II diabetic. Comorbid conditions, such as renal disease and heart failure, complicate HHS. Causes include infections, primarily pneumonia and urinary tract infections (UTIs), stroke, intracranial hemorrhage, myocardial infarction, and pulmonary embolism. Drugs are frequently implicated including: diuretics, β-blockers, histamine-2 blockers, antipsychotics, alcohol, cocaine, and total parenteral nutrition (TPN).

14. What are the four key points in ED management of patients with HHS?

- **Fluid administration:** 1 to 2 L of normal saline should be administered initially. Fluid deficits may be as high as 10 L, however, judicious rehydration should be observed in cardiac and renal patients. Be aware of correcting hypernatremia too quickly.
- **Potassium:** Potassium should be repleted at 10 to 20 mEq/h in patients with normal renal function.
- **Insulin:** Although most patients with HHS do not receive insulin therapy, patients with acidosis, hyperkalemia, or renal failure need insulin to lower glucose levels and resolve metabolic derangements. A starting dose of 0.15 U/kg of insulin given intravenously, with an infusion rate of 0.1 U/kg/h, is reasonable in these patients.
- **Glucose:** Add to IV fluids when levels are less than 250 mg/dL.

15. Which patients with HHS should be admitted to the hospital?

All patients with HHS should be admitted. Most require at least 24 hours of monitoring for treatment of electrolyte abnormalities, fluid administration, and evaluation of precipitating causes.

16. Define hypoglycemia.

Serum glucose $<$50 mg/dL.

17. Who develops hypoglycemia?

Patients who are taking hypoglycemic medications are at greatest risk for hypoglycemia. Other causes include accidental or intentional overdose of insulin, pentamidine, aspirin, haloperidol, insulinomas, renal failure, sepsis, adrenal insufficiency, sepsis, alcoholism, or heart failure.

18. Which overdoses of oral hypoglycemic agents do not cause hypoglycemia?

- Metformin overdose does not cause hypoglycemia because it decreases hepatic production of glucose and increases insulin sensitivity. Instead, symptoms of overdose include nausea, vomiting, and abdominal pain. Lactic acidosis is a known complication of therapeutic and supratherapeutic doses of metformin. Lactic acidosis may be treated with sodium bicarbonate or hemodialysis.
- Thiazolidinediones increase peripheral tissue glucose use and do not cause hypoglycemia. Hepatoxicity has been reported with these drugs.
- α-glucosidase inhibitors decrease gastrointestinal glucose absorption and do not cause hypoglycemia. Symptoms of overdose include bloating, abdominal pain, and diarrhea.

19. What are the presenting signs of a patient with hypoglycemia?

Patients can present with agitation, diaphoresis, tachycardia, decreased level of consciousness, coma, seizures, bizarre and sometimes violent behavior, and even focal neurologic deficits. Symptoms should reverse with administration of glucose. If symptoms do not resolve, seek an alternate diagnosis.

20. Which patients with hypoglycemia require admission to the hospital?

Patients who:

- Have persistent altered mental status or hypoglycemia after glucose administration.
- Have taken excessive amounts of oral hypoglycemic agents or long-acting insulin.
- Are unable to tolerate oral intake.
- Are suicidal.

21. Can patients who have been treated for hypoglycemia in the field by paramedics refuse transport?

Yes. This is a common scenario. Patients most commonly have taken their normal or recently adjusted dose of insulin and have skipped a meal. If these patients can eat, they may refuse transport. Patients who may have taken an intentional overdose of insulin or oral hypoglycemic agents must be transported.

22. What is metabolic syndrome (syndrome X or dysmetabolic syndrome)?

Insulin resistance (hyperglycemia), obesity (particularly abdominal obesity), hypertension, and dyslipidemia.

23. Describe gestational diabetes mellitus (GDM).

GDM is any degree of glucose intolerance that develops or is diagnosed during pregnancy. It usually develops in the second or third trimester and occurs when a woman's pancreatic function cannot overcome the insulin resistance created by placental anti-insulin hormones. It affects approximately 4% of women in the United States but varies according to ethnicity. These women are at increased risk of developing type II diabetes later in life. Untreated GDM can have serious health effects for the fetus, including fetal macrosomia, hypoglycemia, hypocalcemia, and hyperbilirubinemia.

24. What types of infections are seen more commonly in diabetics than in other patients?

Diabetic patients are more susceptible to UTIs, candidal vaginitis, cystitis, balanitis, pneumonia, influenza, tuberculosis, lower-extremity skin and soft-tissue infections, and bacteremia.

- **Rhinocerebral mucormycosis** is a rare, rapidly progressive invasive fungal infection of the nasal and paranasal sinuses. Computed tomography (CT) scan should be obtained to define extent of disease. Early surgical debridement is essential for good outcomes, with a mortality rate as high as 50% despite optimal management. The IV antifungal of choice is amphotericin B.
- **Malignant otitis externa** is usually caused by *Pseudomonas aeruginosa*. Patients present with unilateral otalgia, swelling, and discharge. The external auditory canal is initially affected; it can then cause adjacent cellulitis, osteomyelitis, and temporoparietal abscess. CT scan should be used to image affected regions. IV antipseudomonal antibiotics, debridement, and hyperbaric oxygen are required for extensive disease.
- **Emphysematous pyelonephritis** and **cholecystitis** are more common in diabetics. Findings include gas on plain film, although CT may be required for diagnosis. IV antibiotics and surgical treatment are indicated. The mortality rates even with prompt treatment are 40% and 15%, respectively.

25. What are the common manifestations of diabetic neuropathy?

Patients typically present with a peripheral symmetric neuropathy, which often follows a stocking-glove pattern. Symptoms include bilateral pain, hyperesthesia, and anesthesia.

Neuropathic pain is opioid resistant and is best treated with gabapentin, amitriptyline, and μ-opioid agonists, such as oxycodone. Mononeuropathy multiplex affects motor and sensory nerves, often resulting in wrist or footdrop and affecting cranial nerves III, IV, and VI.

KEY POINTS: DIABETES MELLITUS

1. Infections in diabetics must be aggressively treated because they may spread rapidly.
2. Always measure the serum glucose in patients who are agitated, violent, diaphoretic, or comatose to rule out hypoglycemia as an easily treatable cause of these findings.

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THYROID AND ADRENAL DISORDERS

Daniel H. Bessesen, MD

CHAPTER 46

1. What thyroid-related conditions are considered true emergencies?

Thyroid diseases including hyperthyroidism, hypothyroidism, and nodular thyroid disease are quite common. The two true emergencies are severe hyperthyroidism: thyroid storm and severe hypothyroidism: myxedema coma. The mortality of thyroid storm and myxedema coma without treatment is 80% to 100%. Rarely, eye complications of Graves disease may also require emergent treatment.

2. What are the common clinical signs and symptoms of thyrotoxicosis?

- **Constitutional**: Fatigue, heat intolerance, diaphoresis, weight loss, and uncommonly fever.
- **Neuropsychiatric**: Tremor, hyperreflexia, apathy, anxiety, irritability, emotional lability, and uncommonly psychosis.
- **Ophthalmologic**: Exophthalmos, lid lag, injection, and uncommonly diplopia and reduced visual acuity.
- **Cardiovascular**: Tachycardia, palpitations, and uncommonly atrial fibrillation, chest pain, and congestive heart failure.
- **Gastrointestinal**: Increased frequency of bowel movements or frank diarrhea, nausea, and uncommonly vomiting.
- **Reproductive**: Amenorrhea, infertility in women, and uncommonly gynecomastia in males.
- **Dermatologic**: Hair loss, onycholysis

3. What are the most common causes of hyperthyroidism? How do they present?

Excessive thyroid hormone production:

- Graves disease (85% of all cases): diffuse homogenous enlargement of the thyroid gland, often with proptosis.
- Toxic multinodular goiter: you can typically feel multiple thyroid nodules.
- Hyperfunctioning nodule: you can typically feel a large thyroid nodule, but the rest of the gland is reduced in size or suppressed.

Leakage of thyroid hormone: Thyroiditis typically develops acutely, has a hyperthyroid phase that lasts 1 to 2 months, and is followed by hypothyroidism.

- Subacute thyroiditis: usually presents with pain and tenderness over the thyroid gland with signs and symptoms of hyperthyroidism following a viral infection.
- Painless thyroiditis: Same as subacute thyroiditis but without the pain and tenderness of the thyroid gland.
- Postpartum thyroiditis: Painless thyroiditis that occurs 2 to 6 months following the delivery of a child.
- Radiation-induced inflammation: Exacerbation of Graves disease that occurs 7 to 10 days following the administration of radioactive iodine therapy.

Exogenous thyroid hormone administration:

- Thyrotoxicosis factitia: Munchausen-like; thyroid hormone is taken to cause illness or is taken with the goal of losing weight.
- Thyroid hormone overdose.

4. What lab tests should be ordered in a patient with suspected hyperthyroidism?

When hyperthyroidism is suspected, the best tests to order are the thyroid-stimulating hormone (TSH) level and either a free thyroxine (T_4) level or total T_4 with a triiodothyronine (T_3) resin uptake. When hyperthyroidism is caused by overproduction of thyroid hormone, TSH should be completely suppressed ($<$0.03 mIU/L). A patient with a suppressed TSH and a normal T_4 level has *subclinical hyperthyroidism*.

5. What is apathetic thyrotoxicosis?

A frequently missed presentation of hyperthyroidism seen most commonly in the elderly but may present at any age, even in children. The typical patient is 70 to 80 years old *without* goiter or ophthalmologic findings. The diagnosis should be considered in elderly patients with chronic weight loss, proximal muscle weakness, depressed affect, new-onset atrial fibrillation, or congestive heart failure.

6. What is thyroid storm?

Thyroid storm is simply an exaggerated form of hyperthyroidism that carries with it a risk of serious morbidity or even mortality. The clinical features that are characteristic of thyroid storm include fever (temperature $>$100°F [37.7°C]), altered mental status, and cardiovascular decompensation. A common clinical challenge is to determine if one of these features is due to the thyroid hyperfunction or some other underlying disease such as drug or alcohol intoxication, an infectious process, or underlying cardiac disease. A clinical determination of thyroid storm should be made without necessarily waiting for confirmatory laboratory tests.

7. Which patients with hyperthyroidism should be admitted to the hospital?

While clinical judgment is needed, patients who are suspected of thyroid storm should be admitted. Those with atrial fibrillation should be admitted and anticoagulated because there is an increased risk of atrial thrombus in this condition. Patients who are unable to maintain adequate oral intake due to nausea need to be admitted. Patients who have serious hyperthyroidism and signs and symptoms of heart failure should be admitted because it is often difficult to determine the appropriate dose of β-blockers for these individuals in the outpatient setting. Tachycardia alone, even if marked, is not an indication for admission in an individual who is otherwise young and healthy because β-blockade can safely be instituted as an outpatient.

8. What conditions are included in the differential diagnosis of thyroid storm?

Toxicity caused by cocaine, amphetamines, other sympathomimetics, and anticholinergics; alcohol withdrawal syndromes; or infections such as encephalitis, meningitis, and sepsis. A history of goiter, thyroid disease or previous treatment with an antithyroid medication is helpful in distinguishing thyroid storm from these other conditions.

9. What conditions precipitate thyroid storm?

Thyroid storm typically is precipitated by another problem. Although it is important to treat the thyroid storm directly, it is also important to identify and treat the underlying precipitant. Some of the more common precipitating events include:

- Infection or serious illness
- Surgery
- Trauma
- Childbirth
- Myocardial infarction
- Withdrawal of antithyroid therapy
- Recent ^{131}I thyroid ablation therapy

10. How is hyperthyroidism treated in the ED?

For most patients seen in the ED with hyperthyroidism, treatment with a β-blocker can be initiated. Although propranolol blocks the conversion of T_4 to T_3 and is preferred by some, it needs to be taken at least three times a day in the hyperthyroid patient because of more rapid metabolism. Metoprolol or atenolol are reasonable options and can be given twice a day resulting in better compliance as compared with propranolol. Although Tapazole or propylthiouracil can be initiated, a thyroid scan cannot be done while the patient is taking these medications. For this reason, it is typically best to start a β-blocker and refer the patient for follow-up. ED management of thyroid storm is outlined on Table 46-1.

TABLE 46–1. STEP THERAPY OF DECOMPENSATED THYROTOXICOSIS

1. **Supportive care**
   - General: Oxygen, cardiac monitor
   - Fever: External cooling, acetaminophen (aspirin is contraindicated because it may increase free T_4)
   - Dehydration: IV fluids
   - Nutrition: Glucose, multivitamins, including folate (deficient secondary to hypermetabolism)
   - Cardiac decompensation (atrial fibrillation, congestive heart failure): β-blockers. Atenolol or metoprolol 25 to 100 mg two times per day. Effective dose may be higher, typically used as the metabolism is increased with hyperthyroidism. Esmolol administered intravenously is preferred if there is evidence of congestive heart failure. Begin with 500 mg/kg load over 1 minute, followed by 50 mg/kg/min intravenously. Repeat load and double infusion as necessary
   - Treat precipitating event: Therapy as indicated
2. **Inhibition of hormone biosynthesis—thionamides**
   - PTU,* 1200–1500 mg/day, given as a loading dose of 600–800 mg followed by 200–300 mg every 6 hours PO, by nasogastric tube, or rectally (also blocks peripheral conversion of T_4 to T_3)

   or
   - Methimazole, up to 120 mg/day, given as 20 mg PO every 4 hours (or 40 mg crushed in an aqueous solution rectally).
3. **Blockade of hormone release—iodides* (at least 1 hour after step 2)**
   - Lugol's solution or supersaturated potassium iodide (SSKI), 30–60 drops/day orally divided three or four times per day

   or
   - Ipodate (Oragrafin), 0.5–3 g/day (especially useful with thyroiditis or thyroid hormone overdose)
4. **Blocking the peripheral conversion of T_4 to T_3:**
   - High-dose steroids: Hydrocortisone 100 mg intravenously every 8 hours or prednisone 60 mg PO daily.

IV, intravenous; PO, per os; PTU, propylthiouracil; T_3, triiodothyronine; T_4, thyroxine.
*Preferred medication.
†Consider.
American Thyroid Association: www.thyroid.org

KEY POINTS: THYROID STORM

1. Thyroid disease is extremely common in the ED population.
2. Thyroid storm and myxedema coma are true medical emergencies.
3. Include thyroid storm in your differential for toxic ingestions.
4. Treatment for thyroid storm is initiated without waiting for laboratory work-up.

11. How is acute thyroid hormone overdose treated?

Fatalities are rare with acute ingestion. Toxicity after massive acute overdose usually occurs within 4 to 12 hours but may be delayed for days, particularly with T_4 ingestion. Acute overdose management is as usual, including charcoal and work-up for coingestants.

12. What is Graves ophthalmopathy?

Clinical features include proptosis, injection, chemosis (edema of the conjunctiva), and rarely diplopia with poor eye movement, especially on upward gaze. A loss in visual acuity is a particularly concerning finding. Some eye findings are seen in about half of patients with Graves disease.

13. When is treatment of Graves ophthalmopathy an emergent condition?

Patients with compression of the optic nerve or corneal ulceration require immediate ophthalmologic consultation. Visual blurring that persists with eye closure and diminished color brightness suggests compression of the optic nerve. Severe proptosis can cause keratitis or corneal ulceration presenting as eye pain, photophobia, conjunctival infection, visual loss, and a flare of cells in the anterior chamber. Optic neuropathy is initially treated with high-dose steroids (e.g., prednisone, 1–2 mg/kg per os [PO]). Corneal ulcers, with or without keratitis, require culture and topical antibiotics.

14. What are the common clinical manifestations of hypothyroidism?

- **Constitutional**: Fatigue, cold intolerance, weight gain, lethargy, hoarse or deep voice, slow speech, and drowsiness
- **Neuropsychiatric**: Delayed relaxation phase of deep tendon reflexes (hung up reflex), depression, moodiness, and rarely dementia or psychosis
- **Cardiovascular**: Bradycardia and less commonly congestive heart failure, and rarely pericardial effusion
- **Respiratory**: Occasionally dyspnea, hypoventilation, and rarely pleural effusions
- **Musculoskeletal**: Joint swelling and muscle cramps
- **Dermatologic**: Cool, dry skin and hair loss
- **Gynecologic**: Metromenorrhagia

15. What are the most common causes of hypothyroidism?

Primary hypothyroidism due to thyroid gland dysfunction: In these conditions TSH is increased and T_4 is decreased. A patient with an increased TSH but a normal T_4 has *subclinical hypothyroidism*.

- Autoimmune thyroid destruction: Hashimoto's thyroiditis (90% of all cases). Thyroid gland may be firm or small.
- Thyroiditis: Following a period of hyperthyroidism, the gland may be hypofunctioning permanently or transiently (1–2 years).
- Hypothyroidism following thyroidectomy or radioactive iodine treatment.

Pituitary or hypothalamic insufficiency resulting in inadequate TSH secretion: In these conditions, TSH is typically normal (or low), and T_4 is also low. These patients typically show

signs and symptoms of follicle-stimulating hormone/luteinizing hormone (FSH/LH) deficiency (amenorrhea in women, hypogonadism in men).
- Pituitary tumor
- Pituitary infarction, Sheehan's syndrome, or pituitary apoplexy
- Meningioma or craniopharyngioma near hypothalamus

16. What additional features are present in myxedema coma?

The hallmark clinical features are hypothermia (75%), bradycardia, hypoventilation, and coma in a patient with a history of thyroid disease. Laboratory evaluation may reveal anemia, hyponatremia, hypercarbia and a respiratory acidosis, or respiratory failure. Electrocardiogram (ECG) may show bradycardia with low voltages that may be due to a pericardial effusion. The chest radiograph may show pleural effusions, or frank congestive heart failure.

17. What precipitates myxedema coma in the hypothyroid patient?

As is true with thyroid storm, myxedema coma is typically precipitated by an intercurrent illness such as a pulmonary or renal infection, sedatives and anesthetic agents (including etomidate), trauma, myocardial infarction, cerebrovascular accident, or gastrointestinal hemorrhage. Even moderate hypothyroidism may be life-threatening in patients with underlying hypoxia, hypercapnia or congestive heart failure.

18. What is the treatment for myxedema coma?

See Table 46-2.

TABLE 46–2. TREATMENT FOR MYXEDEMA COMA

1. Supportive care
- Airway control, oxygen, IV access, and cardiac monitor (ABCs).
- Hypotension is treated with crystalloids.
- Vasopressors as indicated (ineffective without thyroid hormone replacement).
- Baseline thyroid function studies should be sent.
- Hypothermia is treated with passive rewarming.
- Perform a Cortrosyn stimulation test and then empirically treat with hydrocortisone (100 mg intravenously every 8 hours) until results are available. This is because of increased metabolism of cortisol that will occur when thyroid hormone is replaced, which may precipitate adrenal insufficiency if there is underlying adrenal insufficiency.

2. Thyroid replacement therapy
- IV T_4 (4 μg/kg; followed in 24 hours by 100 μg intravenously, then 50 μg intravenously until oral medication is tolerated)
- T_3 (liothyronine), at 20 μg intravenously followed by 10 μg intravenously every 8 hours until the patient is conscious (given because of the risk of decreased T_3 generation from T_4 in severely hypothyroid patients). This is not widely available and typically IV T_4 is sufficient.

3. Identify and treat precipitating factors

4. Treat concomitant metabolic abnormalities, including hyponatremia, hypoglycemia, and hypercalcemia

ABCs, airway, breathing, circulation; IV, intravenous; T_3, triiodothyronine; T_4, thyroxine.
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19. What is the significance of a palpable thyroid nodule in an asymptomatic patient?

Palpable thyroid nodules are a common physical finding in the general population occurring in 5% to 8% of all adults. Most are benign adenomas that are not a threat to health. Because a small percentage of solitary nodules are thyroid carcinomas, referral for fine-needle aspiration biopsy is indicated for all patients with palpable nodules who have normal thyroid function tests (TSH, free T_4). Biopsy results identify 70% of nodules to be benign, 5% to be malignant, and the remainder to be cytologically indeterminate.

20. What advice should be given to the patient when a nonpalpable thyroid nodule is incidentally found on a radiologic study?

Thyroid nodules smaller than 1 cm are usually not detected on physical examination but may be identified incidentally on magnetic resonance imaging, computed tomography, or ultrasound done for another reason. These types of nodules are quite common and may occur in 30% to 50% of the general population. Serum levels of TSH and T_4 should be measured and patients should be told of the finding. They should be reassured that the finding is common and does not definitely indicate the presence of cancer. However, the risk of cancer is the same in small nodules as it is in large nodules. These patients should ideally have a formal thyroid ultrasound to look for features such as microcalcification that raise the concern for cancer. If thyroid function tests are normal and the ultrasound is not concerning, the neck ultrasound should be repeated in 6 to 12 months to monitor for growth of the nodule.

21. What are the adrenal emergencies that I need to worry about?

There are two serious adrenal emergencies: acute adrenal insufficiency and pheochromocytoma. Hypercortisolism due to a tumor secreting pituitary adrenocorticotropic hormone (ACTH), ectopic ACTH secretion, or an adrenal tumor that may present with weight gain, hypertension, amenorrhea in women, insulin resistance, or frank diabetes. The specific physical findings in this condition include wide (>1 cm) purple striae, easy bruising, and proximal muscle weakness. Hyperaldosteronism is an unusual cause of hypertension that may present with spontaneous hypokalemia.

22. List the signs and symptoms of primary adrenal insufficiency.

- Fatigue
- Weakness
- Weight loss
- Anorexia
- Hyperpigmentation: This sign is due to increased melanocyte stimulating hormone (MSH), which is oversecreted with ACTH when adrenal insufficiency is due to adrenal gland failure.
- Gastrointestinal symptoms: Nausea, vomiting, abdominal pain, and diarrhea. Abdominal pain may be severe and mimic an acute abdomen.
- Hypotension: This typically presents with orthostatic changes. You should think of adrenal insufficiency when hypotension does not respond to vasopressors.
- Fever: Temperatures as high as 40° C may be seen in acute adrenal insufficiency.

23. What is the difference between primary and secondary adrenal insufficiency?

Primary adrenal insufficiency is due to destruction of the adrenal gland. Secondary adrenal insufficiency is due to inadequate production of ACTH.

24. List the causes of adrenal insufficiency.

See Table 46-3.

25. What are the most common causes of primary adrenal insufficiency?

Tuberculosis and autoimmune destruction account for 90% of the cases of primary adrenal insufficiency.

TABLE 46–3. COMMON CAUSES OF ADRENAL INSUFFICIENCY

Primary adrenal insufficiency	Secondary adrenal insufficiency
Idiopathic (autoimmune)	**Exogenous glucocorticoid administration**
Tuberculosis	**Pituitary or suprasellar tumor**
Bilateral adrenal hemorrhage or infarction	**Pituitary irradiation or surgery**
AIDS	**Head trauma**
Drugs: Adrenolytic agents (metyrapone, aminoglutethimide, or mitotane) or ketoconazole	**Infiltrative disorders of the pituitary or hypothalamus:** sarcoidosis, hemochromatosis, histiocytosis X, metastatic cancer, or lymphoma
Infections: Fungal or bacterial sepsis	**Infectious diseases:** Tuberculosis, meningitis, or fungus
Infiltrative disorders: Sarcoidosis, hemochromatosis, amyloidosis, lymphoma, or metastatic cancer	**Isolated ACTH deficiency**
Bilateral surgical adrenalectomy	
Hereditary: Adrenal hypoplasia, congenital adrenal hyperplasia, adrenoleukodystrophy, or familial glucocorticoid deficiency	

ACTH, adrenocorticotropic hormone.

26. What is the most common cause of secondary adrenal insufficiency?

Long-term therapy with pharmacologic doses of glucocorticoids (e.g., prednisone, methylprednisolone, and dexamethasone) is the most common cause of secondary adrenal insufficiency. These drugs are used to treat a wide variety of medical problems, and if they are used for any significant time, some degree of suppression of the hypothalamic-pituitary-adrenal (HPA) axis occurs.

27. How long must a patient be treated with steroids to cause suppression of the HPA axis, and how long does it take them to recover normal function?

The body needs higher levels of glucocorticoids when stressed. For this reason, signs and symptoms of adrenal insufficiency are most pronounced when patients are sick with an intercurrent illness and are not able to respond with an adequate level of cortisol. Some patients who are on maximal stress doses of steroids (e.g., >60 mg/day of prednisone) for longer than 1 week may have a blunted response to ACTH. This will typically resolve over a few weeks or months. If a person has been on maximal stress doses of steroids for many months or years and then is tapered gradually, they may be able to make enough cortisol for normal daily functioning, but if they are septic, have a myocardial infarction, or sustain severe trauma, they may exhibit signs and symptoms of adrenal insufficiency even 1 to 2 years later.

28. What are the characteristic laboratory findings of primary adrenal insufficiency?

Hyperkalemia may be present because of a lack of aldosterone in addition to cortisol deficiency. Hyponatremia may be present and is due to the SIADH. Cortisol is one of the counter-regulatory hormones that increase liver glucose production with fasting. In the setting of adrenal insufficiency hypoglycemia may develop if the patient has not eaten. Anemia and an increase in eosinophils may be seen. Rarely adrenal insufficiency causes hypercalcemia.

29. How is the presentation of secondary adrenal insufficiency different from that of primary adrenal insufficiency?

In secondary adrenal insufficiency, there is no deficiency of aldosterone secretion. As a result these patients do not have hyperkalemia. Hypotension and hyponatremia can be seen and do not help distinguish primary from secondary adrenal insufficiency. Patients who have adrenal insufficiency from a suppressed HPA axis due to chronic steroid use may have a Cushingoid appearance. If the patient has a pituitary or hypothalamic cause for the adrenal insufficiency, findings may include symptoms of other pituitary hormone deficiencies, such as hypothyroidism, amenorrhea in women, or hypogonadism in men.

30. What is adrenal crisis?

Adrenal crisis is an acute and exaggerated form of adrenal insufficiency. It typically presents in a patient with chronic adrenal insufficiency who undergoes some form of stress, such as an acute myocardial infarction, a systemic infection, surgery, or trauma, and is unable to mount a stress response by increasing circulating cortisol levels.

31. What is the most frequent iatrogenic cause of acute adrenal crisis?

Rapid withdrawal of steroids in patients with adrenal atrophy secondary to long-term steroid administration.

32. Describe the common clinical features of acute adrenal insufficiency.

Patients appear to be profoundly ill. The lack of cortisol makes them vasodilated and makes them appear significantly volume depleted with hypotension and shock. The presence of aldosterone deficiency, anorexia, and nausea with vomiting potentiates this picture. Severe abdominal pain that may mimic an acute abdomen can be present. Fever may occur as a result of infection or the adrenal insufficiency itself. Central nervous system symptoms of confusion, disorientation, and lethargy may also be present.

33. How is adrenal crisis diagnosed?

You should suspect adrenal crisis when some of the typical signs and symptoms are present in a patient who has a reason to have adrenal insufficiency. Although many of the signs and symptoms are non-specific (e.g., fever, abdominal pain, hypotension, fatigue, anorexia) they should raise your suspicion if the patient has a history of being treated with steroids, has a history of a pituitary tumor, has AIDS, or has known metastatic cancer or other predisposing conditions. When faced with this situation the proper test is the **rapid ACTH stimulation test**. Sometimes clinicians simply treat and do not get a test. The problem with this approach is that a day or two later the patient may be better, but you do not know if they indeed had adrenal crisis to begin with and unfortunately by then, the steroids used to treat them make diagnostic testing more difficult. A second approach is to get a random cortisol level with the thought that "the patient is sick, so the level will probably be high." The problem with this approach is that if the cortisol level is 10 to 20 ug/dl, you can neither say the patient is normal or insufficient. For this reason the best test is the ACTH stimulation test.

34. How is the rapid ACTH stimulation test performed?

A baseline sample of blood is drawn at time 0 for a cortisol level. Then 0.25 mg of cosyntropin (synthetic ACTH) is given intravenously. Cortisol levels are checked at 30 minutes and 1 hour later.

35. But what if the patient needs emergent treatment with steroids? Should I withhold treatment until the rapid ACTH stimulation test has been done?

No! If your patient is unstable, you can begin treatment using a glucocorticoid that will not cross react with the cortisol assay. A cortisol level can be drawn and then dexamethasone (6–10 mg) can be administered intravenously. Then cosyntropin 0.25 mg is given intravenously and serum cortisol levels drawn 30 and 60 minutes later. By using this approach, the patient has the benefit of receiving stress-dose steroids in a timely manner

and also has had the appropriate diagnostic test so that the nature of their illness can be better understood as their clinical course develops.

36. How is acute adrenal insufficiency treated?

Stress-dose steroids should be promptly administered once the diagnosis of acute adrenal insufficiency is considered and the ACTH stimulation test initiated. IV administration of hydrocortisone (100 mg minimum) and crystalloid IV fluids containing dextrose is the standard approach. A detailed history and examination should be done to elicit what may have precipitated the stress that caused the acute adrenal insufficiency. If a cause is found, supportive and definitive measures need to be instituted in the ED. If there is uncertainty, empiric administration of broad-spectrum antibiotics may be prudent while waiting for culture results. Mineralocorticoid replacement is usually unnecessary if salt and water replacement is adequate and if the patient receives hydrocortisone—100 mg of hydrocortisone has the salt-retaining effect of 0.1 mg of fludrocortisone.

KEY POINTS: ADRENAL CRISIS

1. Consider adrenal crisis in all hypotensive patients, especially if unresponsive to pressors.
2. All patients in adrenal crisis require rapid administration of IV steroids.
3. Dexamethasone may be initiated in adrenal crisis without affecting the cosyntropin (ACTH) stimulation test.
4. Only 2 weeks of high-dose steroid use can cause adrenal suppression, making a patient more prone to adrenal crisis.

37. What should be done for the patient with chronic adrenal insufficiency who presents to the ED with a minor illness or injury?

These patients usually require supplemental steroid hormone that is appropriate for their degree of medical illness. When seeing a patient who has an acute illness, a dose of hydrocortisone or prednisone that is between the daily replacement dose and the maximal stress dose that is appropriate for the degree of illness that they are experiencing should be administered. A usual daily replacement dose of steroids for someone with adrenal insufficiency who is otherwise healthy would be 20–30 mg/day of hydrocortisone or 5–6 mg of prednisone. Someone who is critically ill would typically be given 100 mg of hydrocortisone three times per day (300 mg) or 60 mg per day of prednisone. This increased dose should be continued for 24–48 hours until symptoms improve. Adding a mineralocorticoid is usually not necessary. Follow-up care should be coordinated closely with the primary care physician or endocrinologist. Patients should be told that if nausea or vomiting develops and they are unable to keep down the medication, they should immediately seek medical attention. Patients should be reminded that they should have a medic-alert bracelet so that emergency physicians can treat them appropriately in the future should they be critically ill and not able to communicate their medical history.

38. What are the signs and symptoms of pheochromocytoma?

Pheochromocytoma is a tumor of the adrenal medulla or sympathetic ganglia that makes excessive catecholamines (e.g., epinephrine, norepinephrine, or dopamine). The classic symptoms of a pheochromocytoma include headache that is typically severe, palpitations, and sweating. These symptoms, occurring in the setting of severe hypertension, especially if the symptoms are episodic, should raise the question of pheochromocytoma. Other symptoms include nervousness, tremor, weight loss, and hyperglycemia.

39. When should the diagnosis of pheochromocytoma be considered?

A diagnosis of pheochromocytoma should be considered in a patient who has the typical symptoms and severe hypertension, especially episodic hypertension, hypertension that requires four or more medications to control, or hypertension that began before the age of 35 years or after age 60. Patients who are hypertensive and have a family history of severe episodic hypertension, or components of multiple endocrine neoplasia type 2 (medullary thyroid cancer, hyperparathyroidism, and pheochromocytoma) should also be considered at risk.

40. What is unique about the treatment of hypertension in a patient with pheochromocytoma?

The most important thing to remember is to not use beta blockers as a first-line treatment when a diagnosis of pheochromocytoma is being considered. This is because β-blockade will result in unopposed α-receptor activation, which will increase vasoconstriction and worsen hypertension. Pure vasodilators can be used in the acute setting. It is important to institute good α-blockade early using medications such as phenoxybenzamine or prazosin. Labetalol has the advantage of having both α- and β-blocking activities and is also useful in this setting.

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4. Hyperthyroidism, thyroid storm, and Graves disease: www.emedicine.com.
5. Myxedema coma or crisis: www.emedicine.com.
6. Thyroid Disease Manager: www.thyroidmanager.org

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SEPSIS SYNDROMES

Stephen J. Wolf, MD

1. What is systemic inflammatory response syndrome (SIRS)?
As its name implies, it is a syndrome of inflammation, not necessarily infection.

KEY POINTS: SIRS CRITERIA (Two of Four Needed for Diagnosis)

1. Temperature >38°C or <35°C
2. Heart rate >90 beats per minute
3. Respiratory rate >20 breaths per minute or $PaCO_2$ <32 mm Hg
4. Serum white blood cell count >12,000 mm^3 or <4,000 mm^3 or 10% band forms

2. How is sepsis defined?
In the ED, sepsis is defined clinically as a syndrome that has the presence of both SIRS and *presumed* bacteremia.

3. What distinguishes sepsis from severe sepsis?
Severe sepsis is sepsis complicated by organ dysfunction. Severe sepsis is now considered to be the most common cause of death in noncoronary critical care units. Approximately 150,000 people die annually in Europe and more than 200,000 die annually in the United States from sepsis.

4. What is the significance of an elevated lactate level in sepsis?
An elevated serum lactate concentration identifies tissue hypoperfusion in patients who are not hypotensive. Although lactate measurements may be useful and correlate with mortality, they lack precision as a measure of tissue metabolic status.

5. What organ systems can become dysfunctional, suggesting severe sepsis?
- **Cardiovascular**: Vasodilation, poor myocardial contractility and increased cardiac oxygen demand, systemic hypotension, or cardiac ischemia
- **Central nervous system**: Altered mental status
- **Global tissue hypoperfusion**: Elevated lactate ⩾ 4.0 mmol/L
- **Hematologic**: Increasing prothrombin time (PT), international normalized ratio (INR), partial thromboplastin time (PTT), hemolysis and thrombocytopenia, or disseminated intravascular coagulopathy (DIC)
- **Liver**: Coagulopathy, jaundice, or elevated transaminases
- **Renal**: Acute renal failure determined by increase in blood urea nitrogen (BUN) and creatinine or decreased urine output to less than 0.5 mL/kg/h
- **Pulmonary**: Acute respiratory distress syndrome, respiratory failure, or unexplained hypoxia

6. What is the mortality rate of sepsis versus severe sepsis?
The mortality rate of sepsis is 15% to 20%, whereas severe sepsis has a 30% to 40% mortality rate.

7. What is the primary goal of resuscitation in a septic patient?
Aggressive resuscitation aims to ensure that oxygen delivery meets oxygen demand of tissues affected by the septic state.

8. What is an easy way to decrease an affected tissue's increased oxygen demand from sepsis?
Early appropriate antibiotics. The Institute for Healthcare Improvement recommends initiation of antibiotics within 3 hours of ED presentation.

9. What are two means of increasing oxygen supply to affected tissues in a septic state?
- High-flow supplemental oxygen
- Early goal-directed therapy (EGDT)

10. What is the mortality benefit to initiating EGDT in severe sepsis patients?
There is a 10% to 20% reduction in mortality.

11. What are the goals outlined in EGDT for patients in severe sepsis?
During the first 6 hours of resuscitation, the goals for initial resuscitation of sepsis-induced hypoperfusion should include all of the following as part of a treatment protocol:
- Central venous pressure (CVP): Goal of 8 to 12 mm H_2O
- Mean arterial pressure (MAP): Goal of $>$ 65 mm Hg
- Urine output: Goal of $\geq$ 0.5 mL/kg/h
- Central venous ($ScVO_2$ obtained from the pulmonary artery) or mixed venous oxygen saturation (SvO_2) obtained from the superior vena cava: Goal of $>$70%

12. What intervention should be used for a CVP that is less than 8 mm H_2O?
Intravenous (IV) fluid resuscitation is the first-line treatment and is given in bolus increments over 30 minutes or until CVP is at the desired goal. Use boluses of 500 to 1000 mL of crystalloids or 300 to 500 mL of colloids, repeated based on response. Caution needs to be used in patients with contraindications to significant volume resuscitation (e.g., patients with congestive heart failure [CHF] or renal failure).

13. What intervention should be initiated for a MAP that is less than 65 mm Hg?
After adequate attempts to raise the patient's CVP to between 8 and 12 mmH_2O with fluid resuscitation (at least 20–40 mL/kg), initiate vasopressor support to increase MAP to 65 mm Hg.

14. Does one vasopressor have a proven benefit over another in the setting of severe sepsis?
Some evidence suggests that norepinephrine is a better first-line vasopressor than dopamine in the setting of sepsis. Although the jury may still be out on that point, it is agreed that both dopamine and norepinephrine are good first- and second-line agents for supportive care in sepsis. Epinephrine is associated with higher mortality in animal models and is generally reserved for use if the patient is failing both dopamine and norepinephrine.

15. What are the implications of a low SvO_2?
This simply means there is a global tissue hypoxia. An SvO_2 of less than 70% suggests that the tissue extraction of O_2 is greater than the delivery needed to sustain the metabolic demands (i.e., poor perfusion).

16. What intervention should be initiated for an SvO_2 <70%?

- If the SvO_2 is less than 70% despite a CVP of at least 8 to 12 mmH_2O and a MAP of at least 65 mm Hg, then consider the use of dobutamine for its inotropic properties to help with cardiac pump function, perfusion, and O_2 delivery.
- Additionally, one may consider transfusing packed red blood cells to increase the patient's hematocrit to a level of 30%. This will help increase oxygen-carrying capacity.

17. What are the drawbacks to transfusion?

Transfusion of blood is initially helpful. There are, however, several potential drawbacks. Acute transfusion reactions and systemic response to minor antigens and storage breakdown products may further increase the immunocompromised state associated with sepsis. Additionally, the optimal end point of transfusion is unclear.

18. Is SvO_2 a reasonable surrogate measure to central venous oxygen saturation ($ScVO_2$)?

Recommendations from the latest international sepsis forum suggest that SvO_2 from a central line placed in the superior vena cava is a comparable and reliably accurate estimate of the $ScVO_2$ gained from a pulmonary artery catheter, Swan-Ganz catheter.

19. What are the implications of meeting these goals as quickly as possible?

There is a clear benefit from aggressively clearing lactate and reversing tissue hypoperfusion in severe sepsis using the goals of EGDT. Rivers and colleagues demonstrated a 16% decrease in absolute 28-day mortality by implementing EGDT through the first 6 hours of patient presentation to the ED.

20. How is septic shock defined?

Septic shock can be defined as severe sepsis with ongoing tissue hypoperfusion refractory to resuscitation.

21. What is the role of vasopressin?

Currently, vasopressin is a second- to third-line vasopressor and is reserved for failure of other vasopressors in the setting of septic shock with refractory hypotension. Vasopressin does not confer a mortality benefit and causes extreme peripheral vasoconstriction that may result in digital ischemia.

22. What is the role of recombinant activated protein C (rhAPC) in sepsis?

Recombinant APC (Drotrecogin Alfa) is a novel sepsis therapy that has demonstrated a 6.1% to 13% absolute reduction in mortality in some studies. The mortality benefit appears to be highest in those who are the sickest with an Acute Physiology and Chronic Health Evaluation II (APACHE II) score >25. The use of rhAPC in all septic patients is controversial due to the lack of efficacy in studies of patients with APACHE II scores <25 and the expense of the treatment. The cost of each treatment is currently around $8,000.

23. What is the role of tight glycemic control in sepsis?

There are data to demonstrate that in critically ill patients there is a 50% reduction from 8% to 4.6% in intensive care unit (ICU) mortality with tightly controlled glucose between 80 and 110 mg/dL. Therefore, it is recommended that an aggressive insulin-controlled glucose protocol be started in critically ill patients in the ED.

WEBSITE

Institute for Healthcare Improvement: www.ihi.org/IHI/Topics/CriticalCare/Sepsis/

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SOFT-TISSUE INFECTIONS

Harvey W. Meislin, MD, FACEP, FAAEM and Megan A. Meislin, MD

CHAPTER 48

1. How do I differentiate cellulitis from an abscess?

Cellulitis is a soft-tissue infection of the skin and subcutaneous tissue usually characterized by blanching erythema, swelling, pain or tenderness, and local warmth. A cutaneous abscess is a localized collection of pus that results in a painful soft-tissue mass that is often fluctuant but surrounded by firm indurated granulation tissue and erythema.

2. What are the causes of cellulitis? How does it progress?

Although most often acute, cellulitis may be subacute or chronic. Cellulitis occurs most often in the lower extremities, then the upper extremities, followed by the face. Minor trauma is often the predisposing cause, but hematogenous and lymphatic dissemination may account for its appearance in previously normal skin. Bacterial cellulitis may progress to ascending lymphangitis and septicemia. Cellulitis caused by bacterial infection tends to spread radially with associated swelling, whereas nonbacterial or inflammatory cellulitis tends to stay localized.

3. What are the causes of abscesses? How do they progress?

Abscesses occur on all areas of the body, although they have a predominance for the axilla, perirectal region, head and neck, and extremities, respectively. The cause of localized abscesses depends on the anatomic region involved; abscesses on the extremities are usually caused by interruptions of the integrity of the protective epithelium; head and neck abscesses tend to be associated with obstruction of the apocrine glands; oral and perineal abscesses originate from the mucous membranes. Superficial abscesses tend to remain localized and often rupture spontaneously through the skin if not incised and drained.

KEY POINTS: CELLULITIS VERSUS ABSCESS

1. Abscesses contain pus; cellulitis does not.
2. Fluctuance surrounded by induration signifies fluid, usually pus. Fluctuance, if present, signifies abscess formation; the absence of fluctuance does not rule out an abscess.
3. There is an increasing incidence of community-associated MRSA.
4. Community-associated MRSA has different sensitivities and susceptibilities to antibiotics than does hospital-associated MRSA.
5. Treatment for cellulitis is immobilization, elevation, heat, analgesics, and antibiotics.
6. Cutaneous abscesses can, in general, be treated solely with incision and drainage.

4. What is pus? Why is the presence of pus significant?

Pus is a heterogeneous mix of cellular material in various stages of digestion by polymorphonuclear leukocytes (PMNs). These PMNs are drawn to sites of inflammation,

infection, or trauma by various chemotactic factors to defend the host against potential pathogens. Abscesses contain pus, whereas cellulitis does not. Thus abscess and cellulitis may be present in the same anatomic area; the presence of pus defines the diagnosis of an abscess and the need for incision and drainage.

5. How do I know if pus is present?

In cutaneous abscesses, a raised painful mass with a fluctuant center surrounded by indurated erythematous tissue signifies the presence of pus. Adjunctive radiographic techniques, such as ultrasound or computed tomography (CT) scan, may be useful for deeper soft-tissue infections but are rarely indicated with cutaneous abscesses. The use of a localizer needle is often helpful, especially in wounds in which the purulence is loculated. Needle aspiration of the involved area with a needle large enough to withdraw thick pus often helps to define the location of purulence for incision and drainage and makes the process more comfortable by decreasing the pressure and pain in the area.

6. What are the differential diagnoses for cellulitis and abscess?

The differential for cellulitis and abscess is one of bacterial versus nonbacterial infection. The etiologies of nonbacterial cellulitis include arthropod envenomation, chemical or thermal burns, arthritis, and healing wounds. Nonbacterial cellulitis is usually localized and often lacks lymphangitic streaking. The differential diagnosis of abscesses includes sterile abscesses, cutaneously borne bacterial abscesses, and mucous membrane abscesses. Abscesses of the oral and anorectal area usually originate from flora indigenous to those areas. Sterile abscesses, which occur approximately 5% of the time, tend to be associated with drug abuse and subcutaneous injections. Most abscesses are isolated; recurrent abscess formation signifies a more complicated or systemic disease process.

7. Is it useful to culture cellulitis or abscesses?

Culturing cellulitis is often futile because a causative agent is identified only 10% of the time. Often there is secondary skin contamination leading to misidentification of the cause. Cultures can be useful, however, in patients who do not respond to initial management, in patients with recurrent disease, or in patients with sepsis. Culturing the portal of entry may be useful, even if distal to the site of the cellulitis. Culturing of cutaneous abscesses seldom is clinically indicated in patients with normal host defenses because they tend to contain and localize the process. In patients with AIDS, diabetes mellitus, leukemia, vascular insufficiency, trauma, burns, or recurrent abscess, or when concerned for community-associated methicillin-resistant *Staphylococcus aureus* (MRSA) or with failure of initial therapy, Gram stain and culture may be indicated.

8. What is the yield of blood cultures when treating cellulitis?

A number of studies have demonstrated that the routine ordering of blood cultures in the ED is not warranted in an immunocompetent host with uncomplicated cellulitis, except in suspected cases of *Haemophilus influenza*e, type B. The impact on clinical management is marginal. One study reported that blood cultures were twice as likely to be contaminated as to be true positives. Blood cultures should be considered prior to starting antibiotic therapy in immunocompromised patients, in those with an exposure to unusual organisms, and in patients with a potentially complicated cellulitis.

9. What is community-associated MRSA?

MRSA was first recognized as a community-associated pathogen in the early 1980s. Community-associated MRSA skin and soft-tissue infections are spread within the community and are genetically distinct from hospital-associated MRSA infections. Community-associated MRSA has different sensitivities and susceptibilities to antibiotics than hospital-associated MRSA as well.

10. Who is at risk for acquiring community-associated MRSA?

The prevalence of MRSA colonization in the community ranges from 0.2% to 2.8%. Highest rates are seen among poor urban populations. There is a high prevalence among injection drug users, as well as in prison populations, athletes sharing equipment, and isolated American Indian communities.

11. Is there a role for routine laboratory studies?

Laboratory studies are generally not helpful in the treatment of superficial soft-tissue infections, unless signs or symptoms of systemic illness are present or the patient is immunocompromised. These patients are often not systemically ill, and even an elevated white blood cell (WBC) count does not differentiate bacterial from nonbacterial infection, identify the presence of abscess or cellulitis, or show systemic involvement. An exception may be *H. influenzae* cellulitis, in which WBC counts often exceed 15,000/mm^3 with a left shift, often occurring in children.

12. Summarize appropriate treatment of soft-tissue infections.

The time-honored treatment for cellulitis is immobilization, elevation, heat or warm moist packs, analgesics, and antibiotics directed toward suspected pathogens. The treatment for cutaneous abscesses is a properly performed incision and drainage.

13. Should I routinely prescribe antibiotics for patients with an abscess?

No. The treatment for most cutaneous abscesses is incision and drainage, and neither antibiotics nor cultures are indicated in patients with normal host defenses as long as the abscess is localized. In patients with complications of diabetes, AIDS, leukemia, neoplasms, significant vascular insufficiency, trauma, thermal burns, or suspicion for MRSA, antibiotics should be considered as prophylaxis to prevent spread of bacteria into local tissues or the bloodstream. Prophylactic antibiotics, although usually not necessary, may also be considered for abscesses of the face, groin, and hand. For abscesses associated with immunocompromised patients, progressing cellulitis, hospital-acquired MRSA, and penetration into deeper soft tissues, incision and drainage (often in the operating room), antibiotic therapy, culture, and Gram stain constitute a reasonable initial approach.

The selection of antimicrobial agent can be facilitated by knowing the flora associated with the anatomic area involved, if the abscess is from a cutaneous or mucosal process, and the most likely cause of the infection. Gram stain results of the purulence in these cases may be helpful.

14. How do you treat community-associated MRSA?

Often simple abscesses can be treated solely with incision and drainage, but when antibiotics are deemed appropriate in the treatment of skin and soft tissue infections, it is no longer recommended to use a β-lactam such as cephalexin. Antimicrobial susceptibility patterns of MRSA all demonstrate uniform resistance to oxacillin. Susceptibilities appear highest to trimethoprim-sulfamethoxazole, clindamycin, tetracycline, levofloxacin, and vancomycin.

15. How does the presence of community-associated MRSA change the management of soft-tissue infections in the ED?

Previously, it was recommended that all suspected MRSA abscesses should be cultured in the ED prior to starting antimicrobial therapy. Antibiotics active against community-associated MRSA should be used in the treatment of skin and soft-tissue infections determined to require antimicrobial treatment. However, it does appear that localized cutaneous abscesses with community-acquired MRSA will respond to incision and drainage alone without the need for adjunctive antibiotics.

16. Are there anatomic areas of significance in a patient with an abscess or cellulitis?

Cellulitis of the midface, especially in the area of the orbits, must be treated aggressively. The venous drainage of these infections is through the cavernous sinus of the brain, with the potential for causing cavernous sinus thrombosis. In true orbital cellulitis, aggressive intravenous (IV) antibiotic therapy is warranted. Often a CT scan is performed to detect abscess formation. *H. influenzae* cellulitis usually occurs in children, resulting in high fevers, high WBC counts, and bacteremia. Perirectal or perianal abscesses that are large or extend into the supralevator or ischiorectal space often need intraoperative management, removing not only the abscess but also the fistulae that are often associated with it. Deep space abscesses of the groin and head and neck region also must be drained in the operating room because of their proximity to major neurovascular structures.

17. Describe appropriate follow-up care.

Most patients with simple cellulitis and localized abscesses can be treated on an outpatient basis. Abscess packing from an incision and drainage procedure can be removed after 48 to 72 hours, and the patient can clean the abscess cavity by bathing or showering at home. It is important to ensure that cellulitis is responding to therapy and, in the case of abscess, that all pus has been drained and evacuated. Further follow-up is indicated only when the processes are recurrent, when there is no response to therapy in 48 to 72 hours, or when the patient is immunocompromised.

18. Who should be admitted to the hospital?

Patients who appear septic, are immunocompromised, are not responding to treatment; patients with soft-tissue infections in certain anatomic sites, such as the central area of the face; and patients with infections, such as sublingual and retropharyngeal abscesses and Ludwig's angina that potentially may cause airway closure, should be admitted. Close attention must be paid to immunosuppressed patients, who may develop abscesses or cellulitis as secondary infections from gram-negative or anaerobic gas-forming organisms. Abscesses in the perineal area may spread quickly through the fascial planes, resulting in Fournier's gangrene.

19. Is there an association between abscesses or cellulitis and systemic disease?

Patients who are immunocompromised or have peripheral vascular disease have a tendency to develop superficial soft-tissue infections. Recurrent abscesses in the head and neck or groin regions may be associated with hidradenitis suppurativa, which is a disease of chronic suppurative abscesses of the apocrine sweat glands. Inflammatory bowel disease, diabetes, malignancies, and pregnancy have been associated with a higher incidence of perirectal abscesses. Recurrent abscesses in the perineal and lower abdominal area may signify the presence of associated inflammatory bowel disease. All patients with recurrent soft-tissue infections, whether superficial or deep, should be evaluated for underlying systemic disease such as diabetes.

20. What is the best advice overall for treating cellulitis and abscesses?

Cellulitis usually responds to antibiotic therapy and immobilization. Cutaneous abscesses usually respond to adequate incision and drainage; antibiotics are not indicated. All soft-tissue infections should be observed to ensure that healing is occurring. Selection of antibiotics, when indicated, is guided by the location and cause of the infection.

21. What is necrotizing fasciitis?

A life-threatening and limb-threatening bacterial infection of the fascia often extending to the skin and subcutaneous tissue. Multiple bacteria are usually involved. The most common are gram-positive cocci (*Streptococcus* and *Staphylococcus*), gram-negative organisms (*Enterococcus, Proteus*, and *Pseudomonas*), and anaerobes (*Clostridium, Escherichia coli,*

Bacteroides fragilis). Bacteria usually enter the subcutaneous tissue through a break in the skin, often caused by minor or trivial trauma. Bacterial substances or exotoxins cause separation of the dermal connective tissue resulting in inflammation and necrosis. Blood-borne and postoperative infection may lead to necrotizing fasciitis.

22. How is necrotizing fasciitis diagnosed?

The diagnosis should be considered in any patient with a soft-tissue infection who has pain and tenderness out of proportion to the visible degree of cellulitis. It also should be considered in patients without any skin changes who have exquisite tenderness without any obvious reason such as a history of musculoskeletal trauma. Some patients may have subcutaneous emphysema. Most patients develop sepsis late in the course, and in severe cases disseminated intravascular coagulopathy develops. A soft-tissue radiograph may be helpful to visualize subcutaneous emphysema. CT and magnetic resonance imaging (MRI) are helpful when this diagnosis is suspected. Later in the course, the skin may reveal bullous lesions and necrotic patches.

KEY POINTS: NECROTIZING FASCIITIS

1. Pain and tenderness are out of proportion to the visible degree of cellulitis.
2. Subcutaneous emphysema may be present.
3. This is a disease requiring early surgical consultation.

23. What is the laboratory risk indicator for necrotizing fasciitis (LRINEC) score?

The LRINEC score is a scoring system developed from a retrospective analysis to differentiate between necrotizing fasciitis and other soft-tissue infections. The score is calculated by totaling up the six predictive factors in Table 48-1. A score of 6 or greater had a positive predictive value of 92% and a negative predictive value of 96%.

24. Why should I get a surgical consultation?

Necrotizing fasciitis is a disease that must be rapidly treated with extensive incision, drainage, and debridement of necrotic tissue. Additional therapy includes IV antibiotics and in-hospital supportive care.

25. What is Fournier's gangrene?

A necrotizing subcutaneous infection of the perineum occurring primarily in men, usually involving the penis and scrotum. It most commonly affects individuals who are immunologically compromised or diabetic. Typically, it begins as a benign infection or small abscess with symptoms of pain and itching that quickly progresses and leads to end-artery thrombosis in subcutaneous tissues. Ultimately, it leads to widespread necrosis of adjacent areas. Any patient complaining of lesions or pain in the aforementioned areas should be approached with this diagnosis in the differential.

26. List the most common (and concerning) organisms found in the following wounds and their accompanying cellulitis.

- **Cat bites**: *Pasteurella multocida* (80%), *Staphylococcus, Streptococcus*
- **Dog bites**: *Pasteurella, Enterobacter, Pseudomonas, Capnocytophaga canimorsus* (rare, but 25% fatality in immunocompromised patients)
- **Human bites**: *Streptococcus, Staphylococcus*, *H. influenzae*, *Eikenella corrodens*, *Enterobacter, Proteus*
- **Open water wounds**: *Aeromonas hydrophila, Bacteroides fragilis, Chromobacterium, Mycobacterium marinum, Vibrio*

TABLE 48–1. THE LRINEC SCORE

Variable	Score
C-reactive protein (mg/L)	
<150	0
150 or more	4
Total white cell count (per mm^3)	
<15	0
15–25	1
>25	2
Hemoglobin (g/dL)	
>13.5	0
11–13.5	1
<11	2
Sodium (mmol/L)	
135 or more	0
<135	2
Creatine (μmol/L)	
141 or less	0
>41	2
Glucose (mmol/L)	
10 or less	0
>10	1

LRINEC, laboratory risk indicator for necrotizing fasciitis).

To convert the values of glucose to milligrams per deciliter, multiply by 18.015. To convert the values of creatinine to milligrams per deciliter, multiply by 0.01131.

27. What question must be asked of all patients presenting with cellulitis or abscesses?

When was your last tetanus booster? Current recommendations suggest tetanus-diphtheria toxoid (Td), 0.5 mL IM × 1, if it has been more than 5 years since the previous booster.

TABLE 48–2. ORAL THERAPY FOR SUPERFICIAL SOFT-TISSUE INFECTIONS

Drug	Dose
Group A *Streptococcus*	
Penicillin V (phenoxymethylpenicillin)	250–500 mg qid
First-generation cephalosporin	250–500 mg qid
Erythromycin	250 mg–1 g q 6 h
Azithromycin	500 mg × 1 dose, then 250 mg q day × 4
Clarithromycin	500 mg bid
***Staphylococcus aureus* (not methicillin-resistant *Staphylococcus aureus*)**	
Dicloxacillin	125–500 mg qid
Cloxacillin	250–500 mg qid
First-generation cephalosporin	250–500 mg qid
Erythromycin (variable effectiveness)	250–500 mg qid
Azithromycin	500 mg × 1 dose, then 250 mg q day × 4
Clarithromycin	500 mg bid
Clindamycin	150–450 mg qid
Amoxicillin-clavulanate	250–500 mg tid
Ciprofloxacin	500 mg bid
***S. aureus* (community-associated methicillin-resistant) MRSA**	
Trimethoprim (TMP)-sulfamethoxazole (SMX)	160 mg TMP/800 mg SMX bid
Clindamycin	150–450 mg qid
Tetracycline	250–500 mg qid
Linezolid	600 mg bid
Haemophilus influenzae	
Amoxicillin-clavulanate	875/125 mg bid
Cefaclor	250–500 mg tid
TMP-SMX	160 mg TMP/800 mg SMX bid
Azithromycin	500 mg × 1 dose, then 250 mg q day × 4
Clarithromycin	500 mg bid

bid, twice a day; d, day; q, every; qid, four times a day; tid, three times a day; TMP-SMX, trimethoprim-sulfamethoxazole.

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SEXUALLY TRANSMITTED DISEASES AND HIV INFECTION

CHAPTER 49

Kerry B. Broderick, MD, Erica Douglass, MD, and Jason Haukoos, MD, MSc

1. What are the most common sexually transmitted diseases (STDs)?

The true incidence of most STDs is unknown because not all cases are reported. The Centers for Disease Control and Prevention (CDC) estimate that 19 million new STD infections occur annually in the United States, nearly half of them among persons aged 15 to 24 years.

- **Chlamydia** is estimated to infect 3 million people annually and is a major health problem for young women because of the sequelae of infertility and ectopic pregnancy. In 2007, over 1.1 million cases were reported to the CDC.
- The incidence of **gonorrhea** peaked at more than 1 million cases per year in the late 1970s. In 2007, 355,991 cases were reported to the CDC. The rate of gonococcal infections is highest among adolescent girls.
- **Trichomoniasis** is the most common curable STD in young sexually active women. An estimated 7.4 million new cases occur each year.
- Genital **human papillomavirus** (HPV) is estimated to have a prevalence of over 26% in women. More than 30 types of HPV can cause genital tract infection. Genital warts usually are caused by HPV type 6 or 11. Several types are associated with cervical dysplasia. There is now a vaccine that prevents the types of genital human papillomavirus (HPV) that cause most cases of cervical cancer and genital warts. The vaccine, Gardasil®, is given in three shots over 6 months. The vaccine is routinely recommended for 11- and 12-year-old girls. It is also recommended for girls and women age 13 through 26 who have not yet been vaccinated or completed the vaccine series. With the implementation of this vaccine, it is expected that the number of HPV cases will begin to decline in upcoming years.
- **Genital herpes** occurs in 1 in 5 adolescents and adults.
- **Syphilis** was on the decline with an all-time low in 2000. Since 2001, however, the rate of both primary and secondary syphilis has risen every year. In 2007, 11,466 cases of syphilis were reported. The greatest increase was seen in males having sex with males (MSM). Syphilis is substantially more common in non-Hispanic blacks than in other ethnic groups with an estimated occurrence rate of over seven times that seen in non-Hispanic whites.
- Cases of **HIV**—the most deadly STD—continue to rise. Worldwide, it is estimated that approximately 56 million people are infected with HIV, and more than 26 million have died of AIDS. An estimated 1.1 to 1.2 million people in the United States are living with HIV, of which there are approximately 250,000 individuals who do not know they are infected. In 2006, 56,300 people were newly diagnosed with HIV. It is estimated that more than 36,000 patients currently live with AIDS in the United States.

2. How should I evaluate abnormal vaginal discharge?

The first thing to do is to take a complete sexual history:

a. How many partners has the patient had in the last several months (male or female)?
b. Has she used protective barriers such as condoms and dental dams with each episode?

c. Ask about previous STDs.
d. Obtain a pregnancy test to decide on the most appropriate method of treatment if needed.

- The appearance of the discharge on **pelvic examination** is important. Always take a sample for wet preparation and potassium hydroxide.
- **Vulvovaginal candidiasis** (not an STD) causes a white, curdlike discharge that clings to vaginal walls. Hyphae are present on potassium hydroxide preparation. Recent antibiotic use is a risk factor for this, as are diabetes and HIV. Treatment is single-dose oral fluconazole or any of the topical imidazoles.
- **Bacterial vaginosis** is not an STD but an alteration of the microbial ecosystem with overgrowth of *Gardnerella vaginalis* and other species. Diagnosis is made by noting clue cells on the wet preparation, and treatment is with metronidazole.
- ***Trichomonas vaginitis***, the third common cause, is a true STD. It causes a green, frothy discharge, and the cervix may be erythematous and friable (strawberry cervix). Diagnosis is based on finding the motile trichomonads on wet preparation or in urine. Treatment is with metronidazole.
- A discharge with significant leukocytes that does not include yeast, clue cells, or *Trichomonas* may be due to **mucopurulent cervicitis** (MPC).

3. A sexually active young man presents with dysuria. How likely is it that it resulted from a urinary tract infection?

About as likely as getting gonorrhea from sitting on a toilet seat. Dysuria in young men almost always is due to urethritis from an STD. The urinalysis will be positive for leukocytes, making a urinalysis not helpful and confusing to the novice. The likely pathogens include gonorrhea, *Chlamydia, Ureaplasma, Trichomonas*, and herpes simplex virus (HSV). A purulent discharge most likely is caused by gonorrhea, whereas a mucoid discharge most likely is caused by infection with *Chlamydia*. The patient should be tested for both of these pathogens. *Chlamydia* also can infect the urethra of women, and they may present only with dysuria. Consider this diagnosis in a woman with dysuria and no bacteria on urinalysis, and do a pelvic examination.

4. Are there any single-dose treatment regimens for uncomplicated chlamydial infections?

Yes, a single 1-g dose of **azithromycin** is an effective treatment for lower tract chlamydial infections, including urethritis and cervicitis. Single-dose therapy is *not* appropriate for upper tract disease, such as epididymitis and pelvic inflammatory disease (PID) or in patients who have had a recent chlamydial infection and may be a treatment failure. This simplified therapy should lead to more effective treatment in noncompliant patients.

5. Are there suitable oral alternatives to parenteral therapy for gonorrhea?

Uncomplicated urethral, endocervical, or rectal gonorrheal infections can be treated adequately with a single intramuscular (IM) injection of ceftriaxone (125 mg) or an equivalent third-generation cephalosporin antibiotic such as cefixime (400mg). Fluoroquinolones are no longer recommended for the treatment of gonorrheal infections given the high rates of resistance and rise in fluoroquinolone-resistant *Neisseria gonorrhoeae (QRNG)*.

6. What is the significance of finding mucopurulent cervicitis (MPC) in a woman with lower abdominal pain?

The normal endometrial secretion, as noted on exit from the endocervical canal, should be transparent. The presence of a mucopurulent secretion from the os, which may appear yellow when viewed on a white cotton-tipped swab (positive Q-Tip sign), suggests MPC. MPC, most commonly caused by gonorrhea or *Chlamydia*, is a precursor to upper genital tract infection.

7. How do I evaluate a sexually active young person who presents with an acutely swollen, warm, painful right ankle?

This patient, with acute monarticular arthritis, should be presumed to have **disseminated gonococcal infection.** This is a syndrome of gonococcal bacteremia that leads to peripheral

manifestations of disease, including dermatitis, tenosynovitis, and septic arthritis. Arthrocentesis should be done on the involved joint, and the fluid should be sent for Gram stain, culture for gonococcus (GC) and regular aerobic cultures, and cell count. GC is cultured from less than 50% of joints. A genitourinary examination must be done to culture the cervix, rectum, and urethra as appropriate for GC. A patient suspected of having disseminated gonococcal infection should be admitted initially and treated with parenteral antibiotics (ceftriaxone, 25–50 mg/kg/day intravenously [IV] or intramuscularly in a single daily dose for 7 days).

8. What are the most common causes of genital ulcers?

Genital ulcers can represent infection with HSV, chancroid, or syphilis. It is difficult to make a diagnosis based solely on history and physical examination. Always ask about travel history and exposure to prostitutes. Genital ulcers are an important risk cofactor for HIV transmission.

- **HSV.** Genital herpes due to HSV is the most common cause of genital ulcers in the United States. Primary HSV infection results in severely ill patients who are toxic with fever, malaise, and inguinal adenopathy. Diagnosis is made by viral culture or antigen testing. HSV is a recurrent disease, and patients may shed the virus while they are asymptomatic. It cannot be cured, but treatment with antiviral agents can shorten the duration of symptoms. Long-term suppressive therapy can prevent outbreaks of ulcers.
- **Chancroid.** Also called *soft sore*, this disease is caused by *Haemophilus ducreyi*, a bacterium that is difficult to culture. Clinically, this syndrome causes a painful nonindurated papule that erodes into an ulcer. Painful inguinal adenopathy is found in more than 50% of cases. Treatment options include single-dose azithromycin or ceftriaxone or 3 days of ciprofloxacin or 1 week of erythromycin.
- **Syphilis.** Primary syphilis presents with a painless indurated ulcer called a *chancre*. Diagnosis is best made by dark-field examination for spirochetes, although this is usually available only in public health laboratories. A Venereal Disease Research Laboratory (VDRL) test should be done on anyone with possible syphilis. Treatment for primary syphilis is penicillin G benzathine, 2.4 million U intramuscularly.

9. What is the Jarisch-Herxheimer reaction?

After initiation of treatment for syphilis, onset of fever, chills, myalgias, headache, tachycardia, increased respirations, increased neutrophil count, and mild hypotension. This occurs approximately 2 hours after initiation of treatment with peak temperatures at approximately 7 hours, with defervescence at 12 to 24 hours. This reaction occurs in 50% of primary syphilis, 90% of secondary syphilis, and 25% of early latent syphilis patients. In secondary syphilis patients, the mucocutaneous lesions may become more edematous and erythematous.

10. Proctitis is a problem primarily seen in men who have sex with men. Discuss the approach and treatment.

Any individual, male or female, with the onset of acute proctitis symptoms (e.g., rectal pain, discharge, tenesmus) who recently has had unprotected, receptive anal intercourse is at risk for an STD-related problem. These patients should be examined by anoscopy and should be tested for gonorrhea, *Chlamydia*, and HSV. All patients should have serologic testing for syphilis. These patients should have empirical treatment for gonorrhea and *Chlamydia*. If ulcers are apparent on anoscopy, consider empirical antiviral therapy with acyclovir.

11. Do I need to report STD cases to the health department?

Yes. Accurate reporting of STDs is essential to national and local STD control efforts. HIV, gonorrhea, and syphilis are reportable infections in every state. Chlamydial infection is reportable in most states. It is the responsibility of each clinician to know his or her local reporting requirements. If you are unsure of what to report about a specific patient, contact your local health department.

12. What are the important points to address in the discharge instructions for STD patients?

a. Education about STDs is the responsibility of every ED physician because you may be the only contact the patient has with the medical system.
b. Instruct patients to refer *all* their sexual partners for evaluation and treatment. Some physicians in the United States routinely provide additional antibiotic prescriptions for sexual partners. Although it is well intentioned, it is controversial to provide a prescription for a person you have not interviewed or examined. That person may be allergic to the medication or may have additional infections that you are not treating.
c. All patients should be instructed to avoid sexual contact with their partners until all parties have finished treatment. Because it is unrealistic to expect all patients to follow this advice, explain the importance of using condoms with every sexual contact to avoid further infections and to prevent infection with HIV.

13. What is the significance of HIV infection in patients seen in the ED?

Disease caused by HIV infection, ranging from asymptomatic infection to AIDS, with serious, possibly life-threatening complications, is encountered commonly in the ED. Seroprevalence among ED patients varies greatly, depending on the location and type of hospital. Among inner-city ED patients, seroprevalence ranges from approximately 5% to 10%. Knowledge of HIV infection and its related diseases is essential to diagnose and treat patients and to ensure adequate protection of health care workers.

14. How is the diagnosis of AIDS made?

AIDS is diagnosed by laboratory evidence of HIV infection and the presence of one of the AIDS-defining illnesses, some of which are listed in Table 49-1. HIV infection should be suspected in all patients with known behavioral risk factors or with presenting symptoms suggestive of an opportunistic infection. Questioning the patient directly about risk factors may be crucial to diagnosing HIV-related disease. High-risk behaviors commonly associated with HIV infection include unprotected sexual intercourse, unprotected insertive or receptive sex between men, and injection drug use.

TABLE 49-1. AIDS-DEFINING CONDITIONS

Laboratory evidence of HIV infection plus any of the following:		
Esophageal candidiasis	Brain lymphoma	HIV wasting syndrome
Cryptococcosis	*Mycobacterium avium* complex	Disseminated histoplasmosis
Cryptosporidiosis	*Pneumocystis carinii* pneumonia	Isosporiasis
Cytomegalovirus	Progressive multifocal	Disseminated *Mycobacterium* retinitis leukoencephalopathy *tuberculosis* disease
Herpes simplex virus	Brain toxoplasmosis	Recurrent *Salmonella* septicemia
Kaposi's sarcoma	HIV encephalopathy	CD4 lymphocyte count <200/mL
Pulmonary tuberculosis	Invasive cervical cancer	

15. Should EDs test for HIV infection?

Testing for HIV has not been traditionally performed in the ED because of difficulty in maintaining confidentiality and ensuring appropriate reporting and counseling. An increasing number of EDs are now performing HIV testing, recognizing that integrating HIV testing into ED operations is possible. The most common HIV testing approach is diagnostic testing (i.e., where physicians are able to test patients based on clinical signs or symptoms), although others, including the CDC, have advocated for performing routine opt-out rapid HIV screening. Several rapid tests are available that are highly accurate and have quick turn-around times. Reactive rapid tests should be confirmed on an outpatient basis by performing an enzyme-linked immunoassay (EIA) and a Western blot (WB). Regardless of whether or not HIV testing is performed in the ED, outpatient referral for high-risk patients is appropriate.

16. How do patients with HIV infection present to the ED?

Patients may present with involvement of virtually any organ system. HIV infection should be suspected in any patient thought to be immunocompetent but with an infectious disease (e.g., community-acquired pneumonia or cellulitis in an otherwise healthy adult), those with unexplained leukopenia or lymphopenia, and those who present with chronic symptoms (e.g., weight loss, fever, or diarrhea) or with symptoms of opportunistic infection. Among patients with HIV infection, systemic infection, or malignancy always must be considered and may present with malaise, anorexia, fever, weight loss, gastrointestinal (GI) complaints, or other symptoms. Because of the wide spectrum of disease related to HIV infection, many specific diagnoses cannot be made definitively in the ED; treatment focuses on recognition of disease, institution of initial therapy, and admission to the hospital or close outpatient follow-up.

17. What tests should be done for the HIV-infected patient with systemic symptoms?

In addition to a complete history and physical examination, appropriate laboratory investigations may include electrolytes, complete blood count, blood cultures (i.e., aerobic, anaerobic, and fungal), urinalysis and culture, lactate dehydrogenase, liver function tests, chest radiography, serologic testing for syphilis, blood tests for cryptococcal antigen, and *Toxoplasma* and *Coccidioides* serologies. Lumbar puncture also may be appropriate if no other source of fever is identified.

18. Explain the significance of fever in patients with HIV infection.

Fever may indicate bacterial, fungal, viral, or protozoal infection. The most common causes of fever include HIV-related fever, systemic infections such as *Mycobacterium avium* complex, cytomegalovirus, Hodgkin's disease, and non-Hodgkin's lymphoma.

Many HIV-infected patients with fever may be managed as outpatients, although this will depend heavily on the patient's CD4 count. A CD4 count less than 200 cells/uL defines AIDS, and these patients should be hospitalized for further evaluation. Patients with high CD4 counts (e.g., >350 cells/uL) may be managed as an outpatient if the patient appears clinically well. Outpatient management may be attempted if the fever source is found and does not dictate admission, if appropriate laboratory studies have been initiated, if the patient is able to function adequately at home (able to ambulate and tolerate oral intake), and if appropriate close medical follow-up can be arranged.

19. What are the common neurologic complications of AIDS?

The most common acute symptoms are altered mental status, seizures, and headache. Because these patients are immunosuppressed, they commonly do not manifest symptoms thought to be associated with central nervous system (CNS) infections. For example, meningismus is rare and patients with meningitis may only present with mild headache. ED evaluation should include a complete neurologic examination and, when appropriate, computed tomography (CT) or magnetic resonance imaging (MRI), and lumbar puncture. Specific cerebrospinal fluid studies that may be of value include cell count, glucose,

protein, Gram stain, cryptococcal antigen bacterial culture, viral culture, fungal culture, *Toxoplasma* and cryptococcal antigen, and coccidioidomycosis titer. The most common causes of neurologic symptoms include *Cryptococcus neoformans*, *Toxoplasma gondii*, HIV encephalopathy, and CNS lymphoma.

20. What is HIV encephalopathy?

Also referred to as *AIDS dementia*, it is an organic brain syndrome manifested by decline in attention, cognitive reasoning, speech, motor function, and motivation. HIV encephalopathy is the most common neurologic problem and affects 33% to 60% of patients. It may be the presenting sign of overt AIDS in 25% of patients. Other causes of dementia and altered mental status must be ruled out.

21. What are the pulmonary complications of HIV infection? How are they managed?

Common presenting pulmonary complaints are cough, hemoptysis, shortness of breath, and chest pain. After history and lung examination, arterial blood gases, chest radiography, sputum culture, Gram stain, acid-fast stain, and blood cultures should be obtained if clinically indicated. The most common pulmonary infection is *Pneumocystis carinii* pneumonia, which occurs in 70% to 80% of seropositive patients and typically presents with dyspnea, dyspnea with exertion, nonproductive cough, fever, and weight loss. Rapid institution of therapy with IV trimethoprim-sulfamethoxazole (TMP-SMX), based on weight for dosing (dapsone or pentamidine if TMP-SMX allergic), and oral steroids may prevent excessive morbidity and mortality. Other causes include *Mycobacterium tuberculosis* pneumonia, *Histoplasma capsulatum*, other traditional community-acquired pneumonia organisms, and neoplasm.

ED management includes administration of supplemental oxygen, volume repletion if indicated, and antibiotic therapy. Admission should be considered for patients with new-onset pulmonary symptoms or patients with a significant deterioration in respiratory status.

Patients with *Pneumocystis* pneumonia should be treated with intravenous TMP-SMX, 15 to 20 mg/kg/day in divided doses. Alternate treatments may be used with primaquine plus clindamycin, or atovaquone, or pentamidine. Patients with hypoxemia ($pO_2 < 70$) or a large Aa gradient (>35) should also be treated with corticosteroids (such as prednisone taper of 40 mg twice daily for 5 days followed by prednisone 40 mg daily for 5 days followed by prednisone 20 mg daily for 11 days).

22. How should GI complaints be managed?

Approximately 50% of AIDS patients present with GI complaints at some time during their illness. Esophageal complaints are common and may be most commonly caused by *Candida* esophagitis or herpes simplex esophagitis. Patients with esophagitis should receive a 2-week empiric course of oral antifungal agents, followed by endoscopy if not successfully treated. The most common presenting symptoms are abdominal pain, bleeding, and diarrhea. Diarrhea is the most common GI complaint and is estimated to occur in 50% to 90% of AIDS patients. Helpful laboratory studies include microscopic examination of stool for leukocytes, acid-fast stain, examination for ova and parasites, and bacterial culture of stool and blood. *Cryptosporidium* and *Isospora* infections in particular are common causes and are associated with prolonged watery diarrhea. Other common infectious agents include *Candida*, Kaposi's sarcoma, *M. avium* complex, HSV, cytomegalovirus, *Campylobacter jejuni, Entamoeba histolytica, Shigella, Salmonella, Giardia, Cryptosporidium*, and *Isospora*. Management should be directed at repletion of fluid and electrolytes and appropriate antibiotic coverage.

23. What are the common cutaneous presentations of AIDS and how are they treated?

Kaposi's sarcoma is the most common unique cutaneous manifestation of AIDS. Usually it is widely disseminated and may involve mucous membranes. Exacerbation of underlying dermatologic conditions is common in the HIV-infected population. Complaints such as

xerosis (dry skin) and pruritus are common and may be manifested before development of opportunistic infections. Xerosis may be treated with emollients and, if necessary, with mild topical steroids. Pruritus may respond to oatmeal baths and, if necessary, antihistamines. Infections, including *Staphylococcus aureus* (presenting as bullous impetigo, ecthyma, or folliculitis), *Pseudomonas aeruginosa* (which may present with chronic ulcerations and macerations), herpes simplex, herpes zoster, syphilis, and scabies are common and should be treated with standard therapies.

Other dermatologic conditions that occur with increased frequency in HIV-infected patients include seborrheic dermatitis, psoriasis, atopic dermatitis, and alopecia. Dermatologic consultation generally is indicated. Admission may be indicated for patients with any disseminated cutaneous infection requiring IV antibiotics or antiviral agents.

24. Describe ophthalmologic emergencies that occur in AIDS patients.

Eye complaints such as change in visual acuity, photophobia, redness, and pain are common and may represent retinitis or invasion of eye or periorbital tissues with a malignant or infectious process. Cytomegalovirus retinitis occurs in 30% of AIDS patients and accounts for most retinitis among AIDS patients. It has a characteristic appearance of fluffy white retinal lesions, often perivascular (sometimes referred to as "tomato and cheese pizza" appearance). Ophthalmology consultation is indicated, followed by treatment with foscarnet or ganciclovir for 2 weeks and long-term maintenance therapy.

25. Should HIV-infected patients receive tetanus and other immunizations?

According to the U.S. Public Health Service Immunizations Practices Advisory Committee, routine immunization recommendations for diphtheria (DPT); tetanus (Td); and measles, mumps, and rubella (MMR) are unchanged for HIV-infected patients. Smallpox and polio vaccines are not recommended in the HIV-infected population.

26. How should symptoms of side effects from drugs be managed?

Reactions to pharmacologic therapy are common in HIV-infected patients and always must be considered as the cause of new symptoms. In one study, 30% of hospitalized patients with HIV disease had an identified probable or definite adverse drug reaction. The most common type of reaction was cutaneous. Certain commonly used pharmaceutical agents cause a particularly high incidence of adverse drug reactions, including TMP-SMX, which has a 65% incidence of adverse drug reactions in AIDS patients, and pentamidine, which has a 50% incidence of adverse reactions. A decision about discontinuing therapy depends on balance between the benefit of the drug and the severity of side effects.

27. How can health care providers protect themselves from acquiring HIV?

Health care workers often are exposed to HIV-infected patients and their body fluids. Precautions in handling potentially infectious fluids are crucial. Because HIV infection is often undiagnosed at the time of the ED encounter, the use of universal precautions is imperative and should be performed without exception, including the appropriate use of gown, gloves, mask, and goggles for procedures performed in all patients. The Needlestick Safety and Prevention Act of 2000 mandates that safety-engineered devices be used whenever possible and that institutions maintain exposure control plans. With the use of universal precautions, the risk of acquiring HIV infection by occupational exposure is extremely low.

28. What constitutes high-risk exposure to HIV?

- Substantial risk from **nonoccupational exposures** are those from an HIV-infected source with blood, semen, vaginal or rectal secretions, breast milk, or any body fluids with visible blood, through the vagina, rectum, eye, mouth, or other mucous membrane, nonintact skin, or percutaneous contact.
- For **occupational exposures**, higher-risk percutaneous exposures associated with an increased likelihood of transmission include deep injuries, visible blood on a device, and

injuries sustained when placing a catheter in a vein or artery. Percutaneous exposures that are superficial or involve solid needles are considered lower-risk exposures. High-risk sources are patients with symptomatic HIV, AIDS, acute seroconversion, or high viral load. Patients with asymptomatic HIV or viral load <15,000 copies/mL are considered lower risk.

29. Should postexposure prophylaxis (PEP) be administered after exposure to blood and body fluids?

PEP should be considered following all occupational and nonoccupational exposures. Decisions to treat should be based on the type of exposure, the risk of HIV in the source patient, and careful consideration of the risks and benefits of therapy. PEP is most effective if administered within 30 minutes of the exposure. PEP may consist of a basic regimen (such as zidovudine plus lamivudine) or an expanded regimen for high-risk exposures (such as zidovudine, lamivudine plus either indinavir or nelfinavir). Ideally, each health care institution should have written protocols that are formulated in consultation with occupational medicine and infectious disease specialists for occupational exposures in health care workers and patients with nonoccupational exposures.

30. What is highly active antiretroviral therapy (HAART)?

HAART is recommended for HIV-infected patients with CD4 counts less than 200 cells/uL or those with symptomatic disease. The use of HAART has led to significant reductions in morbidity and mortality. HAART should be prescribed by infectious disease specialists and typically includes nonnucleoside reverse transcriptase inhibitors (NNRTIs) such as efavirenz, nucleotide reverse transcriptase inhibitors (NRTIs) such as zidovudine or lamivudine, and protease inhibitors such as lopinavir or ritonavir. Adverse reactions to HAART are common and may include bone marrow suppression, cutaneous reactions, GI distress, jaundice, nephrolithiasis, abnormal lipid profiles, neuropathy, and others.

KEY POINTS: SEXUALLY TRANSMITTED DISEASES

1. STDs affect 19 million people a year in the United States.
2. Single-dose therapy for Chlamydia and GC is effective for treatment of uncomplicated cervicitis and urethritis.
3. The most common causes of genital ulcers include herpes simplex, chancroid (*H. ducreyi*), syphilis, and HPV.
4. Patients with suspected *Pneumocystis* pneumonia should be treated with TMP-SMX (weight-based) and corticosteroids (if hypoxic).
5. All patients with high-risk HIV exposure should be considered for postexposure prophylaxis therapy.

WEBSITES

www.cdc.gov/std/hpv/STDFact-HPV-vaccine-young-women.htm

www.cdc.gov/std/treatment

www.ucsf.edu/hivcntr/PEPline/index.html

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CHAPTER 50

TOXIC SHOCK SYNDROME

Joshua B. Gaither, MD

1. What is toxic shock syndrome (TSS)?

TSS is characterized by a rapidly progressing constellation of symptoms, caused by one of several different bacterial exotoxins that act as a superantigen to stimulate an excessive immune response. The syndrome was first described in seven children presenting with high fever, headache, confusion, conjunctival hyperemia, and gastrointestinal symptoms, which were accompanied by a scarlatiniform rash and severe shock. All seven cases were linked to *Staphylococcus aureus*, which was cultured from infected or mucosal sites, prompting fears that the bacterium was expressing a newly discovered toxin. Over the past 30 years the same toxin-mediated syndrome has been described in association with other bacterial infections, including community acquired methicillin-resistant *Staphylococcus aureus* (MRSA), which produces the same toxin, as well as streptococcus infections and clostridial infections, each of which cause TSS-like symptoms through the production of different endotoxins.

2. Why did a bacterium begin to express a new and deadly exotoxin in the late 1970s?

Todd's report led to additional associations between TSS and *S. aureus.* In retrospect, the syndrome may have been described as early as 1927. Some researchers believe that the syndrome may have been responsible for the plague that ended the Golden Age of Athens.

KEY POINTS: RISK FACTORS FOR TSS

1. Air-containing foreign bodies (e.g., tampon, nasal packing)
2. Recent surgery
3. Postpartum
4. Burns
5. Focal infections

3. Who gets TSS?

Menses was associated with 91% of cases reported by 1980, which quickly pointed to the use of new high-absorbency tampons as a risk factor. Such tampons, made with cross-linked carboxymethylcellulose and polyester foam, were thought to provide an ideal environment for the expression of TSS toxin and subsequently were removed from the market. With decreased use of these tampons and increased awareness of TSS, more recent epidemiological surveys have found that less than half of all staphylococcal TSS cases are associated with menses.

4. Is tampon use required for the patient to develop TSS?

No. Three of the patients identified in the 1978 report were male. Although the media focused on the association with high-absorbency tampons, clinical interest in the syndrome identified a wide

variety of causes in the early 1980s. TSS has been reported in all age groups, in burn and postsurgical patients, after childbirth, in association with the nasal packing commonly used to control epistaxis, and in many types of local infections. Recent data have suggested that sinusitis may be responsible for up to 21% of TSS in the pediatric population.

5. Describe the pathophysiology of TSS.

Three stages have been identified in the progression of the syndrome:

- Local proliferation of the toxin-producing strain of bacteria
- Toxin production
- Immune response to the toxin, which sets off the inflammatory cascade and leads to multisystem organ involvement

Although many different bacteria from a wide variety of sources have been reported to cause TSS, the common link between infection and TSS is the production of a superantigen, which stimulates massive cytokine release and a systemic inflammatory response leading to shock.

6. There was heavy media coverage of TSS in the early 1980s because of the high case fatality rate. Has mortality been reduced?

The initial fatality rate was 13% for the first 55 cases, with white females in the 15- to 19-year-old range thought to be at greatest risk. Further analysis of early cases suggests that significant reporting bias was a factor in the high fatality rate, which is now thought to be stable at 5%. However, there have been few recent epidemiologic studies in the United States. A more recent French study suggested that less than half of reported staphylococcal TSS cases were menstrual and that that subset of patients had a 0% mortality rate, while nonmenstural TSS accounted for 62% of cases and was associated with a 22% mortality rate.

KEY POINTS: COMMON CHARACTERISTICS OF TSS

1. Fever
2. Rash
3. Hypotension—shock
5. History of air-containing foreign body, abscess, local infection, or recent surgery or childbirth
6. Multisystem organ involvement

7. List the criteria for defining a case of *S. aureus* TSS.

- Fever $\geq$ 38.9°C
- Diffuse macular erythematous rash
- Desquamation, usually of the palms or soles, after 1 to 2 weeks
- Orthostasis or hypotension (with systolic blood pressure $<$90 mm Hg in adults or less than the sixth percentile in children)
- Involvement of three or more of the following organ systems:
- Gastrointestinal: vomiting or diarrhea
- Muscular: myalgias or elevated creatine phosphokinase (twice normal)
- Mucous membrane: vaginal, oropharyngeal, or conjunctival hyperemia
- Renal: elevated blood urea nitrogen or creatinine (twice normal) or pyuria in the absence of urinary tract infection
- Hepatic: total bilirubin, alanine aminotransferase, aspartate aminotransferase levels at least twice the upper limit of normal
- Hematologic: platelets $<$100,000/mm^3
- Central nervous system: disorientation or alteration in consciousness without focal neurologic signs (when fever and hypotension are absent)

- Negative results for the following, if obtained:
  - Blood, throat, or cerebrospinal fluid cultures (blood cultures may be positive for *S. aureus)*
  - Rise in titer to Rocky Mountain spotted fever, leptospirosis, or rubeola

8. What is *Streptococcal* TSS?

Several different groups of streptococcus (most commonly *Streptococcus pyogenes*) can cause a severe systemic reaction similar to TSS. Their toxin is similar to that of TSS. Diagnosis requires the isolation of streptococci from a sterile or nonsterile site, hypotension, and multisystem organ involvement (at least two or more of the following: renal impairment, coagulopathy causing disseminated intravascular coagulopathy or thrombocytopenia, hepatitis, adult respiratory distress syndrome, necrotizing soft-tissue infections, or skin changes similar to those seen in TSS).

9. Describe the rash associated with TSS.

The rash is a macular erythroderma that blanches and is not pruritic. It may be diffuse or localized and often is described as sunburn-like. It appears early in the illness and fades in about 3 days. It may be subtle and can be missed in dark-skinned patients.

10. When is desquamation likely to occur?

Loss of skin, usually of the distal extremities, invariably occurs in survivors 5 to 12 days after the illness starts. Delayed alopecia and fingernail loss may occur later and seem to depend on the level of hypotension during the acute illness.

11. Given the previously mentioned criteria for TSS, list the differential diagnosis.

- Kawasaki disease
- Staphylococcal scalded skin syndrome
- Streptococcal scarlet fever
- Rocky Mountain spotted fever
- Leptospirosis
- Stevens-Johnson syndrome
- Erythema multiforme
- Toxic epidermal necrolysis
- Sepsis
- Drug reactions
- Measles
- Colorado tick fever

12. Summarize the treatment for TSS.

- Supportive care including intravenous fluids for hypotension, with supplemental vasopressor support as needed
- Identification and removal of the source of infection (e.g., tampon, abscess, nasal packing)
- Antibiotics

13. Do antibiotics help?

No prospective studies show that antibiotics alter the severity or the course of TSS. However, antibiotics reduce the recurrence rate (which can be 28%) and a delay in antibiotic administration has been associated with increased mortality in other types of severe sepsis and septic shock. Therefore early administration of antibiotics is considered the standard care.

14. What antibiotics should I use?

Vancomycin and clindamycin are considered the empiric antibiotics of choice. Clindamycin has the added advantage of a direct antitoxin effect. When a source organism can be identified, additional targeted antibiotic coverage is appropriate. For non-MRSA Staphylococcal TSS, a penicillinase-resistant penicillin such as Nafcillin should be used, whereas for Streptococcal TSS high-dose penicillin should be given.

15. Are there other therapies that can help control the immune response to the toxin?

Intravenous immunoglobulin (IVIG) may decrease mortality rates by up to 3.6 times that of patients receiving standard therapy; however, data on IVIG have been limited due to the low incidence of TSS and a clear benefit has not been established. Most authorities currently

recommend the use of IVIG for severe cases in which prompt antibiotic and source control fail to result in clinical improvement. Theoretically, **steroids** should help attenuate the systemic response to the toxin, but there are no prospective data to show they are effective. Steroid use in sepsis is still debated, and steroids are not routinely used in the management of TSS.

16. Do all patients with TSS need admission?

Patients in whom TSS is suspected should be admitted because this toxin-mediated disease can progress rapidly. In most patients, the systemic signs of illness (e.g., hypotension, fever, and multisystem organ involvement) are present in the ED, clearly indicating the need for inpatient supportive care.

KEY POINTS: TREATMENT FOR TSS

1. IV fluids with pressor support when needed
2. Removal of infectious source
3. Antibiotics
4. Consider IVIG in severe cases refractory to traditional therapy

WEBSITES

CDC website: www.cdc.gov/ncidod/dbmd/diseaseinfo/toxicshock_t.htm

http://emedicine.medscape.com/article/169177-overview

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CHAPTER 51

TETANUS, BOTULISM, AND FOOD POISONING

John E. Houghland, MD

TETANUS

1. What is the causative agent of tetanus and its mechanism of action?

Clostridium tetani is an obligate anaerobic bacteria whose spores produce two distinct toxins—tetanolysin, which causes local tissue destruction, and tetanospasmin, which causes clinical tetanus. Tetanospasmin travels in a retrograde fashion through peripheral nerves to the central nervous system (CNS) where it crosses to presynaptic neurons and disables inhibitory neurotransmitter release (γ-aminobutyric acid [GABA] and glycine) in both the autonomic and somatic nervous systems. Spores are extremely resilient, able to survive household disinfectants, extremes in temperatures and humidity, and for several minutes in boiling water.

2. What are the forms of tetanus?

- Generalized tetanus (>80% of cases) involves rigidity and spasm of all muscles in the body, usually starting cranially and proceeding caudally.
- Localized tetanus is seen with lower toxin loads and peripheral injuries; spasm, rigidity, and pain are limited to the injured body area.
- Cephalic tetanus occurs after a head wound and presents as cranial nerve paralysis (most commonly lower motor neuron weakness of cranial nerve VII), and frequently proceeds to generalized tetanus.
- Tetanus neonatorum is the most common cause of tetanus worldwide and is caused by poor umbilical hygiene. It is rare in developed countries and is preventable by vaccination.

3. How is tetanus contracted?

Tetanus generally originates from a deep wound that facilitates anaerobic bacterial growth and is usually grossly contaminated with soil, manure, or metal. Other sources include burns, ulcers, snakebites, middle ear infections, tattooing, piercings, septic abortions, childbirth, surgery, and intramuscular injections. The low pH of quinine, a common diluting agent in heroin, may cause more aggressive cases of tetanus by facilitating cellular entry of the toxin. A prior episode of tetanus is not protective and does not confer lifelong immunity.

4. What are the presentation and prognosis of neonatal tetanus?

Neonatal tetanus presents commonly during the first week of life in infants of nonimmunized mothers. The bacteria enter through the umbilical cord stump, especially after the application of mud or feces, a practice in some developing countries. General irritability and poor feeding progress to generalized spasms, pneumonia, and pulmonary or CNS hemorrhage. Toxin load is high; mortality is variable (10%–100%).

5. What is the presentation of generalized tetanus?

Initial symptoms include masseter and parapharyngeal spasm, causing trismus. Facial and neck muscle spasms lead to dysphagia, neck pain, and *risus sardonicus* (the ironic smile of tetanus). Muscle spasms proceed caudally to the paraspinous and abdominal wall muscles.

Opisthotonos may compromise respiratory function and cause vertebral fractures. Minor stimuli (e.g., light touch, drafts, or noises), pain, and anxiety may trigger severe spasms. Death can result from glottis spasm, respiratory failure, and autonomic instability (e.g., labile hypertension, dysrhythmias, hyperpyrexia, tachycardia, or myocardial infarction); the latter of which may present late and carries a 40% mortality rate.

6. How do I treat generalized tetanus in the ED?

Initial management involves injection of tetanus immunoglobulin 500 to 6000 IU intramuscularly at the site of the wound, given prior to surgical debridement of devitalized tissue to prevent the release of preformed toxin. Metronidazole 500 mg by mouth (PO) or intravenously (IV) every 6 hours is the antibiotic of choice. Penicillin G, formerly the treatment of choice, has less penetration into devitalized tissue and abscesses and carries the theoretical harmful effect of lowering seizure threshold due to GABA antagonism. Other alternatives include doxycycline, erythromycin, and tetracycline.

7. Where should I admit patients with tetanus?

Patients with tetanus should be admitted to an intensive care unit (ICU) setting, with a dark and quiet environment to minimize external stimuli.

8. How do I vaccinate someone against tetanus?

Previously unimmunized patients require a primary series: three doses, with the second dose given 4 to 8 weeks after the first, and the third dose given 6 to 12 months later. Following the primary vaccine series, individuals should receive a booster every 10 years, or after incurring a tetanus-prone wound if their last vaccination occurred more than 5 years ago. Tetanus-prone wounds include those with devitalized tissue or gross contamination and wounds from crush injuries. The Td formulation, which contains a lower dose of the diphtheria component, should be used in all individuals older than age 7 years. Consider giving tetanus immunoglobulin in addition to tetanus vaccine to those with grossly contaminated wounds who lack a primary vaccination series.

9. What is the time course of tetanus?

The incubation period after exposure averages 7 to 10 days (range 1–60 days). The first week of illness is characterized by muscle rigidity and spasm, followed by autonomic disturbances that last for 1 to 2 weeks. Muscle spasms generally subside after 2 to 3 weeks, but patients may experience persistent stiffness.

10. What are the side effects of tetanus vaccine?

Side effects are generally limited to local reactions including erythema, induration, tenderness, nodule, or sterile abscess at the site of infection. Mild systemic reactions can occur and include fever, drowsiness, “fretfulness,” and anorexia, but all are self-limited.

11. Is tetanus vaccine safe for pregnant and immunocompromised patients?

Tetanus vaccine is a toxoid (inactivated toxin) that is safe and effective in pregnancy and can help prevent neonatal tetanus. Likewise, it is safe for administration in immunocompromised patients.

KEY POINTS: TETANUS

1. Tetanus vaccine is safe for pregnant and immunocompromised patients.
2. Metronidazole is the antibiotic of choice.
3. High mortality rate is due to autonomic instability.

BOTULISM

12. What is the causative agent of botulism? How does it cause disease?

Botulism is caused by the toxin produced by *Clostridium botulinum*. This toxin binds to peripheral presynaptic nerves cholinergic membranes, preventing the release of acetylcholine and producing a life-threatening, paralytic illness. Adrenergic synapses are unaffected. By weight, botulism toxin is the most potent toxin known.

13. State the five ways a patient can contract botulism.

- Adult botulism (10%–20% of U.S. cases) results from the foodborne ingestion of preformed toxin. Undercooked home-canned food is a common source; cases occur in isolation or in small clusters.
- Infant botulism (approximately 60% of U.S. cases) is caused by the ingestion of *C. botulinum* spores, which proliferate in the gastrointestinal (GI) tract. It is usually seen between the ages of 2 weeks and 1 year, with a median age of 10 weeks. Contaminated raw honey ingestion accounts for only up to 20% of cases. It is believed that changes in gut flora can also lead to *C. botulinum* colonization.
- Wound botulism (approximately 15%–25% of U.S. cases) is caused by the contamination of traumatic wounds with *C. botulinum* spores found in soil. Rare in surgical wounds, wound botulism is increasing dramatically in the United States. It is seen almost exclusively in injection drug users, particularly among users of *black tar heroin* and those who partake in *skin popping* (injection into subcutaneous tissue).
- Hidden botulism is an idiopathic form, in which the patient's stool contains *C. botulinum*, and the patient has signs and symptoms of clinical botulism, yet no contaminated food or wound can be identified.
- Iatrogenic botulism is a complication of cosmetic or therapeutic injections. Although cosmetic doses are too low to cause systemic disease, cases have been reported in patients receiving higher doses for cosmetic purposes or neuromuscular disorders. Such patients may present with moderate to severe clinical weakness. Focal neurologic deficits can be seen by craniofacial migration of injected toxin.

14. What are the differential diagnoses of botulism?

Myasthenia gravis, Lambert-Eaton myasthenic syndrome, Guillain-Barré syndrome, tick paralysis, diphtheritic neuropathy, stroke syndromes, tetrodotoxin and shellfish poisoning, and Miller-Fischer variant of Guillain-Barré syndrome.

15. What is the presentation of infant botulism?

Constipation is often the first presenting symptom. This can be followed by a weak cry, prolonged or poor feeding, hypotonia, and decreased gag or suck reflex. As in adults, infants can develop descending motor weakness, flaccid paralysis, and autonomic dysfunction. Unlike tetanus toxin, botulinum toxin acts peripherally and does not cross the blood-brain barrier; fever is uncommon and cerebrospinal fluid (CSF) analysis is normal.

16. How does a patient with adult botulism present?

Early symptoms are nonspecific and usually begin 12 to 36 hours after ingestion (range 6 hours to 8 days) and include nausea, vomiting, weakness, malaise, and dizziness. Prominent anticholinergic symptoms ensue, including extreme dry mouth, decreased lacrimation, constipation, and urinary retention. Symmetric cranial nerve palsies occur, sometimes delayed 3 days after the appearance of anticholinergic symptoms. Ocular and bulbar symptoms include ptosis, diplopia, blurred vision, photophobia, dysphonia, and dysphagia. A flaccid, symmetric, descending paralysis of the voluntary muscles may follow and can lead to respiratory failure.

17. What is the treatment of classic (food) botulism?

Treatment is mostly supportive, including early elective intubation of patients at risk of respiratory failure. Type-specific equine-derived antitoxin is also recommended within

24 hours to arrest the progression of and decrease the duration of paralysis. Anaphylaxis, more common when higher doses of antitoxin were used, is now a rare adverse event (<1%) when the recommended one vial is used.

18. What is treatment for infant botulism?

Treatment of infant botulism includes supportive care, including intubation and mechanical ventilation for respiratory failure. Human botulism immunoglobulin (Baby-BIG) is recommended, which reduces duration of hospitalization, mechanical ventilation, and tube feedings. Equine-source antitoxin is not indicated for infant botulism.

19. Are systemic antibiotics indicated for infant botulism?

No. Aminoglycosides are absolutely contraindicated because they may potentiate neuromuscular blockade and increase duration of symptoms. Antibiotic administration may cause bacterial lysis in the gut and theoretically increase the free toxin load.

KEY POINTS: BOTULISM

1. Adult botulism presents as nonspecific anticholinergic symptoms followed by symmetric cranial nerve palsies and descending paralysis.
2. Infant botulism usually presents as constipation followed by weak cry, prolonged or poor feeding, hypotonia, and decreased gag or suck reflex.
3. Human botulism immunoglobulin (Baby BIG) is available for infant botulism.

20. Are antibiotics indicated in wound botulism?

In addition to surgical debridement, topical antibiotics may be of use in wound botulism, but their use is unproved.

FOOD POISONING

21. Name the causes of food poisoning.

Food poisoning is caused by viruses (e.g., Norwalk, Calicivirus, or Rotavirus), direct bacterial invasion or endotoxins (e.g., *Escherichia coli, Vibrio vulnificus, Vibrio parahaemolyticus, Campylobacter, Salmonella,* or *Yersinia enterocolitica*), secreted exotoxins (e.g., *Staphylococcus aureus, Shigella*, *Bacillus cereus, Clostridium perfringens,* or shellfish-associated algal toxins), toxins innate in food (e.g., Puffer fish tetrodotoxin or *Amanita phalloides* mushrooms), and parasites (e.g., *Giardia lamblia, Cryptosporidium,* or *Entamoeba histolytica*).

22. What is scombroid and how is it treated?

Scombroid fish poisoning is caused by ingestion of fish with high levels of histamine. Multiple fishes in addition to the scombroid family (e.g., tuna, mackerel, or saury) have been implicated (e.g., mahi-mahi, sardines, pilchards, anchovies, herring, marlin, and bluefish). Improperly refrigerated fish becomes spoiled with bacteria that proliferate and convert naturally occurring histidine to histamine, which persists despite cooking. Poisoning is associated with high levels of histamine (>50 mg/100 g contaminated fish). Affected fish typically retain normal appearance and odor. Not a true fish *allergy*, poisoning causes histaminergic symptoms approximately 20 to 30 minutes after ingestion, typically an urticarial rash of the face, neck, and upper chest. Other symptoms include flushing, nausea, vomiting, diarrhea, headache, palpitations, abdominal cramping, dizziness, dry mouth, metallic taste in the mouth, urticaria, and conjunctival injection. Severe hypotension resembling anaphylactic

shock and requiring pressor support, as well as acute pulmonary edema, can occur. Treatment with H1 and H2 antagonists is generally effective.

23. Describe the toxic syndromes associated with ingestion of shellfish.

Algal toxins are produced by numerous species of marine algae that contaminate shellfish, crustaceans, and some fish. Diagnosis is based on history of recent ingestion and clinical picture; treatment is essentially supportive. Syndromes include:

- **Amnestic shellfish poisoning (ASP)** can present with nausea, vomiting, dizziness, headache, confusion, respiratory difficulty, and coma with loss of short-term memory that may be permanent. The causative agent is domoic acid, a preformed agent with neuroexcitatory glutaminergic activity, found primarily in infected scallops, mussels, and crab. Onset is within 24 hours of ingestion.
- **Diarrhetic shellfish poisoning (DSP)** is caused by okadaic acid found in affected mussels, cockles, scallops, oysters, cockles, whelks, and green crabs. Symptoms are self-limited, characterized by acute onset within 30 minutes of severe diarrhea, nausea, vomiting, and abdominal cramps. Recovery generally occurs within 3 to 4 days.
- **Paralytic shellfish poisoning (PSP)** is caused by saxitotoxin in affected mussels, clams, oysters, scallops, abalone, crabs, and lobster, which blocks sodium channels of nerve and muscle cell membranes. Initial perioral paresthesias spread to the face, head, and neck within 30 minutes of ingestion; large ingestion may lead to respiratory arrest and death within 2 hours.
- **Ciguatera poisoning** occurs after the ingestion of coral reef fish that contain ciguatoxin, which stimulates sodium channels at the neuromuscular junction. Neurologic symptoms occur in >90% of patients, including reversal of temperature perception (cold objects cause burning or electric shock-like sensation), facial and perioral paresthesias, coma, and death (overall mortality approximately 0.1%). Non-neurologic symptoms (e.g., hair loss, arthralgias, myalgias, itching, vomiting, diarrhea, or insomnia) resolve within a few days; however, severe neurologic symptoms may persist for weeks or years.
- **Neurotoxic shellfish poisoning (NSP)** is caused by the brevetoxin family of toxins, commonly found in cockles, mussels, and whelks off the coast of Florida and the Gulf of Mexico. Symptoms begin 3 to 6 hours after ingestion, last up to 48 hours, and can include perioral paresthesias, abdominal pain, dizziness, diplopia, gait deficits, chills, reversed temperature perception, headache, musculoskeletal pain, and respiratory difficulty.

24. Describe the clinical course and treatment for Puffer fish poisoning.

Puffer fish poisoning results from the consumption of tetrodotoxin in improperly prepared Puffer fish, commonly found in Japan, Singapore, Hong Kong, and Australia. Tetrodotoxin is resistant to cooking and is produced in the viscera and skin of the fish; safe consumption is predicated on expert chefs removing these areas. Tetrodotoxin blocks sodium channels in the central and peripheral (including autonomic) nervous systems and interferes with axonal nerve transmission in muscle. Symptoms begin with perioral paresthesias, which can spread to the entire body, as well as vomiting and dizziness; most patients develop a rapid ascending paralysis. Tetrodotoxin has direct effects on the respiratory and vasomotor centers in the medulla oblongata. Gastric lavage and activated charcoal have been recommended for large ingestions because they can result in respiratory failure, cardiovascular collapse, coma, and death within 6 hours.

25. Which population of patients is at risk from eating raw oysters?

Patients with pre-existing liver diseases, including cirrhosis and hemochromatosis, have an 80 times higher risk of invasive *Vibrio* disease and 200 times higher risk of mortality than those without liver disease. Consumption of raw oysters, especially from warmer waters between March and November, has a high incidence of *V. vulnificus* and *V. parahaemolyticus*.

26. Describe the four stages of *Amanita phalloides* mushroom toxidrome.

- **Stage 1**: Latent stage: patient remains asymptomatic for 8 to 14 hours post-ingestion.
- **Stage 2**: Violent onset of nausea, vomiting, and diarrhea (often bloody), and severe abdominal pain lasting 1 to 2 days. This stage may include acid-base disturbances, electrolyte abnormalities, hypoglycemia, dehydration, and even hypotension. Physical examination may be significant for epigastric tenderness and hepatomegaly with normal liver function tests.
- **Stage 3**: The patient clinically appears to improve over the next 12 to 24 hours, although liver function tests begin to rise and renal function begins to deteriorate.
- **Stage 4**: Beginning 2 to 4 days post-ingestion, the patient develops hepatic and renal failure, with marked rise in liver function tests, cardiomyopathy, hepatic encephalopathy, convulsions, coma, and death.

KEY POINTS: FOOD POISONING

1. *Amanita phalloides* toxic patients may appear to be improving before rapid deterioration.
2. Antibiotics are not recommended in children with bloody diarrhea.
3. Patients with liver disease are at highest risk for *Vibrio* disease from eating raw oysters.

27. What is the time course and geographic incidence of traveler's diarrhea?

Traveler's diarrhea is one of the most common illnesses of international travelers, affecting 20% to 60% of travelers. High risk destinations include Latin America, Africa, the Middle East, and Asia (20%–50% prevalence). Southern Europe, China, Russia, and the Caribbean are lower risk (up to 15% prevalence). Median time to onset is 6 to 7 days after arrival, although it may occur at any time, and up to 10 days after travel return. It is usually self-limited, lasting 3 to 4 days; however, 10% of patients may have more than 1 week of symptoms, and 3% may have more than 1 month of symptoms. Approximately 20% of individuals will have symptoms of dysentery (e.g., bloody stool, fever).

28. What are some of the more serious complications of traveler's diarrhea? What are the causative agents?

- Postinfectious inflammatory syndromes (e.g., Reiter's syndrome [arthritis], urethritis, or conjunctivitis): *Campylobacter jejuni, Salmonella, Shigella, Y. enterocolitica*
- Guillain-Barré syndrome: *C. jejuni*, especially with Human Leukocyte Antigen (HLA)-B27
- Hemolytic uremic syndrome (HUS): *Shigella dysenteriae* and enterohemorrhagic *E. coli*
- Amebic hepatitis and amebic abscesses: *E. histolytica*
- Bacteremia leading to endocarditis, aortitis, septic arthritis, osteomyelitis: *Salmonella*

29. Is there any role for prophylaxis against traveler's diarrhea?

Patients at high risk for complications, including those with inflammatory bowel disease, insulin-dependent diabetes, heart disease, or immunosuppression, may benefit from prophylaxis. Fluoroquinolones have been shown to have a more than 90% efficacy in preventing traveler's diarrhea. Ciprofloxacin 500 mg PO once daily can be started 2 days prior to arrival and taken 1 week after return, up to 3 weeks total. Patients who are otherwise healthy may wish to consider bismuth subsalicylate (Pepto-Bismol, taken as 524 mg four times a day) as a nonantibacterial option that has fewer side effects but lower efficacy (approximately 65%). *Campylobacter* strains are often resistant to fluoroquinolones; azithromycin should be considered for prophylaxis in higher risk areas such as South Asia and Southeast Asia (especially Thailand) (Fig. 51-1).

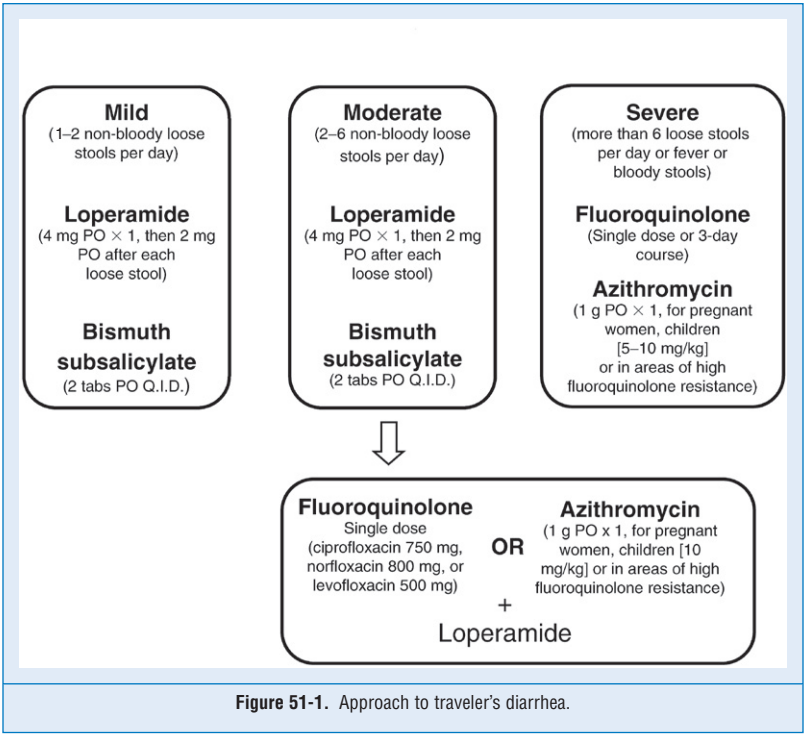

Figure 51-1. Approach to traveler's diarrhea.

30. Should antibiotics be used for infectious diarrhea?

The use of antibiotics in acute diarrhea remains controversial. Short courses or single-dose regimens of broad-spectrum antibiotics (e.g., ciprofloxacin 750 mg, norfloxacin 800 mg, or levofloxacin 500 mg as single doses) have been shown to reduce the frequency of stool by 50% and duration of illness to 12 to 24 hours. Azithromycin (1,000 mg PO as a single dose, or weight-based equivalent) can be considered in pregnant women and children, for whom fluoroquinolones are contraindicated. In areas where amebiasis and giardiasis are prevalent, consider metronidazole 500 mg three times a day for 5 days. Antibiotic therapy is not generally recommended in children with bloody diarrhea because of the association with enterohemorrhagic *E. coli* (O157.H7) infections, in which antibiotics do not significantly improve outcome and can promote the development of HUS from increased lysis of organisms with release of endotoxin and Shiga-like toxin.

31. Which diarrhea-producing agent is associated with febrile seizures in children?

Shigella infections in young children can present with high fevers, generalized toxic appearance, abdominal cramps, bloody mucoid stool, and seizures with or without encephalopathy. Other complications include dehydration, hyponatremia, hypoglycemia, and surgical emergencies (e.g., toxic megacolon, rectal prolapse, or intestinal perforation). Endemic *Shigella* is responsible for 75% of diarrhea-related deaths in developing countries.

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CHAPTER 52

MOSQUITO- AND TICK-BORNE DISEASES OF NORTH AMERICA

Andrew M. Kestler, MD, MBA, DTMH

1. Why should I read this chapter?

Mosquitoes are the most important disease vectors worldwide, and ticks are the most important arthropod vectors in North America. West Nile virus and the Lyme disease spirochete are common endemic pathogens within the continental United States and Canada. In the case of rarer diseases, such as Rocky Mountain spotted fever (RMSF) and tularemia, early recognition and treatment can be life-saving. Other diseases, such as dengue fever and malaria, frequently occur in travelers and recent immigrants from the tropics who present to EDs across North America.

2. What are the most important tools in diagnosing these vector-borne diseases?

A detailed history and a skin examination. Many of these illnesses initially present with a nonspecific syndrome of fever, headache, and myalgia. A history of travel, exposure to the vector, or a characteristic rash can provide the key to a difficult diagnosis.

3. Does malaria occur in North America?

Most definitely. The Centers for Disease Control and Prevention (CDC) was established in Atlanta in 1946 for the specific purpose of controlling malaria in the southeastern United States. Malaria remains endemic in the Caribbean and parts of Mexico. The CDC registered 1,505 cases of malaria in the United States for 2007. *Anopheles* mosquito species, the night-biting vector of malaria, are still widely found across North America and have caused transmission in the United States from one household member to another.

4. What causes malaria?

There are four species of the protozoan *Plasmodium: Plasmodium falciparum, Plasmodium vivax, Plasmodium ovale*, and *Plasmodium malariae*. Malaria is usually acquired by the bite of an infected female *Anopheles*, but it also may be transmitted by transfusion of infected blood or from mother to child in utero. *P. falciparum*, the most common and the most life-threatening type of infection, causes virtually all of the 1 million annual malaria deaths worldwide.

5. Can malaria be prevented?

Although no one method of protection is 100% effective, proper use of prophylactic drugs, bed nets, insect repellent, and proper patient education would prevent most cases of traveler's malaria seen in the United States. Tropic-bound travelers should consult a local travel clinic and the CDC website to assess malaria risk and plan prophylaxis accordingly.

6. Describe the clinical presentation of malaria.

Symptoms develop 10 to 14 days after *P. falciparum* infection but may be latent for 1 year with *P. vivax*. Patients complain of flu-like symptoms, with fever, chills, headache, nausea, vomiting, abdominal pain, cough, and myalgias. Physical examination may reveal jaundice, hepatomegaly, or splenomegaly but is often normal. The presence of a rash or significant lymphadenopathy suggests another diagnosis or a coinfection. Anemia,

thrombocytopenia, and hemoglobinuria are common laboratory findings. Patients may develop renal failure, pulmonary edema, shock, disseminated intravascular coagulation, profound anemia, acidosis, and hypoglycemia. Cerebral malaria, the most common fatal manifestation of malaria in adults, presents with altered mental status, seizures, and coma.

7. How is malaria diagnosed?

A high index of suspicion is the key to an early clinical diagnosis and survival because early initiation of treatment is a time-critical action. Definitive diagnosis is established with light microscopy visualization of protozoa on blood smears. Thick blood smears are more sensitive, but thin blood smears are necessary for speciation and calculation of a parasitemia percentage. Repeat blood smears at least three times at 12-hour intervals, although false-negatives will still occur. Antigen rapid detection kits are useful in case of limited access to an experienced laboratory technician.

8. How is malaria treated?

Unless *P. falciparum* infection can be ruled out, prompt treatment should be initiated empirically and the patient admitted. Severe malaria is treated with intravenous quinine (quinidine in the United States), or with artesunate (investigational new drug through CDC in the United States). There are several oral options for treating uncomplicated malaria, including mefloquine, atovaquone-proguanil (Malarone), and artemether-lumefantrine (Coartem, licensed by the Food and Drug Administration in 2009). Those receiving intravenous medications should receive telemetry monitoring because quinine and quinidine cause dysrhythmogenic QT prolongation. The latter two drugs also require combination therapy with clindamycin or tetracyclines to avoid recurrence and resistance.

KEY POINTS: MALARIA

1. Malaria is a common and lethal disease of the returning traveler or recent immigrant.
2. Normal blood smears do not rule out the disease.
3. Begin early empirical treatment in a sick patient with the right travel history.
4. More people die from malaria than from any other bite- or sting-induced disease.

9. What is dengue and where does it occur?

Dengue is a mosquito-borne flavivirus endemic to tropical regions the world over. Only 41 U.S. cases were confirmed by the CDC in 1999 and 2000, but given that many symptoms are nonspecific and that the disease is not reportable on a national level, it is thought to be significantly underdiagnosed and under-reported. Of note, Florida, Texas, and Hawaii report autochthonous transmission, meaning that the disease was acquired within the state. Puerto Rico has a few thousand cases per year. The principal vector, *Aedes aegypti*, is found across the southeastern United States.

10. How do dengue fever and dengue hemorrhagic fever (DHF) present?

Many infections, especially in children, are asymptomatic or go unnoticed as a mild febrile illness. Classic dengue fever occurs after 5 to 6 days of incubation and lasts about 1 week, presenting with fever, retro-orbital headache, nausea, vomiting, arthralgias, and severe myalgias, earning it the nickname of “breakbone fever.” A confluent blanching, macular rash is characteristic but only occurs in 50% of cases. The illness provides immunity to one of the four virus serotypes, but reinfection with another serotype may lead to the more severe form,

DHF. DHF is characterized by capillary leakage and thrombocytopenia, manifesting with dermal and mucosal hemorrhage or pleural and peritoneal effusions. DHF can develop into dengue shock syndrome, which if untreated carries a mortality of more than 40%.

11. How do I diagnose and treat dengue fever?

The diagnosis is usually made with enzyme-linked immunosorbent assay (ELISA) serology, which is often negative during the acute illness. The ELISA assay may cross-react with antibodies to other arboviruses such as West Nile. Laboratory abnormalities include thrombocytopenia, which occurs frequently even in the absence of DHF, nonspecific elevation of liver enzymes, and hemoconcentration in the case of DHF. Treatment is supportive: fluids and analgesics for dengue fever and intensive care for DHF, which in experienced centers can reduce the mortality below 1%. Nonsteroidal anti-inflammatory drugs (NSAIDs) and aspirin are to be avoided because of platelet inhibition.

12. What is West Nile virus?

The West Nile virus is a flavivirus that is acquired through the bite of *Culex* mosquitoes. Previously endemic in Africa, it first was reported in the United States in an outbreak in New York City in 1999, where it was associated with a die-off of infected crows. Since that time, the disease has spread westward to the Pacific, with a U.S. peak incidence of 9,862 in 2003, decreasing to 720 cases in 2009.

13. What are the symptoms of West Nile infections?

Only one in five infections is symptomatic, and only 1 in 140 to 320 present with central nervous system involvement. Symptomatic infection with the West Nile virus occurs 3 to 15 days after exposure. Initial symptoms include fever, headache, weakness, nausea, vomiting, and rash. Those with central nervous system involvement present most frequently with an encephalitic syndrome, for example, altered mental status and/or seizures. Isolated aseptic meningitis also occurs. More rarely, patients suffer polio-type paralysis or Parkinsonian movement disorders. The elderly are at much higher risk for severe disease and death. Neuropsychiatric sequelae occur in more than 50% of those with severe disease.

14. How is West Nile encephalitis diagnosed and treated?

Diagnosis is made with ELISA antibody assays on serum or cerebrospinal fluid (CSF). Treatment is supportive. Young, nontoxic patients without central nervous system involvement may safely be discharged home.

15. Are ticks a significant vector of disease?

Yes. They are responsible for the greatest variety and number of cases of vector-borne human illness in North America. Tick-transmitted Lyme disease is the most common vector-borne illness in the United States, with more than 28,921 confirmed cases in 2008 and an additional 6,277 probable cases. There are two major families of ticks: the hard ticks and the soft ticks (*Ornithodoros*). Hard ticks transmit all tick-borne diseases, with the exception of relapsing fever. Ticks typically spread disease in the summer months, when ticks are actively seeking blood meals, and when most potential human hosts are engaged in outdoor activities.

16. List the principal vectors and distribution of tick-borne diseases.

See Table 52-1.

17. How is Lyme disease transmitted?

Ixodes scapularis ticks transmit Lyme disease in eastern and central North America, whereas *Ixodes pacificus* is the vector on the Pacific Coast. Tick nymphs pick up the *Borrelia* spirochetes from mice, then transmit the Lyme infections to people. Transmission of Lyme disease rarely occurs before 48 hours of attachment. Although white-tailed deer do not harbor the disease, they are the preferred hosts of the adult tick. New cases of acute Lyme disease peak between April and September, when nymphs are feeding.

TABLE 52–1. PRINCIPAL VECTORS AND DISTRIBUTION OF THE TICK-BORNE DISEASES

Disease	Vector	Pathogen	U.S. Distribution	First-Line Treatment
Babesiosis	*Ixodes scapularis*	*Babesia microti*	Northeast	Quinine plus clindamycin
Colorado tick fever	*Dermacentor andersoni*	*Coltivirus*	Western mountains	Supportive
Ehrlichiosis	*Amblyomma americanum*	*Ehrlichia chaffeensis*	Southeast, south-central	Doxycycline
Anaplasmosis	*Ixodes* species	*Anaplasma phagocytophila*	Same as Lyme	Doxycycline
Lyme disease	*Ixodes* species	*Borrelia burgdorferi*	Northeast, midwest, west	Doxycycline
Relapsing fever	*Ornithodoros* species	*Borrelia* species	Western mountains	Doxycycline
RMSF	*Dermacentor* species	*Rickettsia rickettsii*	Nationwide, mostly Southeast	Doxycycline
STARI	*A. americanum*	*Borrelia lonestari*	Southern United States	Doxycycline
Tick paralysis	Multiple	Toxins	Nationwide	Tick removal
Tularemia	*A. americanum* and *Dermacentor* species	*Francisella tularensis*	West, south-central	Streptomycin

RMSF, Rocky Mountain spotted fever; STARI, southern tick-associated rash illness.

18. Describe the three clinical stages of Lyme disease.

- **Early acute or localized infection.** The classic skin lesion, erythema migrans (EM), develops at the bite site within 3 to 32 days in 80% of infections. If rash develops at the bite site in less than 48 hours, it is a hypersensitivity reaction (or cellulitis) not Lyme. More than 50% of patients will not recall a tick bite. EM expands slowly as an erythematous macule to a size of at least 5 cm and often much larger. Central clearing is common; it is usually painless but at times pruritic. Many individuals will develop systemic flulike symptoms with fever, suggesting some degree of early dissemination. Untreated, EM will typically resolve spontaneously over 3 to 4 weeks.
- **Early disseminated disease.** This may occur in untreated individuals days to weeks after the tick bite. Most will have fever and adenopathy, and many will have multiple secondary skin lesions that are smaller than the initial EM. Lyme arthritis is the most common secondary Lyme manifestation in the United States, occurring in 70% of patients with untreated EM. This mono- or oligoarticular process most commonly produces effusions in the knee and other large joints. Neurologic manifestations include cranial neuritis,

such as unilateral or bilateral facial nerve palsy (Bell's), and aseptic meningitis. Radiculoneuritis, similar to postherpetic neuralgia, with burning and paresthesias also occurs. Carditis occurs in less than 10% of cases and presents most typically with atrioventricular blocks.

- **Late or chronic disease.** Late manifestations occur months to years after infection. Lyme arthritis is the most common, presenting in 10% of those with untreated EM. The arthritis typically does not cause joint destruction and may resolve spontaneously after several years. Chronic neurologic disease may include polyneuritis, multiple-sclerosis-like encephalomyelitis (0.1%), and subtle encephalopathy. Chronic dermatitis (acrodermatitis chronicum atrophicans) and keratitis are relatively rare in the United States.

19. Does Lyme disease remind you of another spirochetal disease?

Primary, secondary, and tertiary phases, and the organ systems involved, are reminiscent of syphilis.

20. How is Lyme disease diagnosed?

The typical EM rash in an endemic area is sufficient for diagnosis. For disseminated disease, ELISA serology is used to detect Lyme antibodies. Suggestive clinical findings are key because positive serology does not prove active infection in an endemic area. A positive test in an asymptomatic individual is not an indication for treatment.

21. How is Lyme disease treated?

Oral therapy with doxycycline or amoxicillin is effective for early disease and for mild early disseminated disease, such as arthritis. Neurologic and cardiac manifestations (with the exception of an isolated Bell's palsy) typically require parenteral therapy with ceftriaxone over 2 to 3 weeks, with a good prognosis. Admission to telemetry is recommended in Lyme carditis even for a first-degree atrioventricular block if the PR interval is greater than 300 ms, given the risk of progression. Temporary pacing may be needed for third-degree blocks. Late Lyme disease may not always respond to treatment.

22. Can Lyme disease be prevented?

The mainstays of prevention remain avoiding tick exposure (difficult for outdoor enthusiasts), preventing attachment with protective clothing, and removing ticks promptly if they attach (twice daily tick checks). The only vaccine was removed from the market in 2002.

23. An ED patient presents with a tick bite; should you treat prophylactically for Lyme disease?

Yes and no. If you practice in an endemic area, if you can identify the tick as *Ixodes scapularis*, and if it likely was attached for more than 48 hours (suggested by engorgement, or known exposure time), treatment with a single 200 mg dose of doxycycline is effective in preventing Lyme. If you cannot meet these three conditions, simply give your patient appropriate return precautions.

KEY POINTS: LYME DISEASE

1. It is the most common vector-borne disease in the United States.
2. A classic erythema migrans rash develops at site of tick bite.
3. Heart block, Bell's palsy, and arthritis develop in advanced disease.
4. Positive serology test is not diagnostic of infection.
5. Treatment is doxycycline, amoxicillin, or ceftriaxone.

24. What on earth is STARI and what can be done about it?

STARI stands for southern tick-associated rash illness because it causes EM just like Lyme disease. It is caused by a nonculturable spirochete, *Borrelia lonestari*, transmitted by the lone star tick, *Amblyomma americanum*. If a patient presents with EM in the southern United States, a Lyme nonendemic area, the diagnosis is likely to be STARI. Treat just as you would early localized Lyme disease.

25. What is relapsing fever?

Relapsing fever is caused by several *Borrelia* species transmitted by soft ticks. Most cases are linked to stays in rural, rodent-infested cabins, in the mountains of the western United States. Abrupt onset of flulike symptoms (i.e., fever, myalgias, headache, and vomiting) occurs 2 to 18 days after exposure. A generalized macular rash or a pruritic eschar at the bite site may develop. After 3 days of fever, symptoms resolve and then *relapse* on a weekly basis up to 10 times, with declining severity. Diagnosis is made by detection of spirochetes on stained thick and thin blood smears or by special culture. The very young and very old suffer increased morbidity and mortality. The disease responds well to doxycycline, penicillin, and erythromycin, but a Jarisch-Herxheimer reaction may occur (malaise and hypotension.)

26. What is RMSF?

RMSF is a life-threatening infection caused by *Rickettsia rickettsii* and transmitted by *Dermacentor*, or dog ticks. Its name derives from its original description in Montana and Idaho in the late 19th century and from the typical petechial rash occurring initially on the wrists and ankles. Currently, most cases are reported from the southeastern and south-central United States. The rash later involves the palms and soles, then spreads to the trunk, and often progresses into purpuric lesions. About 60% of infections will present with the classic triad of rash, fever, and tick exposure, although the rash is rarely present during the first 3 days of the illness. Abrupt onset fever, severe headache, and myalgias are the most common presenting symptoms, 5 to 7 days after the tick bite.

27. How dangerous is RMSF? What can be done about it?

Untreated, RMSF mortality hovers around 25%. In 2003, 1,091 cases were reported in the United States. The rickettsial pathogen induces a vasculitis that leads to end-organ dysfunction, including confusion, respiratory failure, and renal failure, and most typically kills by disseminated intravascular coagulation. Appropriate and timely antibiotics can reduce the mortality to below 5%, but delay is frequent because of the relatively late onset of the characteristic rash. Doxycycline remains the drug of choice, whereas chloramphenicol is recommended for younger children (younger than 8 years). Because antibody production lags behind clinical disease, serology is confirmatory rather than diagnostic. Consider early empiric treatment in the spring and summer in endemic regions.

28. What are ehrlichiosis and anaplasmosis?

Ehrlichiosis and anaplasmosis are tick-borne diseases caused by the rickettsia-like bacteria, *Ehrlichia chaffeensis*, which infects monocytes, and *Anaplasma phagocytophila* (formerly known as *Ehrlichia phagocytophila* or *Ehrlichia equi*), which infects neutrophils. Ehrlichiosis is transmitted by *A. americanum* in the southeastern and south-central United States, whereas anaplasmosis is transmitted by *Ixodes* ticks in a similar distribution to Lyme disease. Around 600 cases of each were reported to the CDC in 2006. Both diseases present with fever and flulike symptoms, progressing to respiratory and renal failure, and coma in severe cases. Rash may occur in ehrlichiosis but not in anaplasmosis. Both diseases preferentially attack older or immunocompromised adults, but anaplasmosis induces additional immunosuppression and has a greater case fatality rate.

29. How are ehrlichiosis and anaplasmosis diagnosed and treated?

High clinical suspicion is needed in endemic areas during the summer months. Thrombocytopenia, leukopenia, and mildly elevated liver enzymes, in the context of possible

tick exposure or bites, are highly suggestive and should prompt treatment in an ill patient. Microscopic examination of buffy coat or peripheral blood may reveal characteristic inclusion bodies in monocytes or neutrophils. Separate serologic and polymerase chain reaction (PCR) tests exist for both pathogens but are only useful for confirmation of diagnosis. First-line therapy consists of doxycycline. Chloramphenicol, fluoroquinolones, and rifampin have also been reported to be effective.

30. What is Colorado tick fever?

Colorado tick fever is caused by an RNA Coltivirus transmitted by *Dermacentor* ticks in the western United States. Patients present 3 to 6 days after a bite, with sudden fever, headache, myalgias, and photophobia. A transient petechial rash may occur. In about 50% of cases, symptoms resolve and then recur after 3 days. Prognosis is excellent, although complications such as encephalitis, meningitis, and pericarditis have been reported. Diagnosis is by serology, although many cases go undiagnosed. Treatment is supportive.

31. What is babesiosis?

Babesiosis is a malaria-like illness, caused by the *Babesia microti* protozoan. It is transmitted by *Ixodes* ticks in the northeastern United States but has also been acquired by transfusion. Like malaria, the protozoan infects red blood cells, causing fever, drenching sweats, myalgias, and headache. Though most disease is mild, life-threatening disease occurs in elderly and asplenic patients. Concurrent infection with Lyme disease occurs in 20%, causing a more severe illness. Diagnosis is made by serology or by detecting ring forms on stained thin or thick blood smears. Treatment typically consists of quinine and clindamycin in combination, although azithromycin and atovaquone together are also effective.

32. What is tularemia?

Tularemia is a rare disease (125 cases/year in the United States) caused by *Francisella tularensis*, a virulent gram-negative coccobacillus. Most cases occur in the south-central United States in hunters through tick bites or contact with infected tissue of rabbits or rodents. The more common ulceroglandular form manifests with an ulcer at the tick bite, painful regional adenopathy, fever, headache, and myalgia. The severe typhoidal form presents with abdominal pain, fever, and prostration without skin and lymphatic manifestations and carries an untreated mortality of 30% to 60% by septic shock. Transaminitis and patchy pulmonary infiltrates develop in severe disease.

33. How is tularemia diagnosed and treated?

Prompt diagnosis and life-saving treatment depend on a high index of suspicion because serology and culture results will not be acutely available. Streptomycin remains the drug of choice, but if unavailable, other aminoglycosides (gentamicin and tobramycin), tetracyclines, ciprofloxacin, and chloramphenicol are alternatives.

34. What is tick paralysis?

Tick paralysis is a syndrome caused by neurotoxins in the saliva of gravid females of many tick species. Young girls in western North America are at highest risk, especially after prolonged tick attachment. The syndrome usually presents as an ascending paralysis similar to Guillain-Barré, with sparing of sensorium and sensory function. Mortality does occur as a result of respiratory failure. Tick removal usually brings about prompt and complete recovery.

35. What is the proper method for tick removal?

At times, simpler is better: use direct traction with a gloved hand and forceps as close as possible to the tick mouthparts, avoiding twisting. It is not necessary to dig after embedded mouthparts. Cleanse the area well after removal. Please forget every other tick removal method ever devised or imagined.

36. Beside ticks and mosquitoes, do other North American arthropods transmit disease?

Yes. Fleas transmit plague in the southwestern United States. Deerfly and flea transmission of tularemia has been reported. Body lice transmit *Bartonella quintana*, the cause of trench fever, to the urban homeless. Mites (chiggers) and body lice transmit types of typhus but not in North America.

WEBSITES

Malaria, Centers for Disease Control and Prevention: www.cdc.gov/malaria

Stari, Centers for Disease Control www.cdc.gov/ncidod/dvbid/stari

West Nile virus, Centers for Disease Control and Prevention www.cdc.gov/ncidod/dvbid/westnile

World Health Organization www.who.int

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CHAPTER 53

ARTHRITIS

Catherine B. Custalow, MD, PhD

1. What are the signs and symptoms of arthritis?

Arthritis is the inflammation of a joint. The process may be monoarticular (involving a single joint) or polyarticular (multiple joints). Patients typically report pain, swelling, redness, and limitation of motion about the involved joint. On examination there may be tenderness, swelling, effusion, erythema, and decreased range of motion. Preverbal children may present with a limp or avoid using the extremity.

2. What are the common causes of acute arthritis?

Arthritis has many causes, including:

- Infection (bacterial, fungal, or viral)
- Trauma (fracture, overuse)
- Hemorrhage (traumatic hemarthrosis, inherited coagulopathy, or anticoagulant induced)
- Crystal deposition disease (gout or pseudogout)
- Neoplasm (metastasis)
- Inflammatory conditions (rheumatoid arthritis, rheumatic fever, lupus)
- Degenerative conditions (osteoarthritis)

3. What is the difference between an intra-articular and a periarticular process?

In a true intra-articular process the inflamed synovium typically causes diffuse, generalized joint pain, effusion, tenderness, warmth, swelling, and an increase in pain with active/passive range of motion and axial loading. A periarticular processes, such as bursitis, tendinitis, or cellulitis, tends to have a more localized area of tenderness, and no joint effusion. With a periarticular process, moving a joint throughout the entire range of its motion may not reproduce the pain in all directions. Instead the pain may be exacerbated only by stretching the specific muscles or tendons over the areas that are affected.

4. What are some examples of diseases that are monoarticular, polyarticular, and periarticular?

See Table 53-1 for a list of diseases by the number of joints involved.

5. What other physical findings may be helpful in diagnosing a patient with arthritis?

A careful physical examination may provide additional clues to certain rheumatologic diseases. Examples include genital ulcerations, purulent urethral discharge, and conjunctivitis in Reiter's syndrome; urethral or cervical discharge in gonococcal arthritis; tophi or concomitant renal stones in gout; malar rash in lupus; swan-neck deformity in rheumatoid arthritis; erythema chronicum migrans rash in Lyme disease; and high fever/chills in septic arthritis.

6. What does the location and distribution of the joint pain reveal about the diagnosis?

Some diseases have a predilection for certain joints. Gout most frequently affects the first metatarsophalangeal (MTP) joint. Rheumatoid arthritis commonly affects the metacarpophalangeal (MCP) and proximal interphalangeal (PIP) joints. Osteoarthritis often

TABLE 53-1. JOINTS INVOLVED IN DISEASE

Monoarticular	Polyarticular	Periarticular
Septic arthritis	Systemic lupus erythematosus	Cellulitis
Gout and pseudogout	Rheumatoid arthritis	Bursitis
Osteoarthritis	Rheumatic fever	Tendinitis
Hemarthrosis	Osteoarthritis	
Trauma	Reiter's syndrome	
	Lyme disease	
	Serum sickness	

affects the distal interphalangeal (DIP) and the first metacarpophalangeal joints. In 45% of cases septic arthritis attacks the knee.

7. Are X-rays helpful in the diagnosis of arthritis?

Often the only radiographic evidence of inflammation is soft-tissue swelling; however, plain radiographs may reveal foreign bodies, fractures, effusions, osteoporosis, or osteomyelitis. The radiographic changes of degenerative arthritis include asymmetrical joint space narrowing, marginal osteophytes, ligamentous calcifications, and subchondral sclerosis. In advanced gout, there may be *punched out* subchondral and marginal erosions, joint space narrowing, and periarticular calcified tophi.

8. Are the erythrocyte sedimentation rate (ESR) and peripheral white blood count (WBC) useful for the evaluation of acute arthritis?

No. The ESR represents the body's acute phase reaction to inflammation and infection. Unfortunately, the ESR is neither sensitive nor specific enough to confirm or exclude any particular disease. False-negative ESRs in septic arthritis can be as high as 20% to 30%. Likewise, the peripheral WBC count does not contribute meaningfully to the diagnosis of an inflamed joint as evidenced by one recent study in which 52% of patients with septic arthritis had a peripheral WBC count less than 11,000 cells/mm^3 (normal). Therefore, when clinical suspicion is high, a joint should be aspirated regardless of the ESR or WBC count.

9. What is the most important diagnostic test for determining the etiology of acute arthritis?

Arthrocentesis is the most important diagnostic procedure for evaluation of an acutely inflamed joint. Synovial fluid analysis provides rapid, critical diagnostic information and should be performed on all patients with an acute joint effusion who have no contraindications. Other indications include drainage of a tense hemarthrosis and injection of analgesic and inflammatory medications into the joint. The procedure is simple and safe, and complications are rare when performed under sterile conditions and with proper technique. If a prosthetic joint infection is suspected, an orthopedist should perform the joint aspiration if possible.

10. How is arthrocentesis performed?

The detailed anatomic approach for each joint is beyond the scope of this chapter, but the general steps are described here. Place the patient in a comfortable position with a cushion supporting the joint if needed. Palpate the bony landmarks. Cleanse and prep the skin. Provide anesthesia by local infiltration with an anesthetic solution such as 1% or 2% lidocaine. Using a 19-gauge needle attached to a syringe, aspirate gently while carefully advancing the needle

into the joint. Avoid abrasion or puncture of the articular cartilage. Withdraw as much synovial fluid as possible. If necessary, inject anesthetic solution into the joint for pain relief. Withdraw the needle. Send the synovial fluid to the lab for analysis for WBC count, differential, crystals, Gram stain, and culture.

11. What are some causes of arthritis with fever?

Diseases causing arthritis with fever include septic arthritis, Lyme disease, rheumatic fever, Reiter's syndrome, and toxic synovitis.

12. How do I interpret the results of the arthrocentesis?

See Table 53-2 for interpretation of synovial fluid analysis.

13. Does a synovial fluid white blood cell count of less than 50,000 cells/mm^3 completely rule out the diagnosis of a septic joint?

No. Typical synovial fluid counts in septic arthritis are greater than 50,000 WBC/mm^3, with predominantly polymorphonuclear neutrophilic (PMN) white blood cells, and a Gram stain positive for bacteria. But a recent study showed that 36% of patients with septic arthritis had synovial fluid counts less than 50,000 cells/mm^3 (168 cells in one patient). For this reason a high index of suspicion should be maintained when a septic joint is in the differential and the threshold for starting antibiotics must be low if the clinical examination suggests bacterial arthritis.

14. What is the most serious cause of arthritis?

Bacterial arthritis is by far the most serious cause of acute monoarticular arthritis because it can cause rapid cartilage destruction and significant in-hospital mortality. The most important risk factor for septic arthritis is pre-existing joint disease, with almost half of septic arthritis patients having previous joint problems. Permanent joint damage may occur in as little as 7 days if untreated, and this can result in chronic disability and pain. In children, septic arthritis can cause epiphyseal damage, resulting in growth impairment and limb length discrepancy.

15. What organisms cause bacterial arthritis?

Staphylococcus aureus is the most common cause of septic arthritis overall. Methicillin-resistant S. *aureus* (MRSA) causes 25% to 50% of septic arthritis cases with risk factors including advanced age, comorbid medical conditions, and significant exposure to the health care system (80% of patients hospitalized in the preceding 6 months). Other causative organisms include *Streptococcus agalactiae (group B streptococcus), Streptococcus pneumonia, Neisseria gonorrhea, Escherichia coli, Pseudomonas aeruginosa, Kingella kingae*, and *Haemophilus influenzae*. In children the incidence of septic arthritis due to *H. influenza* has decreased by 95% since widespread vaccination against this organism.

16. How is bacterial arthritis treated?

The patient should be admitted to the hospital and immediate orthopedic consultation obtained for arthroscopic joint drainage, open joint drainage, or daily joint aspirations. Intravenous (IV) antibiotics should be administered based on the Gram stain and culture of the synovial aspirate if available and are generally continued for about 3 weeks. See Table 53-3 for a list of antibiotic recommendations for each organism. If the Gram stain is negative, then empiric antibiotics can be administered according to the patient's epidemiology. MRSA coverage should be administered if the patient has risk factors such as being elderly, a recent hospitalization, comorbid medical conditions, IV drug use, or living in a location with a high prevalence of community-acquired MRSA.

17. What are some causes of arthritis with rash?

See Key Points.

TABLE 53-2. SYNOVIAL FLUID ANALYSIS

Diagnosis	Appearance	Total WBC Count (per mm^3)	PMN (%)	Mucin Clot Test	Fluid/Blood Glucose (diff.) (mm/dL)	Miscellaneous (Crystals/Organisms)
Normal	Clear, pale	0–200 (200)	<10	Good	NS	—
Group I (noninflammatory; degenerative joint disease, traumatic arthritis)						
	Clear to slightly turbid	50–4,000 (600)	<30	Good	NS	—
Group II (noninfectious, mildly inflammatory; SLE scleroderma)						
	Clear to slightly turbid	0–9,000 (3,000)	<20	Good (occasionally fair)	NS	Occasionally LE cell, decreased complement
Group III (noninfectious, severely inflammatory)						
Gout	Turbid	100–160,000 (21,000)	70	Poor	10	Uric acid crystals
Pseudogout	Turbid	50–75,000	70 (14,000)	Fair-poor	Insufficient data	Calcium pyrophosphate
Rheumatoid arthritis	Turbid	250–80,000	70	Poor	30	Decreased
Group IV (infectious, inflammatory)						
Acute bacterial	Very turbid	150–250,000 (80,000)	90	Poor	90	Positive culture for bacteria
Tuberculosis	Turbid	2,500–100,000 (20,000)	60	Poor	70	Positive culture for *Mycobacterium tuberculosis*

LE, lupus erythematosus; NS, not significant; PMN, polymorphonuclear cells; SLE, systemic lupus erythematosus.
(From Wyngaarden JB, Smith LH, editors: *Cecil textbook of medicine*, ed 18, Philadelphia, 1988, W. B. Saunders, p 1994, with permission.)

TABLE 53-3 ANTIBIOTIC TREATMENT FOR SEPTIC ARTHRITIS

Organism	Gram Stain	Antibiotics	Dosage
Methicillin-sensitive *Staphylococcus aureus*	Gram-positive cocci clusters	cefazolin or nafcillin or oxacillin	cefazolin 2 g IV q 8 nafcillin 2 g IV q 4 oxacillin 2 g IV q 4
Methicillin-resistant *S. aureus*	Gram-positive cocci clusters	vancomycin	vancomycin 15 mg/kg IV q 12
Streptococcus pneumonia	Gram-positive cocci chains	penicillin G or ampicillin	penicillin G 12–18 mU IV q day divided
Penicillin sensitive			Ampicillin 2 g IV q 4 h;
S. pneumonia Penicillin resistant	Gram-positive cocci chains	ceftriaxone or cefepime	ceftriaxone 1 g IV q 24 h cefepime 2 g IV q 8 h
Neisseria gonorrhea	Gram-negative cocci	ceftriaxone or cefepime	ceftriaxone 1 g IV q 24 h cefepime 2 g IV q 8 h
Pseudomonas aeruginosa	Gram-negative rods	ceftazidime or cefepime plus gentamicin or tobramycin	ceftazidime 2 g IV q 8cefepime 2 g IV q 8 h gentamicin 5 mg/kg IV q 24 tobramycin 5 mg/kg IV q 24

g, gram; h, hour; IV, intravenously; mU, million units; q, every.

KEY POINTS: CAUSES OF ARTHRITIS WITH A RASH

1. Lyme disease: Erythema chronicum migrans
2. Reiter syndrome: Keratoderma blennorrhagicum
3. Disseminated gonococcal infection: Pustular rash
4. Psoriatic arthritis: Psoriatic lesions
5. Systemic lupus erythematosus: Malar rash

18. What is the difference between gout and pseudogout?

Gout develops when sodium urate crystals precipitate in a joint and pseudogout develops when calcium pyrophosphate crystals precipitate in the joint. Both are released from the cells lining the synovium and initiate an inflammatory reaction. Under polarized light microscopy, gout crystals are needle shaped and negatively birefringent, whereas pseudogout crystals are rhomboid in shape and positively birefringent.

19. What are the risk factors for gout and which joints are most commonly affected?

Risk factors for gout include obesity, hypertension, diabetes, dietary excess, alcohol consumption, proximal loop diuretics, increased uric acid levels, and stress (illness or surgery). Middle-aged men and postmenopausal women are at an increased risk for gout. The MTP joint of the great toe is the most frequently affected joint (up to 75%). In this joint gout is known as podagra. Other commonly involved joints are the tarsal joints, the ankle, and the knee. Gout is polyarticular in as many as 40% of patients.

20. What medications can be used to treat gout emergently?

Nonsteroidal anti-inflammatory drugs (NSAIDs) are the primary agents used to treat gout. Indomethacin is commonly used at a dose of 75 to 200 mg/day for several days and tapering off as inflammation decreases. Colchicine inhibits microtubule formation, which results in a decreased inflammatory response and is also effective in treating acute attacks. It may be administered orally at a dosage of 0.5 to 0.6 mg every hour until symptoms improve, until diarrhea or vomiting develops, or until the maximum dose of 6 mg has been reached. A single IV dose of 1 to 2 mg of colchicine administered over 10 minutes may also be quite effective. Once bacterial infection has been ruled out, oral corticosteroids may also be administered, such as prednisone at a dose of 40 mg/day for 3 days and then tapering off. Drugs that alter serum uric acid levels such as allopurinol and probenecid should not be administered acutely because changing serum uric acid levels can exacerbate the condition.

21. Which tick-borne infection causes arthritis?

See Chapter 52.

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CHAPTER 54

SKIN DISEASES

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1. How are skin lesions best described?

It is helpful to remember dermatologic terms (Table 54-1). Describing in plain English how the lesions appear is acceptable, however. Communication with other care providers may be improved by the use of plain but accurately descriptive terms.

Characteristics helpful in establishing a diagnosis include the following: location and distribution, color, size, presence or absence of scale, contour (e.g., raised, depressed, or pitted), tactile characteristics (e.g., firm, spongy, fluctuant, blanchable, or nonblanchable), and apparent depth (e.g., superficial, dermal, or subcutaneous).

2. What categories of skin conditions are life-threatening or associated with life-threatening disease?

- Diseases resulting in extensive compromise to the cutaneous barrier
- Skin signs of systemic infection (e.g., meningococcemia)
- Cancers (e.g., melanoma)
- Urticaria or angioedema with airway compromise or anaphylaxis
- Skin signs of vascular compromise (including hemorrhage, emboli, thrombi, and vasculitis)
- Skin findings of an introduced toxin (e.g., venomous snake bite)
- Skin signs of physical abuse

3. List some cutaneous red flags (i.e., skin signs indicating an increased likelihood of disease requiring emergency attention).

- Extensive blisters or denuded areas of skin
- Acute total-body erythema, particularly in the elderly or frail
- Extensive erythematous eruption in a person who is febrile and systemically ill
- Petechiae, purpura, and ecchymoses
- Necrosis
- Urticaria
- Isolated, abnormal-appearing mole

4. What types of skin diseases result in potentially life-threatening compromise to the skin barrier?

Most of these are blistering diseases. When the blister breaks, the barrier is removed, and the individual is at risk for infection, fluid and electrolyte imbalance, and difficulties with heat regulation. Skin conditions that can be associated with an extensively compromised barrier include toxic epidermal necrolysis (TEN), Stevens-Johnson syndrome (SJS), pemphigus and pemphigus-like chronic blistering diseases, and burns. Patients with erythroderma (total or near-total body erythema) also may have problems with infection, fluid and electrolyte balance, and heat regulation, particularly patients who have significant chronic health problems, such as congestive heart failure. Lesions of the oral cavity may compromise life if they are severe enough to prevent food or fluid intake.

TABLE 54-1. BASIC DERMATOLOGIC TERMS

Skin Lesion	Description	Example
Macule	Flat, circumscribed color change (nonpalpable) <1 cm	Café-au-lait spot
Patch	Flat color change >1 cm	Vitiligo
Papule	Raised lesion <1 cm	Molluscum contagiosum
Plaque	Elevated, flat-topped lesion >1 cm. Lesions with epidermal changes (e.g., scale) would be considered plaques.	Psoriasis
Nodule	Raised lesion with a deeper palpable portion	Erythema nodosum
Vesicle	Raised, usually dome-shaped lesion filled with fluid and <1 cm	Varicella
Bulla	Fluid-filled lesion >1 cm	Bullous pemphigoid
Pustule	Raised lesion filled with exudative fluid, giving it a yellow appearance	Folliculitis
Cyst	Nodule filled with semisolid-to-solid material	Epidermoid cyst
Wheal	Flat-topped, firm, raised, edematous lesion; a hive	Urticaria

KEY POINTS: PHYSICAL FINDINGS OF LIFE-THREATENING DISEASES WITH COMPROMISE TO SKIN BARRIER

1. Extensive blistering
2. Erythroderma
3. Extensive oral erosions preventing food or fluid intake

5. Describe the skin findings in meningococcemia, Rocky Mountain spotted fever, toxic shock syndrome, and necrotizing fasciitis.

- In **meningococcemia**, lesions may be irregularly shaped petechiae or purpura with dusky centers, located most commonly on the trunk and extremities. The lesions may involve the palms and soles.
- In **Rocky Mountain spotted fever**, skin lesions appear on about day 4 of the acute febrile illness. Lesions begin on the distal extremities, may involve the palms and soles, and spread centripetally. After a few days, the lesions become petechial or purpuric. In practice, this eruption may be difficult to distinguish from that of meningococcemia.
- Patients with **toxic shock syndrome** may have a scarlatiniform eruption, edema of the face and extremities, conjunctival erythema, and erythema of the oral or genital mucosa. There is desquamation of the hands and feet 1 to 2 weeks later.
- **Necrotizing fasciitis** is characterized by a rapidly progressive painful erythema with development of duskiness and frank necrosis. There may be blisters. The overlying skin change is often mild compared with the necrosis occurring underneath.

6. Describe the skin findings of common or distinctive childhood exanthems.

- **Scarlet fever** occurs in children between the ages of 2 and 10 years. Red macules and papules start on the neck and usually spread to the trunk and extremities. The skin may have a rough *sandpaper* feel on palpation and sometimes can be petechial. Erythema is usually most intense in the axillae, groin, and abdomen. Patients may exhibit Pastia's lines, which are petechiae in a linear pattern along the major skin folds. Palms and soles characteristically are spared. The face appears flushed with circumoral pallor. Desquamation usually occurs as the eruption resolves in 1-3 weeks.
- **Staphylococcal scalded skin syndrome** begins as faint erythema on the face, neck, axilla, and groin in a child younger than 5 years, usually following upper respiratory symptoms. The skin is tender, and crusting typically occurs around the mouth, eyes, and skin folds. The skin separates through the epidermis with even slight rubbing, leaving a red, moist surface underneath (Nikolsky's sign). Mucous membranes are not involved. In neonates, the entire body is involved, whereas in infants and children the upper body is affected preferentially.
- **Varicella** (chickenpox) presents with abrupt onset of crops of faint macules that progress through several stages. The macules become edematous papules, then vesicles over 24 to 48 hours. Often there is a small vesicle on a larger erythematous macule, described as a "dew drop on a rose petal." The vesicles develop moist crusts and leave shallow erosions. Lesions tend to begin centrally and spread to the extremities. The palms, soles, and mucous membranes frequently are involved. Characteristically, lesions in multiple stages of development (i.e., macules, papules, vesicles, crusts, erosions) are present in the same patient. The number of lesions ranges from 10 to 1,500 (average 300). The lesions are usually pruritic. In general, lesions heal without scarring, although scarring may occur in some instances.
- **Erythema infectiosum (fifth disease)** is a parvovirus B19 infection that results in intense erythema on the bilateral cheeks (*slapped cheeks*). In some patients, after the facial erythema, a diffuse pink-to-red macular eruption develops with a reticular or *lacy* appearance. The macular eruption tends to *reappear* with stimulation of cutaneous vasodilation, such as warm baths, exercise, or sun exposure. This can last for 4 months. Some patients develop a petechial eruption of the distal hands and feet with parvovirus B19 infection. This is the *purpuric gloves and socks syndrome.*
- **Roseola (exanthem subitum)** is a disease of infants and toddlers caused by infection with human herpesvirus 6. After 2 to 3 days of sustained fever, abrupt defervescence is followed by a pink maculopapular eruption. Periorbital edema is sometimes seen.
- **Hand-foot-and-mouth disease** caused by Coxsackieviruses exhibits an abrupt onset of a few scattered papules that progress to oval or linear vesicles with an erythematous rim. As the name suggests, most lesions occur on the oral mucosa, palms, and soles. The oral lesions appear as small, discrete, whitish-gray erosions.
- **Kawasaki disease** is typified by an irritable child with conjunctival injection and slightly swollen, distinctly red lips. The hands may be edematous or desquamating. The skin findings are nonspecific and variable, ranging from macules to maculopapules to vesicles. The child must meet the clinical criteria of fever for 5 or more days plus four of five of the following:
  a. Nonpurulent conjunctivitis
  b. Mucosal changes
  c. Edema or desquamation of the distal extremities
  d. Exanthema
  e. Cervical lymphadenopathy

7. What is erythema multiforme (EM)?

EM is usually an eruption of acute onset that is characterized by multiple, fixed red papules. Because the keratinocyte is the target of inflammatory insult, there is keratinocyte necrosis or apoptosis, manifest clinically as a central dusky center. The characteristic lesion is the

target lesion: a papule with a central dusky zone and an outer zone of erythema. The majority of EM lesions are erythematous, whereas typically only a few lesions are truly target-like. Some lesions may develop vesicular changes in the center, due to intense necrosis.

EM frequently follows herpes simplex (HSV) infection. Mucous membranes are usually spared or affected mildly. Lesions are found on the dorsal hands and extensor extremities, and palms and soles frequently are involved. Patients with HSV-associated EM are not generally ill-appearing, and although the eruption can be quite impressive, it can be managed on an outpatient basis. The eruption lasts 10 to 14 days and may recur after subsequent episodes of HSV. Antiviral therapy is useful only as suppressive therapy to prevent HSV recurrences. Treatment is usually reassurance or oral antihistamines to treat any associated burning and itching. Topical or oral steroids are not indicated.

8. Which disease is most often mistaken for EM?

Acute urticaria. Urticarial lesions may be annular with concentric color changes and may occur on the palms and soles. Usually, urticarial lesions have a pale edematous center with an erythematous border, whereas EM lesions tend to have dusky centers. Lesions in urticaria are transient (<24 hours), whereas the target-like lesions of EM are fixed and can be present for one week or more. Urticarial lesions clear with subcutaneous epinephrine, whereas EM lesions do not.

9. What is the most common clinical presentation of a cutaneous adverse drug reaction?

Exanthematous (morbilliform, maculopapular, or urticarial) eruptions are the most common presentation of a cutaneous adverse drug reaction. The eruption is characterized by erythematous macules and papules that are usually widespread and symmetrically distributed. Lesions can sometimes show central clearing, often leading to the incorrect diagnosis of EM. The eruptions usually begin 7 to 14 days after the onset of the new medication, but in the cases of rechallenge, time to onset can be shorter. Treatment includes stopping the offending drug and prescribing antihistamines for symptomatic relief of pruritus.

10. What drugs are most often implicated in exanthematous drug eruptions?

Aminopenicillins, sulfonamides, cephalosporins, and anticonvulsants.

11. What is the main diagnosis in the differential of exanthematous drug eruptions?

Viral exanthems are often clinically and histologically indistinguishable from morbilliform drug eruptions. History of recent drug administration or symptoms of viral illness will help distinguish. Peripheral blood eosinophilia favors drug eruption. In children, 10% to 20% of exanthematous eruptions are drug induced, whereas 50% to 70% are drug induced in adults. Skin biopsy will not distinguish viral from drug exanthematous eruptions and is not helpful to distinguish which drug is the cause.

12. What clinical signs should prompt consideration of a more serious adverse drug reaction?

Facial edema; marked peripheral blood eosinophilia; hepatosplenomegaly; hemorrhagic and tender mucous membrane lesions; painful, dusky papules or plaques; or blistering and sloughing of the skin should prompt consideration of the serious drug reactions.

13. Describe the signs and symptoms of the most serious drug eruptions.

Three severe adverse drug reactions that can result in death are SJS, TEN, and drug reaction with eosinophilia and systemic symptoms (DRESS) (See Table 54-2). Urticaria and angioedema can also be life-threatening if the airway is compromised.

TABLE 54–2. SIGNS AND SYMPTOMS OF THE MOST SERIOUS DRUG ERUPTIONS

	Causes	Clinical Signs/ Symptoms	Mortality Rate	Treatment
SJS	Drugs (70%–90%): antibacterial sulfonamides, anticonvulsants, NSAIDs, allopurinol *Mycoplasma pneumoniae* infection	Prodrome 1–14 days before the onset of mucosal involvement is common (e.g., fever, malaise, headache, sore throat, rhinorrhea and cough) Acute eruption with red to dusky, tender papules at times targetoid with potential for blistering/sloughing (usually <10% of the BSA involved) Extensive, hemorrhagic necrosis of two or more mucous membranes (mouth and eye most common)	5%	Prompt withdrawal of drug Supportive care (wound care in an ICU or bum unit setting if sloughing of skin prominent, enteral nutrition, fluid/electrolyte replacement, pain management, monitoring for infection) Steroid use is controversial; some evidence that steroid use results in worse prognosis; if part of treatment should be started very early in the course
TEN	Drugs (95%): antibacterial sulfonamides, anticonvulsants NSAIDs, allopurinol	Onset with fever and tender skin several days before blistering/sloughing begins Painful, dusky red macules appear, become dusky, then confluent with islands of sparing, then blisters/sloughing of the skin begins (usually >30% BSA involved) Mucous membrane involvement in 90% of cases Poor prognostic factors include increasing age, delay in withdrawal of offending agent, and greater extent of epidermal attachment	30%–35%	Prompt withdrawal of drug* Supportive care in an intensive care burn unit setting (see SJS section) The following are used in treatment of TEN, but efficacy is controversial: intravenous immunoglobulin, systemic steroids (see SJS section)

TABLE 54-2. SIGNS AND SYMPTOMS OF THE MOST SERIOUS DRUG ERUPTIONS—cont'd

	Causes	Clinical Signs/ Symptoms	Mortality Rate	Treatment
DRESS	Drugs: anticonvulsants, sulfonamides, allopurinol, minocycline, dapsone, gold salts	Fever and a morbilliform skin eruption are the most common presenting features. The eruption then becomes edematous with follicular accentuation. Facial edema is a hallmark of DRESS. Lymphadenopathy, eosinophilia, and atypical lymphocytosis are common features. Visceral inflammation (hepatitis most common)	10%	Prompt withdrawal of the drug Systemic steroids (severe cases with visceral inflammation, although efficacy unproven in controlled clinical trials) Topical steroids (milder cases for cutaneous manifestations)

BSA, body surface area; DRESS, drug reaction with eosinophilia and systemic symptoms; ICU, intensive care unit; NSAIDs, nonsteroidal anti-inflammatory drugs; SJS, Stevens-Johnson syndrome; TEN, toxic epidermal necrolysis.
*Garcia-Doval I, LeCleach L, Bocquet H, et al: Toxic epidermal necrolysis and Stevens-Johnson syndrome: does early withdrawal of causative drugs decrease the risk of death? *Arch Dermatol* 136: 323–327, 2000.

KEY POINTS: USUAL TIME TO ONSET FOR CUTANEOUS DRUG ERUPTIONS

1. DRESS syndrome begins 2 to 6 weeks after the responsible drug is started.
2. One to 3 weeks are required for the development of SJS and TEN.
3. Four to 14 days are required for exanthematous drug eruptions.

14. How do I recognize a melanoma?

Melanoma requires urgent diagnosis and treatment. Recognition of the potential and referral for surgical removal of melanoma before it has metastasized can be life-saving. Findings suggestive of melanoma are irregular pigment; irregular borders; and presence of red, white, or blue-black color. An underemphasized finding that can be extremely helpful is a difference in appearance between the lesion in question and the patient's other moles. A brown macule on a fair-skinned person should be viewed with suspicion if the person does not have other similarly pigmented lesions, even if the brown macule is regularly pigmented, small, and perfectly round. A history of change in a mole is a risk factor, as is personal or family history of melanoma.

KEY POINTS: CUTANEOUS FEATURES CONCERNING FOR MELANOMA

1. Different from patient's other pigmented lesions
2. Recent changes in size, shape, and color
3. Markedly irregular borders
4. Markedly irregular pigmentation with colors of red, white, or blue
5. Areas of pigment regression
6. Any one of these may be an indicator of melanoma without the other features being present.

15. Describe common benign skin conditions that mimic melanoma.

- **Seborrheic keratoses** are extremely common benign lesions that usually first appear in middle age. They are benign growths of keratinocytes and can look alarmingly dark or irregularly pigmented. The scaling produced by the seborrheic keratosis may be so compact that it is difficult to discern, but detection of this rough scaling helps considerably in distinguishing this lesion from melanoma.
- **Venous lakes** are vascular growths that often appear on the helix of the ears and on the lips of older persons with sun damage. The purple color may mimic that of a melanoma. Pressing firmly on the lesion drains much of the blood from the lesion and reveals it as a vascular growth.

16. Which spider bites can cause a necrotic reaction?

Only a few species of spiders in North America produce bites that may lead to skin necrosis. Of these, bites of the brown recluse *(Loxosceles reclusa)* and the hobo spider *(Tegenaria agrestis)* are important because they may be fatal. The range of the brown recluse is centered in Arkansas, Tennessee, and Missouri. Outside endemic areas, brown recluse bites are uncommon, although they may occur because the spider may be transported on clothing or in boxes. The range of the hobo spider is the northwest United States and western Canada. Other *Loxosceles* species may cause necrotic reactions, and many of these spiders live in the deserts of the southwest United States. If one is outside an endemic area, the diagnosis of necrotic reaction to spider bite should be made with caution.

17. What skin lesions may be confused with a necrotic reaction to a spider bite?

Necrotizing fasciitis, ecthyma, pyoderma gangrenosum, vasculitis, and clotting disorders. Erythematous reactions to stings or bites, such as bee stings or tick bites, occasionally may be confused with early reactions to spider bites. Most recently, methicillin-resistant *Staphylococcus aureus* (MRSA) skin infection is often confused with spider bites.

18. What are the cutaneous findings of skin and soft-tissue infections (SSTI) with MRSA?

SSTIs are the most common clinical manifestation of community-acquired MRSA (CA-MRSA). This presents most frequently as a skin abscess or furunculosis. Often the patient will give a history of an "infected spider bite." There is no evidence to conclude that MRSA SSTIs are clinically distinguishable from infection with susceptible strains of *S. aureus,* but several risk factors are more common with MRSA such as a history of a spider bite, antibiotics in the past month, history of previous MRSA, close living quarters, and activities with skin-to-skin contact (athletes). Primary treatment is incision and drainage of abscesses. Adjunctive therapy of purulent skin infections with antibiotics has not been proven to be necessary in several studies in healthy, immunocompetent patients. Antibiotics are indicated if there is fever or

surrounding cellulitis. For further recommendations for the treatment of CA-MRSA infections, see: www.cdc.gov/ncidod/dhqp/pdf/ar/CAMRSA_ExpMtgStrategies.pdf

19. Describe common benign skin conditions that mimic purpura resulting from systemic disease.

- **Solar purpura** is common on the forearms and backs of hands of people who have chronic sun damage. Large areas of purpura may be evident and may have occurred with minimal, sometimes unnoticeable trauma. Solar purpura is restricted to chronically sun-damaged skin and is particularly common in patients on long-term systemic steroid therapy.
- **Schamberg's purpura** is a benign condition characterized by petechiae primarily on the lower legs. The lesions tend to be pinpoint, nonpalpable, and extremely numerous. By contrast, purpuric lesions of leukocytoclastic *(hypersensitivity)* vasculitis tend to be slightly larger in diameter (often 2–4 mm), and frequently some of the lesions are palpable. (Although leukocytoclastic vasculitis sometimes is referred to as *palpable purpura*, it is common to find that most lesions are flat and only a few are palpable.)

20. Describe common skin conditions that mimic cellulitis.

- A **kerion** caused by fungal infection in the scalp (tinea capitis) may be so intensely inflamed that it is mistaken for cellulitis. Because a kerion may produce permanent, scarring alopecia, it is important to recognize and institute therapy early. Kerions occur almost exclusively in children.
- **Stasis dermatitis** sometimes may be confused with cellulitis. Stasis dermatitis is usually bilateral, whereas leg cellulitis is more often unilateral. Stasis dermatitis is characterized by scaling and mild-to-moderate erythema. If the erythema is fiery red, the redness is rapidly progressive, the patient is systemically ill, or there is a leukocytosis, cellulitis may be the presumptive diagnosis.
- **Allergic contact dermatitis**, such as poison ivy dermatitis, may result in lesions that are intensely inflamed. The distribution of the lesions often suggests an exogenous cause. In plant dermatitis, linear erythema, often with blisters, is an indicator of where the plant has brushed against the skin. Antibiotic creams containing neomycin are another relatively common cause of allergic contact dermatitis. Because antibiotic creams often are used on wounds, allergic contact dermatitis caused by the cream may be mistaken for a resistant or on-going wound infection.

21. In which disease associated with leg ulceration is débridement generally contraindicated?

Pyoderma gangrenosum.

22. When should patients with dermatitis (eczema) be treated with systemic steroids?

Systemic steroids generally should not be given to patients with *chronic* dermatitis. Topical steroids should be used to avoid systemic side effects. Topical ointments and creams target one of the primary problems in chronic atopic dermatitis, which is a skin barrier defect. Patients taking systemic steroids also may exhibit a rebound of disease when the steroids are tapered. Patients with acute dermatitis, such as severe poison ivy dermatitis, that is expected to be self-limited may be given systemic steroids if the severity of disease merits and there are no contraindications.

23. Should steroids be used for psoriasis?

Systemic steroids are generally considered to be contraindicated for the treatment of psoriasis because severe rebound with generalized pustular psoriasis may occur following withdrawal of the steroid.

24. What are the divisions of the classes of topical corticosteroids? On which areas of the skin are they appropriately applied?

- **Low-potency** topical corticosteroids (class 6 or 7 topical steroids, such as 1% and 2.5% hydrocortisone, 0.05% desonide) are appropriate to use on the face, axillae, groin, breasts, and genitalia, where the skin is thinner and more prone to cutaneous side effects.
- **Moderate-potency** topical corticosteroids (class 4 or 5 topical steroids, such as 0.025% fluocinolone, 0.1% triamcinolone, 0.2% hydrocortisone valerate) are useful on the neck and body, avoiding the more sensitive areas mentioned previously. This class is given most appropriately as first-line therapy for skin conditions diagnosed in the ED.
- **High-potency** topical corticosteroids (class 2 or 3 topical steroids, such as 0.05% fluocinonide, 0.1% halcinonide, 0.25% desoximetasone) should not be applied to the face, breasts, genitalia, axillae, or groin. Topicals in this class are more likely to produce cutaneous side effects if used diffusely or for long periods (>2 weeks). This class should be prescribed only if moderate-potency topical corticosteroids have not been effective or if the condition is limited to the particularly thick skin of the palms and soles.
- **Superpotent** topical corticosteroids (class 1 topical steroids, such as 0.05% clobetasol, 0.05% betamethasone dipropionate in optimized vehicle, 0.05% halobetasol, 0.05% diflorasone) are usually reserved for chronic, recalcitrant conditions, often of the palms and soles. The risk of cutaneous side effects is greatest with this class, and this class of steroid should be dispensed in a continuity-of-care rather than an ED setting.

25. Does the vehicle of the topical corticosteroid affect potency?

Yes. The same corticosteroid may be significantly more or less potent, depending on the vehicle. In general, ointments are most potent, followed by gels, emollients, creams, lotions, solutions, and sprays.

WEBSITE

www.cdc.gov/ncidod/dhqp/pdf/ar/CAMRSA_ExpMtgStrategies.pdf

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LIGHTNING AND ELECTRICAL INJURIES

Gabrielle A. Jacquet, MD, and Timothy R. Hurtado, DO, FACEP

LIGHTNING INJURIES

1. What causes lightning?

The etiology of lightning is a highly complex science involving physics and meteorology. First of all, a large charge separation must occur within a cloud, turning it into a giant *battery* or capacitor. This occurs from hailstones and raindrops settling at various rates in a convectively active cloud. Charged particles get *stripped off*, and the cloud separates into a dipole or tripole (two or three, respectively, equal and opposite electric charges or magnetic poles separated by a small distance). Most often, the cloud base becomes negatively charged. In response, an *induced shadow* of positive charge forms on the ground. The potential between the ground and the cloud can be as great as 7,500 volts per inch. Lightning is initiated by the formation of a *stepped leader*, a zigzag, short, stepped, downward series of branching ionized plasma channels. At the same time, a positively charged *pilot stroke* arises from the ground, usually from the tops of tall objects. When the two meet at about 50 to 100 m above the ground, the *return stroke* is initiated. This is what we think of as the lightning bolt. It is a high-voltage, high-current, high-velocity discharge that travels up the plasma channel. There is an average of four to five return strokes. Lightning only *sees* objects that are in a radius of 30 to 50 m from the tip. Thus, tall objects (such as a tall tree on a ridge) that are greater than 50 m from the stepped leader may not offer protection from a strike.

2. How about thunder?

Thunder is an acoustic wave caused by the sudden heating of the air from lightning. It is the result of explosive shock waves from instantaneous superheated ionized air. This can cause a pressure rise up to 10 atm. The sound of thunder is rarely heard over 10 miles away and has a lower pitch at greater distances. In addition, atmospheric turbulence may decrease the distance the sound travels.

3. What are the mechanisms of lightning injury?

Lightning causes injuries in three basic ways:

a. Its electrical effects
b. The heat it produces
c. The concussive forces it creates.

Another way to consider lightning mechanisms is as follows:

- **Direct strike or contact injury**: This occurs when the victim is in direct contact with the lightning or an object or structure that is struck by lightning. It usually results in the highest mortality and morbidity.
- **Splash or side flash**: Lightning arcs from an object to a nearby person. Current seeks a path of least resistance and can "jump" from its primary target to others.
- **Step or stride voltage**: The lightning current strikes the ground, then spreads out. Current moving underground can cause an induced current, up one leg and down another. This is a reason for multiple victims of a single strike. Humans offer less resistance than the ground thus lightning frequently spreads into their bodies after the nearby ground is struck.

- **Rising upward streamer**: This newly described mechanism happens with an injury from the rising positively charged streamer, which does not connect with a pilot stroke to create the plasma channel that allows return strokes, what we think of as a lightning bolt.
- **Blunt trauma and blast trauma**: Blast trauma may occur from the thunderclap. Common findings from this blast include *pulmonary contusions*, *tympanic membrane rupture*, and *conductive hearing loss*. A victim may also be "thrown" by diffuse muscle contractions.
- **Secondary trauma and burns from fires**: Trauma from falls or falling objects and thermal burns from ignited clothing, heated metal, and burning surroundings can also complicate lightning injuries.

4. True or false: During a thunderstorm, get into a car because the rubber tires will insulate it?

False. You are safer inside a car than out during a thunderstorm but not because of the tires. Think about it: The lightning bolt just passed several kilometers through the air to get to the ground. It won't be intimidated by 6 inches of rubber (or the rubber soles of your tennis shoes, for that matter). The real reason that the car is safer is the metal skin of the body. It acts as a **Faraday cage** (Table 55-1) allowing electromagnetic flow over the outer surface, isolating the occupants. Now, of course, that may not protect the occupants from the flash, thunderclap, splash current, or induced electromagnetic currents through the interior.

5. True or false: People are safe from lightning if they are indoors?

False. Maybe safer, but a significant number of lightning injuries occur to people who are inside buildings. One mechanism for this is a side flash through plumbing, telephone wires, and electrical appliances connected to the outside of the building by metal conductors.

6. True or false: Lightning never strikes twice in the same place?

False. The Empire State Building in New York is struck about 23 times a year; once, it was struck eight times in 24 minutes.

7. Is a lightning bolt an alternating current (AC) or a direct current (DC)?

It is neither. It is a unidirectional current impulse (technically neither AC nor DC, although the closest model would be a large DC discharge). It is very high voltage (100 million to 2 billion volts), very large current (20,000–300,000 peak amps), and very high energy (1 billion joules or 280 kilowatt hours). The bolt is also very hot: 8000°C to 25,000°C (the surface of the sun is *only* 6000°C). The good news is that it's a very short duration phenomenon (0.1–1 msec). This ultrashort duration of exposure is the most important factor that separates lightning injuries from other high voltage electrical injuries. The energy released in a lightning bolt is more electrical energy than is produced by all of the electrical generators in the United States

TABLE 55-1. LIGHTNING GLOSSARY

Fulgurite	The word is from Latin, meaning lightning stone. When lightning strikes the ground, it can spread out underground for up to 60 ft in radial horizontal arcing. Depending on soil's characteristics, there can be enough heat generated to fuse silica into branching configurations. These stones are the fulgurites.
Faraday cage	An enclosure formed by conducting material or by a mesh of such material; blocks out external static electrical fields.
Flashover phenomenon	Given the short duration of a lightning bolt, current is often conducted over the skin without penetration. When this happens, it significantly lowers the mortality of a direct strike victim from 85% with signs of penetration to 40% without.

at that instant. However, because it is such short duration, it is only enough energy to light a single light bulb for about a month. The energy is dissipated as light, heat, sound, and radio waves.

8. Does lightning ever hit airplanes? What happens?

Yes, and usually not much. There are at least 160 lightning strikes on aircraft annually. Typically, these strikes happen at 10,000 ft to 15,000 ft, in rain and light turbulence, within a cloud and near the freezing level. Because the aircraft skin is metal, the lightning almost always ***flashes over***, leaving only minor damage. However, the blast effect of the thunderclap can interrupt jet engines; the bright flash can temporarily blind pilots, and the induced electromagnetic field can disrupt avionics and communication equipment—just what you don't want at 10,000 ft to 15,000 ft, in rain and light turbulence, within a cloud and near the freezing level. There have been a few aircraft lost to fuel vapor explosions within the fuel tanks induced by lightning.

9. How common is lightning?

There are up to 8 million lightning flashes worldwide each day. Across the globe, there are 2,000 active thunderstorms right now. Those storms produce 100 cloud-to-ground strikes per second. In the United States, there are 50 to 300 lightning fatalities every year. This number is hard to pin down exactly due to the majority of data being gathered from newspaper and news accounts. "Storm Data" reports an average of 58 fatalities per year for the past 30 years, but it suggests that there are probably closer to 70 deaths per year.

10. In what types of locations do lightning injuries occur?

In 40% the location goes unreported. Of those that are reported, 27% happen in open fields and recreation areas; 14% happen under trees; 8% are water-related (e.g., boating, fishing, or swimming); 5% happen on golf courses; 3% are related to the use of heavy equipment and machinery; 2.4% are telephone related, and 0.7% are related to radio transmitter and antennae. Central Florida is thunderstorm alley with the greatest number of thunderstorm days in the United States.

States that are consistently in the top 10 for both total casualties and the highest casualty rate are Florida, Colorado, and North Carolina. See Table 55-2.

11. Am I more likely to get struck as a woman or a man?

Remarkably, 84% of victims are male, likely reflecting a higher number of men engaging in outdoor recreational and occupational activities.

TABLE 55-2. Top States for Lightning Incidence and Injury

Top Three States for Lightning Incidence	Bottom Three States for Lightning Incidence
Florida	Alaska
Texas	Hawaii
Colorado	North Dakota
Top Five States for Lightning Injuries	**Top Five States for Lightning Deaths**
Florida	Florida
Michigan	Michigan
Pennsylvania	Texas
North Carolina	New York
New York	Tennessee

12. What time of year am I more likely to get struck?

Summer seems to be the most dangerous time: the monthly incidence is June 21%; July 30%; August 22%.

13. What factors predispose somebody or something to be hit by lightning?

- Height of the object
- Isolation
- Pointiness of the object (this does not apply to humans)

14. I'm treating a farmer who was found unconscious in his field after a thunderstorm. He has no recollection of what happened. How can I tell if he was struck by lightning?

The easiest way to tell is to look at his skin and look in his ears. His memory of the events won't help; in fact, it is said that 100% of direct-strike victims have amnesia. Victims of nondirect strikes may be able to recall being hit by lightning, but they often develop anterograde amnesia. However, arborescent superficial erythema (known as Lichtenberg's flowers, ferning, arboration, or fractals) is pathognomonic for lightning. See Figure 55-1.

Unfortunately, it is seen in only seen in 20% of confirmed cases and fades away over hours. It is not a true burn but the effect of a strong electromagnetic field on wet skin. It has been postulated to be secondary to red blood cell extravasation into the superficial layers of the skin from capillaries from the electrical injury. In addition, there may be partial-thickness linear or punctate burns in moist areas. Tympanic membrane rupture (one or both) occurs in 50% of victims. Tinnitus is also common and usually resolves in hours to days. Seven percent to 12% of victims experience temporary hearing loss, and a few have permanent hearing loss.

15. The farmer is also tachycardic and hypertensive, with cool, pale skin, and diminished peripheral pulses. Although awake, he seems unable to move his extremities. Why?

He is likely suffering from *lightning paraplegia* (keraunoparalysis or Charcot's paralysis). It is probably primarily due to intense adrenergic stimulation, vasospasm, and hypoperfusion, not direct nerve injury. If that is the case, it typically resolves over the next several hours. However, you should be wary of occult injuries and diligent in your work-up for traumatic injuries.

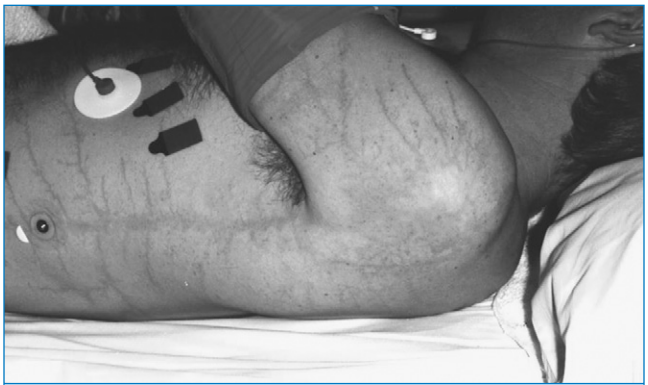

Figure 55-1. Ferning developed after the victim received a side flash lightning strike. (From Auerbach PS: *Wilderness medicine*, ed 5, Philadelphia, 2007, Mosby. Courtesy Sheryl Olson, RN.)

16. My practice at multiple casualty incidents (MCIs) is to allocate resources to victims who are not breathing and not moving *only after* I have taken care of those with signs of life. Is that rule applicable in a lightning strike MCI?

No. Lightning strike is the one exception to the usual MCI triage rule. The first priority should go to those who are not breathing and not moving. The reasons are that only those who present in cardiac arrest are at high risk of dying. Bystander cardiopulmonary resuscitation (CPR) doubles survival from about 24% without CPR to 50% with CPR. Those who have not arrested have little chance of dying, and so in this case the first priority goes to "the dead." Of note, victims' cardiac automaticity often returns before spontaneous respirations; as such, they may need prolonged rescue breathing during CPR.

17. I have heard that prolonged CPR is beneficial in lightning injuries. Is this true?

No. There is no evidence that prolonged CPR improves recovery. It is reasonable to stop CPR after 20 to 30 minutes as long as other reversible causes of cardiopulmonary arrest have been addressed.

18. I am caring for a patient who has been struck by lightning and he has fixed and dilated pupils. Can I stop CPR based on this finding?

No. One should not use dilated/fixed pupils as an indicator of brain death or to gauge the prognosis in lightning victims until all other anatomical and functional etiologies have been evaluated.

19. True or false: Lightning strike victims typically suffer extensive burns?

False. The *crispy critter* phenomenon is largely untrue. Victims do not burst into flame and become reduced to a pile of ashes. Of the lightning strike victims who have burns, only 5% have deep or significant burns. Lightning most often flashes over a victim with few, if any, burns. Victims who experience burns typically demonstrate linear burns, punctuate burns, feather burns (Lichtenberg's Flowers), or thermal burns.

20. Which organ systems can be damaged by lightning?

See Table 55-3.

21. True or false: A lightning strike is always fatal?

False. Lightning is a surprisingly inefficient killer. The mortality may be as low as 10% to 30%. However, lightning is the third most frequent cause of death caused by natural phenomena (after floods and extremes of temperature).

22. True or false: If a victim of lightning is not killed outright, he or she will likely be fine?

False. The majority of victims suffer some sequelae. These complications can include neurologic, psychiatric, cardiac, pulmonary, otic, ophthalmologic, and musculoskeletal disorders (see Question 20).

23. What are the best ways to reduce lightning risk?

- Avoiding the following: thunderstorms, being the *tallest target*, holding a *lightning rod*, touching conductors.
- Seeking shelter indoors or in a car.
- Staying away from groups (especially people who know CPR; they are your potential rescuers).
- Holding feet together and crouching down to reduce your stride potential.

24. What's the 30-30 rule?

It is a lightning safety recommendation. If you see lightning and cannot count to 30 before hearing thunder, you are in danger and should seek shelter. This is based on the *flash-to-bang* method of determining your distance from a lightning strike: the time between the

TABLE 55-3. LIGHTNING INJURIES BY ORGAN SYSTEM

System	Injury
Cardiac	Asystole, ventricular fibrillation (usually from hypoxia), nonspecific dysrhythmias, myocardial stunning, nonspecific ST-T changes, ST elevation, T wave inversion, hypertension, tachycardia, acute myocardial infarction (rare)
Respiratory	Apnea from inhibition of the brainstem respiratory centers, hypoxia, pulmonary contusions and hemorrhage, hemothorax, pneumothorax
Nervous	Loss of consciousness, coma, transient aphasia, confusion, disorientation, amnesia, autonomic dysfunction (with loss of pupillary function), coagulation of brain substance, epidural and subdural hematomas, intraventricular hemorrhage, skull fractures, seizures, transient or permanent paralysis, EEG abnormalities, extrapyramidal symptoms, sensory disturbances, SIADH, cerebral edema, ataxia, vertigo, cerebral artery thrombosis, spinal atrophic paralysis, headaches
Psychiatric	Hysteria, phobias, psychosis, depression, posttraumatic stress disorder, "storm apprehension," memory impairment, personality changes
Skin	Feathering, linear burns, punctate burns, true thermal burns
Musculoskeletal	Dislocations, fractures, muscle necrosis (rare)
Renal	Myoglobinuria (rare)
Gastrointestinal	Gastric atony, ileus, perforations (uncommon)
Ophthalmologic	Mydriasis, loss of light reflex, anisocoria, Horner's syndrome, cataracts (in 20% of victims within 3 years)
Otologic	Tinnitus, hearing loss, ruptured tympanic membranes

EEG, electroencephalographic; SIADH, syndrome of inappropriate antidiuretic hormone.

lightning flash and the thunderclap in seconds divided by five is the distance in miles. Further, you shouldn't resume outdoor activities until at least 30 minutes after the last flash of lightning is seen and the last clap of thunder is heard. "*If you see it, flee it; if you hear it, clear it.*"

25. I want to appear as if I read this whole chapter. Is there one number to memorize that will impress my friends and colleagues?

Sure, try 70%:

- Thunderstorms that happen between noon and 6 pm: approximately 70%
- Thunderstorms that happen in June, July, or August: 70% (June, 21%; July, 30%; August, 22%)
- Lightning victims who survive: approximately 70% (although this may be as high as 90%)
- Survivors with sequelae: approximately 70%
- Singular victims: approximately 70% (fatal singular victims as high as 91%; victims in couples, 15%; victims in groups of three or more, 15%)

ELECTRICAL INJURIES

26. What are the basic physics of electricity?

Electricity is simply the flow of electrons. The electromotive force moving those electrons from high concentration to low concentration is voltage (V). The number of electrons flowing is known as current or amperage (I). Resistance to the electron flow (R) is a property of the medium through which they pass, measured in ohms (O). These three factors are related as shown in Ohm's law: $I = V/R$.

27. What factors determine nature and severity of the injury seen in electrical accidents?

The most harmful effects of electricity on the body are thermal. The heat generated is related to the current, tissue resistance, and duration of contact:

$$\text{Heat} = (\text{amperage})^2 \times \text{resistance} \times \text{time}$$

Because amperage is squared, it contributes the most to tissue injury. However, in real accidents, the amperage is often unknown. Therefore, we use voltage as an approximate indicator because high voltages are usually associated with high amperages. Voltages are classified as:

- High = 1000 volts or more
- Low = less than 1000 volts

KEY POINTS: DIFFERENCES IN LIGHTNING AND HIGH-VOLTAGE ELECTRICAL INJURIES

1. Duration: Instantaneous (lightning) versus prolonged contact (electrical)
2. Energy: 1 billion volts (lightning) versus 1,000 to 10,000 volts (electrical)
3. Current: DC (lightning) versus AC (electrical)
4. Shock wave: Present in thunder (lightning) versus absent (electrical)
5. Flashover: Present (lightning) versus absent (electrical)
6. Cardiac effect: Asystole (lightning) versus ventricular fibrillation (electrical)
7. Burns: Minor and superficial (lightning) versus deep (may appear benign or superficial) (electrical)
8. Urinary failure: Rare (lightning) versus myoglobinuric (electrical)
9. Fasciotomy: Rarely indicated (lightning) versus common and early (electrical)

28. What is the epidemiology of electrical injuries?

There are about 800 to 1000 injuries from high-voltage electricity per year in the United States. Between 3% and 4% of burn center admissions are for electric burns. Serious electrical burns carry a 40% mortality. Electrical injuries follow a bimodal age distribution with a peak in children younger than 6 years and another peak around age 20. The first peak represents children exploring electrical cords, wall sockets, or extension cord outlets. This second peak represents occupational accidents (one third of them are electric workers, and one third are construction workers). In fact, electrocution is the fifth leading cause of occupational death. See Table 55-4.

TABLE 55-4. ELECTRICITY GLOSSARY

Threshold for Sensation	In contradistinction to DC, in which there is an unchanging direction of current flow, AC is an electric source that changes its direction of current flow as a particular frequency. Human tissues, particularly muscles, respond well to AC frequencies between 40 and 150 Hz (cycles per second). In the United States, homes are supplied with exactly that: household current is 60 Hz, 110 to 120 volts. The threshold for sensation is the minimum current that is perceptible, about 1 to 4 mA.
"Let Go" Threshold	"Let go" threshold is the current below which an individual can release their grip on the electricity source. The average child has a "let go" threshold of approximately 3 to 5 mA. The average adult has a "let go" threshold of approximately 6 to 9 mA. Between 8 and 22 mA, AC can cause tetanic muscle contractions to the degree that one may not be able to let go of the electric source. The resulting phenomenon is dangerous because it prolongs the time of contact, thus producing more injury.
Ventricular Fibrillation Threshold	This is the current at which ventricular fibrillation can be induced. The ventricular fibrillation threshold (50–100 mA) is not much higher than the "let go" threshold.

AC, alternating current; DC, direct current.

29. What are some prehospital considerations in the treatment of electrical injuries?

Scene safety for the rescuers is the first priority. Rescuers should not become victims—even if that means taking time to turn off the power source before approaching a victim. The essentials of resuscitation are the ABCs (airway, breathing, circulation). It is prudent to assume traumatic injuries and immobilize the spine. Cardiac dysrhythmias are treated in the standard manner. Two large-bore intravenous (IV) catheters should be established, and aggressive fluid replacement should be started.

30. How is tissue resistance related to injury?

Nerves offer the least resistance and, therefore, allow the deepest penetration but the least heat injury. Blood vessels have the next most resistance, then mucous membranes, muscle, skin, tendon, and fat. The tissue with the greatest resistance is bone. Because of its high resistance, it suffers the greatest heat injury but the least penetration.

Interestingly, the resistance of skin can be highly variable, depending on its thickness, vascularity, and degree of hydration. The thick, dry skin of callused feet and hands is much more resistant (100,000 ohms) than thin, wet skin (2,500 ohms). Immersion in water further drops skin resistance to 1,500 ohms. When high-resistance skin is injured by electricity, the heat produces burns. As the skin chars, it loses resistance and allows for greater penetration.

31. Which organ systems can be damaged by electrical injury?

See Table 55-5.

TABLE 55-5. ORGANS DAMAGED BY ELECTRICAL INJURY

System	Injury
Skin	Thermal burns such as entrance and exit wounds (typically in AC burns, the entrance and exit burns look similar; in DC burns, the entrance is smaller than the exit), flexor crease burns, mouth commissure burns (risk of delayed bleeding from labial artery when the eschar separates)
Cardiac (the most frequent causes of immediate death from electrical injury)	Cardiac arrest from asystole (DC) or ventricular fibrillation (AC), atrial and ventricular ectopy, atrial fibrillation, first-degree and second-degree heart blocks, bundle-branch blocks, and QT interval prolongation, nonspecific ST-changes (common), acute myocardial infarction (rare)
Vascular	Hemorrhage, arterial and venous thrombosis, ischemia, progressive necrosis (from "skip" lesions in which the current, presumably, skips from traveling within the blood column to traveling within the wall of the vessel)
Nervous	CNS or peripheral; immediate or delayed, loss of consciousness, amnesia, confusion, disorientation, concentration and memory problems, apnea or respiratory depression, seizures, paralysis, paresthesias, motor nerves more commonly injured than sensory, poor rate of recovery
Musculoskeletal	Muscular pain, muscle necrosis (rhabdomyolysis), compartment syndrome, tendon rupture, dislocations (one of the few mechanisms for posterior shoulder dislocations), fractures, "electroporation" or the formation of cell membrane pores in bone, aseptic necrosis, periosteal burns, "bone pearls" (osteo schisis)
Respiratory	Inhibition of the brainstem respiratory centers
Gastrointestinal	Hollow visceral and solid visceral injury (both rare), stress ulcers
Renal	Acute tubular necrosis (from rhabdomyolysis and myoglobinuria), renal failure, hyperkalemia, hypocalcemia, hyperglycemia, acidosis
Ophthalmologic	Cataracts (6–24 months after the incident), corneal burns, intraocular hemorrhage and thrombosis, retinal edema, retinal detachment, uveitis, optic nerve atrophy
Pregnancy	Spontaneous abortion (fetal death rate is 73%), oligohydramnios, IUGR (amniotic fluid and fetal tissues conduct 200 times more than dry, intact adult skin)

AC, alternating current; CNS, central nervous system; DC, direct current; IUGR, intrauterine growth retardation.

32. My patient is a 24-year-old, 75-kg man who was working on a high-voltage line when he received a shock. He has a burn on his palm where it contacted a wire and another burn on his knee where it was in contact with a ladder. These two burns together are about 2% of his total body surface area (TBSA). How much IV fluid should he receive?

Traditional burn formulas for calculating volume repletion, such as the Parkland formula (4 mL × weight in kg × %BSA), are not applicable to electrical injuries because the surface damage does not reflect the degree of deeper tissue damage. The surface burn is just the "tip of the iceberg." For this reason, electrical injuries correlate more with crush injuries than they do with thermal burns. IV fluids should be administered at a rate to ensure a urine output of 1 to 2 mL/kg/h (for this patient 75–150 mL of urine per hour). The objective of early, aggressive fluid resuscitation is to prevent renal failure secondary to rhabdomyolysis.

33. When should I include a computed tomography (CT) of the brain in my work-up?

A brain CT scan should be considered as part of the diagnostic procedure in every case of high-voltage electrical injury presenting with signs of central nervous system (CNS) involvement during or after the event. Due to the low resistance of neural tissue, subarachnoid hemorrhage needs to be ruled out, as well as other sources of traumatic intracranial hemorrhage.

34. True or false: One should treat high-voltage electrical victims as one would lightning victims?

False. Lightning and high-voltage electrical injuries can have very different effects and require different treatment approaches. For example, high-voltage injuries often produce deep burns that may require fluid resuscitation and even fasciotomy. These victims often have renal failure from myoglobinuria. If they are in cardiac arrest, it is often ventricular fibrillation. In contrast, lightning victims rarely have deep burns that need fluid resuscitation and fasciotomy. Their kidneys are rarely affected. If the lightning strike victim is in cardiac arrest, it is typically asystole (unless he or she is also hypoxic).

35. What medications should I consider using for lightning and electrical injuries?

There are no specific pharmacotherapeutics for lightning injuries. Patients with electrical injuries and rhabdomyolysis with myoglobinuria may benefit from forced diuresis with mannitol or furosemide. Consideration should also be given to the use of sodium bicarbonate for urine alkinalization because this may increase myoglobin clearance. Finally, nonsteroidal anti-inflammatory agents have been shown to be effective in treating electrical injuries and may be indicated in lightning injuries as well (although this has not been studied).

36. What should be the disposition of lightning and electrical victims?

All patients with cardiac abnormalities (including abnormal electrocardiograms [ECGs]), neurologic findings, or significant burns require hospital admission. Current guidelines recommend 24-hour telemetry monitoring for all patients with high-voltage injuries and for patients with low-voltage injuries who have an abnormal initial ECG. The American Burn Association burn unit referral criteria include any electrical burns and lightning injuries. Patients with associated trauma or burns may need admission, depending on the extent of the injury. All others may be discharged. It is prudent to arrange for ophthalmology follow-up in 6 months and otolaryngology and psychiatry follow-up, as needed.

37. What about children who get injured by a household electrical cord or appliance?

In contrast to adults who typically sustain electrical injuries in the workplace, children often sustain electrical injuries in the home; these injuries are usually associated with electrical cords (60%–70%) and wall outlets (15%–20%). Healthy children exposed to common household currents (120–240V) without any water contact, who are asymptomatic at ED presentation and without a ventricular dysrhythmia or cardiac arrest in the field, are at very low risk for developing cardiac dysrhythmias. Patients with a normal initial ECG do not

develop late dysrhythmias. Those with nonfatal dysrhythmias or nonspecific ECG abnormalities typically resolve spontaneously within 24 hours. As such, literature supports safely discharging these patients without an initial ECG evaluation or inpatient cardiac monitoring after a common household current exposure.

KEY POINTS: LIGHTNING AND ELECTRICAL INJURIES

1. Rescuers should not become victims.
2. Assume the presence of occult trauma and remember the tip of the iceberg phenomenon.
3. Typically, only high-voltage electric victims need aggressive hydration.
4. Lightning MCI priority goes to the dead, (i.e., reverse triage).

38. Who gets admitted, and who goes home?

See Table 55-6.

WEBSITES

American Burn Association: www.ameriburn.org/

Lightning Injury Research Program: www.uic.edu/labs/lightninginjury/

Lightning and Atmospheric Electricity Research at GHCC: http://thunder.msfc.nasa.gov/primer/

National Lightning Safety Institute: www.lightningsafety.com/

Table 55-6. CRITERIA FOR ADMISSION AND DISCHARGE

Admit (Consider Burn or Trauma Center)	Discharge*
Major burns, circumferential burns, significant hand burns	Asymptomatic low-voltage injury
Patients requiring CPR	No ECG findings
High voltage, especially transthoracic path	No significant burns
Abnormal ECG, dysrhythmias	*Ophthalmology follow-up in 6 months
Loss of consciousness	*ENT, psychiatry follow-up as needed
Neurologic abnormalities	
Hypoxia, myoglobinuria	
Mouth commissure burns (because of problems with feeding and bleeding from the labial artery)	
OB consultation is recommended for all pregnant patients	

CPR, cardiopulmonary resuscitation; ECG, electrocardiogram; ENT, ear, nose, and throat; OB, obstetric.
*If discharged, these follow-up arrangements should be provided.

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SUBMERSION INCIDENTS

Jedd Roe, MD, MBA, FACEP

CHAPTER 56

1. Define terms associated with submersion accidents.

Traditional nomenclature applied to submersion incidents has included the following:

- **Drowning** is death by suffocation from submersion in liquid.
- **Near-drowning** is survival (at least temporarily) after a submersion event.
- **Immersion syndrome** is sudden death after submersion in very cold water, probably secondary to vagal nerve mediated asystolic cardiac arrest.
- **Wet drownings** are those in which aspiration of water occurred during the event; 80% to 90% of drownings are classified as wet drownings.
- **Dry drownings** are those in which asphyxia is caused by laryngospasm without aspiration.

In an effort to simplify classification, the use of the term ***submersion incident*** has been adopted to encompass any adverse event sustained by a patient as a result of submersion in a liquid.

2. How many people drown each year?

Each year in the United States, more than 8,000 people die from drowning (an estimated 500,000 worldwide). It is the third leading cause of accidental death in all ages. Drowning is the fourth leading cause of accidental death, and an estimated 50,000 persons annually survive a submersion event.

3. Who drowns and why?

The incidence of drowning peaks in two groups—teenagers and toddlers. In **teenagers** (ages 15–24), nearly 80% of drowning and submersion victims are male. Teenage boys are victims because of risk-taking behavior during swimming, boating, diving, or other water-related activities. Alcohol is a contributing factor in more than 60% of all teenage drownings.

Of all drowning victims, 40% are younger than 4 years. **Toddlers** are at risk because of their inherently inquisitive nature and their physical inability to extricate themselves from hazards such as pools, buckets, tubs, toilets, or washers. Inadequate supervision, even for brief moments, is the primary cause of drowning in toddlers. One always must consider the possibility of abuse when evaluating a child drowning victim because inflicted submersions account for 1.5% to 8% of all events for children younger than age 5. An estimated 59% of drownings in people younger than 1 year of age occur in bathtubs, and 56% of these are a result of child abuse.

Other risk factors in all age groups are as follows:

- Inability to swim
- Seizures
- Trauma
- Ethanol
- Hyperventilation
- Substance abuse
- Hot tubs/spas
- Hypothermia
- Cardiovascular or cerebrovascular disease
- Child abuse/neglect
- Diabetes
- Suicide

In the United States, 50,000 new pools are added annually to the 4.5 million pools that already exist. The increasing prevalence of hot tubs, pleasure craft, and outdoor sports has increased greatly the number of people at risk of drowning. Of drownings, 90% occur tantalizingly close to safety, within 10 yards.

4. What kills a drowning victim?

Historically, emphasis has been placed, incorrectly, on the significance of drowning in salt water versus fresh water because of presumed differences in the pathophysiology of the aspirated water. In fresh-water aspirations, the hypotonic fluid was thought to diffuse into the circulation, increasing blood volume and decreasing the concentration of serum electrolytes. This also causes a loss of surfactant and results in alveolar collapse. Sea water was thought to pull fluid into the alveoli, decreasing the blood volume and increasing the electrolyte concentrations. This transudated fluid would cause a pathologic effect on pulmonary alveolar membranes, leading to noncardiogenic pulmonary edema. In fact, such pathologic changes have rarely been seen in patients who have survived to hospital arrival. It has been suggested that a person must ingest 22 mL/kg to cause electrolyte changes, and it is unusual for submersion victims take in more than 3 to 4 mL/kg.

Of submersion victims, 10% to 20% have not aspirated water, and most victims of submersion do not aspirate enough fluid to cause a significant alteration in blood volume or electrolytes or a life-threatening pulmonary shunt secondary to perfusion of fluid-filled alveoli. Death is most often the result of asphyxia caused by laryngospasm and glottis closure. Although this mechanism is less common, more successful resuscitations (80%–90% of all patients) occur in this group of patients. The aspirated water is a significant pulmonary irritant and contaminant that may increase intrapulmonary shunting, resulting in hypoxemia.

5. What happens in a drowning?

The first event is an unexpected or prolonged submersion. The victim begins to struggle and panic. Fatigue begins and air hunger develops. Reflex inspiration ultimately overrides breath holding. The victim swallows water, and aspiration occurs, causing laryngospasm that may last for several minutes. Hypoxemia worsens, and unconsciousness ensues. If the victim is not rescued and resuscitated promptly, central nervous system damage begins within minutes.

6. Describe the presenting symptoms of near-drowning victims.

The presenting pulmonary symptoms are varied. The patient may be completely asymptomatic, have a mild cough, show mild dyspnea and tachypnea, or be in fulminant pulmonary edema. The clinical spectrum of central nervous system findings may range from confusion or lethargy to coma. Some patients may be found in cardiac arrest.

7. What is the pulmonary pathophysiology?

The central clinical feature of *all* submersion incidents is hypoxemia caused by laryngospasm or aspiration. The PO_2 decreases; the PCO_2 increases, and there is a combined respiratory and metabolic acidosis. If the patient is successfully resuscitated, the recovery phase often is complicated by aspirated water or vomitus. Aspiration can cause airway obstruction by particulates, bronchospasm by direct irritation, acute respiratory distress syndrome (ARDS) due to pulmonary edema from parenchymal damage, atelectasis from loss of surfactant, and pulmonary bacterial infections. Some patients may later develop pulmonary abscesses or empyema.

8. How is the cardiac system affected in drowning?

Cardiac decompensation and dysrhythmias are caused by hypoxemia and complicated by the ensuing acidosis. The heart is relatively resistant to hypoxic injury, and successful resumption of cardiac activity is common, but severe central nervous system damage often occurs. Response of the heart to therapy, particularly antiarrhythmic medications, may be limited by hypoxia, acidosis, and hypothermia. Primary therapy is aimed at reversal of these three problems.

9. What is the prehospital treatment?

The most important part of treatment of a near-drowning victim is delivered in the prehospital phase with immediate resuscitation. If a submersion victim has appropriate airway management and ventilation is rapidly established, anoxic brain injury is avoided, and prompt and full recovery is anticipated. The patient without rapid airway management and ventilation suffers irreversible anoxic brain injury and either is unresponsive to resuscitation or has a progressively deteriorating course after initial resuscitation. Therapy must correct hypoxia, associated acidosis, and hypotension as rapidly as possible. Establish a patent airway using appropriate cervical spine precautions if indicated because diving injuries often are associated with cervical spine injury. Apply a nonrebreather oxygen mask to patients with spontaneous respirations. Initiate bag-mask breathing or endotracheal intubation if indicated. Correct hypoxia and acidosis by hyperventilation with 100% oxygen. Intravenous (IV) access is needed.

NOTE: There is no convincing evidence for the effectiveness of postural drainage maneuvers, and their use is not recommended.

10. When is endotracheal intubation indicated?

Any person with altered mentation or an inability to protect the airway needs intubation. In the initially stable patient, an inability to maintain a pO_2 greater than 60 to 90 mm Hg with high flow oxygen by nonrebreather mask indicates that extensive pulmonary compromise or ARDS may exist, and early airway management with positive-pressure ventilation and positive end-expiratory pressure is appropriate to decrease intrapulmonary shunting.

One important point is to determine if the submersion event may have occurred as a result of diving into water. In these cases, the patient may have sustained a cervical spine injury, and appropriate precautions should be taken with in-line stabilization of the neck during intubation.

11. If aspiration is suspected, what treatment is needed?

Pulmonary treatment is supportive. Close observation for signs of a developing pulmonary infection or ARDS is needed. Some cases with significant aspiration may require bronchoscopy to remove particulate matter and tenacious secretions. Bronchodilator therapy with β-agonists is appropriate if bronchospasm is evident.

12. Does a normal chest radiograph rule out pulmonary injury?

No. A normal chest radiograph may be seen in 20% of cases early in their course. Typical X-ray findings include perihilar infiltrates and pulmonary edema, although these classic descriptors of ARDS (noncardiogenic pulmonary edema) may take hours to develop.

13. Is there a role for prophylactic antibiotics?

When contaminated water is involved (e.g., sewage), prophylactic antibiotics may be considered. In all other instances, prophylactic antibiotics are of no proven benefit.

14. Is there an indication for the use of sodium bicarbonate during resuscitation?

No. Respiratory and metabolic acidosis should be treated by mechanical ventilation and hyperventilation.

15. Discuss the approach to patients with a decreased level of consciousness or coma.

Hypoxic injury leads to cerebral edema and a concomitant rise in intracranial pressure. Although there was initial enthusiasm for treatment of presumed elevated intracranial pressure with the usual modalities of muscle paralysis, hyperventilation, mannitol, barbiturate coma, hypothermia, and steroids, more recent studies have shown no improvement in outcome with these therapies. Supportive care is the mainstay of therapy. Be attentive to the possibility of cranial or spinal injuries in all boating or diving injuries in patients with altered level of consciousness. Do not forget the possibility of suicide or child abuse. If the history is in doubt, assume a cranial and a cervical injury. The possibility of toxicologic conditions also should be investigated with appropriate toxicologic screens performed.

16. Are glucocorticoids, barbiturate coma, or induced hypothermia indicated?

In the case of glucocorticoids and barbiturate coma, *no*. These therapies are unproven and remain controversial. However, therapeutic hypothermia has recently been shown to be of benefit in cardiac arrest, and case reports have suggested similar outcomes for victims of submersion. In fact, Hein and colleagues reported on twin toddlers who suffered submersion incidents, and the patient treated with induced hypothermia recovered without neurologic deficit.

Investigators are also evaluating surfactant therapy as a new treatment for acute respiratory failure secondary to submersion. Cubattoli and colleagues reported on two submersion patients in acute respiratory failure who underwent bronchoscopy and instillation of surfactant into both lungs with marked physiologic improvement.

17. What is unique about cold-water submersion?

Cases in which victims of prolonged submersion in cold water have been resuscitated successfully without apparent neurologic sequelae are reported occasionally. The number remains small, however. Sudden submersion in cold water theoretically induces the mammalian diving reflex, in which blood is shunted from the periphery to the central core. The induced hypothermia causes a decrease in metabolic demand, reducing potential hypoxic injury from prolonged asphyxia. Cold water does have potentially deleterious effects. Most significant are the induced cardiac irritability from hypothermia, exhaustion, and altered mental status. Resuscitation of hypothermic near-drowning victims should be continued until patients are adequately rewarmed or to the level required for therapeutic hypothermia (see Chapter 57).

18. When should resuscitative efforts be withheld?

Generally, all patients should receive initial resuscitative efforts. One child recovered successfully from a 66-minute submersion in cold water, and other studies have reported that patients requiring cardiopulmonary resuscitation (CPR) in the field may make a full recovery. When their core temperature has normalized and therapeutic efforts remain unsuccessful, patients can be pronounced dead.

19. What is the disposition of a submersion victim?

All submersion victims with cardiac arrest deserve aggressive in-hospital resuscitation until all reasonable efforts prove futile and the patient is near normothermic. All other submersion victims require close observation. Some respiratory complications of drowning are delayed in presentation and usually appear within 8 hours. A patient with any respiratory complaints or symptoms, chest radiograph abnormalities, or a demonstrated oxygen requirement should be monitored closely in a hospital for at least 24 hours. Similarly, any patient who received resuscitative efforts or had a reported loss of consciousness, cyanosis, or apnea should be monitored closely. Patients without any symptoms and completely normal evaluation may be discharged after 6 to 8 hours of observation with instructions to return immediately if respiratory distress ensues.

KEY POINTS: SUBMERSION INCIDENTS

1. Toddlers and teenagers are most at risk for death due to submersion.
2. Prehospital treatment is critical and directed at correcting underlying hypoxia.
3. A normal chest radiograph does not rule out pulmonary injury.
4. All asymptomatic victims of submersion must be observed at least 6 to 8 hours prior to discharge.
5. Many submersion incidents are preventable.

20. What are the most important factors in estimating prognosis?

The most important factor in determining outcome is the patient's response to resuscitation as measured by serial neurologic examinations. Poor prognostic factors include:

- A Glasgow Coma Scale score less than or equal to 5
- Prolonged submersion (>5 minutes)
- Delay in initiating CPR
- pH less than 7.0
- Water temperatures of greater than 10°C (77°F)
- Asystole on arrival to the ED

Patients who arrive aware and alert have a 100% complete neurologic recovery, whereas 95% of arousable patients with altered mentation have a complete neurologic recovery.

Szpilman has proposed a clinical classification based on the analysis of 1,831 cases of submersion seen in Brazil over a 19-year period. The classification is based on clinical findings in the field, and the mortalities are shown in Table 56-1.

21. Can we prevent submersion incidents?

Many of the factors contributing to death by submersion *are* preventable and can be directed at those groups at risk, particularly children. Efforts include:

- Fencing of private and public swimming pools
- The use of personal flotation devices
- Improving supervision of infants and young children near water
- Increasing public knowledge of the risks of the day's water conditions
- Understanding the limitations of personal health conditions
- Stressing the separation of alcohol from water-related activities.

WEBSITE

Drowning: http://emedicine.medscape.com/article/772753-overview

TABLE 56-1. SZPILMAN CLASSIFICATION OF NEAR-DROWNING AND DROWNING

Grade	Clinical Findings	Mortality Rate (%)
1	Normal pulmonary auscultation ± cough	0
2	Rales or crackles in some lung fields	0.6
3	Crackles in all fields without hypotension	5.2
4	Crackles in all fields with hypotension	19.4
5	Respiratory arrest without cardiac arrest	44
6	Cardiopulmonary arrest	93

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HYPOTHERMIA AND FROSTBITE

Daniel F. Danzl, MD, and Mary Nan Mallory, MD

CHAPTER 57

HYPOTHERMIA

1. What is accidental hypothermia?

An unintentional decrease in core temperature to less than 35°C (95°F). The preoptic anterior hypothalamus normally maintains a diurnal temperature variation within 1°C.

2. What factors are important in the epidemiology of hypothermia?

Primary accidental hypothermia results from direct exposure to the cold. Secondary hypothermia is a natural complication of many systemic disorders, including sepsis, cancer, and trauma. The mortality of secondary hypothermia is much higher. Although outdoor exposure is common, many elderly victims are found indoors.

3. How is body temperature normally regulated?

The normal physiology of temperature regulation is activated by cold exposure, producing reflex vasoconstriction and stimulating the hypothalamic nuclei. Heat preservation mechanisms include shivering, autonomic and endocrinologic responses, and adaptive behavioral responses. Although acclimatization to heat stress is efficient, humans can't acclimate to a *three-dog night.*

KEY POINTS: COMMON MECHANISMS OF HEAT LOSS

1. Radiation
2. Conduction
3. Convection
4. Respiration
5. Evaporation

4. Describe the common findings in mild, moderate, and severe hypothermia.

- **Mild hypothermia** (32.2°C–35°C [90°F–95°F]) depresses the central nervous system and increases the metabolic rate, pulse, and amount of shivering thermogenesis. Dysarthria, amnesia, ataxia, and apathy are common findings.
- **Moderate hypothermia** (27°–C32.2°C [80°F–90°F]) progressively depresses the level of consciousness and the vital signs. Shivering is extinguished, and dysrhythmias commonly develop. The QT interval is prolonged, and a J wave (Osborn wave) may appear at the junction of the QRS complex and ST segment. Patients become poikilothermic and cannot rewarm spontaneously. A cold diuresis results from an initial central hypervolemia, which is caused by the peripheral vasoconstriction.
- **Severe hypothermia** ($<$ 27°C [80°F]) results in coma and areflexia with profoundly depressed vital signs. Carbon dioxide production decreases 50% for each 8°C fall in temperature; there is little respiratory stimulation.

5. What three factors predispose to hypothermia?

- Decreased heat production
- Increased heat loss
- Impaired thermoregulation

6. What decreases heat production?

Decreased heat production is common:

- At the age extremes
- With inadequate stored fuel
- With endocrinologic or neuromuscular inefficiency

Neonates are poorly adapted for cold, even without being subjected to emergent deliveries and resuscitations. The elderly have progressively impaired thermal perception. Anything from simple hypoglycemia to more severe malnutrition represents a threat to the core temperature. Examples of endocrinologic failure include myxedema, hypopituitarism, and hypoadrenalism.

7. What are the common causes of increased heat loss?

Increased heat loss results mainly from exposure or dermatologic problems that interfere with the skin's integrity. Iatrogenic causes include emergency childbirth, cold infusions, and heatstroke treatment.

8. How is thermoregulation impaired?

Impairment is via central, peripheral, metabolic, or pharmacologic mechanisms. A variety of central nervous system processes affect hypothalamic function. Traumatic or neoplastic lesions and degenerative processes induce hypothermia. Acute spinal cord transection extinguishes peripheral vasoconstriction, which prevents heat conservation. The abnormal plasma osmolality common with metabolic derangements, including diabetic ketoacidosis and uremia, is an additional cause. Innumerable medications and toxins can impair central thermoregulation when present in either therapeutic or toxic doses.

9. When should hypothermia be suspected?

The diagnosis is simple when a history of exposure is obvious. The history may not be available or helpful, however, and subtle presentations are far more common in urban areas. Ataxia and dysarthria may mimic a cerebrovascular accident or intoxication. The only safe way to avoid missing the diagnosis is to routinely measure the patient's temperature.

10. Are there decoys that confuse the physical examination?

If there is tachycardia disproportionate for the temperature, suspect hypoglycemia, an overdose, or hypovolemia. Most patients with vasodilation require volume administration. Hyperventilation during moderate or severe hypothermia suggests a central nervous system lesion or one of the systemic acidoses, such as diabetic ketoacidosis or lactic acidosis. A cold-induced rectus spasm and ileus may mask or mimic an acute abdomen. Suspect an overdose, ETOH intoxication, or central nervous system insult whenever the decreased level of consciousness is not consistent with the temperature.

11. What options are available to measure the core temperature?

Rectal, esophageal, tympanic, and bladder sites can be measured. The rectal temperature may lag or be falsely low if the probe is in cold feces. Esophageal temperature is falsely elevated during heated inhalation. The reliability of tympanic measurements is unclear.

12. How does temperature depression affect the hematologic evaluation of patients?

Anemia is masked because the hematocrit increases 2% per 1°C drop in temperature. Do not rely on leukocytosis to predict sepsis because the leukocytes often are sequestered.

There are no safe predictors of values. The increased viscosity seen with cold hemaglutination often results in either thrombosis or hemolysis, and a type of disseminated intravascular coagulation syndrome can occur. Coagulopathies are not reflected by the deceptively normal international normalized ratio because this test is done routinely on blood rewarmed to 37°C.

13. Should arterial blood gases be corrected for temperature?

No. Correction implies acidosis is beneficial. An uncorrected pH of 7.4 and pCO_2 of 40 mm Hg confirm acid-base balance at all temperatures.

14. What is the key decision regarding rewarming?

The primary initial decision is whether to rewarm the patient passively or actively. Passive rewarming is noninvasive and involves simply covering the patient in a warm environment. This technique is ideal for previously healthy patients with mild hypothermia.

15. What conditions mandate active rewarming?

- Cardiovascular instability
- Temperature $<$ 32.2°C (90°F)
- Age extremes
- Neurologic or endocrinologic insufficiency

16. What is core temperature afterdrop?

The commonly observed continued drop in core temperature after initiation of rewarming. There are two causes:

- Temperature equilibration between tissues
- The circulatory return of cold peripheral blood to the core

17. Are there unique considerations with active external rewarming?

The external transfer of heat to a patient is accomplished most safely when the heat is applied directly to the trunk. In chronically hypothermic patients, rapidly rewarming the vasoconstricted extremities may overwhelm a depressed cardiovascular system and result in cardiovascular collapse. Forced heated air rewarming blankets and circulating water blankets are commonly used. Monitoring in a heated tub can be difficult, and vasoconstricted skin is burned easily by electric blankets.

18. What constitutes active core rewarming?

Techniques that deliver heat directly to the core. Options include heated inhalation, heated infusion, lavage, and extracorporeal rewarming.

19. When is airway rewarming indicated?

Heated, humidified oxygen can be administered via mask or endotracheal tube. Heat transfer is not as significant by mask, but respiratory heat loss is eliminated while the patient is rewarmed gradually.

20. What are the techniques for heated irrigation?

Heat transfer from irrigation of the gastrointestinal tract is minimal. Irrigation should be considered only in severe cases and in combination with other techniques. Thoracostomy tube irrigation with two tubes is a more efficient alternative in severe cases. Intravenous (IV) fluids heated to 40°C to 42°C are particularly helpful during major volume resuscitations.

21. When should heated peritoneal lavage be considered?

Double-catheter peritoneal lavage can efficiently rewarm seriously hypothermic patients. This invasive technique generally should be reserved for severely hypothermic and unstable patients, or in patients with certain overdoses. Infuse 2 L of isotonic dialysate at 40°C to 45°C, and suction after 20 minutes dwell time.

22. When is extracorporeal rewarming indicated?
Cardiopulmonary bypass, continuous arteriovenous and venovenous rewarming, and hemodialysis can be life-saving in cardiac arrest situations. Patients with completely frozen extremities, severe rhabdomyolysis, and major electrolyte fluxes are also easier to manage in this manner.

23. What are the contraindications to cardiopulmonary resuscitation (CPR) in accidental hypothermia?
CPR should be initiated unless do-not-resuscitate status is verified, lethal injuries are identified, no signs of life are present, or the chest wall is frozen and cannot be compressed. Because a profoundly hypothermic patient may appear dead, and because vital signs may be difficult to obtain, a cardiac monitor should be applied for 30 to 45 seconds to ensure that there are no signs of life.

24. Are there unique pharmacologic considerations during hypothermia?
Protein binding increases as body temperature drops, and most drugs become ineffective. Pharmacologic manipulation of the pulse and blood pressure generally should be avoided.

25. What is the significance of atrial and ventricular dysrhythmias?
Atrial dysrhythmias normally have a slow ventricular response. They are innocent and should be left untreated. Pre-existent ventricular ectopy may resurface during rewarming and confuse the picture. Ventricular dysrhythmia treatment is problematic because the cold heart may be unresponsive to cardiovascular agents. If the patient is in ventricular fibrillation, only one defibrillation attempt (2 J/kg) is indicated until the core temperature exceeds 30°C to 32°C.

FROSTBITE

26. What is frostbite?
Frostbite is the most common freezing injury of tissue. It occurs whenever the tissue temperature decreases to less than 0°C. Ice crystal formation damages the cellular architecture, and stasis progresses to microvascular thrombosis.

27. Which factors predispose to frostbite?
Tissue rapidly freezes when in contact with good thermal conductors, including metal, water, and volatiles. Direct exposure to cold wind (wind-chill index) quickly freezes acral areas (e.g., fingers, toes, ears, nose). A variety of conditions can impair the peripheral circulation and predispose to frostbite. Constrictive clothing and immobility reduce heat delivery to the distal tissues. Vasoconstrictive medications, including nicotine, can exacerbate cold damage, especially when coupled with underlying vascular conditions, such as atherosclerosis.

28. What peripheral circulatory changes precede frostbite?
Humans possess a *life-versus-limb* mechanism that helps prevent systemic hypothermia. Arteriovenous anastomoses in the skin shunt blood away from acral areas to limit radiative heat loss.

29. Before frostbite occurs, what other cutaneous events take place in the prefreeze phase?
As tissue temperatures decrease to less than 10°C, anesthesia develops. Endothelial cells leak plasma, and microvascular vasoconstriction occurs. Crystallization is not seen as long as the deeper tissues conduct and radiate heat.

30. What happens during the freeze phase of frostbite?
The type of exposure determines the rate and location of ice crystal formation. Usually, ice initially forms extracellularly, causing water to exit the cell and inducing cellular dehydration hyperosmolality, collapse, and death.

31. Immediately after thawing, what may occur?

In deep frostbite, progressive microvascular collapse develops. Sludging, stasis, and cessation of flow begin in the capillaries and progress to the venules and the arterioles. The tissues are deprived of oxygen and nutrients. Plasma leakage and arteriovenous shunting increase tissue pressures and result in thrombosis, ischemia, and necrosis.

32. What is progressive dermal ischemia?

This is an additional insult to potentially viable tissue that is partially mediated by thromboxane. Arachidonic acid breakdown products are released from underlying damaged tissue into the blister fluid. The prostaglandins and thromboxanes produce platelet aggregation and vasoconstriction.

33. What delayed physiologic events occur?

Edema progresses for 2 to 3 days. As the edema resolves, early necrosis becomes apparent if nonviable tissue is present. Final demarcation often is delayed for more than 60 to 90 days. Hence the aphorism, "Frostbite in January, amputate in July."

34. What are the symptoms of frostbite?

Sensory deficits are always present, affecting light touch, pain, and temperature perception. *Frostnip* produces only a transient numbness and tingling. This is not true frostbite because there is no tissue destruction. In severe cases, patients report a "chunk of wood" sensation and clumsiness.

35. What imaging techniques might help assess frostbite severity?

Routine radiography at presentation and later at 4 to 10 weeks post-injury may demonstrate specific abnormalities. Scintigraphy may predict tissue loss and monitor the efficacy of treatment. Magnetic resonance angiography can also predict tissue demarcation.

36. What is chilblain (PERNIO)?

Repetitive exposure to dry cold can induce chilblain (cold sores), especially in young women. Pruritus, erythema, and mild edema may evolve into plaques, blue nodules, and ulcerations. The face and dorsa of the hands and feet are commonly affected.

37. What is trench foot?

Prolonged exposure to wet cold above freezing results in trench foot (immersion foot). Initially, the feet appear edematous, cold, and cyanotic. The subsequent development of vesiculation may mimic frostbite. Liquefaction gangrene is a more common sequela, however, with trench foot than with frostbite.

38. How should frostbite be classified?

Classification by degrees as is done with burns is unnecessary and is often prognostically incorrect. Superficial or mild frostbite does not result in actual tissue loss; deep or severe frostbite does.

39. What do the various signs of frostbite indicate?

The initial presentation of frostbite can be deceptively benign. Frozen tissues appear yellow, waxy, mottled, or violaceous-white. Favorable signs include normal sensation, warmth, and color after thawing. Early clear bleb formation is more favorable than delayed hemorrhagic blebs. These result from damage to the subdermal vascular plexi. Lack of edema formation also suggests major tissue damage.

40. How should frozen tissues be thawed?

Rapid, complete thawing by immersion in 40°C to 41°C circulating water is ideal. Reestablishment of perfusion is intensely painful, and parenteral narcotics are needed in severe cases. Premature termination of thawing is a common mistake because an incomplete thaw increases tissue loss. Never use dry heat or allow tissues to refreeze. Rubbing or friction massage may be harmful.

KEY POINTS: COMMON SEQUELAE OF FROSTBITE

1. Paresthesias
2. Hyperhidrosis
3. Thermal misperception
4. Epiphyseal damage
5. Nail deformities

41. What steps should immediately follow thawing?

- Handle tissues gently, and elevate the injured parts to minimize edema formation.
- If cyanosis is still present after thawing, monitor the tissue compartment pressures.
- Consider streptococcal and tetanus prophylaxis.
- Avoid compressive dressings, and use daily whirlpool hydrotherapy.
- Consider phenoxybenzamine (α-blocker that reduces vasoconstriction) in severe cases.
- Whenever possible, defer surgical decisions regarding amputation until clear demarcation is demonstrated.
- Magnetic resonance angiography may predict demarcation earlier than clinical demarcation.

42. How are blisters treated?

Clear blisters may temporarily be left intact or sterilely aspirated. After debridement, apply antibiotic ointment or a specific thromboxane inhibitor, topical aloe vera. When coupled with systemic ibuprofen, this strategy can minimize accumulation of arachidonic acid breakdown products. In contrast, hemorrhagic blisters should be left intact to prevent tissue desiccation.

43. Are any ancillary treatment modalities really helpful?

A variety of vasodilatory treatment regimens, including medical and surgical sympathectomies, dextran, heparin, and a variety of anti-inflammatory agents, do not conclusively increase tissue salvage. In select cases, with less than 6 hours of warm ischemia time, thrombolytic therapy may decrease the need for amputation.

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HEAT ILLNESS

Christopher B. Colwell, MD, and Gina Soriya, MD

CHAPTER 58

1. How does the body regulate temperature?

The hypothalamus controls thermoregulation. The posterior region functions to conserve heat and the preoptic region is involved in heat dissipation. This is a critical function as human body systems operate under a narrow range of temperatures.

2. What are the four mechanisms for cooling the body?

- **Conduction**: the transfer of thermal energy between objects in direct contact with each other due to a temperature gradient; heat moves from the warmer to the cooler object, equalizing the temperature difference.
- **Convection**: heat transfer in a gas or liquid by the circulation of currents from one region to another.
- **Radiation**: energy emitted by one body travels through a medium or space to be absorbed by another body.
- **Evaporation**: molecules in a liquid state spontaneously become gaseous, such as sweat into the ambient air.

3. Which mechanism is the most effective for heat loss?

Evaporation is the most effective means of cooling the body.

4. How does the relative humidity of the atmosphere affect the normal body mechanisms of cooling?

The moisture gradient has to be such that the air is dryer than the body. As humidity rises, evaporation becomes less effective. Heat is removed from the body at a slower rate, causing greater heat retention.

5. How does heat cause damage to the body?

Heat is directly toxic to cells, causing protein denaturation, as well as breakdown of cellular membranes and nuclei, leading to cell apoptosis and necrosis. Stress from heat causes the release of several inflammatory cytokines, which can precipitate a severe systemic inflammatory response. In addition, heat directly injures the vascular endothelium, causing increased vascular permeability, activation of the coagulation cascade, and disseminated intravascular coagulation. Heat also may accelerate biochemical reactions, which in turn may cause metabolic abnormalities.

Temperatures above 41.6°C (106.9°F) are considered to be above the critical thermal maximum for humans and can cause cellular damage within hours of exposure. Temperatures above 49°C (120°F) cause nearly immediate cell death and necrosis. Lower temperatures over longer periods of time can cause the same degree of damage as higher temperatures over shorter periods of time.

6. List the spectrum of heat illnesses and briefly describe.

- Heat edema—transient swelling of hands, feet, and ankles due to water retention in a nonacclimated person
- Heat rash—prickly heat; maculo-papular rash caused by excessive sweating and blockage of the sweat ducts; primarily occurs on parts of the body covered by tight clothing

- Heat cramps—painful involuntary spasms of large muscle groups, usually occurring after strenuous exercise
- Heat tetany—occurs in the setting of heat stress with hyperventilation; presents with carpopedal spasms, perioral and peripheral paresthesias
- Heat syncope—dehydration from intense sweating, followed by peripheral vasodilation and decreased motor tone
- Heat exhaustion—excessive dehydration and electrolyte depletion; precursor to heat stroke
- Heat stroke—life-threatening condition in which the body loses its ability to control its temperature

Some have advocated for describing heat illness in basically two categories: heat stroke and heat exhaustion (with heat exhaustion essentially including everything that does not qualify as heat stroke).

7. How are heat cramps treated?

Heat cramps are treated with oral salt solutions or intravenous saline. Passive stretching of the affected muscles may also help relieve the cramps.

8. What are the symptoms of heat syncope and how is it treated?

Patients with heat syncope have a sudden onset of lightheadedness or fainting in the setting of high temperature. Physical examination usually reveals tachycardia, and skin is pale, cool, and sweaty. Patients may or may not have orthostatic vital signs. An important aspect of heat syncope is that any alteration in mentation resolves immediately after the event. If there is any sustained altered mental status, the patient is suffering from heat stroke. Treatment for heat syncope consists of rest in a cool environment, as well as oral or intravenous fluid replacement.

9. What are the symptoms of heat exhaustion and how is it treated?

Patients with heat exhaustion complain of flu-like symptoms, weakness, thirst, nausea/vomiting, sweating. Their heart rate may be normal or tachycardic. Body temperature is usually normal, but will not exceed 40°C (104°F). Patients should be removed from the warm environment and hydrated. Hydration can occur by either the oral route (if the patient can tolerate this) or intravenously. The most important aspect of treating heat exhaustion is ensuring that it does not progress to heat stoke. Remember that a patient's perception of temperature does not always correlate with core temperature.

10. What is heat stroke?

Heat stroke is a life-threatening medical emergency. It is characterized by an elevated core body temperature ($>$40°C), accompanied by mental status changes and varying degrees of organ dysfunction. The patient may or may not be sweating. Signs and symptoms may include confusion, seizures, coma, acute renal failure, respiratory failure, liver failure, disseminated intravascular coagulation, or ischemic bowel.

11. Describe the two types of heat stroke and how they are differentiated.

- **Exertional** heat stroke is usually observed in athletes, laborers, military, or other young healthy individuals involved in strenuous physical activity who may not be acclimatized to their environment. The thermoregulatory system functions normally but becomes overwhelmed.
- **Nonexertional**, or classic, heat stroke occurs in individuals who are exposed to excessive temperatures and who are unable to alter their environment. People that are particularly vulnerable to this condition include the poor, the elderly, infants, alcoholics, patients with chronic medical conditions, or patients that do not have access to cooling mechanisms such as fans or air conditioning.

12. What laboratory abnormalities would be expected in patients with heat illness?

Laboratory abnormalities may include acute renal failure, hemoconcentration, elevated liver function tests, rhabdomyolysis, hyper/hyponatremia, hyper- or hypokalemia, leukocytosis, and

disseminated intravascular coagulation. Renal failure, rhabdomyolysis, and disseminated intravascular coagulation are more common in victims of exertional heat stroke than in victims of classic heat stroke.

13. What is the treatment of heat stroke?

Core body temperature should be monitored continuously via rectal thermometer. The patient should be rapidly cooled, with a goal temperature of <38.9°C (102°F) within 30 minutes of presentation to minimize organ damage. You cannot cool too quickly. Evaporative cooling methods are the safest and may involve continually wetting the patient with tepid water while the skin is fanned, allowing for continuous airflow over moist skin. Cold water is not necessary and may induce shivering and vasoconstriction. A cooling blanket may be utilized, or ice packs applied to the axillae, neck, and groin. Emersion in ice water baths is also an effective way of cooling patients, although not practical in all settings. Invasive cooling techniques, such as iced gastric or peritoneal lavage, have been studied but do not have a clear advantage over evaporative cooling.

Along with rapid cooling, supportive care should be initiated. Intubation may be necessary if the patient is unable to protect their airway. Hemodynamics and urine output should be monitored carefully, with intravenous fluids and electrolyte replacement. Although patients with heat illness are almost uniformly dehydrated, over-resuscitation should be avoided.

14. Are any medications indicated in the treatment of heat stroke?

Benzodiazepines may be used to control shivering. Antipyretic agents such as acetaminophen or salicylic acid have not been shown to be beneficial. Dantrolene, which is used to treat malignant hyperthermia, is not effective in the treatment of heat stroke.

15. What are the risk factors that can lower the threshold for heat stroke?

Risk factors include age (infants, young children, and the elderly), inability to care for oneself, alcohol abuse, obesity, dehydration, non-acclimatization, and chronic medical conditions and medications (such as β-blockers) that can limit the ability to respond to changes in temperature.

16. What is the differential diagnosis for hyperthermia with central nervous system dysfunction?

- Acute infection, including sepsis and meningitis
- Status epilepticus
- Serotonin syndrome
- Neuroleptic malignant syndrome
- Drugs, such as cocaine, alcohol, caffeine, diuretics, psychotropics, and phenothiazines
- Thyroid storm
- Stroke

17. What is the mortality rate associated with heat stroke?

Mortality rate varies depending on a number of factors: age, underlying comorbidities, and most importantly the degree and duration of hyperthermia. In addition, the setting of heat stroke affects the mortality rate. Mortality rates can exceed 50%. Poor prognostic indicators include advanced age, hypotension, and respiratory failure requiring intubation. Aggressive treatment may reduce the mortality, particularly in patients with exertional heat stroke.

18. What about prevention?

Acclimatization is the key. The process can take a week or more and involves limited activity during that period of time. Acclimatization allows sweating to increase and proteins to change to better tolerate heat. It is important to resist the temptation to participate in strenuous activity early in a heat wave and allow the acclimatization process to take effect.

KEY POINTS: HEAT ILLNESS

1. Heat stroke is life-threatening and is defined by altered mental status. Heat stroke victims may or may not be sweating.
2. You cannot cool a patient too quickly.
3. Evaporative cooling is quick, safe, and effective.

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ALTITUDE ILLNESS AND DYSBARISMS

CHAPTER 59

Jeffrey Druck, MD, and Christopher Davis, MD

1. What are the three disease states that comprise high altitude illness?

High altitude illness is comprised of three distinct clinical entities:

- Acute mountain sickness (AMS)
- High-altitude pulmonary edema (HAPE)
- High-altitude cerebral edema (HACE)

2. What are the symptoms of AMS?

AMS is defined as the presence of a headache in the setting of recent ascent to high altitude with one of the following additional complaints: anorexia, nausea, vomiting, fatigue, weakness, dizziness, lightheadedness, or difficulty sleeping. It is an entirely clinical definition.

3. How quickly do symptoms of AMS develop? What elevation is the minimum elevation at which one can see AMS?

Usually, symptoms begin within 6 to 10 hours of ascent. The minimum elevation at which AMS has been documented is 2000 m (6562 ft).

4. How do I treat AMS?

AMS is usually a self-limited disease that resolves with acclimatization (usually within 1–2 days); the real concern is for further progression to HACE or development of HAPE with further ascent. Treatment is tailored to the severity of the symptoms and includes descent, acetazolamide (250 mg twice a day), supplemental oxygen, and dexamethasone.

KEY POINTS: TREATMENT FOR ALL TYPES OF HIGH-ALTITUDE ILLNESS

1. Descent (best treatment)
2. Supplemental oxygen
3. Hyperbaric therapy

5. What is the number-one risk factor for AMS?

Age. Although controversial, in one study, subjects younger than 25 years were 2.6 times *more* likely to develop AMS than those older than 55 years. However, studies on young children show no increased risk. Additional predisposing factors associated with AMS are as follows:

- Rate of ascent
- Exertion on arrival
- Elevation attained
- Previous history of AMS
- Duration of stay at altitude
- Cold temperatures

A recent study demonstrates that the hyperventilatory capacity (oxygen saturation after 1 minute of hyperventilation) may be directly related to one's risk of developing AMS.

6. Is there any treatment that will prevent AMS?

The easiest way to prevent AMS is by minimizing risk. Slow ascent, avoidance of sedatives (alcohol included), and decreased physical activity on arrival minimize risk. Data support using either acetazolamide at 125 mg twice a day starting the day prior to ascent and continuing for 2 days at maximum altitude, or dexamethasone at 4 mg twice a day for the same time period. The data on *Gingko biloba* for the treatment of AMS remain controversial. However, recent studies show that varying purities in Gingko biloba likely contribute to the variability in efficacy.

KEY POINTS: PREVENTION FOR ALL TYPES OF HIGH-ALTITUDE ILLNESS

1. Slow ascent
2. Limitation of exertion
3. Avoidance of sedatives
4. Acetazolamide or dexamethasone

7. What is the definition of HACE?

HACE is a clinical diagnosis defined by a change in consciousness and associated ataxia in the setting of ascent to altitude. Cerebral edema is often present on computed tomography (CT) scans, and death results from brain herniation.

8. When does HACE occur?

The onset of HACE usually occurs 3 to 5 days after arrival to elevation.

9. What is the treatment for HACE?

Treatment includes immediate descent, supplemental oxygen, and dexamethasone. If descent is not an option, hyperbaric therapy (simulating descent) is a possibility; other modalities, such as diuretics or acetazolamide, are untested and are of unproven benefit.

10. Is there anything that will prevent HACE?

Because HACE is thought to be the end point on the spectrum of altitude illness, prevention strategies for AMS are the same for HACE.

11. What is HAPE?

HAPE is defined as two of the following symptoms in the setting of a recent gain in altitude:

- Dyspnea at rest
- Cough
- Weakness or decreased exercise tolerance
- Chest tightness or congestion *and* two of the following signs:
  - Crackles or wheezing in at least one lung field
  - Central cyanosis
  - Tachypnea
  - Tachycardia

HAPE is thought to be noncardiogenic pulmonary edema resulting from failure of the alveolar-capillary barrier. Cold stress and exertion increase pulmonary arterial pressure, which contributes to an increase in pulmonary edema.

12. When does HAPE occur?

Usually, HAPE occurs within 1 to 3 days of arrival at altitude and is rare beyond 4 days.

13. How do I treat HAPE?

Descent, supplemental oxygen, and hyperbaric therapy are the mainstays of treatment. Temporizing measures aimed at decreasing pulmonary artery pressures (nifedipine and expiratory positive airway pressure masks) have been shown to help, but the recovery period from HAPE is measured in days, so definitive therapy such as descent is highly recommended. β-agonists that promote alveolar fluid clearance may also be beneficial. Other vasodilators such as nitric oxide, tadalafil, and sildenafil are currently being researched and show promise. In contrast with HACE and AMS, dexamethasone has no effect.

14. Is there any preventative therapy for HAPE?

Nifedipine has been shown to decrease the recurrence of HAPE in patients with previous HAPE. New data suggest acetazolamide does not prevent HAPE, although further studies are ongoing. In general, the principles designed to decrease the incidence of AMS (slow rate of ascent, decreased activity at altitude, no sedatives) are also true for HAPE.

15. Do you ever see HAPE, HACE, or AMS at the same time?

HACE is thought to be *end-stage* AMS, so you do not see both at the same time. HACE often occurs in association with HAPE, but you can see HAPE without any signs of AMS or HACE.

16. Which form of altitude illness is most common? Which is most deadly?

The incidence of altitude illnesses depends on the altitude achieved in the group studied:

- AMS: Incidence, 15% to 70% (most common); mortality rate, 0%
- HACE: Incidence, 1% to 2%; mortality rate unknown because of usually coexistent HAPE
- HAPE: Incidence: 1% to 15%; mortality rate, as high as 44% (most deadly)

17. What is dysbarism?

Dysbarism refers to pressure-related diseases but is commonly limited to diseases resulting from diving injuries (underwater pressure changes). This category includes diseases related specifically to pressure changes and their physical effects (e.g., middle ear barotrauma [MEBT], pneumothorax, arterial gas embolism, pneumomediastinum, and barosinusitis) as well as disease related to *bubble formation* (e.g., pulmonary decompression sickness, spinal decompression sickness, and the *bends*).

18. How much pressure does a diver experience at 33 ft underwater?

Each 33 ft, or 10 m, is equivalent to 1 atmosphere. Because sea level is equivalent to 1 atmosphere, 33 ft underwater is 2 atmospheres, which is equal to 29.4 psi or 1,520 mm Hg.

19. What are the bends?

The *bends*, also known as caisson's disease (named after caisson workers, who work in pressurized underwater chambers), is one of the more common forms of dysbarism. It occurs when nitrogen comes out of solution and forms bubbles in tissues, causing muscle and joint pain.

20. When would you see someone with the bends?

People experience the bends when they ascend too rapidly from scuba diving.

21. Why would nitrogen *precipitate* in tissues?

According to Boyle's law of gases, pressure is inversely proportional to volume ($P_1V_1 = P_2V_2$). Add into this mixture Henry's law that states that the amount of gas in solution is directly proportional to the partial pressure of that gas. Thus, with increased pressure underwater, the volume of gas decreases, and the amount of gas in solution (dissolved) increases. However, with rapid ascent, gas will expand and come out of solution, resulting in increased gas bubble

size and possible precipitation in tissues. With a slow ascent, the gradual increase in bubble size and slow change in amount of gas in solution allow the gases to remain dissolved in circulating blood and expelled through the respiratory system.

22. What is nitrogen narcosis?

As stated previously, the amount of each gas that goes into solution in the blood increases with increased pressure (or increased depth, because increased depth causes increased pressure). With nitrogen being the largest component of air, a large amount of nitrogen goes into solution in the blood, ever increasing with increasing pressure. This high concentration of nitrogen causes an anesthetic-like effect that causes lack of motor control and inappropriate behavior, and eventually causes unconsciousness. Nitrogen narcosis usually is seen at depths of 100 ft or more. To avoid nitrogen narcosis, alternative mixtures containing decreased nitrogen are recommended for dives greater than 100 ft.

23. What is MEBT?

MEBT occurs when the pressure of the water on the tympanic membrane during descent is not equalized by the eustachian tube. Usually, a diver will mechanically increase the pressure in his or her middle ear by forcing air through the eustachian tube to equilibrate the pressure across the tympanic membrane; if this does not occur, the increased external pressure will cause pain until rupture of the tympanic membrane eventually occurs, which may cause severe vertigo.

24. How would one get a pneumothorax with ascent?

If a diver held his or her breath to go underwater to 33 ft (2 atm), the volume in the lungs would decrease to half the prior volume (1 atm $\times$ normal lung volume $=$ 2 atm $\times$ ½ normal lung volume). If he or she is scuba diving and replaces that lung volume back to normal, with ascent, he or she could double the lung volume (2 atm $\times$ normal lung volume $=$ 1 atm $\times$ 2 normal lung volume). If there is nowhere for this additional gas to escape (i.e., breath holding), the lung may rupture, causing a pneumothorax to develop.

25. What is arterial gas embolism (AGE)?

This condition occurs when expanding gas ruptures an alveolus and the gas is forced into the pulmonary vasculature. The gas is then distributed through the arterial system, with typical symptoms of loss of consciousness, apnea, and cardiac arrest. It is the second most common cause of diving-related deaths.

26. What about the movies that show people bleeding from their eyes when diving? Does that really happen?

With typical diving masks, an artificial air space is created in front of the eyes. When a diver descends, this air space is subject to the same gas laws as the diver, with the volume of air in the mask decreased by one half at 1 atmosphere underwater (effectively 2 atmospheres), one third at an effective 3 atmospheres, and so forth. This pressure change creates a vacuum effect in the mask, which can cause petechial hemorrhage, subconjunctival hemorrhage, and even optic nerve damage, termed *facial barotrauma*. The usual way divers avoid this problem is by wearing a mask that encompasses their nose and then equalizing the pressure by blowing air into their mask.

27. What is decompression sickness (DCS)?

This term that describes the diseases that occur when gas (usually nitrogen) precipitates out of solution. The earliest form of DCS is the bends, the disease that presents as limb and joint pain. Prior thought was that the bends resulted from gas precipitation within joints themselves, but further research has shown that the gas distension occurs along ligaments and tendon sheaths. Other components of DCS include pulmonary DCS ("the chokes"), skin DCS (skinny bends), and spinal cord DCS. Type I DCS includes skin and musculoskeletal symptoms; type II DCS includes all other symptoms.

KEY POINTS: TYPES OF DCS

1. Type I: Skin DCS (skinny bends) and musculoskeletal DCS (the bends)
2. Type II: Pulmonary DCS (the chokes), spinal cord DCS, and CNS DCS

28. What are the chokes?
The chokes is the common term for pulmonary DCS. Pulmonary DCS manifests as cough, shortness of breath, and chest pain resulting from massive venous gas embolism, which enters into the pulmonary artery.

29. What are the skinny bends?
Skinny bends refers to cutaneous DCS, which is the appearance of a diffuse, reticulated, blotchy rash caused by endothelial damage from bubbles, resulting in blood extravasation. It can also refer to a syndrome of cutaneous itching that only appears in the artificial environment of a hyperbaric chamber.

30. What is spinal cord DCS?
Spinal cord DCS is a syndrome characterized by ascending paresthesias and paralysis resulting from venous outflow obstruction by venous gas emboli in the epidural plexus of the spinal cord.

31. Is there a central nervous system (CNS) form of DCS?
Yes. It commonly presents with headache, blurred vision, dysarthria, diplopia, and inappropriate behavior. The exact mechanism of CNS DCS is poorly characterized; there was some thought that it was caused by venous gas embolism going across a patent foramen ovale (PFO), but the incidence of PFOs in patients with CNS DCS is equivalent to the general population.

32. How do you tell the difference between CNS DCS and AGE?
One main point is that loss of consciousness is uncommon with CNS DCS. However, because both are treated the same way, there is little use in distinguishing between the two.

33. How are dysbarisms treated?
In general, DCS and AGE should be treated with immediate recompression in a hyperbaric oxygen chamber. The longer the delay to treatment, the higher the morbidity and mortality. Acute pressure-related injuries (e.g., pneumothorax, pneumomediastinum) should be treated with standard therapy, whereas tympanic membrane rupture and inner ear disturbances should be referred to an otolaryngologist. Facial barotrauma victims should be assessed for more serious injuries, but there usually is no further treatment needed. The best way to treat dysbaric injuries is to prevent them from occurring in the first place. Some data suggest immediate oxygen therapy may result in better outcomes.

34. Is there anything that makes a particular person susceptible to DCS?
Although controversial, there are data that increased age is a risk factor for DCS. Also, inexperience is linked to an increased incidence in DCS.

35. Is there anything that I can do to reduce my risk of DCS?
Slow ascent is the key. There are some data that show a decrease in the incidence of DCS in a rat model with exercise 20 hours prior to diving, but further research needs to be done. Air mixtures such as *nitrox*, a nitrogen and oxygen gas mixture with greater than 21% oxygen, or *heliox*, a mixture of helium and oxygen, also show promise.

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EVALUATION OF FEVER IN CHILDREN YOUNGER THAN AGE THREE

Genie E. Roosevelt, MD, MPH

1. **What is fever?**
   Fever is generally defined as a rectal temperature of 38.0°C (100.4°F). Be aware that parents often consider a fever below the 38.0°C mark, as when parents say, "She had a fever of 99.2°."

2. **How should temperature be measured in infants and young children?**
   For infants from birth up to 3 months of age, the most reasonable and accurate method is the rectal temperature. Tympanic temperatures are appropriate in children older than 3 months of age. Oral temperatures are generally not attempted in young children for obvious logistical reasons. Axillary temperatures are unreliable and should not be used despite the ease with which they may be obtained.

3. **Is it safe to measure temperatures rectally?**
   Many parents, and even health care providers, are anxious about doing this. British studies investigating safety and efficacy demonstrate an extremely low risk of injury.

4. **What is a serious bacterial infection (SBI)?**
   SBI includes:
   - Bacteremia
   - Urinary tract infection (UTI)
   - Bacterial meningitis
   - Pneumonia (established by a focal infiltrate on chest X-ray)

5. **Does it matter *how much* fever the child has?**
   Hyperpyrexia (temperature of 40.5°C) has been associated with higher rates of SBI (4%) in patients 3 to 36 months of age. However, any child who appears toxic should be evaluated for SBI regardless of the temperature.

6. **What is meant by *toxic appearing*?**
   Toxic children may be pale, lethargic, or limp. They may show evidence of poor perfusion (such as cyanosis or peripheral vasoconstriction with mottling) or changes in respiratory drive such as tachypnea or shallow breathing. They may fail to interact with their environment (as evidenced by poor or absent eye contact, poor feeding, or failure to respond to caregivers or objects in their view). These children are generally very ill, requiring immediate resuscitation and evaluation.

KEY POINTS: SIGNS OF TOXICITY

1. Lethargy
2. Cyanosis
3. Tachypnea
4. Poor tone
5. Failure to respond to caregivers

7. Which antipyretics work best for children?

Studies show that acetaminophen (15 mg/kg) and ibuprofen (10 mg/kg) have similar efficacy, and both work well for getting febrile children to defervesce.

Note: Most children's elixirs contain half the amount of an adult tablet per teaspoon (5 mL). For example, an adult tablet of ibuprofen contains 200 mg, whereas the children's elixir contains 100 mg/5 mL.

8. What is the most common cause of antipyretic failure?

Underdosing, either by dose or by schedule. Parents may not know the child's weight, fail to calculate an appropriate dose, or be unfamiliar with units of measure (such as a *teaspoon*). It is also common for parents to believe that antipyretics should *cure* the fever, and they may complain, "I gave her the medicine and it helped for a while, but the fever just came right back." Parental education and provision of an oral syringe frequently help with this issue.

9. What is wrong with baby aspirin; it is for babies, right?

Aspirin administration to children with viral infections has been associated with Reye's syndrome (encephalopathy and acute liver failure). This syndrome, although rare, carries a high mortality rate (20% to 40%). Although some pediatric conditions (such as juvenile rheumatoid arthritis and Kawasaki disease) may involve treatment with aspirin, its use in children with fever of unclear etiology should be strictly avoided.

10. Is there any good reason *not* to treat a fever?

No. Children with fever feel crummy, feed poorly, and worry their caregivers; the quickest way to make them feel better is to bring the fever down.

11. What are febrile seizures?

Febrile seizures are the most common seizure disorder seen in children (incidence 4%) and are typically associated with high fevers. There has been a great deal of discussion about whether these seizures are associated with rate of rise, rather than absolute temperature, but there is no evidence to support this theory. There is no increased risk of SBI in patients with simple febrile seizures as compared to children who present with fever alone. A simple febrile seizure is characterized as a generalized tonic clonic seizure that does not recur in a 24-hour period, is associated with fever, lasts longer than 15 minutes, and occurs in an otherwise neurologically normal child between the ages of 3 months and 5 years. There is excellent long-term data that shows no effect on cognitive development or intelligence. Although benign, simple febrile seizures are very frightening for parents.

12. Does careful administration of antipyretics prevent recurrence of febrile seizures?

No. Placebo-controlled trials with antipyretics show no difference in recurrence rates during subsequent febrile illnesses. About one third of patients will have a second febrile seizure. Risk factors for recurrence include family history, age (younger age at presentation more likely to recur), and height of temperature (lower temperature at presentation more likely to recur). Studies also show no benefit to diazepam prophylaxis to prevent recurrence in patients with simple febrile seizures during subsequent febrile illnesses.

13. How should tiny babies with fever be evaluated?

Febrile infants (temperature of 38.0°C) younger than 1 month should receive a full sepsis work-up:

- Urinalysis and culture (by catheterization or suprapubic aspiration)
- Complete blood count and culture
- Lumbar puncture (LP)
- Chest radiograph (only with respiratory symptoms)
- Stool analysis for white blood cell (WBC) count and culture (if there is a history of diarrhea)

Infants should receive intravenous (IV) antibiotics and be admitted to the hospital.

Note: Age cut-offs for fever evaluation are based on **gestational age**, not age since birth. This means that a 32-week premature infant born 6 weeks ago is still considered to be younger than 1 month.

14. What happens after the magic 1-month mark?

For infants 1 to 3 months of age with a fever without a source, risk stratification is recommended. See algorithm in Figure 60-1.

15. What about older infants and young children?

For children 3 to 36 months with a fever without a source, follow the algorithm in Figure 60-2 (note different temperature cut-off for this older age group).

16. How do we decide when to do a LP in older babies and young children?

A LP should be performed in any child who appears toxic or has signs of meningitis. Be aware that many of the classic signs of meningitis (e.g., Brudzinski's sign, Kernig's sign, neck stiffness, or bulging fontanelle) are frequently absent and unreliable in young children.

17. What if the child has a fever source or one is found during the work-up?

It depends on the source and the age group. A viral source often makes the risk for bacteremia and meningitis low. However, a bacterial source may predispose to these entities and should be strongly considered in patients younger than 3 months of age. If an identified source *completely* explains the clinical presentation, stop looking and treat it. If not, complete the evaluation as previously described. Be aware that urinary tract infections, and occasionally bacteremia, may coexist with viral infections respiratory infections such as bronchiolitis and gastroenteritis.

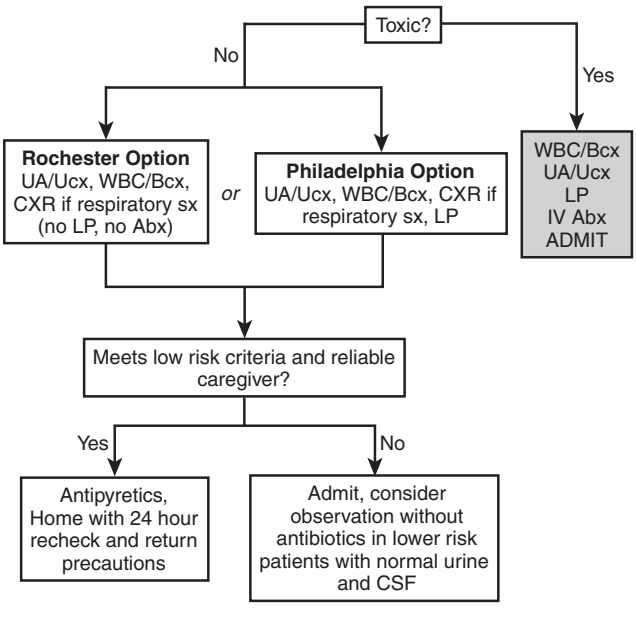

Figure 60-1. Algorithm for infants 1–3 months of age. Abx, antibiotics; Bcx, blood culture; CXR, chest X-ray; LP, lumbar puncture; UA, urinalysis; Ucx, urine culture; WBC, white blood cell count.

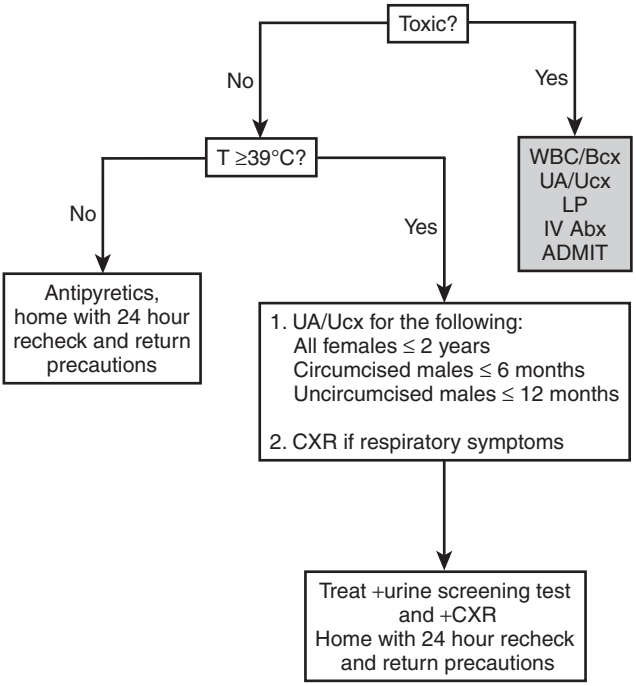

Figure 60-2. Algorithm for children 3–36 months of age. Abx, antibiotics; Bcx, blood culture; CXR, chest X-ray; LP, lumbar puncture; UA, urinalysis; Ucx, urine culture; WBC, white blood cell count.

18. Must we always follow the guidelines, or is there room for clinical judgment in there somewhere?

A study of more than 3,000 febrile infants seen by almost 600 pediatricians throughout the United States demonstrated that selective testing by experienced clinicians in office-based practice was as effective in appropriately identifying and treating SBI as rigid adherence to clinical guidelines. Their findings suggest that if close follow-up is feasible, experienced clinicians may use clinical judgment in select cases rather than published recommendations in their management strategy of febrile infants. Unfortunately the ED physician may not have the luxury of this close follow-up.

19. What if the child looks great; can he or she go home?

Nontoxic-appearing children older than 1 month who meet low-risk criteria may be discharged home with return precautions and close follow-up. This, of course, presumes they have a reliable caregiver, a tenable social situation, and reasonable access to transportation.

20. What are low-risk criteria?

The two sets of low-risk criteria used most often are the Rochester and Philadelphia criteria. Both presume the child is previously healthy and well appearing at the time of evaluation.

Rochester criteria

- WBC between 5,000 and 15,000 with absolute band count (ABC) = 1,500
- Urine WBC < 10/high power field (hpf)
- Stool WBC <5/hpf (in infants with diarrhea)
- Note: No LP performed

Philadelphia criteria

- WBC < 15,000 with band-to-neutrophil ratio <0.2
- Urine WBC < 10/hpf and no bacteria on Gram stain
- Cerebrospinal fluid (CSF) WBC < 8/hpf
- Negative CSF Gram stain
- Stool and chest radiograph negative (if obtained)

21. What is the risk of occult bacteremia (bacteremia without a focal infection)?

Although occult bacteremia usually resolves spontaneously, it may lead to localized infections such as meningitis, pneumonia, or osteomyelitis. Evidence gathered since the widespread use of the heptavalent pneumococcal vaccine suggests that the current rate of occult bacteremia is less than 1% in well-appearing febrile children between the ages of 3 months to 3 years. This risk is low enough that routine blood cultures are no longer recommended for children older than 3 months.

22. What antibiotic should be used for empiric coverage of bacteremia?

Younger than 28 days

- Cefotaxime, 50 mg/kg IV (may consider substituting with Gentamicin 2.5 mg/kg intravenously [IV] in first week of life to treat Group B Strep)

plus

- Ampicillin, 50 mg/kg IV (to cover for Listeria)

28 days to 3 months

- Cefotaxime, 50 mg/kg IV

3 to 36 months

- Ceftriaxone 50 mg/kg IV or intramuscularly ([IM] 24-hour dosing) *or* Cefotaxime, 50 mg/kg IV

23. Which infants should receive acyclovir?

Risk factors for neonatal Herpes simplex virus (HSV) infection include:

- Maternal HSV infection at delivery (although two thirds are asymptomatic)
- Maternal history of HSV or other sexually transmitted diseases
- Vesicular rash
- Seizures
- CSF pleocytosis (20–100 WBCs is typical)

Patients are usually less than 22 days of age. Patients with risk factors should be started on acyclovir pending a HSV polymerase chain reaction (PCR) test from the CSF. Clinical judgment should be used in the decision to discontinue acyclovir given that the initial PCR may be a false-negative. The dose is 20 mg/kg IV every 8 hours (dose and schedule adjusted for gestational age and renal impairment).

24. What about children with fever and rapidly progressive petechial rash?

This is disseminated meningococcemia until proved otherwise. Patients with this type of infection may progress very rapidly. The LP may be deferred until the patient is stable and should not delay antibiotic administration. Dosages below ensure coverage for invasive, resistant pneumococcal meningitis which also may cause purpura fulminans.

- Cefotaxime, 75 mg/kg IV *immediately*
- Vancomycin, 15 mg/kg IV *immediately*

WEBSITE

Pediatric febrile seizures. www.emedicine.com/EMERG/ topic376.htm

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SEIZURES IN INFANCY AND CHILDHOOD

Andrew M. White, MD, PhD

CHAPTER 61

1. How does one determine if an event in a child is actually a seizure?

Many events that appear to be seizures are actually non-epileptic. These events can be classified by the age in which the event occurred and are given in Figure 61-1.

Questions helpful in deciding whether an event was or was not a seizure include:

- Is the movement suppressible? Seizure movements will continue despite someone holding a limb. Tics or stereotypies (a repetitive or ritualistic movement, posture, or utterance) are suppressible.
- Is the event distractible? Seizures will continue if you call a person's name; daydreaming will not.
- Were the movements jerking or thrashing? Typically seizures involve rhythmic jerking and not thrashing movements or pelvic thrusting. It is always good to have a witness demonstrate the movements.
- Are the events always provoked? Seizures are generally not provoked; breath holding spells are.
- Is there tongue biting during the event and, if so, where? During a true seizure the tongue is usually bitten on the side. During a nonepileptic spell, the tongue may be bitten on the tip.
- Was there urination or loss of stool? This can happen during nonepileptic spells, but this favors a diagnosis of true seizure.
- Does it only happen during exercise? Cardiac events are more likely to occur during exercise.
- What do the eyes do during the seizure? During most seizures, the eyes remain open.
- Does it happen only during certain times of day? Usually seizures will happen at all times of day, but there are certain types that are much more frequent in sleep (benign rolandic) and other that are more frequent during waking hours (benign infantile partial seizures)
- Does it only happen upon standing? Likely to be orthostatic syncope.
- What is the child like following the event? Except for absence-type seizures, children are post-ictal following seizures.
- Does the child retain consciousness during the event? If it is a generalized tonic-clonic seizure, this is impossible as both halves of the brain are involved. It *is* possible for simple seizures.
- Does the child remember the event? Typically the child will not remember the entire complex partial seizure or any part of a generalized tonic-clonic seizure.

2. What can be explained about the child's history?

- Get a step-by-step description of the events before, during, and following the seizure.
- What the child was doing prior to the seizure can help differentiate between seizure and breath holding.
- A good description of the seizure itself, including the manner in which it started and evolved, how long it lasted (this is usually significantly overestimated by the parents), and what the patient was like after the event are all useful pieces of information.

Additional questions should be asked to try to establish the cause of the seizure. These include questions with regard to illnesses, trauma, and development. The patients' past medical history should be obtained, as well as a family medical history of seizures.

In an epilepsy patient, inquiries about medication compliance are necessary.

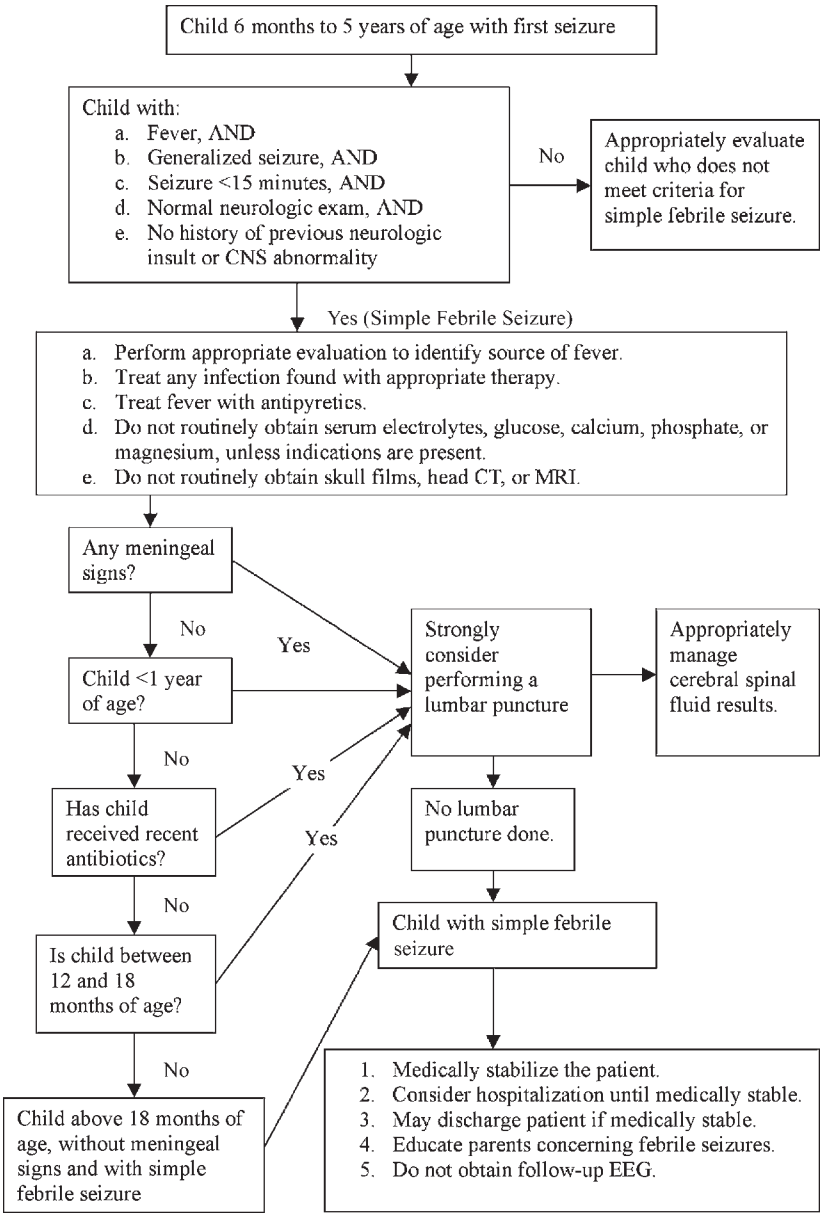

Figure 61-1. Evaluation of child with simple febrile seizure. CT, computed tomography; EEG, electroencephalogram; MRI, magnetic resonance imaging. (Adapted from Committee on Quality Improvement, Subcommittee on Febrile Seizures, American Academy of Pediatrics: Practice parameter: the neurodiagnostic evaluation of the child with a first simple febrile seizure. *Pediatrics* 97:769–775, 1996. Available at www.aap.org/policy/neuro.htm.)

3. What things should be sought on physical examination?

Perform a complete neurological examination on any child with a first time seizure.

Components of the examination include:

- Mental status
- Cranial nerves
- Motor skills
- Coordination
- Reflexes
- Sensation
- Gait

If the patient is febrile, the source of the fever should be sought. A careful search for any evidence of NAT (retinal hemorrhages, bruising, fractures) should be performed.

4. How can I classify a pediatric seizure?

There are several ways in which a seizure can be classified. The first is by its appearance (focal versus generalized). It is important to identify whether or not the seizure started focally and then secondarily generalized or whether it started as a generalized seizure. If the seizure is focal, it is important to obtain an exact description of where it started and if possible, what the child experienced prior to the seizure. This can help significantly in the localization of the epileptic focus. Seizures can also be classified by syndrome, prognosis, and cause.

5. What are common reasons for a seizure in the neonate?

The most frequent cause for neonatal seizures is hypoxic-ischemic encephalopathy. Additional causes include:

- Intracranial hemorrhage (subarachnoid in term, germinal matrix in preterm)
- Metabolic disturbances (hypoglycemia, hypocalcemia, drug withdrawal, amino acidemias, organic acidurias, urea cycle defects)
- Infection: TORCH infections (**TO**xoplasmosis, **R**ubella, **C**ytomegalovirus, **H**erpes simplex virus), *Escherichia coli, Streptococcus pneumoniae*
- Malformations of cortical development
- Benign neonatal or infantile familial convulsions

6. What tests should be done for a neonate experiencing seizures?

Initially, send serum electrolytes, including glucose, calcium, and urine toxicology. Request an ammonia level (free flowing collection), looking for a urea cycle defect. Unless another cause is found, a lumbar puncture (LP), looking for infections, should be performed. Studies on the cerebrospinal fluid (CSF) should include cell count, protein, glucose, amino acids, lactate, pyruvate, Herpes polymerase chain reaction (PCR), and evaluation for xanthochromia (prior bleed). TORCH studies can also be performed. Serum amino acids and urine organic acids can be tested for other inborn errors of metabolism.

Cerebral imaging studies include ultrasound, computed tomography (CT) scan, or magnetic resonance imaging (MRI). MRI is the gold standard. Although expensive and often requiring sedation, MRI will identify malformations of cortical development. Though easier to obtain, CT has less resolution than MRI and exposes the newborn to radiation. Ultrasound is portable and convenient but does not allow the cortical convexities to be well viewed and may have limited availability. An electroencephalogram (EEG) may be ordered on an inpatient basis.

7. What medications are used to treat neonatal seizures?

There is a dramatic lack of evidence that any drug is useful in the treatment of neonatal seizures. For a long time, phenytoin, phenobarbital, and lorazepam have been used. Currently some of the newer medications including Topamax and Keppra are being used for neonatal treatment.

8. What are common reasons for a child to have a seizure?

Common reasons include:

- Fever
- Lack of compliance to antiepileptic medication
- Infection

- Trauma
- Metabolic abnormalities
- Toxins
- Tumor
- Genetics (channelopathies, chromosomal abnormalities)

9. What is the definition of a febrile seizure?

According to the National Institutes of Health (NIH), a febrile seizure is an event in infancy or childhood usually occurring between 3 months and 5 years of age, associated with fever but without evidence of intracranial infection or defined cause. Seizures with fever in children who have suffered a previous nonfebrile seizure are excluded. A similar definition, extending the age range to 1 month, was published by the International League Against Epilepsy.

10. Are genetics involved with febrile seizures?

Genetic factors are definitely involved with febrile seizures. Two syndromes that include febrile seizures are generalized epilepsy with febrile seizures plus (GEFS+) and severe myoclonic epilepsy. There is a family history of febrile seizures in about one third of patients. Presence of febrile seizures in an older sibing confers a 20% chance of febrile seizures in the younger child.

11. What are the types of febrile seizures?

There are two types of febrile seizures: simple and complex. Complex febrile seizures are those that are focal (4%), those that are prolonged (8%), and those that occur multiple times during a single day or illness (15%). Approximately one third of all febrile seizures are complex. If a child has a complex febrile seizure, his or her next febrile seizure is also likely to be complex.

12. What factors make the recurrence of febrile seizures more likely?

The overall risk of recurrence for febrile seizures is about one third. This is true regardless if it was a simple or complex febrile seizure. Risk factors for recurrence include:

- Early occurrence of first seizure
- History of febrile seizures in first-degree relative
- Family history of epilepsy
- Abnormal neurological examination
- Lower temperature at onset of seizure
- Brief period of recognized fever
- More frequent illnesses (daycare)

13. What tests should be done following a febrile seizure?

a. Laboratory
   Consider completed blood count (CBC), glucose, electrolytes, calcium, magnesium, urinanalysis (UA), and stool culture, particularly for complex febrile seizures. (See Chapter 60 for further discussion on pediatric fever.) Strongly consider LP if younger than 18 months with no fever focus.
   LP should also be done if there are meningeal signs.
b. Imaging
   Nonemergent MRI if focal features
   Emergent CT if neurological deficits do not resolve and child does not return to baseline
c. EEG (as outpatient if patient returns to baseline neurologic status)
   If complex features or strong family history of epilepsy

14. Under what conditions should a child having febrile seizures be treated and what treatments should be used?

The two medications that have been shown effective at preventing febrile seizures are phenobarbital and valproate. There is some question as to whether or not phenobarbital

can impact cognitive ability in children. Depakote is not a good drug for children younger than 2 years because it can result in fatal hepatotoxicity. Some of the newer drugs are being tried by practitioners for febrile seizures. Medication should only be started in consultation with a pediatric neurologist and if having the following features: very frequent seizures, prolonged seizures, or a family that is far from medical care. Education for the family is imperative.

Attempts at controlling the fever with agents such as acetaminophen or ibuprofen usually fail; the seizure is typically the first sign of the illness. For patients who have prolonged febrile seizures, the use of Diastat (rectal diazepam) is a reasonable choice.

15. What is the likelihood that a child suffering febrile seizures will eventually develop epilepsy?

The risk of a child with simple febrile seizures developing epilepsy is approximately 1%. This is only slightly above the risk for the general public. The risk of a child with complex febrile seizures developing epilepsy is about 6%.

16. What are infantile spasms and what are some common causes?

Infantile spasms occur in about 1/3,000 of infants. They typically develop between the ages of 3 and 8 months. Causes include TORCH infections, malformations of cortical development, hypoxic-ischemic injury, genetic disorders (tuberous sclerosis, Down syndrome, neurofibromatosis, Incontentia pigmenti), metabolic disorders (phenylketonuria [PKU], maple syrup urine disease [MSUD], pyridoxine-dependent seizures), and trauma.

Infantile spasms first present as a jerking movement in which the body may flex or extend suddenly. It is usually a single jerk initially, but after a while, multiple jerks may occur. There is often a cry during the jerking. The jerk can be unilateral or bilateral. Developmental regression is a poor prognostic sign.

17. What is the standard treatment for infantile spasms?

The standard treatment for infantile spasm is adrenocorticotropic hormone (ACTH). ACTH has side effects including hypertension, osteoporosis, and decreased resistance to infection. Recently this has become exceedingly expensive ($100,000 per course) and people are now using steroids and other newer drugs such as Levetiracetam or Topamax. Vigabatrin is a drug that will soon be available in this country and is the preferred drug in the case of tuberous sclerosis. A side effect of this drug is decreased peripheral vision.

18. What is the prognosis for infantile spasms?

The prognosis for infantile spasms is quite poor. Mortality has been reported as high as 33% but is now significantly less because of better care. Only 12% of children have normal intelligence. Data are not sufficient to demonstrate that any drug is effective or that starting drugs earlier makes any difference.

19. What is the definition of epilepsy?

Epilepsy is defined as the tendency to have unprovoked recurring seizures. Operationally, it is defined as an individual who has had two or more unprovoked seizures.

20. What are some common forms of childhood epilepsy?

- Childhood absence: Begins from 4 to 8 years old. Involves hundreds of seizures per day. Typically associated with normal intelligence and normal imaging. Episodes last 5 to 10 seconds with no postictal period. It can be safely reproduced with hyperventilation. Treatment is with Ethosuximide, or if accompanied by generalized tonic-clonic seizures, Depakote.
- Benign rolandic epilepsy: Begins from 6 to 10 years old. Involves facial and arm twitching, slurred speech, and drooling. It will occasionally generalize. No treatment is necessary unless seizures generalize during the daytime.

- Juvenile myoclonic epilepsy: Begins from 12 to 18 years old. Triad of morning myoclonic, generalized tonic-clonic, and absence seizures. Seizures are brought on by stress, alcohol, and sleep deprivation. It often requires lifelong treatment with an agent such as Depakote or Lamictal.

21. What work-up should be done following an afebrile seizure in an asymptomatic child?

If the child has returned to normal, he or she can go home and follow up with an outpatient EEG and MRI. A neurological consultation should also be scheduled. If the patient does not return to baseline, in addition to standard lab testing (i.e., bedside glucose, CBC, electrolytes, liver function tests [LFTs], ammonia, or urine toxicology), an imaging study (CT or MRI) should be performed emergently. If a clinical indication, an LP should be performed. Occasionally, an acute EEG should also be performed to rule out subclinical status.

22. Under what conditions should afebrile seizures be treated using anti-epileptics?

Treatment of seizures with anti-epileptics balances the risk of recurrence with the risks of the medication. The risk of having a second afebrile seizure after a first is slightly below 50%. Therefore, unless there are other factors, it is customary not to start an antiepileptic medication until after the second seizure. Things that confer additional risk such as dramatically abnormal EEG, very strong family history, or abnormal neurological examination may impact the decision of whether or not to start medication. This decision should be made with a pediatric neurologist.

23. What are the older and newer antiepileptics and how do they vary?

- Older antiepileptics: phenobarbital, phenytoin, carbamazepine, ethosuximide, and valproate.
- Newer antiepileptics: topiramate, lamotrigine, levetiracetam, felbamate, gabapentin, oxcarbazepine, zonisamide, pregabalin, and rufinamide.

Older drugs have the advantage of cost and experience. Newer drugs have the advantages of better side-effect profile, decreased monitoring requirements, less frequent dosing regimens, and decreased interaction with other drugs.

24. What are important side effects of the different antiepileptic drugs?

Recently almost all anti-epileptics have been linked to suicidal behavior. Specific side effects:

- Phenobarbital: sedation, hyperkinesis, and cognitive dysfunction
- Carbamazepine (Tegretol): ataxia, dizziness, sedation, and rash
- Valproic acid (Depakote): alopecia, weight gain, and tremor
- Phenytoin (Dilantin): hirsutism, gingival hyperplasia, and ataxia
- Ethosuximide (Zarontin): gastrointestinal (GI) distress, headaches, drowsiness, and hiccoughs
- Levetiracetam (Keppra): psychotic behavior and irritability
- Lamotrigine (Lamictal): rash
- Topiramate (Topamax): sedation, glaucoma, and kidney stones
- Felbamate (Felbatol): aplastic anemia, insomnia, and anorexia
- Tiagabine (Gabitril): GI intolerance
- Oxcarbazepine (Trileptal): hyponatremia
- Zonisamide (Zonegran): weight loss, kidney stones, headache, and decreased sweating
- Lacosamide (Vimpat): dizziness, headache, nausea, and diplopia
- Carisbamate(Comfyde): dizziness, headache, somnolence, and nausea
- Pregabalin (Lyrica): rhabdomyolysis
- Rufinamide (Banzel): somnolence, nausea, and headache
- Vigabatrin (Sabril): peripheral vision loss

25. If an individual stopped taking their antiepileptic drug because they weren't having seizures, and then started to have them again, at what dose should he restart the medication?

If it has been longer than 1 week, the drug must be started as if you had never been on it. This is especially important for a drug such as Lamictal where a rash or Stevens-Johnson syndrome can occur if it is started too quickly. The individual should be reminded that medication should only be stopped after consultation with a physician.

26. When should antiepileptic drugs be discontinued?

The general rule is that an antiepileptic drug can be stopped after 2 years of seizure freedom. Exceptions to this rule would occur for dramatically abnormal EEG or abnormal neurological examination. Approximately one third of patients remain seizure free after drug withdrawal. If seizures are to reappear, they will within the first 6 months in 80%.

There is no evidence that a trial off of medicine significantly impacts the eventual control of seizures.

27. What happens if a dose of antiepileptic drug is missed?

If it is time for the next dose, it is reasonable to simply continue on without giving additional medication. If it is not time for the next dose, it is reasonable to try to squeeze in the missed dose while slightly delaying the next dose.

28. What if vomiting occurs shortly after taking an antiepileptic drug?

If it is more than an hour since the dose was taken, no action is necessary. If it is between half an hour and an hour, a half dose can be taken. If it is less than half an hour since the dose or fragments of the medicine can be seen in the vomit, the dose should be repeated

29. What is the definition of status epilepticus?

Continuous or intermittent seizure activity for longer than 30 minutes.

30. What is the treatment for status epilepticus?

Airway, breathing, and circulation (ABCs) first, of course. A bedside glucose, chemistry panel (with calcium, magnesium, and phosphorous), urine toxicology and anticonvulsant levels along with intracranial imaging (usually CT) should be obtained. If possible, a continuous EEG (performed in consultation with a neurologist) is helpful and should establish a burst-suppression pattern.

After establishing intravenous (IV) access, a benzodiazepine, such as lorazepam, should be given and repeated multiple times if necessary. If seizures persist, phenobarbital, phenytoin, or levetiracetam should be given next. If the seizure is still not controlled, the next step is a midazolam drip, propofol, or pentobarbital.

31. What should an onlooker do if the child has another seizure?

- If possible, place the child on his or her side.
- Remove anything that may injure the child.
- Place something soft under the head.
- Time the event.
- Do not place anything near the mouth.
- If less than 5 minutes, it may not be necessary to do anything.
- If greater than 5 minutes, administer Diastat (rectal diazepam) or go to the ED.
- If greater than 10 minutes, go to the ED.

32. What cautions should you give to parents of children who have seizures?

- No bathing or swimming alone.
- No activities that may result in harm if there is a temporary loss of consciousness.
- Driving guidelines vary by state.
- A complete list of recommended activities can be found at the Epilepsy Foundation's website.

KEY POINTS: SEIZURES IN INFANCY AND CHILDHOOD

1. Seizures have characteristic patterns that distinguish from nonepileptic events. There are typical childhood seizure patterns that guide prognosis and treatment.
2. ED evaluation of a pediatric seizure is dependent on the age, type, and clinical suspicion for infection or nonaccidental trauma.
3. The evaluation of a child in status or not returning to baseline neurologic state requires consultation with a pediatric neurologist for treatment and EEG monitoring.

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ACUTE RESPIRATORY DISORDERS IN CHILDREN

CHAPTER 62

Kelly Flett, MD, and Joan P. Bothner, MD

1. What are the signs and symptoms of respiratory distress in a child?

The progression of respiratory distress is shown in Figure 62-1. Tachypnea is often the earliest sign in younger children because they cannot significantly increase their tidal volume. Normal respiratory rate in children decreases with age: newborns breathe approximately 50 breaths per minute, while a 12-month-old infant averages 30 breaths per minute. Until respiratory failure leads to hypoventilation and hypercarbia, arterial blood gases are of limited clinical value. Oxygen saturation should not be the sole determinant of severity, with the clinical state primarily dictating any need for intervention. Mental status is often the most important parameter; crying with examination is appropriate (a *well-behaved* child may actually be altered).

2. Why are airway problems more serious in pediatric patients than in adults?

There are several important differences between the adult and the pediatric airway. The child's tongue is large, and is the most common cause of airway obstruction in the obtunded child. The narrowest portion of the pediatric airway is at the cricoid ring, making obstruction with subglottic pathology more likely than in adults. The small size of the pediatric airway means that small changes in diameter cause significant increases in resistance. (Remember physics! Resistance is inversely related to the fourth power of the radius.) Higher oxygen consumption in children contributes to more rapid decrease in arterial oxygen levels after airway obstruction.

3. How can I determine where the problem is?

All noisy breathing is not asthma; a few seconds of observation often helps differentiate upper and lower airway obstruction. Generally, extrathoracic lesions (e.g., epiglottis, croup) produce inspiratory stridor (i.e., harsh, vibratory sound), while intrathoracic lesions (e.g., asthma, bronchiolitis) produce prolonged expiratory wheezing (i.e., high-pitched). Regardless of the location, severe pathology can produce both inspiratory and expiratory sounds.

4. What are common causes of upper airway obstruction in children?

See Table 62-1.

5. Discuss the signs and symptoms of croup, who gets it, what causes it, and what the physician can do for it.

Croup, or laryngotracheitis, is the most common cause of infectious acute upper airway obstruction. The etiology is viral (e.g., parainfluenza, influenza and respiratory syncytial viruses) with erythema and swelling of the trachea just below the vocal cords, and patients classically present with a *barky* or *seal-like* cough. The mean age of affected patients is 18 months with a seasonal increase in autumn and early winter. Patients are often febrile, with a prodrome of mild upper respiratory symptoms that progress to stridor. Symptoms are worse with agitation and at night, classically peaking on day 2 of illness. Because the lungs are not directly affected, oxygen saturation can be maintained even in severe illness. Laboratory data are useless. Diagnosis is clinical; X-rays are not indicated unless diagnosis is unclear and foreign body obstruction is a consideration.

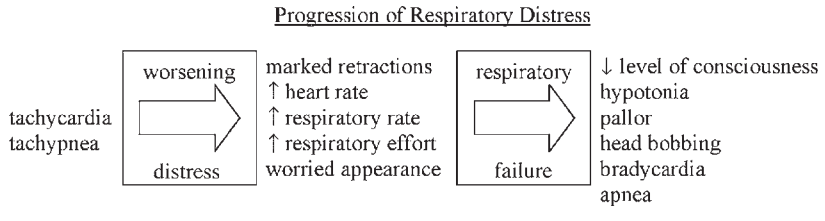

Figure 62-1. Signs and symptoms of respiratory distress in children.

6. Who needs nebulized epinephrine?

Aerosolized epinephrine decreases airway obstruction. It is indicated for children with stridor at rest or marked work of breathing (e.g., tachypnea, retractions). Racemic epinephrine (0.5 mL of 2.25% solution) is used most commonly, but L-epinephrine alone (5 mL of 1:1000 solution) is equivalent. Maximal effect is seen within 30 minutes, with potential rebound to baseline within 3 hours. Patients without resting stridor after 3 hours can be safely discharged home. Criteria for admission include continued stridor at rest, cyanosis, signs of respiratory distress, dehydration, or questionable follow-up. Intubation is rarely needed, but when necessary often requires relatively smaller endotracheal tube (ETT) sizes.

7. What about steroids and croup?

Steroids should be considered for any child who presents to the ED with croup. A single oral dose of dexamethasone 0.6 mg/kg (maximum of 8 mg) decreases the need for hospitalization and return ED visits. There is no evidence to suggest that repeat dosing is indicated or helpful, but this might be considered for young patients presenting prior to the peak of illness (day 1). Nebulized budesonide does not provide benefit over dexamethasone.

KEY POINTS: CROUP

1. Treatment is with oral dexamethasone 0.6 mg/kg (maximum of 8 mg).
2. Racemic epinephrine is used for patients with moderate to severe respiratory distress or stridor at rest.

8. When should I worry about epiglottitis and bacterial tracheitis?

Although both conditions are rare, they warrant careful consideration. Children generally appear toxic with rapid onset of symptoms. Epiglottitis, now rare with universal vaccination against *Haemophilus influenzae* type B, is a bacterial cellulitis of the supraglottic structures, most notably the lingual surface of the epiglottitis. Children lack cough and present with drooling, dysphagia, and a predilection for the *sniffing* position. Radiographic evidence includes a swollen epiglottis (the thumb sign), thickened aryepiglottic folds, and obliteration of the vallecula. Bacterial tracheitis, although rare, may be emerging as a more significant problem. Patients present with croup-like symptoms but toxic in appearance with significant respiratory distress. Radiographs may show shaggy subglottic narrowing and clouding of the trachea. Airway management and broad-spectrum antibiotics (second- or third-generation cephalosporins) are the mainstay of therapy for both disorders.

TABLE 62-1. CAUSES OF UPPER AIRWAY OBSTRUCTION

	Etiology	Age Range	Onset	Toxicity	Drooling	Treatment
Croup	Parainfluenza type 1, influenza A and B, RSV, rhinovirus	6 mo–3 yr	URI prodrome	Mild	Absent	Mist, steroids, aerosolized epinephrine
Epiglottitis	*Haemophilus influenzae*, group A, β-hemolytic *Streptococcus*, *Staphylococcus aureus*, *Streptococcus pneumoniae*, viruses	3–7 yr	Acute	Marked	Frequent	Airway management, antibiotics
Retropharyngeal abscess	Multiple: anaerobes	Infancy–6 yr	URI, sore throat	Variable	Variable	Antibiotics, drainage
Bacterial tracheitis	*S. aureus*, *H. influenzae*, *S. pneumoniae*, *Branhamella catarrhalis* after viral insult	≥3 yr	"Croup" prodrome	Moderate	Usually absent	Airway management, antibiotics

RSV, respiratory syncytial virus; URI, upper respiratory infection.

9. What is the appropriate initial management of a patient with suspected epiglottitis?

Immediately call a surgical or ear, nose, throat (ENT) consultant for anticipated emergent airway management in the operating room; *do not agitate the child in any way*. Direct examination or manipulation of the oropharynx can cause contraction of the pharyngeal muscles and worsen airway obstruction. If the patient will tolerate without agitation, start high-flow oxygen via a nonrebreather bag reservoir mask. Radiographs, blood work, intravenous (IV) lines, and antibiotics can wait; if a child obstructs, bag-valve-mask ventilation should be attempted first.

10. What are retropharyngeal space infections?

The retropharynx is a potential space located immediately posterior to the pharynx, larynx, and trachea. Infection is believed to arise from extension of an acute infection of the ear, nose, or throat, with spread to the lymph nodes in the prevertebral space and subsequent abscess formation. Trauma to the nasopharynx is also a predisposing factor. About 90% of patients are younger than 6 years; the affected child usually appears alert (but mildly toxic), with upper respiratory symptoms, fever, and a stiff slightly extended neck.

11. What imaging studies are helpful in the diagnosis of retropharyngeal infections?

Lateral neck films are often diagnostic (90% sensitivity) but can be difficult to interpret depending on the phase of respiration and neck position. Findings include an increase in the width of the prevertebral space to greater than the anteroposterior width of the adjacent cervical vertebral body, anterior displacement of the airway, and loss of the normal step-off at the level of the larynx. Air-fluid levels may be seen after abscess formation (Fig. 62-2). Computed tomography (CT) scanning is highly sensitive and is used to differentiate abscess from phlegmon or soft-tissue cellulitis.

12. How are they managed?

Treatment includes hospital admission, parenteral antibiotic therapy, and incision and drainage if an abscess is present. Most children do not need acute airway management.

13. When should a foreign body be suspected?

Most patients who present with foreign-body aspiration are males between 5 months and 3 years old. Although a history of an aspiration event (found in 50%–70%) is the most predictive factor, any sudden onset of cough, dyspnea, or wheezing should raise suspicion. Respiratory signs such as stridor or focal wheezing may be absent. Radiographs will only show radiopaque objects. For other objects, expiratory or lateral decubitus films may reveal asymmetric expansion due to a ball-valve mechanism of the foreign body. Endoscopy is diagnostic.

14. How are suspected foreign bodies managed in pediatric patients?

Immediate management depends on the degree of respiratory distress but should be minimal unless respiratory failure is imminent. For unconscious patients, call for emergent ENT evaluation and attempt direct laryngoscopy with removal of any visualized foreign object with Magill forceps. If this fails, attempt bag-valve-mask ventilation and intubation, to push the offending object into one bronchus. If the child cannot be intubated, a needle cricothyroidotomy should be performed.

15. What is bronchiolitis and who does it affect?

Commonly found in children younger than 2 years of age, bronchiolitis is a predominantly wintertime infection (usually from RSV), characterized by inflammation, edema, and mucus accumulation of the bronchioles. Progression of illness leads to lower airway obstruction and consequently ventilation-perfusion mismatch. With small bronchioles more prone to mucous plugging and obstruction, peak incidence and severity is at 3 to 6 months of age.

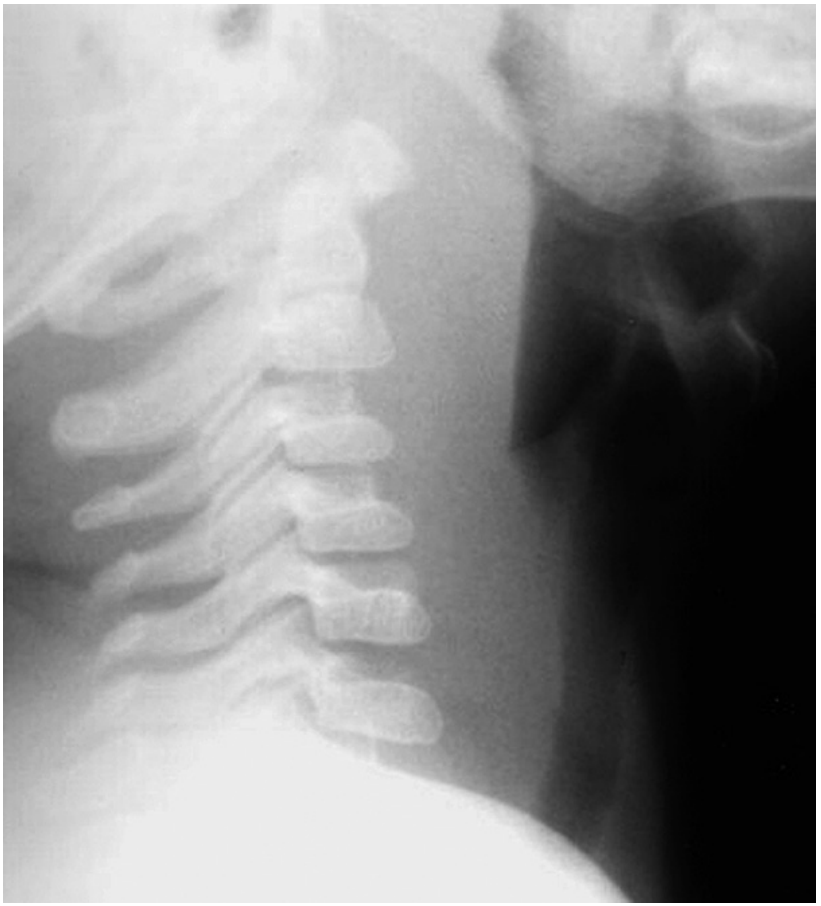

Figure 62-2. X-ray of lateral neck showing thickening of prevertebral space.

16. What are the clinical signs and symptoms of bronchiolitis?

Fever, tachypnea, wheezing, and signs of respiratory distress such as nasal flaring and retractions are coupled with copious mucus secretions. Symptoms follow a predictive course with a 1-to 2-day prodrome of rhinorrhea, cough, and low-grade fever that progresses to lower respiratory signs and respiratory distress. Symptoms peak around days 3 to 4 of illness. Auscultation reveals diffuse wheezing and crackles that often vary between examinations. Children are generally not toxic appearing. Findings of more severe disease include hypoxemia, inability to feed, irritability, and lethargy. Young infants may present with periods of apnea.

17. Do patients with bronchiolitis need chest radiographs?

Generally, infants with classic bronchiolitis do not need any laboratory or radiologic evaluation. Chest X-ray (CXR) findings are nonspecific and include hyperinflation, a flattened diaphragm caused by air trapping, perihilar peribronchial infiltrates, and atelectasis. Findings can be confused with pneumonia and lead to unnecessary use of antibiotics. Children with atypical presentations or examinations may warrant CXR to rule out other causes of first-time

wheezing, including foreign body, congenital airway anomalies, congestive heart failure, or bacterial pneumonia.

18. When are labs needed for bronchiolitis?

Labs are not routinely indicated for bronchiolitis. Infants older than 1 month with bronchiolitis are at low risk for serious invasive bacterial infection. Routine complete blood count (CBC), lumbar puncture, or blood culture is not warranted. Infants with bronchiolitis and fever continue to be at risk for concurrent urinary tract infection (UTI) and should have a catheterized urine specimen for culture performed. Management of fever in neonates (younger than 1 month) with bronchiolitis is unchanged and includes a full septic work-up.

19. What is the treatment for bronchiolitis?

Bronchiolitis treatment is supportive; supplemental oxygen, nasal suctioning, and hydration. There is no evidence to support the routine use of bronchodilators, steroids, or antivirals in the ED. Studies addressing the combined use of these agents, as well as nebulized hypertonic saline, are inconclusive. Children with bronchiolitis and a moderate work of breathing after nasal suctioning are often trialed with a bronchodilator. Albuterol leads to clinical improvement in only 20% to 30% of patients, likely those who have a "reactive component" similar to asthma. Alternatively, albuterol may worsen ventilation/perfusion mismatch and exacerbate hypoxemia. Nebulized epinephrine may be slightly more effective, likely due to vasoconstrictive effects; since there is no outpatient equivalent, it should only be used for admitted patients.

KEY POINTS: BRONCHIOLITIS

1. Less than 20% to 30% of infants with bronchiolitis will respond to inhaled β-agonist or epinephrine therapy. Response should be carefully assessed and treatment continued only in patients with a clear benefit.

20. Who is admitted for bronchiolitis?

Patients who are hypoxemic, have more than mild respiratory distress, have history of apnea, or are unable to adequately self-hydrate should be admitted. Admission should be strongly considered for all children with risk factors for severe disease (Table 62-2). Some centers utilize home oxygen therapy protocols for otherwise well-appearing patients requiring less than one half a liter of oxygen per hour. These patients require 24-hour follow-up, as well as reliable caretaker oversight.

21. How are bronchodilators used in the management of acute asthma?

Selective β_2 agonists are the mainstay of medications to reverse bronchospasm. Delivery of albuterol by nebulizer or meter-dosed inhaler (MDI) with a spacer has been shown to be

TABLE 62-2. BRONCHIOLITIS: RISK FACTORS FOR SEVERE DISEASE

Congenital heart disease
Chronic lung disease (cystic fibrosis, bronchopulmonary dysplasia)
Congenital or acquired immunodeficiency
Major congenital anomalies
Prematurity <37 weeks
Age <6–12 weeks

equally clinically effective, with 4 to 10 puffs of an MDI equivalent to one nebulizer treatment. Delivery by nebulizer remains the preferred route in the ED setting for young patients, as well as those in too much distress to cooperate with MDI use. Albuterol is a racemic mixture of the R and S isomers of the compound, which have bronchodilatory and bronchoconstrictive properties. Levalbuterol, the pure R isomer, may cause less tachycardia but has not, in large clinical trials, been demonstrated to be significantly more effective and is more costly. In children with moderate-to-severe asthma, inhaled anticholinergic therapy (ipratropium bromide) decreases severity and hospitalization rates when given in conjunction with β_2 agonists (albuterol).

22. When and how should steroids be administered?

Controlling inflammation is the cornerstone of asthma treatment. There is lack of consensus regarding the dose and duration of steroid therapy. Generally, a loading dose of prednisone 2 mg/kg (maximum 80 mg) in the ED is followed by a 4-day course of either 1 mg/kg/day as a single daily dose or 2 mg/kg/day divided twice daily. IV steroids are reserved for children who cannot tolerate oral medications. Dexamethasone, with its higher potency and longer half-life, provides an appealing alternative. Multiple small studies have shown that oral dexamethasone 0.6 mg/kg (maximum, 16 mg) given once is equivalent to 5 days of prednisone in children with mild to moderate asthma symptoms.

23. When should a CXR be obtained? What are the typical findings?

A CXR is not indicated in the routine evaluation of a child with asthma but should be obtained if pneumonia, pneumothorax, pneumomediastinum, or foreign body is suspected clinically. CXRs commonly show hyperinflation, atelectasis, and peribronchial thickening, indicating lower airway obstruction. Pneumothorax is rare. Pneumomediastinum is more common in older children (age >10 years), whereas infiltrates are more common in younger children.

24. Outline the evaluation and treatment of an asthma exacerbation in the ED.

Step 1. Initial assessment

- Evaluate vital signs with pulse oximetry, use of accessory muscles, retractions, alertness, auscultation, and peak expiratory flow rate (PEFR) in patients older than 5 years.
- Consider an asthma score for objective assessment and later reassessment (Table 62-3).

Step 2. Initial treatment

- Administer oxygen as needed to keep saturation in the normal range.
- Administer three consecutive treatments of nebulized albuterol (2.5–5 mg or 0.15 mg/kg per treatment) plus ipratropium 250 to 500 μg.
- Administer steroids.

Step 3. Repeat assessment

- Patient should be assessed after initial treatment to determine if additional treatments are needed or if they can be observed for potential discharge. Full effect of albuterol may take 15 minutes beyond the end of the treatment.
- If PEFR is greater than 70% baseline and the patient continues to have no wheezing, retractions, or accessory muscle use at least 2 hours after the last nebulized treatment, they can be safely discharged (Step 4a). If symptoms continue, they should be given additional therapy (Step 4b).

Step 4a. Discharge

- Discharge home with a reliable caretaker, patient education, medications, and follow-up instructions.
- Medications should include albuterol, nebulized or inhaled every 4 hours as needed for wheezing and oral steroids.

Step 4b. Continued therapy

- Continuous albuterol by nebulization (7.5–10 mg/h, can increase to 20 mg/h)
- Ipratropium 500 μg every 4 hours
- Frequent reassessment of patient

Table 62-3. PEDIATRIC ASTHMA SCORE

Score	1	2	3
Resp Rate			
2–3 y	≤34	35–39	≥40
4–5 y	≤30	31–35	≥36
6–12 y	≤26	27–30	≥31
>12	≤23	24–27	≥28
Oxygen requirement	>90% on room air	85%–90% on room air	<85% on room air
Auscultation	Normal or end-expiratory wheeze only	Expiratory wheezes	Inspiratory/Expiratory wheezes or decreased breath sounds
Retractions	0–1 site	2 sites	3+ sites
Dyspnea	Speaks in sentences, coos, babbles	Speaks in partial sentences, short cry	Single words, short phrases, grunting

Modified from Kelly CS, Anderson CL, Pestian JP, et al: Improved outcomes for hospitalized asthmatic children using a clinical pathway. *Ann Allergy Asthma Immunol* 84:509–516, 2000.

Step 5. Admission criteria

- Albuterol treatment required every 2 hours or more.
- Continued hypoxemia by pulse oximetry.
- Continued poor response requiring escalation of treatment.

25. What about magnesium?

The mechanism of action of IV magnesium is not known but is hypothesized to be the counteraction of calcium ions to prevent bronchial smooth muscle contraction. The benefit in patients with mild to moderate exacerbations is unclear, and its use should be reserved for those patients in severe distress or nonresponsive to albuterol and steroids. Dose is 75 mg/kg IV (maximum of 2 g).

26. Does aminophylline have any use?

Aminophylline and theophylline do not have a role in the routine management of the pediatric asthma patient in the ED. IV aminophylline has been shown to improve lung function in children with severe asthma exacerbations, but it does not reduce symptoms, number of nebulizer treatments, or length of stay. Several studies have failed to show benefit of theophylline when added to bronchodilators and steroids in noncritically ill patients. There are inconclusive data to suggest that theophylline may be equally effective to terbutaline in pediatric intensive care unit (PICU) patients.

27. What about parenteral β-agonists?

Use of systemic β-agonists is controversial; few well-designed studies have evaluated their use. They should be considered in patients with severe exacerbations who have failed to respond to maximal inhaled therapy. Terbutaline, subcutaneously or intravenously, may be given as an initial bolus of 10 μg/kg followed by a continuous infusion starting at 0.5 μg/kg/min. Epinephrine may

also be given subcutaneously. These medications, which should not interrupt inhaled therapy, require monitoring of cardiac function and serum potassium levels.

28. What should I do if my patient is going into respiratory failure?

Consider treatment with magnesium, terbutaline, and epinephrine. Bilevel positive airway pressure (BiPAP; set initially at 10/5) has been shown in small studies to improve respiratory rate and oxygenation in children. If intubation is necessary, ketamine (in conjunction with a paralytic) stimulates the release of catecholamines causing bronchodilation, making it the inductive agent of choice (dose: 1-2 mg/kg IV). To optimize oxygenation and prevent barotrauma, initial ventilator settings should be set to a reduced rate of 8 to 12 breaths per minute, allowing for permissive hypercapnia.

KEY POINTS: EVALUATION OF RESPIRATORY DISORDERS

1. Observation prior to auscultation helps to localize and differentiate etiologies of pediatric respiratory complaints.
2. Foreign bodies should be suspected in any child presenting with signs of airway obstruction.
3. Labs and radiographs are not routinely indicated in many childhood respiratory disorders.

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CHAPTER 63

PEDIATRIC GASTROINTESTINAL DISORDERS AND DEHYDRATION

Joshua S. Easter, MD

1. What are the common causes of abdominal pain in children?

Abdominal pain is a common pediatric complaint, the differential for which is guided by age of the patient, history, and physical examination with or without diagnostic studies (Table 63-1).

2. Can you determine if a child is dehydrated based on the history and physical examination?

Historical factors, such as the number of wet diapers, frequency of vomiting or diarrhea, and amount of oral intake, should be used in conjunction with other physical signs to assess dehydration. The presence of sunken eyes, dry mucous membranes, cool extremities, weak pulses, decreased tears, increased heart rate, and sunken fontanelle are often unreliable. Capillary refill time (normally less than 2 seconds), skin turgor, and hyperpnea are better indicators of severe dehydration.

3. How do you manage the different levels of dehydration?

- *Mild or moderate dehydration*: Oral rehydration is the ideal treatment. Using the World Health Organization (WHO) solution or Pedialyte, the patient drinks 50 to 100 (mL/kg) over 4 hours in the form of 5 mL aliquots administered via a medicine dropper or syringe every 5 minutes. An alternate and more simplified strategy involves administration of 1 mL/kg every 5 minutes for 4 hours. If the child vomits, wait 15 minutes and try again. These regimens have similar success rates to intravenous hydration and shorter times to initiation of therapy and shorter ED length of stay.
- *Severe dehydration or patients failing oral rehydration*: These children are ill appearing and should receive intravenous fluids. They often require multiple 20-mL/kg boluses to compensate for their dehydration. By avoiding fatty acid breakdown and ketosis, dextrose containing fluids may lead to more rapid improvement in vomiting than normal saline.

4. How are maintenance fluids determined in a child?

Maintenance fluids per hour are calculated based on weight in kilograms using the 4-2-1 rule: 4 mL/kg for the first 1 to 10 kg, an additional 2 mL/kg for the next 11 to 20 kg, and 1 mL/kg for every additional kg.

5. What are potential causes of vomiting without diarrhea in children?

The differential includes early gastroenteritis, urinary tract infection, appendicitis, diabetic ketoacidosis, otitis media, pneumonia, strep pharyngitis, testicular or ovarian torsion, meningitis, and head injury.

6. How do you differentiate between gastroenteritis and more severe abdominal pathology?

This may be difficult and often requires a period of observation in the ED for other developing signs or symptoms. Focal tenderness in the abdomen makes gastroenteritis less likely.

7. What diagnostic studies should be obtained on children with gastroenteritis?

Most require no tests. Infants with prolonged or severe symptoms can deplete their glycogen stores and therefore should have a bedside glucose test. Electrolyte studies,

TABLE 63-1. DIFFERENTIAL OF NONTRAUMATIC ABDOMINAL PAIN BY AGE

Neonate	**2 months–2 years**
Malrotation	Incarcerated hernia
Necrotizing enterocolitis	Intussusception
Testicular torsion	Urinary tract infection
2–5 years	**6–18 years**
Appendicitis	Appendicitis
Foreign body	Ovarian/testicular torsion
Intussusception	Kidney and gallbladder stones
Ovarian torsion	Diabetic ketoacidosis
Urinary tract infection	Ectopic pregnancy
Strep pharyngitis	Pelvic inflammatory disease
Henoch-Schönlein purpura	Gallbladder disease

looking for hypernatremia or renal insufficiency, may be considered for ill-appearing children. In the setting of hypoglycemia, the infant or child should be given 4 mL/kg of dextrose 10% (if younger than 3 months old) or 2 mL/kg of dextrose 25% (if more than 3 months old).

8. How do you differentiate between bacterial and viral causes of diarrhea?

- Viruses cause the majority of diarrhea in children, with rotavirus the most common agent in younger children and Norwalk virus in older children. Viral diarrhea tends to produce voluminous watery diarrhea with diffuse abdominal cramping.
- Bacterial diarrhea typically causes lower abdominal pain and bloody or mucousy stool.

However, these history and physical examination findings cannot reliably differentiate bacterial from viral diarrhea. Stool cultures should be obtained in patients with significant comorbidities, ill appearance, high fever, bloody stools, severe cramping, recent antibiotic use, travel, or exposure to a patient with a known bacterial diarrhea.

KEY POINTS: GASTROENTERITIS AND DEHYDRATION

1. Young children with gastroenteritis can become dehydrated easily.
2. Vomiting in the pediatric population has a broad differential.
3. Most children can be successfully rehydrated without intravenous fluids.

9. Should narcotics be withheld from children with acute abdominal pain while awaiting a surgical evaluation?

No. Multiple studies have shown diagnostic accuracy from physical examination increases when patients' pain is controlled.

10. How does appendicitis present in younger children?

The diagnosis of appendicitis is commonly missed in younger children who often present with nonspecific symptoms. Vomiting, abdominal pain, fever, diarrhea, irritability, and right hip pain are some typical presentations, often attributed to other causes. Similarly, their physical examinations frequently reveal diffuse abdominal tenderness or abdominal distention, while pain localized to the right lower quadrant is an infrequent presentation. Appendicitis in the infant is typically recognized only after perforation, which occurs in 70% to 95% of these cases.

11. What physical examination findings are found in older children with appendicitis?

The most common findings are tenderness in the right lower quadrant and involuntary guarding. Rovsing's sign (pain in the right lower quadrant with palpation of the left lower quadrant), obturator sign (pain with internal rotation of the flexed hip), and the psoas sign (pain with extension of the right thigh) have not been shown to be particularly sensitive or specific for appendicitis in children. Their absence should not be used to rule out appendicitis.

12. What laboratory tests are helpful in children with appendicitis?

If the history and physical examination are highly suspicious for appendicitis, no further tests are required and a surgeon should be consulted.

The *white blood cell count* (WBC) does not provide useful levels of sensitivity and specificity. A WBC $>10{,}000/mm^3$ has a sensitivity of 88%, but specificity of 53%; a WBC $>15{,}000/mm^3$ improves specificity to over 60%, but the sensitivity declines to 19%. An elevated *C-reactive protein* has similar sensitivity and specificity to a WBC>10,000, with even lower sensitivity and specificity when measured in the first 12 hours. A positive urinalysis cannot exclude appendicitis; 30% of children with appendicitis have pyuria or bacteriuria.

13. What are the advantages and disadvantages of the different radiographic tests for appendicitis?

- **Plain films**: Insensitive and nonspecific, these are normal in 82% of children with appendicitis.
- **Ultrasound (US)**: If available, US should be the initial study of choice in children with suspected appendicitis since lack of peritoneal fat favors ideal imaging with this modality. In experienced hands, US provides a high sensitivity (71%–92%) and specificity (96%–98%). Appendicitis will show an appendiceal diameter >6 mm, wall thickness >2 mm, obstruction of the appendiceal lumen, appendicolith, high echogenicity surrounding the appendix, or pericecal free fluid. Obesity, uncooperative patients, or atypical locations of the appendix may limit this study.
- **Computerized tomography (CT)**: With CT, appendicitis shows an appendiceal diameter >6 mm, wall thickness >1 mm, periappendiceal fat stranding or fluid collection, or an appendicolith. In children, CT has a higher sensitivity (94%–99%) and specificity (87%–99%) than US. Higher cost, potential need for sedation, and radiation exposure are the major drawbacks of CT.

KEY POINTS: APPENDICITIS

1. Younger children have atypical presentations of appendicitis, resulting in delays in diagnosis and high perforation rates.
2. Lab tests are relatively non-specific and should not be used to exclude a diagnosis of appendicitis.
3. In equivocal cases, ultrasound should be the first imaging study in children with suspected appendicitis.

14. How does intussusception present?

Intussusception, an invagination of one portion of bowel into a distal segment (most commonly at the ileocecal junction), afflicts children most commonly between infancy and 3 years of age. The classic triad of colicky abdominal pain, vomiting, and bloody stool is present in less than 25% of children. Intermittent periods of irritability, where children may pull their knees up toward their chest, is often the only symptom. Although often cited in the literature, currant jelly stools are a late, rare, and ominous finding from bowel ischemia. Younger children may present with nonspecific findings such as altered mental status or lethargy.

15. How do you diagnose intussusception?

The classic *crescent sign* on plan radiography from the intussuscepting mass is rarely seen. Nevertheless, abdominal X-rays can be helpful in low risk cases; when air is seen in the ascending colon on at least two of three views (i.e., supine, prone, and lateral decubitus), likelihood of intussusception is substantially reduced. US may identify a *donut* or *target* sign, yielding a sensitivity and specificity of over 90%. Air enema may be utilized to both diagnose and treat intussusception.

16. How should intussusception be treated?

Air enemas provide equivalent success rates to contrast enemas with less radiation exposure. Due to a 1% risk of perforation with enema reduction, a surgeon should be available. Because up to 10% of patients will have recurrence within the first 24 hours, caregivers must ensure appropriate family education on strict return precautions. Shock or suspected intestinal perforations necessitate surgical consultation for operative repair.

17. What is the significance of bilious emesis in a neonate?

Bilious emesis in a neonate is a surgical emergency until proven otherwise because it could represent malrotation with volvulus (midgut volvulus). Congenital malrotation of the midgut predisposes the bowel to twisting on itself, leading to bowel obstruction and vascular compromise, with bowel necrosis developing in as little as 2 hours.

Midgut volvulus classically presents with sudden onset of bilious emesis and abdominal pain; however, early in the course of illness, more than half of patients have normal abdominal examinations and one third have abdominal distention without tenderness. Thus, all infants with bilious emesis should undergo diagnostic testing regardless of their abdominal examinations. Although X-rays can show small bowel obstruction, a *double bubble* sign, or paucity of distal bowel gas with volvulus, imaging is often normal. An upper gastrointestinal (UGI) series with contrast is the gold standard because it will show a cork screwing of contrast or the duodenojejunal junction not crossing to the left of the vertebral column.

If volvulus is suspected, intravenous fluids should be given, a nasogastric tube inserted, broad-spectrum antibiotics administered, and surgical consultation obtained immediately.

18. What characteristics of a patient's history help differentiate pyloric stenosis from other causes of vomiting in infants?

True projectile emesis, where the vomitus shoots away from the patient, is most commonly found with pyloric stenosis. A hypertrophy of the pylorus develops between 1 to 5 weeks of age. Initially, infants vomit only at the end of feeds, later developing more classic projectile vomiting. Unlike more severe conditions such as malrotation, emesis is usually nonbilious due to the stenosis being proximal to the duodenum. The patient will remain hungry and continue attempts to feed. Unlike more benign causes of vomiting such as reflux, the patient does not gain weight appropriately.

19. What diagnostic findings arise with pyloric stenosis?

Vomiting leads to loss of hydrogen ions from the stomach, the kidneys attempt to conserve sodium in a response to dehydration, spilling potassium into the urine, all resulting in a hypokalemic, hypochloremic metabolic alkalosis.

The diagnostic study of choice is an US, which has a sensitivity and specificity of nearly 100%. In pyloric stenosis, the pyloric wall is greater than 4 mm wide or 14 mm long. If the US is equivocal, then an UGI will classically show a string sign as contrast travels through the narrowed pylorus.

Patients with pyloric stenosis require rehydration and eventual surgical correction, although in developing countries often patients can be supported long enough to allow the hypertrophy to resolve without surgery.

20. Are inguinal hernias dangerous?

One percent to 2% of children develop inguinal hernias, with 10% ultimately incarcerating. This happens most commonly in children younger than 1 year of age. If the incarceration persists, the bowel can strangulate, cutting off its blood supply and leading to bowel obstruction or necrosis.

Unless bowel necrosis is already suspected, manual reduction can be attempted on incarcerated hernias to prevent strangulation. With the patient in Trendelenburg, using constant gentle pressure, 95% of inguinal hernias can be reduced and these patients can be safely discharged for outpatient surgical repair. If necessary, sedation can be used to assist in pushing the hernia back through the inguinal ring. If reduction is unsuccessful or strangulation suspected, immediately consult a surgeon.

21. What is the difference between a hernia and a hydrocele?

A hydrocele arises from an incomplete obliteration of the processus vaginalis, which allows the peritoneum to translocate into the scrotum. Unlike hernias, these can be transilluminated. In addition, they are readily separable from the testes. If you cannot differentiate a hydrocele from a hernia on examination, you can obtain a scrotal ultrasound. Hydroceles are benign and often resolve spontaneously.

KEY POINTS: SURGICAL EMERGENCIES IN YOUNG CHILDREN

1. Intussusception rarely presents with the classic triad of colicky abdominal pain, currant jelly stools, and vomiting. More often only intermittent irritability is present.
2. Bilious emesis in a neonate is a surgical emergency until proven otherwise.
3. Infants with pyloric stenosis have projectile emesis but remain hungry and interested in feeding.

22. Why is jaundice concerning in a neonate?

While newborns often have physiologic jaundice that is self-limited, significantly elevated levels of unconjugated bilirubin can lead to kernicterus, with resulting deafness, developmental delay, or death. These elevated levels can arise from a myriad of causes including Rh or ABO incompatibility, prematurity, polycythemia, intestinal obstruction, sepsis, or dehydration. All patients with visible jaundice need a documented bilirubin level, and if elevated a search made for etiology. This may include a blood type, Coomb's test and complete blood count (CBC). Patients with elevated age-specific bilirubin levels may require phototherapy, with exchange transfusion considered for marked elevations (>25 mg/dl).

23. Is it normal for a child to have constipation?

It is normal for infants to strain during bowel movements, but parental anxiety over infant straining or lack of frequent bowel movements may lead to an ED visit. Bottle-fed infants can often pass as few as one stool every other day. Breastfed infants may pass a stool with each feed or as infrequently as once every 7 to 10 days. As infants age, it is typical for stool frequency to decrease; by 4 years of age children average 1.2 bowel movements per day.

Rarely, infants presenting with constipation have more serious conditions. A thorough history and physical examination can give clues to some of these entities: onset of symptoms in the first week of life (Hirschsprung's), abnormal tone, lethargy and weak cry (botulism and hypothyroidism), or worrisome abdomen examination (volvulus).

The diagnosis of constipation should be based on difficulty or pain with passage of a bowel movement rather than absolute frequency. In older children, constipation most commonly arises with changes in diet or inadequate fluid intake. School-age children may have behavioral issues, such as a fear of having a bowel movement at school that ultimately affects their bowel patterns.

24. How can you treat constipation in the ED?

Most patients with constipation not arising from a serious etiology can be managed as outpatients. A trial of a soy-based formula may relieve constipation in infants with suspected cow's milk intolerance. A one-time enema can occasionally help, but hypertonic phosphate enemas and tap water enemas should be avoided because they can cause severe electrolyte abnormalities. Mineral oil (1–4 ml/kg/dose), lactulose (1–2 ml/kg/dose), milk of magnesia (1–3 ml/kg/dose), or MiraLax (mix 17g in 8 oz fluid) can be administered in the short term. Long-term management includes increasing fluid intake and adding fiber to the diet.

25. What is a Meckel's diverticulum?

It is a remnant of the omphalomesenteric duct in children. It is the most significant cause of painless rectal bleeding and can lead to obstruction or intussusception. Sixty percent contain heterotopic gastric, pancreatic, or endometrial tissue. If suspected, a technetium 99m Meckel's scan can identify ectopic gastric mucosal tissue, thus making the diagnosis.

26. What is Meckel's rule of twos?

Meckel's diverticulum occurs in 2% of the population; 2% of patients will manifest symptoms; the diverticulum is typically 2 inches long and within 2 feet of the ileocecal valve; average age at presentation is 2 years.

27. How do you manage an ingested gastrointestinal foreign body?

The management of foreign bodies depends on the nature of what was ingested and its location. Any patient with a known ingestion and hematochezia, melena, or signs of an acute abdomen requires immediate surgical consultation. See Table 63-2.

28. What are the possible complications of an esophageal foreign body?

Airway obstruction, esophageal stricture, esophageal perforation, mediastinitis, or paraesophageal abscess.

29. What diseases are associated with these classic findings on X-ray?

See Table 63-3.

TABLE 63-2. MANAGEMENT OF GASTROINTESTINAL FOREIGN BODIES

Emergent Endoscopy	Gastrointestinologist Consultation
Sharp objects in the esophagus	Button battery past the esophagus
Button battery in the esophagus	Sharp objects past the pylorus
Objects causing difficulty controlling secretions or breathing	Long objects past the pylorus (>5 cm)
Objects in the esophagus for more than 24 hours	Multiple magnets

TABLE 63-3. RADIOGRAPHIC FINDINGS IN PEDIATRIC ABDOMINAL PAIN

Finding	X-ray Description	Disease Process
Double Bubble	Paucity of gas with air bubble in stomach and duodenum	Volvulus
Crescent	Curvilinear mass often found near transverse colon beyond hepatic flexure	Intussusception
Pneumatosis Intestinalis	Air in bowel wall	Necrotizing enterocolitis
Enlarged pylorus	Wall of pylorus >4 mm thick Canal >14 mm	Pyloric stenosis

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PEDIATRIC INFECTIOUS DISEASES

Roger M. Barkin, MD, MPH, FACEP, FAAP

CHAPTER 64

1. Are infectious diseases important to recognize in the pediatric patient?
Infectious diseases account for a significant percentage of pediatric visits to the ED for acute illness. Although most conditions are self-limited and infrequent, some infections are significant in that they may be multisystem or life threatening, requiring consideration in the differential diagnosis of many presenting complaints.

2. What is the mechanism of spread of measles (rubeola)?
By direct contact with infectious droplets or airborne dissemination.

3. What is the incubation period for measles?
From exposure to the onset of symptoms, 8 to 12 days. It is 14 days from exposure to the onset of the rash. Patients are contagious 1 to 2 days before they become symptomatic and 4 days after the rash appears.

4. List the common signs and symptoms of patients with measles.
- High fever.
- Three Cs: Conjunctivitis, coryza, and cough may be observed.
- Rash: Discrete red maculopapular rash first appears on the forehead, becoming coalescent as it spreads down the trunk to the feet by the third day of the illness. The rash fades in the same head-to-feet pattern as it appeared.
- Koplik's spots: 1- to 3-mm bluish white spots on a bright red surface, which appear first on the buccal mucosa opposite the lower molars. They are a pathognomonic exanthem of measles. They appear approximately within 48 hours after the onset of symptoms. The spots may spread to involve the buccal and labial mucosa and disappear on the second day after the onset of the rash.
- Photophobia may be noted.

5. Name the complications of measles.
Otitis media and bronchopneumonia. Encephalitis may occur as well.

6. What is subacute sclerosing panencephalitis?
A rare degenerative central nervous system disease caused by a latent measles infection, occurring an average of 10 years after a primary measles illness. Patients have progressive intellectual and behavioral deterioration and convulsions. This disease is not contagious.

7. Describe the exanthem seen in rubella. Why is it also called 3-day measles?
Numerous discrete rose-pink maculopapules first appear on the face and, as in rubeola, spread downward to involve the trunk and extremities. The rash on the face fades on day 2, and the rash on the trunk becomes coalescent. By the third day, the rash disappears, which is why rubella is also called *3-day measles.* Rubella is now rarely reported in the United States secondary to the efficacy of immunizations.

8. What are Forschheimer spots?
Pinpoint red macules on the soft palate seen early in rubella; however, in contrast to Koplik's spots, they are *not* pathognomonic.

9. What is the incubation period for mumps, and when is the patient contagious?
The incubation is 12 to 18 days. The patient is contagious 1 to 2 days (up to 7 days) before the onset of parotid swelling. Patients are no longer infectious 7 to 9 days after the onset of parotid swelling.

10. List the major complications of mumps.
- Meningoencephalitis in 0.5% of cases
- Orchitis after puberty with secondary sterility (rare)
- Arthritis, renal involvement, thyroiditis, mastitis, and hearing impairment (all rare)

11. Describe the characteristic rash in erythema infectiosum.
Erythematous ears and a maculopapular rash on the cheeks that coalesce to form the classic *slapped-cheek appearance*. The rash spreads to the extremities 1 to 2 days later with a reticular, lacelike pattern caused by central clearing of the confluent rash. Human parvovirus B19 is the causative agent.

12. What is the typical progression of findings of roseola (erythema subitum)?
Typically, a child between 6 months and 2 years old (up to 4 years old) presents with a history of high fever of 3 days' duration and mild symptoms, if any. The fever abates abruptly, followed by the appearance of a macular rash on the trunk and thighs. It is caused by human herpesvirus-6.

13. What is the incubation period for varicella (chickenpox), and when are patients infectious?
The incubation period is 10 to 20 days. Infectivity occurs 1 to 2 days before the appearance of the rash until no new lesions are forming (usually 7–10 days after the appearance of the rash). Children are generally not considered to be infectious once the lesions are crusted and dry.

14. Name the mode of transmission and the cause of infectious mononucleosis (IM).
IM is transmitted through direct and prolonged contact with oropharyngeal secretions. It is caused by the Epstein-Barr virus.

15. List the clinical manifestations of IM.
- Fever lasting 1 to 2 weeks
- Lymphadenopathy (usually nontender, no overlying erythema, most often bilateral cervical location, with epitrochlear nodes being suggestive of IM)
- Tonsillopharyngitis (usually an exudate is present—need to obtain a throat culture to exclude group A streptococci)
- Spleen or liver enlargement
- Young children: May also have rashes, abdominal pain, upper respiratory infections with cough, failure to thrive, and early-onset otitis media

16. Which parenteral antibiotic is correlated with a rash in older children and adults with IM?
Ampicillin and amoxicillin, by an unknown mechanism of action, can cause a rash in patients with IM.

17. What are the hematologic findings in IM?
A relative lymphocytosis of greater than 50% of all leukocytes and a relative atypical lymphocytosis of 10% of leukocytes are the typical findings, although the relative percentage of atypical lymphocytes in children may be lower than in adults.

18. What are heterophil antibodies?
Serum immunoglobulin M (IgM) antibodies with the capability to agglutinate horse (better than sheep or bovine) erythrocytes. The ability to absorb to beef red blood cells but not guinea pig kidney distinguishes heterophil antibodies in IM from both Forssman antibodies

(found in normal serum) and the antibodies in serum sickness. A heterophil antibody titer greater than 40 with a good clinical history for IM strongly supports the diagnosis. It is positive in 90% of cases of IM, with few false-positive results except in young children, in whom Epstein-Barr virus serology is needed to establish the diagnosis.

19. What is the monospot test?

This qualitative, rapid slide test is used to detect serum heterophil antibodies in IM. It is positive in 70% of patients during the first week of illness and in 85% to 90% of patients during the third week. In children younger than 4 years, this test may be negative because of lower levels of detectable heterophil antibodies requiring the more sensitive Epstein-Barr virus serology to be done.

20. Describe the treatment of uncomplicated IM.

Supportive therapy and rest are the mainstays of treatment, with emphasis on analgesia for sore throat, headaches, and myalgias; oral fluids to prevent dehydration secondary to discomfort with swallowing; and a decrease in normal activity. Acetaminophen and ibuprofen may be useful.

21. Summarize the complications of IM.

Respiratory
- Airway obstruction due to tonsillar hypertrophy
- Sinusitis
- Pneumonia

Hematologic
- Thrombocytopenia
- Hemolytic anemia
- Granulocytopenia

Neurologic
- Encephalitis
- Cerebellar ataxia
- Guillain-Barré syndrome
- Transverse myelitis
- Bell's palsy

Cardiac
- Pericarditis
- Myocarditis

Eye
- Optic neuritis
- Uveitis, keratitis

Other
- Splenic rupture
- Chronic fatigue

22. What is the role of corticosteroids in the treatment of IM?

Steroids may reduce the risk of progression to upper airway obstruction by reducing edema and hyperplasia of the lymphoid tissue. There is usually improvement in 6 to 24 hours after administration. It is not used routinely in uncomplicated cases.

23. How long does the patient need to worry about the risk of splenic rupture?

Although rare, rupture of the spleen usually occurs during the second or third week of the illness. Patients must avoid contact sports while the spleen is enlarged. Follow-up examinations determine when it is safe to play contact sports.

24. What are the most common findings associated with botulism in children?

Botulism results from ingestion of preformed toxins (e.g., canned vegetables), ingestion of spores in infant botulism (honey), or spore contamination of open wounds. One third of the 100 annual cases in the United States are food borne; the remainder are cases of infant botulism. *Clostridium botulinum* produces a neurotoxin that blocks the presynaptic release of acetylcholine after an incubation period of 12 to 48 hours. Clinically, patients develop symmetric descending paralysis with weakness and equal deep tendon reflexes associated with a normal sensorium. Pupils are fixed and dilated with oculomotor paralysis, blurred vision, diplopia, ptosis, and photophobia. Associated findings may include slurred speech, nausea, vomiting, constipation, vertigo, dry mouth, dysphagia,

and urinary retention. Dyspnea and rales, progressing to respiratory failure, may be noted.

25. Are there specific measures that should be initiated in the patient with botulism?

Initial management must focus on support, airway maintenance, and monitoring. Botulism equine antitoxin should be administered and is available from the Centers for Disease Control (770-488-7100) or from local state health departments.

26. What are the distinct clinical presentations of diphtheria?

Corynebacterium diphtheriae, an unencapsulated, club-shaped gram-positive bacillus, produces an exotoxin that results in four patterns of clinical findings. The pharyngeal-tonsillar complex consists of a sore throat, fever, vomiting, dysphagia, and malaise associated with a gray, closely adherent pseudomembrane. Respiratory obstruction may develop. Less common presentations include laryngeal diphtheria with hoarseness and loss of voice; respiratory tract edema may lead to obstruction. Serosanguineous nasal discharge may persist for weeks, usually without systemic findings. A sharply demarcated ulcer may develop on the skin with a membranous base. This latter cutaneous form is found mostly in the tropics but may present in alcoholics and lower socioeconomic populations. The diagnosis is confirmed by Löffler's medium and tellurite agar cultures and Gram stain.

27. What is the therapeutic approach to management of diphtheria?

After ensuring stability of the airway and absence of associated cardiovascular dysfunction secondary to myocarditis, antitoxin should be initiated after intradermal or conjunctival tests for horse serum sensitivity. Concurrently, antibiotics should be initiated with penicillin or with erythromycin in a penicillin-allergic patient. Carriers should be treated with antibiotics.

28. What clinical findings must be present to make the diagnosis of Kawasaki's disease?

It is a multisystem disease occurring predominantly in children younger than 5 years. Kawasaki's disease is also known as *mucocutaneous lymph node syndrome*. The cause is thought to be related to lymphotropic retrovirus, although the epidemiology is undefined. The syndrome is triphasic in clinical presentation. An acute febrile episode (temperature $>$38.5°C for at least 5 days) is accompanied by the appearance of five major diagnostic criteria, at least four of which must be present for confirmation of the typical presentation.

1. Bilateral, nonexudative conjunctivitis usually occurs within 2 days of the onset of fever and lasts up to 2 weeks.
2. Mouth lesions appear 1 to 3 days after onset and possibly last for 1 to 2 weeks. Mouth lesions include erythema, fissuring, crusting of the lips; diffuse oropharyngeal erythema; and strawberry tongue.
3. Peripheral extremity lesions begin after 3 to 5 days and last 1 to 2 weeks. The hands and feet may be indurated. Erythema of the palms and soles is present; desquamation of the tips of fingers and toes occurs 2 to 3 weeks after the onset of illness.
4. Erythematous, polymorphous rash occurs concurrently with the fever and spreads from the extremities to the trunk. It usually disappears within 1 week.
5. Enlarged lymph nodes are present, usually cervical and greater than 1.5 cm.

29. What is the most significant complication of Kawasaki's disease?

The most significant complication is **coronary artery disease** caused by arteritis, aneurysm, or thrombosis. Other findings include diarrhea, vomiting, hydrops of the gallbladder, leukocytosis, cough, proteinuria, arthritis, meningismus, and cerebrospinal fluid pleocytosis. Treatment includes anti-inflammatory agents (i.e., aspirin) and intravenous (IV) immune globulin.

30. What infectious conditions should be considered in a child presenting with diffuse erythroderma?

Several acute infectious entities may present with diffuse erythroderma:

- A scarlatiniform rash caused by group A streptococcus
- A viral illness
- Scalded skin syndrome (*S. aureus*)
- Toxic epidermal necrolysis or erythema multiforme caused by a variety of infections and drugs
- Kawasaki's disease
- Toxic shock syndrome (*S. aureus*)
- Leptospirosis

31. Describe the three stages of clinical progression of a child with pertussis.

Pertussis (or whooping cough) is caused by *Bordetella pertussis*, a gram-negative coccobacilli, occurring in all age groups. It peaks in late summer and early fall with an incubation period of 7 to 10 days.

- Initially, patients have respiratory complaints of fever, rhinorrhea, and conjunctivitis lasting 2 weeks (catarrhal).
- The **paroxysmal phase** follows; unremitting coughing paroxysms, accompanied by vomiting may occur for 1 to 6 weeks. Apnea, pneumonia, pneumothorax, seizures, and hypoxia may complicate the illness.
- In the **convalescent phase**, there is an associated residual cough.

32. What are the typical stages of Reye's syndrome?

Reye's syndrome is an uncommon, acute, noninflammatory encephalopathy with altered level of consciousness, cerebral edema without perivascular or meningeal inflammation, and fatty metamorphosis of the liver, probably secondary to mitochondrial dysfunction. It is a multisystem disease that probably has many associated causes, the findings often being referred to as *Reye-like syndrome*. Salicylate ingestion has been incriminated, especially when occurring in association with chickenpox or influenza. Clinically, patients present with a respiratory or gastrointestinal prodrome followed in several days with an encephalopathic picture that is marked by behavioral changes and a deteriorating level of consciousness. Progression of brain stem dysfunction occurs in a cephalocaudal pattern:

0 Alert, wakeful

I Lethargy. Follows verbal comments, normal posture, purposeful response to pain, brisk pupillary light reflex, and normal oculocephalic reflex

II Combative or stuporous, inappropriate verbalizing, normal posture, purposeful or nonpurposeful response to pain, sluggish pupillary reaction, and conjugate deviation on doll's eye maneuver

III Comatose, decorticate posture and decerebrate response to pain, sluggish pupillary reaction, conjugate deviation on doll's eye maneuver

IV Comatose, decorticate posture and decerebrate response to pain, sluggish pupillary reflexes, and inconsistent or absent oculocephalic reflex

V Comatose, flaccid, no response to pain, no pupillary response, no oculocephalic reflex

KEY POINTS: PEDIATRIC INFECTIOUS DISEASES

1. Infectious diseases represent the most frequent cause of ED visits for children. It is important to differentiate self-limited from life-threatening conditions.
2. Infections in children are often age specific, and their management must reflect the child's age and concurrent medical conditions.
3. Immunizations have changed the pattern of infectious diseases in children.
4. Multisystem infections in children often present with dermatologic findings but require management of potential complications.

WEBSITES

Centers for Disease Control and Prevention: www.cdc.gov

American Academy of Pediatrics: www.aap.org

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EMERGENCY DEPARTMENT EVALUATION OF CHILD ABUSE

CHAPTER 65

Catherine McIlhany, MD, FAAP, and Kathryn Wells, MD, FAAP

1. What is child abuse?

Simply, it is "the physical or mental injury, sexual exploitation, negligent treatment, or maltreatment of a child by a person who is responsible for the child's welfare under circumstances which indicate harm or threatened harm to the child's health or welfare." (Federal Child Abuse Prevention Treatment Act. 42, United States Code 5106g [4]). Specific definitions of abuse can vary across cultures, because they are influenced by social, racial, and ethnic norms and values.

- **Physical abuse**, or nonaccidental trauma (NAT), is any physical injury to a child as a result of acts (or omissions) on the part of the caregivers.
- **Emotional abuse** is a repeated pattern of caregiver behavior that conveys to the child that he or she is worthless, flawed, unloved, unwanted, endangered, or only of value in meeting another's needs. This may include name-calling, intimidation, and harassment.
- **Neglect** occurs when the child's basic needs are not met. This includes denial of basic needs such as medical/dental care, food, shelter, clothing, emotional support, education, or protection.
- **Sexual abuse** is engaging any child in sexual activities that the child cannot comprehend, for which he or she is developmentally unprepared and cannot give informed consent, or which violate the sexual and legal taboos of society. This includes all forms of oral-genital, genital, or anal contact by or to the child. It also includes exhibitionism, voyeurism, and child pornography.

2. How common is abuse?

In 2007, 3.2 million referrals of maltreatment were made concerning 5.8 million children. Of these, nearly 800,000 cases of child abuse were confirmed (approximately 8% of which were sexual abuse), yielding a rate of 10.6 per 1000. Additionally, in 2007, 1,760 children died as a result of abuse and neglect. Among fatalities, 75% were younger than 4 years of age and almost 70% were younger than age 2. This, however, is likely a gross underestimation. Abused children are over-represented in the population presenting for evaluation in the ED, with 1.3% to 15% of childhood injuries seen in the ED caused by abuse.

3. Who is at risk for being abused? Who is at risk for abusing a child?

Child abuse or neglect occurs in all socioeconomic, racial, and ethnic groups and across cultures. However, certain child and family characteristics increase a child's risk of being abused:

Child Factors: These characteristics influence caregivers' perceptions of the child, sometimes causing unrealistic expectations for the child's behavior, which further increase the child's risk.

- Children with special needs
  - Prematurity
  - Developmental delay
  - Behavioral or psychiatric problems
  - Giftedness
  - Chronic medical illness
- Children not biologically related to caregiver (who may feel less emotionally bonded with child)

- Children of unplanned pregnancies
- Gender disparities: physical abuse: male > female victims; sexual abuse: female > male victims

Family or Caregiver Factors:

These include both parental behaviors and dysfunction within the parent-child relationship.

- Lower educational level (less understanding of age-appropriate child behavior)
- Poor social support (causes caregiver isolation)
- Stressors: financial, marital/relationship
- Violence in current home or parental history of family violence
- Substance abuse
- Mental illness

4. What are the *red flags* that suggest child abuse?

Ten red flags must be considered when evaluating children for possible abuse:

- Injury unexplained by history or inconsistent with child's developmental age
- Absent, changing, or evolving history
- Delay in seeking care
- Unusual or concerning caregiver affect
- Triggering event causing loss of control in caregiver (i.e., crying, toilet accident)
- Unrealistic expectation for the child
- Crisis or stress in child's environment
- Social or physical isolation of child or family
- Pattern of increasing severity or escalation of event over time
- Prior history of abuse in caregiver as a child

5. How reliable is a child's disclosure of physical or sexual abuse?

ED providers should take children's disclosures of abuse seriously.

Assume the child is telling the truth and respond accordingly. Your job is to notify authorities for suspicion of abuse, not to prove a child is being abused. As a mandated reporter, you legally must report disclosures to authorities for them to investigate further. Your observations are a single *snapshot* of a child, not an all-encompassing encounter, so you should be careful making assumptions about the *appropriateness* or *likelihood* of a family to abuse a child.

Young children, unless they have extensive exposure to pornography or violence, generally lack the capacity to invent a story of sexual abuse by an adult. They should not have the knowledge of such behavior to falsely accuse someone of such acts. Fabrications regarding abuse are rare and may be a warning sign of other difficulties in the home.

6. What happens if abuse is suspected and not reported?

It is the physician's ethical and legal responsibility to report suspected child abuse; not reporting could result in legal action against the medical provider. As a mandated reporter, your responsibility is to report suspicion of abuse, not to prove abuse is occurring prior to reporting. Social work and law enforcement authorities then investigate reported cases to assess if the suspicion is founded.

7. What if a physician makes a report and investigation reveals that no abuse actually occurred?

The Federal Child Abuse Prevention and Treatment Act (CAPTA) provides immunity from civil and criminal liability for those making reports in good faith. Remember that failure to find sufficient evidence to prosecute does not necessarily mean the physician's suspicions were incorrect. For more information on local and national resources for reporting, responding to, and preventing child abuse, visit the Child Welfare Information Gateway website (*http://www.childwelfare.gov/index.cfm*), a service of the Children's Bureau, Administration for Children and Families, U.S. Department of Health and Human Services.

8. What types of injuries are often seen in children who sustain physical abuse?

- Skin (e.g., bruises, bites, burns, lacerations)
- Skeletal (especially long bone and rib fractures)
- Neurologic (e.g., intracranial hemorrhage, hypoxic brain injury, cerebral edema)

Any injury that doesn't have a history or doesn't fit the history provided should raise your suspicion for abuse. Remember the developmental stages of infancy and childhood, and ask yourself, "Could this child have done what the caregiver is reporting?" For example, a 2-month-old infant cannot roll over, so he or she likely didn't "roll off the couch" and sustain a fracture.

9. What injuries are particularly worrisome for abuse or neglect?

- **Skin injuries**: Always interpret bruises in the context of a child's history and developmental stage. Any bruises on a child who is not independently mobile should raise concerns of abuse. Accidental bruises generally occur on skin overlying bony prominences (e.g., anterior tibia, knee, forehead, scalp). Bruises on areas that are not commonly injured (i.e., ears, neck, lower cheeks, back of hands, chest, abdomen, back, genital area, and backs of legs) are suspicious. Human bites with an intercanine distance greater than 2 cm, immersion burns (well demarcated, often without splash marks) to the extremities or buttocks, loop-shaped bruises (suggesting blows from an electrical cord or belt), or pattern bruises with an impression of any recognizable object (such as a hair brush or hand) are concerning for abuse and warrant further evaluation. Genital bruises should make you suspicious for physical or sexual abuse. Finally, oral injuries in infancy should be considered abuse until proven otherwise (e.g., frenulum tears, posterior pharyngeal lacerations). These are generally caused by a caregiver forcefully inserting an object (e.g., bottle, utensil, pacifier) into the infant's mouth.
- **Fractures very concerning for abuse:** metaphyseal chip; posterior rib; spinous process; complex skull; scapula/sternum (unless major mechanism, motor vehicle crash [MVC]); any fracture in child younger than 2 years of age (especially femur, humerus, or tibia); multiple, complex, diastatic, or occipital skull fractures following report of minor head trauma.
- **Fractures moderately concerning for abuse:** multiple fractures; fractures of different ages; vertebral body fractures; epiphyseal separations.
- **Suspicious closed head injuries:** Suspect physical abuse with any major head injury that occurs after a reported minor trauma (i.e., short fall, stairway fall). Major injuries include: intracranial hemorrhage (subdural hematoma, subarachnoid hemorrhage), retinal hemorrhages, cerebral edema, and sudden, unexplained changes in neurologic status.
- **Other injuries concerning for abuse:** Abdominal injuries (particularly to the liver, duodenum, jejunum, and pancreas) without clear history of significant trauma. Lack of abdominal bruising should not prevent further workup in a child with an abnormal abdominal examination because serious internal injuries can occur without external bruising. Other sites of NAT include: hypopharynx, genital area (scrotal hematomas, penile bruising).
- **Overall appearance:** Always obtain growth parameters in children (i.e., weight, height, head circumference). These can be clues to neglect (e.g., failure to thrive, growth delay from malnutrition).

10. What are metaphyseal fractures, and why are they suggestive of abuse?

Also called *bucket handle*, *corner*, or *metaphyseal chip* fractures, metaphyseal fractures in young children strongly suggest physical abuse. These fractures occur at the junction between the metaphysis and epiphysis, and they are caused by biomechanical forces rarely produced by accidental trauma in infants. They are thought to be caused by rotational ***or*** shearing forces (from shaking or pulling/twisting). Bucket handle fractures and corner fractures are architecturally similar but have slightly different appearances on plain film, depending on angle of view and severity. Remember, however, that all fractures should be interpreted in light of

the child's age, mobility, developmental stage, and history provided. No single fracture is pathognomonic for abuse.

11. What is a skeletal survey? What is its purpose?

A skeletal survey is a series of X-rays designed to evaluate each bone individually, used in cases of suspected abuse in preverbal children. It is rarely used in children older than age 2. **A *babygram* (single anterior-posterior view of infant's body) is not an adequate skeletal survey.** A skeletal survey should include at least two views of the skull, spine, and chest, plus at least one view of the humeri, forearms, hands, femora, tibias, and feet. Most tertiary centers obtain oblique rib X-rays, which are more sensitive for detecting rib fractures. Repeat X-rays (particularly rib films) in 2 weeks often show healing fractures not seen acutely on the original skeletal survey; close follow-up of infants undergoing workup for abuse is therefore important.

12. Is visual dating of bruises accurate?

No. Recent data show that physician estimates of bruises' ages are unreliable. Physicians were only 40% accurate at estimating bruises' ages to within 24 hours of injury. There is no predictable order or pattern of color change within bruising. When a bruise appears yellow, it is likely at least 18 hours old. However, other colors in bruising (i.e., red, blue, black, purple) can occur at various stages of bruising and cannot be used to predict the age of a bruise.

13. Which types of abusive injuries are most likely to be deadly?

Abusive head trauma is the leading cause of child abuse fatalities and includes intracranial hemorrhage, hypoxic-ischemic injury, and cerebral edema. Mortality estimates range from 25% to 30%, with an estimated 1,200 to 1,400 injuries per year. Permanent disability (including blindness, seizure disorder, mental retardation, and cerebral palsy) is seen in the majority of survivors. Visceral injuries due to blunt abdominal trauma are the second most common cause of death in fatal child abuse. Mortality in abdominal NAT is greater than 50%.

14. What is the most common neurologic injury seen in abused infants?

The classic constellation of abusive brain injury includes subdural hemorrhage, traumatic brain injury, and retinal hemorrhages (present about 80% of the time). *Abusive head trauma* or *inflicted traumatic brain injury* have become preferred terms for what was previously called *shaken baby syndrome*. Because multiple mechanisms can cause these injuries, these more inclusive terms are felt to better describe the injuries themselves without suggesting the mechanism. Symptoms can vary between patients, making diagnosis difficult. Patients can present unresponsive and seizing, with obvious external bruises, or they may simply have irritability or vomiting without any bruising visible. Caregivers rarely give a history of inflicted injury, and many cases simply lack any history at all to account for the child's clinical presentation. Given these challenges, it's not surprising that one study showed that 31% of children with abusive head trauma were initially misdiagnosed with such conditions as viral gastroenteritis, reflux, and rule out sepsis. Laskey found that 29% of neurologically asymptomatic young children who underwent head CT for evaluation of suspected physical abuse had occult intracranial hemorrhage. Delays in presentation for care and in diagnosis often result in worse outcomes.

15. Which conditions can mimic injuries seen in child abuse?

- Coagulation disorders
- Bony fragility syndromes (e.g., osteogenesis imperfecta, rickets, scurvy)
- Collagen disorders (e.g., Ehlers-Danlos)
- Other dermatologic conditions (e.g., purpurae/petechiae from infection, mongolian spots, phytophotodermatitis = skin rash at sites of citrus/plant juices on skin exposed to sun)
- Traditional remedies: Coining (e.g., vigorous rubbing with a coin results in linear ecchymoses), cupping (circular suction ecchymoses)
- Accidental injuries (important to obtain detailed history and mechanism)

16. What about an infant who presents dead on arrival without external signs of injury?

Any child who presents in this way should be termed an *unexplained infant death* and should be referred to the coroner's office for further evaluation and investigation. Once a thorough assessment (including a complete autopsy, a review of clinical history, and a death scene investigation) is completed and does not uncover another cause of death, the death may be ruled a sudden unexplained death of infancy (SUDI). This term has largely replaced sudden infant death syndrome (SIDS). The family should still be treated with respect and compassion, as further information may not uncover any other cause for the death or the cause may be a previously unidentified medical illness.

KEY POINTS: CHILD PHYSICAL ABUSE

1. Any fracture in an infant who is not independently mobile should be assumed secondary to physical abuse until proven otherwise.
2. Children younger than 2 years of age should have a skeletal survey performed in cases of suspected physical abuse.
3. Abusive head trauma is the leading cause of child abuse fatalities.

17. What evaluation should children who disclose sexual abuse undergo in the ED?

ED evaluation of sexual abuse depends on when the abuse occurred, what was alleged, and if the child has evidence of trauma on examination. A report of suspected sexual abuse needs to be made to the law enforcement jurisdiction where it allegedly occurred. Furthermore, if the contact is intrafamilial (or there are concerns that the parent is not protective), a report should also be made to the Human Services Department for the county where the child lives.

- **Sexual abuse within the past 72 hours is an emergency.** Child victims need a full physical examination (looking for trauma) and possibly evidence collection, depending on the incident. Any questions about evidence collection should be directed to a regional child abuse specialist/team. Young children rarely need a full *rape kit* performed, and collection should be specific to the allegation and exam and guided by an expert team to minimize further trauma to the child. Vaginal speculum examinations should not be done on a child or virginal teen. Rarely, children with severe genital trauma require sedation for examination and repair in the operating room.
- **Sexual abuse that occurred over 72 hours ago, although distressing to the child and family, is not an emergency.** These patients should have a full physical examination and be referred to their primary care provider or a regional child abuse specialist/team for further outpatient evaluation. Evidence collection is not recommended for children who report an abusive event more than 72 hours ago, as the yield is extremely low. Additionally, a report for investigation should be made.

18. What if no specialist is available?

In teens, do as you would for an adult sexual assault. This means a full physical examination, including genital and rectal examinations, evidence collection with care given to maintain the chain of evidence, and pregnancy test (and prophylaxis), if appropriate. In children, discuss evidence collection with a local child abuse specialist by phone.

19. What is the difference between sexual abuse and sexual play?

Sexual abuse is defined as any sexual activity involving a child who is unable to consent or understand the activity. Sexual play, on the other hand, is defined as unsophisticated acts

between preadolescent children fewer than 4 years apart in the absence of coercion, including bribes or threats.

20. Should a normal physical examination decrease the suspicion of abuse?

No. **A normal physical examination does not confirm or rule out sexual abuse.** The vast majority of children who report sexual abuse have normal physical examinations. Mechanisms of child sexual abuse (e.g., fondling, oral-genital contact) often cause no physical trauma, but the emotional trauma of such incidents may be significant.

21. Should child victims of sexual abuse be treated empirically for sexually transmitted diseases (STDs)?

Prophylactic treatment of prepubertal children reporting sexual abuse is not usually indicated, unless the perpetrator is known or presumed to be infected or sexual contact included genital trauma or contact with bodily fluids. In cases of acute assault with high-risk exposure, prophylactic antibiotics and emergency contraception may be offered as with an adult assault. Consultation with a child abuse specialist/team may be helpful.

KEY POINTS: CHILD SEXUAL ABUSE

1. A normal physical examination does not rule out sexual abuse.
2. Most children do not require STD testing or prophylaxis after sexual abuse.

22. What are the long-term consequences for victims of childhood abuse?

Early identification of abuse and intervention in children's lives is important and likely increases the victim's chances for a positive outcome. Prospective studies have shown lower levels of educational achievement (42% of maltreated children finished high school, compared to 66% of nonmaltreated children). Abused children are more likely to require special education than nonabused children (24% vs. 14%). These associations remained after adjustment for family and social differences. Not surprisingly, adult victims of childhood abuse also held lower-skilled, lower-paying jobs as adults than did controls (62% vs. 45%).

Childhood abuse has significant consequences for childhood and adult mental health. Abused children suffer from increased depression, anxiety, and behavior problems in childhood. Depression in adulthood is more common among victims of childhood abuse; 25% to 33% have major depression by their late twenties, based on *Diagnostic and Statistical Manual* (DSM) criteria. They also have higher rates of post-traumatic stress disorder than controls. Physical and sexual abuse are linked to twice the average risk for suicide attempt in victims studied into their late twenties. Alcohol and possibly drug abuse are more common among adult victims of childhood abuse.

Childhood abuse may ultimately affect adult physical health. Strong evidence suggests that childhood physical abuse, sexual abuse, and neglect are linked to obesity in adulthood, even adjusting for family and social factors. There may be increased rates of promiscuity, STDs, teen pregnancy, and prostitution in adult victims of child sexual abuse. Chronic pain in adulthood may be associated with childhood physical or sexual abuse, but further research is needed. Clearly, childhood abuse has significant physical and emotional effects on survivors and profound effects on society. More research is needed to study the effects of child neglect (the most prevalent type of abuse) on adulthood.

23. What can we do to help childhood victims and prevent abuse?

Most physical injuries in young children heal well; outcome is determined, in large part, by the social management of children in abusive homes. The primary responsibility of physicians

suspecting abuse is to report it and to arrange for safe disposition. It is perfectly acceptable (and in fact, imperative) to admit the child, if no safe disposition can be arranged in the community.

ED providers have critical opportunities to educate families about infant behavior and potentially prevent future child abuse. A provider who explains normal infant crying patterns and coping strategies to the family of a colicky infant may protect that infant from abuse. Giving families encouragement and reassurance that their child's behavior is normal and age-appropriate can help decrease caregivers' frustration and improve their coping skills. Finally, national and local campaigns to educate families about crying behavior in infants may help decrease the chance that a frustrated caregiver will injure a child. Even in a busy ED, providers can, in a short conversation with families, alter the life of a child by either preventing abuse or recognizing it and responding accordingly.

WEBSITE

http://www.childwelfare.gov/index.cfm

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CHAPTER 66

PROCEDURAL SEDATION AND ANALGESIA OF THE PEDIATRIC PATIENT

Joe E.Wathen, MD, and Guy Upshaw, MD

1. Why is it called procedural sedation and analgesia or (PSA)?
What used to be called *conscious sedation* is now more accurately referred to as PSA. This is defined as using sedatives, dissociative agents, and analgesics alone or in combinations to assist patients in tolerating unpleasant procedures, while maintaining cardiorespiratory function. An **analgesic** treats pain, whereas a **sedative** or **anxiolytic** relieves fear and anxiety. Some analgesics, particularly narcotics, have sedative and analgesic properties, which make them useful in certain procedures. If a procedure is painful and frightening (e.g., chest tube insertion, fracture reduction), the child would benefit from both sedation and analgesia.

2. Do I need sedation and analgesia when performing procedures on children?
Children undergoing frightening or painful procedures would benefit from agents providing sedation or analgesia. These procedures include reduction of fractures or dislocations, laceration repair, incision and drainage of abscesses, burn care, examinations after sexual assault, and diagnostic procedures such as lumbar puncture, computed tomography (CT), or magnetic resonance imaging (MRI). Systemic sedatives or analgesics may not be needed in some older children who can remain calm and where local anesthetics provide adequate pain control. A comforting staff or family member may be the needed calming ingredient. Many EDs have also employed *child life advocates* for this very purpose.

KEY POINTS: WHY PROVIDE PROCEDURAL SEDATION AND ANALGESIA TO CHILDREN?

1. Relieve fear and anxiety.
2. Provide analgesia as needed.
3. Provide amnesia for an unpleasant procedure.
4. Facilitate optimal outcome of the procedure.
5. Provide a standard of care now expected and appreciated by most parents.

3. What is brutaine and should I use it?
Brutaine, or simply holding a child down without medications to perform a procedure, although tempting as a fast approach, is not ideal. Using sedation and analgesia helps prevent or reduce crying and thrashing. Not only does PSA allow the provider to have a better chance of actually performing the procedure, but it also provides pain control, reduces anxiety, and in some cases results in amnesia for the event. Continuous crying leaves the child, family, and staff exhausted and appears to onlookers as torture. Sometimes, the addition of a sheet wrap or papoose in combination with sedation is needed. The ability to provide PSA for children is an accepted and expected part of emergency medicine.

4. What are the different levels of sedation?

- **Mild sedation** or anxiolysis refers to very little to no depression of **level of consciousness** (LOC). This is the ideal level for procedural sedation in the older child where anxiolysis alone is needed.
- **Moderate sedation/analgesia,** previously considered *conscious sedation*, is a drug-induced depressed LOC where patients respond to verbal or tactile stimulation while protecting their airway reflexes. The child is still awake but with droopy eyes and slurred speech. Only minimally painful procedures will be tolerated with this level of sedation (i.e., suture repair).
- **Deep sedation/analgesia** implies a depressed LOC from which the child is not easily aroused and may need airway and ventilatory assistance. This level may be needed for more painful procedures (i.e., fracture reduction).
- **General anesthesia** represents the end of this continuum, which many sedatives can achieve if given in sufficient doses. This is not desirable because of the risk of cardiorespiratory depression, loss of airway reflexes, and aspiration.

5. List the ideal characteristics of an agent used for PSA?

- Produces effective anxiolysis, even during painful procedures
- Is safe; produces a predictable degree of sedation for a given dose and has minimal effects on airway reflexes and cardiorespiratory status
- Minimizes movement, facilitating an optimal procedure
- Provides amnesia for the procedure
- Produces no adverse interactions with other agents that may be used concurrently
- Is reversible
- Can be administered painlessly
- Is titratable (advantage of intravenous [IV] administration)
- Has rapid onset, short duration, and rapid recovery (most important)

6. What routes of administration are available for administrating a sedative?

There are several potential routes available for administration of PSA. The route can parallel the depth of sedation needed and the type of procedure to be performed. Routes include oral, transmucosal (i.e., nasal, oral mucosal, rectal), intramuscular (IM), intravascular, or inhalational. IV and inhalational routes allow for the important quality of titrating to effect. However, it may be difficult in some pediatric patients to obtain IV access. In those cases where moderate or deep sedation is needed, the intramuscular route may be ideal (e.g., IM ketamine). Likewise, if anxiolysis or mild sedation is needed, oral or nasal midazolam may be sufficient.

7. What are the key items to ask in the medical history prior to PSA?

- When was the last oral intake of liquids and solids? (aspiration risk)
- Are there allergies to any sedative or analgesic agents?
- What are the current medications? Will there be any interactions?
- Have recent medications (e.g., narcotics) been given? (additive effect)
- Any chronic medical problems (e.g., chronic lung disease or airway abnormalities)?
- Prior complications if received prior sedatives/analgesics or general anesthesia?

8. Are there guidelines for presedation fasting?

There are official guidelines for **elective** procedures per the American Society of Anesthesiologists (ASA) guidelines. However, adherence to these presedation fasting guidelines has not been shown to alter the rate of adverse events. Regarding emergency medicine practice, the majority of ED procedures with indications for PSA are urgent or **emergent** with variable prearrival fasting times. The American College of Emergency Physicians (ACEP) consensus committee has offered clinical practice guidelines for these ED PSA patients. They suggest targeting the depth and length of PSA based on the nature of oral intake 3 hours prior to the procedure, balancing the patient risk factors and the urgency of the procedure.

For truly emergent procedures, the ACEP consensus committee advisory "permits all levels of procedural sedation and analgesia regardless of fasting status or underlying patient risk factors."

9. What physical examination findings are important to note prior to PSA?

- Items to note are the presence of airway abnormalities such as large tonsils or adenoids, congenital abnormalities that may have a floppy or anatomically susceptible airway (Down syndrome, Pierre Robin syndrome, Treacher-Collins syndrome) or lower respiratory findings such as wheezing and rales.
- Obese children may have associated sleep apnea and be at increased risk of adverse respiratory event.
- A visual inspection of the open mouth will tell you what the upper airway looks like (Mallampatti score) and will remind you to look for loose teeth or dental hardware (retainers).
- A careful cardiac and neurologic exam should also be performed.

10. Are there any children who should not receive PSA?

Relative contraindications to procedural sedation in the ED relate to the risk of complications, including aspiration and potential difficulty in managing the airway. Children who may be better candidates for operating room procedures under more controlled conditions include:

- Unstable patients (children with abnormal mental status or hemodynamic instability)
- Infants younger than 6 months old
- Children with craniofacial malformations, such as Pierre-Robin syndrome
- Children with cerebral palsy (abnormal swallowing mechanisms)
- Children with snoring, stridor, apnea, or abnormal breathing regulation
- Children with seizure disorders
- Children with vomiting or gastroesophageal reflux
- Children with severe systemic disease

11. What is the monitoring that should occur with PSA?

The level of monitoring can parallel the degree of sedation. The best monitor is a skilled, dedicated observer who is not involved in the procedure and who can observe the child's level of consciousness, response to verbal and physical stimulation, airway patency, respiratory function, and perfusion. Sedated children should not be left unobserved.

Monitoring and resuscitation equipment may include: cardiorespiratory monitor, pulse oximetry, capnography, blood pressure cuff, suctioning equipment, proper-sized bag-mask ventilation connected to oxygen source, and proper-sized advance airway equipment (i.e., endotracheal tubes and laryngoscope).

12. What are the agents used for pediatric PSA?

See Table 66-1.

13. What agents would you use if you needed to obtain a CT scan on a young child?

Radiologic diagnostic procedures are common and may prove to be difficult to achieve without adequate sedation. The newer CT scanners however, are faster diagnostic procedures with the possibility of being performed without sedatives. If medications are needed, sedatives alone are usually adequate. Potential agents include pentobarbital (Nembutal), midazolam, methohexital, or etomidate. Pentobarbital has been shown to more effectively sedate a child for radiologic imaging (97%) versus midazolam (19%).

14. Would the agents used for obtaining a CT scan work for an MRI?

MRIs are not particularly rapid events, so the child must remain motionless for a longer time period. The ultrashort-acting sedatives would not be the best choice. Instead, agents that can either be continuously infused (propofol) or have a longer duration of action (chloral hydrate) would be preferred.

TABLE 66-1. PROCEDURAL SEDATION AND ANALGESIA AGENTS

Agent	Dose	Route	Comment
Anxiolytics			
Midazolam	0.1 mg/kg	IV, IM	Titrate to effect
	0.3 mg/kg	IN	
	0.5 mg/kg	PO, PR	15 mg max
Sedative Analgesics			
Fentanyl	1–3 μg/kg	IV	Avoid rapid or high-dose infusion
Morphine	0.1 mg/kg	IV, IM	
Meperidine	1.0 mg/kg	IV, IM	
Dissociative Agents			
Ketamine	1–2 mg/kg	IV	Give intravenous dose over 1–2 min
	2–4 mg/kg	IM	Longer recovery
Pure Sedatives			
Pentobarbital	4–6 mg/kg	IM	
	2–4 mg/kg	IV	
Etomidate	0.1–0.2 mg/kg	IV	Ultrashort
Propofol	0.5–1.0 mg/kg*	IV	Rapid onset and offset
Methohexital	1 mg/kg	IV	Ultrashort, limited studies
	20–30 mg/kg	PR	
Chloral Hydrate	50 mg/kg	PO,PR	Unpredictable effect, long duration, avoid in newborns
Inhalational agents			
Nitrous oxide	30%–50% NO_2	Inhalation	Older children, able to hold mask
Reversal Agents			
Naloxone	0.01 mg/kg followed by 0.1 mg/kg if first dose ineffective	IV, IM, ETT	Can repeat every 5 min, 4 mg max
Flumazenil	0.01 mg/kg	IV	Titrate to max of 1.0 mg

ETT, endotracheal tube; IM, intramuscularly; IN, intranasally; IV, intravenously; PO, by mouth; PR, per rectum.
*Can be given as a continuous infusion: 25–150 μg/kg/minute or in additional boluses of 0.5 mg/kg IV every 3 minutes as needed

15. What are the advantages and disadvantages of propofol for PSA?

Advantages	Disadvantages
Sedative hypnotic qualities	Risk of apnea
Rapid onset and offset	Hypoxia-hypoventilation, 2% to 31%
High efficacy	Dose-related hypotension
Amnesia	Lipophilic suspension = pain at injection
Constant infusion for longer procedures	Needs opiate for painful procedures
	Contraindicated with egg or soy allergy

16. What medications would you use for a 2-year-old with a facial laceration?

For the majority of patients, local anesthetics such as topical lidocaine, epinephrine, tetracaine (LET) or local injection with lidocaine is sufficient. The difficulty becomes reducing the child's anxiety. Effective sedation can be provided with midazolam, administered intravenously, intranasal, or orally. When this does not provide adequate sedation and motion control for a difficult repair (i.e., laceration crossing the vermillion border of the lip), an agent such as ketamine either intravenously or intramuscularly works well.

17. What medications would you consider for a 6-year-old needing reduction of an angulated forearm fracture?

Fracture reduction is associated with significant pain and anxiety. Both need to be treated. Several options can be effective and include the following: fentanyl or morphine plus midazolam, ketamine, propofol plus an opiate, or nitrous oxide with a hematoma block. Ketamine has been shown to have fewer adverse respiratory events when compared to fentanyl and midazolam.

18. What makes ketamine or *kidamine* useful as a PSA agent?

Ketamine, a dissociative agent causing a trancelike cataleptic state, has become a more commonly used medication for pediatric PSA. It provides strong sedation, analgesia, and amnesia while maintaining cardiovascular stability and protective airway reflexes. Ketamine onset is within a couple of minutes intravenously and 5 to 10 minutes intramuscularly. Because ketamine can increase salivation, coadministration with an antisialogogue such as atropine was previously advised, however recent studies suggest this is unnecessary, with no increase in adverse airway effects. Coadministration of midazolam has *not* been shown to decrease recovery agitation or emergent phenomena (vivid dreams, hallucinations, delirium), but can decrease recovery emesis, which occurs in 15% to 20%. Ondansetron has also been shown to reduce recovery emesis associated with ketamine. Ketamine, although protective of airway reflexes, may be associated with hypoxia in approximately 5%, and rarely laryngospasm or apnea ($<$1%).

19. What are the contraindications for ketamine?

Glaucoma or globe injury, increased intracranial pressure (ICP) or central nervous system (CNS) mass lesion, seizure disorder, hypertension, congestive heart failure, major psychiatric disorder, porphyria, previous adverse reaction, procedures or conditions that can exacerbate laryngospasm (pharyngeal procedures, endoscopy, upper respiratory infections), or age younger than 3 months.

20. What complications are seen with PSA?

With **oversedation**, there is risk for:

- Respiratory events: aspiration (from vomiting and loss of airway reflexes), hypoventilation, hypoxia, laryngospasm, and apnea
- Cardiovascular events: hypotension, bradycardia
- Vomiting

During the **postsedation** recovery period, children may vomit, become agitated, ataxic, dysphoric, or manifest other *emergence reactions*. In addition, the chance of respiratory depression is increased when the painful stimulus of the procedure is complete. Close observation and parental reassurance is essential. Because of the risks involved, at least verbal informed consent should be obtained and documented.

KEY POINTS: HOW TO AVOID ADVERSE EVENTS WITH PEDIATRIC PROCEDURAL SEDATION AND ANALGESIA

1. Beware of infants, children with systemic disease processes, obstructive airway disease, severe obesity, or active respiratory infections.
2. Become acquainted and comfortable with PSA drug regimens.
3. Verify the weight is in kilograms, not pounds prior to dosing.
4. Monitor carefully, both with equipment and a dedicated medical staff per American Academy of Pediatrics or ACEP guidelines.
5. Be attentive to the end of the procedure when the painful stimulus is over and the child is more prone to developing respiratory depression.
6. Prior to starting PSA, have advanced airway equipment ready including suction, oxygen, and *proper-sized* bag-valve mask.

21. What are the complications associated with fentanyl?

Fentanyl is a commonly used narcotic in the ED because it provides analgesia and sedation with a rapid onset and recovery. However, a few reminders about fentanyl are important. When fentanyl is given rapidly or in high doses, it can cause the *rigid-chest syndrome* (thoracic and abdominal wall rigidity). This muscular rigidity can be reversed by naloxone (Narcan) or with neuromuscular blockade. In addition, fentanyl can cause apnea without the usual concomitant decrease in mental status. Full monitoring is essential, including frequent blood pressure checks.

22. Are some agents safer than others?

Using proper monitoring, most agents can be utilized and adverse events promptly treated; reversal agents are seldom needed. Certain drug types used are associated with different adverse event profiles. See Table 66-2.

TABLE 66-2. ADVERSE EVENTS BY DRUG TYPE

Sedation Drugs	Respiratory Events* (%, OR†)	Vomiting (%, OR†)
Ketamine alone	6%, 1	10%, 1
Ketamine/Midazolam	10%, 1.7	5%, 0.5
Fentanyl/Midazolam	19%, 3.7	2%, 0.2
Midazolam alone	6%, 0.9	0.8%, 0.07

*Respiratory events included hypoxia, laryngospasm, and apnea
†OR represents odds ratio.

23. What reversal agents are available for children?

For narcotics and benzodiazepines, specific reversing agents are available. Naloxone (0.01 mg/kg-0.1 mg/kg intravenously, intramuscularly, or endotracheal, up to 4 mg per dose) reverses narcotic effects, and flumazenil (0.01 mg/kg intravenously, up to 1.0 mg) reverses benzodiazepine overdose.

General measures: Discontinue sedative or narcotic administration. Maintain the airway and provide assisted ventilation, initially with bag-valve-mask ventilation, then with endotracheal intubation if necessary. If poor perfusion or shock is present (e.g., capillary refill time >2 seconds, cool extremities, weak pulses, poor tone), obtain vascular access and initiate treatment with a bolus infusion of 20 mL/kg of crystalloid solution.

24. When can I discharge a child home after performing PSA?

The child should have normal vital signs, be reasonably alert, able to sit without assistance, tolerate liquids by mouth, and respond to commands given in a normal voice.

WEBSITE

Agency for Healthcare Research and Quality. National guideline clearinghouse:
http://www.guideline.gov

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PEDIATRIC AND NEONATAL RESUSCITATION

Katherine M. Bakes, MD

CHAPTER 67

1. What is the pediatric assessment triangle?

The pediatric assessment triangle is a quick tool used to assess small children for end-organ perfusion. The three points of the triangle are work of breathing, general appearance, and circulation to the skin. This provides a rapid way to assess a child in less than a minute, thereby enabling the provider to determine physiologic status with regard to oxygenation, ventilation, perfusion, and brain function.

2. How do I prepare myself for a pediatric arrest coming to the ED?

- Know your equipment. If you use it, review the Broselow tape frequently.
- Be familiar with your difficult airway equipment for children and consider scenarios for use.
- Keep a list of equipment that should be stocked and regularly checked in the event of a pediatric arrest.

Finally, have scheduled mock pediatric resuscitations in your department so that you can identify needed equipment, medications, and process impediments. Resuscitation of a child requires a team effort, wherein everyone needs to be aware of the resources available and specific age- and weight-based needs.

3. Is survival rate after cardiopulmonary arrest better in children or adults?

Due to inconsistencies in terminology, estimating exact survival after cardiac arrest in children has been difficult. Investigators who have studied the two populations in parallel have shown a slightly better predicated survival for adults after out-of-hospital cardiopulmonary arrest. Survival to hospital discharge rates for pediatric arrests range from 2% to 12%, with neurologically intact survival at approximately 2%. These poor outcomes can be explained by the irreversible etiologies of pediatric arrests. Underlying causes of pediatric arrests can be divided into age groups: leading causes of death in children younger than 1 year of age include congenital anomalies, sudden infant death syndrome and sepsis; trauma and respiratory infections leading to sepsis top the causes in children older than 1 year of age. Although outcomes are poor, overall fewer children arrest.

4. What are predictors of outcome in pediatric arrest?

Predictors of mortality include:

- Age younger than 1 year
- Bradyasystolic rhythms
- Need for epinephrine administration and greater than 15 minutes of cardiopulmonary resuscitation (CPR)

A pediatric respiratory arrest with a pulse has an estimated 75% chance of survival, whereas a pediatric patient in pulseless arrest has an estimated survival of 2% to 12%.

5. What are the fundamental differences between the pediatric versus the adult airway?

The pediatric airway is relatively more anterior and cephalad, as well as more malleable due to underdeveloped cartilage and supporting structures. These features make the pediatric airway very susceptible to kinking with flexion and extension maneuvers, a feature the physician should be aware of for proper airway positioning. The younger the child, the more submental

and submandibular tissue and the larger the tongue, making any compression to these areas during bag-valve-mask a greater risk of iatrogenic upper airway compression. The pediatric airway is hour-glass in shape, with the cricoid cartilage composing the narrowest portion of the airway.

In contrast, the adult airway is more conical in shape, with the inlet of the vocal cords being the narrowest portion. Finally, the pediatric epiglottis is bigger, floppier and more omega in shape than the adult. Due to this, for endotracheal (ET) intubation, a straight blade is placed under the epiglottis, allowing the epiglottis to be lifted up such that the vocal cords can be viewed.

6. Does the pediatric patient require cricoid pressure during active bagging?

Yes. It is a common misconception that because the cricoid is the smallest part of the airway, cricoid pressure is not necessary when bagging children. Just like with adults, the reasons for cricoid pressure is to compress the esophagus in patients presenting with a presumed full stomach. In addition, due to children's anatomically smaller thorax, preventing gastric insufflation is essential to prevent the pediatric stomach from distending and impeding ventilation during bagging. Care must be taken to provide adequate pressure for esophageal compression, yet not so much as to prevent bag-valve-mask ventilation. This can be accomplished by gently pushing down on the cricoid ring until ventilation is impeded and then lifting up slightly.

7. How does the approach to the B (breathing) of ABCs differ in pediatrics relative to adults?

Children function with only 40% the functional residual capacity of adults relative to their size. Their metabolic rate is also proportionally higher, and thus their oxygen consumption per minute is higher. As such, they are prone to respiratory stress earlier. In addition to this, the younger the child, the less able they are to change cardiac contractility and thus are primarily dependent on increasing heart rate to maintain blood pressure. The result of all of these is that children can decompensate quickly and suddenly. The practitioner should be aware of subtle changes in mental status and work of breathing, which can herald such an event.

8. What are alternative airway devices that can be used in the pediatric population?

Laryngeal mask airway (LMA) is considered Class Indeterminate in the latest Pediatric Advanced Life Support (PALS) guidelines because most of the literature comes from the adult population. Although a rescue option, the LMA may cause an upper airway obstruction in children under 30 kilograms, as the tip of the airway device can rest in the vallecula and fold the larger pediatric epiglottis into the airway inlet.

Due to the slit-like cricothyroid membrane and the easily transectable airway, an open surgical cricothyrotomy is not recommended in children younger than 8 years of age. Needle cricothyrotomy is a rarely utilized alternative. After a 16- to 18-gauge needle is placed into the airway via the cricothyroid membrane, the lungs are insufflated using a jet ventilation device that allows for controlled pressures of 25 to 30 PSI.

9. What drugs and dosing should I have committed to memory in my armamentarium for pediatric resuscitations?

Epinephrine is the most commonly used first-line medication in pediatric resuscitations. The 1:10,000 (standard dose) epinephrine should be used at 0.1 mL/kg (0.01 mg/kg). 1:1,000 can be considered via the ET route if an intravenous/intraosseous (IV/IO) access has not been established. However, because the ET route has not been proven of benefit, this administration should not delay establishing IV/IO access.

10. At what point should chest compressions be initiated in children?

Chest compression should be initiated for pulseless arrest or when the heart rate is $<$60 beats per minute with evidence of poor end-organ perfusion. Chest compressions should be

performed at 100 per minute at a depth ½ to ⅓ of the anterior-posterior diameter of the chest. Like advanced cardiac life support, PALS emphasizes continuous, hard, and fast chest compressions with minimal interruptions.

11. Where should I try for vascular access in the pediatric patient?

Vascular access should be first attempted in the peripheral veins in a child with a blood pressure. If a child presents in cardiac arrest, the provider should immediately perform IO access placement. The anterior proximal tibia is the easiest site of entry in most circumstances. Care should be made to angle the needle just off 90 degrees away from the growth plate. In the young neonate, the distal femur may be a more stable and less easily breakable bone to perform IO placement. For central venous access, the femoral vein is the most commonly used site in children. Whatever the location for central venous access, ultrasound guidance if available should be used to facilitate placing these lines, both in adults and children.

12. What is the earliest gestational age that a newborn has been successfully resuscitated after birth?

In 2006, at 21 weeks and 6 days gestational age and weighing less than 10 ounces, Amillia Sonja Taylor was successfully resuscitated after birth.

13. What spontaneous activity should you expect in a normal newborn at the time of birth?

Almost all infants have a grimace-like facial expression at birth and make some attempt to move the upper and lower extremities. Infants will almost always make an effort to breathe spontaneously and cry within 15 to 20 seconds following birth.

14. After delivery in the ED, what is the first priority in the care of the newborn?

The first priority is to prevent body heat loss. The infant must be dried immediately to prevent evaporative heat loss and be placed under a radiant warming unit to lessen loss of radiant heat. These initial steps take only seconds to accomplish and may prevent serious metabolic derangements. These steps should be taken even prior to the initiation of CPR.

15. When should central cyanosis resolve in a healthy newborn following delivery?

Central cyanosis and cyanosis of the oral mucous membranes should clear after the first minute of life. Peripheral cyanosis of the hands and feet may persist for several minutes in an otherwise healthy newborn. Peripheral cyanosis restricted to the hands and feet is referred to as acrocyanosis and usually has no clinical significance.

16. How do I approach the meconium-stained newborn?

Meconium (either thin or thick) presents a real and serious risk to the respiratory system of the infant if it is aspirated. The material contains noxious substances such as bile acids that can lead to pulmonary injury. The latest neonatal resuscitation guidelines call for ET intubation and suctioning of meconium prior to bulb suctioning in newborns that are non-vigorous, defined as weak respiratory efforts, poor muscle tone, and a heart rate $<$100.

17. After the infant is dried, suctioned, and placed under the warmer, how do I decide whether further active intervention is needed?

If the infant is active, is crying, has a heart rate of greater than 100 beats per minute, and does not have evidence of central cyanosis, further intervention is seldom needed. If the infant demonstrates apnea, bradycardia, or central cyanosis, then use of bag and mask ventilation must be considered after attempts at stimulation have failed. Most of the time, if the infant is near term gestation, the heart rate will respond quickly to a few effective assisted breaths.

18. For the newborn, define bradycardia and indications for intervention.

A heart rate of less than 100 beats per minute at 30 seconds following birth is considered bradycardia in the newborn. Positive pressure ventilation and oxygenation should be initiated if the newborn is cyanotic or has a heart rate of <100 after 30 seconds of drying, stimulating and giving blow by oxygen.

If the heart rate is below 60 beats per minute after providing respiratory support, the provider should initiate chest compressions at a rate of 100 per minute using a two-finger chest encircling technique. Avoid pressure over the liver because a liver laceration can occur. Heart rate should be evaluated about every 30 seconds. Compressions should be performed in between breaths. When a sustained heart rate of greater than 100 beats per minute is achieved, compressions can be discontinued.

19. How many infants will require intubation to provide adequate ventilation?

Almost all newborn infants can be ventilated with bag-mask technique. Bag-mask ventilation should be done only with equipment designed for newborn and premature infants. The most effective bag for this type of assistance is an infant self-inflating unit connected to wall oxygen. There is currently a great deal of discussion in the literature as to whether 100% oxygen is the safest gas for this type of intervention. For the time being, in the setting of the ED, 100% oxygen is the best choice until this debate is resolved. To avoid barotrauma, inflation pressures should not exceed 40 cm H_2O in the term and 25 cm H_2O in the preterm newborn. All neonatal bags should be equipped with pressure manometers.

20. When should I attempt vascular access? What vessel should I use?

As soon as it is fairly obvious that drugs or volume expanders may be needed, an umbilical venous line (3.5 French for preterm and 5.0 French for term infants) should be attempted. It is uncommon for this to be needed. For such events, an umbilical venous tray should always be available in the ED. Remember that there is one larger umbilical vein and two umbilical arteries (Table 67-1).

21. What drugs should be available for use in newborn resuscitation use, and when should they be given?

Drugs are rarely needed in the resuscitation of the newborn infant, especially if bag-and-mask ventilation is started early. Usually, no more than two agents are needed.

- **Epinephrine 1:10,000 dilution** is used if there has been no heart beat noted for 6 to 10 seconds at any point during the event or if heart rate remains less than 60 beats per minute after 30 seconds of bag-and-mask ventilation and chest compressions. The standard dose

TABLE 67-1. CENTRAL VENOUS CATHETER SIZE BASED ON PATIENT AGE, LENGTH/HEIGHT, AND WEIGHT

Age	Weight (kg)	Length/Height (cm)	French Size
Premature	≤2.5	<50	2.0–2.5
Birth–30 days	3–4	50–55	3.0
>1 mo–1 yr	4.5–10	5–75	3.0–4.0
>1–7 yr	11–25	7–120	4.0–5.0
>7–15 yr	26–60	125–175	5.0–8.0

From Nadel FM: Vascular access. In Baren JM, Rothrock SG, Brennan J, et al, editors: *Pediatric emergency medicine*, Philadelphia, 2008, Saunders.

is 0.1 to 0.3 ml/kg (0.01 to 0.03 mg/kg), either via the umbilical vein (preferable) or via the ET tube. The drug can be given as frequently as every 5 minutes if bradycardia persists.

- **Volume expanders** may be used via the umbilical vein catheter (UVC) if there is evidence of blood loss from the infant. Rapid volume expansion must be done with caution in infants less than 32 weeks' gestation because of the risk of central nervous system (CNS) bleeding. The usual agents used are normal saline or 5% albumin in a dose of 10 mL/kg.

22. What is the best means of documentation of the results of resuscitation in the neonate?

The Apgar score remains the standard, despite some limitations (Table 67-2). The score is calculated at 1 minute and at 5 minutes and at every 5 minute interval thereafter.

KEY POINTS: PEDIATRIC AND NEONATAL RESUSCITATION

1. For preparedness of the critical child, all caregivers should be familiar with their ED pediatric resuscitation algorithms, equipment and support resources.
2. Due to underlying etiologies, pediatric patients in cardiac arrest have poor prognoses.
3. The highest impact intervention for a newborn or pediatric resuscitation is appropriate respiratory support.

TABLE 67–2. APGAR SCORING SYSTEM

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

bpm, beats per minute.

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GENERAL APPROACH TO POISONINGS

Katherine M. Hurlbut, MD

1. List the 15 most common causes of death from acute poisoning reported to poison centers.

- Sedative/hypnotics/antipsychotics 30.4%
- Opioids 26.7%
- Antidepressants 17.8%
- Acetaminophen in combination 16.8%
- Cardiovascular drugs 16.4%
- Stimulants and street drugs 15.2%
- Alcohols 13.7%
- Acetaminophen alone 11.3%
- Anticonvulsants 8.0%
- Fumes/gases/vapors 6.5%
- Cyclic antidepressants 6.5%
- Muscle relaxants 5.7%
- Antihistamines 5.6%
- Aspirin alone 5.1%
- Chemicals 3.6%

Note: Despite a high frequency of involvement, these substances are not the most toxic but rather may be the most readily accessible.

Percentages total more than 100% because multiple substances are involved in some fatal exposures. Percentages are based on the total number of human exposures rather than the total number of substances.

2. What is the current role of syrup of ipecac in treating acute poisoning?

Although syrup of ipecac induces vomiting within 20 to 30 minutes in most people who are given a therapeutic dose, little poison is removed; there are more effective means of decontaminating the gastrointestinal (GI) tract. Ipecac may have a limited role in treating children at home and who frequently can be given a dose soon after ingestion; however, its use in the prehospital setting is rapidly declining (use of ipecac recommended in 0.07% of all poison center cases in 2007). By the time most patients present to a hospital, too much time has elapsed for syrup of ipecac to be of benefit. Its use also delays the administration of activated charcoal, which needs to be given as quickly as possible for maximal benefit.

3. What is the current role of gastric lavage in treating acute poisonings?

Gastric lavage has not been shown to alter clinical outcome in large series of patients presenting with overdose. Although serious sequelae of gastric lavage are rare, it carries the risk of aspiration, laryngospasm, and esophageal injury. The risk of injury appears to be greater in uncooperative patients. Endotracheal intubation should precede gastric lavage in patients with altered mental status or the inability to protect the airway. Although lavage can be accomplished without prior tracheal intubation in most patients, airway equipment, including suction, should be immediately available at the bedside. Placing the patient on the left side in mild Trendelenburg position helps prevent aspiration if vomiting occurs. Nasogastric tubes are too small to remove pills or large pill fragments; whenever gastric lavage is done, a large-bore tube (36 French or 40 French in adults) should be placed through the mouth. A bite-block with an oral airway prevents the patient from biting the tube. Proper location of the lavage tube in the stomach must be verified clinically or radiographically before lavage or administration of fluid or charcoal. Deaths have been reported resulting from charcoal instillation into the trachea by nasogastric tube. Gastric lavage generally is reserved for the small number of patients with potentially serious or life-threatening overdose

who present within 1 to 2 hours after ingestion. It was used in 0.36% of poison center cases in 2007.

4. What is the current role of activated charcoal?

Activated charcoal has been shown in numerous studies to be superior to gastric emptying procedures for the treatment of acute overdose. Gastric emptying procedures involve time and some risk to the patient. The time involved in lavaging the patient is time during which drugs are being actively absorbed. By giving a dose of activated charcoal immediately on patient presentation, the most effective means of GI decontamination already has been performed. Not all drugs are adsorbed to charcoal, however. Drugs that are not well adsorbed include lithium, potassium, iron, some metals, and alcohols. Activated charcoal is contraindicated after ingestion of hydrocarbons because toxicity from gastric absorption is generally not a major concern with these substances, and activated charcoal may induce vomiting, which increases the risk of aspiration pneumonitis. Activated charcoal is also not indicated after ingestion of acids or alkalis because the primary toxicity associated with these agents is local mucosal burns rather than systemic absorption. Patients with trivial ingestions (generally children) do not require activated charcoal therapy.

5. What about the asymptomatic overdose patient?

It has been advocated by some that simple observation of asymptomatic overdose patients, with treatment only if symptoms develop, is a management option. Although this approach is safe for many patients who have ingested trivial overdoses, if a patient ingested something quite toxic, an opportunity to prevent absorption may have been lost if nothing is done until symptoms develop. Administering a dose of activated charcoal to all patients with a history of deliberate drug overdose is done easily (although it is often messy) and helps to ensure safe and timely patient disposition. If a reliable history indicates ingestion of substances with minimal toxicity, activated charcoal may not be necessary.

6. Is there a role for cathartics in treating acute poisoning?

The theory behind cathartics is that they speed up GI transit time, allowing activated charcoal to catch up with pills in the bowel and prevent desorption of drug from activated charcoal. Cathartics have not been shown to reduce drug absorption or improve outcome significantly after overdose, but they can cause vomiting, abdominal pain, and electrolyte abnormalities. Use of cathartics is *not* warranted.

7. What is the current role of whole-bowel irrigation in the treatment of acute poisoning?

Whole-bowel irrigation uses a polyethylene glycol electrolyte solution, such as GoLYTELY or Colyte, which is not absorbed, and flushes drugs or chemicals rapidly through the GI tract. This procedure seems to be most useful when radiopaque tablets or chemicals have been ingested because their progress through the GI tract can be monitored by radiography. It should also be considered when toxic amounts of substances that are not well adsorbed by activated charcoal (i.e., iron, lithium, heavy metals) are ingested. This procedure also is commonly used when multiple packets of street drugs, such as heroin or cocaine, have been ingested and need to be passed through the GI tract as quickly as possible, and should be considered after overdose of sustained-release products. The limitations of the procedure are that, unless the patient is awake, cooperative, and able to sit on a commode, there is a risk of vomiting and aspiration in addition to the logistical problem of having an unconscious patient in bed with massive diarrhea.

8. What is the role of multiple-dose charcoal in the treatment of acute poisoning?

Multiple-dose charcoal has been shown to enhance the elimination of many drugs that already have been absorbed from the GI tract or that are given intravenously. This process has been called **gastrointestinal dialysis** and has been shown to be effective for theophylline and

perhaps phenobarbital poisoning. Numerous other drugs have been shown to have their pharmacokinetics altered by multiple-dose charcoal, but it is not clear if this makes a difference in clinical outcome. Many of these drugs have large volumes of distribution, and increasing elimination of the small amount present in the blood is unlikely to be of benefit. Multiple-dose activated charcoal is used most commonly after overdose of theophylline, phenobarbital, carbamazepine, and quinine.

9. Is forced diuresis of benefit in the treatment of acute poisoning?

Few drugs are excreted unchanged in the urine so that even increasing urine flow significantly above baseline is unlikely to be of benefit. By manipulating the pH of the urine with infusions of bicarbonate solution along with enhanced urine flow, however, drug elimination can be increased in certain cases. This most commonly is used for salicylates and phenobarbital. By placing three ampules of sodium bicarbonate in 1 L of D_5W along with potassium chloride and infusing this solution at rates sufficient to produce at least a normal urine flow and a urine pH of 7.5 or greater, the elimination of salicylate and phenobarbital can be increased. Intake and output and urine pH should be monitored hourly with a Foley catheter in place. In the presence of pulmonary or cerebral edema, which may occur in severe salicylate intoxication, alkaline diuresis is dangerous and should not be undertaken.

Alkaline diuresis also may work in a similar manner for chlorophenoxy herbicides, but acute poisonings by these agents are rare. The use of high-volume normal saline to treat lithium intoxication is common, and it is important to maintain adequate urine output and serum sodium in this scenario. It is not clear, however, that forced-saline diuresis for lithium intoxication is of extra benefit over simply ensuring normal renal flow.

10. When are extracorporeal techniques, such as hemodialysis or hemoperfusion, indicated?

Drugs can be removed successfully by extracorporeal maneuvers only if they have relatively small volumes of distribution and are found in significant quantities in the circulation, as opposed to having rapid and thorough tissue distribution. This is the case for only a few drugs. In practice, the toxins most commonly dialyzed after overdose include aspirin, lithium, methanol, ethylene glycol, and perhaps theophylline. Dialysis has the advantage over charcoal hemoperfusion in that it is usually easier and faster to get started, and it can correct fluid and electrolyte abnormalities as it removes drugs. Because protein binding may be saturated in overdose, hemodialysis may be effective for treatment of severe overdose of some drugs that are highly protein bound at therapeutic concentrations. As protein binding is saturated, increasing quantities of drug are present as free, unbound drug in the serum, and may be removed by hemodialysis (one example is valproic acid).

Charcoal hemoperfusion may be more effective at removing drugs that are highly bound to plasma proteins, because the affinity for charcoal may be higher than the affinity for the protein carrier. The disadvantages of hemoperfusion are that it is less widely available, it frequently causes hypocalcemia and thrombocytopenia, and it can result in frequent canister clotting. Drugs for which charcoal hemoperfusion is frequently employed include theophylline, phenobarbital, and a few other less common agents such as paraquat and amatoxin.

11. How can the diagnosis of a drug overdose be made when the patient is unconscious and history is unavailable?

The diagnosis of acute overdose is difficult to make sometimes and requires some detective work on the part of the physician. All unconscious patients should receive a rapid bedside serum glucose determination (or intravenous dextrose if bedside glucose measurement is unavailable); naloxone should be administered if the presentation is consistent with opioid overdose (central nervous system [CNS] and respiratory depression, miosis); a positive response to either is diagnostic. Whenever possible, examine the pill bottles available to the patient, review medical records, and interview family and friends to determine prescribed

drugs. It may be useful to call the pharmacies where the prescriptions were filled to determine if other prescriptions were filled there for different drugs. Discovering which chemical agents were available to the patient, including street drugs, is always important. If needle track marks are seen, consider street drugs commonly used intravenously, such as opiates, cocaine, and amphetamine. The physical examination is useful in narrowing the diagnosis to a class of drug or chemicals. This concept is commonly called **toxic syndromes** (Table 68-1) or toxidromes.

12. How can a toxicology screen and other ancillary laboratory tests make the diagnosis of acute poisoning?

The blood and urine toxicology screen should be done on any patient who has significant toxicity (persistent altered mental status or abnormal vital signs) and when the diagnosis is uncertain. Alternatives to a full toxicology screen include testing discrete serum levels of the toxins in question, doing a urine qualitative test for drugs of abuse, or drawing specimens but holding them until it is determined that a toxicology screen is definitely indicated. The results of urine toxicology screens rarely alter patient management; routine use is *not* warranted.

More drugs and chemicals *are not* found on typical toxicology screens than *are* found on the screens, although most drugs that commonly are ingested are found on comprehensive toxicology screens. It is important to communicate with the laboratory about which drugs are suspected, which drugs the patient takes therapeutically, and the clinical condition of the patient. Whenever there is a discrepancy between clinical suspicion and findings from the toxicology screen, it is useful to communicate with the toxicology laboratory personnel and determine if other tests are likely to be of benefit. Toxicology screens are expensive, frequently are inexact, and frequently do not give all the information that is expected by the clinician. It is important to interpret toxicology screens carefully and to know which drugs and chemicals were not screened for.

13. What other studies are useful in the evaluation of a poisoned patient?

- An **acetaminophen level** should be obtained in patients with deliberate overdose because this substance is widely available, frequently involved in overdoses, and causes little in the way of initial symptoms; and treatment with *N*-acetylcysteine is most effective if begun within 8 hours of ingestion.
- **Nontoxicologic laboratory tests** that are frequently useful include an electrocardiogram (ECG), which can help diagnose overdose of tricyclic antidepressants or cardiac medications; a chest radiograph in patients with pulmonary symptoms or hypoxia, which if demonstrative of acute lung injury would make one think of salicylates or opiates; and, rarely, a kidneys-ureters-bladder (KUB) screen, looking for radiopaque material, which would make one suspicious of ingestion of a heavy metal, iron, phenothiazines, chloral hydrate, or chlorinated hydrocarbon solvents.
- **Liver enzymes** may help to diagnose ingestion of hepatotoxins, such as acetaminophen or carbon tetrachloride late in the course of poisoning.
- A **urinalysis** may show the presence of calcium oxalate crystals, suggesting the diagnosis of ethylene glycol poisoning.
- The **acid-base status** of the patient is important and should be evaluated in all patients with deliberate overdose. Persistent unexplained metabolic acidosis always should prompt the search for other diagnostic clues to aspirin, iron, methanol, or ethylene glycol poisoning. Many other drugs can cause a persistent, unexplained metabolic acidosis, including the ingestion of acids themselves, cyanide, carbon monoxide, theophylline, and others.
- In the work-up of persistent acidosis, **a serum osmolality** done by freezing point depression can be useful *if* it is elevated. A difference between the measured osmolality and the calculated osmolality of greater than 10 is always significant, although a normal osmolal gap does *not* rule out toxic alcohol ingestion.

TABLE 68-1. MOST COMMON TOXIC SYNDROMES

Syndrome	Common Signs	Common Causes
Anticholinergic	Agitated delirium, often with visual hallucinations and mumbling speech, tachycardia, dry flushed skin, dilated pupils, myoclonus, temperature slightly elevated, urinary retention, decreased bowel sounds. Seizures and dysrhythmias may occur in severe cases.	Antihistamines, antiparkinsonism medication, atropine, scopolamine, amantadine, antipsychotics, antidepressants, antispasmodics, mydriatics, skeletal muscle relaxants, many plants (most notably jimson weed)
Sympathomimetic	Delusions, agitation, paranoia, tachycardia, hypertension, hyperpyrexia, diaphoresis, piloerection, slight mydriasis, hyperreflexia. Seizures and dysrhythmias may occur in severe cases.	Cocaine, amphetamine, methamphetamine (and derivatives MDA, MDMA, MDEA), over-the-counter decongestants (phenylpropanolamine, ephedrine, pseudoephedrine). Caffeine and theophylline overdoses cause similar findings secondary to catecholamine release, except for the organic psychiatric signs.
Opiate/sedative	Coma, respiratory depression, miosis, hypotension, bradycardia, hypothermia, acute lung injury, decreased bowel sounds, hyporeflexia, needle marks	Narcotics, barbiturates, benzodiazepines, ethchlorvynol, glutethimide, methyprylon, methaqualone, meprobamate
Cholinergic	Confusion/central nervous system depression, weakness, salivation, lacrimation, urinary and fecal incontinence, GI cramping, emesis, diaphoresis, muscle fasciculations, pulmonary edema, miosis, bradycardia (or tachycardia), seizures	Organophosphate and carbamate insecticides, physostigmine, edrophonium, some mushrooms (*Amanita muscaria, Amanita pantherina, Inocybe, Clitocybe*)
Serotonin	Fever, tremor, incoordination, agitation, mental status changes, diaphoresis, myoclonus, diarrhea, rigidity	Fluoxetine, sertraline, paroxetine, venlafaxine, clomipramine; the preceding drugs in combination with monoamine oxidase inhibitors

GI, gastrointestinal; MDA, methylenedioxyamphetamine; MDEA, methyl diethanolamine; MDMA, 3,4-methylenedioxymethamphetamine.

KEY POINTS: MANAGEMENT OF SUSPECTED TOXIC INGESTION

1. Activated charcoal is sufficient decontamination for most overdose patients.
2. Urine toxicology screens are not indicated in patients with normal mental status and vital signs.
3. Serum electrolytes and acetaminophen concentration should be obtained in patients with deliberate overdose.
4. Although there are a few antidotes for specific toxins, most poisoned patients recover with supportive care.

14. Discuss some other useful antidotes for common poisonings.

- **Naloxone** and **dextrose** are the most common antidotes and should be given routinely to unconscious overdose patients. Intravenous administration of 2 mg of naloxone that results in awakening of the patient is diagnostic of acute opiate overdose. Small, incremental doses of 0.2 mg can be used if it is suspected that the patient is opioid dependent, because the 2-mg dose of naloxone will precipitate withdrawal. Many drugs and chemicals can cause hypoglycemia, including ethanol, and for this reason dextrose likewise should be given, unless it can be determined quickly that the blood glucose is normal.
- **Physostigmine** is an antidote for the anticholinergic syndrome. Physostigmine can be used diagnostically and therapeutically when the diagnosis of the anticholinergic syndrome is suspected. It should *not* be used to treat tricyclic antidepressant poisoning (or in patients with ECG changes suggestive of tricyclic antidepressant poisoning such as QRS widening or a large R wave in AVR). Seizures and bradydysrhythmias have been reported when used in this setting. A dose of 1 to 2 mg given slowly intravenously to an adult is usually sufficient.
- **Digoxin immune Fab (Digibind, Digitab)** is a safe and effective antidote for digitalis glycoside poisoning and can rapidly reverse dysrhythmias and hyperkalemia, which can be life-threatening. In contrast to naloxone, Digibind does not work immediately, and a full response to therapy may not be seen until approximately 20 minutes after administration. For a life-threatening digitalis overdose when the dose and the serum level are currently unknown, 10 vials of Digibind should be given.
- **Atropine** and **pralidoxime (Protopam)** are antidotes used for cholinesterase inhibitor toxicity. This group of pesticides includes the organophosphates and carbamates, which commonly are found in household insecticides. Atropine is used to dry up secretions, primarily pulmonary, and pralidoxime is used primarily to reverse the skeletal muscle toxicity of these agents, including weakness and fasciculations.
- **Flumazenil** is a benzodiazepine antagonist that has been shown to be useful in cases of acute benzodiazepine overdose resulting in significant toxicity. Its use may precipitate benzodiazepine withdrawal, including seizures. It should not be used when tricyclic antidepressants or other pro-convulsants have been coingested with benzodiazepine. The usual adult dose is 0.2 mg followed in 30 seconds by 0.3 mg, followed in 30 seconds by 0.5 mg, repeated up to a total of 3 mg.
- **Ethanol and fomepizole** are alcohol dehydrogenase blocking agents that are used to treat methanol and ethylene glycol poisoning. They prevent the metabolism of methanol and ethylene glycol to their toxic metabolites. Intravenous ethanol is less expensive than fomepizole but is somewhat more difficult to use. The initial intravenous dose is 8 mL/kg of 10% ethanol over 30 minutes, followed by an infusion of 0.8 mL/kg/h in a nondrinker, 1.4 mL/kg/h in an average drinker, and 2 mL/kg/h in a heavy drinker. Blood ethanol concentration should be measured immediately after the loading dose and repeated every hour initially, and the dose adjusted to maintain a blood ethanol of 100 to 125 mg/dL. The

loading dose of fomepizole is 15 mg/kg intravenously over 30 minutes with subsequent doses of 10 mg/kg every 12 hours. The dose of both agents must be increased in patients undergoing dialysis.

- ***N*-acetylcysteine** is extremely effective in preventing acetaminophen-induced liver injury. It is most effective if administered within 8 hours of ingestion but reduces morbidity and mortality even in patients with acetaminophen-induced acute liver failure. It can be administered orally (loading dose 140 mg/kg, subsequent doses 70 mg/kg every 4 hours) or intravenously (initial dose 150 mg/kg in 200 ml D_5W over 15 minutes, followed by 50 mg/kg in 500 ml D_5 over 4 hours, followed by 100 mg/kg in 1 L D_5W infused over 16 hours).

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CHAPTER 69

THE ALCOHOLS: ETHANOL, ETHYLENE GLYCOL, METHANOL, AND ISOPROPYL ALCOHOL

Louis J. Ling, MD

1. Is a patient with altered mental status, who smells of alcohol, simply intoxicated?

In most cases, yes. However intoxicated patients are at increased risk for injury, immunosuppression, poor nutrition, poor thermal regulation, and many medical conditions. Every patient assumed to be drunk (only) needs a thoughtful initial evaluation and subsequent serial evaluations.

2. How should intoxicated patients be evaluated?

Patients who arrive primarily with intoxication and without a history of trauma or injury should have vital signs taken; a rapid scan for recent trauma; a rapid glucose determination; and assessment for level of consciousness, responsiveness, and respiratory depression. Examination should include a look for unequal pupils, ecchymosis, deformity, and palpation everywhere for abdominal and bony tenderness. If no concerns are identified, it is important to return and reexamine the patient every hour or two to ensure that the patient is improving.

3. When should an acutely intoxicated patient be intubated?

Hypopnea and hypoventilation are rarely the issue, but the inability of the patient to protect the airway is. For patients who are heavily intoxicated but not deemed to require intubation, lateral decubitus positioning is preferred. Restraining a patient supine or prone can be dangerous because of the risk of aspiration and airway compromise.

4. Which medications are best for management of alcohol withdrawal?

Benzodiazepines, usually diazepam or lorazepam, can be given orally, intravenously, or in combination and titrated by clinical response. Patients with mild withdrawal (normal vital signs, no hallucinosis) may be discharged with a 2- to 3-day course of a single agent (e.g., lorazepam, 1–2 mg twice a day). Haloperidol is an appropriate adjunct for hallucinosis. Theoretical concerns over haloperidol lowering seizure threshold and exacerbating hemodynamic abnormalities have not been substantiated. More severe withdrawal syndromes require increasingly aggressive therapy with these same agents while under observation by medical personnel.

5. What is an appropriate work-up for alcohol withdrawal seizures (AWDs)?

Typically, AWDs occur approximately 6 to 96 hours after the last drink and in clusters of one to four seizures. The seizures are usually generalized and self-limited. Coincident features of withdrawal may be lacking, and lateralizing findings during the seizure, the postictal state, or both, are often present because of underlying structural pathology.

In a first-time evaluation, other causes of or contributors to seizures should be sought. Laboratory studies (i.e., electrolytes, glucose, magnesium, calcium, toxicologic screen) are rarely useful unless history or physical examination is suggestive. Noncontrast computed tomography (CT) shows traumatic, infectious, vascular, or other abnormalities in nearly 10% of patients Generally, electroencephalography is not integral to the work-up. Lumbar puncture is indicated when meningitis, meningoencephalitis, or subarachnoid hemorrhage is suspected.

Subsequent visits for suspected AWDs demand scrupulous history and physical examination to ensure that other pathologic causes have not developed in the interim. If the presentation

matches prior episodes and the findings on current neurologic examination are baseline, no other work-up, including CT, is necessary. Lingering postictal confusion warrants a check of glucose and electrolytes. If the history or examination has changed significantly or is worrisome, the clinician should start from scratch.

6. How should AWDs be managed?

- **Acute**: As with all seizures, ensure a patent airway and administer 50% dextrose (D_{50}) and benzodiazepines intravenously (as needed). An observation period of 6 hours is optimal because recurrent seizures are common within this period. Benzodiazepines in the immediate and 2-day postseizure period decrease the incidence of additional seizures during this time.
- **Chronic**: Patients whose seizures have an epileptogenic focus (e.g., old subdural) should have an anticonvulsant, such as **phenytoin**, administered. However, compliance is typically poor. In the patient with pure AWDs, long-term anticonvulsant therapy is *contraindicated*. Physicians must resist the imperative to *prescribe something* unless there is clear justification.

7. Can AWDs be prevented?

Benzodiazepines in the acute withdrawal period, particularly in patients with a history of AWDs, can decrease seizures.

8. Who is at risk for alcohol-induced hypoglycemia (AIH)? What is the clinical presentation?

AIH results from:

a. Insufficient glycogen stores
b. Alcohol-induced impairment of gluconeogenesis

The three groups vulnerable to AIH are:

a. Chronic alcoholics
b. Binge drinkers
c. Young children

AIH may occur during intoxication or up to 20 hours after the last drink. Manifestations of neuroglycopenia (e.g., headache, depressed mental status, seizure, or coma) predominate. Evidence of catecholamine excess, typical of insulin-induced hypoglycemia (tremulousness, diaphoresis, anxiety), is unusual. Seizures are a frequent presentation in children. Localized central nervous system signs, including a stroke-like picture (alcohol-induced hypoglycemic hemiplegia), often occur in adults.

9. What causes alcoholic ketoacidosis (AKA)?

This common metabolic disturbance occurs early after heavy binge drinking and is heralded by starvation and vomiting and occasionally shortness of breath (Kussmaul respirations) and abdominal pain. Ketoacidosis results from accumulation of acetoacetate and, particularly, β-hydroxybutyrate. Because the latter is not measurable on routine blood and urine tests, the patient may have trace or absent ketones at presentation. Similarly, as the patient improves and β-hydroxybutyrate is metabolized to acetoacetate, there may be a paradoxical spike in urine and serum ketones.

At presentation, serum pH and bicarbonate average 7.1 and 10, respectively. These values vary widely because of the frequently overlapping ketoacidosis (metabolic acidosis), withdrawal-related hyperventilation (respiratory alkalosis) and protracted emesis (metabolic alkalosis). When all three are coincident, the result is a triple acid-base disturbance. This allows you to interpret arterial blood gases and electrolytes pretty much any way you wish and be at least partially correct. Decreased body stores of potassium and phosphate are typical. In AKA, serum glucose is usually normal or low, a distinguishing feature from diabetic ketoacidosis.

10. How should AKA be managed?

Treatment consists of rehydration with dextrose-containing crystalloid, antiemetics if needed, benzodiazepines for withdrawal, and multivitamins, potassium, and phosphate as indicated. Bicarbonate is rarely required, and insulin therapy is proscribed. Metabolic abnormalities usually resolve with 12 to 16 hours of therapy.

11. What is the relationship between alcohol and metabolic acidosis?

- **Ethanol**: Acute ethanol ingestion results in a mild increase in the lactate-to-pyruvate ratio. Clinically significant metabolic acidosis does not ensue.
- **AKA**: This ethanol abstinence syndrome produces marked elevations in acetoacetate and β-hydroxybutyrate with resultant and occasionally profound increased anion gap metabolic acidosis. During the correction phase, a non-anion gap, hyperchloremic picture often develops (because some of the bicarbonate-bound ketoacids are excreted in the urine) on the road to normalization.
- **Ethylene glycol and methanol**: Toxic metabolites of these compounds produce increased anion-gap metabolic acidosis. In the suspected alcoholic patient who presents with significant metabolic acidosis, a quick method of distinguishing the presence of ethylene glycol or methanol from AKA is the osmolal gap. If this exceeds 25 mOsm/Kg, it is 88% specific for the presence of ethylene glycol or methanol.
- **Isopropyl alcohol**: A significant portion of isopropyl alcohol is metabolized to acetone. This is a ketone but not a ketoacid, causing ketosis and ketonuria but not acidosis.

12. How is coagulation affected in a chronic alcoholic?

Bone marrow depression from ethanol, folate deficiency, and hypersplenism secondary to portal hypertension all cause thrombocytopenia. Platelet counts less than 30,000/mL, resulting from alcohol usage alone, are unlikely. Qualitative platelet defects also occur.

Hepatocyte loss from chronic alcohol abuse depletes all coagulation factors except VIII, particularly II, VII, IX, and X. Alcoholics often have inadequate vitamin K, a requisite cofactor for the production of factors II, VII, IX, and X because of hepatobiliary dysfunction and poor diet. When faced with gastrointestinal hemorrhage in a chronic alcoholic, an intravenous vitamin K supplementation trial is warranted. The far more likely culprit is hepatocellular destruction, however, for which vitamin K would not be helpful. Vitamin K does not begin to restore factor levels for 2 to 6 hours, so for emergent scenarios, fresh frozen plasma provides immediate factor supplementation.

13. How should the combative alcoholic patient be managed?

When the patient or staff is in jeopardy, the first step is physical restraint of the patient. A sufficient number of competent personnel and restraint devices are necessary. Closed head injury, hypoxia, or a full bladder may be the source of distress and should be excluded, managed, or relieved.

For chemical sedation, haloperidol (5–10 mg intravenous push) has rapid onset of sedation (5 minutes), but repeat doses may be required. This agent is not detrimental to airway patency, ventilation, or hemodynamics. There is a 5% to 10% incidence of extrapyramidal reactions, usually within 12 to 24 hours. Droperidol (2.5–5 mg intramuscularly) is another effective butyrophenone, but it received a black-box warning in 2004 due to reports of QT prolongation and torsades de pointes. In any case, haloperidol and droperidol have been shown to be relatively comparable in efficacy and side effects.

14. When is an intoxicated patient safe to discharge from the ED?

From a management perspective, there are two fundamental concerns:

- Acute intoxication obfuscates the verification of certain diagnoses and the exclusion of others.
- A physician who discharges an acutely intoxicated (i.e., incompetent) patient may be held accountable for the actions of that patient subsequent and proximate to discharge from the ED.

The degree of clinical intoxication at a specific serum alcohol level is variable in accordance with the patient's chronicity and severity of drinking. A veteran drinker with a level in excess of 500 mg/dL can look less drunk than a teenager at 100 mg/dL. Documentation of the discharge examination includes mental status, gait, and the fact that the patient is **clinically sober**. Particular attention should be paid to potential abdominal or closed head trauma. The patient should be discharged to an appropriate environment. Serum or breath alcohol determinations sometimes can be helpful at the outset of care but unneeded at discharge.

KEY POINTS: ALCOHOL-RELATED DISORDERS

1. Airway protection: Clinical judgment is the key factor in determining whether an acutely intoxicated patient requires intubation for airway protection.
2. Phenytoin should only be given to patients with clear indication of an epileptogenic focus. Otherwise, its use for prevention of alcohol withdrawal seizures is strictly proscribed.
3. Discharge documentation: The critical determination in the discharge of the patient is the progression to and documentation of clinically sober.

15. Must thiamine be administered before glucose in the alcoholic patient?

Wernicke-Korsakoff syndrome develops over hours to days. The precipitous initiation of Wernicke-Korsakoff syndrome by dextrose has not been substantiated. The consequences of neuroglycopenia begin within 30 minutes and are easily prevented. In alcoholic patients with known or suspected hypoglycemia, promptly administer glucose and deliver thiamine empirically as soon afterward as possible. Because magnesium is a cofactor of thiamine and because alcoholics are frequently hypomagnesemic, 2 g of IV magnesium should be administered when there is suspicion of Wernicke-Korsakoff syndrome.

16. Is it dangerous to administer thiamine intravenously?

Orally administered thiamine is often absorbed poorly in the alcoholic patient. The intramuscular route is painful and can result in hematomas or abscesses, particularly in patients with impaired coagulation status. The experience with intravenous thiamine is extensive. Thiamine may be given as part of fluid hydration with multivitamin preparations or by bolus infusion.

17. Is there a cure for a hangover?

Probably not, at least not one with solid scientific credentials. There is no shortage of remedies, however, from the well-worn "hair of the dog that bit you" (i.e., start drinking again) to a more recently acclaimed concoction of vitamin B_6, nonsteroidal anti-inflammatories, and hydration. The only sure-fire measure is the avoidance of drinking in the first place.

18. Are there specific criteria for the diagnosis of Wernicke-Korsakoff syndrome?

Criteria require two of the following four signs to be present:

a. Dietary deficiencies
b. Oculomotor abnormalities
c. Cerebellar dysfunction
d. Either an altered mental state or mild memory impairment.

19. Why is it important to understand the metabolism of methanol?

The metabolites of methanol are the toxins and depend on alcohol dehydrogenase (ADH) for their conversion from the non-toxic parent methanol. Ethanol and fomepizole both saturate ADH and greatly slow the metabolism of methanol to the toxic metabolite. Folate is a cofactor in the breakdown of formic acid, and in monkeys (and other primates) folate supplementation

maximizes its metabolism and decreases injury. Knowledge of the metabolism directs the treatment.

$$\text{Methanol} \xrightarrow{\text{ADH}} \underset{\text{(toxic)}}{\text{Formaldehyde}} \xrightarrow{\text{Folate}} \underset{\text{(toxic)}}{\text{Formic Acid}} \rightarrow \underset{\text{(nontoxic)}}{CO_2 + H_2O}$$

20. List the signs and symptoms of methanol poisoning.

Gastrointestinal toxicity
- Nausea and vomiting
- Abdominal pain

Central nervous system toxicity
- Headache
- Decreased level of consciousness
- Confusion

Ocular toxicity
- Retinal edema
- Hyperemia of the disc
- Decreased visual acuity

Other toxicity
- Metabolic acidosis

KEY POINTS: METHANOL

1. Symptoms and acidosis are delayed.
2. Osmolal gap is often absent.
3. Persistent acidosis correlates with a poor prognosis.
4. Fomepizole, ethanol, and dialysis can all be used to treat the poisoned patient.

21. Why is it important to understand the metabolism of ethylene glycol?

As with methanol, ethanol and fomepizole saturate ADH, inhibiting conversion of ethylene glycol into its harmful metabolites. Pyridoxine (vitamin B_6) and thiamine are cofactors in the final steps to form nonharmful end products and should be given to ensure maximal metabolism. Oxalate crystals may not appear until late in the course of the poisoning (Fig. 69-1).

22. Why are the symptoms of ethylene glycol and methanol overdose often delayed?

It may take 6 to 12 hours for sufficient quantities of the toxic metabolites to accumulate and cause symptoms. The delay in symptoms is even greater with concurrent ethanol intoxication because the ethanol slows down the rate of methanol and ethylene glycol metabolism and delays the appearance of the toxic metabolites.

KEY POINTS: ETHYLENE GLYCOL

1. Symptoms and acidosis are often delayed.
2. Urinary oxalate crystals, fluorescence, early osmolal gap, and metabolic acidosis all suggest ethylene glycol poisoning.
3. Fomepizole, ethanol, and dialysis can all be used to treat the poisoned patient.

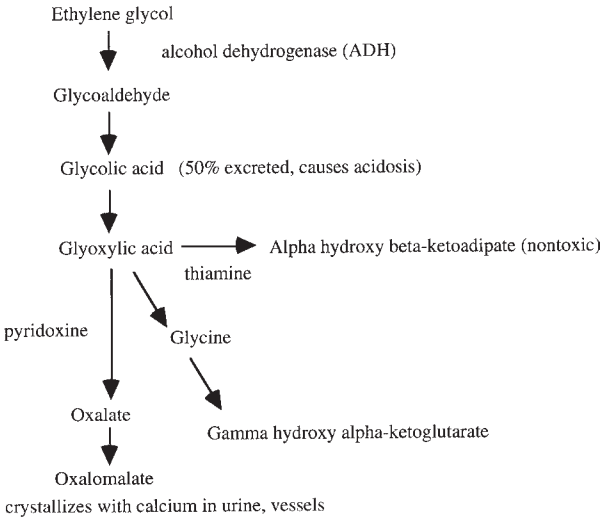

Figure 69-1. Metabolism of methanol and ethylene glycol.

23. How are methanol and ethylene glycol poisonings similar?

Methanol and ethylene glycol are metabolized initially by ADH. Methanol is metabolized further to formic acid, and ethylene glycol is metabolized to glycolic acid, glyoxylic acid, oxalate, and several nontoxic metabolites. Because of these end products, both poisons result in metabolic acidosis with an anion gap. Because of their low molecular weight, both increase the osmolar gap.

24. What is an anion gap?

A normal anion gap is the difference between measured and unmeasured anions (e.g., various proteins, organic acids, phosphates) and measured and unmeasured cations (e.g., potassium, calcium, and magnesium). The anion gap can be calculated from the formula:

$$\text{Anion gap} = (Na^+) - (HCO_3^- + Cl^-)$$

25. What causes an increased anion gap?

When metabolic acidosis results from an ingestion of nonvolatile acids, there are increased hydrogen ions with positive charges. Because there is an equal increase in unmeasured negatively charged anions but no increase in chloride, the difference between the measured cations and measured anions is increased, causing an increased anion gap. The normal anion gap is about 6 to 10 mEq/L. The causes of increased anion gap can be remembered by the mnemonic **A MUD PILES.**

A = **A**lcohol, **M** = **M**ethanol, **U** = **U**remia, **D** = **D**iabetic ketoacidosis, **P** = **P**araldehyde, **I** = **I**ron, **I**soniazid (INH), **L** = **L**actate, **E** = **E**thylene glycol, **S** = **S**alicylate

26. What is an osmolal gap?

Small atoms and molecules in solution are osmotically active, and this activity can be measured by a depression in the freezing point or an elevation in the boiling point of the solution. If there is an increase in low-molecular-weight molecules, such as acetone, methanol, ethanol, mannitol, isopropyl alcohol, or ethylene glycol, the osmolality is greater than what is calculated from the usual serum molecules. The difference between the actual measured osmolality and the calculated osmolality is the osmolal gap, and a gap greater than about 10 mOsm is considered abnormal.

27. How is an osmolal gap calculated?

One formula is $2 \times Na^+$ (mEq/L) + glucose (mg/dL)/18 + blood urea nitrogen (BUN) (mg/dL)/2.8 + ethanol (mg/dL)/4.3. The inclusion of the ethanol level excludes patients who have an elevated osmolal gap from ethanol ingestion alone. Using International System (SI) units, the calculated osmolality = $2 \times$ Na (mEq/L) + glucose (mmol/L) + BUN (mmol/L) + ethanol (mmol/L). The calculated osmolality is 285 $\pm$ 5 mOsm/L. A toxic ethylene glycol level of 25 mg/dL can be predicted to increase the osmolal gap 5 mOsm/L. Because of the small effects on the osmolality and the imprecision of the measurement, this test is not precise enough to be definitive so a normal osmolal gap does not exclude toxic levels of methanol or ethylene glycol. The laboratory must use the method of freezing-point depression so that volatile alcohols contributing to an osmolal gap are not boiled away during a boiling point elevation procedure.

28. What comes first, the anion gap or the osmolal gap?

With initial absorption, the small parent molecules cause an early osmolal gap, but with metabolism, acidic metabolites are formed causing a late metabolic anion-gap acidosis.

29. How toxic are methanol and ethylene glycol?

Death is reported after 15 to 30 mL (1–2 tablespoons) of methanol. Others have survived larger ingestions, however. A minimal lethal dose for ethylene glycol is approximately 1–2 mL/kg.

30. What is the toxicity of ethylene glycol?

Initially, there is central nervous system intoxication and gastrointestinal irritation, followed by metabolic acidosis. Renal failure occurs frequently and typically is delayed in presentation. Cranial nerve deficits are a rare complication.

31. Why is ethylene glycol so dangerous to animals?

Ethylene glycol is a frequent cause of death in animals who ingest antifreeze (especially dogs, who drink almost anything). The taste is sweet and a small volume is deadly. The cause of death for these animals may not be apparent because toxicity is delayed, and death occurs long after the animal has left the scene.

32. Why does antifreeze have such a bright color?

Antifreeze is a bright color that fluoresces with ultraviolet (UV) light so that leaks from auto radiators can be detected more easily. If the mouth and the urine are examined with a UV light, fluorescein can be detected in about 30% of patients after ingestion. A positive test should encourage immediate treatment, but a negative test misses two thirds of ingestions.

33. How should patients with methanol and ethylene glycol poisoning be treated?

Airway protection is paramount in patients with decreased level of consciousness or respiratory depression. Small volumes and rapid absorption limit the effectiveness of gastric lavage and charcoal. Acidosis (pH $<$7.2) should be treated aggressively with sodium bicarbonate. Ethanol and 4-methylpyrazole (4-MP) are antidotes that competitively block the conversion of methanol and ethylene glycol to their toxic metabolites, allowing for elimination of the unchanged poison without injury.

34. What are the indications for ethanol or 4-MP therapy?

They should be used if ethylene glycol or methanol levels exceed 20 mg/dL; if acidosis is present, regardless of drug level; and if there is a history of a toxic ingestion while awaiting confirmatory blood methanol or ethylene glycol levels.

35. How do you choose between fomepizole (4-MP) and ethanol?

Ethanol is difficult to give consistently; ethanol blood levels are required to adjust the dose and infusion can cause pain, resulting in the use of a central catheter. Ethanol may cause hypoglycemia and respiratory depression, especially in children. These patients usually require the close monitoring of an intensive care unit (ICU). 4-MP is replacing ethanol because it does not cause sedation, does not require blood testing, is easily given as a bolus, and does not require ICU management.

36. How do you use 4-MP?

The dose is 15 mg/kg every 12 hours and is increased to every 4 hours during dialysis. Typical treatment is for 48 hours.

37. What are the indications for hemodialysis?

Dialysis used to be the primary treatment for these poisons and should be done in patients with blood levels greater than 50 mg/dL, when the metabolic acidosis is not correctable, with pending renal failure, or with visual symptoms in a methanol overdose. Many clinicians recommend dialysis when blood levels exceed 25 mg/dL, if dialysis is readily available. Hemodialysis can be avoided with prolonged fomepizole treatment.

38. What if dialysis is unavailable?

Patients with ethylene glycol poisoning can be treated successfully with 4-MP alone without dialysis if there is no acidosis or renal failure. Because the half-life of ethylene glycol is prolonged to 17 hours, the treatment may be extended but avoids invasive treatment of dialysis. In methanol poisoning, 4-MP slows the metabolism and increases the half-life of methanol to 30-52 hours. The use of 4-MP alone would not suffice for these patients.

39. How is isopropyl alcohol poisoning different from methanol and ethylene glycol poisoning?

Isopropyl or rubbing alcohol is metabolized in the liver to acetone, which results in measurable ketonemia in the serum. Acetone is excreted by the kidney, resulting in ketonuria, and is exhaled through the lungs, giving patients an acetone aroma on their breath. Because these metabolites are not acidic, isopropyl alcohol poisoning does not result in metabolic acidosis and is far less toxic than either methanol or ethylene glycol.

40. What are the symptoms of isopropyl alcohol ingestion?

Isopropyl alcohol has a three-carbon chain rather than the two-carbon chain of ethanol. Because of this, it crosses the blood-brain barrier faster and is about twice as intoxicating as ethanol. Because it is commonly found in concentrated solutions and is more potent, the central nervous system depression can occur rapidly and can continue from residual poison in the stomach. Isopropyl alcohol is much more irritating than ethanol to the gastric mucosa and often causes abdominal pain, vomiting, and hematemesis.

41. Why is isopropanol so frequently abused?

Isopropanol is easy and legal to obtain; rubbing alcohol is 70% isopropanol. Unlike consumable beer, wine, and liquor, it is not taxed and is very inexpensive.

42. What treatment is advisable for isopropyl alcohol poisoning?

Patients need observation to watch for respiratory depression similar to patients intoxicated with ethanol. An isopropyl alcohol level is roughly equivalent to an ethanol level twice as high. An isopropyl level usually does not add greatly to clinical observation. In the rare instance of coma or hypertension corresponding to isopropyl levels greater than 500 mg/dL, intubation and ventilation may be necessary, and hemodialysis can greatly enhance removal of isopropyl alcohol from the body. An antidote is not available for isopropyl alcohol (nor is one needed).

KEY POINTS: ISOPROPANOL

1. Symptoms and toxicity are completely different from methanol and ethylene glycol.
2. Ketosis occurs, but acidosis does not.
3. Supportive treatment is adequate in almost all cases.

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ANTIPYRETIC POISONING

James C. Mitchiner, MD, MPH

CHAPTER 70

SALICYLATE POISONING

1. What are the causes of salicylate overdose?

A salicylate overdose may be intentional or accidental. Parental administration of adult doses of aspirin to a child may cause toxicity. Bismuth subsalicylate (Pepto-Bismol), which contains 130 mg/tablespoon of salicylate, is occasionally the culprit. In adults, concurrent ingestion of aspirin and aspirin-containing prescription or non-prescription medications may lead to unintentional overdose with possible formation of gastric concretions. Liquid methyl salicylate (oil of wintergreen) is especially toxic because of its high salicylate content (1 teaspoon = 7 g of salicylate) and rapid absorption. Dermal application of salicylic acid ointment is a rare cause of acute salicylism. The minimal acute toxic ingestion is 150 mg/kg.

2. What are the characteristics of a patient who presents with an acute salicylate overdose?

Patients may present with nausea, vomiting, tinnitus, vertigo, fever, diaphoresis, and confusion. Hyperventilation may be ascribed mistakenly to anxiety. Patients also may present with headache or chronic pain, which prompted the excess ingestion of salicylate.

3. List some common signs of salicylate intoxication.

See Table 70-1.

4. Describe the acid-base disturbances associated with salicylate toxicity.

Acute **respiratory alkalosis**, without hypoxia, is due to salicylate stimulation of the respiratory center. If the patient is hypoxic, salicylate-induced noncardiogenic pulmonary edema should be considered. Within 12 to 24 hours after ingestion, the acid-base status in an untreated patient shifts toward an anion gap **metabolic acidosis.** A mixed respiratory alkalosis and metabolic acidosis typically is seen in adults. In patients presenting with **respiratory acidosis**, concomitant ingestion of a central nervous system depressant should be suspected. **Metabolic acidosis** is the predominant acid-base disturbance in children, patients who take massive amounts of salicylates, hemodynamically unstable patients, and patients of all ages who have chronic salicylate toxicity.

5. What are some of the other metabolic disturbances seen in acute salicylate poisoning?

The patient may be dehydrated secondary to vomiting, the diuretic effects of increased renal sodium excretion, or diaphoresis in response to the hyperpyrexic state. Insensible losses are increased in patients with hyperventilation. Hypokalemia is due to renal potassium excretion and respiratory and metabolic alkalemia (secondary to bicarbonate therapy).

6. I thought aspirin was an antipyretic. How does it cause a fever?

At a cellular level, salicylate poisoning leads to the uncoupling of oxidative phosphorylation. When this occurs, the energy obtained from oxygen reduction and reduced nicotinamide adenine dinucleotide oxidation that is normally captured in the form of adenosine triphosphate (ATP) instead is released as heat.

TABLE 70-1. COMMON SIGNS OF ACUTE SALICYLATE TOXICITY

General	Hyperthermia, dehydration
Respiratory	Hyperventilation (may be mistaken for anxiety), noncardiogenic pulmonary edema
Central nervous system	Confusion, delirium, seizures, coma
Gastrointestinal	Nausea, vomiting, gastrointestinal hemorrhage
Dermatologic	Eyelid petechiae
Laboratory	Acid-base disturbances, azotemia, hyperkalemia or hypokalemia, hypoglycemia (children), elevated CK levels (rhabdomyolysis), coagulopathy

CK, creatine kinase.

7. Name some of the hematologic abnormalities.

These are rare in an acute overdose. Features include decreased production of prothrombin (factor II) and factor VII, an increase in capillary endothelial fragility, and a decrease in the quantity and function of platelets (i.e., decreased adhesiveness). Significant hemorrhage is unusual.

8. How is the severity of salicylate overdose assessed?

Salicylate levels should be obtained at the time of initial ED evaluation and repeated several hours apart, *while the patient is still under observation in the ED*, so that the severity of poisoning can be trended. The aspirin (**Done) nomogram** is of historic interest only and is no longer recommended.

9. Which laboratory tests are indicated?

Serial serum salicylate levels (initial and 6 hours postingestion) should be obtained, along with a complete blood cell count, serum electrolytes, blood urea nitrogen (BUN), creatinine, glucose and a urinalysis. Prothrombin time (PT), international normalized ratio (INR), and arterial blood gases should be considered. If the patient presents less than 6 hours after an acute ingestion, a salicylate level should be repeated at 6 hours. A quantitative acetaminophen level is also recommended because many patients confuse these two drugs or mix both kinds in the same bottle.

10. What is the initial ED treatment for an acute salicylate overdose?

If poisoning is through dermal contact, the skin should be washed copiously with tap water. For acute ingestions, intravenous normal saline should be given initially, with conversion to alkaline diuresis if the patient is toxic. A slurry of activated charcoal mixed with cathartic (sorbitol or magnesium sulfate) should be given orally or by gastric lavage tube at a dose of 1 g of charcoal per kg. Lavage may be useful even if the patient presents several hours after ingestion because large amounts of aspirin may form gastric concretions with ongoing absorption.

11. What else needs to be done in the ED?

After the patient has responded with diuresis, potassium losses should be replaced with potassium chloride at a dose of 20 to 40 mEq/L. Patients with hyperthermia should be cooled with a cooling blanket. Hypoglycemia should be treated with intravenous D50. Patients with aspirin-induced noncardiogenic pulmonary edema should be treated with oxygen, noninvasive

ventilation (continuous positive airway pressure [CPAP] or bi-level positive airway pressure [BiPAP]), or intubation and positive end-expiratory pressure (PEEP). **If possible, sedation should be avoided because of the risk of respiratory depression leading to respiratory acidosis and exacerbation of central nervous system toxicity.**

12. Is there a role for repetitive dosing of activated charcoal?

Because of aspirin release from the aspirin-charcoal complex in the gastrointestinal tract and subsequent reabsorption, salicylate levels may not decline significantly after a single dose of activated charcoal. Repeated doses of charcoal (25 g every 3 hours, without cathartic) may be indicated to enhance elimination.

13. What is the rationale for alkaline diuresis?

Because aspirin is an organic acid, administration of bicarbonate intravenously raises the pH of the blood and *traps* salicylate ion, limiting the amount of salicylate that crosses the blood-brain barrier. Similarly, an alkalotic urine retains salicylate ion, preventing its reabsorption by the renal tubules. Isotonic alkaline diuresis is achieved by adding 3 ampules of $NaHCO_3$ to 1 liter of D5W, with infusion at a rate of 2 to 3 mL/kg per hour. The patient should be monitored for the development of pulmonary edema.

14. Explain the paradox of a decreasing serum salicylate concentration and increasing clinical toxicity.

The serum salicylate level by itself does not reflect tissue distribution of the drug. If the patient's blood is acidemic, salicylate acid remains un-ionized and more penetrates the blood-brain barrier, resulting in central nervous system toxicity. **Salicylate levels should be interpreted in light of the patient's clinical condition and a concurrent blood pH;** an acidotic pH is associated with toxicity regardless of the salicylate level.

15. What are the indications for hemodialysis?

Standard indications include persistent, refractory metabolic acidosis (arterial pH <7.10), renal failure with oliguria, cardiopulmonary dysfunction (e.g., pulmonary edema, dysrhythmias, cardiac arrest), central nervous system deterioration (e.g., seizures, coma, cerebral edema), and an acute salicylate level greater than 130 mg/dL at 6 hours post-ingestion. Because ingestion of more than 300 mg/kg predicts severe toxicity, a nephrologist should be contacted early in anticipation of the possible need for dialysis.

16. What are the most common findings in chronic salicylate poisoning?

In contrast to acute salicylate poisoning, chronic salicylism is usually accidental. The principal diagnostic feature is a change in mental status manifested by weakness, tinnitus, lethargy, confusion, drowsiness, slurred speech, hallucinations, agitation, or seizures. Because these signs are common to many other disorders, the diagnosis frequently is missed, resulting in a mortality rate of 25%. Most patients are tachypneic, which is a compensatory response to an anion gap metabolic acidosis. The serum salicylate level may be normal or minimally elevated.

ACETAMINOPHEN POISONING

17. Is there anything new in acetaminophen toxicology?

Yes. There's much more to worry about now that we have extended release preparations and reports of hepatotoxicity due to unintentional supratherapeutic ingestions.

18. What are the characteristics of acetaminophen overdose?

Acetaminophen is the drug most commonly involved in acute analgesic ingestions, either as a single agent or in combination with various cough, cold, or pain remedies. Early diagnosis of acute (phase I) acetaminophen toxicity is important because early symptoms may be subtle or absent; the onset of hepatotoxicity, the major manifestation, is delayed by several days after

ingestion. Failure to recognize and treat toxicity within 16 hours of ingestion results in significant morbidity and mortality. **The main issue in treatment is the prevention of hepatotoxicity.**

19. Outline the four phases of acetaminophen overdose.
See Table 70-2.

20. What are the initial central nervous system manifestations of acetaminophen poisoning?
Gotcha! In the early stages, there are none, and abnormalities in mental status or level of consciousness should be attributed to other drugs (e.g., salicylates, opiates, sedatives) or to other disease states. Hepatic encephalopathy can occur in phase III.

21. Describe the pathophysiology of acetaminophen toxicity.
Acetaminophen is metabolized primarily by the liver. About 90% of it is conjugated with glucuronic or sulfuric acid to form nontoxic compounds that are excreted in the urine. About 2% of the drug is excreted unchanged in the urine. The remainder is metabolized by the cytochrome P-450 mixed-function oxidase system. This involves formation of a toxic intermediary compound, which is conjugated rapidly with hepatic glutathione. The resulting conjugate is metabolized further, and its byproducts are excreted in the urine. Because the liver normally has a fixed amount of glutathione, this compound is depleted rapidly in an acute overdose. The toxic intermediary then accumulates, unmetabolized, and binds to the sulfhydryl groups of hepatic enzymes. The result is irreversible centrilobular hepatic necrosis.

22. How is hepatotoxicity predicted?
An acute ingestion of 7.5 gm or more in an adult or 140 mg/kg in a child is generally predictive of hepatotoxicity. The most accurate predictor of hepatotoxicity is the serum acetaminophen level obtained between 4 and 24 hours after **acute** ingestion. The **Rumack-Matthew nomogram**, which plots serum concentration against hours postingestion, is the standard reference for predicting hepatotoxicity in an acute overdose. Certain drugs, such as cimetidine, compete with acetaminophen for metabolism by the P-450 pathway and theoretically offer some protection from hepatotoxicity. Other drugs, such as phenytoin and phenobarbital, may induce P-450 enzymes and facilitate acetaminophen metabolism to the toxic intermediary, thereby increasing the risk of toxicity.

TABLE 70–2. PHASES OF ACETAMINOPHEN TOXICITY

Phase	Onset	Clinical Characteristics	Laboratory Findings
I	<24 hours	Anorexia, nausea, vomiting, diaphoresis (patient may be asymptomatic)	Toxic acetaminophen level
II	24–72 hours	Right upper quadrant abdominal pain	Mild elevation in LFTs
III	3–5 days	Vomiting, jaundice, encephalopathy, oliguria	Marked elevation in LFTs, coagulopathy, azotemia, hypoglycemia, hypophosphatemia
IV	about 1 week	Gradual resolution of toxicity	Improvement in laboratory values

LFTs, liver function tests.

23. Are serial serum acetaminophen levels helpful?

If an accurate estimate of the time of ingestion cannot be obtained, the nomogram cannot be used and serial levels should be obtained. Patients with rising levels are at risk for hepatotoxicity and should be treated with *N*-acetylcysteine (NAC).

24. Why is hepatotoxicity in children rare?

No one knows for sure. Toxicity in children is rare, even when toxic levels of acetaminophen are found. One theory holds that acetaminophen metabolism in children shows a preference for alternative pathways other than the P-450 system. The conversion from juvenile to adult metabolism is believed to occur between 6 and 9 years of age.

25. Which laboratory tests are helpful?

If a serum acetaminophen level is in the toxic range on the nomogram, additional blood should be obtained for a complete blood cell count, electrolytes, BUN, glucose, PT, INR, and liver function tests. A **limited** toxicology screen also should be ordered, with attention to treatable concomitant ingestions, such as salicylates, opiates, barbiturates, ethanol, and cyclic antidepressants.

26. Outline the general treatment of acetaminophen poisoning.

Activated charcoal (1 g/kg) mixed with cathartic (e.g., sorbitol or magnesium sulfate) should be administered orally or by gastric lavage tube, if there is an indication for gastric lavage (see Chapter 68). The specific antidote is NAC. This agent is a glutathione substitute with a high therapeutic-to-toxic safety ratio. It should be given orally or intravenously as soon as possible after an acute overdose with a toxic plasma acetaminophen level, as documented by the nomogram. NAC should also be given intravenously to patients with hepatic failure where acetaminophen poisoning is suspected, even if the acetaminophen level is in the normal range.

KEY POINTS: ED APPROACH TO ANALGESIC TOXICITY

1. Serial salicylate levels should be used to exclude toxicity prior to admitting the suicidal patient to the psychiatric floor.
2. Salicylate levels must be interpreted in light of the patient's clinical condition, the formulation of the drug (pills, capsules or liquids), and a concurrent blood pH.
3. The primary goal in the treatment of acetaminophen toxicity is the prevention of hepatotoxicity.
4. The antidote for acetaminophen overdose is *N*-acetylcysteine. It is most effective when administered within 10 hours, regardless of whether it is given orally or intravenously.

27. How is NAC administered?

Oral NAC (Mucomyst™) is given by mouth or nasogastric tube after diluting it 1:5 with water or juice. This dilution produces a 20% solution, which is given as a loading dose of 140 mg/kg, followed by a maintenance dose of 70 mg/kg every 4 hours for 17 additional doses. If the patient vomits a dose within 1 hour, the dose should be repeated. Antiemetics should be given if vomiting is persistent. The intravenous NAC formulation (Acetadote™) is indicated for patients in whom oral NAC is contraindicated (e.g., persistent vomiting, encephalopathy, gastrointestinal bleeding, bowel obstruction, concomitant ingestion with anticholinergic drug). The dose is 150 mg/kg in D_5W over 15 minutes, followed by 50 mg/kg over 4 hours, then 100 mg/kg over 16 hours.

28. Which route is better for administering NAC, oral or intravenous?

It depends. Compared to the oral route, intravenous NAC is easier to administer, takes less time (20.25 hours vs. 72 hours for the oral route), and can be given to patients who are vomiting. Disadvantages include a higher rate of adverse reactions (up to 17%) and greater expense. There is no evidence to date that one route is preferable to the other in terms of reducing the risk of hepatotoxicity.

29. Is there a critical window in time to administer NAC?

Yes. Whenever possible, NAC should be given within 10 hours of acute acetaminophen overdose. NAC still may be of benefit if given more than 10 hours after acute ingestion, particularly in patients who have taken extended-release formulations or staggered overdoses, and in patients with persistently toxic acetaminophen levels or elevated liver enzymes. The intravenous route is recommended in these cases.

30. Should I be concerned about potential adverse reactions to intravenous NAC?

Yes. The incidence of such reactions is 5% to 17%, and they tend to occur during infusion of the loading dose. Typical symptoms not requiring therapy include nausea, vomiting, and flushing; mild urticaria can be treated with diphenhydramine. Interruption of NAC therapy is not necessary, but the initial infusion rate should be slowed. Serious reactions, such as bronchospasm, angioedema, and hypotension, require aggressive therapy with antihistamines, steroids, and epinephrine and discontinuation of the NAC.

31. What is the acetaminophen-alcohol syndrome?

Acute alcohol ingestion is said to be protective because alcohol competes with acetaminophen as a substrate for cytochrome P-450. In contrast, chronic alcohol abuse affects acetaminophen detoxification in two ways:

a. It lowers hepatic glutathione stores, resulting in a reduced capacity to detoxify the toxic intermediate compound.
b. It induces the cytochrome P-450 system, increasing the proportion of ingested acetaminophen that is converted to the toxic intermediate.

Diagnostic findings include a history of acetaminophen ingestion and elevated aspartate transaminase levels (usually $>$800 IU/L) in patients with known or occult alcohol abuse who regularly take acetaminophen. The diagnosis initially is missed in one third of cases, and the mortality rate is greater than 30%. Treatment is generally supportive, although NAC has been tried, and liver transplantation is an option.

32. What is the treatment for chronic acetaminophen toxicity?

In chronic acetaminophen poisoning, the nomogram is not helpful in predicting toxicity. Repetitive ingestion is thought to be of serious concern only in alcoholics, patients on anticonvulsants, children with febrile illnesses, individuals taking large doses (e.g., $>$10 g/day), and patients with symptoms of toxicity. NAC is recommended only for patients with detectable acetaminophen levels and evidence of liver injury.

IBUPROFEN POISONING

33. What are the characteristics of ibuprofen overdose?

Ibuprofen is readily available as an over-the-counter medication used in the treatment of mild-to-moderate pain and fever. Rapid absorption leads to peak drug levels within 2 hours. Symptoms usually are seen within 4 hours of ingestion and are more likely to be serious in children. Toxicity is limited in patients who ingest less than 100 mg/kg, whereas patients, primarily children, who ingest more than 400 mg/kg may be at risk for more severe symptoms.

34. List the primary symptoms of ibuprofen toxicity.

- **Gastrointestinal toxicity** is manifested by nausea, vomiting, abdominal pain, and hematemesis.
- **Nephrotoxicity** results in acute renal failure.
- **Central nervous system toxicity** (seen mostly in children) includes somnolence, apnea, seizures, and coma.
- **Severe metabolic acidosis** and **thrombocytopenia** have also been described.

35. Should a serum ibuprofen level be obtained?

No, because the serum ibuprofen level does not correlate with clinical symptoms, there is no role for this test in medical decision-making.

36. Describe the treatment for ibuprofen toxicity.

Treatment is directed at alleviating symptoms and providing supportive care (see Chapter 68). If hematemesis is present or there is blood in the stool, a nasogastric tube should be placed; if blood is present, the stomach should be irrigated with saline. A limited toxicology screen to search for other readily treatable toxins (i.e., salicylates, acetaminophen, opiates, barbiturates, cyclic antidepressants, and ethanol) is recommended. Seizures should be treated with intravenous diazepam. Renal and hepatic function tests should be ordered. Children with ingestions of greater than 400 mg/kg should be observed in the hospital. Forced diuresis, alkalinization, and hemodialysis are not indicated.

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CHAPTER 71

BITES AND STINGS

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ARACHNIDA (CHIGGERS. SCABIES, SCORPIONS, AND SPIDERS)

1. What is a tarantula?

It is a large spider of the family Theraphosidae. The largest is the South American *Grammostola mollicoma*, with a leg span of up to 27 cm and a body length of up to 10 cm! Not much is known about tarantula venom, although it seems to contain a mixture of hyaluronidase, nucleotides, and polyamines (which act as neurotransmitters to paralyze the prey). On the whole, these bites tend to be of low toxicity in humans with a mild, briefly active venom causing pain, numbness, and lymphangitis. The bites usually do not cause necrosis or serious sequelae. The spider has little urticating hairs that are barbed and can cause skin and mucous membrane irritation with edema and pruritus that can last for weeks. Eye exposure can cause a severe keratoconjunctivitis and ophthalmia nodosa.

2. What spider bites are likely to be an issue?

Although all spiders possess venom, there are two spiders of particular clinical importance in the United States: *Latrodectus* (black widow) and *Loxosceles* (brown recluse or fiddle back). In 2007, the American Association of Poison Control Centers (AAPCC) reported 2,514 bites from *Latrodectus* and 1,783 from *Loxosceles.* There were no deaths and only 16 major reactions attributed to Latrodectus bites (0.64%). Similarly, no deaths and 15 major reactions (0.84%) were attributed to *Loxosceles.* The envenomation syndromes (and treatment) of these two spiders are quite distinct (Table 71-1).

3. What is *Mustov's* disease?

It is a play on words. Although there were 13,479 bites attributed to spiders reported to the AAPCC in 2007, this number is likely an inaccurate estimate of the true incidence because:

- These are only the cases that were reported to poison control centers.
- The effect of Mustov's disease (as in, "Doc, I woke up with this. I ***must've*** been bitten by a spider in my sleep."). It seems that a number of nonbite skin lesions (especially community acquired methicillin-resistant *Staphylococcus aureus* abscesses) are unfairly blamed on spiders. Mustov's disease is not specific to spider bites.

4. A 5-year-old boy presents with genital itching that started several hours after sitting on the lawn watching a fireworks display. His examination reveals intensely pruritic, erythematous papules around his groin. What is this? How can it be treated? (Clue: he had been wearing shorts.)

Chiggers. They are tiny mite larvae that cause intense pruritus. The diagnosis is based on identifying the characteristic skin lesions in a person with an outdoor exposure. Itching begins within a few hours of exposure, and the papules can enlarge to form nodules in 1 to 2 days. There may be fever and erythema multiforme. Treatment is with antihistamines and steroids (topical or oral) for the symptoms, and lindane, permethrin, or crotamiton for definitive therapy.

TABLE 71-1. COMPARISON OF BLACK WIDOW AND BROWN RECLUSE SPIDERS

Latrodectus: Black Widow	Loxosceles: Brown Recluse
Markings	**Markings**
Red hourglass shape on the ventral abdomen (♀)	Dark, violin-shaped spot anterodorsally
Presentation	**Presentation**
Pain at the bite within 1 hour Target-shaped erythema, swelling, diaphoresis ■ Diffuse large muscle cramping, including the back, chest, and abdomen (which may mimic peritonitis) ■ Latrodectisima: characteristic facial muscle spasm, lacrimation, photophobia, and periorbital edema ■ Headache ■ Light-headedness ■ Nausea and vomiting Severe envenomations may result in dysphagia, hypertension, respiratory failure, shock, and coma	Typically, an initially mild bite characterized by erythema Bite becomes necrotic over 2–4 days Systemic reaction may occur in 1–2 days: ■ Fever ■ Chills ■ Vomiting ■ Arthralgia ■ Myalgia ■ Hemolysis ■ Coagulapathy May result in renal failure and death
Treatment	**Treatment**
■ Wound care ■ Analgesics, benzodiazepines for spasm ■ Tetanus prophylaxis ■ Calcium gluconate (IV) is ineffective and is no longer recommended ■ Horse-serum IgG antivenom: administer a subcutaneous test dose, and then, if no severe reaction, 1 vial mixed in 50 mL saline over 30 min IV ■ New *Latrodectus* immune F(ab)2 antivenom being studied	■ Wound care ■ Analgesics ■ Tetanus prophylaxis ■ Surgical debridement and possible grafting for lesions greater than 2 cm ■ Transfusion or dialysis, as necessary ■ Hyperbaric oxygen therapy, corticosteroids, and dapsone have been advocated by some, but there is no clear evidence of efficacy in humans ■ Currently, there is no commercially available antivenom

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5. What are the distinguishing features of scabies?

The bites are typically in the web spaces between the fingers and toes (also the penis, face, and scalp in children). They create *burrows* of pruritic, white, threadlike patterns with small gray spots at the closed end, where the parasite rests. Treatment is with a thorough application of permethrin from the neck down and may require a second course of treatment in 2 weeks.

6. How dangerous are scorpion stings?

Scorpions are generally not aggressive. They do not hunt for prey, but rather hide and wait. Scorpions have paired venom glands located in the last of the five abdominal segments (the tail). They hold their prey with their pincers, arch their tail over their body, and sting (not bite) the victim. The principal toxins are polypeptides and low-molecular-weight proteins, histamine, and indole compounds (including serotonin). The venom causes an increase in the sodium permeability of presynaptic neurons, which leads to continuous depolarization. Most scorpions indigenous to the United States are of low toxicity except for the bark scorpion *(Centruroides)*. In North America, *Centruroides exilicauda* (found in Baja California) and *Centruroides sculpturatus* (in Sonora, Mexico, and in the Southwestern U.S.—Arizona, Utah, New Mexico, Nevada, and California) are capable of producing systemic toxicity. They can hitch a ride in unsuspecting travelers' luggage. In 2007, the AAPCC reported 16,937 patients with scorpion stings. There was moderate morbidity in 3.9% of patients, major morbidity in 0.18%, and no deaths.

7. What are the signs of scorpion envenomation?

The sting is acutely painful. Systemic manifestations are rare and mainly occur in small children and the elderly where a larger venom-to-body weight ratio exists. The principal signs of systemic toxicity are salivation, tachycardia, roving eye movements, involuntary muscle jerking, opisthotonos, and tongue fasciculations.

8. What is the treatment for a scorpion sting?

Supportive care with local wound care, analgesia, and benzodiazepines for the neuromuscular symptoms is the mainstay of treatment. There was a *Centruroides* scorpion antivenom available only in Arizona, but it is no longer produced. A new scorpion-specific fragment antigen binding (Fab) F(ab)2 antivenom (Anascorp™) is available in Mexico and is undergoing evaluation in the United States. In critically ill children with neurotoxic effects the antivenom has been shown to resolve the clinical syndrome within 4 hours, to reduce the need for concomitant sedation with benzodiazepines, and to reduce the levels of circulating unbound venom.

FORMICOIDEA (ANTS)

9. I have a patient who received multiple stings from fire ants. What do I do?

Don't panic. Treatment is the same as for a bee sting. Fire ants swarm during an attack, and each sting contributes to the total antigen load. The individual stings result in intensely pruritic papules that may evolve to sterile pustules within 24 hours. Local necrosis and scarring may occur.

HYMENOPTERA (BEES AND WASPS)

10. What types of reactions occur from *Hymenoptera* stings?

There are four types of reactions:

- The **toxic reaction** is a nonantigenic response to the venom characterized by local irritation at the sting site and, potentially, vomiting, diarrhea, light-headedness, and syncope. There may also be headache, fever, drowsiness, involuntary muscle spasms, edema without urticaria, and occasionally convulsions. Local toxic reactions are treated with supportive care, including cool packs and analgesics.

- **Anaphylactic reactions** are most commonly seen in Vespidae stings (i.e., wasps, hornets, yellow jackets). These reactions can range from mild to fatal and occur from 15 minutes to 6 hours after the sting. These reactions are treated like any other allergic reaction.
- **Delayed reactions** present as a serum sickness-like syndrome 10 to 14 days after the sting. The delayed reactions are treated with antihistamines and corticosteroids.
- **Unusual reactions** reported after *Hymenoptera* stings include encephalitis, neuritis, vasculitis, and nephritis.

11. How does a bee sting differ from a wasp sting?

Bees have barbed stingers that usually remain in the victim, pulling the venom sac off of the bee. Whereas the bee dies after a single sting, a wasp is capable of stinging multiple times. It is better to remove the stinger from a bee by scraping it out with a credit card rather than by pinching and plucking it with fingers or tweezers and risking the inadvertent injection of more venom. Removal of the stinger should be done as soon as possible because the venom sac continues to pulse venom after it has detached from the bee.

12. What about *killer bees*?

African honeybees *(Apris mellifera scutellata)* were introduced into Brazil in 1956 as a potential honey producer in the tropical environment. There is little difference between the Africanized bees and European bees in terms of appearance, the nature of their venom, and the amount of venom that they carry. The difference lies in their aggressive behavior. Victims typically receive multiple stings during a swarming attack and, therefore, a greater venom burden. For this reason, the Africanized honeybees have been called *killer bees*.

13. After a patient has survived an anaphylactic reaction to a bee sting, what should be done to prepare the patient in case he or she is stung again in the future?

- First, avoid bees and wasps.
- Second, carry medical identification describing the bee sting allergy, such as a MedicAlert bracelet.
- Third, carry and learn to use an epinephrine self-injector (the Ana-Kit or the EpiPen).

KEY POINTS: BITES AND STINGS RESPONSIBLE FOR THE GREATEST NUMBER OF DEATHS AND MODERATE-TO-SEVERE MORBIDITY IN THE UNITED STATES

1. Snakes (35%)
2. Insects (34.5%)
3. Spiders (22.6%)
4. Mammals (4.3%)
5. Aquatic animals (3.6%)

HELODERMA (LIZARDS)

14. Are there any venomous lizards in the world?

Yes, two species: the Mexican beaded lizard *(Heloderma horridum)* and the Gila monster *(Heloderma suspectum)*. Both animals live in the desert areas of the southwestern United States and in Mexico. The venom of these lizards is somewhat similar to Crotaline venom, although the clinical course is typically milder. The more serious problem with these reptiles

is their powerful jaws (and their tendency to hold onto their victims). They deliver their venom by chewing and dripping the venom into the lacerations created by their teeth. Their teeth also commonly break off in the wounds and become foreign bodies and a nidus for infection if not removed. The teeth, by the way, are difficult to visualize on radiographs. Envenomation is present in about 70% of the bites.

CULICIDAE (MOSQUITOES)

15. What is the major clinical significance of mosquito bites?

There are more than 3,000 species of mosquito, and they are found on every continent, except Antarctica. They are responsible for more bites than any other blood-sucking organism. They are attracted by carbon dioxide, lactic acid, body heat, and sweat. Children younger than age 1 year rarely show a skin reaction to the bite; however, by age 5 years almost all children react. Both immediate and delayed hypersensitivity reactions can occur. The major significance, however, is in the role of the mosquito as a disease vector: They can transmit more than a half dozen forms of encephalitis, malaria, yellow fever, dengue fever, filariasis, West Nile Virus, Ross River virus, Chikungunya fever, and Rift Valley fever. They transmit disease to over 700 million people annually with at least 2 million resultant deaths in Africa, South and Central America, Mexico, and Asia. (See Chapter 52 for more information about mosquito-borne diseases).

MAMMALS (BATS, DOGS, CATS, FOXES, HORSES, HUMANS, RACCOONS, SKUNKS, AND WOODCHUCKS)

16. How many dog and cat bites are there annually in the United States? What is the risk of infection? What is the mortality from these bites?

The majority of pet bites that require medical attention are from dogs. It is estimated that more than 4.7 million dog bites occur annually, causing up to 800,000 victims to seek medical attention. The annual incidence of cat bites is about 1 million. The risk of infection from a bite is determined by multiple factors, including the location of the bite (hands are worse), the type of wound (crush injury from dogs and punctures from cats are worse), the biting species, and host factors (immunocompromising comorbidities). Dog bites to the hand, for example, may have a risk of infection as high as 30%. Cat bites carry a 15% to 80% rate of infection (the broad spread is likely due to the determination of what constitutes the denominator). Dog attacks are fatal for about 10 to 20 people annually. The victims are often infants and children and usually die from exsanguinating neck injuries.

17. Should I give prophylactic antibiotics to the victim of a dog or cat bite?

This is controversial. Meticulous wound care is the most effective means of reducing infection potential. In a meta-analysis, Cummings showed a number needed to treat of 14 to prevent one wound infection after a dog bite. However, if one of the studies that Cummings included had shown an abnormally high infection rate, the conclusion would have been that low-risk wounds (immunocompetent patients with nonpuncture wounds that do not involve the hand or foot, which are treated within 12 hours and show no signs of infection) do not benefit from antibiotics. High-risk wounds, however, may do better with antibiotics in addition to meticulous wound care. When choosing antibiotic(s), consider the polymicrobial nature of these infections (*Staphylococcus, Streptococcus, Pasteurella multocida*, anaerobes, etc.) and the cost of the antibiotic.

18. What is *Capnocytophaga canimorsus*?

Capnocytophaga canimorsus (DF2) is a fastidious gram-negative rod that can cause sepsis after a dog bite. Eighty percent of the patients who become seriously ill from this infection are immunocompromised (i.e., splenectomy, hematologic malignancy, cirrhosis, HIV/AIDS, or long-term steroids). Fortunately, it is a rare infection because it carries a 25% to 36% mortality.

19. What is the origin of the phrase "the hair of the dog that bit you"?

It is a very, very old expression, probably arising from the Roman belief that *similia similibus curantur* (like cures like). To treat the victim of a dog bite, one would obtain samples of its hair and either burn the hair and apply it to the wound or use the hair in creating a poultice for the wound. It was thought that this would speed wound healing and recovery from rabies. By extension, another drink or two after a drinking binge would be the cure for a hangover.

20. What types of bites are at risk for the transmission of rabies?

Rabies is a disease caused by an RNA rhabdovirus transmitted by inoculation with infectious saliva. It is prevalent in parts of Latin America, Asia, Africa, South America, Europe, the Middle East, India, and Southeast Asia. Rabies-free areas include Hawaii, England, Australia, Japan, and parts of the Caribbean. The virus primarily affects the central nervous system and is almost always fatal. In the United States, animal bites from skunks, raccoons, bats, foxes, and woodchucks should be considered a risk. Exposures from livestock, rodents, and lagomorphs should be considered individually but rarely require postexposure prophylaxis because the host dies before the rabies virus can replicate sufficiently. Consult your state health department for local recommendations.

21. What does postexposure prophylaxis for rabies consist of?

Postexposure prophylaxis means trying to prevent the disease before it manifests after a high-risk exposure. It begins with a thorough cleansing of the wound. Then administer 20 IU/kg of human rabies immunoglobulin (50% injected in and around the wound, if possible, and 50% given intramuscularly in the gluteal muscle). Inject 1 mL of human diploid cell vaccine into the deltoid muscle (or the anterolateral thigh in young children) on days 0, 3, 7, and 14. Tip: Do not administer the rabies vaccine and the immunoglobulin in the same site.

22. What is a *fight bite*?

A *fight bite* or *clenched fist injury* is a human bite that occurs when a fist strikes the teeth of an opponent. This usually involves the knuckles of the dominant hand. The laceration can involve the extensor tendon and its bursa, the superficial and deep fascia, and the joint capsule. These structures are contaminated with oral flora at the time of injury and are notorious for becoming infected. There are at least 42 species of bacteria in human saliva. The most frequently cultured organism from fight bites is *Streptococcus*, followed by *S. aureus* (usually penicillin resistant); 31% of these wound infections are due to gram-negative organisms, and 43% are due to mixed gram-negative and gram-positive organisms. Up to 29% of these infections may be due to a facultatively anaerobic gram-negative rod, *Eikenella corrodens.* This harmful organism is typically resistant to the semisynthetic penicillins, clindamycin, and the first-generation cephalosporins. However, it is usually sensitive to penicillin and ampicillin. These wounds should be meticulously cared for, with special attention given to thorough exploration and irrigation. Consider the polymicrobial nature of these infections when choosing antibiotics.

MARINE FAUNA (JELLYFISH, SHARK, AND VENOMOUS FISH)

23. How do I treat jellyfish or other coelenterate stings?

Jellyfish envenomate by injecting small harpoon-shaped spines from their nematocysts into their prey. The discharge is triggered by either physical or chemical stimulation. Frequently, *undischarged* nematocysts within the jellyfish tentacles remain in contact with the victim's skin. The concern, of course, is that the undischarged nematocysts may be stimulated to release additional venom into the victim. Acetic acid (vinegar) inhibits nematocyst discharge in *Chironex fleckeri* stings but has no effect on cysts that have already discharged. Further, inhibition of nematocyst discharge is probably species specific. Effective inhibitors in one species may trigger nematocyst discharge in another.

Acetic acid has been demonstrated to inhibit nematocyst discharge in all of the species of medically important Australian jellyfish.

If vinegar is not available, acidic drinks (soft drinks and fruit juices) may be tried. Although popular in folklore, urine has not been proven to inhibit nematocyst discharge and may even stimulate nematocyst discharge.

Immersion of the affected extremity in hot water may be of some benefit (after acetic acid decontamination). The nematocysts that remain in the skin can be removed by applying shaving cream, talc, baking soda, or flour and then shaving the area. The same treatment can be used for the stings from sea anemones or fire coral. There is an Australian box jellyfish *(Chironex fleckeri)* antivenin available. The recommended dosage for the antivenom is 1 to 3 ampules.

24. Name some venomous fish, and state what their venoms have in common. How can that feature of their venom be used in treatment?

Stingray, lionfish, scorpionfish, stonefish, catfish (i.e., freshwater catfish, sea catfish, coral catfish), oldwife fish, zebrafish, butterfly cod, spiny dogfish, rabbit fish, raffish, stargazer fish, surgeon fish, toadfish, weever fish, bullrout, sculpin, and stinging sharks all have heat-labile toxins (heat destroys the toxin). Barbs and spines may remain embedded in the wound and should be promptly removed. The venoms can be rendered nontoxic by placing the affected extremity of the victim (usually the foot) into hot water (45°C) for 90 minutes.

25. Question: How do I acquire antivenom for exotic snake or marine envenomations?

Call your local or regional poison center in the United States (1-800-222-1222) for assistance. They will have the most success in obtaining antivenom from the local zoo or aquarium or from another poison center. Note that although zoos and aquaria may possess antivenom for exotic envenomations, the supply is primarily intended for their workers in case of an emergency. They may choose to release specific antivenom under a *compassionate use* clause, but they are not obligated to do so.

26. How many people are killed by sharks worldwide annually?

Between 2001 and 2006 an average of 4.3 people died worldwide annually from unprovoked shark attacks. In contrast, approximately 100 million sharks are killed by people annually.

CROTALINAE ELAPIDAE (RATTLESNAKES, COPPERHEADS, WATER MOCCASINS, AND CORAL SNAKES)

27. What is a *dry* snakebite?

A dry snakebite is a bite in which no venom was introduced. About 20% to 25% of all U.S. rattlesnake bites do not result in envenomation. Coral snakes, lacking fangs, envenomate by *chewing* the skin; thus, as many as 50% of their bites are dry. Quick observations helpful in determining whether rattlesnake envenomation has taken place include the presence of fang marks that ooze nonclotting blood with surrounding ecchymosis and severe burning pain. These signs combined with microhematuria are characteristic of severe envenomation and a poor prognosis. In coral snake envenomation, there is usually little local tissue damage, and systemic signs may be delayed for as long as 12 hours. The earliest signs and symptoms include nausea, vomiting, headache, abdominal pain, diaphoresis, and pallor. Coagulopathy is not a feature of coral snake envenomation, but respiratory paralysis is the feared outcome.

28. True or false: Snakebites are uncommon but are highly lethal?

True and false. In the United States, snakebites are uncommon, and mortality is rare. The 2007 AAPCC report documented 6,550 snakebites (venomous and nonvenomous), and only two deaths (one from a rattlesnake and one from an unknown crotaline snake). Rattlesnakes accounted for more than half of the major medical outcomes, and copperheads one sixth.

29. List some of the epidemiologic characteristics of snakebites in the United States.

- Seventy-five percent occur from April to September.
- Forty-five percent occur between the hours of 2 P.M. and 6 P.M.
- Male-to-female victim ratio is 7:1.
- Fifty-five percent of victims are age 17 to 27 years.
- Eight-five percent of bites are on the fingers or hand; 15% involve the foot or ankle.
- Ethanol intoxication was present in 30% to 60% (especially if "pet" snakes are involved).
- Fifteen percent had previous snakebites.

30. List the three main clinical effects and the clinical signs of crotaline (pit viper) envenomation.

Crotaline snakebite manifests with three main clinical effects:

- Local (i.e., pain, edema, or ecchymosis)
- Systemic (i.e., hypotension, fasciculations, or multiorgan dysfunction syndrome)
- Coagulopathy (i.e., low platelets, elevated international normalized ratio [INR], low fibrinogen)

Signs of Crotaline snakebite include the following:

- Rapid onset of slow spreading edema (80%)
- Pain out of proportion to the puncture (72%)
- Weakness (65%)
- Light-headedness (52%)
- Nausea (48%)
- Erythema at the bite site (53%)
- Bleeding diathesis (52%)
- Lymphangitis, hypotension, shock, diaphoresis, and chills (58%)
- Paresthesias, taste changes, and fasciculations (33%)

31. What is *CroFab*? When should it be administered?

CroFab (Crotalidae Polyvalent Immune Fab) is an antivenom produced from the pooled serum of sheep immunized with one of four crotaline snake venoms, then digested with papain to produce antibody fragments (Fab and Fc). The more immunogenic Fc portion is eliminated during purification, leaving the four monospecific Fab preparations that are combined to form the final antivenom. It is provided as lyophilized powder and must be reconstituted (this takes 30 minutes). The goal is to attain initial control (defined as halting progression of all components of envenomation including local effects, systemic effects, and coagulopathy) by administering an initial loading dose of 4 to 6 vials. After control has been established, additional 2-vial maintenance doses are infused at 6, 12, and 18 hours. Since Food and Drug Administration (FDA) approval in 2000 of CroFab, Wyeth has recently ceased manufacture of Polyvalent Crotalidae Antivenin (a whole immunoglobulin [IgG] preparation), although some institutions may have supplies that have not yet been used.

Due to the relatively small Fab molecular size and consequent renal clearing, the effective CroFab duration of action may be insufficient for one-dose treatment of crotaline envenomation. Scheduled maintenance dosing is recommended after achieving initial control.

The Mexican pharmaceutical company Bioclon has developed an equine-derived polyvalent crotaline immune F(ab)2 antivenom for use in Mexico that is effective in neutralizing 15 venoms from North American snakes.

32. Can a crotaline bite cause a compartment syndrome?

Compartment syndrome may result, but it is unlikely because venom is usually deposited in the subcutaneous tissue, not in fascial compartments. Children, however, with the smaller body mass and potential for relatively deeper envenomations are more prone to develop compartment syndrome. It cannot be diagnosed reliably without directly measuring intracompartmental pressures because the signs and symptoms of compartment syndrome (i.e., paresthesias,

decreased pulses, and pain on motion) are similar to signs and symptoms of envenomation. Antivenin may improve compartment pressure. If the pressure is elevated initially, monitor the compartment pressure while administering antivenin. Perform fasciotomy only when pressures remain persistently elevated above 30 to 40 mm Hg. One exception may be the envenomated finger (i.e., tense, blue or pale with absent or poor capillary refill), which should be discussed with a toxicologist and surgeon. It can be treated with a digit dermotomy on clinical grounds.

33. What is the importance of the coloring of coral snakes, and what are the active components of its venom?

This small, thin, brightly colored snake is venomous; however, the king snake, which is nonvenomous, has similar coloration but a different pattern. Remember:

- "Red on yellow, kill a fellow" (coral snake)
- "Red on black, venom lack" (harmless snake)

This rhyme helps only with identifying North American Coral snakes. Coral snake venom contains a neurotoxin that irreversibly binds to presynaptic nerve terminals and blocks acetylcholine receptors. It may take weeks to regenerate the receptors. The clinical effects are slurred speech, ptosis, dilated pupils, dysphagia, and myalgias. Death results from progressive paralysis and respiratory failure. There is virtually no local tissue destruction.

34. How is coral snake envenomation treated?

Supportive care with good wound care, in addition to early treatment with 3 to 5 vials of elapid equine antivenom (Wyeth), is indicated for eastern coral or Texas coral snake envenomation. Western coral snake envenomation does not require antivenom. Wyeth no longer produces the antivenom, but some stock may still exist. Coralmyn®, a coral snake antivenom produced by the Mexican pharmaceutical company Bioclon, must undergo review by the FDA before being adopted for use in the United States. Coralmyn is effective in the neutralization of both clinically important coral snake venoms in the United States.

35. What prehospital treatments have been advocated for rattlesnake bites that are now considered to be ineffective or harmful?

Incising the wound and attempting to extract the poison by oral suction (cut and suck), venom extraction devices, electric shock to denature the toxin proteins, carbolic acid, strychnine, enemas, urine, cauterization, prophylactic antibiotics, ice packs (cryotherapy), and arterial tourniquets are ineffective and, in some cases, harmful. Venom extraction devices do not remove a significant amount of venom (0.04%–2% in one study). Nonsteroidal anti-inflammatory drugs may compound a crotaline venom-induced thrombocytopenic bleeding diathesis and should be avoided.

36. What prehospital nonantivenin treatments do make sense?

It is prudent to remain calm, avoid activity, remove jewelry or constricting items, use a lymphatic band, immobilize the extremity, follow good basic life support principles, and transport rapidly to the ED. A lymphatic constriction band (broad and flat band as opposed to a ropelike tourniquet) can be applied to exert a pressure great enough to occlude superficial veins and lymphatic channels (typically $>$20 mm Hg) but loose enough to admit one or two fingers. It has been shown in experimental models to delay the systemic absorption of venom and may have use in cases with prolonged transport time. In an animal model, the time to death after injection of venom can be prolonged, and the median lethal dose of venom can be increased, simply by immobilizing all four limbs (including the unbitten extremities). This technique has not been studied clinically.

37. What about exotic snakes (at least exotic by North American standards)?

In 2007 the AAPCC reported 153 exotic snake exposures (i.e., poisonous, nonpoisonous, and "unknown if poisonous"). There were 0 deaths, 4 life-threatening outcomes, and 32 moderate outcomes. An Antivenom Index exists that includes a catalog of all of the antivenoms stocked by North American zoos and aquariums. Possession of exotic venomous snakes may be restricted by law, and these cases should be reported to the authorities.

WEBSITES

American Association of Poison Control Centers: *http://www.aapcc.org/*

Scorpion envenomation: *http://emedicine.medscape.com/article/168230-overview*

Snake bite antivenom—resources and producers: *http://globalcrisis.info/latestantivenom.htm*

Widow spider envenomation: http://emedicine.medscape.com/article/772196-overview

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CHAPTER 72

SMOKE INHALATION

Richard E. Wolfe, MD

1. What is the most common way to die in a fire?
Smoke inhalation accounts for as high as 80% of fire-related deaths. It increases mortality by up to 20% over that predicted by age and extent of cutaneous burn alone.

2. Is smoke inhalation so lethal because it causes thermal injury to the lungs?
Not usually. Air has such a low heat-carrying capacity that it rarely produces lower airway damage. The upper respiratory tract generally cools hot air before it reaches the vocal cords. Steam, however, has 4,000 times the heat-carrying capacity of air and causes severe upper airway burns with fatal glottic edema, as well as bronchial mucosal destruction and alveolar hemorrhage.

3. Why is smoke inhalation so dangerous?
Carbon dioxide and carbon monoxide (CO), the major components of smoke, are responsible for a drop in the concentration of ambient oxygen from 22% to 5% to 10%. CO and, more rarely, hydrogen cyanide block the uptake and use of oxygen, leading to severe tissue cellular hypoxemia. Depending on the fuel, temperature, and rate of heating, smoke contains a wide variety of toxins. Soot may act as a vehicle in transporting these toxic gases to the lower respiratory tract, where they dissolve to form acids and alkali. Removal of the soot is impaired by action of certain of these toxins on respiratory cilia, leading to severe, delayed pneumonia.

4. Name the four clinical stages of smoke inhalation.
- **Stage 1:** Acute respiratory distress occurs 1 to 12 hours post-injury and is due to bronchospasm, laryngeal edema, and bronchorrhea.
- **Stage 2:** Noncardiogenic pulmonary edema (adult respiratory distress syndrome) occurs 6 to 72 hours post-injury secondary to increased capillary permeability.
- **Stage 3:** Strangulation occurs 60 to 120 hours post-injury from cervical eschar formation in patients with circumferential neck burns.
- **Stage 4:** Onset of pneumonia 72 hours after injury, usually from *Staphylococcus aureus, Pseudomonas aeruginosa*, or gram-negative organisms.

5. How should smoke inhalation victims be managed in the field?
All victims should be placed on a 100% nonrebreather mask, even if they are asymptomatic. Oxygen administration dramatically accelerates the washout of CO, shortening the half-life from 4 hours at room air to about 90 minutes. Endotracheal intubation is indicated for patients in respiratory distress. When intubated, the patient should be suctioned aggressively to remove inhaled soot. Patients with a loss of consciousness or altered mental status should be transported to a facility capable of providing hyperbaric oxygen (HBO) therapy.

6. What should I ask the emergency medical technicians (EMTs) about the fire?
Ask if the patient was trapped in a closed space because significant inhalation injury would not occur in an open area. Try to determine what material was burning. The fuel is of primary importance in determining the composition of smoke and the risk to the patient.

7. Name some toxins produced by smoke and the materials from which they derive.

- **Hydrogen cyanide:** combustion of wool, silk, nylons, and polyurethanes found commonly in furniture and paper.
- **Aldehydes, acrolein:** wood, cotton, paper, and plastic materials.
- **Hydrogen chloride, phosgene:** pyrolysis of chlorinated polymers; polyvinyl chloride (wire insulation materials); chlorinated acrylics; and wall, floor, and furniture coverings.
- **Oxides of nitrogen:** nitrocellulose film.
- **Sulfur dioxide, hydrogen sulfide:** rubber.

8. What are the earliest clinical manifestations of acute inhalation injury following smoke exposure?

Inflamed nares, cough, sputum production, and hoarseness are the first signs of injury. This is because the nasopharynx and larynx are exposed to the highest concentration of inhaled toxins leading to the most severe chemical burns. Furthermore, the proximal airway is usually the only part of the airway subjected to thermal burns. However, even when injured, nasopharyngeal and laryngeal edema may be delayed. Furthermore, rapid progression to complete airway obstruction may occur in patients with mild symptoms. For this reason, close observation followed by early airway management is often necessary to ensure patient safety.

9. Why is HBO therapy thought to be beneficial in smoke inhalation?

- HBO therapy provides increased oxygen to poorly functioning mitochondrial enzymes inhibited by CO and cyanide.
- HBO therapy at 3 atmospheres (atm) decreases the half-life of CO to 23 minutes.
- HBO therapy has been shown to reduce smoke-induced pulmonary edema.
- At a cellular level, HBO therapy decreases the formation of intercellular adhesion molecule-I on the endothelial membrane, which prevents neutrophils from infiltrating the central nervous system and causing a damaging inflammatory reaction and permanent neurologic sequelae.

10. How do I make the diagnosis of smoke inhalation injury?

Bronchoscopy is needed to confirm the presence of inhalation injury. Soot deposition in the airway, extensive edema, mucosal erythema, hemorrhage, and ulceration confirm that smoke inhalation has occurred. The initial bronchoscopy may be relatively normal because hyperemia and edema formation may take some time to evolve. A normal proximal airway does not rule out more distal injury.

11. How should asymptomatic patients be managed?

Observe the patient in the ED for a few hours first. If still asymptomatic, provide comprehensive discharge instructions on when to return. Although the physical examination cannot reliably rule out complications such as delayed noncardiogenic pulmonary edema or pneumonia, ancillary studies and ED or in-hospital observation are not cost effective. The patient should be instructed to return to the ED if shortness of breath, chest pain, or fever occurs.

12. If the patient's pulse oximetry is normal, would arterial blood gas analysis yield additional information?

In the presence of carboxyhemoglobin (CO), pulse oximetry may yield a falsely elevated (normal) reading. Arterial blood gases are of limited use and may be helpful only if the oxygen saturation is measured directly and not derived from the PaO_2 measurement. Although an increased alveolar-arterial gradient may correlate with smoke inhalation injury, it does not predict the severity of injury. Arterial blood gases are most useful in determining hypoventilation (increased PCO_2) and the presence of a metabolic or respiratory acidosis.

13. Should I get a chest radiograph on all patients with a history of smoke inhalation?

No. A chest radiograph offers little benefit in the ED. Chest radiographs are normal immediately after smoke inhalation injury, and abnormalities appear only on a delayed basis. A chest radiograph is not indicated in asymptomatic patients, and in most instances, it is useful only as a baseline in symptomatic patients.

14. Can I use the standard burn formula for intravenous fluids if smoke inhalation is present?

Patients with cutaneous and inhalation injuries pose a difficult problem because their fluid requirements are usually greater, but because of leaky capillaries, they are much more likely to develop membrane permeable pulmonary edema. Intravenous fluids must be guided by regular clinical reevaluation (i.e., breath sounds, oxygen saturation, urinary output, vital signs) rather than by formulas. Swan-Ganz monitoring may be required.

15. Is HBO therapy the only available therapy for cyanide poisoning?

No. Either the *Lilly* cyanide antidote kit or hydroxocobalamin (Cyano kit) can be used for victims of cyanide toxicity.

16. Tell me about hydroxocobalamin.

Hydroxocobalamin (vitamin B_{12}) reduces cyanide concentrations by combining with cyanide to form cyanocobalamin. It can be considered for victims who are comatose, in cardiac arrest, or have clear signs of cardiovascular extremis. If hydroxocobalamin is used, it should be given as early as possible. The usual dose is 5 g intravenously (IV).

17. How does the Lilly cyanide antidote kit work?

Cyanide binds to the ferric ions, blocking the mitochondrial cytochrome oxidase pathway and cellular respiration. The cyanide antidote kit acts in two ways to limit this:

- Nitrites generate methemoglobin, creating heme-ferric ions to compete with cyanide with mitochondrial ferric ions
- Sulfur transferase (rhodanese) binds cyanide molecules to sulfur-forming thiocyanate, which is nontoxic and eliminated in the urine. Thiosulfate accelerates this process by increasing available sulfur molecules. (Fig. 72-1.)

18. When should I use the cyanide antidote kit?

Symptomatic patients can have CO or cyanide toxicity. Nitrites can cause more prolonged asphyxia in patients with hypoxemia and elevated carboxyhemoglobin (COHb) fractions. These drugs should be reserved for patients in extremis or who remain critically ill after intubation and 100% oxygenation. The sodium thiosulfate portion of the kit can be used safely even when the measured oxygen saturation is low. High lactate levels can help distinguish cyanide from CO because elevations in serum lactate correlate well with cyanide toxicity.

KEY POINTS: SMOKE INHALATION

1. Obtain CO level and treat any patient who has inhaled smoke in an enclosed space with nonrebreather high-flow mask oxygen.
2. Consider cyanide poisoning when the patient inhaled smoke from burning furniture fabric (e.g., wool, silk, or polyurethanes).

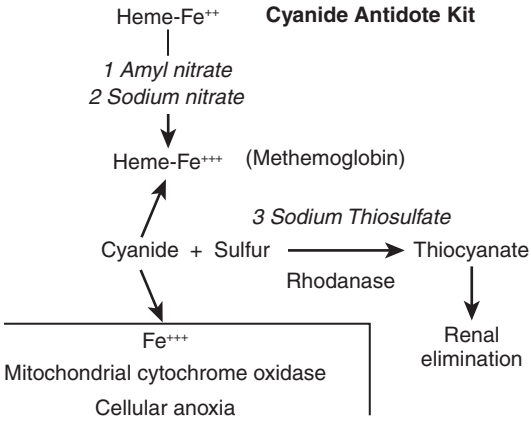

Figure 72-1. Lilly cyanide antidote kit.

19. How do I administer the cyanide antidote kit?

Administer a dose of 12.5 g of intravenous sodium thiosulfate. Amyl nitrite inhalers in patients in extremis without intravenous access can be given every 3 to 4 minutes. If a patient is apneic, break one of the amyl nitrite inhalants inside the resuscitation bag. When an intravenous line is established and indications for nitrites are present, the full amount of a 10-mL ampule or 5 to 10 mg/kg of sodium nitrite should be administered IV over 4 minutes.

20. Why is CO so dangerous?

CO is a colorless, odorless gas that has a 210 times greater affinity for hemoglobin than does oxygen. Even when exposed to low levels, it accumulates resulting in impaired cellular oxygen utilization. Fetal hemoglobin has an even greater affinity for CO.

21. How do I make the diagnosis in the ED?

The obvious history of any exposure to fire or smoke in a confined space. The more subtle presentation is early morning headache, which improves after exiting a residence with a defective heating system. A CO level should be obtained on all patients in whom the diagnosis is considered.

CONTROVERSY

22. Isn't the early respiratory failure seen in smoke inhalation victims worsened by aggressive crystalloid resuscitation?

Respiratory failure from interstitial fluid accumulation is a rare event. When it occurs, it is caused by capillary leakage due to inflammation of pulmonary tissue. The amount of crystalloid used during resuscitation does not increase the risk or the severity of the resultant pulmonary edema. Fluids should not be withheld in a patient with severe cutaneous and respiratory burns.

23. How do I treat CO poisoning?

All patients should be placed on high-flow O_2 via a nonrebreather bag reservoir mask, which will reduce the half-life of CO from 4 to 5 hours on room air to 1 hour. Although the long-term

benefit of HBO therapy has been called into question with conflicting published studies, most still recommend its use in the following patients:

- A pregnant woman with a CO level greater than 15
- Any patient with a neurologic abnormality (i.e., coma or altered momentum)
- Any patient with cardiac ischemia or instability

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COMMON DRUGS OF ABUSE

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CHAPTER 73

1. Aren't the occurrences of heroin and other opioid abuse decreasing in frequency?

Actually, it is just the opposite. The data collected by the American Association of Poison Control Centers (AAPCC) demonstrated an increase from 5% to 13% in opioid-related deaths from 1995 to 2003. Analgesics (opioids and non-opioids) are the most common killer in toxic ingestions of pharmaceuticals every year since 1995. Opioids represent approximately half of those deaths each year. The Drug Abuse Warning Network in the United States showed a 25% increase in ED visits involving heroin from 1998 to 2002.

2. What do the terms *opium, opiate, opioid,* and *narcotic* mean?

- **Opium** is a mixture of alkaloids, including morphine and codeine, extracted from the opium poppy.
- An **opiate** is a natural drug derived from opium (e.g., heroin, codeine, and morphine).
- An **opioid** is any drug that has opium-like activity, including the opiates and all synthetic and semisynthetic drugs that interact with opioid receptors in the body (e.g., hydrocodone and oxycodone).
- The term ***narcotic*** is nonspecific; it refers to any addictive drug that reduces pain, alters mood and behavior, and usually induces sleep or stupor and more specific to law enforcement than medicine.

3. What is the typical clinical presentation of opioid poisoning?

The classic triad of opioid poisoning is **central nervous system (CNS) depression, respiratory depression**, and **miosis**. Patients who have overdosed on opioids are hyporeflexic and have decreased bowel sounds. They may be hypothermic, cyanotic, and mildly hypotensive and bradycardic.

4. Do all opioid intoxication cases present with miosis?

No. Mydriasis or normal pupils can occur with:

- Intoxication of specific synthetic opioids (i.e., meperidine, propoxyphene, or pentazocine)
- Diphenoxylate-atropine (Lomotil)
- After naloxone use
- With hypoxia
- With mydriatic eye drops use
- Coingestion of other mydriatic drugs (e.g., anticholinergics)
- Occasionally, phenylephrine, instilled into the patient's nares by paramedics for nasal intubation, may spill into the patient's eyes causing mydriasis (Table 73-1).

5. How should a patient with respiratory compromise from opioid overdose be treated?

Resuscitation takes precedence over naloxone administration. Support the patient's ventilation with a bag-mask until the opioid antagonist is administered. Intubate the apneic or cyanotic patient if the patient does not awaken after naloxone. Obtain a serum glucose level, administer oxygen, and consider thiamine administration in the patient with altered consciousness. Administer activated charcoal if the opioid ingestion was recent.

TABLE 73-1. COMMON CAUSES OF NONOPIOID-RELATED MIOSIS	
Sympatholytic agents	Clonidine, antipsychotics, oxymetazoline, and tetrahydrozoline
Cholinergic agents	Organophosphates, carbamates, nicotine, pilocarpine, phencyclidine, and similar congeners
Miscellaneous	Pontine infarct and Horner's syndrome

6. What is the appropriate naloxone dose?

For children younger than 5 years old or less than 20 kg, administer 0.1 mg/kg intravenously. In a patient who has CNS depression only, infuse an initial dose of 0.2 to 0.4 mg intravenously. If there is no response to this dose, repeated doses up to 2 mg can be given. Administer 2 mg intravenously initially for the apneic or cyanotic adult or child. For patients who abuse opioids or who use opioids for chronic pain, infuse 0.1 mg to wake the patient without inducing opioid withdrawal. Additional doses should be given judiciously to patients who consume opioids chronically. Opioid withdrawal is unpleasant to the patient but is not life-threatening.

7. Can naloxone be administered by other routes besides intravenously?

Yes. If venous access cannot be accomplished, administer the naloxone intramuscularly or subcutaneously. A dose of 0.8 mg subcutaneously has an equal time to effect as 0.4 mg intravenously. Naloxone can also be administered through the endotracheal tube, intranasally, intraosseously, or injected sublingually. Naloxone is not effective orally because of significant first pass metabolism.

8. Do all patients respond to a standard dose of naloxone?

No. Larger doses of naloxone may be required to reverse the effects of synthetic opioids, such as codeine, diphenoxylate-atropine (Lomotil), propoxyphene (Darvon), pentazocine (Talwin), codeine, dextromethorphan, and the fentanyl derivatives. If an opioid overdose is suspected and the patient does not respond to an initial naloxone dose, repeat additional doses until a response is noted or until 10 mg has been given. If there is no response to 10 mg of naloxone, it is unlikely to be an isolated opioid overdose.

9. How long does the clinical effect of naloxone last?

The duration of action of intravenous naloxone is 40 to 75 minutes, although the serum half-life is shorter. Many oral and some injected opioids produce clinical effects that last for 3 to 6 hours. Although the duration of action of most opioids is much longer than that of naloxone, resedation is uncommon, particularly with short-acting parenteral opioids (e.g., heroin). Most oral opioids, particularly long-acting agents (e.g., methadone or sustained release morphine) last several hours and may require additional naloxone doses and hospital admission.

10. How should recurrent sedation and respiratory depression resulting from a long-acting opioid be treated?

Treat most patients with boluses of naloxone as needed, along with hospital admission, supplemental oxygen, and close monitoring in an intensive care setting. On occasion, patients require several doses of naloxone over a short time interval to maintain normal oxygenation. In these cases, a continuous naloxone infusion may be started. A naloxone infusion is administered at an hourly rate that provides two thirds of the dose needed to reverse the respiratory depression. Thus, multiply the bolus dose being given by 6.6, mix it into one liter of crystalloid, and infuse it at 100 mL/h. The infusion can be adjusted based on the patient's symptoms of withdrawal or sedation.

11. Should naloxone be administered empirically to every patient with altered mental status?

No. Although naloxone is a safe medication, the response to naloxone has been shown to occasionally cloud the diagnostic picture. If a patient presents with an obvious sympathomimetic or anticholinergic syndrome (i.e., agitated and stimulated), the patient will not benefit from naloxone. In addition, if the opioid toxidrome is obvious and the patient's ventilatory status is adequate, naloxone may stimulate opioid withdrawal, which is more difficult to control in a busy ED than a slightly sedated patient.

12. Who should be observed in the ED, and for how long?

It depends. Patients who inject heroin can be observed for at least 2 hours after a dose of naloxone, as resedation and noncardiogenic pulmonary edema almost always occur during this period. Most consider observation for up to 4 hours after the last dose of naloxone adequate in an asymptomatic patient who used a parenteral opioid. This extended period may allow for recognition of coingestants and recurrent respiratory depression. Occasionally, patients who have inadequate ventilation, which necessitates treatment, or who develop complications of opioid use must be admitted. Patients who ingest long-acting opioids, such as methadone, may require admission for 24 hours or longer. Patients who inject long-acting opioids should be observed for 4 to 8 hours or admitted. Patients should be normoxic off oxygen, awake, and ambulatory before discharge, and preferably discharged into the care of a competent adult.

13. What are the signs of opioid withdrawal?

Signs of withdrawal include anxiety, yawning, lacrimation, rhinorrhea, diaphoresis, mydriasis, nausea and vomiting, diarrhea, piloerection, abdominal pain, and diffuse myalgias. Opioid withdrawal typically occurs approximately 12 hours after last heroin use and 30 hours after last methadone use. Seizures, dysrhythmias, and other life-threatening complications are not consistent with opioid withdrawal.

14. How is opioid withdrawal best treated?

Treatment is symptomatic. Intravenous fluids, sedation, antiemetics, and antidiarrheal agents are mainstays of treatment. Clonidine, 0.1 to 0.2 mg orally, may also be helpful. However, published cases describe a concomitant abuse of clonidine, because the user feels it enhances the opioid euphoria. If naloxone is given, the most severe withdrawal symptoms typically resolve in 45 to 75 minutes.

15. What are body packers and stuffers?

- *Body packers* are individuals who carefully pack large amounts of illegal drugs into small, glass, or plastic vials. The vials are sealed and ingested by the human carrier along with an antimotility agent. The individual then travels by plane or other vehicle to another location. *Body packing* is used to transport illegal drugs, such as heroin or cocaine to other countries. The individual then defecates the vials and delivers them to the recipient. The packets rarely rupture, but it can be life-threatening if they do.
- *Body stuffers* are individuals who quickly ingest *(stuff)* poorly wrapped illegal drugs while attempting to evade law enforcement. The wrapping containing the drug is usually referred to as a *baggie*. Commonly it is a much smaller amount of drug than body packers handle and is loosely wrapped. The drug is typically absorbed quickly, and the patient usually develops symptoms shortly after ingestion.

16. How should body stuffers/packers be managed?

Urine drug screening is not helpful for determining which drug or if any drug was ingested. In addition, the patient's history for timing, content, and amount of the ingestion is unreliable.

- Body stuffers should receive activated charcoal and be observed in a monitored setting for at least 8 hours. Radiographs are not helpful. If the patient develops symptoms, admit the patient to an intensive care setting for observation.

- The packets from body packers can be seen on plain abdominal radiographs, radiographs with oral contrast material (Gastrografin), or abdominal computed tomography (CT). Based on limited data, contrasted radiographs and CT scan are the most sensitive. Body packers should receive activated charcoal and polyethylene glycol electrolyte solution (Colyte, GoLYTELY) to enhance elimination through the colon. Polyethylene glycol may be administered through a nasogastric tube at approximately 2 L/h until all packets have cleared. Clear rectal effluent is not a sufficient end point to end decontamination. Repeat radiologic testing with abdominal CT or a radiograph with oral contrast should be used to determine when all packets have cleared. Enemas may be used if the packets are in the distal colon or are felt on digital rectal examination. Typically, bowel irrigation for more than 6 hours is futile in moving stubborn vials. Surgery is rarely needed to remove retained packets. Occasionally, packets may take days to evacuate.

17. How useful are toxicologic screens for opioids, and which opioids are not often detected?

Toxicologic screens are not generally helpful in acute management. Not only are the results delayed, but the clinical presentation is also more helpful than the insensitive test. Opiate screens do not detect methadone or other synthetic opioids, such as fentanyl, pentazocine, meperidine, oxymorphone, oxycodone, and propoxyphene. Ingestion of poppy seeds does not commonly cause a positive screen because the lower limit threshold has been raised. However, with further testing, this erroneous cause of positive screens can be excluded. Fluoroquinolones can cause a false-positive for opiates.

18. Are there any other tests that should be checked in patients with opioid ingestions?

Obtain acetaminophen levels in all patients because it is often combined with hydrocodone, oxycodone, propoxyphene, and codeine. Also obtain a metabolic panel, salicylate level, and electrocardiogram.

19. What is the most common pulmonary complication of opioid use?

Noncardiogenic pulmonary edema occurs in 3% of nonhospitalized opioid intoxications. The mechanism is unclear, but it may be a result of capillary permeability and fluid leak, or from breathing deeply and quickly against a closed glottis. The patient presents with pink frothy sputum, cyanosis, and rales. Bilateral alveolar infiltrates are seen on the chest radiograph. Naloxone does not reverse the process, and many patients will need mechanical ventilation. Heroin, methadone, morphine, and propoxyphene have been associated with noncardiogenic pulmonary edema.

20. Can opioids cause seizures?

Seizures are rare in patients with therapeutic doses of opioids, but they have been reported with use of synthetic opioids (i.e., meperidine, tramadol, pentazocine, and propoxyphene) and chronic use of morphine.

21. Is it safe to give dextromethorphan or meperidine to patients on antidepressant medications?

The combination of these opioids with antidepressants may precipitate serotonin syndrome. Meperidine and dextromethorphan inhibit serotonin reuptake similar to selective serotonin reuptake inhibitors. A combination of these opioids and monoamine oxidase inhibitors (MAO-Is) is also contraindicated as MOA-Is decrease serotonin metabolism.

22. Why should I avoid prescribing meperidine (Demerol)?

The duration of action of meperidine is only 2 to 3 hours, shorter than morphine or hydromorphone. In contrast to morphine, meperidine's half-life is prolonged by hepatic disease, resulting in toxic effects after repeated doses in patients with liver disease. Seizures are an adverse effect of normeperidine, a renally cleared metabolite of meperidine. Normeperidine levels are elevated with repetitive administration of oral meperidine, renal

failure, and concomitant use of drugs that induce hepatic enzymes, such as phenytoin, phenobarbital, and chlorpromazine. Naloxone does not terminate the seizures. Normeperidine can cause CNS agitation, tremors, and psychosis. Meperidine can produce serotonin syndrome when combined with other serotonergic agents.

23. Which antidiarrheal agent can cause significant toxicity if ingested?

Lomotil (diphenoxylate 2.5 mg + atropine 0.025 mg). Most toxic cases occur in children. Classically the overdose is a two-phase toxicity: Phase 1, anticholinergic symptoms (flushing, dry mouth) and Phase 2, opioid effects. However, this pattern is uncommon. Delayed presentations have been reported, and all children should be observed in a monitored setting for at least 24 hours.

Loperamide is a nonprescription antidiarrheal agent derived from diphenoxylate. Acute overdoses usually produce only mild drowsiness.

24. Which opioid can produce ventricular dysrhythmias, a wide QRS complex, mydriasis, and seizures?

Propoxyphene has a quinidine-like effect that blocks sodium channels, similar to cyclic antidepressants. Large doses of naloxone (10 mg) may reverse the CNS depression, but not the cardiotoxic effects. Sodium bicarbonate has been used successfully for propoxyphene-induced dysrhythmias. Propoxyphene is no more effective for analgesia than salicylates, acetaminophen, or codeine.

25. What are designer drugs, and what are the two most notorious designer drugs that have been used?

Designer drugs are substitutes for other chemicals or drugs that are popular with illicit drug users. They are made inexpensively in clandestine laboratories. 3-Methylfentanyl is an analog of fentanyl known as *China white* or *Persian white*. It is 2,000 times more potent than morphine and 20 times more potent than fentanyl. It can cause respiratory compromise quickly. It does not cause the abbreviated *rush* of heroin, but instead causes a longer duration of euphoria.

MPTP (1-methyl-4-phenyl-1,2,5,6 tetrahydropyridine) is a compound that was produced accidentally during the synthesis of MPPP, a meperidine analog. MPTP is cytotoxic for dopaminergic neurons in the substantia nigra. It produces a Parkinson-like syndrome that is permanent and occurs after a single ingestion of MPTP. The symptoms do not respond to typical anti-Parkinsonism medications.

26. What over-the-counter cold remedy is sometimes abused by teenagers?

Dextromethorphan (DM) is the d-isomer of codeine. Its metabolite stimulates the release of serotonin and acts at the phencyclidine receptor site, which accounts for its abuse as a hallucinogen. While *Coricidin* is the trade name most well known, DM is available in many other cough medications. It is also known as *ROBO, DEX, red devils, triple C, CCC,* and *skittles*. DM toxicity may present with symptoms of opioid toxicity but more commonly presents with slurred speech, nystagmus, hyperexcitability, vomiting, and ataxia. Not all individuals can metabolize dextromethorphan to its psychoactive metabolite. Naloxone does not usually reverse the symptoms of toxicity. DM does cause false positive phencyclidine results on urine screening, but it usually does not produce positive results for opiates. Coingredients may cause a predominance of that clinical syndrome (anticholinergic or sympathomimetic toxidrome). Acetaminophen is commonly a coingredient and should be screened for in all patients abusing DM.

27. Name another analog of codeine.

Tramadol (Ultram) is a synthetic analog of codeine. The usual effects with overdose are mild sedation and opioid effects. Overdoses have occasionally been associated with seizures, hypertension, respiratory depression, and agitation. The seizures do not respond to naloxone. Although the drug has a low abuse potential, it is not recommended for patients with an opioid dependence history.

SEDATIVE-HYPNOTICS

28. What is a sedative-hypnotic?

Sedatives-hypnotics are drugs that primarily cause relaxation and tranquilization and induce drowsiness and sleep. There is no consistent structural relationship among the agents of this group. In sufficient quantities all drugs of this group result in CNS depression.

29. What medications fall into this category?

There are three groups: benzodiazepines, barbiturates, and miscellaneous. Some examples of miscellaneous sedative-hypnotics are chloral hydrate, ethanol, and gamma-hydroxy butyrate (GHB). Many of the miscellaneous agents are also pharmaceutical agents. For example, ethanol is used in the treatment of methanol and ethylene glycol toxicity and GHB (Xyrem) is used for narcolepsy. These drugs are also commonly abused.

30. What is a typical presentation of sedative hypnotic intoxication?

Mild intoxication presents with slurred speech, ataxia, and loss of coordination. Moderate to severe intoxication presents with greater CNS depression. Respiratory depression may occur with large ingestions, and is compounded by other agents that suppress respiratory drive, such as opioids or ethanol. Pupils are usually midsize and reactive, and may be disconjugate. There are also symptoms specific to individual drugs. Some examples are choral hydrate (pear odor), ethchlorvynol (pulmonary edema, vinyl odor), and glutethimide (anticholinergic effects) (Table 73-2).

31. Don't many overdoses present this way?

Many overdoses present with CNS depression. However, some intoxications also present with a pattern of symptoms known as a toxidrome (Table 73-3). Signs of antipsychotic intoxication include sedation and are similar to sedative-hypnotics but also commonly include tachycardia, mild hypotension, and occasionally miosis. CNS depression is also a common presentation of illness other than intoxication. Maintain a broad differential while evaluating these patients for such illnesses as meningoencephalitis, intracranial hemorrhage, hypoglycemia, shock, and sepsis.

32. How do sedative-hypnotics cause CNS depression?

Most sedative-hypnotics, particularly benzodiazepines and barbiturates, cause CNS depression by enhancing the effects of gamma-aminobutyric acid (GABA), an inhibitory neurotransmitter in the brain. Benzodiazepines increase the frequency of the opening of chloride channels associated with GABA. Propofol and barbiturates directly open the chloride channels, potentially causing greater sedation and respiratory suppression. Propofol may also inhibit excitatory brain neurotransmitters, adding to the GABA effects.

TABLE 73-2. CLINICAL PRESENTATIONS OF LESS COMMON SEDATIVE-HYPNOTICS

Chloral Hydrate	Vomiting and ventricular dysrhythmias
Ethchlorvynol	Vinyl-like odor on breath, prolonged coma, and noncardiogenic pulmonary edema
Glutethimide	Cyclic coma, anticholinergic symptoms (tachycardia uncommon), and thick secretions
Methaqualone	Hyperreflexia, clonus, and muscle hyperactivity
Meprobamate/Carisoprodol	Euphoria and concretions in stomach may be seen on radiographs

TABLE 73-3. CLINICAL PRESENTATION OF TOXIDROMES RESULTING IN DEPRESSED OR ALTERED MENTAL STATUS

Toxidrome	Presentation
Opioid	Central nervous system depression, miosis, respiratory depression, hypothermia, and mild bradycardia
Sympathomimetic	Psychomotor agitation, mydriasis, hypertension, tachycardia, diaphoresis, hyperthermia, and seizure
Cholinergic	Bradycardia, bronchorrhea, miosis, salivation, lacrimation, urination, diaphoresis, diarrhea, vomiting, diarrhea, altered mental status, and seizures
Anticholinergic	Delirium, sedation, mydriasis, dry/flushed skin, tachycardia, decreased/absence of bowel sounds, seizures, and mild pyrexia

33. How do I make the diagnosis of sedative/hypnotic overdose in a patient with undifferentiated CNS depression?

Making the diagnosis can be difficult. Elicit help from friends, family, and involved prehospital providers and police. Review the patient's medical records for previous visits and search the belongings for paraphernalia and empty bottles. Often a specific agent will not be identified; rather, only a constellation of symptoms seen with sedative hypnotic intoxication may be recognizable. Routine laboratory and radiologic studies including chemistries, cerebrospinal fluid analysis, and cranial CT scans may assist in ruling out metabolic, infectious, and CNS disorders as the cause. Urine drug screens are available, but typically are not helpful.

34. Is there a role for drug screens or specific drug levels?

Routine drug screens are often not useful in acute patient management. The sensitivities and specificities of the assays for detecting specific drugs are variable. For example, the assay for benzodiazepines in the most commonly used urine drug screen is designed only to detect the metabolite of some older, long-lasting benzodiazepine medications. Many newer benzodiazepines will not be detected. The same is true for many barbiturates. Most sedative-hypnotics are not tested for on the routine urine drug screen and thus their use cannot be excluded. If the screen is positive, it only indicates use within the past several days and may not correlate with clinical presentation. Other agents can cause a false-positive result and lead to missing the true etiology for the altered mental status. In addition, many other chemicals and drugs cause altered mental status but will not be present on urine toxicology screening (e.g., jimson weed, isopropanol, inhalant toxicity, lithium, ketamine, chloral hydrate, and bromides).

Because the most important treatment in sedative-hypnotic intoxication is supportive care, recognizing the intoxication pattern is more helpful than toxicology testing.

35. What is the treatment for sedative-hypnotic overdose?

Rapid resuscitation is the initial treatment. Manage the patient's airway, assess the respiratory effort and oxygenation, evaluate the circulation and perfusion, and examine for neurological deficits (ABCDs). After resuscitation, initiate gastrointestinal decontamination with activated charcoal (within approximately 1 hour of ingestion) and then exclude other causes for altered mentation, acid-base disturbances, or unstable hemodynamics. Do not use flumazenil in the undifferentiated intoxication.

36. How do patients die of sedative-hypnotic overdose?

Respiratory depression and resultant hypoxia is the cause of most deaths.

37. What is the appropriate way to decontaminate the gastrointestinal tract?

Administer 1 g/kg of activated charcoal to all patients with significant ingestions who arrive within approximately 1 hour of ingestion. Pulmonary aspiration of activated charcoal causes a significant pneumonitis and occasionally permanent sequelae. Intubate patients with decreased mental status and airway reflexes prior to administering activated charcoal. Do not perform orogastric lavage for sedative-hypnotic intoxication.

38. Are there specific antidotes for sedative hypnotic intoxication?

Flumazenil can be used for overdose of benzodiazepine and related medications such as zolpidem.

39. How does flumazenil work?

Benzodiazepines and zolpidem act as GABA-A receptor agonists. Flumazenil antagonizes the effects of these drugs by competitively inhibiting the GABA receptor. Administer 0.2 to 0.5 mg of flumazenil intravenously in increasing doses to a generally accepted maximum dose of 5 mg. If there is no response with 5 mg, consider another intoxication, coingestant, or etiology for the patient's responsiveness.

40. Should flumazenil be given empirically to all patients with depressed mental status?

No. Flumazenil may be used for a patient with iatrogenic toxicity during procedural sedation or in an unintentional ingestion by a benzodiazepine-naïve child or adult. Consider it with a sole benzodiazepine overdose causing significant CNS depression. It has no role in undifferentiated or mixed overdose because it can induce seizures, unmask the effects of a coingestant, and rarely cause life-threatening dysrhythmias. Flumazenil may also induce seizures and withdrawal symptoms in chronic benzodiazepine users. The onset of flumazenil is 1 to 5 minutes and duration of effect is 1 to 4 hours. Sedation will resume after its effects have worn off. Most patients with a benzodiazepine overdose will do well with only supportive care and do not require flumazenil.

The ideal patient for flumazenil use would be iatrogenic oversedation with none of the following contraindications:

- Prior seizure history
- Electrocardiogram (ECG) evidence of cyclic antidepressants
- Chronic benzodiazepine use
- Abnormal vital signs, including hypoxia
- Coingestants that provoke seizures or dysrhythmias

41. What is GHB?

It is an abbreviation for gamma-hydroxybutyrate, which is a naturally occurring human neurotransmitter similar in structure to GABA. GHB has been used as a sleep aid, anesthetic, and muscle builder. It is sold on the Internet and abused for its mild sedating and euphoric effects. Although restricted by the Food and Drug Administration in the 1990s, GHB is available again (trade name Xyrem) as a tightly controlled treatment for narcolepsy. However, it is easily synthesized; recipes and materials are widely available. Congeners, including gamma-butyrlactone and 1,4-butanediol, are metabolized to GHB and have the same effects and are common.

42. How does a GHB overdose present?

Most ingestions of GHB are mild and produce minimal sedation and euphoria. Rarely, patients overdose on GHB and present to the ED with a decreased level of consciousness. In contrast to other sedative hypnotic intoxications, the level of consciousness tends to fluctuate between mild agitation and severe CNS depression. Airway reflexes are usually intact and often hypersensitive. An attempt at direct laryngoscopy may cause the patient to quickly sit up and be agitated for several minutes. Because the clinical effects of GHB usually last less than 6 hours, decisions about airway management should be based on the patient's respiratory status and the ability to monitor oxygenation closely in the ED. Although naloxone, flumazenil,

and physostigmine have been described as reversal agents in GHB intoxication, no antidote has consistently been shown to be effective. Death from GHB intoxication is generally from respiratory failure.

43. What are the effects of GHB withdrawal?

Recreational users of GHB manifest withdrawal symptoms of anxiety, insomnia, disorientation, tachycardia, hypertension, and visual and auditory hallucinations. GHB withdrawal is similar in presentation to benzodiazepine withdrawal, but with greater intensity.

44. How do zolpidem and benzodiazepine overdose differ?

Zolpidem (Ambien) acts similarly to benzodiazepines by binding at the alpha-1 subunit of the GABA-A receptor, and is useful as a sleep aid. Due to its specific affinity for the alpha-1 subunit, zolpidem has minimal muscle relaxant, anxiolytic, and anticonvulsant properties. Therefore, zolpidem intoxication presents with CNS depression; respiratory depression is uncommon.

45. What is a *Mickey Finn*?

A *Mickey Finn* is a drug-laced drink named for a mafia-associated bartender from Chicago in the 1920s. Specifically it refers to a mixture of chloral hydrate and alcohol. Alcohol and chloral hydrate act to potentiate each other's effects, and prolong their duration of effect as they are metabolized via the same pathway. Mr. Finn would use the drink to induce his victims into unconsciousness, and then relieve them of all their valuables.

MUSHROOMS

46. What are the symptoms and signs of mushroom poisoning?

Many mushrooms contain toxins that cause gastrointestinal manifestations including nausea, vomiting, and diarrhea. Certain species have toxins that are associated with more severe gastrointestinal manifestations or other characteristic symptoms and signs (Table 73-4).

47. Which mushroom's toxins cause the most concern?

Amatoxins, which are cyclopeptides found in *Amanita* and some *Galerina* species. The classic presentation of amatoxin poisoning includes an initial asymptomatic 6- to 12-hour period followed by gastrointestinal symptoms. Severe hepatotoxicity becomes evident 24 hours to several days after the initial ingestion.

TABLE 73-4. MANIFESTATIONS OF MUSHROOM POISONING

Mushroom Species	Toxin	Symptoms and Signs
Amanita phalloides, Galerina	Amatoxins	Delayed-onset GI manifestations, hepatic failure
Gyromitra	Monomethylhydrazine	Delayed-onset GI manifestations, CNS manifestations, hemolysis
Psilocybe	Muscimol, psilocybin	Anticholinergic (including hallucinations and seizures)
Clitocybe	Muscarine-containing	Cholinergic
Coprinus	Coprine	Disulfiram-like reaction with ethanol

CNS, central nervous system; GI, gastrointestinal.

48. Do symptoms within 6 hours absolutely exclude amatoxin ingestion?
No. Not all patients exhibit the classic presentation. In addition, mushroom ingestion often involves more than one species. Consider the possibility of amatoxin ingestion in all cases.

49. How do I treat someone who has ingested mushrooms?
Therapy is primarily supportive including volume resuscitation, seizure control, and treatment of agitation. Identify the mushroom species ingested, if possible, and monitor for delayed onset of symptoms when orellanine, amatoxin, or monomethylhydrazine are ingested. Specific antidote therapy is available for some mushroom toxins.

HALLUCINOGENS

50. What are hallucinogens?
Typically, the term *hallucinogen* refers to agents that are used recreationally for their mind-altering effects. Many substances (including mushrooms and stimulants) can cause hallucinations, perceptions without any basis in reality, or alterations in the perception of reality.

51. List some examples of hallucinogens.
- *N, N*-Diisopropyl-5-methoxytryptamine (Foxy-Methoxy)
- Lysergic acid diethylamide (LSD)
- Marijuana
- Mescaline
- 3,4-Methylenedioxymethamphetamine (MDMA, Ecstasy)
- 1-(1-Phenylcyclohexyl) piperidine (PCP)

52. List the life-threatening effects of hallucinogens.
Common effects are seizures, hyperthermia, metabolic acidosis, hypertension, and dysrhythmias. Rhabdomyolysis can develop subsequently. The effects of hallucinogens are unpredictable and different with each use. Trauma frequently occurs as a result of the disinhibition and aggressiveness caused by hallucinogen abuse.

53. Why would you "lick a toad"?
Hallucinations are produced by bufotenine, the substance in the skin secretions of Bufo *(Bufo vulgaris, Bufo marinus)* toads. Bufotenine and many other natural toxins have been used for years for hallucinogenic effects. Mescaline is the toxin in peyote, a cactus found in the southwestern United States and Mexico. Psilocybin (4-phosphoryloxy-*N, N*-dimethyltryptamine) is found in some species of mushrooms; *N, N*-dimethyltryptamine (DMT) is in many plants and seeds. Natural agents (such as these) and their synthetic derivatives are used for hallucinogenic purposes.

54. What is the treatment for hallucinogen toxicity?
Reassurance, a calm environment, avoidance of further trauma, and good supportive care are important. Administer a benzodiazepine to calm agitated patients or to treat seizures. Consider an antipsychotic for patients experiencing hallucinations and psychosis. Occasionally, physical restraint may also be necessary to protect the patient or staff from harm.

STIMULANTS

55. What are examples of stimulants?
Cocaine, crack cocaine, amphetamine, methamphetamines, ecstasy (MDMA), and caffeine

56. What is the difference between cocaine and amphetamines?
Both drugs are stimulants and both work by increasing release of norepinephrine, epinephrine, dopamine, and serotonin. Cocaine has direct vasoconstrictive effects,

blocks nervous system and cardiac sodium channels, and has a shorter duration of action than amphetamines.

57. How should I screen for cocaine use?

The best way to screen for recent cocaine use is with a urine drug screen. Cocaine is metabolized rapidly, and detection of the parent compound in blood indicates recent use. However, blood tests for cocaine are rarely used. Cocaine undergoes nonenzymatic degradation to benzoylecgonine and ecgonine methyl ester. These metabolites are excreted renally and may be detected in the urine for several days after the initial exposure. Common urine drug screens are positive for degradation products of cocaine.

58. What are *free-base* and *crack* cocaine?

Cocaine usually arrives in the United States as a white powder, cocaine hydrochloride (CHCl). This powder is highly water-soluble and therefore crosses mucous membranes and intestinal mucosa very quickly. Vaporization requires very high temperatures, so the powder is not suitable for smoking. The powder can be dissolved with sodium bicarbonate (baking soda) or ammonia and water. This solution may subsequently be treated with diethyl ether, decanted, and dried to form *freebase*; or it can be boiled, ice added to reduce the temperature, and dried to form *crack* (so called due to the popping sound that occurs during heating). Freebase and crack are resistant to pyrolysis and can be smoked.

59. What is the significance of chest pain after using cocaine?

Pneumothorax or pneumomediastinum may occur after a Valsalva maneuver when cocaine has been smoked. Aortic dissection is rare. Myocardial infarction and acute coronary syndrome have followed intranasal, intravenous, and smoked cocaine, even in young patients with normal coronary arteries. Benzodiazepine is the initial treatment of choice for cocaine-induced chest pain of cardiac etiology.

60. Does concomitant ingestion of ethanol change the effects of cocaine?

Yes. In the presence of ethanol, cocaine is metabolized to cocaethylene, a metabolite that retains the cocaine's vasoconstrictive properties. Cocaine and ethanol cause synergistic depression of ventricular contraction and relaxation. Simultaneous ethanol ingestion and intranasal cocaine increase peak plasma concentration of cocaine by 20%, compared with intranasal cocaine alone. The increased cocaine concentration increases euphoria, and thus concomitant abuse.

61. What is *ice*?

Ice is the smokable form of methamphetamine, named for its appearance of transparent crystals. In contrast to cocaine HCl, this pure base form of methamphetamine HCl evaporates easily at room temperature and is absorbed rapidly from the lungs. Similar to intravenous methamphetamine, it causes an immediate euphoric effect but without the risks of intravenous drug administration. The clinical manifestations of methamphetamine are secondary to heightened catecholamine activity and are the same, regardless of the route of administration. Potential adverse effects include hypertension, dysrhythmias, intracranial hemorrhage, seizures, and hyperthermia.

62. What is *ecstasy* and what is *Eve*?

Adam, ecstasy, E, and XTC are street names for 3,4-methylenedioxymethamphetamine (MDMA). Eve is a street name for MDEA (3,4-methylenedioxyethylamphetamine) and is less commonly used. These are *designer drug* analogs of amphetamines and are illegal. These drugs increase serotonin release and reduce degradation more potently than other amphetamines. Their unique chemical structure results in greater euphoria and less sympathomimetic toxicity. MDMA causes long-term neurotoxic damage in brains of experimental animals. Large overdoses of MDMA or MDEA, both phenylethylamines, can resemble amphetamine toxicity. Hyperthermia (caused by the drug, and hot, crowded conditions at the raves [dances]) and seizures are associated with death. In addition, ecstasy

use has been associated with severe hyponatremia related to increased water intake during raves and drug-induced increased secretion of antidiuretic hormone. Ecstasy and other designer drugs are not detected on routine urine drug screens for amphetamines.

63. How should I treat someone with toxicity from stimulants?

The triple C method:

- **C**alm them
- **C**ool them
- Uncover **C**omplications

Treat agitation and seizures with a benzodiazepine. Large and repeated doses may be required. Treat hyperthermia aggressively by reducing psychomotor agitation with sedation and by adding cooling measures (i.e., evaporation, cooling blanket, and cool intravenous fluids). Stimulant complications include rhabdomyolysis, pyrexia, acidosis, intracranial hemorrhage, pneumomediastinum, abdominal ischemia, and injection-related complications (i.e., abscess, endocarditis, and cellulitis). Evaluate patients for these complications through history, examination, and testing as needed. Cocaine is more likely to cause complications because it directly causes vasoconstriction, in addition to the secondary effects of increased release and decreased uptake of norepinephrine, epinephrine, serotonin, and dopamine.

64. How do I treat stimulant-induced high blood pressure?

High blood pressure from stimulant toxicity is usually short-lived. Most cases can be treated with benzodiazepines. A true hypertensive emergency, although rare, can be treated with benzodiazepines and nitroglycerin. Phentolamine, nitroprusside, and calcium channel blockers are rarely needed and supportive data is limited. Nitroglycerin and other cardiac interventions may be used in patients with ischemic chest pain from vasoconstriction or myocardial infarction. β-blockers, such as propranolol, should be avoided in the cocaine-toxic patient because they allow unbridled alpha agonism, which can result in elevated blood pressure and coronary artery vasoconstriction.

KEY POINTS: COMMON DRUGS OF ABUSE

1. The classic triad of opioid poisoning is CNS depression, respiratory depression, and miosis.
2. In patients with respiratory compromise secondary to opioid intoxication, patient resuscitation takes precedence over administration of opioid antagonists, such as naloxone.
3. Flumazenil is an antidote to benzodiazepine intoxication, and it is a specific antagonist to the GABA A receptor. It has no role in the undifferentiated or mixed overdose.
4. The use of routine toxicologic screens in undifferentiated overdose is generally not helpful due to the false-positive results, limited number of drugs screened, lack of correlation to clinical presentation, and prolonged length of time to obtain the results.
5. Mushrooms of the *Amanita* species are associated with delayed-onset fulminant hepatic failure produced by amatoxin.
6. Simultaneous cocaine and ethanol use depresses myocardial contractility.
7. Ecstasy (MDMA) can cause hyponatremia and pyrexia.
8. Evaluate patients with stimulant-induced agitation for the OTD: pyrexia, acidosis, and rhabdomyolysis.

65. What is the stimulant-induced *OTD*?

OTD is the occult triad of death: acidemia, rhabdomyolysis, and pyrexia. These three occult effects occur often, can be easily missed in the agitated patient, and, if not detected early, can lead to death. With the moderately or severely agitated patient, obtain an arterial or venous blood ph. Also, obtain a creatine kinase and repeat it if the patient continues to be physically agitated. Finally, check the patient's core temperature early and again before disposition.

WEBSITES

http://dawninfo.samhsa.gov
www.aapcc.org

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CHAPTER 74

CARDIOVASCULAR TOXICOLOGY

Jennie A. Buchanan, MD

1. How do different poisons affect heart rate, blood pressure, and QRS duration?
See Table 74-1.

2. What drugs cause cardiovascular toxicity by blocking cardiac sodium channels?
The major clinical manifestations of sodium channel blockade are QRS prolongation and ventricular dysrhythmias.

- **Drugs with primary toxic effects on sodium channel** include quinidine, flecainide, mexiletine, disopyramide, and procainamide.
- **Drugs with sodium channel effects and other serious effects** include tricyclic antidepressants, propranolol, cocaine, diphenhydramine, carbamazepine, lidocaine chloroquine, cyclobenzaprine, and norpropoxyphene (a metabolite of propoxyphene). Patients poisoned with these agents present with other symptoms but should be observed and treated if prolonged QRS duration or arrhythmias develop.

3. What is the antidote for drugs that cause sodium channel blockade?
Sodium bicarbonate 1 to 2 mEq/kg as a bolus is used for the treatment of dysrhythmias or prolongation of the QRS duration, which occurs after the ingestion of any of these agents. If the QRS duration does not narrow after administration of sodium bicarbonate, a second bolus should be given. Hyperventilation should be initiated to induce a serum pH of 7.5 to 7.55. Hypertonic saline can also be administered in a dose of 200 mL of 7.5% solution or 400 mL of a 3% solution. In addition to bicarbonate, patients with cardiovascular toxicity also often require fluids and vasopressors for hypotension, benzodiazepines for seizures, and endotracheal intubation for altered mental status.

4. How do patients with a calcium channel blocker (CCB) overdose present?
CCBs decrease calcium influx into cardiac tissue and vascular smooth muscle. The heart depends on calcium for automaticity, conduction through the atrioventricular node, and contractility. Vascular smooth muscle requires calcium to maintain tone. Patients with CCB overdose present with hypotension (secondary to decreased contractility and decreased vascular tone), bradycardia, and atrioventricular blocks. If hypotension is significant, patients may have altered mental status, organ ischemia, and acidosis. These patients may also be hyperglycemic.

5. What is the treatment for CCB overdose?
Begin treatment by addressing airway, breathing, and circulation (ABCs). Gastric decontamination may proceed after the airway is adequately protected. Hypotension is treated initially with fluid boluses (i.e., 2 L normal saline), and symptomatic bradycardia is treated with atropine or pacing. Inotropic agents, such as dopamine, norepinephrine, or epinephrine sometimes at high doses, are used next. Calcium is usually the next step in treatment for toxicity. The dose is 1 to 2 g of calcium chloride or calcium gluconate intravenously (calcium chloride requires a central line or large intravenous (IV) line because of risk for vein necrosis) and may be repeated every 10 minutes three to four times. Occasionally, an infusion of calcium may be needed. Glucagon (5–10 mg IV push) may be given and if improvement is

TABLE 74-1. CARDIOVASCULAR EFFECTS OF DIFFERENT POISONS

Bradycardia with hypertension

- Centrally acting presynaptic α_2 agonists (clonidine, oxymetazoline, and tetrahydrozoline): Patients progress to bradycardia and hypotension by the time they reach the hospital; the initial hypertension and bradycardia is transient.

Bradycardia with hypotension and narrow-complex QRS

- Centrally acting presynaptic α_2 agonists (clonidine, oxymetazoline, and tetrahydrozoline): Inhibit sympathetic outflow in the central nervous system, resulting in hypotension, bradycardia, pinpoint pupils, and somnolence
- β-blockers (BBs) without sodium channel effects
- Calcium channel blockers (CCBs)
- Cardiac glycosides
- Sedative-hypnotics, opioids, benzodiazepines and barbiturates decrease CNS sympathetic outflow. Hypotension and bradycardia are usually minimal.
- Organophosphates and carbamates by increasing vagal tone

Bradycardia with hypotension and wide-complex QRS

- Lidocaine, tocainide (class 1b antiarrhythmics): Bradycardia with hypotension and wide-complex QRS
- BBs with sodium channel effects (i.e., propranolol, acebutolol, or metoprolol)
- CCBs (severe toxicity causes ventricular escape rhythms)
- Cardiac glycosides (severe toxicity causes ventricular escape rhythms)
- Propafenone and flecainide (class 1c antiarrhythmics that cause sodium channel blockade): Initially, patients bradycardic with wide QRS, due to decreased cardiac conduction, that may degenerate into ventricular tachycardia
- Quinidine, procainamide, and disopyramide (class 1a antiarrhythmics that cause sodium channel blockade, prolonged QRS and QT intervals): Patients may present with bradycardia, due to decreased cardiac conduction that may degenerate into ventricular tachycardia.
- Hyperkalemia from cardiac glycosides, BBs, and potassium-sparing diuretics

Tachycardia with hypertension

- Sympathomimetics (amphetamines, cocaine, ephedrine, pseudoephedrine) by stimulating the sympathetic nervous system
- Anticholinergics (diphenhydramine and atropine): Due to decrease in vagal tone and agitation from delirium

Continued

TABLE 74-1. CARDIOVASCULAR EFFECTS OF DIFFERENT POISONS—cont'd

Tachycardia with hypotension

- Monoamine oxidase inhibitors: Inhibit the breakdown of catecholamines in central nervous system synapses, tachycardia with hypotension and narrow-complex QRS. In overdose, hypertension can also be profound.
- α_1 antagonists (i.e., prazosin, terazosin, doxazosin): Cause vasodilation and reflex tachycardia
- Phenothiazines: Due to α_1-antagonism causing vasodilation and reflex tachycardia
- Diuretics: Tachycardia and hypotension usually mild secondary to dehydration
- Nitrates: Cause vasodilation and reflex tachycardia
- Theophylline and caffeine: Inhibition of adenosine receptors, β-adrenergic stimulation from catecholamine release resulting in tachycardia and hypotension

Tachycardia with hypotension and wide-complex QRS

- Tricyclic antidepressants (amitriptyline, and imipramine), cyclobenzaprine and diphenhydramine: Sodium channel blockade causing widening of the QRS complex. (In severe toxicity, this can lead to hypotension despite tachycardia from anticholinergic effects.)
- Cocaine: Sodium channel effects that, late in the course, override the ability to maintain blood pressure from tachycardia and vasoconstriction

CNS, central nervous system.

noted, a drip at 5 to 10 mg/hr is initiated. Hyperinsulinemia euglycemia (HIE) therapy or high-dose insulin (1 unit/kg bolus, 25 gm dextrose bolus, then 0.5 units/kg/hr with supplemental dextrose 0.5 gm/kg/hr and potassium) is the next line of therapy. Heroic measures, such as Intralipid (1–2 mL/kg of a 20% lipid emulsion followed by an infusion of 0.25 mL/kg/min for 30–60 minutes), extracorporeal membrane oxygenation, intraaortic balloon pump, and cardiopulmonary bypass, may be used in severe refractory cases.

6. How do patients with β-blocker (BB) overdose present?

BBs compete with endogenous catecholamines for receptor sites; this blunts the normal adrenergic response, leading to bradycardia, atrioventricular blocks, and hypotension from decreased contractility. Patients suffering from BB toxicity present similarly to patients with CCB overdose. There can be a few differences, however, depending on which BB is involved. Some BBs, such as propranolol, are lipid soluble. This allows entry into the central nervous system, leading to seizures and altered mental status unrelated to blood pressure. Some BBs (i.e., propranolol, acebutolol, alprenolol, and oxprenolol) antagonize sodium channels, leading to a widened QRS. Sotalol also blocks potassium channels, causing a prolonged QT interval and torsades de pointes. Hypoglycemia may also be evident.

7. Describe the treatment for BB toxicity.

Treatment is similar to that for CCB overdose. Glucagon is utilized for treatment after fluids, vasopressors, and atropine. The dose of glucagon is the same as for CCB overdose. High-dose insulin therapy may be beneficial as well. Calcium has not been well studied for treatment of BB overdose. Seizures unrelated to hypotension should be treated with benzodiazepines; sodium bicarbonate is used for QRS widening. Refractory sympathetic bradycardia should be treated with external cardiac pacing.

8. Describe the manifestations of acute and chronic digoxin poisoning.

- **Acute digoxin toxicity** occurs after accidental or intentional ingestion of a supratherapeutic amount of digoxin-containing products. A dose of more than 1 mg in a child and more than 3 mg in an adult is potentially toxic. Patients with acute digoxin toxicity often develop gastrointestinal symptoms, such as nausea or vomiting. The most common cardiac effects are bradycardia and heart block. After acute digoxin ingestion, blockade of the cellular sodium/potassium exchange pump leads to systemic hyperkalemia. Severe hyperkalemia (serum level >5.5 mEq/L) is associated with a mortality of greater than 90% if untreated.
- **Chronic digoxin toxicity** occurs when there is a change in the dose or clearance of digoxin in a patient who is receiving digoxin therapy. Initiation of treatment with quinidine, amiodarone, spironolactone, or verapamil may change the steady-state clearance of digoxin and result in toxicity. Decreased clearance of digoxin may occur when patients develop renal insufficiency. Symptoms of chronic digoxin toxicity are often subtle and nonspecific, including confusion, anorexia, vomiting, visual changes, and abdominal pain. The patient is often bradycardic with varying degrees of heart block. Patients may develop premature atrial and ventricular contractions, supraventricular tachycardia, ventricular tachycardia, or ventricular fibrillation. In contrast to acute digoxin toxicity, serum potassium is often normal or depressed, unless the patient has hyperkalemia from renal insufficiency.

9. What are the indications for digoxin immune antibody fragments (Fab)?

The most common indications are symptomatic bradycardia, complete heart block, ventricular tachycardia, or ventricular fibrillation. Often, digoxin immune Fab must be administered to critically ill patients without laboratory confirmation of elevated digoxin levels. Fab should be administered to patients who present after an acute ingestion with hyperkalemia or hemodynamically significant dysrhythmias. The indications for Fab therapy in patients with chronic digoxin toxicity are not well defined. Therapy should be considered for patients with hemodynamically significant bradycardia, multifocal ventricular ectopy, and ventricular dysrhythmias. Because serum digoxin levels correlate poorly with symptoms, there is no specific serum digoxin level that is considered an absolute indication for digoxin Fab.

10. How is digoxin Fab dosed?

Digoxin Fab may be dosed in one of several ways, depending on the information available to the clinician:

- If the patient is critically ill, 10 to 20 vials should be given empirically.
- If the amount of digoxin ingested is known, the following formula should be used: Dose ingested (mg) $\times$ 0.48 = number of vials.
- If the steady-state serum level is known, the following formula should be used: Serum digoxin level (ng/mL) $\times$ patient wt (kg)/100 = number of vials. (This normally results in a patient with chronic toxicity receiving one to three vials.)

KEY POINTS: CARDIOVASCULAR TOXICOLOGY

1. For sodium channel blocking agents, use sodium bicarbonate if there is QRS widening and clinical signs of toxicity.
2. There is no single proven successful treatment for CCB and BB overdose. Severe ingestions often require multiple interventions. Start with symptomatic and supportive care first (ABCs), IV fluids, and pressors. Remember atropine, glucagon, calcium, and high-dose insulin.
3. There is no serum digoxin level that is considered an absolute indication for digoxin immune Fab.

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ONE PILL CAN KILL: PEDIATRIC INGESTIONS

Shan Yin, MD, MPH

CHAPTER 75

1. What comprises the pediatric *one pill can kill* list?

This is a list of pharmaceuticals that may be lethal in a toddler at a therapeutic adult dose. In actuality, the literature may not support the fact that one pill can kill. In other words, there may not be reports of single pill ingestions causing fatalities in children. Regardless, any ingestion by a child from this list should be considered to have the potential to produce serious toxicity at low doses.

2. What drugs may be found on the one pill can kill list?

Although not a consensus list, these drugs are often mentioned:

- Lomotil
- Tricyclic antidepressants
- Calcium channel blockers
- β-blockers
- Sulfonylureas
- Clonidine
- Camphor
- Salicylates
- Phenothiazines
- Opioids

3. Describe the epidemiology of pediatric ingestions reported to U.S. poison centers.

Approximately two thirds of ingestions reported to U.S. poison centers are pediatric, with 80% of those occurring in children younger than 6 years of age. The epidemiology of pediatric ingestions is bimodal, with the majority of ingestions in children younger than 6 years and then a second smaller peak in adolescence. Children younger than 6 years are mobile and exploratory, not having yet developed the cognitive capacity to completely understand the potential danger of the ingestion. The increase in adolescence is largely due to self-administration from self-harm or abuse purposes. The vast majority of pediatric ingestions result in minimal or no clinical effects. Approximately 2% of pediatric exposures reported to U.S. poison centers result in moderate or major outcomes or death with approximately 50% of those in the adolescent age group.

4. What are the components of Lomotil and what are their mechanisms of action?

Lomotil is an antidiarrheal agent composed of diphenoxylate (an opioid) and atropine (an anticholinergic).

5. What is the presentation of a Lomotil ingestion?

Classically, Lomotil ingestions were considered to have a two-phase presentation. The first phase consists of an anticholinergic toxidrome followed by an opiate toxidrome. This classic presentation is unusual, and Lomotil ingestions should be considered as a long-acting opiate ingestion, which may include features of atropine toxicity; Lomotil ingestions should be observed for 24 hours.

6. What is the potential lethal dose of a tricyclic antidepressant (TCA)?
The potentially lethal dose is 15 mg/kg. So in a 10-kg child, 150 mg (a one tablet adult dosing option for amitriptyline) is potentially lethal. Doses of less than 5 mg/kg are considered to be safe.

7. What electrocardiogram (ECG) finding in TCA ingestions is helpful in children?
The terminal 40 ms QRS axis has been shown in adults to be a useful marker in identifying TCA overdose. In a retrospective study of 35 children presenting with TCA ingestions, the terminal 40 ms QRS axis was not helpful in predicting TCA ingestions in children. In a study of children and adolescents, increasing QRS duration was associated with serum tricyclic levels, which suggested that QRS duration could be of prognostic value in a similar manner to TCA ingestions in adults.

8. Have deaths been reported in single ingestions of dihydropyridine (e.g., nifedipine) ingestions in children?
Yes. Although ingestions of dihydropyridines are considered to be less serious than ingestions of phenylalkylamine (e.g., verapamil) and benzothiazepines (e.g., diltiazem) due to direct cardiotoxicity, there is a report of a death in a 14-month-old child from a single 10-mg ingestion of nifedipine.

9. What is the pediatric dose of calcium for calcium channel blocker ingestions?
Calcium is considered one of the first-line treatments for calcium channel blocker ingestions and can improve inotropy and hypotension. 0.1 to 0.2 mL/kg of 10% calcium chloride or 0.3 to 0.6 mL/kg of 10% calcium gluconate can be bolused and repeated every 10 to 20 minutes up to three to four doses. However, in severely poisoned patients, the beneficial effect of calcium is often negligible or short-lived. Furthermore, calcium chloride can be sclerosing to veins, an issue when dealing with the smaller-caliber veins in children. In severely poisoned patients, it is prudent to begin other treatments such as vasopressors and inotropes simultaneously with calcium administration.

10. What new therapy has shown some promising results in treatment of calcium channel blockers?
Hyperinsulinemia/euglycemia has shown to be effective in animal models of calcium channel blocker toxicity. Human case reports support improved hemodynamics with insulin/dextrose administration, with published reports of its use in a 5-month-old child and two teenagers. The typical suggested starting dose is 1 U/kg insulin bolus followed by a continuous infusion of 0.5 to 1 U/kg/hr titrated to effect. Dextrose should be administered to maintain euglycemia.

11. What is a potential side effect of β-blocker ingestions other than cardiovascular toxicity in children?
There have been reports of severe hypoglycemia associated with propranolol ingestions. A prospective series of 208 children, however, suggested that exposure to one or two β-blockers pills are very unlikely to result in any toxicity.

12. For how long should a sulfonylurea ingestion in a child be observed?
A child should be observed for 24 hours. There are case reports of hypoglycemia occurring up to 21 hours after the initial ingestion. Ingestions of single tablets of glipizide have caused hypoglycemia in children, as well as in naive adults.

13. How often should blood sugars be monitored?
Initially, blood sugars should be monitored hourly.

14. After a sulfonylurea ingestion in a child, should prophylactic dextrose or maintenance fluids with dextrose be given?
No. Dextrose may potentiate the insulin release caused by sulfonylureas. In many reports of delayed hypoglycemia, the child received prophylactic dextrose. The child should be allowed

to eat a normal diet free of concentrated sweets. If the child's blood sugar drops, then dextrose should be administered to bring their blood sugar up.

15. What is the *rule of 50*?

The *rule of 50* is a mnemonic for calculating a dextrose dose for pediatric resuscitation. When the concentration of the dextrose solution times the dose in mL/kg equals 50, 0.5 g/kg bolus of dextrose is provided. For example, a 10% dextrose solution at 5 mL/kg or a 25% dextrose solution at 2 mL/kg, both provide 0.5 g/kg.

16. What is considered the antidote of sulfonylurea ingestions?

Octreotide. Glucose (and sulfonylureas) opens voltage-gated calcium channels, which triggers insulin secretion via intracellular signaling. Octreotide independently closes these channels, resulting in decreased insulin secretion. It is important to note that octreotide does not raise the serum blood sugar, but only stops further insulin secretion; dextrose is still needed to normalize blood sugar when giving octreotide.

17. How is octreotide dosed in pediatric sulfonylurea ingestions?

The appropriate dose, dosing frequency, and side effect profile in pediatric sulfonylurea ingestions has not been rigorously studied. Adults typically received 50 to 100 μg subcutaneously every 8 to 12 hours. A suggested pediatric dose is 1 μg/kg subcutaneously with an initial dosing interval of every 6 hours.

18. What are the cardiovascular effects that may be seen with clonidine ingestions?

Bradycardia and hypotension are most commonly reported. However, hypertension has also been reported in children. This likely occurs from activation of peripheral α-2 receptors. The hypertension tends to be transient and does not usually require specific treatment. Other commonly reported effects are central nervous system (CNS) depression, respiratory depression, hypothermia, and miosis. No specific antidote exists; treatment is generally focused on general respiratory and hemodynamic support.

19. Can naloxone be used in pediatric clonidine ingestions?

The experience with naloxone in pediatric clonidine ingestions largely parallels the experience with adult ingestions—it only works a fraction of the time. In a review of pediatric ingestions receiving variable doses, naloxone was observed to have a positive response in 16% of patients. Although clonidine ingestions often present similar to opiate ingestions, naloxone's effect is not completely understood.

20. How much clonidine do the 0.1 mg/day, 0.2 mg/day, and 0.3 mg/day patches contain?

2.5 mg, 5 mg, and 7.5 mg respectively. Given that pediatric ingestions as low as 0.1 mg have produced symptoms, ingestions of transdermal patches are particularly worrisome.

21. What are some common over-the-counter products that contain pharmaceuticals with similar mechanisms of action to clonidine?

Oxymetazoline, naphazoline, xylometazoline, and tetrahydrozoline are imidazolines with the same mechanism of action as clonidine. They are found in ophthalmic solutions and nasal decongestants. Ingestion of these products can cause significant effects. As little as 2.5 to 5 mL of a 0.05% tetrahydrozoline solution caused drowsiness, bradycardia, respiratory depression, cool extremities, and miotic pupils in a 1-year-old girl. Onset of symptoms is typically rapid occurring in 15 to 30 minutes.

22. How do ingestions of camphor present?

Ingestions initially cause gastrointestinal symptoms such as burning of the mouth and throat and vomiting. Severe toxicity manifests as neurologic symptoms such as seizures, hyperreflexia, myoclonic jerks, and coma. The onset of symptoms tends to be rapid, occurring 5 to 90 minutes following the exposure. There is no specific antidote and treatment is primarily symptomatic and

supportive. A recent case series suggested that camphor should be considered as a cause of undifferentiated seizures in children from communities of widespread use.

23. Where is camphor typically found?

Campho-Phenique, Vick's Vaposteam, Vick's VapoRub, Tiger Balm, Anbesol Cold Sore Therapy Ointment, BenGay UltraStrength, and many other over-the-counter topical creams. The Food and Drug Administration (FDA) has ruled that no product sold in the United States can contain greater than 11% camphor. Foreign products, however, may contain much higher percentages of camphor.

24. What is the potentially toxic dose of camphor?

Five hundred mg can cause serious toxicity in a child. In an 11% solution, this would equal approximately 4.6 mL.

25. At what dose of salicylate do children begin to manifest toxicity in an acute ingestion?

Both children and adults manifest acute toxicity at approximately 150 mg/kg. Serious toxicity is likely to occur at 300 mg/kg.

26. How does the potency of methyl salicylate compare to salicylate?

One mg of methyl salicylate is roughly as potent as 1.4 mg of salicylate. Methyl salicylate is found in oil of wintergreen, many topical over-the-counter creams, and many Asian herbal remedies.

27. Five mL of 100% methyl salicylate is equal to approximately how much aspirin (or acetylsalicylate)?

Five mL (or 1 teaspoon) of 100% methyl salicylate is equal to approximately 7,000 mg of salicylate or almost 22 regular strength adult aspirin tablets. In a 10-kg child, this would be 700 mg/kg, easily a life-threatening ingestion. Oil of wintergreen often contains 98% to 100% methyl salicylate and ingestions of 4 mL have caused death in children.

28. What phenothiazine is believed to be the most dangerous in pediatric accidental ingestions?

Chlorpromazine (Thorazine) is responsible for nearly every serious documented pediatric ingestion. As little as 280 mg resulted in the fatality of a 2-year-old child. An available high-concentration chlorpromazine elixir contains 100 mg/mL. Phenothiazine toxicity manifests as CNS depression, hypotension, and anticholinergic symptoms. Fatal pediatric cases of neuroleptic malignant syndrome have been reported after acute ingestions of phenothiazines. Serious morbidity or mortality has not been reported from isolated ingestions of small doses of the antiemetic phenothiazines, promethazine (Phenergan), and prochlorperazine (Compazine).

29. What is the pathophysiology of chloroquine and hydroxychloroquine ingestions?

These drugs are believed to exhibit quinidine-like effects and inhibit cardiac sodium and potassium channels and may manifest with QRS prolongation, atrioventricular block, ST- and T-wave depression, and QTc prolongation. Chloroquine is generally not found in U.S. households because of its primary use as malarial prophylaxis or treatment. However, hydroxychloroquine is increasingly being used as an anti-inflammatory agent. Although hydroxychloroquine is considered safer than chloroquine, both have the potential to cause serious toxicity including cardiotoxicity, respiratory depression, CNS depression, and seizures.

30. What other drug besides standard therapy has been used to treat chloroquine poisoning?

Other than sodium bicarbonate for QRS widening, diazepam (2 mg/kg intravenously over 30 min) may be tried. Although its mechanism of action is unclear and randomized trials have failed to demonstrate a clear benefit, diazepam may be considered in severe poisonings.

31. What newer opioid can result in significant toxicity with ingestions of one pill?

Buprenorphine is a new opioid that is often marketed as Suboxone, contains naloxone, and is most often used to treat opiate addiction. It is typically dosed as a dissolving sublingual tablet, increasing the potential for toxicity in children. Ingestions of one pill have been associated with significant respiratory depression, requiring children be observed for 24 hours after exposure. Naloxone, used for this or any other opioid poisoning, is dosed in children at 0.01 mg/kg intravenously for respiratory depression and 0.1 mg/kg intravenously (up to 2 mg) for apnea.

KEY POINTS: PEDIATRIC INGESTIONS

1. Pediatric patients can easily reach toxic doses of common medications from small amounts of ingestions.
2. Risk of toxicity and antidote dosages are weight dependent.
3. Because specific ingested amounts are often difficult to determine in small children, prolonged observation is often required to rule out a potentially toxic ingestion.
4. Although range of toxicity may vary, treatment for children usually mirrors that for adults with similar ingestions.

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PELVIC INFLAMMATORY DISEASE

Leslie L. Armstrong, MD, and Susan Brion, MD, MS

1. What is pelvic inflammatory disease (PID)?

PID represents a spectrum of disorders that are usually secondary to one or more sexually transmitted diseases involving the upper genital tract of women. PID can include any of the following: mucopurulent cervicitis, endometritis, salpingitis, tubo-ovarian abscess, or pelvic peritonitis.

2. What are the risk factors for PID?

Although there are no definitive risk factors for PID, there are several likely contributors. Age younger than 25 is seen in 75% of cases and confers 10 times the risk for the development of PID for several reasons. Females 15 to 24 have increased number of sexual partners, have a cervical barrier more easily breached by pathogens, often have less frequent use of barrier contraceptives, and tend to seek health care later. Other risk factors include earlier age at first intercourse, instrumentation including induced abortion and intrauterine device insertion (not the IUD itself), and the period immediately following menses.

3. What are the signs and symptoms of PID?

There are no specific signs and symptoms that are diagnostic for PID. Studies have shown that clinical diagnosis of PID may be no better than chance when compared to biopsy-proven diagnosis. Patients present with complaints of lower abdominal pain with or without dyspareunia, abnormal bleeding, or abnormal vaginal discharge. On examination, patients have lower abdominal tenderness, cervical motion tenderness, and bilateral adnexal tenderness.

4. What are the microbiologic causes?

Most cases of PID are polymicrobial. Typically, the first time a patient is diagnosed with PID it is from a sexually transmitted disease, most commonly *Neisseria gonorrhea* or *Chlamydia trachomatis*. Other microbials that are frequently found include pelvic anaerobes, endogenous pelvic flora, gram-negative rods, group B strep, *Mycoplasma hominis, Staphylococcus aureus*, *Gardnerella vaginalis*, and *Haemophilus influenzae*. About 15% of PID occurs in relation to recent pelvic instrumentation (i.e., endometrial biopsy, dilation and curettage, hysterectomy, and IUD placement).

5. What are the diagnostic criteria for PID?

Delay in diagnosis and treatment contributes to inflammatory sequelae in the upper reproductive tract. Therefore a low threshold for the diagnosis of PID should be maintained. The diagnosis should be considered in any female patient with a chief complaint of lower abdominal or pelvic pain, fever, vaginal discharge, abnormal bleeding, dyspareunia, or dysmenorrhea. Diagnostic criteria for PID present on pelvic examination include cervical motion tenderness or uterine tenderness or adnexal tenderness. The presence of signs of lower genital tract inflammation in addition to one of these criteria increases the specificity of the diagnosis and empirical treatment is indicated.

Additional criteria used to support a diagnosis of PID include the following:

- Oral temperature >38.3°C
- Abnormal cervical or vaginal mucopurulent discharge

- Presence of increased white blood cells on microscopy of vaginal secretions
- Elevated erythrocyte sedimentation rate
- Elevated C-reactive protein
- Laboratory documentation of cervical infection with *N. gonorrhoeae or C. trachomatis*

The most specific criteria for diagnosing PID include the following:

- Endometrial biopsy suggesting endometritis
- Transvaginal sonography or magnetic resonance imaging (MRI) showing thickened, fluid-filled tubes with or without free fluid or Doppler studies showing tubal hyperemia
- Laparoscopic abnormalities consistent with PID

6. Which diagnostic tests should be performed in patients suspected of PID?

- A pregnancy test is necessary to rule out complications with pregnancy.
- Ultrasound for all positive pregnancy tests to exclude ectopic pregnancy, patients being considered for admission, or for those with a possible tubo-ovarian abscess.
- A catheter-obtained urinalysis may reveal a urinary tract infection.
- Cultures for *C. trachomatis* and *N. gonorrhoeae* should be obtained.

Short of laparoscopy, there is no reliable test to exclude PID. Although abnormal laboratory results may provide supportive evidence, all laboratory studies may be normal in a patient with PID.

7. What other diseases should be considered?

The differential diagnosis for this nonspecific presentation includes cervicitis, ectopic pregnancy, endometriosis, ovarian cyst, ovarian torsion, spontaneous abortion, septic abortion, cholecystitis, gastroenteritis, appendicitis, diverticulitis, pyelonephritis, and renal colic. In a few patients, the cause of pelvic pain is never diagnosed, despite extensive testing.

8. What are the consequences of PID?

PID is associated with a number of serious clinical sequelae. Tubo-ovarian abscess is reported in up to one third of patients hospitalized for PID. The incidence of infertility drastically increases with each episode of PID and is thought to be primarily due to tubal occlusion from scarring and adhesions within tubal lumens. Ectopic pregnancy risk also increases by 6- to 10-fold after an episode of PID, and the rate of a potentially fatal ectopic is 12% to 15% higher in women who have had PID. Chronic pelvic pain may occur in up to one in three women with PID. Fitz-Hugh-Curtis syndrome (perihepatitis) and pelvic abscess may be seen in up to 5% to 10% of patients.

9. Who should be hospitalized?

In women with PID of mild to moderate clinical severity, outpatient therapy is reasonable. The decision to hospitalize should be based on the severity of the patient's presentation at the discretion of the health care provider. However, some criteria for hospitalization are suggested by the Centers for Disease Control and Prevention (CDC) and include the following:

- Surgical emergencies (i.e., appendicitis) cannot be excluded
- Pregnant patients
- Patients who do not respond clinically to a trial of oral antimicrobial therapy
- Patients who are unable to follow or tolerate an outpatient oral regimen
- Patients with severe illness, nausea and vomiting, or high fever
- Patients with tubo-ovarian abscess

10. Summarize the recommended antibiotic regimens for PID treatment.

See Table 76-1.

11. Are there alternative outpatient treatment regimens for PID?

The CDC no longer recommends fluoroquinolones for treatment of gonococcal infections and associated conditions such as PID. Comparative evidence suggests that azithromycin is more effective than doxycycline when given with ceftriaxone for mild PID. In a trial of

TABLE 76–1. OUTPATIENT TREATMENT OF PELVIC INFLAMMATORY DISEASE

Recommended Oral Regimen

Ceftriaxone 250 mg IM in a single dose
Plus
Doxycycline 100 mg PO bid for 14 days
with or without
Metronidazole, 500 mg PO bid for 14 days
OR
Cefoxitin, 2 g IM, plus probenecid, 1 g PO in a single dose concurrently once; *or* other parenteral third-generation cephalosporin (e.g., ceftizoxime or cefotaxime)
Plus
Doxycycline, 100 mg PO bid for 14 days
with or without Metronidazole 500 mg PO bid for 14 days

Parenteral Regimen A

Cefotetan, 2 g IV every 12 hour; *or* cefoxitin, 2 g IV every 6 hours
Plus
Doxycycline, 100 mg IV or PO every 12 hours
Note: Because of pain associated with infusion, doxycycline should be given orally when possible, even when patient is hospitalized. Oral and intravenous administration of doxycycline provide similar bioavailability. If intravenous administration is necessary, lidocaine or another short-acting local anesthetic, heparin, or steroids with a steel needle or extension of the infusion time may reduce infusion complications. Parenteral therapy may be discontinued 24 hours after a patient improves clinically, and oral therapy with doxycycline (100 mg bid) should continue for 14 days. When tubo-ovarian abscess is present, clindamycin or metronidazole may be used with doxycycline for continued therapy rather than doxycycline alone because it provides more effective anaerobic coverage.

Parenteral Regimen B

Clindamycin, 900 mg IV every 8 hours
Plus
Gentamicin, loading dose IV or IM (2 mg/kg body weight), followed by a maintenance dose (1.5 mg/kg) every 8 hours. Single daily dosing may be substituted.
Note: Although use of one daily dose of gentamicin has not been evaluated for the treatment of PID, it is efficacious in analogous situations. Parenteral therapy may be discontinued 24 hours after a patient improves clinically, and continuing oral therapy should consist of doxycycline, 100 mg PO bid, or clindamycin, 450 mg PO qid, to complete 14 days of therapy. When tubo-ovarian abscess is present, clindamycin may be used for continued therapy rather than doxycycline because clindamycin provides more effective anaerobic coverage.

bid, twice a day; IM; intramuscularly; IV, intravenously; PID, pelvic inflammatory disease; PO, per os; qid, four times a day.
Adapted from Centers for Disease Control and Prevention: Sexually transmitted diseases treatment guidelines. *MMWR Morb Mortal Wkly Rep* 56 (14):332, 2007.

women with mild PID treated with ceftriaxone 250 mg intramuscularly and randomized to azithromycin 1 g orally once weekly for 2 weeks, versus doxycycline 200 mg per day for 14 days, results demonstrated a 90.3% versus 72.4% cure rate for azithromycin versus doxycycline.

12. Does the presence of an intrauterine pregnancy effectively rule out PID?

A common misconception is that PID cannot occur in a pregnant woman. Although rare, it most commonly occurs in the first trimester in primigravida patients. Infection can take place concurrently with fertilization or throughout the first trimester, after which the uterine cavity is obliterated by the pregnancy.

13. Does a history of tubal ligation preclude the diagnosis of PID?

Bilateral tubal ligation (BTL) does not preclude PID. Histologically, inflammation occurs both proximal and distal to the occlusion site. Tubo-ovarian abscesses have been reported up to 20 years following ligation.

14. What is the appropriate follow-up for patients with PID?

Patients treated as outpatients need close follow-up. Response to treatment should be assessed within 48 to 72 hours. For reliable patients, a phone follow-up may suffice. For all patients, a test of cure by repeat examination and cervical cultures is recommended 2 to 4 weeks after the initial intervention.

15. Summarize the principles of management of acute PID.

- Rule out pregnancy.
- Maintain a low level of suspicion for PID as the consequences of undiagnosed PID include infertility and chronic pelvic pain.
- Treat early with broad spectrum antibiotics if PID is suspected.
- Recommend all patients with PID be tested for other sexually transmitted diseases, particularly syphilis, Hepatitis B and C, and HIV.
- Inform the patient that her partner(s) also need to be treated to prevent reinfection.

KEY POINTS: PELVIC INFLAMMATORY DISEASE

1. Maintain a high suspicion for PID in any patient younger than age 25 with pelvic pain.
2. Although early diagnosis and treatment can prevent significant morbidity, there are no historical, physical, or laboratory findings that can conclusively diagnose PID.
3. Most cases of PID are polymicrobial, requiring broad spectrum antibiotics for treatment.
4. Abnormal uterine bleeding may be the only sign of PID.
5. Neither pregnancy nor tubal ligation excludes a diagnosis of PID.

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CHAPTER 77

SEXUAL ASSAULT

Katherine M. Bakes, MD, and Bernadine L. Mellinger, BSN, RN, SANE-A, CFN

1. What is the definition of sexual assault?

Definitions may vary from state to state, but sexual assault generally refers to any deliberate sexual contact to areas of the genitalia, anus, or mouth, or manual penetration of the victim's body by way of force, threatened physical abuse, or abuse of authority or when the victim does not or cannot consent. Individuals who have an impaired mental function due to alcohol, drugs, sleep, or unconsciousness are unable to give consent by definition. The more traditional term *rape* is defined as forced vaginal, anal, or oral penetration. This definition of sexual assault includes attempted rape of male, female, heterosexual, and homosexual victims.

2. How common is sexual assault?

Sexual assault is one of the most under-reported crimes; research indicates that only 40% of sexual assaults are reported to law enforcement. In 2007, 248,300 men and women became victims of rape, attempted rape, or sexual assault and reported to law enforcement. That means every 2 minutes, somewhere in the United States, someone is sexually assaulted. One in 6 women and one in 33 men will report a completed or attempted rape during their lifetime. Women ages 18 to 26 are four times more likely to be sexually assaulted. Of women who report being raped, 54% were younger than the age of 18 years when they were victimized. Rape in the United States has been termed a *tragedy of youth*. Women who are sexually assaulted as children and adolescents are at greater risk of being sexually assaulted as an adult. Although most victims of sexual assault are women, men can be assaulted by other men, and occasionally women perpetrate sexual assaults against other women or men. The vast majority of sexual assaults (72%) are perpetrated by someone whom the victim knows, with only 6% of those perpetrators spending time in jail.

3. What role does a medical provider have in cases of alleged rape?

Rape is a legal term, and usually law enforcement is requesting for evidence to be gathered. The ED is the most common place for a sexual assault victim to present for acute medical care *and* forensic evidence gathering. 32% of women older than 18 years who are sexually assaulted report being injured in the assault and 36% seek some type of medical treatment, which includes care for traumatic injuries. Always remember that the physician's primary responsibility is to provide for the patient's physical and psychological well-being first; then, if the patient consents, to provide police with corroborative forensic evidence. Victims should be encouraged to undergo an evidentiary examination as soon as possible because critical evidence may be lost if this examination is delayed. The victim may later choose not to proceed through the criminal justice system because collection of forensic evidence does not commit him or her to seek prosecution.

Many hospitals now have Sexual Assault Nurse Examiners (SANE) who have been specially trained to care for these victims of violence. These nurses are educated and trained to complete a comprehensive and compassionate medical-legal examination. If a SANE is not available, a comprehensive ED sexual assault protocol that addresses medical care and the evidentiary collection is necessary for optimal patient care.

4. What information should be elicited in the patient history?

- Information regarding the patient's general health, medications, and allergies should be obtained along with a complete gynecologic history, including birth control usage, date and time of last consensual intercourse, last menstrual period, and history of recent gynecologic symptoms before the assault. Questions as to body surface injuries prior to the assault should also be documented.
- A directed history of the assault includes the date, time, and location of the assault; information concerning the relation the victim had with the assailant; and the type and details of the sexual acts, including type of force or threats used. The history must be obtained in a private setting; law enforcement personnel should not be present.

5. What should comprise the physical examination?

Rape is a crime of violence and control, not a crime of passion. The purpose of the physical examination is to detect injuries requiring treatment and to record and gather forensic evidence for prosecution. A complete head-to-toe medical examination should be done, regardless of whether law enforcement has requested a forensic examination and regardless of whether the patient has consented to a forensic examination. *General body trauma* occurs more frequently than genital trauma. Injuries may include abrasions and bruises on the arms, head and neck, signs of restraint (such as rope burns or mouth injuries), broken teeth, fractured nose or jaw from being punched or slapped, muscle soreness, or stiffness from restraint in positions allowing sexual penetration. These injuries should be documented (i.e., size, color, and shape) on a body diagram with photographic documentation if possible.

The gynecologic examination should include a thorough search for contusions, abrasions, lacerations, bleeding, or tenderness. Most semen or saliva will fluoresce under a Wood's lamp. Toluidine blue dye does not reveal injury that is not visible to the naked eye but highlights genital injuries such as tears or abrasions. A colposcopic examination may help to identify outer genital and cervical injuries, and if possible, photographs of the genital area should be taken. A careful rectal examination should be done in cases of rectal penetration, and if blood is present, anoscopy or sigmoidoscopy should be done to identify internal injuries.

6. What evidence is gathered as part of the forensic examination?

The forensic evidence may be divided into four categories: control samples from the victim, evidence that might identify the assailant, evidence or proof of recent sexual contact, and evidence or proof of force or coercion (Table 77-1).

7. What laboratory studies are indicated?

A urine or serum pregnancy test will rule out a preexisting pregnancy. If pregnant, the patient should be reassured that this pregnancy was not the result of the assault. The routine collection of gonorrhea or chlamydial cultures is debatable. From a medical-legal perspective, positive cultures indicating preexisting sexually transmitted diseases (STDs) have been used by the perpetrator's defense attorney as evidence of the victim's sexual promiscuity. A preexisting infection is present in approximately 5% of assault victims, the same rate as in the general population. It is reasonable to only test those patients with signs or symptoms of infection and presumptively treat all victims for possible exposure. If the victim does not wish to receive prophylactic antibiotics in the ED, Chlamydia and gonorrhea cultures should be repeated in 2 weeks.

8. What about blood alcohol levels and tests for drug use?

In general, routine drug screens and routine alcohol levels are not recommended; proof of intoxication or drug use may or may not be used against the victim in a court of law, however, collecting blood or urine may help to prove the victim was too intoxicated to consent. If tests are medically indicated and will influence your treatment (such as a patient with an unexplained altered mental status, or unexplained tachycardia), then laboratory testing may be indicated.

TABLE 77-1. FORENSIC EVIDENCE KIT CONTENTS

Control samples from victim

- Head hair samples
- Saliva sample
- Pubic hair samples

Samples to identify assailant

- Skin swabbing for assailant's saliva or sperm
- Fingernail scrapings or clipping (from victim)
- Pubic hair combing
- Trace evidence (such as stray hair, bits of clothing, foreign matter)

Evidence for proof of recent sexual contact:

- Oral, vaginal, or anal swabs for semen
- Skin swabbing for saliva or semen
- Any tampons, vaginal pads, or condoms left in vaginal vault if present

Evidence for proof of force or coercion:

- Documentation and photographs of injuries found on examination
- Fingernail scrapings or clippings
- Urine or blood for toxicologic testing (if drug-facilitated sexual assault is suspected)
- All clothing

Adapted from Patel M, Minshall L: Management of sexual assault. *Emerg Med Clin North Am* 19:817–831, 2001; and Feldhaus KM: Female and male sexual assault: In Tintinalli JE, Kellen GB, Stapcznski JS, editors: *Emergency medicine: a comprehensive study guide*, ed 6, New York, 2004, McGraw-Hill, pp 1851–1854.

9. What historical features might indicate a drug-facilitated rape?

A history of a period of amnesia or a history of being out drinking and then suddenly feeling *very intoxicated* should raise concerns about a drug-facilitated sexual assault (DFSA). Sometimes the patient simply relates a history of waking up without clothes on, unsure of what occurred, with genital or pelvic soreness. In these situations, urine and blood should be obtained for drug testing. This testing can be collected in the ED and handed directly to law enforcement to preserve chain of evidence, and victims should be informed that any previous volitional, recreational drug use (such as cocaine or marijuana) may also be revealed in the toxicologic screening. The samples should be refrigerated after receipt by law enforcement to preserve the detection of drugs of abuse. Conviction of a DFSA increases the legal penalties significantly.

10. What are the most common STDs that may be contracted as a result of a sexual assault?

Sexual assault victims are at risk of contracting chlamydial infection, gonorrhea, bacterial vaginosis, hepatitis B, and HIV. The risks of contracting a new chlamydial, gonorrheal, or bacterial vaginosis infection as the result of sexual assault are hard to estimate; risk varies according to geographical area and type of assault. In general, the risk of contracting chlamydial infection or gonorrhea is 4% to 17%, and the risk of contracting bacterial vaginosis is slightly higher. Hepatitis B is efficiently transmitted via sexual contact.

11. Is empirical antibiotic treatment of sexual assault victims indicated? How about vaccinations?

Because of historically poor follow-up rates by sexual assault victims, along with the significant risk of contracting a new STD, prophylaxis should be offered to all victims. Effective regimens include azithromycin, 1 gm orally in a single dose, or doxycycline, 100 mg orally twice a day for 7 to 10 days for chlamydial prophylaxis; ceftriaxone, 125 mg intramuscularly in a single dose or cefixime 400 mg orally in a single dose for gonorrhea coverage; and a single 2 g oral metronidazole dose to treat *Trichomonas* and bacterial vaginosis. The Centers for Disease Control and Prevention (CDC) also recommends that pregnant patients should receive ceftriaxone 125 mg intramuscularly (IM) in a single dose (avoid quinolones and tetracyclines) for gonorrhea coverage, as well as erythromycin base 500 mg four times a day (qid) for 7 days or amoxicillin 500 mg three times a day (tid) for 7 days to cover *Chlamydia.* Contracting bacterial vaginosis during pregnancy carries a risk of premature rupture of membranes, preterm labor, and chorioamnionitis; pregnant women should be encouraged to follow up with gynecology and receive treatment if they develop bacterial vaginosis.

The CDC also recommends that hepatitis B vaccine be administered at the time of the initial examination if victims have not been previously vaccinated. Follow-up doses of vaccine should be administered 1 to 2 and 4 to 6 months after the first dose.

12. What is the risk of pregnancy after sexual assault?

Although the risk of pregnancy after an isolated sexual encounter during nonfertile periods of the menstrual cycle is thought to be less than 1%, it is significantly higher at midcycle. Approximately 5% of all sexual assault victims become pregnant as a result of the assault. The presence of a preexisting pregnancy must be identified in the ED.

13. What are the current options for pregnancy prophylaxis?

When a preexisting pregnancy has been ruled out, postcoital contraceptives can be used to prevent pregnancy by inhibiting or disrupting ovulation or inhibiting fertilization or implantation. Emergency contraception is not effective once implantation has occurred, and it will not disrupt an existing pregnancy. These products may be taken up to 5 days after sexual contact, but because their effectiveness decreases over time, ideally they should be taken within 72 hours. There are two Food and Drug Administration (FDA)-approved oral products for postcoital emergency contraception. Preven is a combination of ethinyl estradiol and levonorgestrel. Plan B contains only levonorgestrel. Common side effects include nausea, vomiting, and vaginal spotting. The latest research shows that taking both tablets of Plan B at one time (total of 150 μg levonorgestrel) is as effective as separating the two tablets by 12 hours. The failure rate with Plan B is less than 2%. If a dedicated emergency contraceptive product is not available, a levonorgestrel-containing oral contraceptive pill, such as Ovral, may be used, dosed as two tablets of Ovral now, two in 12 hours.

14. What are special characteristics of the male sexual assault victim?

The male sexual assault victim should be treated similarly to a female victim. Special attention should be paid to the mouth, genitalia, anus, and rectum. Men represent approximately 5% of reported sexual assault victims.

15. Discuss the special characteristics of pediatric sexual assault.

In pediatric sexual assault, the assailant is often known to the victim, and there is often a history of repetitive assaults. In addition to documenting signs of acute trauma, the physician should look for signs of recurrent abuse, such as healed hymenal tears, a large vaginal opening, vaginal discharge, or relaxed rectal sphincter tone. The gynecologic examination should take into account the nature of the assault and the age of the child, keeping in mind the exquisite sensitivity of the prepubertal vaginal introitus and hymen. In the evaluation of a small child in whom a speculum examination is indicated, a nasal speculum may be used in place of a vaginal speculum. If possible, this examination is best performed in conjunction with physicians who specialize in child abuse. Sometimes the vaginal or rectal examination

must be done under general anesthesia because of the emotional state of the child. The child should be protected from further abuse by admission to the hospital or by immediate referral to the appropriate social service agency.

16. Should pediatric patients be given prophylactic antibiotics?

Prophylactic antibiotics are not generally indicated when sexual abuse of children is suspected. The baseline infection rate in children is significantly lower than in adults, and the presence of an STD in a child is strong evidence that abuse has occurred. It is important to document the presence of the infection before treatment. In the child, chlamydial and gonorrhea cultures should be obtained from the urine or vagina instead of the cervix.

17. State the important aspects of follow-up care for any victim of sexual assault.

Follow-up medical care should ensure that any physical injuries have healed properly (follow up photographs may be taken), adequate pregnancy prophylaxis has been administered, STDs have been treated properly, and the victim has accessed supportive counseling. Provision of written aftercare instructions and information on community resources is essential.

18. What types of emotional trauma might sexual assault victims experience?

The development of a posttraumatic stress disorder, manifested by sleep disturbances, feelings of guilt, memory impairment, and detachment from the world and others may occur in the days to weeks following the assault. Long-term psychological sequelae in the form of rape trauma syndrome may also occur. Many communities have rape crisis centers with social workers and volunteers who are trained to provide counseling for sexual assault survivors. Sexual assault response teams have been organized in other areas to provide a coordinated approach to the sexual assault victim, including emotional support after the event. Physicians should be aware of the availability of such services so that they can recommend them to their patients.

19. My patient is terrified of contracting HIV after her sexual assault. What do I do now?

Offer nonoccupational postexposure prophylaxis (nPEP).

20. What is nPEP?

nPEP refers to the provision of postexposure antiretroviral therapies for individuals who are exposed to potentially infected blood or bodily fluids from sexual contact, from injection drug use, or in other nonoccupational settings (i.e., non-health care, sanitation, public safety, or laboratory employment settings).

21. What is the risk of acquiring HIV after a sexual assault?

The estimated risk is dependent on the HIV status of the assailant, the type of sexual contact, and the amount of mucosal trauma involved. The HIV status of the source should be considered—is the assailant known to be HIV positive? Is the assailant known to be from a group with a high prevalence rate of HIV (i.e., injection drug users, commercial sex workers, or homosexual or bisexual men)? Studies in prison populations reveal that HIV infection rates are higher in male sexual assailants than in the general male population (1% vs. 0.3%). In most cases of sexual assault, the HIV status of the assailant will not be known. Genital trauma, bleeding, and inflammation associated with sexual assault increase the risk of HIV transmission. In general, receptive anal intercourse carries a risk of seroconversion of 50 per 10,000 exposures. In comparison, a percutaneous needle stick from an infected source carries a 30 per 10,000 risk of contracting HIV. The risk of contracting HIV following receptive penile-vaginal intercourse is 10 per 10,000.

22. How exactly do I provide nPEP for my patient?

Baseline HIV testing should be performed on the victim, preferably with an FDA-approved rapid-test kit (results available within 1 hour). nPEP should begin within 72 hours of exposure, the sooner the better. If more than 72 hours have lapsed since the assault, the risks of

antiretroviral therapies may outweigh the benefit. No evidence indicates that any specific antiretroviral medication or combination of medications is optimal for use as nPEP. Based on the degree of experience with certain agents, there are preferred regimens, including nonnucleoside reverse transcriptase inhibitor (NNRTI)-based therapies and protease inhibitor (PI)-based therapies.

- Preferred NNRTI regimens include efavirenz (Sustiva) *plus* lamivudine (Epivir, 3TC) or emtricitabine *plus* zidovudine or tenofovir.
- PI regimens include lopinavir/ritonavir (coformulated in one tablet as Kaletra) *plus* zidovudine (AZT, Retrovir) *plus* either lamivudine or emtricitabine.

Efavirenz should be avoided in pregnant women and women of child-bearing potential. Regardless of the regimen chosen, the exposed person should be counseled about the potential associated side effects and adverse events that require immediate medical attention. The use of medications to treat symptoms (e.g., antiemetics or antimotility agents) might improve medication compliance. A 3- to 5-day starter pack is recommended with a follow-up visit scheduled to review the results of HIV testing (if a rapid test was not used), review baseline laboratory data, discuss medication side effects, and change therapies if needed. A full 28-day course of medications should be provided at that time.

KEY POINTS: CARE OF THE SEXUAL ASSAULT VICTIM

1. First and foremost, care for the victim's medical and emotional needs.
2. Collection of forensic evidence is requested by law enforcement and may not be performed without the victim's consent.
3. Victims must be told of the options they have as to reporting and evidence collection. They can report to law enforcement and have evidence collected, they may decline reporting to law enforcement and have evidence collection, or they can decline to report and decline evidence collection but choose only treatment.
4. All victims should be offered prophylactic antibiotics for sexually transmitted diseases.
5. Women of child-bearing age should be informed about emergency contraception; if it is not offered to the victim at the hospital, a referral should be made so that the patient may receive it in a timely manner.
6. Written referral to community resources for post-assault counseling is critical.

WEBSITES

Antiretroviral postexposure prophylaxis: www.cdc.gov/mmwr/preview/mmwrhtml/rr5402a1.htm

Facts about sexual assault: www.rainn.org/statistics

National Crime Victimization Survey 2007: www.ojp.usdoj.gov/bjs/pub/pdf/cv07.pdf

Prevalence, incidence, and consequences of violence against women:

http://ncjrs.org/pdffiles/172837.pdf

Sexually transmitted diseases treatment guidelines: www.cdc.gov/std/treatment/2006/sexual-assault.htm

Sexual violence fact sheet: www.cdc.gov/ncipc/factsheets/svfacts.htm

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SPONTANEOUS ABORTION, ECTOPIC PREGNANCY, AND VAGINAL BLEEDING

Brandon H. Backlund, MD, FACEP

CHAPTER 78

1. What is spontaneous abortion or miscarriage?

Spontaneous termination of intrauterine pregnancy (IUP) before achieving fetal weight or maturity compatible with survival (less than 20–22 weeks' gestation, or fetal weight less than 500 g).

2. State the incidence and timing of spontaneous miscarriage.

Ten percent to 20% of clinically recognized pregnancies less than 20 weeks miscarry; 80% of these occur in the first 12 weeks of gestation. Approximately 70% of all spontaneous abortions occur before pregnancy is clinically detected.

KEY POINTS: MOST COMMON CAUSES OF SPONTANEOUS ABORTIONS

1. First 4 to 8 weeks of gestation: abnormal development of the zygote with chromosomal abnormalities in 50% to 60% of spontaneous abortions
2. Later in the first trimester:
   - Isolated chromosomal abnormalities
   - Maternal factors (insufficient progesterone support, alcohol use, cocaine use, tobacco use, nonsteroidal anti-inflammatory drug [NSAID], or caffeine use)
   - Structural uterine abnormalities

3. What are the five types of miscarriage or abortion?

- **Threatened abortion:** A pregnant patient within the first half of pregnancy with vaginal bleeding and a closed internal cervical os on bimanual examination. Cramping abdominal, pelvic, or back pain may also be present.
- **Inevitable abortion:** Findings of a threatened abortion, but with an open internal cervical os.
- **Incomplete abortion:** A miscarriage in progress. Only parts of the products of conception have been passed and may be visible in the cervical os or the vaginal canal.
- **Complete abortion:** All products of conception have been passed. Pain and significant bleeding should stop after a complete abortion.
- **Missed abortion:** Retention of a nonviable IUP within the uterus. Products of conception are demonstrable, but fetal development has ceased, no cardiac activity is visible, the cervical os is closed, and spontaneous passage has not occurred after 4 to 8 weeks.

4. Describe the early sonographic findings in a healthy pregnancy.

- **Gestational sac:** One of the earliest findings of normal pregnancy, seen at about 4 to 5 weeks gestational age (GA) by transvaginal ultrasound. This is a well-circumscribed fluid collection within the endometrial cavity. A true gestational sac is surrounded by two layers of tissue, forming the "double decidual" sign: an inner layer, called the decidua capsularis,

and an outer layer, called the decidua vera, which distinguish a true gestational sac from a pseudosac that may be seen with an ectopic pregnancy.

- **Yolk sac:** Seen at about 5 weeks' GA by transvaginal ultrasound. This is a well-defined echogenic ring seen within the gestational sac. For the purposes of emergency bedside ultrasound, this is considered by many authors to be the most reliable finding confirming the presence of an IUP and is easily recognized by sonographers of all skill levels.
- **Embryo:** Fetal pole visible within the gestational sac, seen at about 5 to 6 weeks' GA by transvaginal ultrasound. Fetal cardiac activity is generally recognizable by about 6 to 7 weeks.

5. What are important questions to consider during the examination and treatment of spontaneous abortion?

- Is the patient hemodynamically stable?
- Is there abdominal tenderness, guarding, or rebound (indicating a possible ectopic pregnancy [EP])?
- Are products of conception visible in the cervical os or vaginal canal (an incomplete abortion)?
- Is the cervical os open (inevitable abortion) or closed?
- Is the patient febrile, indicating a possible septic abortion?

6. What is a septic abortion?

A spontaneous abortion complicated by endometritis, parametritis, or peritonitis.

7. What are the signs and symptoms of a septic abortion?

- Malodorous discharge from the cervix or vagina
- Pelvic and abdominal pain
- Uterine tenderness
- Fever/hyperthermia
- Sepsis or septic shock

8. What are the earliest symptoms of a miscarriage?

Bleeding or spotting is usually first, followed by crampy abdominal, pelvic, or low back pain.

9. Describe the approach to the patient presenting with a positive pregnancy test and first-trimester vaginal bleeding or pain?

- Establish appropriate intravenous (IV) access. Two large-bore (18-gauge or larger) intravenous (IV) lines are recommended if there is any concern for hemodynamic instability.
- Obtain a measurement of hemoglobin and/or hematocrit or complete blood count (CBC). A blood type and screen should be sent if there is any possibility the patient may require a blood transfusion.
- Treat hypotension or tachycardia with IV fluids or transfusion of blood products, as indicated by patient condition.
- Determine whether an IUP has been previously documented by ultrasound. If no IUP has been documented, perform a bedside ultrasound to evaluate for a possible ectopic pregnancy. Unstable patients should not be allowed to leave the ED for diagnostic studies.
- Perform a bimanual and speculum pelvic examination to assess the source of bleeding, and examine the cervical os to assess for the presence of products of conception in the cervix or vaginal vault.
- If there is ongoing bleeding, remove any visualized tissue using gentle traction with ring forceps. This may help abate ongoing bleeding.
- Consult an ob/gyn for an open cervical os (suggesting inevitable or incomplete abortion), ongoing profuse bleeding, hypovolemic shock, or continued decreasing hemoglobin or hematocrit in the ED.
- Establish maternal rhesus (Rh) type to determine the need for RhoGAM.

Quantitative serum β-human chorionic gonadotropin (β-hCG) assay is helpful in assessing ectopic risk and coordinating follow-up.

10. What is the prognosis for patients with threatened abortion?

Patients with bleeding and a closed internal os have a risk of miscarriage estimated at 35% to 50%. If fetal cardiac activity is shown on ultrasound, risk of subsequent miscarriage is much lower. There is no treatment regimen that influences the course of a threatened abortion. Expectant management for women in early pregnancy failure can be as effective as medical or surgical management if the fetus is less than 13 weeks of gestation and the mother has stable vital signs without fever. Successful spontaneous abortion occurs in 91% of incomplete miscarriages and 26% with missed abortions.

11. Do diagnostic radiographs cause spontaneous abortion?

No. Although there is a risk for the development of fetal chromosomal abnormalities, diagnostic radiographs (<10 rads) place a pregnant woman at little or no increased risk for miscarriage. Therapeutic radiation, as well as antineoplastic agents, *do* increase the incidence of spontaneous abortion.

12. What factors are associated with spontaneous abortion and/or fetal abnormalities?

- Cigarette, alcohol (at high-exposure range), and cocaine use
- Progesterone-containing, but not copper-containing, intrauterine devices increase the risk of spontaneous abortion. Oral contraceptives taken either before or during pregnancy have not been associated with spontaneous abortion.
- Environmental chemicals (i.e., anesthetic agents, arsenic, aniline, benzene, ethylene oxide, formaldehyde, lead)
- Accutane (isotretinoin). Do *not* use in pregnant women or in women planning to become pregnant.
- Increased maternal parity and advanced maternal and paternal age (The frequency increases from 12% in women younger than age 20–26 to 40% in women age 40.)
- Conception within 3 months after a live birth
- Systemic disease of the mother (e.g., diabetes mellitus, cancer, hypothyroidism, or hyperthyroidism)
- Laparotomy: The closer the surgery to the pelvic organs, the greater the risk of spontaneous abortion
- Uterine defects, including leiomyomas (fibroids), where the location is more important than the size (submucosal fibroids carry higher risk); and developmental abnormalities including müllerian duct malformation or fusion and a septate, bicornuate, or unicornuate uterus

13. Is minor trauma a significant factor associated with spontaneous abortion?

No. Fetuses are well protected by maternal structures and amniotic fluid from minor falls or blows, but penetrating trauma, such as a gunshot wound or stab wound, is dangerous to the fetus.

14. Is exposure to spermicide before or after conception deleterious to a pregnancy?

No.

15. Define cervical incompetence.

Cervical incompetence is the painless dilation of the cervix during the second trimester that leads to spontaneous rupture of membranes and subsequent expulsion of uterine contents.

16. Name the drug used to prevent Rh immunization.

Rh immunoglobulin or RhoGAM. Any pregnant woman who is experiencing vaginal bleeding must have an Rh type checked. If she is Rh negative and less than 12 weeks' gestation, she

should receive a mini-dose of RhoGAM, 50 μg intramuscularly. If she is greater than 12 weeks' gestation, the full dose of RhoGAM, 300 μg, should be given.

17. What follow-up instructions should be given to a patient with a threatened abortion?

Careful instructions are given to return for an increase in pain, bleeding, or signs of hemodynamic instability, such as syncopal or near-syncopal episodes. The patient should also be instructed to return for heavy vaginal bleeding. In practice, patients are commonly instructed to return if bleeding exceeds saturation of 1 pad per hour for 4 to 6 hours, regardless of symptomatology. The patient should be instructed to bring any passed tissue to the ED or primary care physician (PCP). Arrangements to repeat quantitative hCG measurements should be made. Patients with a history of recurrent miscarriages need referral to a specialist for further evaluation. Patients are instructed to avoid sexual intercourse and inserting objects into the vagina, such as tampons or douches.

18. What about the emotional aspects of an early miscarriage?

Miscarriage is associated with a significant amount of psychological stress and grieving. Important therapeutic messages include informing the patient that early miscarriages are common and that miscarriages are usually due to spontaneous chromosomal abnormalities and not to the patient's own actions.

19. What is an ectopic pregnancy (EP)?

An EP is a pregnancy in which implantation of the gestational sac occurs outside of the uterus. In most cases, the pregnancy is located in the fallopian tubes, but EPs can occur in the interstitial or cornual portion of the uterus (2%), intra-abdominally (1.5%), on the ovary (0.1%), or within the cervix (0.1%). EP occurs in approximately 1 in 60 pregnancies in the United States; the risk is higher in older women and minorities. EP is still the leading cause of pregnancy-related first-trimester maternal deaths. Most ED series report that about 7% of first-trimester patients presenting to EDs have an EP diagnosed. Typically, patients with EP will present with abdominal pain, amenorrhea, or vaginal bleeding. However, more than 50% of women with EP are asymptomatic before tubal rupture and do not have a risk factor for EP.

20. What are common risk factors for EP?

- Pelvic inflammatory disease, which can be seen histologically in 50% of patients with EP
- Prior EP
- Tubal ligation
- Intrauterine device use
- Pelvic surgery
- Infertility and fertilization procedures (New technology, such as artificial fertilization, ovulation stimulation, and surgical procedures that result in salvage of potentially abnormal fallopian tubes, also may contribute to the increased incidence.)

Odds ratios are given in Table 78-1.

21. Define heterotopic pregnancy. What is the main risk factor for this condition, and what is its incidence?

A heterotopic pregnancy is defined as the simultaneous implantation of an embryo at two or more sites, most commonly manifested as an IUP and EP. The most significant risk factor is assisted fertility treatment. In the general population the incidence is thought to be low and has been historically cited as 1 in 30,000, but it is thought to be increasing, with some estimating the incidence as 1 in 4,000 or higher. Among patients undergoing fertility treatments, the incidence is as high as 1 in 100.

22. How reliable are routine serum and urine pregnancy tests in a patient with EP?

Sensitive serum or urine pregnancy tests are almost always positive in EP. β-hCG is secreted from the time of implantation and is detectable about 7 to 8 days after implantation of the

TABLE 78–1. ODDS RATIO FOR RISK FACTORS FOR ECTOPIC PREGNANCY

Degree of Risk	Risk Factors
High (odds ratios = 2.4–25)	Previous ectopic pregnancy Previous tubal surgery Tubal pathology In utero DES exposure
Moderate (odds ratios = 2.1–21)	Previous genital infections Infertility Multiple sexual partners
Low (odds ratios = 0.9–3.8)	Previous pelvic/abdominal surgery Smoking Vaginal douching Early age of intercourse (<18 years)

DES. diethylstilbestrol.
Adapted from data in Ankum WM, Mol BWJ, Van Der Veen F, et al: Risk factors for ectopic pregnancy—a meta-analysis. *Fertil Steril* 65:1093, 1996.

fertilized ovum. Qualitative pregnancy tests positive at a level of 10 to 50 mIU/mL are positive in 99% of patients with EP. Home pregnancy tests and less sensitive tests with higher thresholds may have false-negative results. Serum and urine tests provide similar accuracy for qualitative testing if their thresholds are similar.

23. What clinical signs and symptoms are useful to increase suspicion of an EP?

The classic picture of EP is of vaginal bleeding, pelvic or abdominal pain, prior missed menses, and an adnexal mass. However, this picture is neither sensitive nor specific. Missed menses occur in only 85% of EP patients. Vaginal bleeding and pain may occur only later, when the growing EP begins to fail or overstretch its abnormal implantation site. Adnexal masses are palpated in only 50% of patients, even under anesthesia; they may also represent the corpus luteum of the pregnancy rather than the ectopic gestation itself. Patients at high risk for EP are those with first-trimester pregnancy and either pelvic pain or risk factors for EP. Peritoneal signs, severe pain on pelvic examination, and cervical motion tenderness increase suspicion of a ruptured EP. There is, however, no constellation of historical factors or findings that confirms or excludes EP with sufficient reliability to obviate the need for evaluation.

24. What are the incidence and risk factors for tubal rupture?

The overall rate of tubal rupture is 18%. Risk factors include the following:

- History of tubal damage and infertility
- Induction of ovulation
- High β-hCG level (=10,000 IU/L) at time of diagnosis of EP

25. Why are corpus luteum cysts frequently confused with EPs?

The corpus luteum of the ovary, originating from the Graafian follicle, supports the pregnancy with secretion of β-hCG and progesterone during the first 6 to 7 weeks of gestation and may become cystic, growing to 5 cm in diameter or more. Cyst rupture can occur in the first trimester, presenting as a patient in early pregnancy with sudden pain, unilateral peritoneal findings, adnexal tenderness, and perhaps a mass.

26. What is the most efficient way to diagnose or exclude EP in the ED?

Ultrasound evaluation of early pregnancy is the best first ancillary study; 50% to 75% of patients have a definitive diagnosis of either IUP or EP. Normal IUPs can be seen by transvaginal sonography by about 5 weeks' GA. EPs can be seen on occasion, but an empty uterus may be the only finding. The risk of EP can be defined further by obtaining a quantitative hCG level if the ultrasound is inconclusive. IUP, if present, should be detected on ultrasound when the β-hCG concentration is above the discriminatory zone.

27. Describe the concept of the *discriminatory zone* as it applies to the serum β-hCG level.

In the early stages of a normal pregnancy, β-hCG levels increase at a predictable rate, correlating to expected stages of fetal development. The discriminatory zone is that β-hCG level at which a normally developing IUP, if present, should be visible by ultrasound. For transvaginal ultrasonography, the discriminatory zone is generally considered to be between 1,000 and 2,000 mIU/mL, depending on institutional protocols. If a patient has a serum β-hCG level above the discriminatory zone, but no IUP can be seen by ultrasound, the suspicion for EP increases significantly.

28. How else is quantitative β-hCG used?

Levels of hCG double every 2 to 3 days during the first 7 to 8 weeks of normal pregnancies. Because many women do not know the date of their last menstrual period, quantitative levels may be useful to estimate gestational age and correlate with expected sonographic findings. With β-hCG above the discriminatory zone, a healthy IUP should be visible by transvaginal sonography. Failure to double normally during the first 7 weeks indicates a failed pregnancy—either within the uterus or at an ectopic site. EP is likely if the ultrasound is indeterminate and the quantitative hCG is above the discriminatory zone or rising on serial measurements. A rapidly falling hCG level (less than half of the original in 48 hours) is unlikely to be an EP, whereas slowly falling levels may be seen with EP. A failed pregnancy is more likely to be ectopic if dilation and curettage fails to detect villi or if no products of conception are found at the time of miscarriage.

29. Does every patient with bleeding or pain in the first trimester require ultrasound before discharge from the ED?

All first-trimester complaints are treated as *rule out EP* until diagnosis of an IUP is established. In general, an ultrasound should be performed in all patients presenting with a positive pregnancy test and vaginal bleeding or pain. Unstable patients or those with peritoneal signs, severe pain, or heavy ongoing bleeding should have their ultrasound performed in the ED. If ED ultrasound is not available, an ultrasound by radiology should be ordered.

30. What are the ultrasound findings in patients with suspected EP?

See Table 78-2.

31. What patients with EPs can be discharged from the ED?

Women who are unstable with significant pain or signs of significant blood loss require admission. ED or inpatient observation may be useful in stable patients with worrisome symptoms, risk factors, or expected poor compliance to facilitate rapid sonography, quantitative hCG interpretation, or specialist consultation. Stable patients with indeterminate ultrasound results (*rule out EP*) may be followed on an outpatient basis. Expectant management or chemotherapy for women with few symptoms and low hormonal levels should be determined in consultation with an ob/gyn. The role of the ED physician is to consider the diagnosis, make every effort to exclude or make the diagnosis of EP expeditiously, and make the patient aware of the differential diagnosis and signs that should be of concern to her, ensuring access to close follow-up care for this potentially fatal condition.

TABLE 78–2. ULTRASOUND FINDINGS IN PATIENTS WITH SUSPECTED ECTOPIC PREGNANCY

Diagnostic of IUP	**Suggestive of EP**
Intrauterine fetal pole or yolk sac	Moderate or large cul-de-sac fluid without IUP
Intrauterine fetal cardiac activity	Adnexal mass* without IUP
Diagnostic of EP	**Indeterminate**
Ectopic fetal heart activity or	Empty uterus
Ectopic fetal pole	Nonspecific fluid collections
	Echogenic material
	Abnormal gestational sac

EP, ectopic pregnancy; IUP, intrauterine pregnancy.
*Complex mass most suggestive of EP but cyst also can be seen with EP.
Modified from Dart RG: Role of pelvic ultrasonography in evaluation of symptomatic first trimester pregnancy. *Ann Emerg Med* 33:310-320, 1999.

32. Which EPs should be treated medically with methotrexate?

Medical treatment is often less expensive than laparoscopic surgery and single-dose methotrexate is effective in 85% of patients. Methotrexate, a folic acid antagonist that inhibits DNA synthesis and cell reproduction, targets rapidly growing cells and has replaced surgery for many patients with EPs at low risk for rupture (no fetal heart tones, <3.5 cm diameter and without peritoneal signs). Because of significant failure rates, patients must be followed closely. Failure rates are higher when the β-hCG is >5,000. Patients commonly have significant pain with or without peritoneal signs several days after treatment with methotrexate. Abnormal vital signs, a decreasing hematocrit, or diffuse peritoneal signs are indications of EP rupture.

KEY POINTS: CANDIDATES FOR MEDICAL TREATMENT OF ECTOPIC PREGNANCY WITH METHOTREXATE†

1. Hemodynamically stable
2. No evidence of ectopic rupture
3. The ability and willingness to comply with post-treatment monitoring
4. Ectopic gestational sac <3.5 cm*
5. No fetal cardiac activity on ultrasonographic examination*

*Sac size >3.5 cm or presence and cardiac activity are relative, not absolute, contraindications.
†Adapted from ACOG Practice Bulletin No. 94: Medical management of ectopic pregnancy.

33. What are contraindications to methotrexate therapy for ectopic pregnancy?

- Hemodynamic instability
- Breastfeeding
- Immunodeficiency
- Alcoholism

- Hepatic or renal disease
- Preexisting blood dyscrasia (e.g., significant leukopenia, anemia, thrombocytopenia)
- Active pulmonary disease
- Peptic ulcer disease
- Known sensitivity to methotrexate

34. Name the sources and causes of third-trimester vaginal bleeding.

The sources are the vagina, cervix, and uterus. In the following list, life-threatening causes are indicated by an asterisk:

- Placenta previa: 0.3% to 0.5% of live births*
- Placental abruption: 0.8% to 1.2% of pregnancies (15%–20% present without vaginal bleeding)*
- Uterine rupture: 0.05% of pregnancies*
- Marginal sinus rupture
- Bloody show
- Local trauma
- Cervical polyps and lesions

35. What is placenta previa? How is it diagnosed?

Placenta previa occurs when the placenta implants on or near the cervical os. Total coverage of the cervical os by placenta is called **complete placenta previa**, whereas subtotal coverage is called **partial placenta previa. Marginal placenta previa** occurs when the margin of the placenta approaches but does not cover any of the cervical os.

Placenta previa should be suspected when a pregnant patient in the second half of pregnancy presents with bright red vaginal bleeding. It is usually painless and may present with or without uterine contractions. Placenta previa is dangerous, because vaginal penetration or manipulation of the cervix during a pelvic examination may rupture placental blood vessels and cause massive hemorrhage. Color flow Doppler ultrasound is 82% sensitive and 91% to 96% specific for placenta previa. It is frequently diagnosed early in pregnancy and followed with serial ultrasound studies until delivery, spontaneously resolving in up to 90% of cases diagnosed before 20 weeks' gestational age.

36. How is placenta previa treated?

Early consultation with ob/gyn should be obtained when the diagnosis is suspected. Administer supplemental oxygen, obtain two large-bore intravenous lines, and perform maternal cardiopulmonary monitoring, and fetal monitoring. Obtain a CBC, hemoglobin, or hematocrit, and blood type and screen for anticipated transfusion. Place the patient in the left lateral decubitus position. In the stable patient, ultrasound may confirm the diagnosis. Because life-threatening bleeding may occur with placenta manipulation, *do not* perform a pelvic examination unless you are in the operating room or delivery suite with an experienced obstetrician. Patients who are hemodynamically stable with small amounts of bleeding may be admitted for ongoing maternal and fetal monitoring. In selected cases, delivery is delayed to optimize fetal development. Unstable patients or patients with life-threatening bleeding are treated by emergency delivery.

37. What is placental abruption (abruptio placentae)? Why is it dangerous?

Placental abruption is the premature separation of the placenta from its insertion on the uterine wall where a large amount of blood may collect between the placenta and the uterine wall, causing maternal shock and fetal demise. Abruption occurs spontaneously or after abdominal trauma. The uterus is firm and the patient reports severe abdominal pain. Hypotension may occur with vaginal bleeding occurring in about 80% of patients. If vaginal bleeding does occur, the blood is dark red. This presentation is in contrast to the painless, bright red bleeding of placenta previa. Placental abruption is diagnosed by ultrasound.

38. Describe the treatment of placental abruption.

Immediately consult an ob/gyn specialist. Start two large-bore intravenous lines, and administer oxygen. Monitor fetal heart tones and the maternal vital signs. Obtain a CBC, hemoglobin, or hematocrit, type and screen for anticipated transfusion, and coagulation studies. If the mother and fetus are stable, arrange an immediate ultrasound. Unstable patients should be transferred directly to the operating room or delivery suite for delivery.

39. What is uterine rupture? Why is it dangerous?

A grave complication of late pregnancy in which the uterus ruptures, usually during contractions. It can produce massive, life-threatening intra-abdominal hemorrhage. Maternal mortality is 8%, and fetal mortality is estimated at 50%. Uterine rupture presents with sudden abdominal pain, uterine contractions and shock late in pregnancy. There may be scant vaginal bleeding, but the abdomen is extremely tender. The most significant risk factor for uterine rupture is prior caesarean section or other uterine surgery.

40. What is the treatment of uterine rupture?

Start two large-bore intravenous lines, and administer oxygen with immediate transfer to the operating room or delivery suite for laparotomy and hysterectomy. Ultrasound may be necessary in selected cases to distinguish uterine rupture from placental abruption.

41. Describe the non-life-threatening causes of third-trimester vaginal bleeding.

- Bloody show is a pink mucous discharge caused by cervical changes that precedes labor by several hours to a week.
- The cervix is prone to hemorrhage during late pregnancy, and local trauma from vaginal penetration, including intercourse, may cause bleeding.
- Cervical erosions or preexisting polyps produce limited bleeding.
- Marginal sinus rupture is a premature separation of the placenta limited to the placental margin.

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THIRD-TRIMESTER COMPLICATIONS AND DELIVERY

CHAPTER 79

Gina Soriya, MD

1. What is preeclampsia?

Preeclampsia is a condition that occurs in pregnancy after 20 weeks' gestation, characterized by new-onset high blood pressure and proteinuria with widespread vascular endothelial malfunction and vasospasm.

2. What level of hypertension and proteinuria qualify as preeclampsia?

- **Hypertension**: In a woman who was normotensive prior to 20 weeks' gestation, a systolic blood pressure (BP) greater than 140 mm Hg and a diastolic BP greater than 90 mm Hg. In a woman who has preexisting hypertension, if the systolic BP has increased by 30 mm Hg, or the diastolic BP by 15 mm Hg.
- **Proteinuria**: Excretion of 300 mg or more of protein in the urine in a 24-hour period.

Note: Edema was previously part of the classic diagnostic triad for preeclampsia; however, this can be a common finding in pregnancy. In addition, up to one third of preeclamptic patients do not have edema. It is no longer considered part of the diagnosis of preeclampsia.

3. What are the diagnostic criteria for *severe* preeclampsia?

The presence of one of the following defines severe preeclampsia: systolic BP greater than 160 mm Hg or diastolic BP greater than 110; pulmonary edema; thrombocytopenia, microangiopathic hemolysis, symptoms suggesting end-organ involvement, such as headache, vision changes, and epigastric or right upper quadrant pain; oliguria defined as less than 500 mL in 24 hours; severe intrauterine growth retardation; oligohydramnios.

4. What causes preeclampsia?

The exact pathophysiology is unknown. Research suggests there may be incomplete invasion of cytotrophoblasts into the uterine spiral arteries. This leads to placental hypoperfusion, which may cause the placenta to release factors that circulate in the maternal blood stream, resulting in widespread endothelial dysfunction and end-organ damage.

5. What are the risk factors for preeclampsia?

Preeclampsia is primarily a complication of first pregnancies. Other risk factors include conception before age 20, personal or family history of preeclampsia, multi-fetal pregnancies, advanced maternal age, high body mass index, and adverse outcome in previous pregnancy. There are also several preexisting medical conditions that can contribute to preeclampsia, including insulin-dependent diabetes, connective tissue disease, renal disease, chronic hypertension, and antiphospholipid antibody syndrome.

6. How common is preeclampsia?

Worldwide, 5% to 18% of pregnancies are complicated by preeclampsia. In the United States, the incidence is 5% to 8%.

7. What is the treatment for preeclampsia?

For mild disease, expectant management is appropriate, with delivery if the patient is at term. Patients with severe preeclampsia should be admitted and given magnesium sulfate. Anti-hypertensives should be used for sustained systolic BP greater than 160 mm Hg or sustained

diastolic BP greater than 110 mm Hg. If symptoms do not resolve, pregnancy termination at less than 24 weeks gestation may need to be considered for maternal safety. For patients between 24 and 34 weeks' gestation, corticosteroids are administered to enhance fetal lung maturity. Expectant management can be considered in these patients. However, if the condition persists or worsens within 48 hours, delivery may be the safest option for the mother. Patients with severe preeclampsia at greater than 34 weeks' gestation should undergo delivery.

8. What antihypertensive medications can be used?

- Hydralazine: 5 to 10 mg intravenously every 20 minutes until desired effect to a maximum dose of 30 mg.
- Labetalol: 20 mg intravenously, may repeat 40 mg within 10 minutes if no effect, then 80 mg every 10 minutes for a maximum of 300 mg.
- Nifedipine: 10 mg orally every 15 to 30 minutes with a maximum of three doses.
- Sodium nitroprusside: This medication can be used in severe hypertensive emergency if the previous medications have been ineffective. However, cyanide poisoning of the fetus may occur and thus its long term use should be limited.

Contraindicated antihypertensives: angiotensin-converting enzyme inhibitors, diuretics

9. What are the complications of preeclampsia?

There are several complications of preeclampsia, the most serious being eclampsia, intracerebral hemorrhage, hemolysis with elevated liver enzymes and low platelets (HELLP) syndrome, renal failure, and posterior reversible encephalopathy syndrome (PRES).

10. Is there a way to prevent preeclampsia?

No proven definitive method of prevention has been identified. However, some studies have described the use of calcium, low-dose aspirin, or vitamins C plus E in the prevention or reduction in severity of preeclampsia.

11. Is any pregnant woman with hypertension considered to have preeclampsia?

No. There are four categories of hypertensive disorders in pregnancy:

- Chronic hypertension
- Gestational hypertension
- Preeclampsia
- Preeclampsia superimposed on chronic hypertension

12. What is gestational hypertension?

Gestational hypertension is defined as a systolic BP greater than 140 mm Hg or a diastolic BP greater than 90 mm Hg after 20 weeks' gestation in a previously normotensive woman. Blood pressure returns to normal less than 12 weeks' postpartum.

13. What is HELLP syndrome?

Hemolysis, elevated liver enzymes, and low platelet count. Patients typically present with epigastric or right upper abdominal pain, nausea, and vomiting. Some patients may also complain of malaise, headache, or visual disturbances. Others may present atypically with nonspecific viral syndrome-like symptoms.

14. What are the complications and mortality rate of HELLP syndrome?

Complications include abruptio placentae, disseminated intravascular coagulation (DIC) with resultant severe postpartum hemorrhage, rupture of a subcapsular liver hematoma, intracranial hemorrhage or infarction, and acute renal failure.

- Maternal mortality is considered to be approximately 1.1%, although some studies have reported the mortality rate to be as high as 25%. The leading causes of maternal death are intracerebral hemorrhage, cerebral infarction, and hepatic rupture.

- Fetal mortality rates range from 7.4% to 34%, with improved survival after 32 weeks' gestation. The most common causes of neonatal death with HELLP syndrome are abruptio placentae, placental insufficiency, and prematurity.

15. How is HELLP treated?

In the patient > 34 weeks' gestation, immediate delivery is the treatment of choice. Between 27 and 34 weeks gestation, corticosteroids are administered for fetal lung development and delivery is recommended within 48 hours of evaluation and stabilization. For gestations < 27 weeks, conservative management can be considered for 48 to 72 hours of evaluation, along with corticosteroid administration.

16. What is eclampsia?

Eclampsia is defined as new onset of grand mal seizure or unexplained coma in a patient with signs and symptoms of preeclampsia.

17. What is the treatment for eclampsia?

Immediate stabilization of airway, breathing, and circulation (ABCs) is necessary, with administration of oxygen, placement of intravenous lines, and monitoring of mother and fetus. Magnesium sulfate 4 to 6 g intravenously, followed by a maintenance dose of 2 g/hr. Administration of a benzodiazepine may be considered if the patient seizes a second time. After magnesium, blood pressure should be controlled with hydralazine or Labetalol. The definitive treatment of eclampsia is delivery.

18. What are the findings of magnesium toxicity, and how should the patient be monitored?

Magnesium toxicity usually presents when levels reach 8 to 12 mg/dL, with loss of patellar reflexes, somnolence, slurred speech, and flushing. At levels of 15 to 17 mg/dL, muscle paralysis and respiratory arrest may occur. Cardiac arrest is seen at levels of 30 to 35 mg/dL. For patients receiving magnesium, patellar tendon reflexes and respiratory rate should be checked every hour and magnesium levels should be drawn every 4 to 6 hours.

19. What is the most common cause of death in a patient with preeclampsia-eclampsia?

Central nervous system complications, including cerebral edema, hemorrhages, and infarctions.

20. Does a woman have to be pregnant to have preeclampsia or eclampsia?

No. It can occur in the postpartum period. Usually symptoms develop within 48 hours of delivery, but it can present as late as 1 month post-partum.

21. Are there atypical presentations of preeclampsia or eclampsia?

Yes. Preeclampsia or eclampsia can occur at < 20 weeks' gestation, primarily in the setting of a molar or partial molar pregnancy. However, hypertension and proteinuria at < 20 weeks' gestation, among other laboratory abnormalities, may indicate another disease process in the woman, such as hemolytic uremic syndrome, antiphospholipid antibody syndrome, lupus nephritis, or thrombotic thrombocytopenic purpura.

KEY POINTS: PREECLAMPSIA AND ECLAMPSIA

1. Preeclampsia or eclampsia can present any time from 20 weeks' gestation to 4 weeks' postpartum.
2. Hydralazine and Labetalol are used to treat hypertension in preeclampsia or eclampsia.
3. Magnesium sulfate is the first-line treatment in eclampsia.

22. What do I need to do to stabilize a pregnant patient brought into the ED?

The unplanned delivery of babies in the ED will occur. After vital signs and chief complaint are obtained, establish intravenous access. Most mistakes in the care of the mother and baby are made as the result of lack of preparation and planning. Help should be sought from Obstetrics and Pediatrics as soon as it is evident that a delivery is going to occur in the ED. Labor and delivery is a natural process, which usually proceeds to its conclusion with limited intervention and without difficulty.

23. What information do I need to care properly for the pregnant patient?

Obtain the patient's age, her due date, and number of past pregnancies. Ask her if she feels the baby moving and if she is having contractions, vaginal bleeding, or leakage of fluid from the vagina. Inquire about any problems with the current pregnancy and clarify that she is only carrying one baby presently. In addition to identifying her current medications, ask about illicit drug use. Find out where she is getting her prenatal care and the name of her doctor or midwife.

24. How are the baby and pregnancy evaluated?

Indirect evaluation of the baby can provide some information. Fetal heart tones should be obtained as soon as the mother is stabilized. A normal fetal heart rate is between 120 and 160 beats per minute. The fundal height (the distance in centimeters from the symphysis pubis to the top of the pregnant uterus) should be measured to give a rough estimate of gestational age. For example, a fundal height of 32 cm would indicate a gestational age of roughly 30 to 34 weeks.

25. How do you check cervical dilation?

Under sterile conditions, a digital pelvic examination is performed. The diameter of the cervical opening in front of the baby's head is estimated in centimeters. The measurements vary from closed up to 10 cm. Practice is required for accuracy. To avoid exacerbating a placenta previa, a digital pelvic examination is contraindicated in the ED for any patient bleeding in the late second and third trimesters; immediate obstetrical consultation should be initiated.

26. What should be in an emergency delivery pack?

The contents of an emergency delivery pack may vary. At a minimum, each pack should contain:

- A bulb syringe to clear the baby's nose and mouth
- Sterile gloves in various sizes for the delivering health care provider
- Sterile towels to dry off baby and a baby blanket to keep the baby warm
- Four Kelly clamps for the umbilical cord or vaginal or perineal bleeders
- Mayo scissors to cut the umbilical cord
- Two ring forceps
- Three packs of 4-×-4 sponges
- Container for placenta

27. How can I determine if a delivery is imminent?

This can be difficult. Some patients will not appear to be in any distress one minute, and there is a baby in the bed the next minute. If delivery is imminent, most patients will feel the urge to push or bear down in response to the pressure of the baby as it moves down the birth canal. The patient may say, "The baby is coming" or "I have to have a bowel movement." She may be visibly bearing down or pushing with contractions. If examination of the patient reveals the cervix is 6 cm or more dilated, delivery may occur before or during transport to labor and delivery.

28. I have a laboring pregnant patient in the ED and the baby can be seen distending the mother's perineum. The obstetrician is on the way but will not make it in time. What do I do now?

Wearing sterile gloves, apply gentle pressure against the presenting part to prevent sudden expulsion and to allow gradual stretching of the perineum.

29. Okay, the baby is part way out. Should I pull on the baby to help the delivery?

In most cases, the mother will push the baby out without help. Pulling on the baby may interrupt the normal delivery process unless you are experienced with deliveries. The best way to help with the delivery is to use your hands to help control and guide the delivery of the rest of the baby's body. The baby may deliver very quickly, so keep a good grip.

30. If the umbilical cord is wrapped around the neck during a delivery, what should I do?

If the cord is wrapped around the neck, it should be pulled gently over the head, if possible, so that it does not tighten as the baby is being born. If the cord is too tight to lift over the head, carefully apply two clamps to the cord and cut the cord between the clamps. Then the cord can be unwound from around the neck and the baby delivered. See Chapter 67 for the approach to neonatal resuscitation.

31. The placenta is still inside. What should I do now?

There is no hurry to deliver the placenta until the obstetrician arrives, unless there is heavy bleeding.

If there is heavy bleeding, have the mother try to push out the placenta. Provide very gentle traction using ring forceps on the distal end of the cord, winding it around the forceps as the placenta is delivered; never forcibly pull on the cord. If the placenta delivers by itself, massage the uterus gently but firmly to keep it contracted into a firm *ball*. As long as the uterus stays firmly contracted, bleeding should be minimal (up to 500 mL is considered within the normal range).

KEY POINTS: CHILDBIRTH IN THE ED

1. Labor and delivery is a natural process, which usually proceeds to its conclusion without difficulty. In most cases, little intervention is required on the part of the health care provider.
2. It is essential to have intravenous access during the delivery process.
3. During delivery, the health care provider should not attempt to speed up the process by pulling or pushing on the baby. In most cases it is unnecessary to do more than attempt to guide and control the process.

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MULTIPLE TRAUMA

Peter Rosen, MD

1. What is multiple trauma?

Significant injury to more than one major body system or organ.

2. What is new in the management of trauma?

- Diagnostic peritoneal lavage (DPL), which for many decades was the principle diagnostic study for an objective evaluation of a traumatized abdomen, has largely been displaced by the ultrasound-focused abdominal sonography for trauma (FAST) examination. Both are overly sensitive, and can tell you that there is blood or fluid in the abdomen, but not whether there is an injury serious enough to warrant laparotomy. A DPL may still be performed on an unstable patient with blunt abdominal trauma, but it is no longer useful to obtain cell counts because one would only operate if the return were grossly positive.
- It has become apparent from the use of computed tomography (CT) scan of the chest, that many patients harbor a small pneumothorax after blunt chest trauma that formerly went unrecognized on plain film imaging of the chest. It is further apparent that most of these cases of small pneumothorax can be watched without a tube thoracostomy. The exception would still be a patient who needs mechanical ventilation, either because of a need for immediate surgery or because of ventilatory failure from another cause.
- Not only is plain film study insensitive to the presence of a pneumothorax, it is dependent upon the position of the patient (more accuracy is obtained when the patient is upright, which of course is impossible for most multitrauma patients.) Ultrasound imaging has shown new utility in the identification of pneumothorax and is part of the utility provided by use of bedside ultrasound for the trauma patient along with the FAST examination. Repeat examinations should be performed in the patient who is not responding to resuscitation, even if the initial examination appeared accurate and negative.
- For many years Ketamine was thought to be a harmful drug to use for airway management in a trauma patient, but recent experience shows that it may be extremely helpful for the combative trauma patient who needs emergent airway management. It does not raise intracranial pressure as was once thought, does raise blood pressure, unlike most sedating agents, provides quick control of a combative patient, and is not only helpful for the emergency intubation, but also for any other acute invasive procedure that needs to be performed. It is also safer than Haldol or Droperidol, which both have concerns for seizure induction and serious dysrhythmia production, especially in the presence of alcohol consumption, perhaps accompanied by other drugs such as cocaine.
- Surgical airway management has become less and less necessary with improvements in intubation equipment, and with more experience in the management of difficult airways. There are still patients in whom it will be necessary, although it is more and more difficult to find the means of providing the technical training to residents in emergency medicine.

3. Define mechanism of injury.

Mechanism of injury refers to the events and conditions that lead to visible and occult traumatic injuries. Significant mechanism of injury is associated with a higher likelihood of multiple trauma. Less obvious mechanism is of greater concern with increasing age or

preexisting disease. A 70-year-old patient with ankylosing spondylitis is much less able to tolerate blunt trauma to the spinal column and pelvis than is a healthy younger person. Even trivial mechanism of injury can produce significant damage in patients with prior disease or in patients of advanced age. Many times the mechanism of injury is obscure or not known when the patient arrives at the ED or trauma unit; it is helpful to know if anyone on the scene was killed, severely injured, or perhaps taken to a different location than the patient being treated as sometimes happens with remote trauma or when there are multiple casualties.

4. Give examples of significant mechanisms of injury.

Blunt trauma

- Automobile crashes: fatality at the scene or in the same vehicle, passenger ejection, vehicle rollover, significant interior damage
- Automobile-pedestrian accidents: high speed, damage to exterior of vehicle
- Falls: Greater than one story (12–15 feet)

Penetrating trauma

- Gunshot wounds to head, neck, or torso
- Stab wounds to neck or torso

5. List the first steps in managing multiple trauma in the ED.

- Activate the trauma resuscitation team.
- Designate a trauma captain and call for O-negative blood if indicated by prehospital course and vital signs.
- Transfer the patient from the ambulance stretcher or other conveyance to the ED resuscitation bed using spinal injury precautions if indicated.
- Quickly obtain a history, including the mechanism of injury, field treatment, and response to this field treatment.
- Obtain vital signs while the patient is being undressed.
- Assess the airway, breathing, and circulation (ABCs) and intervene as needed.
- Draw blood for type, cross-matching, and baseline laboratory testing.

6. How should the patient be undressed?

Because immobilization is necessary until the spine can be cleared, all movement should be avoided. To obtain complete visualization rapidly while protecting the spine, simply cut the clothes away. Keep in mind that one of the purposes of clothing removal is to rid the patient of objects that can cause further damage to the patient or injury to the health care providers, such as shards of broken glass, bits of metal, or weapons.

7. What are the ABCs (and D) of trauma?

- A = Airway
- B = Breathing
- C = Circulation
- D = Disability

8. Discuss assessment of the airway.

Airway patency is evaluated by listening for vocalizations, asking for the patient's name, and looking in the patient's mouth for signs of obstruction (e.g., blood, emesis, or foreign debris). The trauma captain must determine if the patient needs active airway management and verify that supplemental oxygen is being administered continuously to all patients who do not require immediate intubation.

Mandatory indications for airway management in trauma include the following:

- Massive facial injuries
- Head injury with Glasgow Coma Scale (GCS) less than 8
- Penetrating injury to the cranial vault
- Missile penetrating injury to the neck

- Blunt injury to the neck with expanding hematoma or alteration of the voice
- Multisystem trauma with persistent shock
- Any patient with an injury that has the potential to distort the patient's anatomy (e.g. penetrating wound of the neck). It is much more prudent to intubate these patients while it is still possible to do so than to have to try when the anatomy is distorted, the patient is obstructing the airway, and the emergent airway management becomes a difficult flail.

Relative indications for airway management in trauma include the following:

- Upper airway obstruction from any cause
- Any patient with injuries impairing ventilation
- Flail chest with increasing respiratory rate or deteriorating oxygenation
- Any patient with one or more rib fractures who is going to need a ventilator or a general anesthetic
- Patients with bilateral pneumothorax
- Bilateral missile penetrating injuries of the thorax
- Patients with severe hypovolemic shock
- Patients with recurrent hemothorax, or who do not respond to tube thoracostomy

9. How is breathing assessed?

Ventilation is assessed by observing for symmetric rise and fall of the chest and by listening for bilateral breath sounds over the anterior chest and axillae. The chest should be palpated gently for subcutaneous air and bony crepitus. Oxygen saturation should be monitored continuously. The trauma captain determines whether or not tube thoracostomies or ventilatory support is needed immediately.

10. How is circulation assessed?

Circulatory function is assessed by noting the patient's mental status; skin color and character (cool and clammy versus warm and dry); vital signs; and presence or absence of radial, femoral, and carotid pulses. Continuous cardiac monitoring should be started. Prehospital vascular access and type and amount of volume infused are assessed. The trauma captain determines whether additional vascular access or volume of crystalloid is needed and whether blood should be administered. A FAST examination should be performed on any patient with torso trauma to detect free fluid (blood) in the abdomen or perineum. See Chapter 5.

11. How is disability assessed?

The patient's neurologic status should be assessed (level of consciousness and gross motor function). An initial ED GCS rating should be ascertained, and this should be compared with the prehospital GCS. With any alteration of consciousness, it is useful to perform a rectal examination to determine anal sphincter tone.

12. What type of intravenous access should be established in a patient with major trauma?

At least two large-bore (16-gauge) intravenous catheters should be placed. Forearm or antecubital veins are the preferred sites for initial access. Although subclavian and internal jugular catheters allow central venous pressure monitoring, they rarely provide access for high-volume intravenous infusions unless a Cordis introducer is left in place. These routes should be used only if no other access exists, and catheters should be placed on the ipsilateral side of the chest trauma unless a subclavian vascular injury is suspected. Femoral lines and saphenous cutdowns are indicated in patients with a dropping blood pressure because large-volume infusions will be needed quickly. Central line placement is aided by use of an ultrasound probe. This will also make femoral line insertion safer.

13. Where should cutdowns be performed?

The ankle. The distal saphenous vein can be found between the anterior tibialis tendon and the medial malleolus.

KEY POINTS: MULTIPLE TRAUMA

1. Trauma care is a team sport involving prehospital providers and emergency physicians and surgical staff.
2. All trauma patients should be completely undressed and examined.
3. All trauma victims must be assessed for occult bleeding in the cranial vault, chest, and abdomen.
4. When indicated by mechanism of injury, symptoms, or signs, spinal precautions must be maintained until the spine is cleared.

14. What parameters should be monitored in multiple trauma victims?

Vital signs, neurologic status, cardiac rhythm, oxygen saturation, and, if possible, central venous pressure and urinary output. Hypothermia adversely affects outcome, and core temperature can drop rapidly when the patient is disrobed and receives large quantities of cold intravenous fluid. Tachypnea is a sensitive sign of hypoxia and acidosis and should be measured accurately rather than estimated. Neurologic status, skin color and character, and urinary output over time should be monitored.

15. When should blood be administered?

O-negative (universal donor) blood should be reserved for patients who are in arrest from hypovolemic shock. If 50 mL/kg of crystalloid is infused rapidly and there is no significant improvement in the patient's circulatory status, type-specific noncross-matched blood should be administered if available. Otherwise, use type O initially. (For more details, see Chapter 4.)

16. Are laboratory tests useful?

No, although all major trauma victims should have a clot blood tube sent for type and cross-match. Baseline values of hematocrit and serum amylase (or preferably lipase) may be useful in detecting occult injuries and preexisting anemia. Urinalysis should be done to detect hematuria. Many trauma centers obtain an extensive trauma panel, which may be useful if the patient requires surgery or has underlying disease. No laboratory test defines injury, however, and the trauma panel is of little use in determining initial management, disposition, or need for surgery. Common initial lab tests in multiple trauma include the following:

- Complete blood cell count
- Type and cross-match
- Electrolytes
- Urinalysis
- Blood urea nitrogen, creatinine
- Blood alcohol as indicated
- Glucose
- Toxicology as indicated
- Prothrombin and partial thromboplastin times
- Amylase, lipase

17. What is the secondary survey?

The complete physical examination performed after the ABCs have been assessed and stabilized. This survey includes assessment of the chest, abdomen, pelvis, back, and extremities. A repeat neurologic examination and rectal examination also should be done. The purpose of the rectal examination is to determine if there is gross blood in the rectum, if there is adequate sphincter tone and sensation, and if the prostate gland is in a normal position.

18. Which radiologic studies need to be obtained immediately?

- When the patient is stabilized, portable radiographs of the lateral cervical spine, chest, and pelvis should be obtained.
- In gunshot wounds, portable films in two planes may be needed to determine the location of the bullet.
- If the mechanism of injury is an ejection or a fall, a cross-table lumbar spine film should be added to the initial series.

19. How do I prioritize diagnostic tests?

Prioritization is based on potential life threats. After external hemorrhage is controlled, diagnosing intraperitoneal hemorrhage takes precedence. Unless an indication for immediate laparotomy is present, the patient should undergo diagnostic peritoneal lavage, abdominal CT scan, or abdominal ultrasound to assess the intraperitoneal cavity. After these procedures, attention should be focused on ruling out correctable intracranial hemorrhage, such as a subdural or an epidural hematoma. Based on the mechanism of injury and the initial course, other specialized studies to evaluate the aorta and the retroperitoneum should be done. If the patient has a bleeding diathesis (e.g., hemophilia) or is on an anticoagulant, even minor head injury mandates a CT scan.

20. How are fluids managed in pediatric trauma?

Start with a bolus of 20 mL/kg of normal saline (NS) or lactated Ringer's (LR). This can be repeated until up to 50 mL/kg has been reached. At this point, start packed red blood cells at 10 mL/kg. See Chapter 90.

21. What is the significance of blunt abdominal trauma in the pregnant woman?

- During the first trimester, the fetus is well protected, and the best treatment for the fetus is to protect the mother from hypovolemic shock.
- In the second trimester, the fetus is more vulnerable and must be monitored for signs of placental abruption
- In the third trimester, the fetus is the most vulnerable, and even minor trauma necessitates fetal monitoring for several hours. If the signs of abruption occur, emergency caesarian section must be performed. See Chapter 89.

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CHAPTER 81

MAXILLOFACIAL TRAUMA

Ethan M. Ross, MD, and Carlo L. Rosen, MD

1. What are the facial bones?

The facial bones are the frontal, temporal, nasal, ethmoid, lacrimal, palatine, sphenoid, zygoma, maxilla, and mandible.

2. What is the initial approach to a patient with maxillofacial trauma?

The initial management of patients with facial trauma should follow the ABCs (airway, breathing, and circulation) of trauma resuscitation. The airway is the primary concern and can be challenging in these patients. Significant facial trauma may cause swelling or distortion of the airway as a result of bleeding, loose teeth, or fractures. In patients with mandibular fractures, the tongue loses its support and can occlude the airway. Early endotracheal intubation should be considered in patients with significant midface or mandibular trauma, especially if they exhibit any signs of airway distress. Standard methods of intubation, such as rapid-sequence intubation, should be attempted first. However, airway distortion resulting from facial trauma sometimes necessitates a cricothyrotomy. All patients with facial and head trauma should be assumed to have a cervical spine injury. In-line cervical spine stabilization should be used during intubation. The incidence of cervical spine injuries in patients with facial trauma is 1% to 4%.

3. Which procedure is contraindicated in patients with maxillofacial trauma?

Nasogastric tube placement should not be performed because of the risk of intracranial placement through a fracture in the cribriform plate. The small size and flexibility of the nasogastric tube allow it to be misdirected through a fracture into the brain. There is also a theoretical concern about placing a nasotracheal tube through the cribriform plate into the brain. However, an endotracheal tube is larger and more rigid than a nasogastric tube. The literature suggests that the risk of intracranial placement of a nasotracheal tube is low.

4. What is a blow-out fracture? What is the entrapment syndrome?

A blow-out fracture is a fracture of the orbital floor that results from a direct blow to the orbit. Increased intraorbital pressure causes rupture of the floor of the orbit. The entrapment syndrome is binocular diplopia and paralysis of upward gaze that results from entrapment of the inferior rectus muscle in the orbital wall defect. Diplopia is noted by having the patient follow and count fingers on upward gaze. Other physical findings include infraorbital anesthesia and enophthalmos (posterior displacement of the globe into the orbit). Patients may have tenderness or step-offs at the infraorbital rim or subcutaneous emphysema secondary to a fracture into the maxillary sinus. Ophthalmologic evaluation for associated orbital trauma (globe rupture, hyphema, retinal tear or detachment, blindness), despite an initially normal visual acuity and funduscopic examination, should be considered.

KEY POINTS: CLINICAL SIGNS OF ORBITAL FRACTURES

1. Eyelid edema
2. Enophthalmos
3. Proptosis
4. Limitation of upward gaze
5. Diplopia
6. Infraorbital anesthesia
7. Subcutaneous emphysema

5. What are Le Fort fractures?

The Le Fort classification is used to describe maxillary fractures (Fig. 81-1). Midface fractures are diagnosed by grasping the upper alveolar ridge and noting which part of the midface moves.

- **Le Fort I**, a transverse fracture just above the teeth at the level of the nasal fossa, allows movement of the alveolar ridge and hard palate.
- **Le Fort II**, a pyramidal fracture with its apex just above the bridge of the nose and extending laterally and interiorly through the infraorbital rims, allows movement of the maxilla, nose, and infraorbital rims.
- **Le Fort III**, the most serious of the Le Fort fractures, represents complete craniofacial disruption and involves fractures of the zygoma, infraorbital rims, and maxilla.

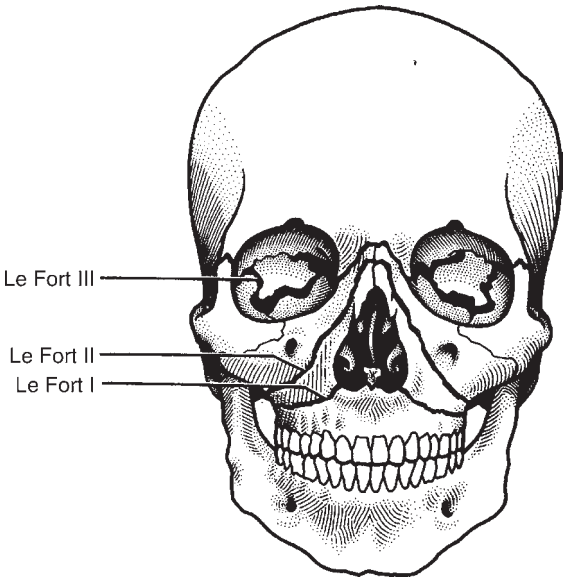

Figure 81-1. Le Fort classification of facial fractures. Le Fort I: palato facial disjunction. Le Fort II: pyramidal disjunction. Le Fort III: craniofacial disjunction. (From Cantrill SV: Face. In Marx JA, Hockberger RS, Walls RM, et al, editors: *Rosen's emergency medicine: concepts and clinical practice*, ed 5, St. Louis, 2002, Mosby, p 325.)

It is rare for these fracture types to occur in isolation; they usually occur in combination (one type on one side of the face and another on the other side).

6. Is there a role for screening patients with LeFort fractures for blunt cerebrovascular injury?

Blunt cerebrovascular injury (BCVI) to the carotid or vertebral artery is becoming increasingly recognized in blunt trauma and is found in nearly 1% of all blunt trauma patients when screening protocols are used. Significant morbidity and mortality is encountered if left untreated and there is frequently a clinically silent period before symptoms occur. Although no standard exists for screening, guidelines are being developed that suggest screening should be undertaken with computed tomographic angiography (CTA) or standard angiography in patients with signs or symptoms of BCVI and patients at high risk. High-risk patients include those with a LeFort II or III fracture, certain cervical spine fracture patterns (subluxation, fractures extending into the transverse foramen, fractures of C1–C3), basilar skull fracture with carotid canal involvement, diffuse axonal injury with Glasgow Coma Scale <6, or near hanging with anoxic brain injury.

7. When are nasal radiographs indicated?

Almost never. Nasal fractures typically are a clinical diagnosis without the need for routine radiographs. Physical examination may reveal swelling, angulation, bony crepitus, deformity, pain on palpation, epistaxis, and periorbital ecchymosis. Nasal radiographs are neither sensitive nor specific for fractures. The results do not alter management.

8. What is a septal hematoma? Why is it important?

All patients with nasal trauma and suspicion of a nasal fracture require inspection of the nasal septum for a septal hematoma. This is a collection of blood between the mucoperichondrium and the cartilage of the septum. It appears as a grape-like swelling over the nasal septum. If left undrained, it may result in septal abscess, necrosis of the nasal cartilage, and permanent deformity. If a septal hematoma is identified, incision and drainage is indicated in the ED, followed by nasal packing, antistaphylococcal antibiotics (prophylaxis for toxic shock syndrome), and prompt referral.

9. When should a consultation be obtained for a nasal fracture?

Most nasal fractures do not require immediate reduction unless there is significant deformity and malalignment. After anesthetizing the nose with lidocaine or tetracaine-soaked gauze or pledgets, early reduction of an angulated fracture is performed by exerting firm, quick pressure toward the midline with both thumbs. Patients should be referred to a maxillofacial or plastic surgeon for follow-up in 4 to 7 days. Immediate consultation is suggested for nasal fractures with associated facial fractures, cerebrospinal fluid rhinorrhea, and sustained epistaxis.

10. How is a frontal sinus fracture diagnosed?

Frontal sinus fracture should be suspected in any patient with a severe blow to the forehead. There is often an associated brain injury. The clinical signs include supraorbital nerve anesthesia, anosmia, cerebrospinal fluid rhinorrhea, subconjunctival hemorrhage, crepitus, and tenderness to palpation. The preferred diagnostic modality is computed tomography (CT) to determine if there is involvement of the anterior or posterior walls of the sinus or intracranial hemorrhage.

11. How are frontal sinus fractures treated?

After surgical consultation, patients with nondisplaced anterior wall fractures may be discharged on prophylactic antibiotics, with instructions to avoid Valsalva maneuvers and to follow up in 1 week with the surgical consultant. Patients with displaced anterior wall and sinus floor fractures require surgical consultation, admission, and antibiotic therapy.

Patients with posterior wall fractures require antibiotics and immediate neurosurgical consultation.

12. What are the classic zygoma fractures?

The zygoma is the third most commonly fractured facial bone (after the nose and mandible). Zygoma fractures are classified into three basic types:

- **Arch:** The bone may be fractured in one or two places and may be nondisplaced or displaced medially. Pain and trismus are caused by bony arch fragments abutting the coronoid process of the mandible. Because the masseter muscle originates on the zygoma, any movement causes further arch disruption. The fracture is diagnosed by the plain radiograph bucket-handle view (submentovertex).
- **Tripod:** Also termed a zygomaticomaxillary fracture, this is the most serious type of zygoma fracture and involves the infraorbital rim, the zygomaticofrontal suture, and the zygomaticotemporal suture. Clinical signs include deformity (flatness of the cheek), infraorbital nerve hypesthesia, inferior rectus muscle entrapment, and diplopia on upward gaze. Although these fractures may be detected on plain radiographs (Waters and Caldwell views), CT is necessary to better define the extent of the fracture. For these fractures, admission and consultation with a maxillofacial surgeon are required.
- **Body:** Fracture of the body of the zygoma, which involves the clinical signs and symptoms of the tripod fracture, results from severe force and leads to exaggerated malar depression.

13. What is the tongue blade test?

Patients with mandible fractures have mandibular tenderness and deformity, sublingual hematoma, and malocclusion on physical examination. The jaw appears asymmetric, with deviation toward the side of the fracture. The tongue blade test is performed by asking the patient to bite down on a tongue depressor. The tongue blade should be twisted by the examiner. If there is no fracture, the patient should be able to break the blade. In the presence of a mandible fracture, the patient opens his or her mouth and the tongue blade remains intact.

14. Which imaging studies should be ordered to diagnose a mandible fracture?

Mandible fractures are the second most common facial fracture. Multiple fractures are common (>50%) because of the ring structure of the bone. Always check for a second fracture. If available, the Panorex view is the most useful view for detecting mandible fractures. It provides a 180-degree view of the mandible and can detect fractures in all regions of the mandible, including symphyseal fractures that can be missed with the other views. If a Panorex radiograph is unavailable, the standard mandible series can be used. This comprises a posteroanterior view (for detecting fractures of the angle and body of the mandible), lateral and bilateral oblique views (for detection of rami fractures), Townes view (anteroposterior view that projects the rami and condyles), and often a submentovertex view. A condylar fracture may be missed by plain radiographs. If this fracture is suspected and the plain radiographs are negative, facial CT is indicated.

15. What are the most commonly fractured areas of the mandible?

The most commonly fractured areas are the body, the condyle, and the angle of the mandible.

16. What is the mechanism for a temporomandibular joint dislocation? How is it treated?

Temporomandibular joint dislocation can result from blunt trauma to the mandible, but it also can occur with exaggerated opening or closing of the jaw such as after a seizure or with yawning. Patients with a temporomandibular joint dislocation present with jaw deviation away from the side of the dislocation if it is a unilateral dislocation or with the mandible pushed forward (underbite) if it is a bilateral dislocation. After conscious sedation with benzodiazepine for masseter muscle relaxation and a narcotic for pain relief, the emergency physician should place gauze-wrapped thumbs on the posterior molars while standing above and behind the patient. The mandible is then pushed downward and posterior.

17. When is a CT scan indicated in the evaluation of maxillofacial trauma?

In patients with a history of facial trauma but with minimal physical findings consistent with fractures or an equivocal examination, plain radiography should be used as a screening test. The standard plain film series of the face includes a Waters (occipitomental) view, Caldwell (occipitofrontal) view, submentovertex view, and lateral view. The Waters view visualizes the orbital rim, infraorbital floor, maxilla, and maxillary sinuses and is useful as an initial examination in patients with suspected orbital floor fractures. Performance of this view requires that the cervical spine be clear because the patient is in the prone position. Fluid in the maxillary sinus is indirect evidence of fracture. The Caldwell view allows visualization of the superior orbital rim and the frontal sinuses. The lateral view shows the anterior wall of the frontal sinus and the anterior and posterior walls of the maxillary sinus.

In patients with physical findings that are highly suggestive of facial fractures (tenderness, step-offs, crepitus, or evidence of entrapment), some authors recommend proceeding directly to CT. This allows appropriate surgical planning. High-resolution, thin-cut CT scanning is the preferred modality for the elucidation of bony and soft-tissue injury in maxillofacial trauma. This is the preferred test in any patient with suspected tripod, orbital, or midface fractures. In patients with suspected orbital fractures, CT scan with coronal and axial sections should be ordered (2- to 3-mm cuts).

18. How do I recognize an injury to Stenson's duct?

Stenson's (parotid) duct arises from the parotid gland and courses from the level of the external auditory canal (superficial) through the buccinator muscle to open at the level of the upper second molar (Fig. 81-2). Any laceration at this level may involve the parotid gland, parotid duct, or buccal branch of the facial nerve. Laceration of the parotid system is

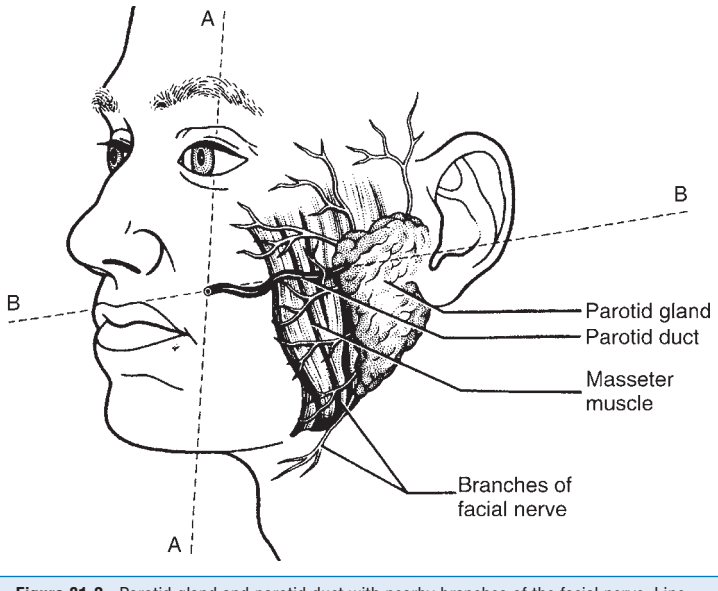

Figure 81-2. Parotid gland and parotid duct with nearby branches of the facial nerve. Line B demonstrates approximate course of parotid duct from parotid gland, entering mouth at junction of lines A and B. (From Cantrill SV: Face. In Marx JA, Hockberger RS, Walls RM, et al, editors: *Rosen's emergency medicine: concepts and clinical practice*, ed 5 St. Louis, 2002, Mosby, p 323.)

recognized by a flow of saliva from the wound or bloody drainage from the duct orifice. Careful exploration reveals whether the flow is from the parotid gland or duct. The buccal branch of the facial nerve travels in close proximity to Stenson's duct; injury leads to drooping of the upper lip, which indicates a possible parotid duct injury. To assess for parotid duct patency, the parotid gland should be milked to see if saliva is expressed from the intraoral opening of the parotid duct. Damage to the duct requires repair over a stent and plastic surgical consultation.

19. When should closure of a facial laceration be deferred?

Closure of facial lacerations in the ED depends on the severity of facial and systemic injuries. Complex lacerations in patients needing operative intervention should be cleansed with normal saline, covered with moist gauze, and deferred for intraoperative closure. Closure of the highly vascular tissues of the face may be delayed for up to 24 hours. Wounds involving the facial nerve, lacrimal duct, parotid duct, and avulsions should be referred on presentation to the appropriate surgeon for definitive care.

20. What deformity may arise from blunt trauma to the ear?

An acute auricular hematoma is a collection of blood separating the perichondrium from the underlying cartilaginous layer that may develop after a blow to the ear. If a hematoma is left undrained or an auricular compression dressing not applied after incision and drainage of a hematoma or repair of a pinna laceration secondary to blunt trauma to the ear, necrosis of the cartilage may develop with resultant deformity known as Cauliflower ear.

21. How is the ear anesthetized?

A subcutaneous circumferential injection of plain lidocaine should be placed at the base of the pinna. Lacerations in the external auditory require topical anesthesia with 4% lidocaine or local injection.

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CHAPTER 82

CERVICAL SPINE AND SPINAL CORD TRAUMA

Michael J. Klevens, MD, FAAEM, and Robert M. McNamara, MD, FAAEM

1. What is the annual incidence of spinal cord injury (SCI)?
Approximately 12,000 new cases per year. Of these causes, the percentage of violent causes has been increasing.

2. Name the most common causes of SCI.
- Vehicular crashes (42.1%)
- Falls (26.7%)
- Violence, primarily gunshot wounds (15.1%)
- Sports (7.6%)
- Other (8.6%)

3. What are the most common levels of injury?
The most frequent level of injury is C5, followed by C4, C6, C7, T12, and L1. Overall about half are cervical injuries.

4. In patients discharged from the hospital with neurologic impairment, what percentage has paraplegia and what percentage has tetraplegia (quadriplegia)?
- Incomplete tetraplegia (30.1%)
- Complete paraplegia (25.6%)
- Complete tetraplegia (20.4%)
- Incomplete paraplegia (18.5%)

One percent has complete recovery by discharge. An injury to one of the eight cervical vertebrae can cause tetraplegia. People with paraplegia have injuries to the thoracic, lumbar, or sacral regions. Over the past 15 years, the percentage of people with incomplete tetraplegia has increased slightly, while complete paraplegia has decreased slightly.

5. If most spinal injuries do not cause neurologic injury, why do I care?
The management of any spinal injury is important because improper care can result in permanent neurologic injury. It is important to have an index of suspicion for injury and proper immobilization and handling of the patient. It is imperative to have good-quality diagnostic imaging because inadequate studies and interpretation errors can lead to permanent injury.

6. What is the financial impact of an SCI?
Huge. The health care and living costs can vary greatly, depending on what severity of injury was sustained and the age of the individual at time of injury. The estimated yearly costs can range from $16,500 to $143,500. This does not include the first year costs, which range from $236,000 to $801,000. The lifetime costs can range from $704,000 to $3.1 million.

7. What is the average age of the person who sustains an SCI?
SCI is devastating in that it primarily affects young healthy adults. Since 2005, the average age at injury is 40.2 years. The predominant age range is between 16 and 30. An overwhelming percentage of injuries are suffered by males. There has been a slight trend toward older age recently, likely reflecting the preserved mobility of an aging population.

8. Name the causes of reduced life expectancy in SCI patients.
Pneumonia, pulmonary emboli, and sepsis.

9. Are there any underlying conditions that could precipitate or heighten the chance of an SCI?
Less force is required to cause fractures in the elderly. Rheumatoid arthritis can lead to subluxation problems at C1 and C2. Normal development of the odontoid may not occur in a Down syndrome patient. Osteoporosis and metastatic cancer may lead to a vertebral fracture with insignificant trauma.

10. How do I immobilize the potential spinal injury patient?
When any spinal injury is suspected, initially, the entire spine should be immobilized with a long board. The cervical spine is immobilized with a hard collar. There are several different types of stiff cervical collars, such as the Philadelphia or Ambu brand collars. The emergency medical service (EMS) stabilization may also include the forehead taped to the board and accessory towel, sandbag, or other bolsters to prevent further neck movement. The sandbag and tape method without a rigid collar support should not be used. It is interesting that total spinal immobilization has never been proven to prevent secondary spinal injury. However, there continue to be sporadic case reports of such injuries. Therefore, it is prudent to follow these initial guidelines. It is recommended that the patient be removed from the long board as soon as possible to prevent soft tissue injury.

11. How should one approach the patient with potential spinal injury?
There are several mnemonics for the initial stabilization of any trauma patient. Advanced trauma life support (ATLS) teaches the **ABCDE** mnemonic:

A = **A**irway
B = **B**reathing
C = **C**irculation
D = **D**isability
E = **E**xposure

According to another mnemonic, a proper history is **A MUST:**

A = **A**ltered mental state. Check for drugs or alcohol.
M = **M**echanism.* Does the potential for injury exist?
U = **U**nderlying conditions. Are high-risk factors for fractures present?
S = **S**ymptoms. Is pain, paresthesia, or neurologic compromise part of the picture?
T = **T**iming. When did the symptoms begin in relation to the event?

*Because fall injuries are common, the physician should get information as to the height of the fall and any preceding events such as syncope, chest pain, or seizure.

12. What should be assessed on physical examination?
There are two key areas: the spine itself and the neurologic examination. The spine is palpated to assess for tenderness, deformity/step-off, and muscle spasm. It is important to understand that the examiner feels only the posterior elements of the vertebrae; therefore, a fracture may be present despite a lack of tenderness. The neurologic examination should include motor function, sensory function, some aspect of posterior column function (position and vibration), and a rectal examination. Regarding sensory testing, the assessment of light touch examines the integrity of the posterior neurological column while pinprick testing assesses the anterior column. In an unconscious patient, the only clues may be poor rectal tone, priapism, absence of deep tendon reflexes, or diaphragmatic breathing.

13. What is neurogenic shock? How is it treated?
Neurogenic shock is a syndrome with loss of neurologic function and accompanying autonomic tone. This is usually exhibited by flaccid paralysis with loss of reflexes and loss of urinary and rectal tone. Accompanying this are bradycardia, hypotension, hypothermia, and ileus. The diagnosis of neurogenic shock should only be made after all other forms of

shock have been eliminated. Hypotension is usually successfully treated with rapid infusion of crystalloid. If intravenous fluids are not adequate to maintain organ perfusion, the use of dopamine or phenylephrine may be beneficial. Bradycardia can be treated with atropine or dopamine. In refractory bradycardia, a pacemaker may be required. In most cases of neurogenic shock, hypotension resolves within 24 to 48 hours.

14. What are the general principles of emergency treatment in the patient with spinal cord trauma?

First, do no harm. As stated previously, that means proper immobilization and coordinated movement of the patient, when absolutely necessary. A higher level of cervical injury results in a more devastating injury to the patient. Any patient with an injury above C5 probably should be intubated because the phrenic nerve roots, which supply the diaphragm, emerge from C3 to C5. Rapid-sequence intubation (RSI) oral-tracheal intubation with manual in-line cervical-spine stabilization is considered the safest way to intubate these patients. Early gastric and bladder decompression are also indicated. Overhydration should be avoided so as to not cause pulmonary edema. The absence of pain below the level of injury can mask other injuries. The patient with neurologic deficit faces a difficult hospital stay, and the ED should use full sterile precautions for any procedure such as urinary catheters or central venous access when possible.

15. How do I determine which patients need spine radiographs?

There are two validated decision rules available to the emergency physician. One is the National Emergency X-Radiography Utilization Study (NEXUS) decision rule. The other is the Canadian C-Spine Rule (CCR). Both have been shown to reduce the number of radiographs necessary to identify important cervical-spine injury.

16. What are the NEXUS criteria?

- No midline cervical tenderness
- No focal neurologic deficit
- Normal alertness
- No intoxication
- No painful distracting injury

If patients meet the criteria, there is a high likelihood that they have a low probability of injury and that cervical radiography is not needed. In this large multicenter study, the overall rate of missed cervical spine injuries was less than 1 in 4,000 patients. Note that there are specific definitions of the previous five criteria that must be reviewed when applying the NEXUS criteria. This rule was 99.6% sensitive and 12.9% specific for significant injury.

17. What is the CCR?

The CCR asks three questions:

- Is there any high-risk factor that mandates radiography? These are:
  a. age $>$ 65
  b. significant mechanism of injury (i.e., fall from $>$1 m, axial loading injury, high speed MVA/rollover/ejection, bike collision)
  c. the presence of paresthesias
- Can the patient be assessed safely for range of motion (simple mechanism, sitting position in the ED. ambulatory at any time, delayed onset of neck pain, or absence of midline cervical spine tenderness)?
- Can the patient actively rotate the neck 45 degrees to the left and the right?

This study had a sensitivity of 100% and a specificity of 42.5% for identifying clinically important cervical-spine injuries. Again, one needs to review the rule completely before applying it to patients.

18. What are distracting injuries?

NEXUS: includes a long list such as long bone fractures, large lacerations, visceral injury, and burns

CCR: injuries such as fractures that are so severely painful that the neck examination is unreliable.

19. Can these decision rules be applied to children?

It is difficult to validate the NEXUS or CCR in children because there has been a paucity of studies in this population. Additionally, some of the criteria in the CCR and NEXUS are difficult to verify in toddlers and children because of their inherent immaturity and unpredictability.

20. Which type of radiographs should be obtained?

The standard three-view of the cervical spine is an anteroposterior (AP), lateral, and open-mouth (odontoid). During the initial evaluation, a cross-table lateral radiograph should be taken because the patient does not have to move. It is extremely important that cervical-spine precautions not be discontinued based solely on the cross-table lateral. Some studies have reported that up to 18% of cervical-spine injuries are missed with the cross-table lateral radiograph alone. The most commonly missed injuries are at C1 to C2, followed by the lower C6-C7-T1 junction. In the elderly, C1 and C2 fractures account for approximately 70% of cervical-spine fractures.

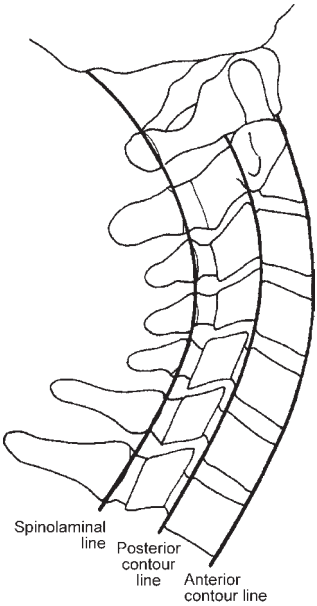

Figure 82-1. Curve of alignment.

21. How do I interpret the lateral cervical spine radiograph?

The first rule is to make sure that the radiograph is technically adequate and that the top of T1 is visible on the film. Next, follow the mnemonic **ABCS:**

- **A** = **A**lignment. Check for a smooth line at the anterior and posterior aspect of the vertebral bodies and the spinolaminar line from C1 to T1 (Fig. 82-1)
- **B** = **B**ones. Check each vertebral body to ensure that the anterior and posterior heights are similar (>3 mm difference suggests fracture); follow the vertebrae out to the laminae and spinous process. Look carefully at the upper and lower cervical segments where fractures are likely to be missed. Examine the ring of C2, which can show a fracture through the upper portion of the vertebral body of C2.
- **C** = **C**artilage. Check the intervertebral joint spaces and the facet joints.
- **S** = **S**oft-tissue spaces. Look for prevertebral swelling, especially at the C2 to C3 area (> 5 mm) and check the predental space (Fig. 82-2), which should be less than 3 mm in adults and less than 5 mm in children. From C4 to C7 the soft-tissue thickness should not be greater than 22 mm.

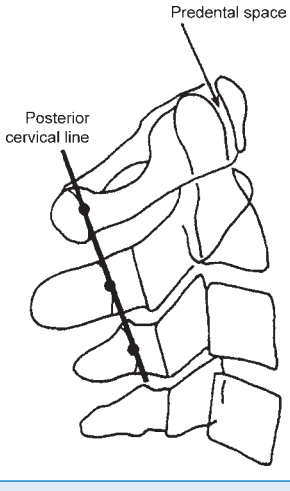

Figure 82-2. Posterior cervical line and predental space.

22. What are the indications for flexion-extension views of the cervical spine?

Based on the NEXUS study, flexion-extension imaging is unnecessary in the acute evaluation of patients

with blunt trauma. A computed tomography (CT) or magnetic resonance imaging (MRI) would give more information in the presence of specific clinical concerns for ligamentous damage or fracture not diagnosed on plain films.

23. When would a CT or MRI be ordered?

Routine indications are when plain radiographs are inconclusive or difficult to interpret and you have suspicion of spinal injury. A CT is good for detection of bony injury and to identify surgical conditions such as hematoma or disk fragments within the spinal canal. With the advent of rapid helical scanning, many centers are increasing their use of the CT scan in place of plain radiographs, especially if the patient has an indication for a head CT. A CT scan is also needed for the clearance of the cervical spine in comatose or obtunded patient. In a study of patients with traumatic brain injury, 5.4% of the patients had C1 or C2 fractures and 4% had occipital condyle fractures that were not visualized on the three-view radiography series. An MRI is useful to identify injury to the spinal cord itself in the face of neurologic deficit. The MRI can show areas of contusion and edema within the spinal space. An MRI can also detect rupture of intervertebral disks and ligamentous injury. CT is better than MRI for the identification of vertebral fractures.

24. What is SCIWORA?

It is **S**pinal **C**ord **I**njury **W**ithout **R**adiographic **A**bnormalities. Children are more susceptible to SCIWORA because of the greater elasticity of their cervical structures. This leads to transient spinal column subluxation and stretching of the spinal cord. These pediatric patients may have a brief episode of upper extremity weakness or paresthesias with delayed development of neurologic deficits that appear hours to days later. MRI should be obtained on all patients with SCIWORA.

KEY POINTS: SPINAL CORD TRAUMA

1. It is important to have a high index of suspicion for injury and ensure proper immobilization and handling of the patient.
2. Use clinical decision rules such as NEXUS or Canadian C-spine Rule to minimize cervical spine radiographs.
3. The use of steroids in blunt spinal cord trauma is not standard of care.
4. Special populations, such as pediatric and geriatric, need a more thorough work-up than the normal adult.
5. The three-view c-spine series is mandatory in evaluations if you are getting plain radiographs, whereas the flexion-extension view is not needed in the acute setting.

25. Describe the Jefferson, Hangman, Clay Shoveler, and Chance fractures.

- **Jefferson fracture** is a burst fracture of the ring of C1 that occurs from axial loading.
- **Hangman fracture** is a disruption of the posterior arch of C2.
- **Clay-Shoveler fracture** is a fracture of the spinous process that is classically caused by forceful cervical extension.
- **Chance fracture** is a vertebral fracture, usually in the lumbar segment, involving the posterior spinous process, pedicles, and vertebral body. It is caused by the flexion forces on the spinal column. This is associated with the use of lap belts.

26. Describe the incomplete cord syndromes or injuries.

- **Anterior cord syndrome** results in loss of function in the anterior two thirds of the spinal cord from damage to the corticospinal and spinothalamic pathways. Findings include loss of voluntary motor function and pain and temperature sensation below the level of the

injury, with preservation of the posterior column functions of position and function. The key issue is the potential reversibility of this lesion if a compressing hematoma or disk fragment can be removed. This condition requires immediate neurosurgical evaluation.

- **Central cord syndrome** results from injury to the central portion of the spinal cord. Because more proximal innervation is placed centrally within the cord, this lesion results in greater involvement of the upper extremities than of the lower extremities. Bowel or bladder control is usually preserved. The mechanism of injury is hyperextension of a cervical spine with a cord space narrowed by congenital variation, degenerative spurring, or hypertrophic ligaments. This syndrome can occur without actual fracture or ligamentous disruption.
- **Brown-Séquard syndrome** is a hemisection of the spinal cord, usually from penetrating trauma. Contralateral sensation of pain and temperature is lost, and motor and posterior column functions are absent on the side of the injury.
- **Cauda equina syndrome** is an injury to the lumbar, sacral, and coccygeal nerve roots causing a peripheral nerve injury. There can be motor and sensory loss in the lower extremities, bowel and bladder dysfunction, and loss of pain sensation at the perineum (saddle anesthesia).

27. What is the significance of sacral sparing and spinal shock?

Sacral sparing refers to the preservation of any function of the sacral roots, such as toe movement or perianal sensation. If sacral sparing is present, the chance of functional neurologic recovery is good. Spinal shock is a temporary concussive-like condition in which cord-mediated reflexes, such as the anal wink, are absent. Spinal shock also may result in bradycardia and hypotension. The extent of cord injury—and prognosis—cannot be determined until these reflexes return.

28. What can physicians do to prevent spinal injuries?

Get involved in injury prevention and education. Due to the predominance of vehicle crashes causing SCIs, one can work to reduce driving under the influence of alcohol and drugs. This includes using cellphones or texting while driving. Further, the use of safety belts should be emphasized at discharge in every ED visit, regardless of the reason the person came in for treatment. Diving and sporting injuries can be reduced by proper public education and coaching.

CONTROVERSY

29. What is the status of steroids in spinal cord trauma?

This has been a very controversial topic. In 1975, the first National Acute Spinal Cord Injury Study (NASCIS) was established. This was followed by NASCIS 2 and NASCIS 3, which was completed in 1998. The dosage of methylprednisolone was an initial bolus of 30 mg/kg intravenously over 15 minutes, followed 45 minutes later by a continuous infusion of 5.4 mg/kg/hr for 23 hours. This was given within 3 hours of injury. When the therapy was initiated 3 to 8 hours after injury, patients were maintained on an infusion for 47 hours. Initial support for the use of steroids was encouraging, but multiple reviews of the NASCIS study and other literature have shown that there is insufficient evidence to support the use of corticosteroids in the treatment of patients with acute SCI. Many believe that there were study design, data presentation, interpretation, and analysis flaws in the NASCIS study. Further, a recent Level I study showed that patients treated with methylprednisolone had a higher incidence of complications, such as gastrointestinal (GI) bleed and respiratory complications. Unfortunately, the overwhelming desire for any improvement has made steroid therapy a de facto standard of care at many institutions. This conflicts with recent position statements by the American Academy of Emergency Medicine, Canadian Association of Emergency Physicians, American Academy of Neurological Surgeons, and the Congress of Neurological Surgeons. In conclusion, the use of steroids is an option but is certainly not a standard of care.

WEBSITES

1. American Academy of Emergency Medicine: www.aaem.org/positionstatements/steroidsinacuteinjury.shtml
2. Canadian Association of Emergency Physicians: www.caep.ca/
3. Eastern Association for the Surgery of Trauma: www.east.org
4. National Spinal Cord Injury Statistical Center: www.spinalcord.uab.edu

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HEAD TRAUMA

Edward Newton, MD

CHAPTER 83

1. What is the scope of head injury in the United States?

There are more than 1.1 million ED visits and approximately 70,000 deaths as a result of head injury every year in the United States. Although the incidence of severe head injury is decreasing, most likely owing to the preventive benefits of helmets and seat belts and air bags in automobiles, head trauma remains the most lethal traumatic injury and accounts for a large proportion of patients with permanent disability. The peak incidence of head injury is in the 15- to 24-year-old age group, with males affected twice as often as females. The spectrum of head injury includes relatively minor problems, such as lacerations and scalp contusions, and major, often lethal, intracranial trauma. Distinguishing between minor and potentially lethal head injuries while using diagnostic resources appropriately is one of the most difficult tasks facing the emergency physician.

2. What groups of patients are at particular risk from head trauma?

Because assessment of mental status is such an integral part of the evaluation of head-injured patients, patients who are unable to communicate because they are preverbal (e.g., infants), intoxicated, mentally impaired, aphasic, or have a language barrier pose a special challenge. When such communication barriers are present, there should be a lower threshold for obtaining a computed tomography (CT) scan.

Certain age groups are at higher risk for intracranial injury:

- **Infants** are at higher risk because of their relatively large head size and compressibility of the skull. Infants also are at high risk for nonaccidental trauma (e.g., **abusive head trauma**, which is also known as **shaken baby syndrome**), in which case an accurate history may be unavailable or deliberately withheld. If the cranial sutures and fontanelles are not closed, the cranium can expand as a result of intracranial bleeding. Infants can bleed sufficiently intracranially to produce hemorrhagic shock, whereas in older children and adults, another source of bleeding is inevitably responsible for shock.
- The **elderly** also are at higher risk of intracranial injury, particularly subdural hematoma (SDH). Cerebral atrophy results in stretching of bridging veins from the dura to the brain parenchyma, making these veins vulnerable to tearing from deceleration forces.
- **Chronic alcoholics** are at risk because of their greater frequency of head trauma, cerebral atrophy, and coagulopathy.
- Patients who are taking anticoagulants or antiplatelet agents or who have intrinsic **bleeding diatheses** bleed more actively than patients with normal coagulation and have higher mortality from brain injury.

KEY POINTS: PATIENTS AT HIGH RISK FOR HEAD INJURY

1. Very young and very old patients
2. Chronic alcoholics
3. Patients with coagulopathy

3. What is a cerebral concussion?

Sudden, transient loss of central neurologic function secondary to trauma. It is characterized by loss of consciousness, transient amnesia, confusion, disorientation, or transient visual changes, without any gross cerebral abnormalities or neurological deficits on examination.

4. What is the postconcussive syndrome?

Although the patient may have a completely normal neurologic examination after a concussion, there are common sequelae from this type of injury. Patients frequently report migraine-type headaches, dizziness, inability to concentrate, and irritability. Although in 90% of cases these symptoms resolve within 2 weeks, they rarely may persist for up to 1 year. Treatment is supportive, and the long-term prognosis is good. A phenomenon known as the **second impact syndrome** is recognized in which a second head trauma during a vulnerable period after a concussion results in severe and often fatal diffuse cerebral edema. Consequently, athletes should be held out of contact sports until all postconcussive symptoms have resolved.

5. How do you detect cerebrospinal fluid (CSF) leaks caused by basilar skull fractures?

A patient with signs of basilar skull fracture (i.e., raccoon eyes, hemotympanum, or Battle sign) with clear drainage from the nose or ear canal should be suspected of having a CSF leak. Analysis of the glucose content of the drainage by Glucometer or laboratory analysis may distinguish CSF (containing 60% of serum glucose levels) from nasal mucus (glucose not present). In cases in which blood is mixed with CSF, applying a drop of the fluid to filter paper reveals CSF in a target shape with blood at the center and pink-tinged CSF forming an outer ring. However, bedside tests are neither specific nor sensitive for detecting CSF leaks.

6. How are CSF leaks treated?

CSF leaks through tears in the dura generally are managed conservatively. The use of prophylactic antibiotics is controversial because they have not been shown to significantly reduce the incidence of meningitis and may select for antibiotic-resistant bacteria. Patients must be followed closely until the dural tear heals because of the risk of meningitis. Dural tears that fail to close spontaneously over 2 to 3 weeks usually require operative repair.

7. How does a patient with epidural hematoma present?

Epidural hematoma occurs in 5% to 10% of severe head injuries. In the classic pattern, a patient loses consciousness from the initial concussion, gradually recovers over a few minutes, and enters a lucid interval wherein he or she is relatively asymptomatic and has a normal neurologic examination. During this interval, accumulation of arterial blood in the epidural space, usually from a lacerated middle meningeal artery, eventually causes compression and shift of brain across the midline. This process is accompanied by a second reduction in the level of consciousness and the pupillary and motor signs of herniation. This classic pattern occurs in only about 30% of cases, however. Many patients remain unconscious after the initial impact or with minor hemorrhages, may not develop increased intracranial pressure (ICP) at all. The characteristic CT scan appearance of an epidural hematoma is a hyperdense lenticular collection of blood that indents adjacent brain parenchyma and does not extend beyond cranial sutures where the dura is attached.

8. How does a subdural hematoma (SDH) present?

SDH may be acute, subacute (6–14 days), or chronic (>14 days after trauma).

- **Acute SDH** is associated with a high incidence of underlying brain injury. The presentation varies with the severity of the underlying injury, but patients commonly present with a diminished level of consciousness, headache, and focal neurologic deficits corresponding to the area of brain injury. If sufficient bleeding occurs, ICP increases and herniation may occur. The characteristic appearance of an acute SDH on CT scan is a collection of hyperdense blood in a crescent-shaped pattern conforming to the convexity of the hemisphere and often extending past cranial sutures. (Fig. 83-1) At times, the injury causes

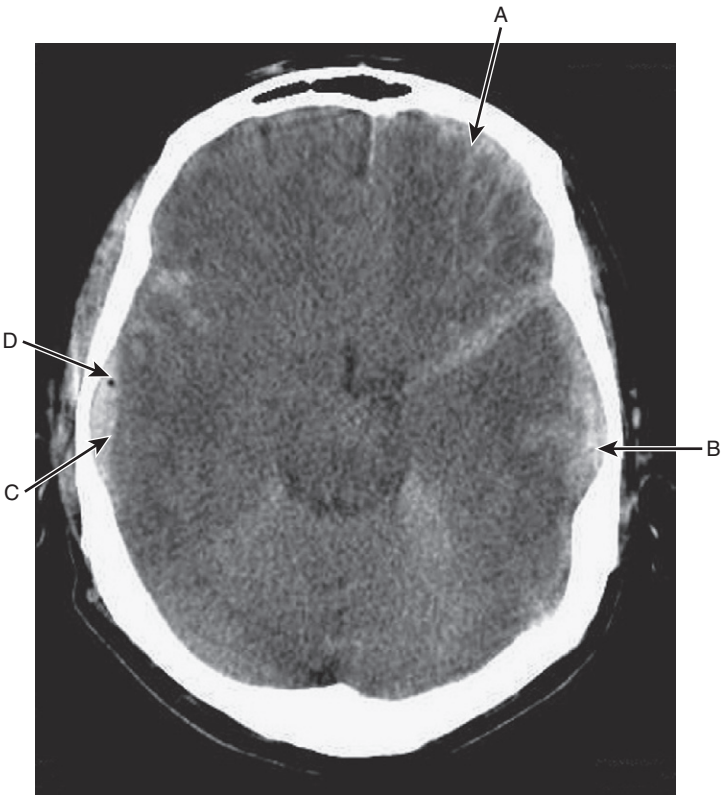

Figure 83-1. Computed tomographic scan of the head showing *A,* Subarachnoid hemorrhage. *B,* Subdural hemorrhage. *C,* Subdural hemorrhage with *D,* intracranial free air (*black dot*).

a minimal amount of bleeding and the patient does not immediately seek medical care. The SDH undergoes lysis over a period of several days and eventually organizes into an encapsulated mass.

- **Subacute** or **chronic SDH** is a difficult clinical diagnosis because the symptoms are vague and common (e.g., persistent headache, difficulty concentrating, lethargy), and the trauma may have been forgotten. Even the CT scan diagnosis is difficult because subacute SDH becomes isodense and indistinguishable from surrounding brain unless special contrast-enhanced CT techniques are used. Chronic SDH appears as an encapsulated lucent collection of fluid in the same position as the acute type.

9. What is axonal shear injury?

Axonal shear injury occurs during abrupt deceleration because white and gray matter have different densities and different rates of deceleration. This produces a shearing force that may tear axons at the white-gray interface, resulting in severe neurologic derangements such as prolonged coma or persistent vegetative state. The CT scan may appear completely normal or show only small petechial hemorrhages. Magnetic resonance imaging (MRI) of the brain is a more sensitive tool in detecting these injuries but is currently impractical in the acute phase.

10. What is brain herniation?

Herniation is caused by increased ICP. Because the cranium is a rigid structure, pressure varies with the volume of its contents. Approximately 10% of intracranial volume is blood, another 10% is CSF, and the remainder is brain parenchyma and intracellular fluid. An increase in any of these compartments by blood, tumor, or edema causes a predictable response. Initially, CSF is forced into the spinal canal, and the ventricles and cisterns collapse. Once this has occurred, ICP rises steeply, and the brain parenchyma shifts away from the accumulating blood and herniates through one of several spaces, eventually causing death by compressing the brainstem.

11. List the four types of herniation syndrome.

- Uncal herniation
- Central herniation
- Cingulate herniation
- Posterior fossa cingulation

12. Describe uncal herniation syndrome.

The uncus is the most medial portion of the hemisphere and is often the first structure to shift below the tentorium that separates the hemispheres from the midbrain. As the uncus is forced medially and downward, the ipsilateral third cranial nerve is compressed, producing pupillary dilation, ptosis, and oculomotor paresis. As herniation progresses, the ipsilateral cerebral peduncle and pyramidal tract are compressed, resulting in contralateral hemiplegia.

In approximately 10% of cases, the hemiparesis occurs on the same side as the brain lesion, making this a less reliable finding for localizing the injury. Further progression results in brainstem compression with respiratory and cardiac arrest. Transtentorial herniation of this type is the most common variety.

13. What is central herniation syndrome?

Occasionally, hematomas located at the vertex or frontal lobes cause simultaneous downward herniation of both hemispheres through the tentorium. Clinical findings are similar to uncal herniation, except that bilateral motor weakness occurs.

14. How does cingulate herniation occur?

Rarely, the cingulate gyrus is forced medially beneath the falx by an expanding lateral hematoma, causing compression of the ventricles and impairing cerebral blood flow.

15. Explain posterior fossa herniation.

Bleeding or edema in the posterior fossa can result in herniation of the cerebellar tonsils either upward through the tentorium or downward through the foramen magnum. In the latter case, coma and fatal brainstem dysfunction may occur rapidly and with little warning.

16. What is the ED treatment for increased ICP?

- **Maintain adequate cerebral perfusion pressure.** Although there is often misguided reluctance to hydrate vigorously patients with concomitant head and systemic injuries, cerebral perfusion must be maintained for resuscitation to be successful. Hypotension must be avoided, and often laparotomy to correct intraabdominal bleeding must take precedence over neurosurgical intervention to maintain cerebral perfusion. Patients who experience hypotension (systolic blood pressure $<$ 90 mm Hg) have a twofold increase in mortality.
- **Avoid secondary injuries to the central nervous system.** After brain trauma, there is a cascade of secondary neuronal metabolic injuries that are detrimental to recovery of neurological function. At present, few interventions have proved effective in limiting these changes. Certain other treatable conditions either increase the metabolic demands of the brain or decrease cerebral perfusion and worsen the prognosis unless they are corrected. The **5 Hs** (**hypotension, hypoxia, hypercarbia, hypoglycemia**, and **hyperthermia**) and **seizures** are conditions that should be avoided or corrected in the ED. Anticonvulsant

prophylaxis with diphenylhydantoin or levetiracetam is indicated particularly for penetrating injuries and depressed skull fractures. It is crucial to avoid hypoxia as well because head-injured patients who experience hypoxia ($pO_2 < 60$ mm Hg) also have a twofold increase in mortality. Consequently, early and careful airway management and ventilation are essential. Coagulopathies should be corrected with fresh frozen plasma, and platelet transfusion should be considered in patients who have recently taken aspirin or other antiplatelet drugs.

- **Hyperventilation.** Carbon dioxide is one of the main determinants of cerebrovascular tone. High levels produce cerebral vasodilation; low levels cause vasoconstriction. Hyperventilation to a PCO_2 level of 25 mm Hg decreases blood flow to the brain by 50%, which decreases the vascular compartment of the brain and may "buy some time" for definitive surgical interventions. When blood flow to the brain decreases, delivery of oxygen and glucose also decreases, resulting in ischemic injury and worse edema. The optimal level of hypocarbia is uncertain at present, but most clinicians recommend moderate short-term hyperventilation, with a PCO_2 level no less than 30 to 35 mm Hg as the goal in patients with evidence of herniation. To accomplish this degree of hypocarbia, it is necessary to intubate the patient with rapid-sequence intubation and mechanically ventilate with settings determined by arterial blood gases to maintain the PCO_2 between 30 and 35 mm Hg. The indication for implementing hyperventilation is increased ICP resulting in clinical signs of focal neurologic deficit (i.e., herniation). Hyperventilation is not used prophylactically but is reserved for patients with elevated ICP and rapid clinical deterioration.
- **Diuresis.** The use of an osmotic diuretic, such as mannitol, 0.5 to 1.0 g/kg intravenously over 15 minutes, or a loop diuretic, such as furosemide, 0.5 to 1.0 mg/kg intravenously, is effective in reducing brain edema. Infusion of mannitol creates an osmotic gradient between the intravascular space and the extracellular fluid, drawing fluid from the extracellular fluid and reducing brain water content and ICP. Mannitol is filtered by the kidneys, producing systemic dehydration. Clinical experience and animal studies seem to support the concomitant administration of osmotic diuretics and volume resuscitation in patients with hypovolemic shock.
- **Hypertonic saline.** Various concentrations of hypertonic saline ranging from 3% to 23% have been used to simultaneously decrease brain edema, maintain cerebral perfusion pressure, and restore systemic volume. It has been shown to be at least as effective as mannitol in treating elevated ICP. Patients receiving hypertonic saline will develop significant hypernatremia and hyperosmolarity. Unless serum sodium exceeds 160 mEq/L, these abnormalities should be allowed to correct themselves gradually over a period of several days.
- **Ventriculostomy.** Although generally an intensive care unit (ICU) technique, removal of CSF through a ventriculostomy is occasionally implemented in the ED and is perhaps the most effective way of rapidly lowering ICP.
- **Barbiturates.** Conscious patients who are paralyzed for intubation also must be sedated. A short-acting barbiturate such as thiopental is the ideal agent for this purpose because it lowers ICP, prevents seizures, and decreases cerebral metabolism. Such agents cannot be used in a hypotensive patient, however. In these cases, a reversible agent, such as morphine, 0.1 mg/kg; lorazepam, 0.01 mg/kg; or midazolam, 1 mg/kg/h, is preferred because adverse effects on blood pressure and cardiac output can be reversed by specific antagonists. Etomidate, 0.2 mg/kg, is a short-acting agent that decreases ICP without adversely affecting cardiac output, cerebral perfusion pressure, and systemic blood pressure and can be used for sedation, although suppression of adrenal function is a known complication. Fentanyl, 3 to 5 mcg/kg, causes a slight increase in ICP and is not the preferred agent for sedation of a head-injured patient.
- **Therapeutic hypothermia.** Reducing a patient's body temperature to 32°C to 33°C for 24 to 48 hours has shown some benefit in preserving neurological function in cardiac arrest survivors and it was hoped that it would show the same benefits in brain-injured patients.

However, the results of several trials have been conflicting. If any benefit occurs, it is likely in those who present with a Glasgow Coma Scale(GCS) 5 to 8, but even in these patients the treatment should be considered experimental. Fever should be treated aggressively, however, and patients who arrive in the ED with mild hypothermia should be allowed to passively rewarm.

KEY POINTS: TREATMENT OF HEAD INJURY

1. Maintain cerebral perfusion and avoid hypotension.
2. Maintain oxygenation.
3. Secure airway using RSI if the GCS is less than 8.
4. Seizure prophylaxis with diphenylhydantoin (15 mg/kg IV).
5. Hyperventilate to $pCO_2 = 30$ to 35 mm Hg only if patient has elevated ICP and is clinically herniating.
6. Osmotic therapy using either mannitol or hypertonic saline.
7. Correct coagulopathy.

17. If a patient has a normal CT scan after head trauma, is it completely safe to discharge him or her home?

Nothing is completely safe. There are well-documented instances of delayed epidural and subdural bleeding many hours after injury. Consequently, although it is generally safe to discharge such patients, head injury instructions should be given to responsible family members, and the patient should be instructed to return immediately if symptoms worsen. If the patient is socially isolated or unreliable, a judgment has to be made regarding the seriousness of the mechanism of injury and the risk of discharge. Intoxicated patients should be kept under observation until their mental status can be evaluated properly.

18. Because CT scan is available, is there any role for plain skull films?

The usefulness of plain radiographs of the skull has been far outstripped by more informative imaging modalities such as CT and MRI. Skull films still have certain indications in evaluation of the following:

- Penetrating trauma (gunshot wounds)
- Suspected depressed skull fracture
- Suspected basilar skull fracture
- As part of the skeletal survey for suspected child abuse
- In patients with prior craniotomies or shunts

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TRAUMATIC OPHTHALMOLOGIC EMERGENCIES

CHAPTER 84

Harold Thomas, MD

1. **Name the two most time-critical emergencies in ophthalmology.**
   Central retinal artery occlusion and chemical burns to the eyes.

2. **What is the treatment for a chemical burn of the eye?**
   Immediate copious irrigation of the eyes (for at least 20 minutes). Irrigation should be initiated before transport to the ED.

3. **How do you know when you have irrigated the eye enough?**
   Nitrazine paper can be used to ensure that the pH has been corrected to normal. This usually requires at least 3 L of normal saline in each eye and continuous irrigation for 20 minutes. Alkalis, which cause the most damaging burns, tend to adhere to the tissue of the eye and are difficult to remove completely with irrigation. After irrigation, emergent ophthalmologic consultation is indicated.

4. **What is the significance of pain from an eye injury that is not relieved with topical anesthesia?**
   Complete symptomatic relief with topical anesthesia indicates a superficial injury involving only the cornea. If a patient still has significant pain after application of anesthetic drops, a deeper injury (often traumatic iritis) must be suspected, even in the presence of an obvious superficial injury.

5. **List nine potential injuries that must be considered in a patient sustaining a blunt injury to the eye.**
   - Blow-out fracture of the floor of the orbit
   - Corneal abrasion
   - Anterior hyphema
   - Lens dislocation
   - Traumatic mydriasis
   - Vitreous hemorrhage
   - Retinal detachment
   - Traumatic iritis
   - Ruptured globe (rare after blunt injuries)

6. **What is the most common eye injury seen in the ED?**
   Corneal abrasion with or without a superficial foreign body.

7. **How is corneal abrasion diagnosed?**
   The anesthetized eye can be stained with fluorescein and illuminated by an ultraviolet or Wood lamp; corneal defects fluoresce bright yellow-orange. Visual acuity should be checked, and the eye should be inspected, with particular emphasis on the anterior chamber to look for an anterior hyphema.

8. **What is the treatment for a corneal abrasion?**
   Because this injury is extremely painful, narcotic analgesics are indicated. Sending a patient home with topical anesthesia is controversial; most recommend against it. One frequently

overlooked aspect of therapy is the instillation of a cycloplegic agent, usually cyclopentolate (Cyclogyl), to relieve the ciliary spasm that commonly accompanies this injury. Patients also need evaluation for tetanus prophylaxis. Most should receive topical antibiotics, drops, or ointment. Nonsteroidal anti-inflammatory eye drops have also been proven to be useful.

9. What is the role of an eye patch in treatment of corneal abrasions?

A pressure patch previously was considered the most important aspect of management of a corneal abrasion. Patches were thought to increase comfort and hasten healing. It is now known that not only are eye patches uncomfortable but also they do not increase healing and may promote infection. They do *not* prevent the involved eye from moving and should not be used for most superficial corneal abrasions. If you do use a patch, be sure to instruct the patient not to drive because depth perception depends on binocular vision.

10. How does a corneal abrasion from a contact lens differ from other causes of corneal trauma?

Corneal abrasions secondary to overuse of contact lenses are much more likely to have a bacterial process involved, often *Pseudomonas*. These patients should receive topical antibiotics effective against *Pseudomonas* organisms (tobramycin or gentamicin) and should never be patched. If the emergency physician is unable to do a slit-lamp examination, early ophthalmologic referral to rule out ulcerative keratitis (corneal ulcer) is indicated.

11. What is the most common location of an ocular foreign body?

Foreign bodies are often lodged just beneath the upper eyelid along the palpebral conjunctiva. The eyelid needs to be everted with a cotton swab to examine this area adequately. Conjunctival foreign bodies should be suspected when many vertical linear streaks are noted on the cornea with fluorescein examination.

12. What is the proper treatment for a corneal foreign body?

First, topical anesthesia is applied, usually proparacaine. Nonembedded foreign bodies should be removed with a sterile, moist cotton swab. Embedded foreign bodies are removed with a 27-gauge needle or an eye spud. Most metallic foreign bodies leave a residual rust ring that should be removed in approximately 24 hours, after the cornea has softened.

13. What is an anterior hyphema?

A collection of blood in the anterior chamber of the eye; it is seen as a layering of cells that pool along the bottom of the eye when the patient is sitting upright. When the patient is lying down, a hyphema is not recognized easily; it may appear as a diffuse haziness of the anterior chamber. Small hyphemas, termed **microhyphemas**, may be identified only with a slit lamp.

14. How is an anterior hyphema treated?

The standard in the past was to admit all patients for bed rest; today the dominant tendency is toward outpatient management. The patient should be kept upright, the eye patched, and ophthalmologic consultation initiated, at least by phone. Complications include rebleeding, glaucoma formation (particularly in patients with sickle-cell trait), and corneal staining.

15. What physical findings lead to the suspicion of a blow-out fracture?

Classic findings with a blow-out fracture (fracture of the inferior orbital wall with herniation of the global contents into the maxillary sinus) are (1) decreased sensation over the inferior orbital rim, extending to the edge of the nose and ipsilateral upper lip, secondary to compromise of the inferior orbital nerve; (2) enophthalmos, or a sunken appearance of the eye, which may be masked by edema; and (3) paralysis or limitation of upward gaze (manifested as diplopia), resulting from entrapment of the inferior rectus muscle.

16. What is traumatic mydriasis?

An efferent pupillary defect manifested by a dilated (in most instances irregular) pupil that does not react to direct or consensual light, usually as a result of minor trauma to the eye.

Because such a patient is at risk for other more serious eye injuries, a careful eye examination is mandatory. The possibility of uncal herniation secondary to intracranial injury should be considered if level of consciousness is decreased in the presence of a perfectly round, nonreactive, unilateral, dilated pupil. If level of consciousness is unaltered, this is most likely an isolated ocular injury.

17. Why is a history of hammering metal on metal important in a patient presenting with an eye complaint?

Often a small, high-velocity fragment penetrates the globe with minimal or no physical findings. This injury, which can cause inflammation weeks later, is diagnosed with soft-tissue radiographs of the orbit or a computed tomography (CT) scan of the globe.

18. Which eyelid lacerations should be repaired by an ophthalmologist or plastic surgeon?

Those involving the:

- Lid margin or gray line
- Tear duct mechanism along the lower eyelid
- Tarsal plate or levator muscle

19. When should penetration of the globe be suspected?

The pupil is usually misshapen, pointing in the direction of the penetration. The globe may appear soft because of decreased intraocular pressure. Intraocular pressure should not be tested if a penetrating injury is suspected because the pressure promotes extrusion of aqueous humor.

20. List traumatic ophthalmologic injuries that require immediate ophthalmologic consultation.

- Chemical burns of the eye
- Orbital hemorrhage with increased intraocular pressure
- Perforation of the globe or cornea
- Lacerations involving the lid margin, tarsal plate, or tear duct
- Lens dislocation

21. Name two ophthalmologic injuries that require urgent ophthalmologic consultation (within 12–24 hours).

Anterior hyphema and blow-out fracture.

22. What is solar keratitis?

Also known as **flash burns** or **snow blindness**, solar keratitis is a corneal injury secondary to overexposure to ultraviolet light. Diagnosis is made with fluorescein staining, which shows multiple punctate lesions of the cornea. Treatment consists of resting the eyes with adequate narcotic analgesia. Spontaneous resolution can be expected in 12 to 24 hours.

23. What is the significance of a retro-orbital hematoma?

Bleeding behind the globe (retro-orbital hematoma) can lead to elevated orbital pressure, which can be greater than the perfusion pressure of the retina and result in ischemia. Treatment is a lateral canthotomy, which releases the canthus that holds the eye in its socket. This allows for proptosis of the globe, which (temporarily) relieves the elevated retro-orbital pressure, preserving blood flow to the retina.

24. What is the cause of a dilated pupil that fails to constrict with topical pilocarpine?

A dilated pupil that fails to constrict with topical miotic agents is due to topical application of a mydriatic agent often because of rubbing the eye after application of a scopolamine patch (for motion sickness).

KEY POINTS: OPHTHALMOLOGIC EMERGENCIES

1. Preservation of vision in a chemical burn is directly related from time of exposure to time initiating irrigation; do not wait for the patient to arrive at the hospital.
2. Never patch a patient with an eye injury related to contact lens; a patch provides a perfect environment for bacterial proliferation. These patients should be treated with aminoglycoside ointment.
3. Diplopia on upward gaze is the hallmark of a blow-out fracture of the orbital floor.

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NECK TRAUMA

Jeffrey J. Schaider, MD

CHAPTER 85

1. Why is neck trauma a complicated topic?

The lack of bony protection makes the anterior neck especially vulnerable to severe, life-threatening injuries. The exposed anatomic structure of the neck, which contains many vital structures of the vascular, airway, and gastrointestinal systems, provides a fertile ground for debate and myriad opinions about modality of treatment.

2. What are the most urgent concerns in the initial management of neck trauma?

Airway and **hemorrhage control.** Airway management comes before anything else discussed in this chapter. Early endotracheal intubation is indicated for any patient with existing or potential airway compromise. Delay in airway management increases the difficulty of intubation because of swelling and compression of the anatomic structures. Bleeding should be controlled with pressure rather than with blind clamping. The wound should be examined to determine whether it has violated the platysma. Injudicious probing of the wound may be dangerous, however, because a vascular structure that has ceased to bleed may resume with disastrous consequences when its tamponade is released.

3. What is the preferred method to secure the airway?

Rapid-sequence induction (RSI) with oral tracheal intubation should be the initial airway approach in patients with none to minimal distortion of their airway. Patients with airway distortion with anticipated difficult bag-valve-mask ventilation should have their airway managed with local airway anesthesia or sedative-assisted oral tracheal intubation. The preferred sedative medications include versed and fentanyl because they are reversible or ketamine because it does not depress spontaneous respirations. Although the risks of blind nasal tracheal intubation include breaking away clots and damaging a distorted airway, a recent study from Denver found that blind nasal tracheal intubation had a 90% success rate in the prehospital phase. Surgical airway via cricothyrotomy should be employed if endotracheal intubation is unsuccessful. Tracheostomy is preferred over cricothyrotomy if there is severe damage to the larynx and cricoid cartilage.

4. What common findings indicate significant neck injury?

- Injuries involving the **vascular system** result in hematomas, bleeding, pulse deficit, shock, and neurologic deficit secondary to arterial interruption.
- **Laryngeal trauma** causes voice alteration, airway compromise, subcutaneous emphysema, crepitus, and hemoptysis.
- Signs and symptoms of **esophageal disruption** include pain and tenderness in the neck, resistance of the neck to passive motion, crepitus, subcutaneous emphysema, dysphagia, and bleeding from the mouth or nasogastric tube. The diagnosis of esophageal disruption is difficult because of injuries to other overlying structures. Ancillary testing must be used to assist in the diagnosis of these injuries.

For more details, see Table 85-1.

5. What are the signs and symptoms of blunt carotid artery trauma?

Of patients with blunt carotid trauma, 25% to 50% have no external signs of trauma. Delayed neurologic signs are the rule rather than the exception; only 10% of patients have symptoms

TABLE 85–1. SIGNS AND SYMPTOMS OF SYSTEM INJURIES

Vascular	Aerodigestive
Hematoma	Respiratory distress
Hemorrhage	Stridor
Neurologic deficit	Cyanosis
Pulse deficit	Hemoptysis
Horner's syndrome (carotid injury)	Tracheal deviation
Hypovolemic shock	Subcutaneous emphysema
Vascular bruit or thrill	Pneumothorax
Altered sensorium	Sucking wound
Harsh, machinery-like precordial murmur (air embolism)	Dysphonia, aphonia, hoarseness Dysphagia Odynophagia

of transient ischemic attacks or strokes within 1 hour of injury. Most patients develop symptoms within the first 24 hours, but 17% develop symptoms days or weeks after injury. Carotid artery injuries may present with a hematoma of the lateral neck, bruit over carotid circulation, Horner's syndrome, transient ischemic attack, aphasia, or hemiparesis. The clinical manifestations of vertebral artery injury include ataxia, vertigo, nystagmus, hemiparesis, dysarthria, and diplopia.

6. Name the main controversy regarding management of penetrating neck trauma.

The management of penetrating neck trauma that violates the platysma. In the 1990s, physicians and surgeons changed from a mandatory exploration policy for penetrating neck wounds to a selective management approach.

7. What is mandatory exploration for penetrating neck wounds?

All patients who have wounds that penetrate the platysma muscle in the neck are explored surgically to determine the presence or absence of injury to the deeper structures in the neck. Some ancillary diagnostic testing (i.e., angiography, esophagography, esophagoscopy, and laryngoscopy) may be done preoperatively, depending on the location of the wound and the stability of the patient.

8. What are the advantages and disadvantages of mandatory exploration for penetrating neck wounds?

During the 1940s, mandatory exploration was instituted for all penetrating wounds that violate the platysma. This policy reduced mortality significantly and remained the only mode of therapy until the mid-1970s. Proponents of mandatory exploration warn of the catastrophic complications from delayed treatment and missed injuries. Neck exploration is relatively simple, and a negative exploration has low morbidity and mortality. However, because the negative exploration rate (no injuries found at surgery) is 50%, the cost of the operation and the added length of hospital stay are unwarranted. Many of these operations could be avoided with the selective approach to neck exploration.

KEY POINTS: EARLY AIRWAY MANAGEMENT FOR NECK INJURIES

1. Manage airway early before airway distortion occurs.
2. Use oral tracheal intubation with RSI as initial airway management option.
3. Do not paralyze patients with significant airway distortion who cannot be ventilated.
4. Tracheostomy may be necessary as rescue airway if there is a hematoma over or damage to cricoid cartilage.

9. Describe the theory behind the selective surgical management of penetrating neck wounds.

With the improved sensitivity and specificity of ancillary diagnostic testing (angiography, carotid duplex scanning, computed tomography [CT], esophagography, esophagoscopy, laryngoscopy), a nonoperative approach to a select group of patients, based on physical examination and results of ancillary tests, is safe. The selective approach has reduced the negative exploration rate from 50% to 30%.

10. What are the three anatomic zones of the neck?

- **Zone I** is the area below the cricoid cartilage.
- **Zone II** extends from the cricoid cartilage to the angle of the mandible.
- **Zone III** extends from the angle of the mandible to the base of the skull.

Figure 85-1 illustrates the zones of the neck.

11. Why is the neck divided into three zones?

The location of the injury plays a major role in assessing the need for angiography:

- All **zone I** injuries require angiography to determine the integrity of the thoracic outlet vessels. In stable but symptomatic patients needing surgery, angiography should be done preoperatively because positive findings necessitate a thoracotomy before neck exploration.
- The familiar anatomy of **zone II**, coupled with relative ease of surgical exposure, minimizes the need for angiography in symptomatic patients undergoing surgery. For patients without clinical signs of significant injury, most recommend helical CT angiography of the neck or carotid duplex scanning on asymptomatic patients to detect occult injuries and involvement

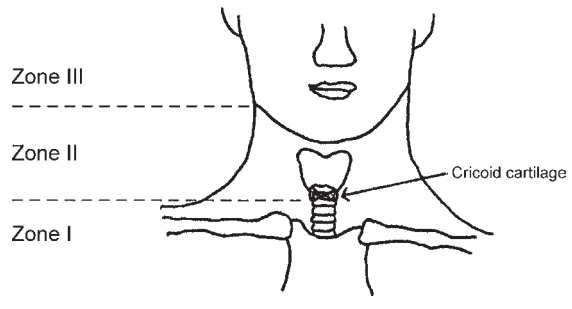

Figure 85-1. Zones of the neck.

of vertebral vessels before observation. Some clinicians observe asymptomatic patients with penetrating injuries without any imaging.

- The management of **zone III** injuries is controversial because of the complex anatomy of the area and the difficulty in obtaining adequate exposure. Most clinicians agree that for asymptomatic patients not undergoing surgery, angiography is necessary to assess the status of the internal carotid artery and the intracerebral circulation. For symptomatic patients, preoperative angiography is helpful because high internal carotid artery injuries are difficult to visualize at operation and may require carotid artery ligation and concomitant extracranial-intracranial bypass.

12. Can carotid duplex scanning or computed tomographic angiography (CTA) replace angiography for detection of vascular injuries in penetrating neck injuries?

With experienced operators, carotid duplex scanning approaches 100% sensitivity for excluding zone II and III vascular injuries in stable, asymptomatic patients with penetrating neck injuries. Because carotid duplex scanning has a lower specificity (85%–95%), positive carotid duplex scanning should be followed by carotid angiography before making a decision regarding surgical intervention.

In recent studies using multidetector helical CT scanners, CTA had sensitivities of 90% to 100%, a specificity of 100%, a positive predictive value of 100%, and a negative predictive value of 98% in detecting carotid artery injuries. The limitations and pitfalls of the helical CTA include artifacts produced by the shoulders of large patients or by bullet fragments and other metallic foreign bodies. Streak artifacts can simulate an intimal tear. In cases with inadequate studies or doubtful CTA results, the study should be considered nondiagnostic, and the patients must undergo conventional angiography. CTA examinations have only a 1.1% reported incidence of nondiagnostic results.

13. What diagnostic testing is preferred in detection of blunt vascular injuries?

- Blunt vascular injuries were found in 27% of high-risk patients screened for blunt vascular injury (combination of injury mechanism [cervical hyperextension or hyperflexion, direct cervical blow, near-hanging] and injury pattern [carotid canal, midface, and cervical spine fracture]). **Angiography** is the study of choice in acutely injured and symptomatic patients. Of lesions, 90% occur at the bifurcation of carotids or higher. Four-vessel angiography is recommended because multiple vessel injuries occur in 40% to 80%. With the improved sensitivity of CT, angiography is shifting to a therapeutic role.
- The diagnostic accuracy of **CTA** has improved with the use of better CT technology. Using a 16 slice CT, Eastman found that CTA had a 97% sensitivity and 100% specificity. CTA has been shown to decrease significantly the time to diagnose the injury.
- **Color flow Doppler ultrasound** provides rapid identification and quantification of arterial dissection, but it is unable to assess distal upper extracranial and intracranial internal carotid artery and is operator dependent. With an experienced operator, ultrasound can be used as a screening test in lower-risk patients.
- **Magnetic resonance angiography (MRA)** accurately detects carotid and vertebral artery injuries with a sensitivity and specificity greater than 95% for carotid artery dissection. It is ideal for follow-up or for stable patients; MRA is difficult to perform in an acutely injured unstable patient.

14. What is the appropriate management of blunt vascular injuries?

Carotid and vertebral artery dissections causing neurologic deficits should be treated with endovascular stent-placement. Asymptomatic dissections are at risk for extension of the dissection and thromboembolic events but often heal with observation and anticoagulation therapy alone.

15. Which diagnostic studies are important in suspected laryngeal injuries?

- Soft-tissue cervical **radiographs** may show a fractured larynx, subcutaneous air, or prevertebral air.
- **CT** accurately identifies the location and extent of laryngeal fractures. CT should be done when the diagnosis of a laryngeal fracture is still suspected despite a negative examination of the endolarynx or when flexible laryngoscopy cannot be done (e.g., intubated patient).
- Flexible **laryngoscopy** provides valuable information regarding the integrity of the cartilaginous framework and the function of the vocal cords.

16. Are diagnostic studies necessary in suspected esophageal injuries?

Yes. Soft-tissue cervical radiographs may show subcutaneous emphysema or an increased prevertebral shadow. Chest radiograph findings include pleural effusion, pneumothorax, mediastinal air, and mediastinal widening. Esophageal contrast studies should be done initially with radiopaque contrast medium (Gastrografin); if negative, studies should be repeated with barium to increase diagnostic yield. Radiographic imaging is difficult because of the high false-negative rate. Esophagography has a 30% to 50% false-negative rate and should be followed by esophagoscopy in patients with suspected esophageal injury. Rigid endoscopy is more sensitive than flexible endoscopy. No one study can exclude esophageal perforation; a combination of physical signs, plain and contrast radiographs, and esophagoscopy should be used to make the diagnosis. Isolated esophageal injuries after blunt injury are extremely rare.

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CHAPTER 86

CHEST TRAUMA

Susan Brion, MD, MS, and Justin C. Chang, MD

1. What is the initial approach to the patient with chest trauma?

Specific life-threatening conditions should be suspected based on mechanism of injury. Key injuries that should be diagnosed and treated during the initial standard survey include airway obstruction, tension pneumothorax, cardiac tamponade, massive hemothorax, open pneumothorax, and flail chest.

- **Inspection.** Completely undress the patient and visually inspect the entire chest, which necessitates rolling over a supine patient. Look for a flail chest (paradoxical movement of the chest wall) and sucking chest wounds. Identify the exact location, number, and type (i.e., penetrating or blunt) of wounds. Look for distended neck veins, swollen face or neck (indicating damage to the mediastinum or underlying subcutaneous emphysema).
- **Auscultation.** Initial auscultation should focus on the axillae where breath sounds are most readily heard. If breath sounds are equal bilaterally, the major bronchi are probably intact. Listen for diminished or absent breath sounds and bowel sounds in the chest. The presence of bowel sounds high in the chest may be the first indication of diaphragmatic injury. Decreased breath sounds usually indicate pneumothorax or hemothorax, although in an intubated patient this may also occur with the endotracheal tube being in too deep. Bony crepitus indicates a rib or sternal fracture with the potential for intrathoracic injury.
- **Palpation.** It is important to palpate the chest wall gently at first to detect bony crepitus and subcutaneous emphysema. Crepitus indicates a major blow to the chest with rib fractures and the potential for underlying organ damage (e.g., pulmonary contusion), and subcutaneous emphysema, depending on location, indicates pneumothorax or pneumomediastinum.

2. What is the differential diagnosis for blunt versus penetrating chest trauma?

- **Blunt injury:** Simple pneumothorax, tension pneumothorax, hemothorax, rib fractures, flail chest, sternal fracture, pulmonary contusion, pneumomediastinum, cardiac contusion/blunt myocardial injury, and aortic injury.
- **Penetrating injury:** Open pneumothorax, cardiac and great vessel injury

3. What is the best way to diagnose simple pneumothorax?

Recent literature supports the use of bedside transthoracic ultrasound in the hands of experienced emergency medicine physicians as a superior modality for the diagnosis of pneumothorax in comparison to plain chest X-ray, particularly in the supine patient. If plain chest X-ray is used, a posterior-anterior (PA) film in full expiration should be used to detect a small pneumothorax. Computed tomography (CT) of the thorax remains the gold standard for diagnosis.

4. When is a pneumothorax likely to cause severe symptoms?

Severe symptoms may be seen if the pneumothorax is greater than 40% of the hemithorax (approximately 2.5 cm from the chest wall in an adult), in patients with shock or pre-existing cardiopulmonary disease, or if it is a tension pneumothorax.

5. How is tension pneumothorax diagnosed?

Tension pneumothorax is a clinical rather than radiographic diagnosis. Clinical signs include dyspnea, distended neck veins, diminished or absent breath sounds on the affected side, tracheal deviation to the opposite side, hyperresonance to percussion, and hyperexpansion of the chest wall on the affected side. If hypotension is present, jugular venous distention may not be noted. Tachycardia, absent breath sounds, and hypotension are the most reliable and easiest to appreciate signs of tension pneumothorax. If a chest X-ray is obtained, a hyperlucent, overexpanded hemithorax with an evident pneumothorax and a mediastinal shift to the opposite side would be observed.

6. What is the treatment for tension pneumothorax?

If tension pneumothorax is suspected, immediate reduction of the intrapleural pressure by placement of a 14-gauge intravenous catheter over the fourth or fifth rib in the midaxillary line, followed by aspiration with a 50-mL syringe, will convert the tension pneumothorax into an open pneumothorax. This should be followed immediately by a tube thoracostomy after vital signs improve. Lack of improvement following decompression indicates another cause of hypoperfusion should be sought immediately.

KEY POINTS: TENSION PNEUMOTHORAX

1. Tension pneumothorax should be a clinical diagnosis.
2. Tachycardia, absent breath sounds, and hypotension are the most reliable means of diagnosis.
3. Immediately decompress the chest with a 14-gauge intravenous catheter whenever tension pneumothorax is suspected.
4. Look for other sources of hypotension if there is no immediate improvement after decompression.

7. What is the definition of massive hemothorax?

Massive hemothorax in an adult is defined as 1500 mL or more, about two thirds of the available space in the hemithorax, occupied by blood. Patients with hemothorax will occasionally continue to bleed. If there is evidence of ongoing hemorrhage after initial drainage exceeding 600 mL over 6 hours, a *massive hemothorax equivalent* is diagnosed.

8. What is flail chest?

A flail chest occurs when a segment of the chest wall becomes unattached from the rest of the chest. It occurs in one of three settings:

- Two or more ribs are broken in two or more places.
- More than one rib is fractured in association with costal cartilage disarticulation.
- The costal cartilages on both sides of the sternum are disarticulated, resulting in a sternal or central flail segment.

The significance of a flail chest lies in the tremendous force that caused it and the near certainty of associated intrathoracic injuries.

9. What is the treatment for flail chest?

The primary treatment should be supportive, including analgesia, coughing, chest physiotherapy, prevention of fluid overload, and subsequent pulmonary edema. If the patient is in respiratory distress either clinically or as indicated by blood gas analysis or oxygen saturation measurements, intubation and positive-pressure ventilation should be initiated. Indications for early ventilatory support include shock, three or more associated injuries, severe head injury, comorbid pulmonary disease, fracture of eight or more ribs, and age older than 65 years.

10. How do I diagnose a rib fracture by physical examination?

A rib fracture and chest wall contusion will both exhibit localized tenderness to palpation. However, only a rib fracture will exhibit referred pain when the rib is compressed posterior or anterior to the area of localized tenderness. In a stable patient in no respiratory distress, a chest X-ray is unnecessary and may not reveal an insolated, nondisplaced rib fracture.

11. What are the radiologic findings of pulmonary contusion?

Characteristic findings on chest radiographs consist of solitary or multiple patchy, ill-defined areas that may be either localized or diffuse resulting from blood accumulating in the alveoli and interstitial spaces of the lung. Areas of opacification of the lung seen on chest X-ray within 6 hours of blunt trauma are usually considered pulmonary contusions. Although often visible within 1 to 2 hours following blunt chest trauma injury, these findings sometimes may not appear until several hours after injury. The findings on chest X-ray often lag behind those seen on clinical examination and chest CT.

12. What is the significance of a sternal fracture?

The significance of a sternal fracture lies in the fact that it is often associated with more serious injuries such as damage to the great vessels or blunt myocardial injury (BMI) requiring further investigation. Sternal fractures are often missed on initial chest X-ray and are best viewed on lateral films or by CT scan.

13. When should BMI be suspected, and what types of injuries occur?

BMI is most commonly caused by high speed motor vehicle accidents, but it is also seen with direct blows to the chest, crush injuries, falls from heights, blast injuries, and athletic trauma. Mechanisms of blunt injury to the heart include sudden anterior-posterior acceleration or deceleration forces causing the heart to impact against the sternum and vertebrae, direct compression from a forceful blow to the chest or abdomen, any sudden increase in intrathoracic and intracardiac pressures, and prolonged cardiopulmonary resuscitation (CPR). BMI injuries include wall rupture, septal rupture, valvular injuries (aortic most common), direct myocardial injury (contusion), coronary laceration or thrombosis, and pericardial injury.

14. What are the symptoms of BMI and how is it diagnosed?

The symptoms of BMI vary with the severity of injury, but most commonly include chest pain, tachycardia unexplained by the degree of blood loss, trauma, pain and dysrhythmias. Although there are no good screening tests available, a reasonable approach to diagnosis includes obtaining an initial electrocardiogram (ECG) for any patient who has sustained blunt trauma to the mid-anterior chest. If normal, the patient may be discharged from the ED. If the initial ECG is abnormal, they should be admitted to telemetry and a repeat ECG obtained in 24 hours. A negative troponin obtained 6-8 hours post injury may further help to exclude BMI.

15. What are the most common ECG abnormalities in a patient with BMI?

A persistent supraventricular tachycardia (after all other causes have been treated or ruled out), premature ventricular contractions (PVCs), transient right bundle branch block, or any other new ECG abnormalities may commonly be seen in patients with BMI.

16. What are the clinical findings of pericardial tamponade?

Pericardial tamponade occurs when blood and clots accumulate in the pericardial space, compromising cardiac filling pressure and ultimately leading to shock and death. Pericardial tamponade should be suspected in any penetrating wound of the chest and is typically associated with hypotension, tachycardia, and elevated central venous pressure (CVP). Paradoxical pulse, characterized by a drop in systolic blood pressure of more than 10 to 15 mm Hg during normal spontaneous inspiration may also be seen. All of these findings are also seen with tension pneumothorax and this must first be clinically ruled out. Bedside ultrasonography is the most rapid and reliable means of diagnosis.

KEY POINTS: CLINICAL FINDINGS OF PERICARDIAL TAMPONADE

1. Hypotension.
2. Tachycardia.
3. Elevated central venous pressure or jugular venous distension.
4. Remember these are also seen in tension pneumothorax!

17. How is pericardial tamponade treated?

Pericardiocentesis should be immediately performed in unstable patients with tamponade. Preferably this should be ultrasound-guided, followed by immediate transfer to the operating room. If vital signs are lost in the ED, an immediate thoracotomy is indicated. Patients with less severe hypotension, or in situations in which an operating room is not immediately available, may benefit from placement of a pericardial catheter to allow for repeat aspirations until arrangements can be made for transfer for more definitive therapy.

18. What is the mechanism and what are common locations of a traumatic aortic tear?

The thoracic aorta is particularly susceptible to acceleration-deceleration shearing forces because the arch is less mobile than the heart and the aorta distal to the ligamentum arteriosum. Frontal or transverse deceleration causes shearing forces at the points of fixation, with the most common site for disruption being just distal to the left subclavian artery. Vertical acceleration-deceleration injuries such as falls may result in a tear of the ascending aorta with coronary artery compromise or acute pericardial tamponade. At least 90% of blunt aortic injuries in patients who reach the hospital alive occur in the isthmus of the aorta, between the left subclavian artery and the ligamentum arteriosum.

19. How is acute traumatic rupture of the aorta (TRA) diagnosed?

Maintaining a high index of suspicion in any patient who has sustained sudden severe deceleration or a high speed impact from the side is the first step to making this diagnosis. TRA should also be considered in the presence of multiple rib fractures or flail chest, although one third of blunt aortic injuries have no obvious external trauma. Physical findings suggesting aortic injury include acute onset of upper extremity hypertension, difference in pulse amplitude between upper and lower extremities, and a harsh murmur over the precordium or space between the scapula. Widened mediastinum visualized on an upright chest X-ray remains the most sensitive and specific finding in patients subsequently shown to have TRA. Less predictive findings on chest X-ray include esophageal deviation, an apical cap, left pleural effusion, obscuration of the aortic knob, loss of the paraspinal stripe, and depression of the left mainstem bronchus. Keep in mind that 10% of initial chest X-rays in patients with TRA are completely normal! Any suspicion based on mechanism or clinical findings warrants further evaluation by CT of the chest with contrast or consideration of transesophageal echocardiogram performed at the bedside for unstable patients.

20. What is the prognosis for blunt aortic injury and what emergent treatment is necessary?

Approximately 85% to 90% of patients with aortic rupture die before medical aid reaches them. The 10% to 15% who survive do so because not all three layers of the aorta are ruptured; the adventitia remains intact and temporarily contains the hemorrhage. Left untreated, this injury usually results in complete rupture and exsanguination, usually in hours to days, but this may be delayed for years in the form of a pseudoaneurysm rupture. Therefore, diagnosis and

treatment is truly lifesaving. Treatment includes use of β-blockade with an esmolol drip infusion to control blood pressure, maintaining a systolic pressure of less than 120 mm Hg, and careful replacement of fluids to prevent worsening tear or rupture. Operative repair is almost always necessary, but the optimal timing or method is determined based on the individual patient.

21. How is penetrating chest trauma managed, and what is the significance of wound location?

Wound location dictates the clinical approach by virtue of the organs at risk. From a functional standpoint, wounds are categorized as central, peripheral, thoracoabdominal, and those in adjacent areas (abdomen and neck). Multiple diagnostic and therapeutic approaches exist, depending on the location of the chest wound and the nature of the wounding implement.

22. How are penetrating wounds of the central region managed?

Patients who are grossly unstable require transfer to an operating room for an emergent thoracotomy with essentially no ED workup. Stable patients should be monitored closely while a diagnostic workup (consisting of aortography, an esophagogram with or without esophagoscopy, and possibly bronchoscopy) is done. If the workup is negative, observation for 24 to 48 hours is appropriate; if positive, surgical intervention is needed. A helical CT scan of the chest can be extremely useful to determine the presence and location of mediastinal hemorrhage.

23. Should all patients with a penetrating peripheral wound of the chest be admitted to the hospital?

Patients with peripheral wounds not in the thoracoabdominal area who are stable and have an initial normal chest X-ray usually do not require admission. They should be observed in the ED and have a repeat upright chest X-ray and hematocrit done in 4 to 6 hours. If repeat studies are normal, the patient may be discharged.

24. Which trauma victims can potentially benefit from ED thoracotomy (EDT)?

Victims of blunt trauma with documented asystole, or who require more than 5 to 10 minutes of prehospital CPR and arrive to the ED with no signs of life (i.e., pupillary response, respiration, or motor activity), are generally regarded as being unsalvageable. Because survival is essentially 0% for this population, EDT is considered futile care. Victims of penetrating trauma who arrest in the field but arrive with less than 15 minutes of prehospital CPR are potentially salvageable and candidates for EDT. The population that appears to benefit the most from EDT are those victims of penetrating chest trauma who arrest immediately on, or after, their arrival to the ED. Immediate release of a pericardial tamponade or temporary repair of a cardiac laceration can be life saving.

KEY POINTS: CONTRAINDICATIONS FOR ED THORACOTOMY

1. Blunt traumatic arrest with documented asystole.
2. Blunt traumatic arrest with prehospital CPR more than 5 minutes and no signs of life.
3. Penetrating traumatic arrest with CPR more than 15 minutes and no signs of life.
4. Penetrating traumatic arrest and asystole without the possibility of cardiac tamponade.

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CHAPTER 87

ABDOMINAL TRAUMA

Max V. Wohlauer, MD, and Ernest E. Moore, MD

1. Why is ABCDE relevant to the evaluation of significant abdominal trauma?

ABCDE (airway [with cervical spine control], breathing, circulation, disability, and exposure) and clinical examination are the most important elements in the evaluation of a trauma patient. Significant disability renders the clinical examination ineffective, requiring the use of diagnostic studies. Exposure is a reminder that a complete examination of the abdomen, back, and pelvis is essential.

Diffuse peritonitis or persistent hemodynamic instability is an absolute indication for prompt laparotomy, regardless of injury mechanism.

KEY POINTS: INDICATIONS FOR LAPAROTOMY

1. Diffuse peritonitis
2. Hemodynamic instability

2. Discuss the key aspects of the history and physical examination in the initial evaluation of abdominal trauma.

A good history is important in establishing the tempo, sequence, and extent of early diagnostic efforts. Glean as much relevant information as possible from the prehospital providers. They were at the scene and can provide a picture of what transpired. Key information includes: extent of vehicular damage, duration of extrication, whether a passenger died at the scene, use of seat belts and airbags, whether the person was ejected from the vehicle, the presence of alcohol or drug use, and the trends in vital signs.

The primary survey consists of the initial identification and correction of life-threatening injuries and is followed by a comprehensive head-to-toe examination of the patient, the secondary survey. Lower thoracic and upper abdominal trauma should be considered as a unit; suspect abdominal injury in any penetrating wound below the level of the nipple. With significant injury, abdominal tenderness and guarding are prevalent, whereas rebound tenderness and rigidity are relatively uncommon. Most importantly, 20% to 40% of patients with serious intra-abdominal injury may appear asymptomatic.

3. What are some of the biomechanical differences between blunt and penetrating trauma?

Blunt trauma results from a combination of crushing, stretching, and shearing forces. The magnitude of these forces is proportional to the mass of the object, rate of change in velocity (acceleration and deceleration), direction of the impact, and elasticity of the tissues. Conversely, penetrating injuries result from the dissipation of energy and consequent tissue disruption along the path of the offending projectile. The magnitude of injury depends on the kinetic energy imparted by the penetrating object and the trajectory. Gunshot wounds can produce extensive tissue damage ($KE = MV^2$), making the injury patterns much less predictable than stab wounds. Gunshot wounds that violate the peritoneum generally require a laparotomy.

4. Lower rib fractures are typically associated with what intra-abdominal injuries?
Lower rib fractures are associated with injuries to the liver and spleen.

5. What is a Chance fracture?
A Chance fracture, a transverse fracture of a low thoracic or lumbar vertebrae, is caused by flexion of the back and is associated with two-point seat-beat use. The incidence of associated intra-abdominal injuries with a Chance fracture approaches 50% and includes small bowel and abdominal aorta.

6. What injuries are associated with pelvic fractures?
Pelvic fractures are associated with injuries to the spleen, liver, or gastrointestinal tract in approximately 10% of patients.

7. What is a seatbelt sign?
It is an ecchymotic imprint of the seatbelt on the anterior chest or abdomen of a restrained passenger, indicating rapid deceleration from a motor vehicle crash. Presence of a seatbelt sign is associated with a 20% incidence of intra-abdominal injury.

8. What is the characteristic finding of diaphragmatic rupture on chest X-ray?
A displaced nasogastric tube representing the stomach through the left hemithorax reveals a diaphragm rupture. However, the chest X-ray is normal in up to half of patients with left diaphragmatic injury and is often normal with right-sided injuries.

9. Does a normal serum amylase exclude pancreatic injury?
No, serum amylase is neither a sensitive nor specific test for pancreatic injury (i.e., a normal amylase does not exclude pancreatic injury) and an elevated amylase may be due to increase in salivary amylase.

10. What is the most commonly injured abdominal organ?
In **blunt trauma**, the spleen, and in **penetrating trauma**, it is the liver. The small bowel is the most commonly injured abdominal hollow viscus.

KEY POINTS: DIAGNOSTIC TOOLS FOR BLUNT TRAUMA

1. Ultrasound FAST examination
2. DPL or DPA
3. CT

11. Which is the initial test of choice?
The focused abdominal sonography for trauma (FAST) is currently the initial test of choice in the evaluation of blunt abdominal trauma. Performed by emergency medicine physicians and surgeons, FAST is a rapid, painless, and sensitive test for identifying intra-abdominal fluid. If the test is initially negative, **repeating the examination in an unstable patient is imperative** because $>$ 250 mL of blood within the abdominal cavity must accumulate in Morrison's pouch before a fluid stripe will appear on FAST.

12. What is the role of computed tomography (CT) scanning?
Abdominal CT is the test of choice for evaluating the abdomen of patients with significant blunt abdominal trauma who are hemodynamically stable. Abdominal CT serves a major role in the decision to manage the injured spleen, liver, or kidney nonoperatively. Because CT can help determine the trajectory of a projectile, it also serves a role in selecting stable patients with penetrating trauma for nonoperative management.

13. What is the role of diagnostic peritoneal lavage (DPL)?

The major advantage of DPL is a sensitivity rate > 95% for the identification of intraperitoneal hemorrhage. Because the technique is invasive and DPL fails to identify the source of bleeding, its use has declined (as FAST has become routine). For hemodynamically unstable patients, FAST is a more rapid, less invasive test, but is operator dependent. For hemodynamically stable patients, CT provides more accurate information. A negative FAST does not exclude injury in penetrating trauma. DPL is used predominantly when CT is unavailable, or if the FAST results are negative but there is no other source to account for a patient's hemodynamic instability. The DPL is often done without the infusion of fluid (i.e., diagnostic peritoneal aspiration [DPA]). If the patient is hemodynamically unstable due to intra-abdominal hemorrhage, gross blood should be retrieved on insertion of the catheter.

14. How are DPL results interpreted?

DPL is considered positive if > 10 mL of free blood is aspirated. Otherwise 1 L of warmed normal saline is infused. A minimal recovery of 75% of lavage effluent is required for the test to be considered valid. The fluid is analyzed for red blood cell (RBC) counts, white blood cell (WBC) counts, lavage amylase, alkaline phosphatase, and bilirubin.

In blunt trauma, unlike in penetrating trauma, there are multiple sources that can cause hemodynamic instability in which a laparotomy would not be the priority in management. These include intrathoracic trauma, pelvic retroperitoneal injury, long bone fractures, or spinal cord injury or intra-thoracic trauma. Therefore, it is important to search beyond the abdomen for causes of hypotension before deciding to proceed to the operating room (Figs. 87-1 and 87-2).

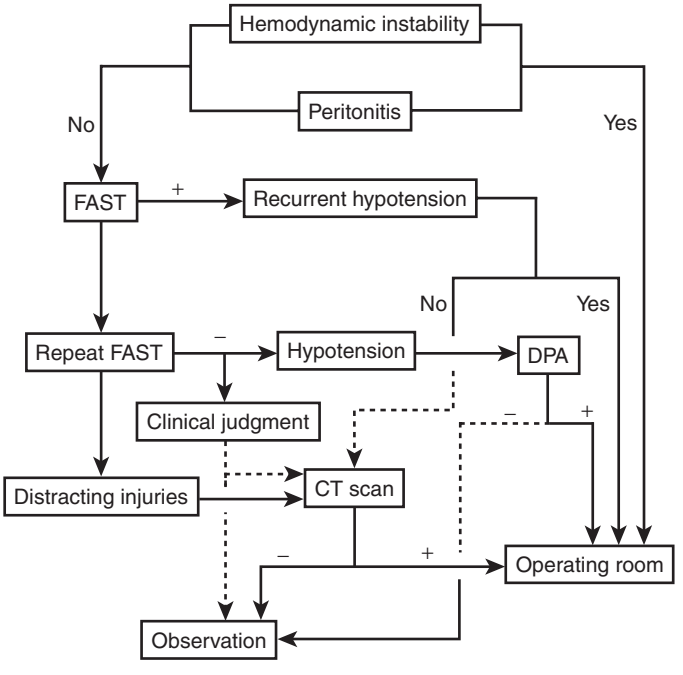

Figure 87-1. Algorithm for management of blunt abdominal trauma.

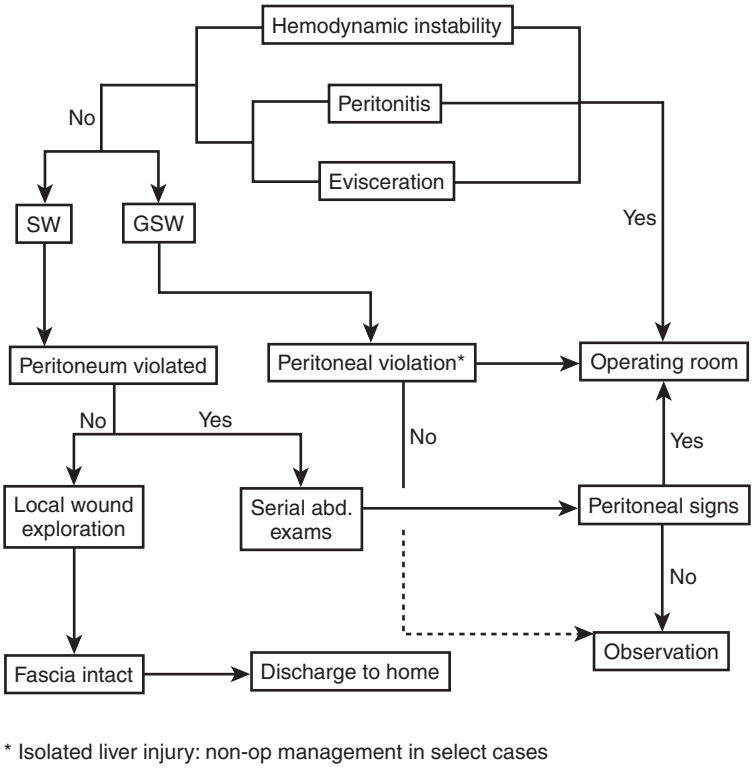

Figure 87-2. Algorithm for management of penetrating abdominal trauma.

15. What are the unique concerns in a pregnant patient with abdominal trauma?

The prevailing rule is that optimal care of the mother ensures the best outcome for the fetus. Noninvasive fetal monitoring is used routinely if the fetus is potentially survivable (i.e, >26 weeks). Hemodynamic instability, uterine rupture, placental abruption, and fetal distress are indications for abdominal exploration.

16. What are the general principles of trauma in the elderly population?

The combination of chronic medical conditions and limited organ reserve makes elderly patients especially vulnerable to trauma. Preinjury β-blocker use inhibits the physiologic response to hemorrhagic shock and is associated with increased mortality. Anticoagulant use (i.e., warfarin) prolongs hemostasis and is associated with increased mortality in patients with head injuries.

Gunshot wounds (GSW) that violate the peritoneal fascia mandate a laparotomy, with the possible exception of a low energy GSW to the liver. **Stab wounds are managed selectively.** Local wound exploration in the ED provides valuable information because only two thirds of stab wounds to the anterior abdomen actually violate the peritoneum. Even if the peritoneum is violated, only 50% of these stab wounds produce injuries that require surgical intervention.

17. In the management of abdominal trauma, are children really just *small adults*?
No, injury patterns are different in children due to their size (see Chapter 90). The elasticity of the child's lower rib cage and the relatively large abdominal cavity increase susceptibility to injury. Although blunt injuries tend to be self-limited, an aggressive operative policy is warranted for pediatric trauma because of the child's limited physiologic reserve.

KEY POINTS: ABDOMINAL TRAUMA

1. Any patient who has persistent hemodynamic instability or signs of peritonitis following abdominal trauma requires emergent laparotomy.
2. A meticulous physical examination is the most important element in the evaluation of a lucid trauma patient.
3. A negative FAST examination does not reliably exclude significant intraperitoneal injury.
4. Observing a trauma patient is an active process, including serial physical examinations and repeat abdominal ultrasonography.
5. CT scan is the most reliable means to evaluate abdominal trauma in the hemodynamically stable patient.

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PELVIC FRACTURES AND GENITOURINARY TRAUMA

CHAPTER 88

Walter L. Biffl, MD, FACS

1. Why are pelvic fractures so deadly?

Pelvic fractures can lead to life-threatening hemorrhage. Sources of bleeding include the pelvic bones themselves, surrounding soft tissue, and the extensive arterial and venous networks running through the pelvic ring. The considerable force required to fracture the pelvis typically results in significant associated injuries in up to 90% of patients. Collectively, these factors account for high rates of morbidity and mortality.

2. What is the approach to the patient with a pelvic fracture?

The evaluation begins with the primary survey (the ABCs) and resuscitation. Unstable patients with pelvic fractures require a multidisciplinary approach, with the fundamental objectives of:

- Control of hemorrhage
- Reversal of shock
- Identification of associated injuries
- Prioritization of treatment based on threat to life

Life-threatening associated injuries are evaluated and treated simultaneously with systematic assessment of the pelvic fractures. Because these patients may require coordinated interventions by multiple specialties, the immediate presence of the attending trauma surgeon, attending orthopedic surgeon, and interventional radiologist in the ED is warranted. (Fig. 88-1)

3. How do you examine the patient with a pelvic fracture?

Very carefully! The physical examination directed at the pelvis includes gentle manual compression of the bony pelvis and inspection of the perineum, rectum, and vagina for ecchymosis, ongoing bleeding, and open wounds. An unstable pelvic fracture is *not* a "teaching case;" every manipulation leads to further hemorrhage because bony edges disrupt clot and lacerate tissue and blood vessels. Plain anteroposterior radiography of the pelvis is a priority in patients with suspected fracture. Hemodynamically stable patients may be evaluated further with additional views (e.g., inlet/outlet) or computed tomography (CT), but this should not interfere with resuscitation or necessary interventions.

4. How are pelvic fractures classified?

The Tile classification, based on pelvic stability, is useful for reconstructive planning:

- Tile A: Rotationally and vertically stable
- Tile B: Rotationally unstable, vertically stable
- Tile C: Rotationally and vertically unstable

A commonly used scheme is that of Young and Burgess, which is based on injury mechanism and is more helpful in assessing the risk of hemorrhage:

Anteroposterior compression (APC)

- APC I: Pubic symphyseal diastasis < 2.5 cm, no significant posterior ring injury
- APC II: Pubic symphyseal diastasis > 2.5 cm, tearing of anterior sacral ligaments
- APC III: Complete disruption of pubic symphysis and posterior ligament complexes

Lateral compression (LC)

- LC I: Posterior compression of sacroiliac (SI) joint without ligament disruption
- LC II: Posterior SI ligament rupture, sacral crush injury
- LC III: LC II, with APC injury to contralateral pelvis

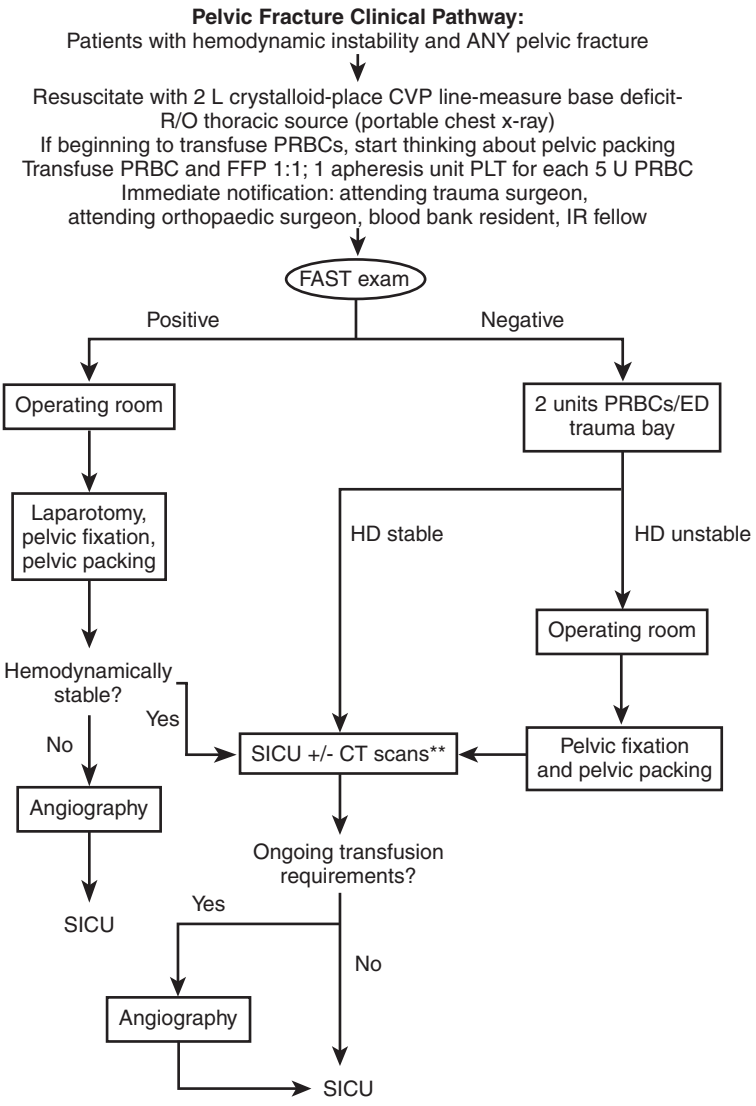

Figure 88-1. Management of patients with a pelvic fracture. CVP, central venous pressure; DPL, diagnostic peritoneal lavage; FFP, fresh frozen plasma; PLTs, platelets; PRBCs, packed red blood cells; TICU, trauma intensive care unit.

Vertical shear injuries consist of displaced fractures of the anterior rami and posterior columns, including SI dislocation.

5. What are the sources of bleeding from major pelvic fractures?

The most frequent source is of venous origin, but arterial bleeding can lead quickly to hemodynamic compromise. Massive bleeding is often associated with vertical shear or APC fractures. The internal iliac arterial system (in particular, the superior gluteal artery bridging the SI joint) may be affected by SI disruption. Significant blood loss can occur from vesicular branches of the pudendal artery in association with pubic diastasis and anterior fractures. Injury to the veins in the superior gluteal and pudendal distributions and the lumbosacral venous plexus also contributes significantly to retroperitoneal and pelvic hemorrhage. LC fractures are not usually associated with major blood loss because they result in compression of local vasculature.

KEY POINTS: APPROACH TO PATIENTS WITH A PELVIC FRACTURE

1. Multispecialty involvement is critical.
2. Bleeding is the leading cause of death.
3. Associated injuries are common.
4. Prompt, definitive decision making is the key to survival.

6. Name three goals of mechanical pelvic stabilization.

- Reduce pelvic volume.
- Promote tamponade of bleeding bone and vessels.
- Prevent further fracture motion.

7. Discuss four methods of pelvic stabilization.

- **Wrapping the pelvis with a sheet and binding the knees and ankles with tape.** This intervention should be performed immediately on discovery of an unstable pelvic fracture, particularly before patient transport. Prolonged use may result in extremity or abdominal compartment syndrome.
- **Anterior external fixation.** This is becoming the standard intervention for acute pelvic stabilization. It is most effective with the anteroposterior open-book fracture. More complex fractures such as vertical shear injury may also benefit from early stabilization, but fixation is not as complete because of the instability of the posterior column.
- **Pelvic C-clamp.** This intervention is more effective than a standard anterior frame in stabilizing the posterior pelvis.
- **Pneumatic antishock garment (PASG).** Use is controversial, particularly in prehospital care in urban areas with short transport times. Given the efficacy of pelvic wrapping, there is little role for the PASG today.

8. When should patients with pelvic trauma undergo laparotomy?

The incidence of active intraperitoneal visceral bleeding is 20% to 30% in association with pelvic fracture. Ultrasound should be utilized during initial evaluation of unstable patients to exclude hemoperitoneum. If ultrasound is not available, diagnostic peritoneal aspirate (DPA) should be done at the supraumbilical ring to avoid dissecting pelvic hematoma. Ultrasound showing overt intraperitoneal fluid, or a grossly positive DPA, should prompt immediate laparotomy. In the patient with a normal ultrasound or DPL positive by red blood cell count only, the pelvic bleeding should be managed first. In this case, the key decision is whether to employ skeletal fixation

alone, pelvic packing, or selective arterial embolization; prompt consultation of orthopedic and interventional radiology specialists is imperative. (See Fig. 88-1.)

9. How frequently are rectal injuries associated with pelvic injuries? How are they managed?

Approximately 5% of major pelvic fractures are associated with rectal injuries. These complex injuries result in a high mortality rate secondary to septic complications. Current management principles consist of fecal diversion, presacral drainage, and perineal debridement as needed. Although some studies have shown that presacral drainage may be unnecessary, these were based on small patient samples.

10. What is the role of pelvic packing for pelvic trauma?

Packing was employed commonly in Europe before being adopted in the United States. Packing is beneficial in patients who do not appear to have an indication for laparotomy but who remain hemodynamically unstable despite blood transfusion (see Fig. 88-1). The interventional radiology suite is not an ideal place for these patients, so packing in the operating room, followed by pelvic stabilization, can help with hemorrhage control. More prospective studies are needed for this to gain wide acceptance in the United States.

11. What types of injuries are associated with genitourinary trauma?

Pelvic fracture can cause posterior (above the urogenital diaphragm) urethral tears or bladder trauma, whereas perineal straddle injury is more likely to cause anterior urethral tear. Fractures of the lower ribs, lower thoracic, or lumbar vertebrae are often associated with renal or ureteral injuries.

12. What is considered a true genitourinary emergency?

Most genitourinary trauma is not life-threatening and can be addressed after stabilization of the patient, including necessary operative control of significant hemorrhage and contamination. However, renal pedicle injury can lead to uncontrolled hemorrhage or renal ischemia. The kidneys are not fixed and move to a limited degree on the vascular pedicle. Complete severance of this pedicle can lead to exsanguination, whereas lesser injury to the renal vessels can cause thrombosis and subsequent ischemia. This is typically seen with deceleration injury. Early diagnosis and surgical intervention are crucial for salvage of the affected kidney.

13. What four clinical signs may indicate injury to the kidney?

- Flank ecchymosis
- Lateral abdominal tenderness or mass
- Hematuria
- Fracture of lumbar posterior ribs or lumbar vertebrae

14. What is the general management strategy for renal injury?

Nonoperative management is appropriate in the large majority of patients because injuries will heal spontaneously. Surgery is indicated for hemodynamic instability, ongoing bleeding, or urinary extravasation. However, minimally invasive techniques, such as angio embolization for hemorrhage and stenting for urinary extravasation, may allow renal salvage.

15. What diagnostic tools can be used to evaluate renal trauma?

CT is the preferred modality for the evaluation of blunt abdominal trauma. It allows for comprehensive evaluation of all intra-abdominal structures. Helical CT has increased sensitivity for ureteral injury. Intravenous pyelography (IVP) is less sensitive and does not allow for evaluation of nonurologic injuries. However, it may still be used in cases of suspected renal or ureteral injury when CT is unavailable, or if urologic imaging is required in the operating room. Renal angiography may be indicated in the presence of a suspected vascular injury, although it has also largely been replaced by CT. Magnetic resonance

imaging (MRI) has imaging capabilities similar to CT but is far more expensive, time consuming, and not as readily available. MRI may be useful in stable patients with contrast allergies.

16. When should ureteral trauma be suspected?

In the presence of penetrating injuries in proximity to the ureter. These are the least common of the genitourinary injuries. Hematuria may be absent when the ureter is completely transected. Ureteral injuries can be detected by CT or IVP and should be managed operatively.

17. What are the associated clinical findings with bladder injury?

Traumatic bladder rupture is an uncommon injury secondary to the protected location of the bladder within the pelvis. This injury most often occurs in conjunction with pelvic fracture but can also be seen with lower abdominal compression due to lap belt or steering wheel injuries. Gross hematuria is present in greater than 95% of patients.

18. How should bladder injury be evaluated?

The two main diagnostic modalities for evaluation of bladder injury are CT cystography and conventional retrograde cystography. The accuracy of either method depends on adequate distention of the bladder. Bladder imaging is mandatory in the setting of gross hematuria with pelvic fracture. Relative indications include gross hematuria without pelvic fracture and pelvic fracture with microhematuria. Penetrating trauma in the vicinity of the bladder should be evaluated with a cystogram regardless of the presence of hematuria.

19. When should urethral injury be suspected?

Blood is visualized at the urethral meatus in 80% to 90% of patients with urethral injury. Other signs of urethral injury are penile, scrotal, or perineal hematomas or a high-riding prostate on rectal examination. If urethral injury is suspected, insertion of Foley catheter should be deferred until retrograde urethrogram can be performed. The ED management of complete urethral disruption is transcutaneous suprapubic cystostomy.

20. How is a retrograde urethrogram performed?

The urethrogram is obtained using a 12-French urinary catheter secured in the meatal fossa by inflating the balloon to approximately 3 mL. Alternatively, a catheter-tipped syringe may be used. Standard water-soluble contrast material (25–30 mL) is injected under gentle pressure as the anteroposterior and oblique views are taken.

KEY POINTS: UROLOGIC TRAUMA

1. Renal injury is the most frequent urologic trauma.
2. Renal pedicle injury can lead to uncontrolled hemorrhage or ischemia.
3. Clinical signs of kidney damage may include flank ecchymosis, lateral abdominal tenderness or mass, hematuria, or fracture of lumbar posterior ribs or lumbar vertebrae.
4. Gross hematuria or persistent microhematuria warrant evaluation.
5. Urologic injury may be present in the absence of hematuria.

21. What is the diagnostic approach to asymptomatic microhematuria in the patient with blunt trauma?

Asymptomatic microscopic hematuria is not a good predictor of genitourinary tract injury. The amount of blood in the urine does not correlate with severity of injury. The relatively low incidence of positive studies requiring surgery does not justify an extensive radiographic

evaluation. Close follow-up of these patients and repeat urinalyses are recommended, with additional studies only if the hematuria persists. Controversy still exists regarding the evaluation of pediatric patients with asymptomatic microhematuria. Pediatric patients are more susceptible to significant renal injury with relatively benign mechanisms, and consequently many advocate imaging studies with any degree of hematuria regardless of symptoms.

22. What is a penile fracture?

A sudden tear in the tunica albuginea with subsequent rupture of the corpora cavernosum. It occurs only in the erect penis and usually is associated with falls or sudden unexpected moves during sexual intercourse. It has also been reported with direct blunt trauma. A sudden intense pain associated with a snapping noise and immediate detumescence usually occurs. Most authors support surgical intervention in an attempt to restore normal function and prevent angulation. Inability to urinate, bleeding from the urethral meatus, or extravasation of urine may indicate injury to the corpora spongiosum and urethra, which occurs in approximately 20% of cases.

23. What is the role of ultrasound in the evaluation of testicular trauma?

Testicular injuries are most often caused by a fall or a kick to the scrotal area. Ultrasound is a valuable tool in assessing the integrity of the testicles. Adequate palpation may be prevented by hematoma formation. Ultrasound can distinguish between simple hematoma and disruption of the parenchyma. Failure to suspect and diagnose testicular rupture may result in subsequent loss of the testicle.

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TRAUMA IN PREGNANCY

Jedd Roe, MD, MBA, and Bophal Sarha Hang, MD

CHAPTER 89

1. What is the most important concept I need to remember from this chapter?
Fetal outcome is largely related to maternal morbidity. The best fetal resuscitation is aggressive maternal resuscitation.

2. How common is trauma in pregnancy?
An estimated 6% to 7% of pregnancies are complicated by trauma. In blunt abdominal trauma, the usual causes are motor vehicle accidents (MVAs; 60%), falls (22%), and direct blows (17%) to the abdomen. One study showed that serious MVAs accounted for a 7% maternal mortality rate, whereas the fetal mortality rate was 15%. Of falls, 80% occur after 32 weeks of gestation.

3. Is physical or sexual abuse seen frequently in pregnant patients?
Yes. One large study reported a prevalence of abuse in pregnant women in urban settings of 32%. Of physically abused women, 60% reported two or more episodes of assault. Injury was more common to the head, neck, and extremities; a fourfold increase in the incidence of genital trauma was noted in this population. When pregnant patients are physically abused, there is a higher incidence of low-birth-weight infants, low maternal weight gain, maternal anemia, and drug and alcohol abuse. Homicides account for one third of maternal trauma deaths. In low-income pregnant women, there have been reported high rates of violence-related abdominal trauma. Another study showed that three screening questions asked of pregnant patients in the ED can detect the majority of patients who are victims of partner abuse, which suggests that screening for domestic violence should be pursued with pregnant trauma patients. (See Chapter 98.)

4. How do physiologic changes in pregnancy affect the evaluation of the trauma victim?
First, decreasing blood pressure and rising heart rate might indicate hypovolemic shock in a nonpregnant woman, but in pregnancy this may merely reflect physiologic changes or supine positioning. The maternal blood volume increases 50%. As a result, signs of shock may not be clinically apparent until 2000 mL or 30% to 40% of maternal blood volume is lost. Furthermore, uterine flow comprises 20% of cardiac output, approximately 600 mL/min. Given the markedly increased blood flow, the uterus is a new potential source of blood loss that requires aggressive investigation. Because physiologic changes result in increased oxygen demand and decreased oxygen reserve, tissue hypoxia develops more rapidly in response to a traumatic insult. Also, placental blood flow has no autoregulation and thus, small changes in blood pressure can result in fetal distress.

5. How do physiologic changes of pregnancy affect laboratory values?
A physiologic anemia is seen as the plasma volume rises by more than twice the amount of red blood cells. It is not unusual for one to see hematocrits of 32% to 34% by the third trimester. Fibrinogen levels are double those seen in other trauma patients. Disseminated intravascular coagulation (DIC) may be seen with normal fibrinogen levels. Because of hormonal stimulation of the central respiratory drive, PCO_2 falls between 27 and 32 mm Hg,

and injury sufficient to cause a respiratory acidosis might be manifested by what ordinarily would be considered a normal PCO_2 of 40 mm Hg.

6. Are serious maternal injuries required for fetal injury to be present?
Not always. Although in utero damage is often associated with maternal pelvic fractures, 7% of maternal cases of minor trauma have been associated with poor fetal outcome. Direct injuries to the fetus in utero are unusual, but given the size of the fetal head, when direct trauma occurs, fetal head injury is the most common injury.

7. Name the most common causes of fetal death.
Maternal death, maternal shock, and placental abruption.

8. How does placental abruption occur?
Abruption results from the separation of a relatively inelastic placenta from an elastic uterus secondary due to a shearing, deceleration force. There may be little or no external evidence of such a mechanism. Although abruption may be present in 50% of patients with life-threatening injuries, it also exists in 2% to 4% of minor mechanisms. Classically, the clinical findings of abruption have included vaginal bleeding and abdominal and uterine tenderness. In many cases, fetal distress may be the only presenting sign because the reduction in placental blood flow to the fetus causes hypoxia and acidosis. DIC may occur with placental injury, and evaluation for DIC can be performed by screening with a serum fibrinogen level, with low levels stimulating the sending of a more complete DIC panel.

9. How often does ultrasound detect cases of placental abruption?
Because a large separation must be present for ultrasound to be diagnostic, it detects only about half of cases. In many instances, fetal distress is present before the clear visualization of an abruption by ultrasound. Fetal mortality from abruption is reported to be 30% to 68%. Usually an abruption large enough to place the fetus at risk becomes apparent within 48 hours. Detection of fetal distress mandates prompt delivery of the fetus.

10. Are radiologic investigations harmful to the fetus?
The fundamental effects of radiation on the developing fetus are intrauterine growth retardation and defects in the central nervous system (microcephaly, mental retardation). The most vulnerable period is between 2 and 15 weeks' gestation. Cumulative exposure of less than 5 rads (0.05 Gy) during pregnancy has not been shown to affect the outcome of pregnancy compared to control populations. In general, all radiographic studies should be undertaken with appropriate fetal shielding. All **clinically indicated** studies should be done regardless of any radiation concerns. Furthermore, there has been no reported adverse effects on neonatal thyroid function with the use of iodinated contrast, and it should be administered if absolutely necessary. Consideration also should be given to nonradiographic alternative evaluation with ultrasound. See Chapter 9 for radiation exposures from diagnostic imaging studies.

11. How should these patients be managed in the field?
Given the reduced maternal oxygen reserve, oxygen therapy is crucial. Intravenous volume resuscitation with crystalloid should proceed as with other trauma patients. Avoid compression of the inferior vena cava by transporting the patient on her left side, or if the patient is immobilized, elevate the right side of the backboard to 15 or 20 degrees. Aside from early transport, the most important aspect of prehospital management is to notify the ED so that the appropriate obstetric consultants may participate on the trauma team.

12. What are the priorities for ED management?
The prehospital therapies mentioned previously should be continued. Of particular interest is the history of this pregnancy with attention directed at estimating gestational age and fetal viability. After the usual primary and secondary survey, a sterile speculum examination should be performed to evaluate for the presence of vaginal fluid, opening of the cervical os, and

genital tract trauma. Continued aggressive resuscitation with warmed lactated Ringer's solution (less acidotic, more physiologic) and blood is especially important given the physiologic changes mentioned previously.

13. How do I begin to evaluate the fetus?

First, determine the size of the uterus and the presence of abdominal and uterine tenderness. Uterine size, measured in centimeters from the pubic symphysis to fundus, provides a rough estimate of gestational age and potential viability. Carefully inspect the vaginal introitus for evidence of vaginal bleeding. Next, assess for fetal distress, which may be the earliest indication of maternal hypovolemia. Abnormal fetal heart rates are greater than 160 beats per minute and less than 120 beats per minute. As soon as possible after patient arrival, continuous cardiotocographic monitoring (CTM) should be initiated to ascertain early signs of fetal distress (e.g., decreased variability of heart rate or fetal decelerations after contractions). Ultrasound should be done promptly thereafter to confirm gestational age, fetal viability, and the integrity of the placenta.

14. Is diagnostic peritoneal lavage (DPL) safe and accurate in pregnant women?

DPL has been reported to be safe and accurate when using an open, supraumbilical technique. Although the cell count thresholds and clinical indications for DPL are the same, ED ultrasound has become the more prevalent investigation. The physiologic changes that take place with pregnancy and the elimination of radiation exposure from abdominal computed tomography (CT) provide persuasive arguments for aggressive use of ED ultrasound as a diagnostic tool. With the exception of concern for diaphragmatic injury secondary to penetrating trauma, the need for DPL as an evaluation modality has largely been supplanted by the use of ED ultrasound (focused abdominal sonogram of trauma [FAST]) to determine rapidly the presence of intraperitoneal hemorrhage.

15. What is fetomaternal hemorrhage (FMH)?

Hemorrhage of fetal blood into the usually distinct maternal circulation. The incidence of FMH in trauma patients has been reported to be 30% (four to five times the incidence of noninjured controls). With FMH, the complications of maternal Rh sensitization, fetal anemia, and fetal death can occur. Laboratory techniques are not sensitive enough to diagnose FMH accurately. The prudent course is to give Rh immunoglobulin to all Rh-negative patients who present with the suspicion of abdominal trauma because a 300-mg dose of Rh immunoglobulin given within 72 hours of antigenic exposure prevents Rh isoimmunization. Massive transfusion ($>$30 mL) into the maternal circulation sometimes is seen with severe abdominal trauma. The Kleihauer-Betke (KB) test detects fetal erythrocytes in the maternal circulation, and positive KB tests have not been shown to alter management except in Rh-negative patients. However, one study showed that the incidence of positive KB tests did not differ between low-risk pregnant patients and maternal trauma patients. Given the inaccuracy of the KB test in the setting of trauma, administration of Rh immunoglobulin should proceed as described previously.

16. When is cesarean section indicated?

The first factor to be considered is the stability of the mother. If the mother has sustained serious injuries elsewhere and is critically ill, she may not be able to tolerate an additional procedure and the blood loss it would entail. Next, fetuses whose gestational age is 24 weeks or whose weight is estimated to be greater than 750 gm are predicted to have a 50% survival rate in the neonatal intensive care unit (ICU) setting and are considered viable. The most common indication for cesarean section is fetal distress. Other indications are uterine rupture and malpresentation of the fetus. Perimortem cesarean section should be done when ultrasound or uterine size suggests viability (i.e., above the umbilicus) and maternal decompensation is acute. Resuscitation should be instituted within 4 minutes, but fetal survival with normal neurologic outcome has been reported 40 minutes after maternal decompensation.

17. Which pregnant patients with abdominal trauma require admission for fetal monitoring?

Any viable (>23-24 weeks) fetus requires continuous fetal monitoring or CTM. CTM is recommended even for patients without external evidence of trauma because it has been well documented that these patients are at risk from placental abruption, and CTM is sensitive for its detection. Current guidelines suggest that these patients be observed for a minimum of 4 hours with a cardiotocograph. If any abnormalities are discovered, including more than four uterine contractions/hour, amniotic membrane rupture, vaginal bleeding, serious maternal injury, significant abdominal pain, and signs of fetal distress, the patient should be hospitalized and monitored for 24 hours.

KEY POINTS: TRAUMA IN PREGNANCY

1. Aggressive maternal resuscitation is the best therapy for the fetus.
2. The fetus may be in acute distress with little or no maternal manifestations.
3. Ultrasound is the investigation of choice to evaluate the maternal abdomen and the fetus.
4. All clinically necessary radiologic investigations should be performed regardless of radiation concerns.

WEBSITE

www.perinatology.com/exposures/Physical/Xray.htm

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PEDIATRIC TRAUMA

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CHAPTER 90

1. Which children get injured? How do they do it?

Every year, nearly one in three children is injured. Trauma is the leading cause of mortality for children younger than 14 years, greater than all other causes combined in children older than 1 year of age. Motor vehicle crashes account for most deaths in all age groups, followed by drownings, house fires, homicides, and falls in descending order. A very common site of lethal pediatric trauma is the home. Boys are injured twice as often as girls. Falls are the most common cause of severe injury in infants and toddlers; bicycle crashes are the most common cause in children and adolescents.

2. Aren't children just little adults?

No. Anatomically, unique characteristics in children require special consideration:

- A smaller body mass results in more force applied per unit area, with a propensity toward multiple injuries in a child. An example of this is Waddell's triad: a femur fracture, truncal trauma (i.e., intra-abdominal or intrathoracic injury), and head injury, typically occurring after a child is struck by an automobile at high speed.
- Due to a greater head-to-body ratio, thinner cranial bones and less myelinated brain tissue, intracranial injury is the leading cause of mortality and morbidity in the pediatric trauma population.
- Relatively larger solid organs with relatively smaller thoracic and pelvic bony structures, coupled with less subcutaneous fat and less mature abdominal musculature, makes intra-abdominal solid organs more anatomically susceptible to injury.
- A child's incompletely calcified and thus compliant skeleton allows internal organ damage without overlying fractures.
- A high body surface area-to-volume ratio results in significant thermal energy loss and early hypothermia in a child.

3. How does prioritization of ABCs differ between children and adults?

Airway, breathing, and circulation always take priority in that order, whether in an adult or a child. Relative to adults, children have a higher baseline respiratory rate due to increased metabolic demands. Blood loss and an increase in lactate production can quickly lead to marked increased work of breathing and respiratory arrest from fatigue. Thus, tachypnea can be the first clue to the child with acute blood loss. Tachycardia may be a manifestation of emotional factors in the injured child but should not be easily dismissed as such. Tracking the patient's capillary refill time and urine output with repeated vital signs can help guide the provider. Bradycardia in a child is often secondary to head injury, hypoxia, or inadequate ventilation. In the setting of trauma without respiratory compromise, bradycardia is an ominous sign.

4. Which factors affect the patency of a child's airway?

Particularly in infants, craniofacial disproportion (the child's occiput is relatively large compared with the midface) results in cervical flexion when the child is lying supine. To align the oral, pharyngeal, and tracheal axes, a roll may be placed under the shoulders in very young children. Compared with an adult, a child has a large tongue, floppy epiglottis, and increased lymphoid tissue; these factors may contribute to airway obstruction. The sniffing

position (slight superior and anterior positioning of the midface) is employed to maintain a patent airway. Infants are preferential nasal breathers, so their nares should not be occluded with a nasogastric tube. Oral airways should be inserted only in unconscious children as they may induce vomiting.

5. Which factors affect endotracheal intubation of a child?

A child's larynx lies higher and more anterior in the neck, and the vocal cords have a more anterocaudal angle; the cords may be more difficult to visualize for intubation. The narrowest part of a child's airway is the cricoid ring, which forms a natural seal with the endotracheal tube. In adults, the narrowest portion is at the level of the vocal cords and a balloon is necessary to stabilize the tube. Uncuffed tubes (using the formula 4 + age/4 to calculate mm of internal diameter) may be used in children younger than 8 years. However, because the size of the airway cannot always be predicted, one may opt for a cuffed endotracheal tube (ETT) half a size smaller to prevent any potential air leak. Insertion depth can be estimated as three times the tube size in centimeters.

6. What are my options if I cannot endotracheally intubate the patient?

A laryngeal mask airway is considered class indeterminate by pediatric advanced life support (PALS) guidelines. Although an option, this device may cause upper airway obstruction, particularly in children less than 20 kg, by folding the larger epiglottis into the larynx (see chapter on pediatric and neonatal resuscitation). In children older than 8 years of age, a surgical cricothyrotomy can be performed. There is debate and limited evidence to support a specific lower age limit for this procedure. Most would agree that in children younger than 6 years of age, the cricothyroid membrane is too small and structures too thin to safely perform this procedure. In this group, a needle cricothyrotomy should be performed using a 16- to 18-gauge needle and a translaryngeal jet ventilation device. Due to limitations in adequate ventilation with needle cricothyrotomy, the treating provider should immediately consult with a surgeon to perform an emergent tracheostomy.

7. How do I recognize shock in a pediatric patient?

Children have an increased physiologic reserve and often maintain vitals signs in the normal range even in the presence of compensated shock. However, young children are less able to increase their cardiac contractility, responding to blood loss only by increasing heart rate. This singular cardiac mechanism can result in precipitous drops in blood pressure when a critical volume is lost. Other signs include poor skin perfusion, decreased pulse pressure, mottling of the skin, cool extremities, capillary refill greater than 2 seconds, increased work of breathing and a depressed level of consciousness. For children older than 1 year, the average systolic blood pressure can be estimated as 90 mm Hg plus twice the age in years, whereas the lower fifth percentile is estimated as 70 mm Hg plus twice the age in years. Hypotension typically indicates a loss of $>$ 45 % of blood volume and may be accompanied by bradycardia.

8. Name the preferred sites for venous access.

In decreasing order of preference: peripheral, intraosseous (particularly in very young critically ill children), percutaneous femoral-subclavian-internal jugular, and saphenous vein cutdown at the ankle. In the unstable patient, intraosseous or central venous access must not be delayed by multiple peripheral attempts. Ultrasound-guided central line access should be utilized, as its use results in a decreased number of access attempts, as well as fewer arterial punctures in the pediatric population.

9. What are some considerations regarding an intraosseous line?

It is most appropriate in children younger than 6 years and allows administration of virtually any fluid and blood product or drug. The preferred site is the proximal tibia below the tibial tuberosity. It should not be placed distal to a fracture and should be removed when peripheral intravenous access is secured. Complications include cellulitis, osteomyelitis, growth plate injury, fat microembolism, compartment syndrome, and iatrogenic fractures.

10. What is a child's normal blood volume?
80 mL/kg

11. How should I resuscitate a pediatric trauma patient?
A 30% reduction in blood volume generally is required to manifest signs of compensated shock. The 3:1 rule (crystalloid resuscitation-to-blood loss) applies as it does in adults. For a 20-mL/kg blood loss, 60 mL/kg should be given. Either warmed normal saline or lactated Ringer's solution can be given in boluses of 20 mL/kg. After 60 mL/kg, consideration should be given to transfusing warmed packed red blood cells (10 mL/kg).

12. Why are children prone to head trauma?
A child's body is like a dart—they lead with their head. Relative to the adult, children's heads are larger in proportion to their bodies.

13. Which kinds of head injuries do children get?
Compared with adults, mass lesions are less common, but cerebral edema and postinjury seizures are more common. Hemorrhagic shock may occur secondary to blood loss in the subgaleal or epidural space because of open cranial sutures and fontanels. Bulging sutures or fontanelles suggest a significant brain injury and/or cerebral edema and warrant aggressive management and neurosurgical consultation before decompensation occurs. Subdural hemorrhages and cerebral edema are typical manifestations of abusive head trauma (also known as *shaken baby syndrome*), where bridging veins are torn and shear injury occurs from a rapid acceleration-deceleration shaking mechanism.

14. Which children need cranial imaging after head trauma?
This has been an area of considerable debate. Many algorithms have lacked sensitivity for identifying intracranial injuries in children, while others would result in overutilization and unnecessary exposure to radiation. In an attempt to identify patients that can be safely discharged from the ED without imaging, the Pediatric Emergency Care Applied Research Network (PECARN) has recently published a large series (over 42,000 patients) of children with head injury.

- In patients younger than 2 years of age, the following characteristics gave a negative predictive value (NPV) of 100% (95% CI, 99.7%–100%) for clinically important brain injuries: normal mental status, no scalp hematoma (except frontal), no loss of consciousness or loss of consciousness less than 5 seconds, nonsevere mechanism, no palpable skull fracture, and acting normally per parents.
- For children older than 2 years of age, the following criteria generated an NPV of 99.95% (95% CI, 99.81%–99.99%): normal mental status, no loss of consciousness, no vomiting, nonsevere mechanism, no signs of basilar skull fracture, and no severe headache.

15. What is SCIWORA?
SCIWORA stands for spinal cord injury without radiographic abnormality. Children are particularly vulnerable to this type of injury due to their horizontally situated facet joints, incomplete spinal ossification, and immature ligamental support structures. Plain spine radiographs are normal in two thirds of children with spinal cord injury. Therefore, in children with evidence of a spinal cord injury and normal plain films, the provider should request magnetic resonance imaging (MRI) and neurosurgical consultation.

16. How common is pseudo-subluxation of the cervical spine?
About 40% of children younger than 7 years demonstrate anterior displacement of the anterior border of C2 on C3. Approximately 20% of children up to 16 years also demonstrate pseudo-subluxation. This radiographic finding can be minimized by placing the patient in a sniffing position. It can be differentiated from true subluxation by evaluating the *line of Swischuk*; drawn along the anterior spinous processes of C1 and C3. Injury is suspected if the line passes greater than 1.5 mm from the anterior spinous process of C2.

17. How common are rib fractures in children?

Not very. The compliant chest wall allows unimpeded transmission of energy to the underlying thoracic organs, potentially resulting in life-threatening contusions. Because of the force required to break elastic bones in young children, two thirds of children with rib fractures have associated organ injuries. The child's mobile mediastinum allows tension pneumothorax to develop more readily than in adults. Bilateral posterior rib fractures should heighten the clinician's suspicion for nonaccidental trauma.

18. What are predictors of pediatric intra-abdominal injuries?

Multiple investigators have attempted to identify predictors of intra-abdominal injury in children. High-risk mechanisms for intra-abdominal injury should be taken into consideration and include high speed motor vehicle collisions, pedestrian versus automobiles, bicycle accidents (including handlebar injuries), and direct blows to the abdomen. Tenderness on palpation, seatbelt bruising, and hemodynamic instability in the field are other associated findings. Holmes et al recently published a prospectively validated prediction instrument that identified six criteria, wherein the absence of all of the following identified pediatric patients at low risk for intra-abdominal injury (95% sensitivity and 37% specificity): low age-adjusted systolic blood pressure, abdominal tenderness, femur fracture, increased liver enzyme levels (serum aspartate aminotransferase $>$ 200 U/L or serum alanine aminotransferase $>$ 125 U/L), microscopic hematuria ($>$ 5 red blood cells/high powered field) and an initial hematocrit level $<$ 30%. Ultimately, because no criteria have been perfect in identifying abdominal injuries on initial evaluation of the pediatric trauma patient, any decision algorithm cannot supplant clinical judgment in determining any individual patient's risk.

19. Compare and contrast the primary diagnostic modalities for evaluating children for abdominal trauma.

CT, diagnostic peritoneal lavage (DPL), and ultrasonography are the primary diagnostic tests. CT is appropriate in stable patients. It is the most specific of the tests, identifying solid organ and (less accurately) hollow viscus injuries. It also evaluates the retroperitoneum. However, it is time consuming, requires the patient to be hemodynamically stable for transfer out of the ED, and often requires sedation in young children. DPL is more than 95% accurate in identifying injuries, but is invasive and nonspecific. Because most solid organ injuries can be managed nonoperatively, the finding of a hemoperitoneum alone, without hemodynamic instability, does not warrant operative exploration. DPL is most useful in patients who are hemodynamically unstable or in patients whose abdomen cannot be serially evaluated, for example, if they require emergency neurosurgical or orthopedic surgery under general anesthesia. Ultrasound is simple, rapid, repeatable, and non-invasive. As with an adult patient, it may be used to triage unstable pediatric patients to the operating room or to rapidly exclude the abdomen as a source of significant blood loss. If equivocal, further imaging or diagnostic peritoneal lavage may be required, depending on the urgency of the situation. Growing experience with focused abdominal sonography for trauma (FAST) in children suggests that it can replace peritoneal lavage for the unstable pediatric patient.

20. What is a handlebar injury?

An intra-abdominal injury from a direct blow (e.g., from a bicycle handlebar) to the right upper quadrant or epigastrium. The classic finding is a pancreatic injury or duodenal hematoma, but renal injury can occur if the impact is off midline.

21. What is the lap belt syndrome?

Ecchymoses of the abdominal wall, a flexion-distraction injury of the lumbar spine (Chance fracture), and intestinal or mesenteric injury.

22. How much of a problem is nonaccidental trauma?

Nonaccidental trauma is the most common cause of traumatic death in the first year of life. Fifty percent of abused children who are released to their abusers will be the victims of repeated nonaccidental trauma, often resulting in death. Identifying victims of abuse in the ED allows for early intervention. Historical clues to abuse include changing reports of the traumatic event, injuries incompatible with reported mechanism, and late presentations. Very young age ($<$ 3 years of age and particularly $<$ 3 months of age), developmental delay, and parental substance abuse have all been associated with nonaccidental pediatric trauma.

KEY POINTS: PEDIATRIC TRAUMA

1. Children are not just little adults. Providers must take into account specific anatomic and physiologic differences when evaluating for pediatric injuries.
2. In the pediatric trauma population, head injury is the leading cause of morbidity and mortality.
3. Unstable vital signs is a late and ominous finding in the pediatric trauma patient.

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CHAPTER 91

MUSCULOSKELETAL TRAUMA AND CONDITIONS OF THE EXTREMITY

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GENERAL PRINCIPLES

1. What are the immediate treatment priorities in an open fracture?

An open fracture is an orthopedic emergency!

As in any trauma patient, the immediate priorities are the ABCs: airway, breathing, and circulation. Any break in the skin near a fracture site should be assumed to communicate with the fracture until proved otherwise. After careful examination with neurologic and vascular assessment, the wound should be cleaned of gross contamination, and a sterile dressing applied. Direct pressure can be used for hemorrhage control. Axial realignment and splinting immobilize the bone, decreasing blood loss and protecting the soft tissue from further damage. Probing of the wound, wound cultures, extensive irrigation, and multiple examinations of the wound should be avoided, owing to the increased potential for secondary contamination and soft-tissue damage. Tetanus prophylaxis and intravenous antibiotics are usually administered.

A first-generation cephalosporin, with or without an aminoglycoside, is used most commonly for antibiotic prophylaxis. When open fractures occur in grossly contaminated environments, such as barnyards, penicillin is added, secondary to the increased risk of anaerobic organisms. Consult an orthopedic surgeon immediately.

2. What percentage of polytrauma patients have unrecognized fractures at the time of admission?

Of patients with multiple system injuries, up to 20% have unrecognized fractures at the time of initial assessment. In general, these do not typically involve the axial skeleton or long bones. These unrecognized injuries are located most commonly in the hands and feet. This important fact shows the need for repetitive examination of the multiply injured patient and discussions with the family of the injured party regarding the potential of unrecognized fractures.

3. What is compartment syndrome?

A condition that develops when the pressure in the confined space of the muscle compartment exceeds the filling pressure of the venules and the arterioles supplying the muscle, resulting in muscle ischemia and edema. This increases intracompartmental pressure, further setting up a vicious cycle, eventually leading to necrosis of muscle and nerves. Conditions or situations that cause an increase in the compartment contents or decrease the expansive nature of the compartment can result in compartment syndrome. Common causes include fracture, crush injuries, hemorrhage, postischemic swelling after repair of vascular injury, tight-fitting casts or dressings, military antishock trousers (MAST), and circumferential burns.

4. What are the signs and symptoms of acute compartment syndrome?

The classic diagnosis of acute compartment syndrome is indicated by the **5 Ps:**

- **Pain** is the earliest and most common symptom associated with compartment syndrome. It is typically more severe than what is normally expected, based on the associated cause,

and is often exacerbated when the muscles of the involved compartment are put on stretch. Pain is typically ischemic in nature and often not relieved by narcotics.

- **Paresthesia** is another common finding but indicates a prolonged period of increased compartment pressures.
- **Pallor**, **pulselessness**, and **paralysis** are late findings, and the condition is often not reversible at that point.

There are various methods of measuring intracompartmental pressure to obtain objective evidence of compartment syndrome. The gold standard of diagnosis is a positive clinical examination combined with a plausible history. Missed or delayed diagnosis of compartment syndrome is catastrophic, warranting a high index of suspicion and early specialist consultation.

Exertional compartment syndrome is characterized by exercise-induced compartmental pain and swelling that resolves with rest. Acute compartment syndrome may complicate this condition. It is therefore prudent *always* to exclude compartment syndrome in a painful limb due to uncertain etiology to avoid disastrous complications.

5. What are the most common sites of compartment syndrome?

The volar compartment of the forearm and the anterior compartment of the leg. The deep posterior compartment of the leg is the site most often missed for this event. Supracondylar fractures of the humerus in children and both-bone forearm fractures are the injuries most commonly associated with this process in the upper extremity. Proximal tibial fractures are the most common cause of compartment syndrome in the leg. Compartment syndrome is also known to occur in the hands, feet, thighs, and upper arm. Fractures may or may not be present.

6. What is the treatment of compartment syndrome?

Surgical release of the investing fascia of the compartment (fasciotomy) is the only effective treatment for this condition. Temporary measures that may be used to prevent compartment syndrome include elevation of the extremity to above heart level and maintenance of a normal arteriole filling pressure by maintaining a normotensive blood pressure.

7. Describe the joint fluid analysis consistent with septic arthritis.

The fluid will appear cloudy. Other causes of cloudy fluid are gout and psuedogout. Cells per mm^3 will be between 2,000 and 50,000 with greater than 90% being polymorphonuclear neutrophils (PMNs). Unlike gout and pseudogout, there will be no crystals and the culture will be positive.

8. What information should be obtained if a possible septic joint is to be aspirated?

The fluid should be sent for Gram stain, culture, cell count, and crystals. Always remember that if attempting to aspirate a joint with surrounding cellulitis, try to stay clear of the red, inflamed, infected skin area so as not to seed the joint.

9. How do I diagnose a traumatic open joint?

Probing of a wound in proximity to a joint is insufficient, may increase the risk of infection, and generally should be avoided. Radiographs of the involved joint may reveal the presence of air in the joint, indicating joint violation. The definitive diagnosis is made by performing an arthrogram. Sterile saline combined with a small amount of methylene blue should be injected in the joint. A significant amount of fluid needs to be injected to distend the joint. The traumatic wound is inspected for egress of the injected fluid. The fluid should then be withdrawn from the joint.

10. When should I order radiographs? How many should I order?

- Radiographs should be ordered based on the physical examination findings.
- Radiographs should include the joints above and below the perceived area of injury.
- Two orthogonal views always should be obtained.

Obtaining radiographs should not obstruct the resuscitation process in the multiply injured patient. In situations in which significant deformity of the limb results in vascular compromise or devitalization of the overlying skin, radiographs should be delayed, pending emergent realignment and splinting of the involved extremity.

KEY POINTS: MUSCULOSKELETAL TRAUMA AND CONDITIONS OF THE EXTREMITY

1. An open fracture is an orthopedic emergency.
2. Compartment syndrome is an important diagnosis for exclusion.
3. Septic joints require immediate orthopedic attention.

HAND AND UPPER EXTREMITY

11. What is the best method to control bleeding in a hand or forearm laceration?
Direct pressure. Tourniquets are rarely necessary. The practice of blindly placing clamps into the wound is dangerous and often can damage structures in close proximity to the offending vessel, such as the median or ulnar nerve.

12. What metacarpophalangeal joint most commonly sustains a laceration when an individual engages in *fist diplomacy*?
The third metacarpophalangeal joint because it is the most prominent knuckle when making a fist.

13. How much deformity can be tolerated in a metacarpal fracture?
Rotational deformity is not tolerated well and should be corrected. Rotation in a metacarpal neck or shaft fracture causes the fingers to cross when the individual makes a fist. Flexion deformity of the fracture is the most common and best tolerated. You can accept 10, 20, 30, and 40 degrees of flexion in the index through small fingers. A greater degree of deformity is tolerated at the small finger, secondary to the increased motion at the carpometacarpal joint. The same is true for a thumb metacarpal fracture, in which 40° of angular deformity can be accepted.

14. What bacterium is often associated with cat bites, and what is the antibiotic treatment?
Pasteurella multocida. Penicillin G, co-amoxiclav (orally [PO]), or ampicillin/sulbactam (Unasyn) (intravenous [IV]). Use doxycycline in penicillin-allergic patients.

15. What should be done with an amputated part that may be replanted?
1. Remove gross contamination by irrigating with saline.
2. Wrap the part in a saline-moistened (not soaked) sterile gauze.
3. Place the wrapped part into a sealed plastic bag or container.
4. Place the bag or container into an ice water bath.
   Never put the amputated part straight onto ice!

16. List traumatic amputations that should be considered for replantation.
- Any amputation in a child
- Multiple finger amputations
- Thumb
- Hand
- Arm

The ultimate decision always should be deferred to a hand surgery specialist. It is usually best to defer detailed discussion of replantation with the patient or family to the consulting specialist.

17. What is the appropriate treatment for a patient with pain in the snuffbox of the wrist and normal radiographs after a traumatic event to the wrist?

The scaphoid is easily palpable in the anatomic snuffbox of the wrist. The snuffbox is the space between the extensor pollicis longus and the extensor pollicis brevis. Tenderness in this area is suggestive of a scaphoid fracture. The absence of fracture on the initial radiographs is common. As the necrotic bone at the fracture site is resorbed, the fracture line often becomes apparent on radiographs approximately 14 days after injury. Individuals with this condition should be immobilized in a thumb spica splint or cast and referred to an orthopedist for evaluation. Bone scans and magnetic resonance imaging (MRI) are not indicated in the acute evaluation.

18. Radiographically, how can one tell the difference between an anterior and posterior shoulder dislocation?

On an anterior-posterior (AP) radiograph, an anterior dislocation would show bony overlap in a medial and inferior direction. An AP radiograph with a posterior shoulder dislocation shows a *vacant glenoid* sign as the humeral head fails to fill most of the glenoid, which should be present on a normal AP radiograph. There is also a positive *rim sign* with space between the anterior rim of the glenoid and the humeral head exceeding 6 mm.

19. State the incidence and common causes of posterior shoulder dislocations.

Incidence is 5% of shoulder dislocations. They are most often the result of falls on an outstretched hand. Other cause include tonic-clonic seizures, electrical shock, and direct anterior shoulder trauma. Reduction can be accomplished with flexion of the arm to 90 degrees and adduction to disimpact the humeral head from the glenoid rim. The arm is then externally rotated until the head has cleared the glenoid rim. A sling should be applied in neutral to 5 to 10 degrees of external rotation and slight abduction. It is critical to avoid internal rotation for 4 to 6 weeks.

20. What percentage of patients with first-time anterior shoulder dislocations experience a recurrent dislocation?

Of patients aged 30 years or younger, 90% experience a recurrent dislocation. For older patients, the percentage is lower and more variable, depending on the mechanism of injury.

21. What are the potential complications of anterior shoulder dislocation?

The axillary nerve is at risk for injury at the time of dislocation. Careful examination of the deltoid muscle should be done to assess motor function. The axillary nerve also provides sensation to the lateral aspect of the shoulder (regimental badge area), and sensation should be checked in this area. In addition to axillary nerve injury, there is high incidence of a rotator cuff tear in the first time dislocator older than age of 40.

22. How is a rotator cuff tear diagnosed?

The patient often complains of pain with overhead activity, night pain, and pain with abduction of the arm. The patient has difficulty abducting the arm and often is unable to lift the arm above the level of the shoulder. With the shoulder in 90-degree abduction, 30-degree forward flexion, and maximal internal rotation, the patient cannot resist against downward pressure on the extremity (supraspinatus strength test). A drop test is done in the same manner with the arm simply at 90-degree abduction. The patient is not able to lower the arm slowly from 90-degree abduction. When these conditions exist, it is important to differentiate a rotator cuff tear from subacromial impingement (condition that irritates the rotator cuff). Inject 10 mL 1% lidocaine in the subacromial space. If the patient obtains pain relief and still cannot initiate abduction, the diagnosis of rotator cuff tear is confirmed.

23. How is a posterior sternoclavicular dislocation diagnosed?
Radiographs are generally nondiagnostic. Computed tomography (CT) scan is the most sensitive diagnostic modality.

24. Describe the significance of anterior versus posterior dislocation of a sternoclavicular joint.
- **Anterior** dislocations are not associated with major complications and are treated easily with a sling.
- Of **posterior** sternoclavicular dislocations, 25% are associated with complications, including rupture or compression of the trachea; esophageal occlusion or rupture; lung contusion; and laceration or occlusion of the superior vena cava, subclavian vein, or artery. Reduction of posterior sternoclavicular dislocation should be done only in the operating room, with a cardiothoracic surgeon immediately available.

25. What is the most common neurological deficit seen with a humeral shaft fracture?
The radial nerve may be stretched **(neurapraxia)** or rarely lacerated **(neurotmesis)**. This condition typically occurs with fractures involving the distal one third of the humerus. Disability includes inability to extend the wrist and fingers at the metacarpophalangeal joints and numbness on the dorsum of the radial side of the hand. Interphalangeal extension, representing ulnar and median nerve function, is preserved. Triceps function is preserved because it is innervated by branches of the radial nerve proximal to the radial groove.

26. What is the difference between a nightstick fracture and a Monteggia fracture?
- A fracture of the ulna (typically the proximal one third) with a radial head dislocation is a **Monteggia** fracture. This fracture occurs as a result of a fall on an outstretched hand with associated valgus force on the extremity. The treatment requires internal fixation of the ulna fracture.
- A **nightstick** fracture is a fracture of the ulna resulting from a direct blow to the shaft of the ulna. There is no associated injury to the proximal radial ulnar humeral joint. In most cases, these injuries can be treated by closed means and early range of motion. Nightstick fractures with significant comminution, greater than 10 degrees of angulation or greater than 50% displacement, may be considered for operative fixation.

27. What nerve is commonly injured in a Monteggia fracture?
The posterior interosseous nerve lies in close proximity to the neck of the radius. When the radial head is dislocated, this nerve is often stretched, resulting in a neurapraxia and inability to extend the thumb or wrist.

LOWER EXTREMITY AND PELVIC FRACTURES

28. Name the major complications directly related to pelvic fracture.
Hemorrhage and urologic injuries, including bladder rupture and urethral tear. (See Chapter 88.)

29. What is the mortality rate in patients with open pelvic fracture?
Mortality has decreased from 50% in the 1980s to 10% to 25% due to a move toward a multidisciplinary approach and advances in critical care.

30. What are the incidence and mechanism of injury in posterior hip dislocation?
Greater than 80% are posterior and result from a force directed posteriorly to a flexed knee, as occurs when the knee strikes the dashboard in a motor vehicle crash.

31. What are the complications of posterior hip dislocation?
- **Sciatic nerve deficit** is found in about 10% of patients, resulting in weakness or loss of hamstring function in the thigh and all of the muscles of the leg.

- **Avascular necrosis** occurs in 10% to 15% of patients but increases almost to 50% if reduction is delayed beyond 12 hours.
- Even with prompt reduction, 20% of patients develop **osteoarthritis**.
- The risk of **recurrent dislocation** is increased during early rehabilitation following traction.

32. How is posterior hip dislocation differentiated clinically from a femoral neck fracture or intertrochanteric femoral fracture?

Both result in lower extremity shortening. In posterior hip dislocation, the hip is flexed, adducted, and internally rotated. This is often referred to as the position of modesty. With a femoral neck or intertrochanteric fracture, the lower extremity is not flexed but is shortened, abducted, and externally rotated.

33. How much blood loss can be expected with a fracture of the femoral shaft?

500 to 1500 mL

34. How are femoral shaft fractures best stabilized in the ED?

Longitudinal traction, involving a self-contained traction unit. Most emergency providers carry these and can place them in the field or ambulance. Traction should not be left in place for more than 2 hours without frequent neurovascular checks because of the potential for compartment syndrome and vascular compromise. A second option is placement of a distal femoral traction pin and in-line traction connected to the bed or gurney. A proximal tibial pin may also be used provided there is no knee injury. Conventional splinting is ineffective.

35. Why do patients with hip pathology present with knee pain?

A patient with a hip problem may complain only of pain to the anterior distal thigh and medial aspect of the knee. The knee and the hip share a common innervation through the obturator nerve. Always suspect a hip problem in a patient who complains of knee pain without corresponding findings on physical examination. Careful examination of the knee and hip, with appropriate radiographs of the hip, is necessary to complete the evaluation.

36. Name the most common injury associated with traumatic hemarthrosis of the knee joint.

Anterior cruciate ligament rupture. If fat globules are noted in the joint aspiration fluid, the possibility of an associated intra-articular fracture should be pursued.

37. Name the ligament most commonly associated with an inversion ankle sprain.

Anterior talofibular ligament. The calcaneofibular ligament can also be injured in more severe sprains.

38. Describe the treatment for ankle sprains.

Ankle sprains are treated by the **RICE** protocol: **R**est, **I**ce, **C**ompression, and **E**levation.

Early protected weight bearing with crutches and an early range-of-motion program should be instituted. More severe sprains may require a short period of immobilization.

39. Discuss the Ottawa rules regarding radiographs of the ankle.

The Ottawa rules were developed from a large study done in Ottawa, Canada, which examined the necessity of routine ankle radiography in the assessment of patients with ankle injuries. It was determined that radiographs are not required when the following conditions are met:

- The examiner is experienced.
- The patient does not have significant deformity of the ankle.
- The examination is consistent with an ankle sprain.
- There is no tenderness on examination over the medial or lateral malleolus (palpate posteriorly from the tip of the malleolus to 6 cm proximally).
- The patient was able to bear weight on the injured ankle immediately after the injury or in the ED.

40. What is a locked knee? What are the most common causes?

The patient is unable to extend the knee actively or passively beyond 10- to 45-degree flexion. True locking and unlocking occur suddenly. The most common causes are a tear of the medial meniscus, a loose body or **joint mouse** (osteochondral fragment) in the knee, or a dislocated patella.

41. What injuries are associated with a calcaneal fracture?

Depending on the exact mechanism of injury and the type of calcaneal fracture, 10% to 50% of patients have an associated compression fracture of the lumbar or lower thoracic spine. Of calcaneal injuries, 10% are bilateral, and about 25% are associated with other lower extremity injuries; 10% can result in a compartment syndrome of the foot, requiring fasciotomy.

42. What is the most common direction of a tibiofemoral knee dislocation?

Anterior (the tibia's relationship to the femur). The mechanism of an anterior knee dislocation is hyperextension of the knee. There is a 30% to 50% chance of a popliteal artery injury following this type of dislocation.

43. What direction is considered the *irreducible* knee dislocation?

Posterolateral (the tibia is posterolateral to the femur). The medial femoral condyle produces a *dimple sign* as it buttonholes through the anteromedial joint capsule becoming entrapped. An open reduction in the operating room is required.

44. What vascular injury must be considered with a tibiofemoral knee dislocation?

Injury or compression of the popliteal artery. Cadaver studies showed that anterior dislocations tend to cause intimal flaps and occlusion, whereas posterior dislocations are more likely to cause a rupture of the popliteal artery. Injuries also occur at the trifurcation just distal to the popliteal fossa. Postreduction angiography should be considered for all patients with abnormal distal pulses or ankle-brachial index.

45. How is the ankle-brachial index (ABI) calculated?

ABI = Doppler systolic arterial pressure in the injured limb (ankle)
Doppler systolic arterial pressure in uninjured limb (brachial)

An ABI value of 0.9 is considered normal. The ABI measurement may be inaccurate in patients with risk factors for peripheral arterial disease, such as diabetes and hypertension. Vessel calcification in the elderly can also increase the risk of a false-positive result.

PEDIATRIC ORTHOPEDICS

46. What is a torus or buckle fracture?

This fracture is typically seen in the metaphysis of the radius but is not limited to this bone. **Torus** means a round swelling or protuberance. In children, the cortical bone and metaphyseal bone fail in compression **(buckling)**, while the opposite cortex remains intact. The area of bone that fails in compression forms a torus. Because the opposite cortex remains intact, these fractures are stable and require splint or cast immobilization for 4 weeks.

47. What is a greenstick fracture?

Children's bones have increased elasticity. An angular force applied to a long bone of a child causes a greenstick fracture. One cortex fails in tension, while the opposite cortex bows but does not fail or fracture in compression. The fracture is similar to what occurs when one attempts to break a green branch of a tree. This fracture pattern is common in the radius and ulna. These fractures require reduction, and often the fracture must be completed to achieve an adequate reduction. Immobilization in a cast is required for 6 weeks.

48. What is the Salter-Harris classification? What is its clinical significance?

A method of classifying epiphyseal injuries (Fig. 91-1). Fractures involving the epiphysis may result in growth disturbance, and parents must be informed of this potential. About 80% of these injuries are Salter-Harris types I and II, both of which have a low complication rate. Salter-Harris types III, IV, and V injuries have a more variable prognosis. Displaced Salter-Harris types III and IV fractures may require open reduction to restore the normal relationship of the epiphysis and articular surface. The five types are summarized as follows:

- Type I: Fracture extends through the epiphyseal plate, resulting in displacement of the epiphysis (this may appear merely as widening of the radiolucent area representing the growth plate).
- Type II: Fracture is as above, with an additional fracture of a triangular segment of metaphysis.
- Type III: Fracture line runs from the joint surface through the epiphyseal plate and epiphysis.
- Type IV: Fracture line also occurs in type III but also passes through adjacent metaphysis.
- Type V: A crush injury of the epiphysis occurs, which may be difficult to determine by x-ray examination.

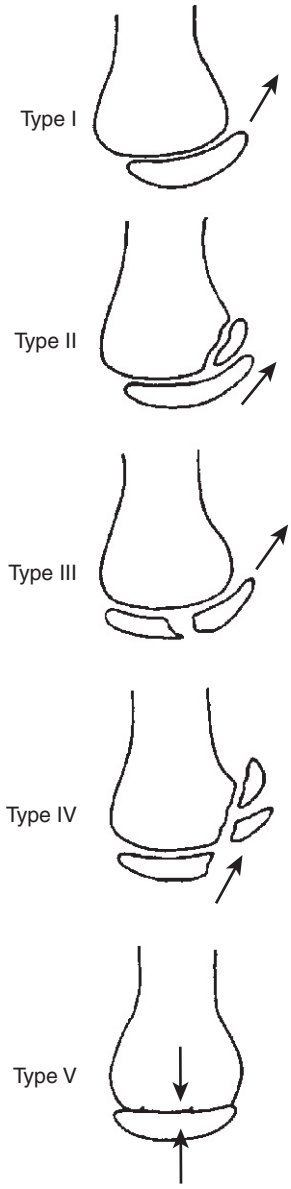

Figure 91-1. Salter-Harris classification of epiphyseal injuries.

49. Describe the vascular complications associated with pediatric supracondylar humerus fractures.

Displaced supracondylar humerus fractures in children have a 5% incidence of vascular compromise. The brachial artery typically is compressed or lacerated by the anteriorly displaced humeral shaft. Posterior lateral displacement of the supracondylar fracture is the fracture pattern most likely to result in vascular injury. The child with a viable hand and absent pulse should undergo prompt reduction and fracture fixation in the operating room, with re-evaluation of the vascular status after the procedure. In the patient with an absent pulse and a devascularized hand, longitudinal traction and splinting should be done in the ED in an attempt to reconstitute flow to the distal extremity. Prompt consultation with orthopedic and vascular surgeons is required.

50. Describe the neurologic complications associated with pediatric supracondylar humerus fractures.

The anterior interosseous nerve (branch of the median nerve) is potentially the most commonly injured nerve. This nerve innervates the deep compartment of the forearm, which consists of the flexor digitorum profundus to the index, the pronator quadratus, and the flexor pollicis longus. The nerve can be checked by evaluating flexor pollicis longus function at the interphalangeal joint of the thumb. The radial nerve is the next most commonly injured nerve, followed by the ulnar nerve. A thorough physical examination must be done to identify these injuries, a difficult task in the small child.

KEY POINTS: MUSCULOSKELETAL TRAUMA AND CONDITIONS OF THE EXTREMITY

1. Never place an amputated part directly on ice or immerse in water.
2. Bleeding from a wound is best controlled with direct pressure
3. Multidisciplinary approach is paramount in the treatment of pelvic hemorrhage.
4. Knee dislocations require a thorough vascular examination.
5. Always rule out infection in a child presenting with atraumatic hip pain.
6. In nonambulating children with humeral or femoral fractures, be suspicious of child abuse.

51. What is a nursemaid's or pulled elbow? What is its management?

A longitudinal pull on the outstretched arm of a 1- to 5-year-old child may result in a subluxation of the annular ligament over cartilaginous radial head. The child typically presents with pseudoparalysis of the injured extremity. Radiographs are negative for fracture or radial head dislocation. Reduction involves simultaneous supination of the forearm and flexion of the elbow. A distinct click over the radial head signifies reduction. The child often begins to use the extremity within minutes of reduction. The parent or caregiver should be educated to avoid longitudinal traction on the arm to prevent this from occurring in the future.

52. Describe the potential implications of a humeral or femoral fracture in a small child.

In the nonambulating child with these fractures, the suspicion of child abuse should be high. An unwitnessed event or a history that does not correspond to the injuries is another potential sign of abuse. Careful examination of the child should be done, looking specifically for skin bruises or burns, retinal hemorrhage, and evidence of previous fracture. A skeletal survey should be considered because the presence of fractures at different stages of healing is a sign of abuse. All cases of suspected abuse need to be reported to the local authorities. (See Chapter 65.)

53. What is Waddell's triad?

The constellation of injuries in a child struck by a car:

- Femoral fracture
- Intrathoracic or intra-abdominal injury
- Head injury

54. Which nontraumatic hip disorders cause a limp in a child?

- Septic arthritis
- Transient synovitis (ages 2–12 years)
- Idiopathic avascular necrosis (boys, ages 5–9 years)
- Slipped capital femoral epiphysis (SCFE) (boys, ages 10–16 years)

- Perthes' disease
- Juvenile rheumatoid arthritis

All are uncommon. Transient synovitis is probably the most common cause of nontraumatic limp in a child but is a diagnosis of exclusion.

Symptomatic treatment is prescribed for transient synovitis, including nonsteroidal anti-inflammatory drugs and non-weight bearing or bed rest. Untreated or delayed treatment of septic arthritis can lead to irreversible and catastrophic sequelae from permanent damage and deformation of the articular cartilage. Infection in a child presenting with atraumatic hip pain must be convincingly ruled out. The white blood cell count, erythrocyte sedimentation rate, and body temperature frequently are elevated in cases of infection. If doubt persists, the gold standard is hip aspiration, usually done in the operating room. Standard anteroposterior and lateral radiographs of the hip differentiate between Perthes' disease and a slipped capital femoral epiphysis.

55. What are the early radiographic findings of an SCFE?

Any asymmetry of the relationship of the femoral head to the femoral neck should raise the suspicion of SCFE, even if evident on only one X-ray view. If anteroposterior and lateral radiographs are normal, frog-leg views should be obtained. Comparison of the two hips may not be helpful in discerning subtle changes because SCFE is bilateral in 20% of cases.

56. What is the ED management of a child with injury and tenderness over an open epiphysis but a normal radiograph?

It is best to assume the child has sustained an undeterminable fracture of the physis (Salter-Harris type I or V). Immobilize the joint in a posterior splint, and keep the child non-weight bearing if the lower extremity is involved. Parents should be notified of the possibility of this type of injury and the potential for growth disturbance. The need for prompt follow-up must be emphasized and is best arranged before discharging the child from the ED. A nondisplaced physeal fracture that becomes displaced because of lack of immobilization can have significant long-term consequences. Short-term extremity immobilization in an appropriately applied splint or cast is well tolerated. When in doubt, immobilize.

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CHAPTER 92

HAND INJURIES AND INFECTIONS

Michael A. Kohn, MD, MPP

1. Are hand problems important in emergency medicine?

Yes. Our hands are our interface to the mechanical world, so they are frequently injured or exposed to infection. There are 4.8 million ED visits per year for hand/wrist injuries. At least one of every eight injury-related ED visits is for a hand/wrist injury.

2. List the essential elements of the history.

- Age
- Dominant hand
- Occupation
- How, where, and when injury occurred
- Posture of hand when injured
- Tetanus status
- Prior injury or disability of the hand

3. List the elements of a complete hand examination.

- Initial inspection of skin and soft tissue
- Vascular examination
- Evaluation of tendon function
- Nerve examination (motor and sensory)
- Determination of joint capsule integrity
- Skeletal examination

4. What is topographical anticipation?

Looking at the skin wound and thinking about which underlying structure (vessel, tendon, nerve, bone, ligament, or joint) could be injured. Know the anatomy. Do not hesitate to consult an atlas.

5. What is the normal posture of the hand at rest?

With the wrist in slight extension, the resting fingers normally assume a *cascade*, progressively more flexed from index to small. (See this by relaxing your own hand with the wrist in slight extension.) An alteration in the normal posture can lead to immediate diagnosis of major tendon and joint injuries.

6. Does dorsal swelling always signify an injury or infection in the dorsum of the hand?

No. Most of the palmar lymphatics drain to lymph channels and lacunae located in the loose areolar layer on the dorsum of the hand. Always check for a palmar pathology when a patient presents with dorsal swelling.

7. What is the Allen test? How is it performed?

The Allen test verifies patency of the radial and ulnar arteries as follows: Occlude radial and ulnar arteries. Have patient open and close hand five or six times. Hand should blanch. Release ulnar artery; blanching should resolve within 3 to 5 seconds. Repeat test, releasing

radial artery instead of ulnar artery Blanching should resolve within 3 to 5 seconds. The most accurate form of the Allen test uses digital blood pressures rather than return of color to monitor reperfusion.

8. How is function of the flexor digitorum superficialis (FDS) tendon tested?
The FDS inserts on the middle phalanx and flexes the proximal interphalangeal (PIP) joint. The flexor digitorum profundus (FDP) inserts on the distal phalanx and flexes the PIP and the distal interphalangeal (DIP) joints. The FDS muscle-tendon units should be independent of one another, whereas the FDP tendons arise from a common muscle belly. Testing the FDS of a finger entails flexing it at the PIP joint, while stabilizing the other three fingers in full extension, thereby taking the FDP out of action as a potential flexor of the PIP joint.

9. In which finger is the test of FDS function unreliable?
Because the FDP to the index finger can be independent of the other profundi, the FDS test is unreliable in the index finger. Flexion at the PIP joint may be due to the FDP, even with the other fingers stabilized in extension. Suspected index finger FDS injuries must be explored.

10. Why is the flexor or palmar aspect of the hand called the *OR side*, whereas the extensor or dorsal aspect is the *ED side*?
In contrast to the extensors, the flexor tendons run through delicate sheaths. Because of these sheaths, repairing flexor tendons requires more expertise and a more controlled environment in the operating room than repairing extensor tendons does.

11. How is a partial tendon laceration diagnosed?
If the location of the skin laceration is suspicious, rule out an underlying partial tendon laceration by exploration and direct visualization under tourniquet hemostasis. Because a flexor tendon runs through its sheath like a piston through a cylinder, a sheath laceration implies a partial tendon laceration, which is visible only when the hand is in the same posture as when injured. A bloodless field and direct visualization of the tendon during full range of motion must occur to rule out a partial tendon laceration. All wounds over tendons must be explored for partial tendon lacerations since range of motion test can still be normal. Never test for tendon integrity against resistance since a partial tendon laceration can be converted to a complete (100%) laceration.

12. How do I test the extrinsic extensor tendons?
The extrinsic extensors alone extend the metacarpophalangeal (MCP) joints, whereas they combine with tendons from the interossei and lumbricals to form the extensor mechanism that extends the interphalangeal joints. To test the extrinsic extensor, ensure that the patient can extend at the MCP joint (but see below).

13. Can extensor function to a finger be intact despite complete laceration of the extensor digitorum communis (EDC) to that finger?
Yes. The juncturae tendinum interlink the EDC tendons at the midmetacarpal level. Even if the EDC to a finger is completely lacerated in the dorsum of the hand, extension at the MCP still may be possible because of the junctura.

14. How do I test sensory nerve function?
Assess nerve function before the use of anesthesia. Test digital nerves by checking two-point discrimination on the volar pad. The two points should be 5 mm apart and aligned longitudinally.

15. What are the sensory distributions of the median, ulnar, and radial nerves?
See Fig. 92-1.

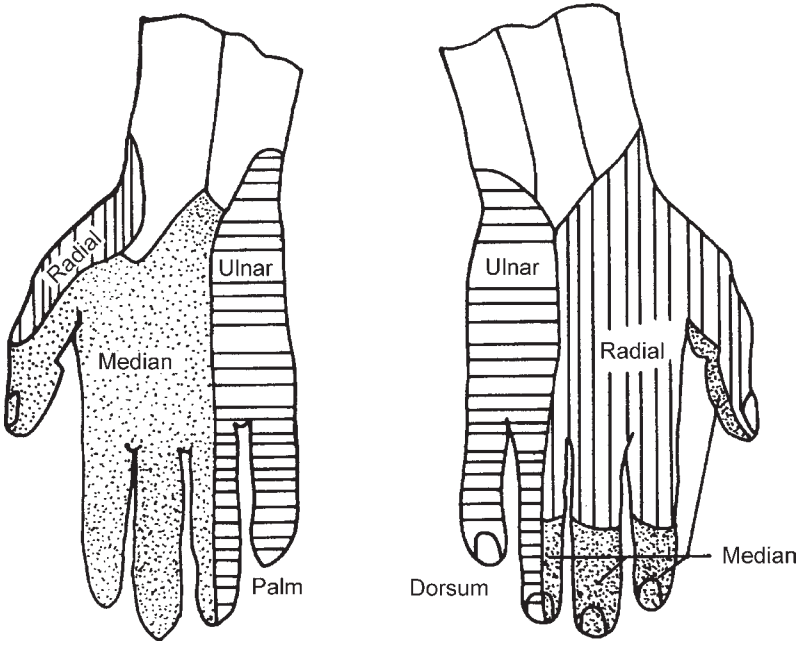

Figure. 92-1. Sensory distributions of the median, ulnar, and radial nerves.

16. How is motor function of the median, ulnar, and radial nerves tested?

- **Median:** Abductor pollicis brevis (APB)—abducts the thumb against resistance while palpating the APB muscle belly.
- **Ulnar:** First dorsal interosseous—abducts the index finger against resistance.
- **Radial** (no intrinsics): Extensor pollicis longus (EPL)—extends the thumb interphalangeal joint against resistance.

17. Which is the most frequently dislocated carpal bone?

The carpal bones from radial to ulnar side are as follows:

- Proximal row—scaphoid, lunate, triquetrum, pisiform
- Distal row—trapezium, trapezoid, capitate, hamate

The **lunate** is most frequently dislocated. Its blood supply comes through the volar and dorsal ligaments from the radius. If both ligaments are ruptured, avascular necrosis results.

18. Which is the most frequently fractured carpal bone?

The scaphoid. Its distal blood supply increases the likelihood of avascular necrosis in the proximal segment after fracture.

19. What is the classic sign of a scaphoid fracture?

Snuffbox tenderness. Even without radiographic evidence of a fracture, the patient with tenderness to palpation of the anatomic snuffbox gets a thumb spica splint and must have a repeat radiograph in 2 weeks.

20. How do I control hemorrhage from a hand injury?

Direct pressure and elevation. This will work 99.9% of the time. Elevate and hold pressure for 10 minutes by the clock. Very rarely, an incomplete arterial laceration requires a proximal tourniquet

for temporary control, followed by sensory examination, anesthesia, irrigation, and exploration under good light and magnification to tie off the bleeding vessel. **Never blindly clamp a bleeder.**

21. Why the rule, no blind clamping of bleeders?

In the hand, the arteries run in close approximation to the nerves. Blindly clamping an artery may irreparably damage the associated nerve. Also, the clamp may damage a section of vessel vital to successful reanastomosis. All hand bleeding can be controlled by direct pressure or a tourniquet.

22. What should be done with an amputated digit?

Gently clean the digit with sterile saline, wrap it in moist gauze, place it in a sterile container, and float the container in ice water. (Avoid direct contact between ice and tissue to prevent freezing.)

23. What should be done with a devascularized but still partially attached digit?

Leave part attached (preserves veins for reimplantation), gently wrap in moist gauze, and apply a bulky dressing.

24. What are the indications and contraindications for reimplantation?

- **Indications:** Multiple finger injury; thumb amputations (especially proximal to interphalangeal joint); single finger injury in children; clean amputation at hand, wrist, or distal forearm.
- **Contraindications:** Severe crush or avulsion, heavy contamination, single-finger amputations in adults, severe associated medical problems or injuries, severe multilevel injury of amputated part, willful self-amputation. Bottom line: Give the hand surgeon the opportunity to decide.

25. Which are the most deceptive of all serious hand injuries?

High-pressure injection injuries (from paint guns, grease guns, or hydraulic lines) initially may seem innocuous, often involving just the fingertip. In one published case series, 6 out of 15 high-pressure injection injuries resulted in some form of amputation, and only one patient regained normal sensation, despite aggressive early surgical treatment.

26. List Kanavel's four cardinal signs of flexor tenosynovitis.

- Slightly flexed posture of the digit
- Fusiform swelling of the digit
- Pain on passive extension
- Tenderness along the flexor tendon sheath

Flexor tenosynovitis requires admission and surgery.

27. What is a paronychia? How is it treated?

A common bacterial infection involving the folds of skin that hold the fingernail in place. In the absence of visible pus, treatment should consist of warm moist compresses, elevation, and antistaphylococcal antibiotics. If pus is present, do the minimum necessary to drain and maintain drainage. This usually consists of simply elevating the eponychial fold or making a small incision. Sometimes removal of a longitudinal section of the nail plate is necessary.

28. How is whitlow different from a paronychia?

Whitlow is infection of the tissue around the nail plate with herpes simplex virus (rather than bacteria). The discharge is serous and crusting rather than purulent. The patient also may have perioral cold sores. Do **not** incise and drain herpetic whitlow.

29. What is a felon? How is it treated?

A painful and potentially disabling infection of the fingertip pulp. Treatment is controversial. Some clinicians argue for immediate drainage of the tensely swollen and painful fingertip pad. Others argue that early treatment with antibiotics, elevation, and immobilization may prevent

the need for surgical drainage. Even if drainage is necessary, the best method is also a matter of controversy. The full fishmouth incision has fallen out of favor, but the three-quarter fishmouth incision and the simple lateral incision are both acceptable.

30. What is a football jersey finger? How is it treated?

Rupture of the FDP occurs commonly when a football player catches his finger in an opponent's jersey. The tendon is avulsed from its insertion at the palmar base of the distal phalanx, often taking a bone fragment along. Surgical repair within the next several days is indicated.

31. What is a mallet finger? How is it treated?

A mallet finger is the opposite of a football jersey finger; the insertion of the extensor tendon, rather than the flexor tendon, is avulsed from the dorsum of the distal phalanx, often pulling off a bone fragment. Appropriate treatment is to splint the DIP joint in extension (not hyperextension) for 6 weeks.

32. Describe a subungual hematoma. How is it treated?

A collection of blood under the nail plate can be painful. Classically, this occurs when a weekend carpenter strikes his or her thumb with a hammer. Relieving the pressure by nail trephination (poking a hole in the nail) will make you a hero to the patient. Use electrocautery, a red-hot paperclip, or an 18-gauge needle (twisting it between your fingers like a drill bit). Removal of an intact nail plate is almost never indicated.

33. What is a gamekeeper's thumb? How is it diagnosed?

A torn ulnar collateral ligament of the thumb MCP joint resulting from forceful abduction of the thumb. In 1955, the injury was reported in 24 Scottish game wardens, arising from their technique for breaking the necks of wounded rabbits. The injury is more properly called **skier's thumb** because it most commonly occurs when a skier either catches the thumb on a planted ski pole or falls while holding a pole in the outstretched hand. The injury is potentially severely disabling. Complete rupture of the ligament always requires surgery. ED treatment consists of a thumb spica splint and referral. One way to test for injury to the ulnar collateral ligament of the thumb MCP is to hand the patient a heavy can (e.g., of soda) or bottle (e.g., of hydrogen peroxide). If the injury is present, the patient will be unable to hold the object in the usual way, either supinating to balance the object in the palm or dropping it.

34. What is a boxer's fracture?

Fracture of the fifth (small finger) metacarpal is common in barefisted pugilists. Because the small finger metacarpal is second only to the thumb metacarpal in mobility, large angles of angulation are tolerated without functional deficit. Nevertheless, attempts to correct significant angulation of an acute boxer's fracture are warranted. Any rotational deformity must be corrected. A laceration accompanying a boxer's fracture is assumed to be a fight bite.

35. What is a fight bite?

The most notorious of all nonvenomous bite wounds is the fight bite. As the name implies, the injury occurs when the soon-to-be-patient punches his or her adversary in the teeth, lacerating the dorsum of one or more MCP joints. Other names for this injury such as **morsus humanus** or **closed fist injury** have been proposed. **Fight bite** is more compact, descriptive, and poetic. All such wounds require formal exploration, including extension of the skin laceration if necessary. They should be débrided, irrigated, dressed open (no sutures), and splinted. If the wound penetrates the extensor hood, the MCP joint requires thorough washout, and strong consideration should be given to hospitalization for intravenous (IV) antibiotics and meticulous wound care. If the wound is already infected, hospitalization is mandatory.

KEY POINTS: HAND INJURIES AND INFECTIONS

1. Because scaphoid fractures are frequently radiographically occult, even without X-ray evidence of a fracture, the patient with tenderness to palpation of the anatomic snuffbox should be treated with a thumb spica splint and repeat evaluation in 1 to 2 weeks.
2. The most deceptive of serious hand injuries is the high-pressure injection injury sustained while testing a hydraulic paint or oil gun because, despite seeming innocuous on initial presentation, these injuries require aggressive, surgical management.
3. Any laceration over the dorsal MCP joint is suspicious for a fight bite. Fight bites require meticulous exploration and wound care. If the wound penetrates the extensor hood, thorough joint washout and IV antibiotics are required.

36. Are human bites more dangerous than other animal bites?

No. The fight bite gave human bites their reputation for being more prone to infection than other animal bites. This probably has more to do with the location of the bite and the typical delay in treatment than with the mix of organisms in the human mouth. True human bites (occlusive bites rather than fight bites) have no higher infection rates than animal bites. If humans punched animals in the teeth, these animal fight bites would have high infection rates also.

37. Name six true hand emergencies.

- Amputation or other devascularization injury
- Compartment syndrome
- Third-degree and circumferential burns
- High-pressure injection injury
- Flexor tenosynovitis
- Septic joint

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CHAPTER 93

BURNS

Jeffrey S. Guy, MD

1. How should thermal burn patients be initially assessed in the ED?

Always start with the ABCs. Airway evaluation for inhalation injury is critical early in the patient's course. Assess respiratory status, and provide supplemental oxygen if necessary. Evaluate for circulation and hemodynamic status with vital signs, pulses, and capillary refill. The ABCs should be followed by a complete secondary survey including evaluation of burn size and depth, associated trauma, and possible cervical spine injury. As with any ED patient, past medical history, medications, allergies, and tetanus immunization status are also important. Also, it is important to speak with the emergency medical service (EMS) transport team to determine if the patient was trapped in an enclosed space or if exposure to toxic substances is a concern.

KEY POINTS: TYPES OF BURNS COMMONLY SEEN IN THE ED

1. Thermal (most common)
2. Solar (i.e., sunburn)
3. Chemical
4. Electrical
5. Radiation

2. How do you evaluate for inhalation injury?

Risk factors for inhalation injury include flame burns, exposure to smoke in an enclosed space, and associated trauma. Signs of possible inhalation injury are burns around the face and mouth, soot in the nose or mouth, and carbonaceous sputum. Respiratory symptoms such as dyspnea, hoarseness, wheezing, and stridor are highly suggestive of inhalation injury.

3. How does smoke from a fire cause asphyxiation?

Two asphyxiates produced in fires include carbon monoxide (CO) and cyanide gas (CN) (see Chapter 72). Both of these molecules cause cellular death from cellular hypoxia and asphyxia. In these cases, the patient often has an airway and is capable of being ventilated, but the tissues are not capable of utilizing the oxygen that is delivered to them. Portable carbon monoxide monitors are available for use in the prehospital environment to aid in the detection of CO. These monitors are helpful in evaluating both patients and firefighters. Treatment of CO toxicity is removal from the source and administration of oxygen. When the patient is placed on 100% oxygen, the half-life of CO-hemoglobin is reduced to 40 to 60 minutes.

Burning plastics and polyurethane produce cyanide gas. Patients with carbon monoxide toxicity should be considered at risk for CN poisoning. A safe cyanide antidote, hydroxocobalamin, is approved and available for prehospital and hospital use in the United States and Europe.

4. What about thermal burns to the airway?

Because of the great capacity for heat dissipation in the oropharynx and nasopharynx, thermal burns to the lower airways are uncommon except in the setting of steam inhalation. Direct thermal burns to the airway are usually limited to the upper airway and present as mucosal edema, erythema, and ulceration.

5. What are the indications for intubation in the burn patient?

Inhalation injury can produce a rapid progression of edema that can make orotracheal intubation difficult or impossible. Evidence of airway involvement (voice changes, stridor, wheezing, mucosal edema) or low oxygen saturation is an indication for observation in an intensive care unit capable of providing advanced airway management or early intubation in the ED. It should also be considered in patients with severe burns to the face and neck, even if initial respiratory status is adequate.

6. Are there any specific issues related to intubation in the burn patient?

Again, early intubation is the key to avoiding complications of airway edema and possible *crash* intubation. Orotracheal rapid-sequence intubation (RSI) is appropriate. Fiberoptic intubation can be considered in the stable patient. Emergency cricothyrotomy may be necessary if airway edema prevents orotracheal tube placement. Remember, many RSI drugs can contribute to hypotension. Avoid complications with adequate fluid resuscitation and careful selection of RSI medications.

7. What about succinylcholine?

Succinylcholine is frequently reported as contraindicated in the burn patient due to changes in muscle receptors that can cause hyperkalemia. These changes generally take place over the first 7 to 10 days after the burn. This is not a concern in the acute burn patient encountered in the ED.

8. What are some options for securing an endotracheal tube in a patient with burns to the face?

Burned facial skin commonly shears off and weeps fluid, making adhesive tapes ineffective. Loss of the airway in these patients can be catastrophic and require an emergent surgical airway. Several commercial devices exist to secure these tubes. An effective alternative to such commercial devices is to secure the endotracheal tube with umbilical tape or intravenous tubing. The provider should use two ties to secure the tube. One tie should go over the ears and one tie should drape under the ears. A common time for displacement of the airway is with movement of the patient. For that reason, the position of the airway should always be reconfirmed following each movement of the patient.

9. How is the burn injury evaluated?

Begin with a complete physical examination including the back and the perineum. Document the locations burned and the depth of the burns.

- **First-degree burns** involve only the epidermis and are erythematous and painful, without blisters. They are usually described as looking like a sunburn. These do not count toward the total body surface area (TBSA) when calculating the burn size.
- **Second-degree burns** are characterized as superficial or deep partial-thickness burns. They are painful, and their color can vary from red to mottled to pale. Blisters may be thin- or thick-walled depending on the depth of the burn. *Superficial partial-thickness burns* are erythematous and have thin-walled fluid-filled blisters. These usually heal in 2 to 3 weeks without scarring. *Deep partial-thickness burns* extend further into the dermis, are usually pale pink or mottled, and have thick-walled blisters. These burns will usually heal in 3 to 9 weeks but tend to develop hypertrophic scars; surgical treatment is usually necessary.
- **Third-degree burns** involve all layers of the dermis. The skin is firm, white, or charred, and is often described as "leathery." This represents complete tissue destruction, and surgery is necessary except in the smallest of third-degree burns.
- **Fourth-degree burns** extend to deeper tissues including subcutaneous fat, muscle, and bone. Significant debridement and reconstruction are required.

10. What is so concerning about circumferential full-thickness burns?

As fluid leaks from damaged tissues into adjacent soft tissues, the leather-like full-thickness burn prevents tissue expansion, thus causing compression of internal structures. Circumferential burns of the neck can lead to compression of the jugular veins and increased intracranial pressure or airway compromise. Circumferential burns of the chest can lead to decreased chest wall compliance, increasing difficulty ventilating, and respiratory insufficiency. Circumferential burns to the extremities can cause vascular compromise similar to compartment syndrome. Evaluation of pulses and Doppler signals is absolutely necessary. Signs of poor perfusion to a distal extremity are cyanosis, deep tissue pain, paresthesias, and cold skin. Prompt escharotomy can be performed by the emergency physician or a surgeon.

11. After a thermal burn, what is the best method to stop the burning process?

The initial care of the burn patient is to stop the burning process. Remove clothing and jewelry as these items will retain residual heat. The most effective cooling technique is irrigation with copious amounts of room-temperature water. Immediate cooling with tap water (15° C) is almost twice as effective in reducing temperature within burned tissue than application of a product such as Melaleuca Alternifola hydrogel. Smaller burns that have been cooled have been shown to have less cellular damage than those that are not cooled. Application of ice is contraindicated. Ice will stop the burning process and provide analgesia but will increase the extent of tissue damage. In fact, cooling the burn immediately with ice is more harmful than application of tap water or no treatment at all. Too aggressive cooling will lead to tissue damage, similar to what is experienced with frost bite. The hazard of cooling larger burns is causing the patient to become hypothermic.

12. How is % TBSA burned calculated? Why is it important?

Estimation of the percentage of body surface area burned helps direct fluid resuscitation, determines appropriate disposition of the burn patient, and allows meaningful communication with consultants and burn units. There are several methods to estimate % TBSA burned. The rule of nines divides the regions of the body into approximate percentages of total surface area (Fig. 93-1). Due to differences in body proportions, the rule of nines is not applicable to children. The Lund and Browder chart has similar divisions but allows for variations in infancy and childhood (Fig. 93-2). With either chart, document the areas of second- and third-degree burns on the chart and calculate a total percentage of TBSA burned.

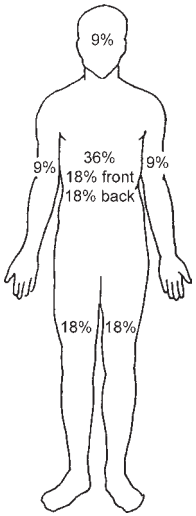

Figure 93-1. Percentages used in determining extent of burn by rule of nines. (From Miller RH: *Textbook of basic emergency medicine*, ed 2, St. Louis, 1980, Mosby.)

13. What is burn shock?

Burn shock is a complex interaction of intravascular fluid loss and release of vasoactive substances and inflammatory mediators. Initial fluid loss is due to tissue destruction at the burn site that causes increased vascular permeability. Fluid shifts into the extravascular space and is quickly lost through the damaged skin.

14. How do you determine appropriate fluid resuscitation in the burn patient?

Following a burn injury, the patient has profound shifts in intravascular fluids and an initial reduction in cardiac output. Several formulas exist for the determination of fluid resuscitation needs; however, the Parkland formula is the most well known and most widely applied. The Parkland formula provides an estimate of the initial fluid needs of the severely burned patient. The fluid requirement (of lactated Ringer's solution) is calculated

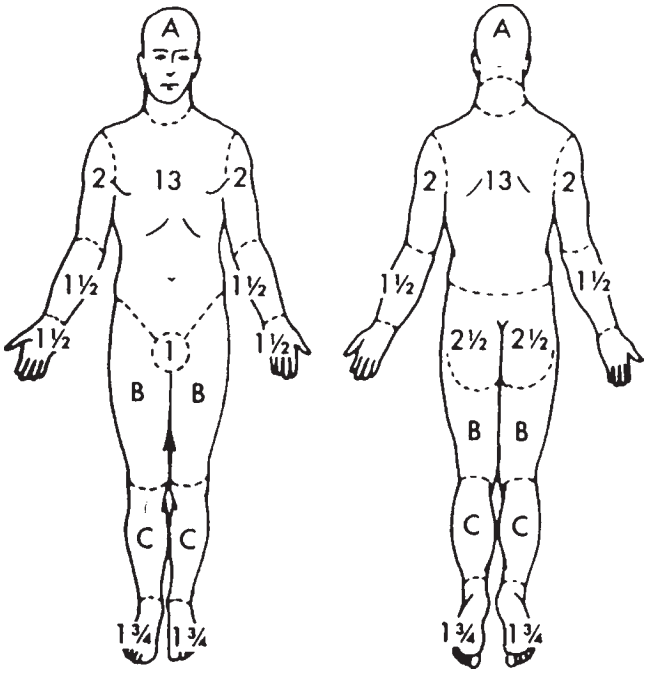

Figure. 93-2 Classic Lund and Bowden chart. The best method for determining percentage of body surface burn is to mark areas of injury on a chart and then compute total percentage according to patient's age. (From Artz CP, Yarbrough DR III: Burns: Including cold, chemical, and electrical injuries. In Sabiston DC Jr, editor: *Textbook of surgery*, ed 11, Philadelphia, 1977, WB Saunders.)

for the first 24 hours. One half of this is administered over the first 8 hours. The second half is administered over the following 16 hours.

Fluid required = body weight(kg) × %TBSA burn (second and third degree) × 4 mL

Remember, this is an estimate. Monitor adequacy of fluid resuscitation by following vital signs and urine output. Goal urine output is 30 mL/hr in adults and 1 to 2 mL/kg/hr in children. (Do not forget the Foley catheter.)

15. Can you give me an example of using the Parkland formula?

Let's use an example of an 80-kg patient who has sustained a 30% body surface area burn.

24-hour fluid total = 4 mL/kg/% TBSA burn
= 4 mL/kg/% TBSA burn × 80 kg × 30% TBSA burn
= 9,600 mL

- Once you have calculated the 24-hour fluid requirement, divide that number by two. Half of that fluid should be given in the first 8 hours from injury, and the second half in the next 16 hours.
- Fluids given in the first 8 hours: 9,600 mL/2 = 4800 mL
- To determine the hourly rate for the first 8 hours, divide this number by 8.
- Fluid rate in first 8 hours = 4,800 mL/8 hours = 600 mL/hr
- The fluid rate for hours 8 to 24 is calculated by dividing 9,600 mL by 16.
- Fluid rate for hours 8 to 24= 4,800 mL/16 hours = 300 mL/hr

16. Are there any pitfalls with using the Parkland formula?

Yes, there are some common pitfalls with fluid resuscitation. The first is that a formal Parkland fluid resuscitation is not required for burns less than 20% TBSA. Another detail concerns those patients who may present to the ED several hours after injury. For example, if the patient presented earlier were to arrive to the ED 2 hours after injury not having received any resuscitative fluids, then the provider would need to recalculate the fluid rate for the fluid to be given for the first 8 hours after injury. The first half of the fluids should be administered within 8 hours from the time of the injury, not from the time after presentation. Therefore, if the patient presents two hours after injury, the initial fluid requirement would now have to be given over the remaining 6 hours, and the rate for fluid resuscitation would be:

- Fluid resuscitation hours 2 to 8= 4,800 mL/6 hours = 800 mL/hr
- The fluid rate for remaining 16 hours would remain at 300 mL/hr

17. How do you manage a patient with burns *and* trauma?

Patients with combined burns and trauma are at greater risk for morbidity and mortality than either alone. These patients are at higher risk for inhalation injury and tend to require greater fluid resuscitation than isolated burn patients. Generally, aggressive burn resuscitation should be started, and life-threatening traumatic injuries should be treated initially. Transfer to a burn unit can be delayed until the traumatic injuries have been stabilized.

18. What are the criteria for referral to a burn center?

The American Burn Association has developed a list of criteria that warrant referral to a specialized burn unit. Their website also contains a list of verified burn centers. Referral does not always require transfer to the burn unit but may include instruction in wound treatment or plans for follow-up.

- Partial-thickness burns > 10% TBSA
- Burns that involve the face, hands, feet, genitalia, perineum, or major joints
- Third-degree burns
- Electrical burns, including lightning injury
- Chemical burns
- Inhalation injury
- Burns in patients with preexisting medical disorders that could complicate management, prolong recovery, or affect mortality
- Any patient with burns and concomitant trauma in which the burn injury poses the greatest risk of morbidity or mortality
- Burned children in hospitals without qualified personnel or equipment for the care of children
- Burn injury in patients who will require special social, emotional, or long-term rehabilitative intervention

19. Are there any burns that can be managed in the outpatient setting?

Partial-thickness burns involving less than 10% TBSA or full-thickness burns involving less than 2% TBSA without risk of functional or cosmetic complications can usually be treated as an outpatient.

20. How are outpatient burns treated in the ED?

All burns should be cleaned with saline or water and a mild cleanser. Ruptured blisters should be débrided. There is controversy in the burn community over treatment of intact blisters; most sources recommend débridement of the blister or aspiration of the fluid to prevent infection of the blister fluid. Topical antibiotics such as bacitracin ointment or silver sulfadiazine should be applied liberally and covered with a nonadhesive dressing and a bulky bandage. The patient should follow up in 24 hours in the ED or with a physician experienced

in burn care. If there are any questions concerning outpatient management, consultation with a burn center is appropriate.

21. Is there anything special about facial burns?

Facial burns should not be treated with silver sulfadiazine because it can cause pigmentation changes in the healing tissue. Antibiotic ointment alone, without dressings, is adequate for facial burns. Again, follow-up in 24 hours is recommended.

22. What about pain control?

Burns are painful, and large doses of narcotics are sometimes necessary. Intravenous narcotics can be started in the field and repeated if needed. Adequate pain control may require substantial doses of pain medications. These should only be withheld if administration is life threatening due to hemodynamic status. Also, don't forget to provide adequate oral analgesics for outpatient management. Superficial partial-thickness burns can be the most painful.

23. Should I check tetanus immunization status?

Always check the status of tetanus immunization and update if needed.

24. What are the special considerations in burn care in children?

Children younger than 2 years have increased morbidity and mortality from burns. Consider admission even in minor burns in this age group. Do not forget the possibility of nonaccidental trauma. Twenty percent of pediatric burns are the result of intentional burn injury. Ask about conditions around the home, and assess for safety. The reliability of the parents is another consideration in evaluating need for inpatient management of a pediatric burn. Do not forget that any suspicion of child abuse must be reported to child protective services.

25. How can I recognize intentional burns in children?

The most common form of intentional burn injury in children is forcible immersion. These types of injuries commonly occur when an adult places a child in hot water as a form of punishment, often associated with toilet training. These children will commonly have second- and third-degree burns of the hands and feet in a glove or stocking type pattern. These injuries are especially suspicious when the burns are symmetrical and lack splash patterns. When a child is lowered into hot water, the child will assume a defensive posture tightly flexing their arms and legs. These burns will produce sparing of the flexion creases in the antecubital fossa, popliteal fossa, and groins.

Contact burns are the second most common form of burn-related child abuse. Accidental contact burns typically have irregular burn depth and edges. This is because dropped hot objects will often strike and deflect off the curvature of the various body surfaces. With intentional contact burns, the instrument causing the burn is typically pressed onto the body surface. This therefore produces a burn pattern that has sharp lines of demarcation.

26. What are the special considerations in adults?

Past medical history is important. Patients with HIV, immune suppression (transplants, steroids), diabetes, cardiac disease, chronic obstructive pulmonary disease (COPD), or substance abuse may all require inpatient management of minor burns to monitor and treat complications of the chronic disease. Also, burn patients older than 60 years are at increased risk of morbidity and mortality from their injuries.

WEBSITE

American Burn Association: www.ameriburn.org

ACKNOWLEDGMENT

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WOUND MANAGEMENT

Maria E. Moreira, MD, and Vincent J. Markovchick, MD, FAAEM, FACEP

CHAPTER 94

1. Why is wound management important?

Annually, approximately 12 million traumatic wounds are treated in EDs across the United States constituting about 10% of all ED visits. Patients often judge the competency of a physician based on the ultimate functional or cosmetic result of the wound repair and the development of complications.

2. What is the difference between functional and cosmetic closure?

Functional closure is closure of a wound prioritizing returning the injured part to full function. A **cosmetic repair** prioritizes obtaining the least amount of scarring.

3. How do I remember what steps to take when repairing a wound?

Use the mnemonic LACERATE:

- **L** = **Look.** Evaluate the wound to determine the most appropriate closure. Examine thoroughly for movement, sensation, and pulsation distal to the wound.
- **A** = **Anesthetize.**
- **C** = **Clip and clean.** Clipping hair leads to less infection than shaving. Methodical irrigation is the best way to decrease infection risk.
- **E** = **Equipment.** Have everything needed for repair at the bedside, including laceration kit, sterile gloves, suture material, and dressing.
- **R** = **Repair.** Perform the repair. Devitalized tissue may need to be débrided.
- **A** = **Assess results.** Reevaluate the wound when the repair is near completion to determine the need for additional sutures.
- **T** = **Tetanus.** Give tetanus prophylaxis for dirty or contaminated wounds when the patient has not had a booster in 5 years or for clean wounds when the patient has not had a booster in 10 years.
- **E** = **Educate.** Educate the patient on how to care for the wound, signs of infection, and the timing of suture removal.

4. Which factors increase the visibility of scars and compromise wound healing? How are they minimized?

See Table 94-1.

5. What aspects of history should be obtained in a patient with a traumatic wound?

The time, setting, and mechanism of injury are essential to determine whether the wound is contaminated, the possibility of foreign body, or the potential for infection. The patient's current medications and immune status (AIDS, diabetes, chemotherapy), the patient's occupation, and dominant hand if a hand injury has occurred are important. The patient's tetanus immunization history and allergies (specifically regarding anesthetics, antibiotics, or latex gloves) must be obtained.

6. What are the most important aspects of the physical examination?

To perform an adequate examination, it is important to be familiar with underlying anatomy, especially in the regions of the face, neck, hands, and feet. Examination of the injured site should begin with identification of any motor, sensory, and vascular deficits. With extremity

TABLE 94–1 MINIMIZING FACTORS THAT INCREASE VISIBILITY OF SCARS

Contributing Factors	Methods to Minimize Scarring
Direction of wound (e.g., perpendicular to lines of static and dynamic tension)	Layered closure; proper direction in elective incisions of wound
Infection necessitating removal of sutures débridement, resulting in healing by secondary intention and a wide scar	Proper wound preparation; irrigation and use of delayed closure in contaminated wounds
Wide scar secondary to tension	Layered closure; proper splinting and elevation
Suture marks	Removal of percutaneous sutures within 7 days
Uneven wound edges resulting in magnification edges and of scar by shadows	Careful, even approximation of wound top layer closure to prevent differential swelling of edges
Inversion of wound edges	Proper placement of simple sutures or use of horizontal mattress sutures
Tattooing secondary to retained dirt or foreign body	Proper wound preparation and débridement
Tissue necrosis	Use of corner sutures on flaps, splinting, and elevation of wounds with marginal circulation or venous return; excise nonviable wound edges before closure
Compromised healing secondary to hematoma	Use of properly conforming dressing and splints
Hyperpigmentation of scar or abraded skin	Use of 15 or greater SPF sunblock for 6 months
Superimposition of blood clots between healing frequent wound edges	Proper hemostasis and closure; H_2O_2 swabbing; proper application of compressive dressings
Failure to align anatomic structures properly such as vermilion border	Meticulous closure and alignment; marking or placement of alignment suture before distortion of wound edges with local anesthesia; use of field block

From Markovchick V: Suture materials and mechanical after care. *Emerg Med Clin North Am* 10: 673–689, 1992.

injuries, examination can be conducted in the absence of hemorrhage by temporarily inflating a sphygmomanometer or placement of a finger tourniquet proximal to the injury. Palpation of the bones adjacent to the site of injury may detect instability or point tenderness from an underlying fracture. Direct inspection and visualization always should be performed when there is a suspicion of a tendon or joint capsule injury or presence of a foreign body.

7. What is the most important step I can take to prevent infection?

1. For all traumatic wounds, irrigation with normal saline at least 8 psi should be done with an 18- or 19-gauge needle and a 30-mL syringe. The optimal volume of irrigant has not been determined; however, 50 mL to 100 mL per centimeter of wound length has been used as a guideline. In the presence of gross contamination, copious irrigation should be done and débridement considered. Tap water is a reasonable alternative for wound irrigation. Detergents, hydrogen peroxide, and concentrated povidone-iodine should not be used for irrigation because they are toxic to tissues. Exploration; débridement when indicated; hemostasis; and proper repair, dressing, and immobilization are essential adjuncts for proper wound management. Antibiotics have no proven prophylactic benefit in the normal host. For contaminated or dirty extensive wounds, a mechanical irrigation device should be used to remove all dirt and decrease the bacterial count. A stiff brush such as a toothbrush or sharp débridement should be used to remove dirt that remains after irrigation.

8. Which anesthetic agent should be used for local anesthesia?

Selection of an appropriate anesthetic depends on many factors, including age of the patient, underlying health, prior drug reactions, wound size and location, and practice environment in the ED. Lidocaine traditionally has been the standard agent for local anesthesia in the ED; however, bupivacaine has advantages over lidocaine, related mainly to duration of anesthesia. Patients receiving bupivacaine experience significantly less discomfort during the 6-hour postinfiltration period. Also, in a busy ED, use of bupivacaine may prevent the need to reanesthetize a wound when repair has been interrupted by the arrival of a higher acuity patient.

9. What causes the pain of local anesthetic infiltration, and how can it be prevented?

Pain from anesthetic infiltration is caused by distention of tissue from too-rapid injection with too large a needle directly into the dermis. The acidity of the agent also contributes to the pain. Pain from infiltration can be minimized by injecting slowly, subcutaneously, with a small, 25- or 27-gauge needle, directly through the wound margins. Buffering the anesthetic agent with 1 mL of sodium bicarbonate for every 10 mL of lidocaine also can help to reduce pain. However, bupivacaine does not lend itself to buffering because it precipitates as its pH rises. Another efficacious and inexpensive method of decreasing the pain of infiltration is by warming the anesthetic.

10. What is the toxic dose of lidocaine and bupivacaine?

Table 94-2 summarizes the maximum dose and duration of action of lidocaine, bupivacaine, and procaine, alone and in combination with epinephrine. When calculating the dose of milligrams infiltrated, 1 mL of 1% lidocaine = 10 mg of lidocaine and 1 mL of 0.25% bupivacaine = 2.5 mg of bupivacaine. Lower maximal doses should be used for patients with chronic illness, for very young or very old patients, or when infiltrating highly vascular areas or mucosa.

11. Describe the presentation of lidocaine toxicity.

In general, toxicity should not occur unless the recommended dosing is met or exceeded. The caveat to that statement is that toxicity may take place at lower than maximal doses when infiltrating highly vascular areas or mucous membranes or in patients who are at the extremes of age or chronically ill. The main effects are on the central nervous and cardiovascular systems. Central nervous system effects present as lightheadedness, nystagmus, and sensory

TABLE 94–2 MAXIMUM DOSE AND DURATION OF ACTION OF ANESTHETICS

Anesthetic	Class	Maximum Dose	Duration
Lidocaine	Amide	4.5 mg/kg	1–2 hours
Lidocaine with epinephrine	Amide	7 mg/kg	2–4 hours
Bupivacaine	Amide	2 mg/kg	4–8 hours
Bupivacaine with epinephrine	Amide	3 mg/kg	8–16 hours
Procaine	Ester	7 mg/kg	15–45 minutes
Procaine with epinephrine	Ester	9 mg/kg	30–60 minutes

disturbances, including visual aura or scotoma, tinnitus, perioral tingling, or a metallic taste in the mouth. Slurred speech, disorientation, muscle twitching, and finally seizures may follow. The cardiovascular effects are manifested by hypotension, bradycardia, and prolonged electrocardiogram (ECG) intervals. In severe toxicity, the end result is seizures, coma, and cardiorespiratory arrest.

12. What can I use to anesthetize a patient who is allergic to amide and ester anesthetics?

Subdermal diphenhydramine may be injected locally to obtain short-acting analgesia. Prepare a 0.5 to 1.0% solution by diluting 1 mL of 50 mg/mL diphenhydramine into 5 to 10 mL of saline. The anesthetic effect may take several minutes to become evident. Do not exceed a total dose of 50 mg in adults or 1 mg/kg in children. The patient may become drowsy after the injection.

13. What are the contraindications to epinephrine as an adjunct to lidocaine and bupivacaine?

Anesthetics with epinephrine should not be used on digits, the pinna, circumferentially around the penis, or in areas with poor or marginal blood supply, such as flap wounds of the anterior pretibial area. Epinephrine decreases resistance to infection because of its potent vasoconstrictor effect. In areas of the body such as the scalp and face, the vasoconstriction and resulting hemostasis aid in the exploration and repair of the wound and do not seem to increase wound infection.

14. What is LET (LAT)?

A topical anesthetic that consists of a mixture of **lidocaine** (4%), **epinephrine (adrenaline)** 1:1000, and **tetracaine** (0.5%). LET has been shown to be efficacious for wound anesthesia and is the topical agent of choice. It has a good margin of safety. For optimal effect it should be placed directly into the wound.

15. What are the contraindications to LET?

They are the same as for lidocaine or bupivacaine with epinephrine.

16. When do I use procedural sedation?

Procedural sedation is a pharmacologic means of lowering the level of consciousness to allow procedures to be performed easily with optimal results. See Chapter 66.

17. What is a contaminated wound?

Any wound that has a high inoculum of bacteria. Some examples are:

- Full-thickness bites
- Wounds of the perineum or axilla where there is normally a high skin flora count
- Wounds that are exposed to contaminated water, such as from ponds, lakes, or coral reefs

18. List factors that contribute to wound infection.

- Wound age
- Presence of foreign material
- Amount of devitalized tissue
- Presence of bacterial contamination
- Advanced patient age
- Ability of the host to mount an adequate immune response

19. Is a dirty wound the same as a contaminated wound?

No. **Road rash**, resulting from road gravel, has a low bacterial count. In contrast, wounds that occur in a barnyard or are exposed to soil contaminated with fecal material have a high bacterial count and are contaminated.

20. What causes tattooing?

The retention of foreign material and incorporation of it in the dermis during the healing process. To prevent this cosmetic complication, all foreign material and dirt must be removed through proper débridement, scrubbing, and irrigation at the time of the initial patient encounter. A stiff brush, such as a toothbrush, and soap are useful to remove dirt and asphalt embedded in the dermis.

21. How do I treat road rash?

Anesthetize the area with viscous lidocaine and circumferential or field block anesthesia. Remove all foreign bodies with the methods described previously. Consider dressing with **silver sulfadiazine**, which greatly reduces the pain and may obviate the need for potent oral analgesics for deep, extensive, painful abrasions.

22. When do I get an X-ray?

Radiographs are useful to search for a foreign body or to look for an associated fracture. Obtain a radiograph if the history is suspicious for a foreign body (e.g., broken glass) and the wound penetrates muscle fascia or the entire depth of the wound cannot be visualized. In the case of some bite wounds or lip lacerations with broken or avulsed teeth, radiographs should be considered to search for teeth. With severe pain or structural instability, radiographs may reveal an underlying open fracture, which necessitates an orthopedic consultation in most cases.

23. Which types of foreign bodies found in wounds are visible on radiographs?

Glass, metal, and gravel. In general, glass larger than 2 mm and gravel larger than 1 mm can be seen on radiographs. Foreign bodies that are radiolucent (not visible on radiographs) include wood, plastics, and some aluminum products.

24. What is the best method for hair removal?

Clipping or cutting hair with scissors as opposed to shaving has been shown to result in lower wound bacterial counts and decreased rates of infection.

25. Define the three different types of wound closure.

- **Primary closure** is closure of wound margins with sutures, staples, glues, or adhesive tapes within 24 hours of the time of injury.
- **Delayed primary** closure is closure of a wound 3-5 days after wounding to decrease the risk of infection.
- **Secondary closure**, or healing by secondary intention, is allowing a wound to heal by granulation without mechanical approximation of the wound margins.

26. Which wounds should be closed primarily?

Any clean (not initially contaminated) wound if it is less than 6 to 8 hours old and is located anywhere on the body except for the face and scalp, which may be closed primarily up to 24 hours because of the rich vascular supply and resistance to infection.

27. When should secondary closure be used?

For contaminated wounds that penetrate deeply into tissue and cannot be irrigated adequately before closure. Examples of such wounds are puncture wounds of the sole of the foot or palm of the hand and stab wounds that penetrate into subcutaneous tissue and muscle.

28. When should delayed primary closure be used?

It should be strongly considered for all contaminated wounds that are gaping or have significant amounts of tension. It decreases the risk of infection, optimizes the cosmetic result, and accelerates the healing process.

29. How is a wound prepared for delayed primary closure?

The wound should be examined thoroughly, débrided, and irrigated. Hemorrhage should be controlled. A fine layer of mesh gauze should be laid in the wound; the wound should be packed open and followed closely. At 3 to 5 days, if there is no purulent drainage or wound-margin erythema, the wound may be closed in the same fashion as if it were being closed primarily.

30. What is the most important step when closing a lip laceration through the vermilion border?

Placement of the first suture at the vermilion border. Use nonabsorbable suture to close the edges of the vermilion border. Be sure to line up the edges precisely. Failure to do so will result in a visible cosmetic defect. The remainder of the lip should be closed with absorbable suture. The skin should be closed with nonabsorbable suture.

31. When are surgical staples indicated?

To reapproximate linear lacerations that do not involve cosmetically sensitive areas such as the face. Two approaches are commonly employed. One approach involves two operators with one everting both wound edges with forceps while the other staples the wound together. If only one operator is available, the wound edges should be aligned and one edge everted with forceps in one hand while stapling with the other. Staples work best in wounds that are perpendicular, that is, 90 degrees to the surface, rather than with shelving angular lacerations because these tend to overlap.

32. What is surgical glue, and how is it used?

Surgical glue is 2-0ctyl cyanoacrylate (Dermabond) is a polymer currently being used as an alternative for wound repair. Cyanoacrylate acts rapidly, polymerizing within 30 seconds at room air. It is best used for linear lacerations under low tension and may replace 5-0 or 6-0 sutures. The wound can be held together manually, and the cyanoacrylate can be painted over the wound in three to four coats to ensure adequate closure. Be careful not to apply any adhesive within the wound because this will impede healing. The adhesive sloughs off in 7 to 10 days. Do not use antibiotic ointment or any other type of ointment on the wound because it destroys the adhesive bond.

33. How do I remove tissue adhesive?

First, avoid getting tissue adhesive into undesirable areas by applying protective covering and petroleum jelly to areas surrounding the wound. Apply light coats of the adhesive and quickly wipe off excess fluid. You have about 15 seconds before the adhesive dries. If the adhesive dries on an undesirable area (e.g., eyelid glued shut), the bond may be loosened with petroleum jelly or antibiotic ointment.

34. Summarize the advantages and disadvantages of the available techniques for wound closure.

See Table 94-3.

TABLE 94–3. ADVANTAGES AND DISADVANTAGES OF WOUND CLOSURE TECHNIQUES

Technique	Advantages	Disadvantages
Sutures	Time-honored method Meticulous closure Greatest tensile strength Lowest dehiscence rate	Removal required Anesthesia required Greatest tissue reactivity Slowness of application
Staples	Rapidity of application Low tissue reactivity Low cost	Less meticulous closure than with sutures May interfere with CT and MRI May result in uneven wound edges
Tissue adhesives	Rapidity of application Patient comfort Resistance to bacterial growth No need for removal Sometimes no need for needle stick	Lower tensile strength than sutures Dehiscence over high-tension areas (joints) Wound healing inhibited if placed in the wound High cost
Surgical tapes	Least tissue reactivity Lowest infection rates Rapidity of application Patient comfort Low cost	Lower tensile strength than sutures Highest rate of dehiscence Cannot be used in hairy areas Must remain dry

CT, computed tomography; MRI, magnetic resonance imaging.
From Singer AJ, Hollander JE, Quinn JV: Evaluation and management of traumatic lacerations. *N Engl J Med* 337:1142–1148. Copyright ©1997 Massachusetts Medical Society. All rights reserved.

35. Which sutures are used, how is the wound repaired, and when do I remove the sutures?

See Table 94-4.

36. How are bites treated?

See Figure 94-1.

37. What should be included in all follow-up instructions?

Instructions on local wound care, signs of infection, and time of suture removal. Antimicrobial ointment may be applied to decrease the risk of infection; however, when tissue adhesives have been used, ointments dissolve the adhesive and may cause separation of the wound. Sunlight should be avoided, and sunscreen should be used to help minimize hyperpigmentation and scarring. Inform patients that all wounds will heal with a scar, all wounds may get infected, and all wounds may have retained foreign material.

TABLE 94–4 USE OF SUTURES FOR WOUND REPAIR

Location	Suture Material	Technique of Closure and Dressing	Suture Removal
Scalp	3–0 or 4–0 nylon or polypropylene	Interrupted in galea; single tight layer in scalp; horizontal mattress if bleeding not well controlled by simple sutures	7–10 days
Pinna (ear)	6–0 nylon or 5–0 SA in perichondrium	Close perichondrium with 5–0 SA interrupted; close skin with 6–0 nylon interrupted; stint dressing	4–6 days
Eyebrow	4–0 or 5–0 SA and 6–0 nylon	Layered closure	4–5 days
Eyelid	6–0 nylon or silk	Single layer simple or horizontal mattress	5-6 days
Lip	4–0 silk or SA (mucosa); 5–0 SA (SC, muscle); 6–0 (skin); 4–0 SA	Three layers (mucosa, muscle, skin) if through and through, otherwise two layers	5-6 days
Oral cavity	4–0 SA	Simple interrupted or horizontal mattress: layered closure if the muscularis of the tongue is involved	7–8 days or allow to dissolve
Face	4–0 or 5–0 SA (SC); 6–0 nylon (skin)	If full-thickness laceration, layered closure desirable	5-6 days
Neck	4–0 SA (SC); 5–0 nylon (skin)	Two-layered closure for best cosmetic results	5-6 days
Trunk	4–0 SA (Sq, fat); 4–0 or 5–0 nylon (skin)	Single or layered closure	7–12 days
Extremity	3–0 or 4–0 SA (SC, fat, muscle); 4–0 or 5–0 nylon (skin)	Single or layered closure is adequate, although a layered or running Sq closure may give a better cosmetic result; apply a splint if the wound is over a joint	7–14 days
Hands and feet	4–0 or 5–0 nylon	Single-layer closure only with simple or interrupted horizontal mattress suture, at least 5 mm from cut wound edges; horizontal mattress sutures should be used if there is much tension on wound edges; apply splint if wound is over a joint	7–12 days
Nailbeds	5–0 SA	Gentle, meticulous placement to obtain even edges. Replace nail under cuticle	Allow to dissolve

SA, synthetic absorbable sutures such as Vicryl and Dexon; SC, subcutaneous.
From Markovchick V: Soft tissue injury and wound repair. In Reisdorff EJ, Roberts MR, Wiegenstein JG, editors: *Pediatric emergency medicine*, Philadelphia, 1993, WB Saunders, pp 899–908.

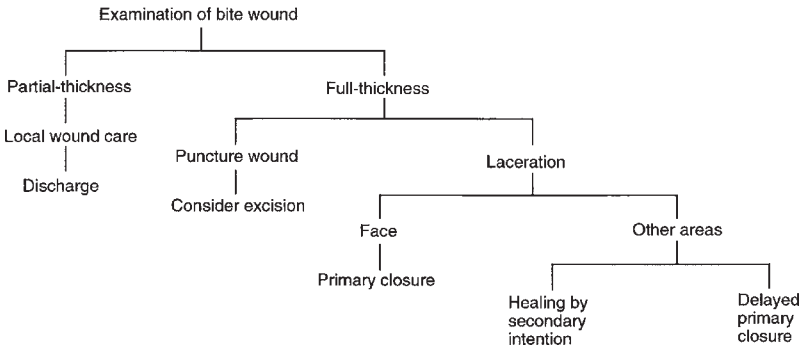

Figure 94-1. Algorithm for treatment of bites.

38. How do I remember the direction of the lines of skin tension?

You don't, unless you have a photographic memory. Refer to Figures 94-2 and 94-3.

39. Are there any controversies in wound care?

The primary controversy relates to the use of prophylactic antibiotics. Their use is widespread and has developed with little scientific support. In general, the use of prophylactic antibiotics is not warranted in the normal host. Antibiotic therapy is indicated in patients with soft tissue wounds who are prone to infective endocarditis. Antibiotics may be indicated when the risk for infection is high, including wounds of the distal foot; contaminated wounds; wounds in which there has been a delay in irrigation and débridement; and wounds that contain fecal material, pus, saliva, or vaginal secretions. Prophylactic antibiotic use should never replace proper wound decontamination. To meet the *standard of care*, as perceived by many, and to decrease cost to the patient, generic antibiotics should be used.

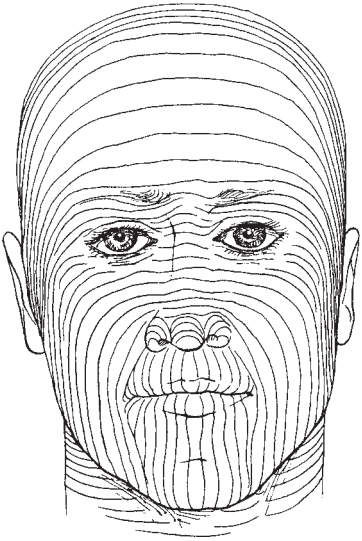

Figure 94-2. Direction of the lines of skin tension for the face. (From Marx J, Hockberger R, Well R, et al, editors: *Rosen's emergency medicine: concepts and clinical practice*, ed 5, Philadelphia, 2002, Mosby, p 738.)

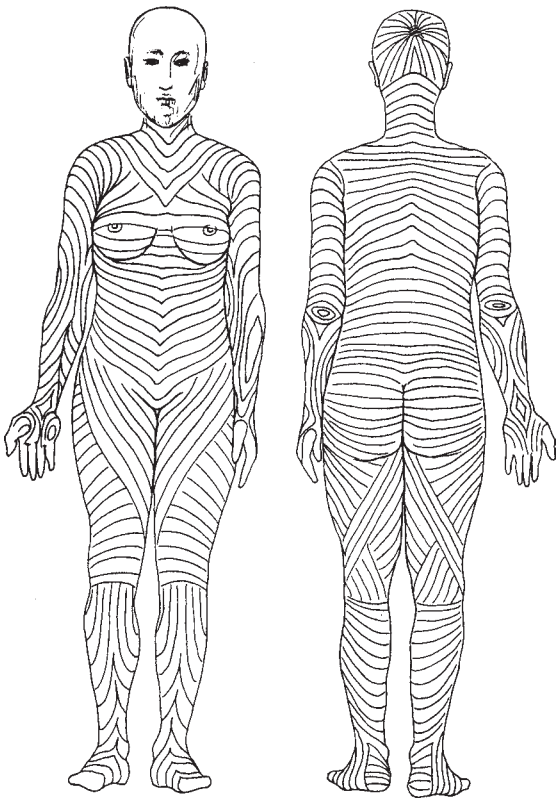

Figure 94-3. Direction of the lines of skin tension for the body. (From Marx J, Hockberger R, Well R, et al, editors: *Rosen's emergency medicine: concepts and clinical practice*, ed 5, Philadelphia, 2002, Mosby p 739.)

KEY POINTS: WOUND MANAGEMENT

1. Use a tourniquet, if necessary, on extremities to adequately examine and repair the wound.
2. Irrigation pressure must be at least 8 psi.
3. Wounds may be irrigated with tap water or sterile saline.
4. It soap is used, irrigation should follow.
5. A stiff brush (toothbrush) will remove ground-in dirt.

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ACUTE PSYCHOSIS

Manish Amin, DO

1. What is psychosis?

According to the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR), the term *psychosis* "has historically received a number of different definitions, none of which has achieved universal acceptance. The narrowest definition of *psychosis* is restricted to delusions or prominent hallucinations, with the hallucinations occurring in the absence of insight into their pathological nature."

2. What are delusions?

"Delusions are erroneous beliefs that usually involve a misinterpretation of perceptions or experiences. Their content may include a variety of themes (e.g., persecutory, referential, somatic, religious, or grandiose). Persecutory delusions are most common; the person believes he or she is being tormented, followed, tricked, spied on, or ridiculed. Referential delusions are also common; the person believes that certain gestures, comments, passages from books, newspapers, song lyrics, or other environmental cues are specifically directed at him or her."

3. What are hallucinations?

"Hallucinations are sensory perceptions without external stimulation. Hallucinations may occur in any sensory modality (e.g., auditory, visual, olfactory, gustatory, and tactile). Auditory hallucinations are by far the most common."

4. How does a patient in a psychotic state typically present to the ED?

Patients who are in a psychotic state may act strangely (i.e., mannerisms, posturing), dress bizarrely, respond to hallucinations, harbor false and delusional beliefs, and consistently confuse the reality of events. They are frequently impulsive and in constant danger of acting on distorted perceptions or delusional ideas resulting in unintentional injury or death. Clarity of oneself and the environment is consistently blurred. The patient is unable to discriminate the stimuli that he or she perceives. Thinking is disorganized and incoherent, as evidenced from the patient's speech. Memory is impaired in registration, retention, and recall. Orientation may be impaired, especially for time. Psychomotor behavior may be hypoactive or hyperactive. Emotions can range from apathy and depression to fear and rage.

5. How should priorities be set when I first encounter a psychotic patient?

- Assess the ABCs (airway, breathing, circulation) if necessary.
- Observe (quickly assess the patient's impulse control and tendency to physically act out).
- Control and manage psychotic behavior, if necessary.
- Obtain a history (gather information from everyone who has been involved with the patient).
- Differentiate between organic and functional causes of psychosis.
- Do a complete physical examination.
- Obtain laboratory tests as deemed appropriate.
- Obtain a psychiatric consultation and disposition.

6. Why is it important to control psychotic behavior immediately?

Patients who present in a psychotic state have no impulse control. They cannot distinguish internal from external stimuli and are unable to prioritize their reactions to them. Because of this dysfunction, they should always be considered a significant danger to themselves or to others. The best way to deal with violent behavior is to prevent it. Emergency physicians should recognize patients who are obviously confused, irrational, paranoid, or excited. Emergency physicians must also develop an intuitive vigilance to detect the possibility of violence in patients who present more rationally and less floridly psychotic. Any history or comment that suggests violence should be taken seriously. The potential for violence in general is particularly high in patients who are psychotic secondary to illicit drug use.

7. Are there behavioral controls that can be used immediately for the psychotic patient?

Yes. Recognizing the potential for violence and physical harm, definitive steps should be taken to avoid confrontation:

- **Environmental:** Keep the environment simple and stimuli-free, and minimize staff changes.
- **Interpersonal:** Assume the role of patient advocate, and engage the patient in a calm and self-assured voice. Recognize the patient's right to privacy and dignity.

8. What options can be exercised if the patient becomes increasingly disorganized, agitated, and violent?

See Chapter 97.

9. How do I obtain a history on a psychotic patient?

Because acutely psychotic patients may not be able to provide an adequate history, all available sources for obtaining information must be explored. This may include speaking to emergency medical services (EMS) personnel, family, friends, neighbors, and law enforcement officers, as well as reviewing old medical records. A telephone conversation with caregivers and significant others can also be helpful.

10. What historical information is important?

- Onset. Did the behavior change suddenly or gradually?
- Longitudinal course. What was the precipitating event? Is this the first such event? What was the behavior like on previous events?
- Psychosocial setting. Obtain some information regarding the patient's support system.
- Previous psychiatric disease, organic brain disease, the use or misuse of medication, history of illicit drug use.
- What are the current medications and have they been taken as prescribed?

11. How should my physical examination be tailored for a psychotic patient?

In retrospective reviews performed by Reeves, Tintinalli, and Riba, a high percentage of missed organic diagnosis in psychotic patients was due to the lack of a complete history and physical examination. Thus a complete and thorough physical examination, including a mental status examination, is imperative. Always note the vital signs and pulse oximetry readings. In most cases, emergency physicians will have built sufficient rapport with patients that they will cooperate with the examination. Tell the patient exactly what you are doing and what you are going to do during the examination. This helps to provide structure for the psychotic patient and avoids confusion or misunderstanding.

12. What is the difference between organic and functional psychosis?

- **Organic psychosis** refers to a reversible or nonreversible dysfunctional mental condition that can be identified as a disturbance in the anatomy, physiology, or biochemistry of the brain (i.e., dementia, withdrawal states and intoxications).

- **Functional psychosis** refers to a dysfunctional mental condition identified as schizophrenia, a major affective disorder, or other mental disorders with psychotic features (i.e., schizophrenia and the affective disorders).

13. Summarize the key points to consider in the differentiation of organic from functional psychosis.

See Table 95-1.

14. List the possible causes of alcohol-related organic psychosis.

- Chronic alcoholism
- Thiamine deficiency (i.e., diet, starvation, and emesis)
- Alcohol-dependent withdrawal states
- Comorbid substance abuse
- Comorbid psychotic and mood disorder
- Alcohol idiosyncratic intoxication (pathologic intoxication)

15. Is there a brief, self-limited, and nonorganic psychosis?

Yes. Some individuals may become acutely and briefly psychotic after exposure to an extremely traumatic experience. If such a psychosis lasts for less than 4 weeks, it is termed a brief psychotic disorder. Precipitants of the psychosis include the death of a loved one, a life-threatening situation, such as combat or a natural disaster, or other life stressors. Patients with hysterical, borderline, and narcissistic personalities are prone to brief psychotic disorder,

TABLE 95–1. MADFOCS MNEMONIC

	Organic	Functional
M = Memory deficit	Recent impaired	Remote impaired
A = Activity	Hyperactivity and hypoactivity Tremor Ataxia	Repetitive activity Posturing Rocking
D = Distortions	Visual hallucinations	Auditory hallucinations
F = Feelings	Emotional lability	Flat affect
O = Orientation	Disoriented	Oriented
C = Cognition	Some lucid thoughts Perceives occasionally Attends occasionally Focuses occasionally	No lucid thoughts Unfiltered perceptions Unable to attend Unable to focus
S = Some other findings	Age > 40 Sudden onset Physical examination often abnormal Vital signs may be abnormal Social immodesty Aphasia Consciousness impaired Confabulation	Age < 40 Gradual onset Physical examination normal Vital signs usually normal Social modesty Intelligible speech Alert, awake Ambivalence

and some studies support a genetic vulnerability. Emotional turmoil, confusion, and extremely bizarre behavior and speech are common symptoms on presentation.

16. Summarize the potentially reversible causes of psychosis.

DEMENTIA mnemonic:

D = **D**rug toxicity
E = **E**motional disorders
M = **M**etabolic disorders
E = **E**ndocrine disorders
N = **N**utritional disorders
T = **T**umors and trauma
I = **I**nfection
A = **A**rteriosclerotic complications

17. Name the life-threatening causes of acute psychosis.

WHHHIMP mnemonic:

W = **W**ernicke's encephalopathy
H = **H**ypoxia or hypoperfusion of the central nervous system
H = **H**ypoglycemia
H = **H**ypertensive encephalopathy
I = **I**ntracerebral hemorrhage
M = **M**eningitis/encephalitis
P = **P**oisonings

18. List pharmacologic agents that can cause acute psychosis.

- Digitalis
- Corticosteroids
- Isoniazid (INH)
- Disulfiram (Antabuse)
- Tricyclics
- Anticonvulsants
- Cimetidine
- Benzodiazepines
- Amphetamines and related drugs
- Antidysrhythmics
- Narcotics
- Barbiturates
- Methyldopa
- Nonsteroidal anti-inflammatory drugs
- Anticancer agents
- Recreational drugs: alcohol, cocaine, amphetamines

19. Is laboratory screening necessary in the work-up of an acute psychotic patient?

Patients, with established psychiatric diagnosis, presenting to the ED with psychiatric chief complaints, benign histories, and normal physical examinations have a low likelihood of clinically significant laboratory findings. Therefore, routine laboratory tests are not recommended. If a patient presents with their first psychotic episode, then laboratory studies are indicated to distinguish functional versus organic psychosis. The following tests are recommended: complete blood count, electrolytes, toxicology screens, pregnancy test, thyroid function tests and computed tomography (CT) scan of the brain.

20. Are there any other clinical *rules of thumb* in the work-up of the acute psychotic patient?

Fever and psychosis = meningitis
Acute psychosis and alcoholism = Wernicke's encephalopathy

Headache and psychosis = tumor or intracranial hemorrhage
Abdominal pain and psychosis = porphyria
Sweating and psychosis = hypoglycemia or delirium tremens
Autonomic signs and psychosis = toxic or metabolic encephalopathy

21. When should hospitalization be recommended?

If this is the patient's first psychotic episode
If the patient is a danger to self or others
If the patient is unable to care for self appropriately
If the patient has no social support system
If the functional psychotic patient is not sufficiently clear after initial ED tranquilization
If an acute organic psychosis does not clear while the patient is in the ED

22. How do I treat the acutely psychotic patient in the ED?

See Chapter 97.

KEY POINTS: ACUTE PSYCHOSIS

1. Definition: delusions or prominent hallucinations
2. Least restrictive restraint: isolation; restraints; psychotropic medication
3. Complete and thorough history and physical examination, including mental status examination, is imperative
4. Organic versus functional disorder

WEBSITE

Consensus guidelines on the medical clearance examination for the evaluation and management of the psychiatric patient in the emergency department: www.macep.org/site/index.php?option=com_content&task=view&id=57&Itemid=74

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DEPRESSION AND SUICIDE

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CHAPTER 96

DEPRESSION

1. What are the symptoms of depression?

The cardinal symptoms of depression are a **dysphoric or sad mood or a loss of interest or enjoyment.** To diagnose depression, one of these must be present nearly every day over a 2-week period. There must also be at least four of the following symptoms during this same period: sleep disturbance, feelings of guilt or worthlessness, lack of energy, decreased concentration or ability to make decisions, appetite disturbance (usually diminished), psychomotor changes (agitated or slowed), and suicidal thinking. The mnemonic **SIG E CAPS** can be remembered by thinking of what you want to do for depressed patients (figuratively): *prescribe energy capsules*.

Five of the following are necessary for the diagnosis of depression, one of which must be the *I*, loss of interests or depressed mood:

S = **S**leep disturbance
I = **I**nterests/mood
G = **G**uilt
E = **E**nergy
C = **C**oncentration
A = **A**ppetite disturbance
P = **P**sychomotor changes
S = **S**uicidal thinking

2. Why is depression considered a mood disorder?

Mood refers to a person's internal state, as subjectively experienced and reported by that person. *Affect* is a person's outward appearance, as objectively experienced by another. The term *mood disorder* has essentially replaced *affective disorder* in much of the psychiatric literature and communications. The main mood disorders are:

- **Major depression** (or unipolar disorder), which is exclusively depression
- **Manic depression** (or bipolar disorder), which is depression with a history of at least one manic episode

3. What is the difference between primary and secondary depression?

Major depression is classified as **primary** if the symptom complex appears before or is causally unrelated to any other significant medical or psychiatric illness. It is considered **secondary** when it follows and is causally related to another medical or psychiatric illness.

4. List medical conditions that might cause secondary depression.

Endocrine disorders
- Hypothyroidism
- Diabetes mellitus
- Cushing's syndrome

Neurologic disorders
- Cerebrovascular accidents
- Subdural hematoma
- Multiple sclerosis
- Brain neoplasm
- Parkinson's disease
- Seizure disorder
- Dementia

Connective tissue diseases
- Systemic lupus erythematosus

Neoplasms
- Pancreatic cancer

5. List medications that might cause secondary depression.

- Antihypertensives (β-blockers)
- Hypnotics and sedatives (benzodiazepines and barbiturates)
- Corticosteroids
- Cimetidine
- Ranitidine

6. Why should the clinician always inquire about alcohol use when evaluating depression?

Alcohol use and abuse is an extremely common comorbid condition with depression and should always be queried for several reasons. First, alcohol use can be disinhibiting with regard to behavior, putting a depressed and suicidal person at increased risk of impulsively acting on suicidal tendencies. Second, depression cannot be treated effectively if there is ongoing alcohol abuse. Third, alcohol is a depressant and is a common cause for depression, a problem known as **alcohol-induced mood disorder.** It may be that the patient's depression is secondary to alcohol use and is treated best by abstaining from alcohol, rather than by administering an antidepressant. This situation is suggested when the onset of the mood disturbance occurs during an extended period of regular (usually daily) alcohol use, rather than before it.

7. When should I suspect depression when a patient presents with what seems to be a medical complaint?

Screen for depression when patients present with nonspecific complaints, such as "sick all over," "weak and dizzy," or "just feeling bad." Using the SIG E CAPS mnemonic (see Question 1) aids in diagnosis. Often depression is expressed in physical rather than emotional terms. Nonspecific physical complaints, such as fatigue, exhaustion, headache, gastrointestinal complaints, muscle aches, and nonspecific pain, are common. Anxiety is seen commonly with depression and can manifest as shortness of breath, nervousness, irritability, and difficulty swallowing, among other symptoms. Panic attacks, a severe form of anxiety that often occurs in the context of depression, are a common cause of ED presentations of atypical chest pain.

8. Are psychotic features ever a manifestation of depression?

Sometimes. If psychotic symptoms accompany depression, it signifies a more severe and dangerous form of depression. When this is the case, psychiatric consultation and often psychiatric hospitalization are indicated. Common psychotic symptoms are hearing guilt-provoking or self-critical voices, called *auditory hallucinations,* and fixed, false beliefs that can be persecutory or paranoid in nature, referred to as *delusions.* Patients with **psychotic depression** are at higher risk for suicide, especially when they have auditory hallucinations commanding them to harm themselves.

9. Name therapies available for treatment of depression.

Antidepressant medications, psychotherapy, and electroconvulsive therapy.

10. What antidepressant medications are used to treat depression?

Tricyclic antidepressants (TCAs) and monoamine oxidase inhibitors (MAOIs) are the two oldest classes of antidepressants, both of which have fallen into relative disuse because of their side effects and dietary restrictions (in the case of MAOIs). Serotonin reuptake inhibitors are still the most commonly prescribed class of antidepressants, mainly because of comparable efficacy, greater ease of use, and fewer adverse effects. These are fluoxetine (Prozac), paroxetine (Paxil), sertraline (Zoloft), citalopram (Celexa), fluvoxamine (Luvox), and Escitalopram (Lexapro).

Newer and perhaps better medications that act on multiple neurotransmitter systems are now available, and also have better side-effect profiles than the older TCAs or MAOIs. These include:

- Venlafaxine (Effexor)
- Bupropion (Wellbutrin)
- Mirtazapine (Remeron)
- Duloxetine (Cymbalta)

Lithium, psychostimulants, and thyroid hormone are common adjunctive treatments.

11. What are some psychotropic-related emergencies or precautions?

- MAOIs in combination with sympathomimetic agents can cause hyperadrenergic crisis, and their combination with meperidine (Demerol) or dextromethorphan can cause cardiovascular instability and central nervous system excitability.
- Neuroleptics can cause dystonias, including laryngeal spasm and neuroleptic malignant syndrome (delirium, rigidity, fever, and autonomic abnormalities), both medical emergencies.
- Anticholinergic toxicity may occur because many psychotropics have anticholinergic properties and often are used in combination; these include benztropine mesylate (Cogentin); trihexyphenidyl (Artane); diphenhydramine (Benadryl); TCAs; and low-potency and mid-potency neuroleptics.
- Many other commonly used agents have dose-related toxic side effects, particularly the *mood stabilizers*, which include lithium and the anticonvulsants valproic acid and carbamazepine.

12. When should the emergency physician prescribe antidepressant therapy?

Because antidepressants generally take weeks to begin working and often require monitoring of side effects and dose titration, prescribing them in the ED should be avoided whenever possible. Exceptions include a patient who is already on treatment and needs a refill or a patient who is initiating new treatment after an emergent consultative evaluation by a psychiatrist. Ideally, in both of these cases, a 1- to 2-week supply of medication can be prescribed, and the patient should follow up with outpatient psychiatric care.

13. What is the most serious complication of depression?

Suicide. Major depression accounts for an estimated 50% of all suicides.

14. Which patients should be hospitalized for depression?

Depressed patients who express suicidal intent or have a plan for suicide should be hospitalized. Psychotically depressed patients should usually be admitted. Also, patients who have just made a violent suicide attempt, have tried to avoid rescue, or are refusing help should be admitted for further observation. Do not forget to institute suicidal precautions while these patients are in the ED.

SUICIDE

15. What is the proper approach to a patient who has attempted suicide?

Medical management of any life-threatening condition precedes psychiatric evaluation. It is important, however, that as the treatment proceeds, the ED team maintain a nonjudgmental approach. Punishment or ridicule is neither therapeutic nor proper conduct for medical professionals. Nearly all patients who attempt suicide are at least ambivalent about the wish to live or die. Demeaning or harsh treatment of such patients, especially by health professionals who are symbols of medical authority, worsens the already low self-esteem and may make subsequent psychiatric care more difficult.

16. Describe suicide precautions.

Because some patients have been known to repeat a suicide attempt while in the ED, suicide precautions are necessary. Such precautions include searching the patient and recovering weapons, pills, or other potential means of self-injury; keeping the patient under close observation; recovering any potential dangerous items from the immediate care area (e.g., needles, scalpels, glass, razors); and not allowing the patient to go anywhere (e.g., bathroom) unaccompanied. When constant staff observation is not possible, physical restraints may be necessary to protect the severely suicidal patient from further self-harm.

17. Are *accidents* ever suicide attempts?

It is important to remember that victims of trauma may have actually attempted suicide. Single-victim accidents, such as a car driven at high speed into a concrete structure, a pedestrian hit by a high-speed vehicle, or a fall from a height, are classic examples of suicide

attempts presenting as trauma. Medical management should be followed by an assessment of suicide intent, including a discussion with family members and perhaps psychiatric consultation.

18. What psychiatric disorders are associated with attempted suicide?
Major depression, alcohol and drug dependence, schizophrenia and other thought disorders, personality disorders, panic disorder, adjustment disorders, and organic brain syndromes.

19. How do I evaluate the risk of a subsequent suicide in someone who attempted suicide?
The following elements are part of an emergency assessment of suicide risks: age, gender, marital status, social supports, physical illness, previous attempts, family history of suicide, risk of the attempt versus likelihood of rescue, secondary gain, nature of any psychiatric illness, alcohol or drug abuse, attitude (hopelessness, impulsivity) affect, and future plans of the suicide attempter. If, after reviewing these factors, the emergency physician is still unsure of the patient's risk, psychiatric consultation is often helpful.

20. How does age relate to suicide risk?
Older patients (especially $>$ 65 years) are statistically more likely to complete suicide than younger patients. Such patients may experience loss of spouse, loneliness, physical illness, or economic hardship in addition to depression. A worrisome increase in suicide among younger persons has emerged, however. Suicide is now the third leading cause of death in youth and young adults (19–24 years of age).

21. What role does gender play?
The rates of completed suicide in men are higher than those for women, whereas the rates of attempted suicide are higher for women than for men. This difference has to do with the lethality of the means. Men attempt suicide more often by violent means, such as shooting, stabbing, hanging, or jumping from a height, whereas women typically use less violent and less lethal methods, such as drug overdose.

22. What is the relationship of marital status to risk of successful suicide?
Never having been married carries the highest risk, followed in decreasing magnitude of risk by being widowed, separated, divorced, and married.

23. What about other social support?
Unemployment, loneliness, loss of home, and relative isolation increase the risks of suicide. Church, family, or community support helps to mitigate suicide risk.

24. Is there a relationship between physical illness and suicide risk?
Yes. Patients with a medical illness, especially a painful, incurable one, may seek a "way out" through suicide. The most common nonpsychiatric diagnoses associated with suicide are chronic medical conditions, such as cancer, chronic obstructive pulmonary disease, and chronic pain. Renal dialysis patients have a suicide rate 400 times higher than the general population, and HIV patients also have a higher than average rate.

25. Does a history of prior suicide attempts signify increased risk?
Yes, especially if each subsequent attempt escalates in severity. The risk of completed suicide is more than 100 times the average in the first year after an attempt—200 times greater for people older than 45 (National Mental Health Association: www.nmha.org). An exception may exist if the previous attempts all have been minor and considered to be manipulative acts.

26. What is the relationship of family history to suicide risk?
Patients with a family history of suicide, alcoholism, or depression have a higher suicide risk than patients without such a family history. A family history of suicide in first-order relatives (e.g., parent or sibling) should cause particular concern.

27. How does the risk of the suicide attempt and the likelihood of rescue affect a suicide evaluation?

In general, a more serious or risky attempt is considered a more likely predictor of subsequent attempts than a minor attempt. An attempt carried out in such a way that rescue is probable is associated with a lower risk of subsequent successful suicide. The patient's *belief* about the lethality of the attempt is at least as important as the physician's assessment of the seriousness of it.

KEY POINTS: SERIOUS SUICIDE ATTEMPTS

1. Patients thought what they did in their attempt to commit suicide was likely to kill them.
2. They did it in such a way as to have a low chance of being rescued.
3. They are not talking much about how they are feeling now.
4. They have little social support and are unwilling to reach out to others or accept help from available resources.
5. They still want to die.

28. What is secondary gain as it applies to suicide attempt?

Sometimes a suicide attempt seems to have a goal other than death. This goal, which is termed *secondary gain*, may be increased attention from parents, friends, or lovers. In attempts with no expected gain other than death, the potential for subsequent successful suicide is great. With the increase in successful suicides among the young, the physician must be careful in ascribing suicide attempts to the desire for attention or secondary gain until a reasonably thorough evaluation can be completed.

29. What is the value of assessing the suicidal patient's attitude and affect?

The patient who appears exhausted, helpless, hopeless, or lonely represents high risk. The patient who attempts suicide because of anger or in an effort to gain revenge has a much better prognosis than one who appears quiet, sad, fatigued, or apathetic.

30. Why is it important to inquire about a specific plan?

Never hesitate to ask the patient about any plans regarding suicide. The patient who continues to express suicidal ideation after one attempt is at risk for a subsequent attempt. The risk is highest if the plan is detailed, violent, or feasible.

31. What is the SAD PERSONS Scale?

In 1983, Patterson et al. used known high-risk characteristics to develop the mnemonic SAD PERSONS Scale. The scale was designed to be used by nonpsychiatrists to assess the need for hospitalization in suicidal patients. Hockberger and Rothstein modified the scale to facilitate use in the ED (see Table 96-1). A score of 5 or less indicates that a patient probably can be discharged safely. Scores of 6 or more require psychiatric consultation, and a score of 9 or more indicates the probable need for psychiatric hospitalization.

32. In general, which suicidal patients should be hospitalized?

- **Absolute indications** for hospitalization after suicide attempts (involuntarily, if necessary) usually include the following: presence of psychosis; a violent, nearly lethal preplanned attempt; and continued suicidal ideation with definite plans for a repeated attempt.
- **Relative indications** include age older than 45; high risk-to-rescue ratio; serious mental illness; alcoholism; drug addiction; living alone with poor social support; and hopelessness, helplessness, or exhaustion.

TABLE 96–1. MODIFIED SAD PERSONS SCALE

	Mnemonic	Characteristic	Score
S	Sex	Male	1
A	Age	<19 or >45 years	1
D	Depression or hopelessness	Admits to depression or decreased concentration, appetite, sleep, libido	2
P	Previous attempts or psychiatric care	Previous inpatient or outpatient psychiatric care	1
E	Excessive alcohol or drug use	Stigmata of chronic addiction or recent frequent use	1
R	Rational thinking loss	Organic brain syndrome or psychosis	2
S	Separated, widowed, or divorced		1
O	Organized or serious attempt	Well-thought-out plan or life-threatening presentation	2
N	No social supports	No close family, friends, job, or active religious affiliation	1
S	Stated future intent	Determined to repeat attempt or ambivalent	2

Scoring: A positive answer to the presence of depression or hopelessness, lack of rational thought processes, an organized plan or serious suicide attempt, and affirmative or ambivalent statement regarding future intent to commit suicide are each scored 2 points. Each other positive answer is scored 1 point.

Score	Risk
<6	Low
6–8	Intermediate
>8	High

Adapted from Hockberger RS, Rothstein RJ: Assessment of suicide potential by non-psychiatrists using the SAD PERSONS score. *J Emerg Med* 99:6, 1988. In Hockberger RS, Smith M: Depression and suicide ideation In Wolfson A.B (ed): *Clinical practice of emergency medicine,* ed 4, Philadelphia, 2005, Lippincott Williams & Wilkins, pp 637–639.

KEY POINTS: POINTS: INDICATIONS FOR SUICIDE PRECAUTIONS AND PSYCHIATRIC CONSULTATION

1. Violent, near-lethal, preplanned attempt
2. Psychotic patient
3. Elderly patient
4. Expression of continued wish to die by suicide

WEBSITE

National Mental Health Association: www.nmha.org

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CHAPTER 97

MANAGEMENT OF THE VIOLENT PATIENT

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1. Is violence a problem in the emergency department (ED)?

Yes. Only a small percentage of ED patients are violent, disruptive, or abusive to staff yet they require disproportionate staff attention and resources. About 4% of patients in the ED need restraints during their stay. According to a prospective observational study from an urban ED, 29.2% of restraint episodes were for violent and disruptive behavior, 25.2% for agitation, and 7.4% for alcohol or drug intoxication. Restraints were less frequently used for confusion (6.4%) or dementia (1.7%), but note that 40% of the patients who were restrained had multiple reasons for restraints. Approximately 30% of agitated patients require a combination of physical and chemical restraint.

2. Why did the patient become violent in the first place?

Common causes of violence and agitation include acute intoxication, acute withdrawal and associated delirium, metabolic disorders, trauma, infectious disease, sepsis, cardiovascular disorders, psychiatric disorders, hypoxia, or intracranial processes such as stroke or hemorrhage. Note that the rate of violence is the same between patients with mental illness and those without mental illness. Better predictors of violence include recent history of violence and the presence of personality disorder or substance abuse.

KEY POINTS: COMMON MEDICAL CONDITIONS THAT MANIFEST AS VIOLENT BEHAVIOR

1. Hypoxia
2. Hypoglycemia
3. Acute intoxication or withdrawal syndromes
4. Meningoencephalitis
5. Intracranial injury or bleed
6. Hyponatremia or hypernatremia
7. Drug side effects

3. What can hospitals do to decrease the risk of violence?

- Limit hospital access to only a few entrances staffed and monitored by trained security personnel.
- Metal detectors should be used to screen patients and visitors for weapons.
- Continuous surveillance or closed-circuit television monitors should be used to help ensure safety in the parking areas and the immediate grounds of the hospital.
- Multiple methods of summoning police or security must be available to the ED without having to go through the hospital operator.

4. What can be done to preempt a violent episode?

- Be aware of early signs of impending violent behavior, such as agitation, intoxication, delirium, abusive language, and challenges to authority.
- Completely undress all patients and place in a gown; remove from reach any items that may be used as a weapon.

5. What is the initial approach a physician can take to control an agitated or violent patient?

The first approach should be verbal redirection. The physician should appear calm and in control but empathetic while explaining to the patient that he or she is in a safe environment and that the ED staff is there to help. Active listening may go a long way toward calming the patient. Keep hands in a neutral position, not crossed across your chest or behind your back. Make an attempt to make the patient more comfortable by offering a drink or a warm blanket. Be clear that violence will not be tolerated and that ED staff must maintain a safe environment for treating patients. Security officers in the patient's presence may dissuade further inappropriate behavior. It may be beneficial to offer nicotine replacement such as gum or a patch or other voluntary meds for agitation including oral haloperidol, lorazepam, olanzapine, or ziprasidone (see section on medications). Care providers must be aware of their own emotions when dealing with agitated patients; yelling or exchanging threats with the patient only further escalates the situation.

KEY POINTS: DE-ESCALATING A VIOLENT PATIENT

1. Use verbal redirection.
2. Use nonconfrontational body language.
3. Reassure the patient's safety.
4. Use empathy.
5. Maintain and emphasize control over the ED.
6. Be honest and direct.
7. Offer nicotine replacement, food, water, or blanket.
8. Offer voluntary meds for agitation.

6. What if that doesn't work?

When aggressive behavior cannot be managed by verbal de-escalation and a patient becomes an increasing risk of violence or elopement, physical or chemical restraint may be indicated. Physical restraint usually involves four-point locking leather cuffs (both wrists and both ankles). A team of five staff members should place the restraints—one care provider for each limb and one at the head of the patient to provide an ongoing explanation to the patient of what is happening and why. The physician should not be involved in this action because it may further jeopardize his or her therapeutic relationship with the patient. Soft restraints or two-point restraints may be best for the disoriented patient at risk of pulling lines and tubes. When the patient becomes calm, he or she is often brought down to two-point restraints or released from restraints completely.

7. What do I need to remember when physically restraining a patient?

Restraints must be snug enough to control the patient but not impede circulation. There should be enough room to allow one finger to move easily between the patient's skin and the cuff. Ensure that side rails are up. Due to risk of suffocation, never restrain a patient in the

prone position and any patient at risk of aspiration should be positioned on his or her side. Patients in restraints must be monitored directly and continuously. As the agitation resolves, restraining measures can be downgraded or discontinued. Documentation is required regarding the patient's behavior and mental status, attempts at less restrictive measures, and monitoring of vital signs. The Joint Commission has specific guidelines for use of physical restraints. There are additional hospital protocols regarding restraints, and the physician should be familiar with all of these guidelines.

8. Am I legally allowed to restrain someone?

Yes. Chemical or physical restraint is indicated when patients become imminently dangerous and less restrictive measures have failed. The courts have held both physicians and hospitals liable for injuries that have occurred when violent or otherwise incapacitated patients escaped hospital grounds or are discharged. The ED staff must therefore prevent certain patients from leaving until they can be examined and thoroughly evaluated. If the patient elopes, avoid personal heroics and instead call the local authorities. Regarding a patient's right to refuse medications, this does not apply to patients who exhibit violent or acutely psychotic behavior in the ED. Courts have routinely held that physicians may involuntarily administer medications to patients who would otherwise present an imminent risk of dangerous behavior.

9. What medications are recommended for chemical restraint?

Three primary classes of drugs are used for chemical restraint: (1) benzodiazepines, such as lorazepam and diazepam; (2) traditional antipsychotics such as haloperidol and chlorpromazine; and (3) atypical antipsychotics such as olanzapine, risperidone, and ziprasidone.

Benzodiazepines: These are useful in agitation, mania, psychosis, alcohol withdrawal, benzodiazepine withdrawal, and sympathomimetic toxidromes like cocaine or amphetamine toxicity. Doses of 1 to 2 mg intramuscularly (IM) or intravenously (IV) of lorazepam may be given every hour as needed. Diazepam may be given as follows: 5 to 10 mg PO, IV, IM, or per rectum (PR) every hour as needed. Another choice is midazolam alone or in combination with haloperidol. Midazolam, 5 mg IM, has been shown to have a more rapid onset than lorazepam or haloperidol and also has the benefit of having a shorter time to arousal.

Traditional Antipsychotics: Although the antipsychotic effects of these medications may take days to achieve, their usefulness in the acute setting with any patient (with or without psychosis) is due to their sedating properties. Haloperidol in doses of 5 to 10 mg per os (by mouth; PO), IM, or IV can be an effective medication for controlling agitation due to psychosis, delirium, or intoxication. These doses may be repeated every hour until the patient is calm; maximum recommended daily dose of haloperidol is 30 mg. Chlorpromazine at a dose of 100 mg PO or 50 mg IM may be given hourly as needed. In 2001, the Food and Drug Administration (FDA) issued a *black box warning* for droperidol, citing a risk of QTc prolongation and torsades de pointes. However, the evidence for this is in dispute and many practitioners believe that the risks associated with use of droperidol are outweighed by the beneficial effects, particularly when routine electrocardiogram (ECG) screening is employed. Whenever using a traditional antipsychotic such as haloperidol, it is advisable to provide protection from possible extrapyramidal symptoms (EPS) by coadministration of an anticholinergic agent such as diphenhydramine at a dose of 50 mg PO, IM, or IV, or benztropine at a dose of 1 mg PO, IM, or IV.

Atypical Antipsychotics: Although the atypicals can be more expensive, they are effective at controlling agitation without overly sedating the patient, thus offering potential benefits with regard to more expedient disposition of the patient. Risperidone 2 to 4 mg PO or olanzapine 10 to 20 mg PO or 10 mg IM can be effective in controlling agitation. Ziprasidone 80 to 120 mg PO or 10 to 20 mg IM (with or without lorazepam) is also indicated for acute agitation. Be advised that coadminstration of benzodiazepines with IM olanzapine is not recommended due to risk of respiratory depression.

10. What if two doses of haloperidol have not sedated the patient?

Don't give a third dose. Supplement the haloperidol with a benzodiazepine; the two together have a synergistic effect. If you used haloperidol 5 mg IM and repeated this once, try adding lorazepam 2 mg IM or IV or diazepam 5 mg IM or IV. Alternatively, add an atypical antipsychotic such as ziprasidone, risperidone, or olanzapine.

11. How do you use chemical restraint for sedation in a pediatric patient?

Very few of the psychotropic medications used for restraint in adults have been approved for use in children. The most frequently used medication for rapid sedation in children is lorazepam due to its rapid onset, short half-life, and multiple routes of administration (see doses), but be wary of dysinhibition/paradoxical agitation. Haloperidol is a safe and effective alternative and may be given IM or PO (see Table 97-1). Intravenous haloperidol has not been approved by the FDA but may be effective. Haloperidol and lorazepam may be mixed together in one syringe; this combination is safe and effective in the pediatric and adult population but may lead to prolonged sedation.

KEY POINTS: PREFERRED DRUGS FOR CHEMICAL RESTRAINT

1. Haloperidol 5 to 10 mg PO or IM; may repeat every hour as needed; max 30 mg; consider adding diphenhydramine 50 mg PO or IM twice a day to reduce risk of EPS. Consider adding lorazepam 1 to 2 mg PO or IM.
2. Olanzapine 10 to 20 mg PO or 10 mg IM; do not combine intramuscular olanzapine with lorazepam due to risk of respiratory depression.
3. Lorazepam 1 to 2 mg PO, IM, or IV; may repeat hourly as needed.

12. Summarize the main side effects to watch for with these drugs.

Benzodiazepines may rarely cause hypotension and respiratory depression. The traditional antipsychotics may cause hypotension and extrapyramidal or other dystonic symptoms (EPS). Hypotension is rare, but EPS occurs in approximately 1% of patients. The most common dystonic reactions are oculogyric crisis, torticollis, and opisthotonos, which are irregular movements of the eyes, neck, and back, respectively. These symptoms may be prevented or reversed with diphenhydramine or benztropine. Neuroleptic malignant syndrome (NMS) is a medical emergency characterized by rigidity, hypertension, hyperthermia, and altered mental status and can been seen after administration of antipsychotics. Treatment for NMS involves supportive care and dantrolene.

KEY POINTS: MAJOR SIDE EFFECTS OF HALOPERIDOL

1. Akathisias
2. Dystonic reactions
3. Neuroleptic malignant syndrome (rare)
4. Anticholinergic effects
5. Hypotension
6. Lowered seizure threshold

TABLE 97–1. QUICK REFERENCE FOR DOSING AND ADMINISTRATION OF MEDS FOR AGITATION

Medication	Adult Dose	Pediatric Dose	Onset	Duration	Side Effects
Benztropine	1–4 mg PO/IM/IV bid prn EPS	0.02–0.05 mg/kg/dose	■ IV 5–15 mins ■ IM 15–30 mins ■ PO 30–60 mins	6–12 hours	Anticholinergic effects
Chlorpromazine	100 mg PO q 60 min prn or 50 mg IM q 60 min prn		■ PO 30–60 mins ■ IM 20–40 mins	4-6 hours	Akathisia, NMS, EPS
Diazepam	2–10 mg PO/IM/IV q 30–60 min prn		■ IV 1–5 mins ■ IM 20–30 mins ■ PO 30–40 mins	30–60 mins	Respiratory depression, paradoxical agitation, dysinhibition
Diphenhydramine	25–50 mg PO/IM/IV q 6 h prn EPS	1.25 mg/kg/dose	■ IV 5–15 mins ■ IM 15–30 mins ■ PO 30–60 mins	4–6 hours	Anticholinergic effects
Droperidol*	2.5–10 mg IM/IV q 60 min prn	0.03–0.07 mg/kg/dose (max 2.5 mg)	■ IV 2–10 mins ■ IM 5–10 mins	2–3 hours	Dystonias, akathisia, NMS hypotension, QTc elevation, lower seizure threshold, anticholinergic effects, hypotension

Haloperidol	2–10 mg PO/IM/IV q 30–60 min prn (max 30 mg)	0.025–0.075 mg/kg/dose	■ IV 2–5 mins ■ IM 30–40 mins ■ PO 30–60 mins	2–4 hours	Dystonias, akathisia, NMS, hypotension, QTc elevation, lower seizure threshold, anticholinergic effects
Lorazepam	0.5–4 mg PO/IM/IV q 30–60 min prn	0.05–0.1 mg/kg/dose PO/IV	■ IM/IV 5–10 mins ■ PO 30–40 mins	4–6 hours	Respiratory depression, paradoxical agitation, disinhibition
Midazolam	1–5 mg IM/IV q 60 min prn	0.05–0.1 mg/kg/dose IV (max 1.0 mg)	■ IV 1–5 mins ■ IM 15–20 mins	1–2 hours	Respiratory depression, paradoxical agitation, disinhibition
Olanzapine	5–20 mg PO/SL q 60 min prn or 2.5–10 mg IM tid prn (max 30 mg daily)		■ PO/SL 30–60 mins ■ IM 20–40 mins	2–4 hours	NMS, akathisia; *do not mix intramuscular preparation with benzos*
Risperidone	2-4 mg PO q 4 h (max 16 mg daily)		■ PO 30–60 mins	2-4 hours	Akathisia, NMS
Ziprasidone	80–120 mg PO bid or 10–20 mg IM q 4 h (max IM dose = 40 mg)		■ PO 30–60 mins ■ IM 20–40 mins	2–4 hours	Akathisia, QTc elevation

*FDA Black Box Warning
EPS, extrapyramidal symptoms; IM, intramuscularly; IV, intravenously; NMS; neuroleptic malignant syndrome; PO, orally; PRN, as needed; q, every.

13. Give a quick reference on dosing and administration.
See Table 97-1.

14. How should restrained patients be monitored?
Continuous pulse oximetry is indicated due to possible respiratory compromise, particularly when ethanol-intoxicated patients receive chemical restraint. The Joint Commission requires that physically restrained patients be directly and continuously monitored by trained staff.

15. Does the ED staff need any treatment?
The effect of violence on ED employees can be devastating; physical and psychological trauma may have a long-lasting impact and such episodes may affect future job performance. A comprehensive program patterned after the critical incident stress debriefing model should be established to provide immediate and long-term psychological support, and staff should be encouraged to avail themselves of this support when the need arises.

WEBSITE

Restraints: www.emedicine.com

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INTIMATE PARTNER VIOLENCE

Debra Houry, MD, MPH

CHAPTER 98

1. Isn't intimate partner violence more of a law enforcement issue than it is a health issue?

No. Research shows that up to one fourth of all women presenting to EDs for care have experienced partner violence within the past year. Injuries and illnesses caused by abuse affect their lives more frequently than diseases such as hypertension, cancer, or diabetes. Survivors of intimate partner violence have higher rates of physical and mental health problems than their peers.

2. Define *domestic violence.*

Domestic violence, in a broad sense, refers to all violence occurring within a family unit. By this definition, partner abuse, child abuse, and elder abuse are subsets of domestic violence. *Intimate partner violence* (IPV) is a more specific term, and it is used in this chapter. IPV includes physical acts, such as battering and sexual assault, and nonphysical acts, such as emotional abuse, economic abuse, threats to harm children and property, and prevention of access to health care or prenatal care. Most battered women state that the nonphysical abuse is more humiliating and distressing to them than physical beatings.

3. What are the risk factors for IPV?

IPV occurs in all socioeconomic classes and in all races. Women at greatest risk include those with male partners who abuse alcohol or use drugs; are unemployed; have mental health issues; have a history of pet abuse; have less than a high school education; or are the former husband, estranged husband, or former boyfriend of the woman. Women who are younger than 30 years; who are single, divorced, or separated; or who abuse drugs or alcohol classically have been viewed as being at increased risk for IPV. It is unclear, however, if some of these risk factors lead to the partner abuse or are a result of living in an abusive situation.

4. Are men *ever* victims of partner abuse?

Yes, men do experience partner violence, but this is less often *battering*. Men may be embarrassed to disclose IPV or worried that they may be the ones arrested if they go for help. However, male IPV is not as lethal: a woman is 13 times more likely to be injured and 30% more likely to be killed.

5. If IPV is so common, why have none of my patients experienced it?

Many of your patients may be experiencing partner abuse. Often, physicians do not know because they do not ask about it.

6. What is the result of a missed diagnosis of IPV?

Failure to diagnose IPV may return the woman to a dangerous situation and increase her risk of future injury. It also furthers the victim's sense of entrapment and helplessness. Inappropriate medications may be prescribed (tranquilizers and antidepressants) without a search for the underlying causes of these symptoms. Patients may be labeled as being hysterical, paranoid, and irrational.

7. State some of the reasons why physicians choose not to inquire about IPV.
The most commonly cited reason is lack of time. Health care providers believe that this issue is too time consuming to deal with, especially in a busy ED. Other reasons include the beliefs that it is none of the physician's business, that women would "tell" if they wanted to, that there is nothing that can be done, that the woman deserved the abuse, and that a woman could just leave the situation if she wanted to.

8. Why are victims of partner abuse reluctant to disclose the abuse to health care providers, even if asked?
Men and women may be embarrassed and humiliated that it is happening to them. There may be cultural or religious beliefs that lead a woman to believe that this is normal or to be expected. A woman may have been told that she deserved the abuse. An abuser might have threatened to harm a woman, her children, or other loved ones if she discloses to others; or she may believe that no one can help her.

9. What are some of the structural and system barriers that might prevent a victim from disclosing abuse?
Lack of privacy is a real concern in the ED. Victims should be interviewed alone, without children or partners present. If necessary, hospital security may be recruited to ensure their safety. Also, family members or children should not be used as translators when inquiring about abuse. The use of computer kiosks can help to identify patients that health providers do not personally screen for IPV as it allows patients to disclose IPV anonymously and obtain information on community resources.

10. What clues to IPV might be evident in a patient's history?
Most importantly, a history that is inconsistent with the physical examination findings should raise physician suspicion for IPV. Partner abuse should also be considered in patients with suicidal intentions or attempts, patients who are depressed, patients who have evidence of drug and alcohol abuse, and patients with frequent visits for chronic pain or other somatic complaints.

11. What clues may be present on physical examination in a victim of IPV?
Common injury patterns include injuries to the face, neck, and throat (especially signs or symptoms of strangulation), chest, breasts, abdomen, and genitals. Any injury that does not "fit" with the history obtained should create suspicion of abuse. Other physical examination findings of concern include evidence of sexual assault or frequent, recurrent sexually transmitted diseases.

KEY POINTS: PHYSICAL EXAMINATION FINDINGS IN IPV

1. Injuries to face, neck, throat, chest, abdomen ("central" pattern)
2. Injuries to chest and abdomen
3. *Any* injury that does not "fit" with the history
4. Evidence of sexual assault or frequent, recurrent sexually transmitted diseases
5. Injuries in multiple stages of healing

12. How can I increase my recognition of partner abuse?
First, ask about IPV. Any woman who presents with an injury should be specifically asked who injured her. Second, raise your level of suspicion in women without injuries. Remember the clues that might be present in the history or physical examination. If you are considering partner abuse, ask about it.

13. What questions about partner violence can I ask a woman without injuries?

a. Have you ever been hurt or injured by a partner or ex-partner?
b. Are there situations in your relationship where you have felt afraid?
c. Has your partner ever abused you or your children?
d. Do you feel safe in your current relationship?
e. Is there a partner from a past relationship who is making you feel unsafe now?

14. What about screening all women for IPV?

Good idea! Because of the prevalence of this problem, many organizations have advocated screening all women for the presence of IPV. One screening tool that has been tested clinically is the Partner Violence Screen. This consists of these three questions:

- Have you been hurt or injured in the past year by anyone? If so, by whom?
- Do you feel safe in your current relationship?
- Is there a partner from a previous relationship who is making you feel unsafe now?

This tool is 71% sensitive for detecting IPV. Women who screen positive for IPV are 11 times more likely to experience physical violence in the next 4 months than women who screen negative for IPV.

15. What comments or questions would be inappropriate when discussing IPV with women?

- "What did you do to him?"
- "What did you do that made him so mad?"
- "This has happened before, and you are still married to him?"
- "Why didn't you tell anyone?"
- "You let him do that to you?"
- "I wouldn't let anyone do that to me."
- "Why don't you just leave?"

16. What do I do if my patient has an injury caused by her partner?

- Treat her injuries.
- Document her history and her injuries carefully in the medical records.
- Provide support and empathy; women should be informed that IPV is a common problem, that no one deserves this abuse, and that help is available. Helping victims access community resources should be a primary goal of ED treatment.
- Inquire about the woman's safety and that of her children. Not all women want or require shelter placement. Women who are experiencing increasingly severe physical injuries or whose batterers have access to firearms are at risk for severe or lethal injuries. Some of these interventions may be by a social worker or by a domestic violence advocate, depending on the clinical setting.

17. Summarize some important points to remember when documenting IPV.

Document what happened in the patient's own words, and document the relationship to the batterer. Record all areas of bruising or tenderness; a body map may be helpful. Photographs may be used, but care should be taken to follow local legal guidelines for photographing injuries. Be *sure* to obtain the patient's permission. Any treatment and intervention should be documented. In cases in which abuse is highly suspected and the patient is denying abuse, document the reason that you suspect abuse (e.g., the history does not match the physical examination findings). A well-documented medical record can mean the difference between convicting an abuser and allowing him to go free.

18. Don't I have any legal responsibilities?

You might. As of March 2001, 45 states had a law that mandates reporting intentionally inflicted injuries; however, these laws vary greatly as to what injuries must be reported (Table 98-1). Each emergency physician must be familiar with the current reporting requirement in his or her state.

TABLE 98–1. LEGAL REQUIREMENT FOR REPORTING INJURIES BY STATE

State (Statute)	Injuries from Weapons	Injuries from Crimes	Injuries from Domestic Violence
Alabama	No	No	No
Alaska (Statute 08.64.369)	Yes	Yes	No
Arizona (Rev Stat 13-3806)	Yes	Yes	No
Arkansas (Code Ann 12-12-602)	Yes	No	No
California (Pen Code 11172 AB74 × 19)	Yes	Yes	Yes
Colorado (Rev Stat 12-36-135)	Yes	Yes	Yes
Connecticut (Acts 269)	Yes	No	No
Delaware (Code Ann 24-17-1762)	Yes	No	No
District of Columbia (Ann 2-1361)	Yes	No	No
Florida (Stat Ann 790.24)	Yes	Yes	No
Georgia (Code Ann 31-7-9)	No	Yes	No
Hawaii (Rev Stat 453-14)	Yes	Yes	No
Idaho (Code 39-1390)	Yes	Yes	No
Illinois (Code Ann 20-2630-3)	Yes	Yes	No
Indiana (Code Ann 35-47-1)	Yes	No	No
Iowa (Code Ann 147.111)	Yes	Yes	No
Kansas (Stat 21-4213)	Yes	No	No
Kentucky (Stat Ann 209.020)	No	Yes	Yes
Louisiana (Rev Stat 403.5)	Yes	No	No
Maine (Rev Stat 17A Ch 21.512)	Yes	No	No
Maryland (Ann Code 336, art 27)	Yes	No	No
Massachusetts (Gen Laws 112-12)	Yes	No	No
Michigan (Comp Laws 750.411)	Yes	Yes	No
Minnesota (Stat Ann 626.52)	Yes	No	No
Mississippi (Code Ann 45-9-31; 93-21-1)	Yes	No	Yes
Missouri (Rev Stat 578-350.1)	Yes	Yes	No
Montana (Code Ann 37-2-302)	Yes	No	No
Nebraska (Rev Stat 28-902)	No	Yes	No
Nevada (Rev Stat Ann 629.041)	Yes	No	No
New Hampshire (Rev Stat Ann 631.6)	Yes	Yes	No
New Jersey (Stat Ann 2C: 58-8)	Yes	No	No
New Mexico	No	No	No

TABLE 98–1. LEGAL REQUIREMENT FOR REPORTING INJURIES BY STATE—cont'd

State (Statute)	Injuries from Weapons	Injuries from Crimes	Injuries from Domestic Violence
New York (Penal Code 265.25)	Yes	No	No
North Carolina (Gen Stat 90-21.20)	Yes	Yes	No
North Dakota (Cent Code 43-17-41)	Yes	Yes	No
Ohio (ORC 2921; 2151)	Yes	Yes	Yes
Oklahoma (Stat 2105-846.1)	No	Yes	No
Oregon (Rev Stat 146.750)	Yes	No	No
Pennsylvania (Cons Stat Anns 18-5106)	Yes	Yes	No
Rhode Island (Gen Laws 12-29-9; 11-47-48)	Yes	No	Yes
South Carolina	No	No	No
South Dakota (Codified Laws 23-13-10)	Yes	No	No
Tennessee (Stat 36-3-621; 38-1-101)	Yes	Yes	Voluntary
Texas (Fam Code 91.003,161.041)	Yes	No	Yes
Utah (Code Ann 26-23a)	Yes	Yes	No
Vermont (Stat Ann 13-4012)	Yes	No	No
Virginia (Code Ann 54.1-2967)	Yes	No	No
Washington	No	No	No
West Virginia (Code Ann 61-2-27)	Yes	No	No
Wisconsin (Stat Ann 146.995)	Yes	Yes	No
Wyoming	No	No	No
Total (including DC) "yes"	42	23	7

*Verified as of March 2001.
From Houry D, Sachs CJ, Feldhaus KM, et al: Violence-inflicted injuries: reporting laws in the fifty states. *Ann Emerg Med* 39:56–60, 2002.

KEY POINTS: WHAT TO DO WITH AN IPV VICTIM

1. Treat the injuries.
2. Document the history and injuries carefully. (Consider drawing a picture or taking a photo.)
3. Provide support and empathy.
4. Inquire about the woman's safety and that of her children.
5. Refer to community resources or social worker.
6. Notify law enforcement if required by your state.

19. Why is she going home to her batterer? Why doesn't she just leave him?

Why a woman does not leave her batterer is the wrong question to ask. It implies that the woman is to blame and that if she would just leave everything would be okay. Battered women are most likely to be killed during the act of leaving or after they have left their abuser. There are many other valid reasons why women stay in an abusive situation. She may:

- Have no money or job skills
- Have nowhere else to go
- Feel she must stay to protect her children

20. What can we do about IPV?

A more appropriate response to IPV is to ask ourselves why society tolerates this behavior and how we, as health care providers, might change those attitudes.

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COST CONTAINMENT AND RISK MANAGEMENT IN EMERGENCY MEDICINE

Stephen V. Cantrill, MD

COST CONTAINMENT

1. What is cost containment in emergency medicine?
An approach to limit medical care expenses without compromising quality of care.

2. Why is cost containment so important?
Medical care currently consumes more than 16% of the U.S. gross domestic product (GDP), and health care costs traditionally have increased at a rate far above inflation. Because the federal government directly or indirectly pays for at least 42% of health care, they are quite concerned about this ongoing increase. This has led to active involvement by the National Quality Forum and the Center for Medicare and Medicaid Services in dealing with what is felt to be excessive patient diagnostic testing. One study concluded that one third of medical care may be unnecessary. The proliferation of health-maintenance organizations (HMOs) and capitated-care contracts has also placed additional pressure on physicians to curtail unnecessary health care costs, often with the health care provider sharing in the financial risk of providing patient care. In addition, many practice environments are developing physician practice profiling systems to identify practitioners who order excessive numbers of tests and procedures.

3. In what area do emergency physicians have the most control in terms of containing costs?
Ancillary tests (clinical laboratory and radiology) constitute 44% of patient ED charges—the largest component. These tests are done at the request of the emergency physician and represent an area directly under our control.

4. List some reasons for excessive test ordering in emergency medicine.
- Peer pressure (e.g., wanting to please a consultant)
- Out-of-date hospital policies
- Intellectual curiosity
- Ignorance of the costs of tests
- Patient expectations (and demands)
- Defensive medicine
- Reflex ordering or old habits

None of these reasons is adequate justification for ordering tests that are not medically indicated based on the patient's presentation.

5. Can just knowing the cost of the tests have an effect?
Yes. This has been demonstrated by several studies, including one in which just having the patient charges for tests on the order slip reduced overall patient test charges by 27%.

6. What is the *golden question* to ask before ordering any test?
The question is "How useful will this test be in establishing a diagnosis, assisting in treatment, or making the appropriate disposition?" If the answer is it won't, serious thought should be given regarding the necessity of ordering the study.

7. List some additional strategies to reduce inappropriate test ordering.

- Avoid ordering reflexively. Carefully consider the benefits before ordering a test.
- Do not order a test because *it would be nice to know*, unless you are willing to pay for the test yourself.
- Learn how much routine laboratory tests and radiographs cost. Prepare yourself for a shock.
- Establish guidelines for the use of new technologies. Medicine is notorious for developing and using new tests without discontinuing tests that are old or outdated.
- Avoid ordering studies for medicolegal reasons. Good medicine is good law. Order only studies that are medically indicated.
- Use patient education to reshape patient expectations when possible.
- Cancel studies that were ordered but later found to be unnecessary.

8. Shouldn't we order tests to *cover* ourselves?

No. Again, good medicine is good law. The criteria for ordering studies should be strictly medical, not based on the physician's notion of what would be helpful to have in a court of law. Laboratory or radiographic studies should not be used as a substitute for a proper history, physical examination, and good documentation.

9. Name some commonly over-ordered tests.

- Extremity radiographs
- Urine culture and sensitivity
- Chest radiographs
- Throat culture (excluding streptococcal screen)
- Abdominal radiographs
- Blood type and cross-match
- Rib radiographs
- Blood ethanol level
- Electrolyte panel
- Arterial blood gases
- Complete blood count
- Toxicology screens

10. How much can be saved with no compromise in patient care?

In a multicenter study of 20 hospital EDs, both teaching and nonteaching, a cost-containment educational program was used. Seventeen tests or groups of tests or studies (including those listed previously) were targeted. A 12.5% decrease in targeted test charges was shown. No decrease in the perceived quality of care could be shown. Careful implementation of clinical decision rules, such as the Ottawa ankle rule, can save up to 35% in ordering X-rays, with no decrease in sensitivity. Implementation of specific cost-effective guidelines has been shown to decrease the hospital ED charge by 28%, with the laboratory charge decreased by 46%. This clearly demonstrates that the costs of medical testing in the ED can be contained by careful, thoughtful ordering without compromising patient care.

11. Can good medication-prescribing habits impact the cost of patient care?

Without question! Common, costly, unnecessary practices in prescribing practices include prescribing:

- Antibiotics when no true medical indication exists (e.g., for a viral upper respiratory tract infections [URI]).
- The latest and greatest antibiotic that gives unnecessarily broad coverage.
- By brand name when a generic has demonstrated adequate bioequivalence.

Avoiding these practices will not only help control costs but will also improve the quality of your care.

12. How is the Center for Medicare and Medicaid Services (CMS) concerned about appropriateness of testing?

CMS requires that documentation be supplied to support that the diagnostic testing ordered was reasonable and medically necessary. Often, routine *screening* tests are disallowed, even if the ordering physician thought them to be appropriate. Retrospective audits have been performed, with some institutions having to return tens of millions of dollars to the federal government for not being able to demonstrate the medical necessity of many diagnostic studies.

13. Is the National Quality Forum (NQF) concerned about appropriate resource use in medicine?

Yes. The National Quality Forum is a nonprofit organization that is involved in the development and endorsement of national consensus standards for measuring and reporting medical performance. As such, the NQF is currently involved in medical performance measurements relating to resource use:

- Imaging efficiency standards regarding the appropriate and effective use of imaging in a clinical setting
- Evaluation of episode-based and per capita resource use measures of 18 specific diseases and conditions (many of which involve emergency medicine).

KEY POINTS: COST CONTAINMENT

1. Only diagnostic studies that directly impact the diagnosis or treatment of the patient's presenting problem should be ordered in the ED.
2. CMS will not reimburse a hospital for diagnostic testing if the documentation does not support that the tests were reasonable and necessary.

RISK MANAGEMENT

14. What is risk management?

Efforts to identify (and, when possible, improve or rectify) situations that place a service provider in jeopardy. Good risk management not only deals with situations as they arise (e.g., dealing appropriately with a patient's complaint about care) but also anticipates health-delivery problems before they occur (e.g., establishing in advance the procedures for dealing with a patient who wishes to leave against medical advice).

15. Why are emergency physicians at high risk for malpractice lawsuits?

The primary reason is the lack of an established physician-patient relationship. The patient often feels little rapport with a physician unknown to the patient before the visit to the ED. The visit is usually not at the patient's wish, occurring at an unscheduled time and in a situation in which the patient is under stress and sometimes pain. All of these factors may contribute to feelings of anger and hostility, laying the groundwork for feelings of dissatisfaction about the provided care. A second major reason is that in emergency medicine, the decisions are often irrevocable. If a mistake or misjudgment is made on a patient who is admitted to the hospital, a second chance to correct the error usually exists because the patient is still accessible. In patients wrongly discharged from the ED, sometimes no such second chance exists.

16. What must be proved in a malpractice case?

- Duty to treat. Was there an obligation for the physician in question to treat the patient? In emergency medicine, this answer is almost always yes. By working in an ED, an emergency

physician automatically assumes the duty to treat any patient presenting to the ED and requesting care. The EMTALA statute mandates a medical screening examination on all patients presenting to the ED. (See Chapter 100.)

- Actual negligence. Was the care provided actually negligent? This often involves showing (to the jury's satisfaction) that the care provided fell below what is to be considered the standard of care. This point is the one most often contested by the opposing sides in a malpractice suit. Negligence may result from acts of commission or omission.
- Damages. Did the patient suffer actual damages? This can include the nebulous pain and suffering.
- Proximate cause. Did the negligence cause the damages? It must be shown to the jury's satisfaction that the alleged damages were truly the result of the alleged negligent care.

17. Give some examples of high-risk patients.

- The hostile or belligerent patient. These patients are difficult to deal with and sometimes get less than complete, careful evaluation. Intoxicated patients represent a significant subgroup of this class of patients. Demanding patients also fall into this class. When confronted with patients in this category, remember that *you don't have to love them to give them proper care*.
- The patient with a problem that may be a potential life threat. With these patients, the challenge is to discover and address the life threat (see Chapter 1). Inappropriately discharging these patients often results in a risk-management issue.
- The returning patient. The patient who returns unscheduled to the ED should raise a red flag. What problem is being missed? These patients deserve extra care in reevaluation. The threshold for admitting an unscheduled returning patient should be low.
- The *private* patient. Patients may be sent to the ED by a private physician for diagnostic studies or treatment but not to be seen and evaluated by the emergency physician. In general, any patient in the ED becomes the responsibility of the emergency physician. If something goes wrong with the care of these patients, the emergency physician also may be held liable. It is advisable to have very clear established policies concerning *private* patients in the ED. These patients should be seen by the emergency physician on duty if the patient so requests, if there is a delay in the arrival of the private physician, or if their triage category so warrants.

KEY POINTS: RISK MANAGEMENT

1. Treat every patient as you would want your mother treated.
2. If possible, avoid writing admission orders.
3. Always address the potential life threats, based on the patient's presentation.

18. What clinical problems tend to get emergency physicians into malpractice difficulty?

There is regional variation in clinical problems that tend to cause malpractice problems for emergency physicians, but the following entities are generally major causes:

- Acute coronary syndromes
- Meningitis/sepsis (especially in young children)
- Missed fractures (including spine and pelvis)
- Appendicitis
- Stroke management
- Retained foreign bodies
- Aortic aneurysms
- Tendon/nerve injuries associated with wounds

- Intracranial hemorrhage (subdural, epidural, and subarachnoid hemorrhages)
- Wound infections

19. What is the most common error emergency physicians make with regard to their malpractice insurance policy?

Failure to read carefully and understand the conditions of the policy (i.e., what is covered, what is not covered, what is required for a malpractice occurrence to be covered, what are the settlement options, and what are the "tail" requirements to provide coverage for past patient encounters when the current policy is no longer in force).

20. What common deficiencies in the medical record exacerbate malpractice problems for emergency physicians?

In a malpractice case, your record of a patient's visit can be your greatest friend or your worst foe. The following problems will place the record on the side of the opposing team:

- An illegible record. Think about how the record will look when it is enlarged to 4 feet by 4 feet by the plaintiff's attorney to show to the jury. Electronic, dictated, or typed records avoid this problem.
- Not addressing the chief complaint or nurses' and paramedics' notes. Make sure your evaluation addresses why the patient came to the ED and what others observed and documented about the patient.
- Not addressing abnormal vital signs. As a rule, patients must not be discharged from the ED with abnormal vital signs. Whenever this is done, the record must contain a discussion of why the physician is taking this action.
- An incomplete recorded history. As with all other parts of the medical record, an attempt will be made to convince the jury that *not recorded equals not done*. The history must include information concerning all potential serious problems consistent with the patient's presentation. Significant negatives should be recorded as well.
- Labeling the patient with a diagnosis that cannot be substantiated by the rest of the record. This not only may cause difficulty if the physician's *guess* is wrong but also leads to premature closure on the part of the next physician to treat the patient, removing the slim chance of correcting the diagnostic error if the patient returns to the ED because of no improvement.
- Inadequate documentation of the patient's course in the ED with inadequate attention to the patient's condition at discharge. Often the patient's condition may improve dramatically while in the ED, justifying discharge, but this fact is not reflected in the record. If this case becomes a malpractice problem, it appears that the patient was discharged in the original (unimproved) condition.
- Inadequate discharge (follow-up, aftercare) instructions. The greatest risk in dealing with patients is being wrong in our judgment. The best insurance is careful and complete patient discharge instructions that include when and where to follow up and under what conditions to return to the ED. It is striking how little effort is put into this component of the record. After completing your evaluation and treatment of a patient, ask yourself, "What if I am wrong, and what is the worst possible complication that can occur?" Address these possibilities completely in your discharge instructions, and document them carefully in the record.

21. What *systems problems* often lead to lawsuits?

- Systems problems are not under the emergency physician's control, but can still cause difficulty. Such problems include:
- Inadequate follow-up on radiology rereads of radiographs
- Inadequate follow-up of cardiology rereads of electrocardiograms (ECGs)
- Inadequate follow-up of delayed clinical laboratory results (e.g., cultures)
- Poor availability of previous medical records
- Inadequate handling of patient complaints (your chance possibly to head off a malpractice suit)
- Inadequate physician and ED staffing patterns (leading to prolonged patient waits and subsequent patient hostility

22. When a patient refuses care, what are the two criteria that must be present?

If a patient desires to leave the ED against medical advice, the patient must:

- Be competent to refuse care
- Understand the possible untoward sequelae that could result from refusal of care

All patients have the right to refuse care if these two criteria are met. Common sense (and most risk managers) would tell you to err on the side of treating the patient if there is any doubt as to competence.

23. What clinical problem-solving approach is most helpful in avoiding lawsuits?

When dealing with any patient, make sure you address the life threats: major problems that could exist, given this presentation for this patient. The safe approach is to assume the presence of these life threats, then set about to disprove them (see Chapter 1).

24. What physician behaviors may help avoid lawsuits?

- Be courteous and kind to the patient and to the patient's family.
- Take time to communicate with the patient. It takes only seconds to tell the patient what is going on, what the results of diagnostic studies are, and what you are thinking concerning his or her case. Make sure all patient questions and concerns are addressed.
- Dress neatly.
- Explain and apologize for inordinate delays in patient care.
- Make sure the medical record accurately reflects the care provided and the thought processes behind the care.

This approach can be summarized in a simple statement: "Treat every patient as you would want your mother treated." This, of course, assumes you love your mother.

25. How can writing admission orders for admitted patients cause problems for the emergency physician?

In many situations, writing admission orders for patients has made the emergency physician liable for untoward events occurring to the patient in the hospital before he or she is seen by the private physician. There is often significant peer pressure for the emergency physician to write such orders. This practice is potentially dangerous and must be discouraged.

26. What are the criteria for reporting a physician to the National Practitioner Data Bank (NPDB)?

The NPDB was established by the federal government in 1989 to track potential problem physicians. The criteria for reporting a physician to the NPDB are any:

- Payment made for a claim or judgment against a physician
- Action taken by a state medical licensing board against a physician
- Disciplinary action lasting more than 30 days taken against a physician by a group or institution.

A hospital must query the NPDB about any physician applying for staff privileges and at the time of reappointment of a physician to the medical staff.

27. How can clinical policies (evidence-based practice guidelines) *decrease* malpractice risk for the emergency physician?

Many groups and organizations are developing evidence-based practice guidelines. If it can be shown that a physician's care was consistent with these guidelines, it may help to show the appropriateness of the care and the lack of negligence.

28. How can clinical policies potentially *increase* malpractice risk for emergency physicians?

Malpractice risk can be increased by applicable evidence-based practice guidelines if the emergency physician is not aware of or if he or she chooses not to follow these guidelines without carefully documenting the reasons for not doing so.

29. Does emergency medicine residency training decrease my malpractice risk?
One study revealed emergency medicine residency-trained physicians had significantly less malpractice indemnity than non-emergency medicine residency-trained physicians. This difference was not due to differences in the average indemnity but was due to significantly fewer closed claims against emergency medicine residency-trained physicians with indemnity paid. This resulted in a cost per physician-year of malpractice coverage for non-emergency medicine residency-trained physicians that was more than twice that of emergency medicine residency-trained physicians ($4,905 versus $2,212).

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National Quality Forum: www.qualityforum.org

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CHAPTER 100

EMTALA, JC, AND HIPAA

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EMERGENCY MEDICAL TREATMENT AND LABOR ACT (EMTALA)

1. What is EMTALA?

In 1986, Congress enacted EMTALA as part of the Consolidated Omnibus Reconciliation Act (COBRA) to ensure public access to emergency services regardless of ability to pay. The intended purpose is to prevent the *dumping* of patients; that is, the inappropriate transfer or discharge of uninsured patients in an unstable condition solely for the economic benefit of the treating hospital. Put simply, EMTALA requires any hospital that participates in Medicare to do a medical screening examination (MSE) on any patient requesting medical evaluation for an emergency medical condition (EMC), without regard for the patient's ability to pay for services rendered. If such a condition is found to exist, the hospital and the treating physician must use all of the resources normally available to them in stabilizing the emergency medical condition before that patient can be discharged or transferred to another facility. Because more than 98% of hospitals in the United States participate in Medicare, the influence of EMTALA on emergency medical care is far reaching. Failure to comply with its provisions can mean criminal sanctions, stiff financial penalties, and exclusion from participating in governmental programs such as Medicare and Medicaid.

2. Define *emergency medical condition*.

Any medical condition (including psychiatric disturbances or symptoms of substance abuse) that without immediate medical attention might result in the patient's loss of life; a serious impairment of bodily function; serious dysfunction of any body organ or part; severe pain; or, in the case of a woman in active labor, the death or disability of the woman or unborn child.

3. Why does such a statute even exist?

Access to medical care in the United States has never been defined as a fundamental right. For much of the 20th century, private hospitals were under no obligation to offer emergency care to the uninsured. Consequently, indigent or *undesirable* patients were often denied such care and forced either to seek care elsewhere or go without any assistance whatsoever. By the mid-20th century, a two-tiered emergency health care system existed in which the properly insured received better care than the poor. To mitigate the situation, in 1946 Congress enacted the Hill Burton Act requiring any hospital receiving federal funds for construction or other expenses to open its doors to all people residing within its territorial area. The statute lacked any real means of enforcement leading to poor compliance.

Over the course of the 1960s and 1970s, the number of civil legal actions taken against hospitals that denied emergent medical treatment to indigent patients grew dramatically. As a result, important legal theories emerged as to how and why hospitals could be held liable for withholding medical care. Essentially, these theories held that any hospital presenting itself as a place offering emergency care must provide that service competently and in a timely fashion to anyone in the public who relied on such advertisement in seeking emergent treatment during a time of need. Paralleling this development was the evolving concept that any hospital receiving public moneys through such programs as Medicare and Medicaid reimbursement in

turn held a duty to serve all sectors of the public equally. Ultimately, these concepts culminated in the 1986 enactment of EMTALA. Many amendments over the years have sharpened the focus and increased both the scope and the enforcement powers of the statute.

4. As a physician, can I personally be penalized for an EMTALA violation?
Yes. Most provisions of EMTALA apply to hospitals. A hospital that has more than 100 beds may be fined up to $50 per violation (fewer than 100 beds may be fined up to $25,000). However, there are a few provisions which apply to physicians. For example, if a physician fails to respond to an emergency situation when he or she is assigned as the on-call physician, a penalty may be imposed. A physician, who signs a certification in support of a patient transfer, is liable if he or she knew or should have known, the certification was false.

5. Will my malpractice insurance cover me for an EMTALA violation?
Probably not. Malpractice insurers generally will not cover monetary sanctions imposed for an infraction of the statute. As a result, the EMTALA penalties amount to a major out-of-pocket expense for the practitioner. Another important difference is that EMTALA is not intended to police standards of medical care per se, but rather to ensure that every patient is treated equally without regard to ability to pay. The patient does not have to suffer damages or have a poor outcome, nor does a practitioner have to commit negligence for a physician or a hospital to be cited for an EMTALA violation. If a patient has a poor outcome from treatment and alleges malpractice in the state courts, EMTALA is invoked only if it can be proven that the care was substantially different from what the hospital would provide uniformly to any other patient presenting with similar complaints and circumstances.

6. Does EMTALA apply when a patient in need presents to any part of a hospital's campus, even if it is not an ED?
Yes. If an individual presents anywhere on hospital property, an EMTALA obligation on the part of the hospital may be triggered if the individual requests examination or treatment for an emergency medical condition or if a prudent layperson would believe that the individual is suffering from an emergency medical condition. The term *hospital property* means the entire main hospital campus including the parking lot, sidewalk, and driveway or hospital departments, including any buildings owned by the hospital that are within 250 yards of the hospital. The patient must be moved to a dedicated ED within the hospital to receive an appropriate MSE.

7. What is a dedicated ED?
A hospital location is defined as a dedicated ED if it meets any one of three criteria: (1) it is licensed by the state to function as an ED, (2) it holds itself out to the public as a place providing care for emergent medical conditions on an urgent basis without requiring a previously scheduled appointment, or (3) if a representative sample of its patient population seen over the previous year demonstrated that at least one third of all outpatient visits were for urgent patient complaints that did not require a previously scheduled appointment.

8. Is a hospital still obligated under EMTALA to medically screen and stabilize any patient presenting to an ambulance it owns and operates?
No. As long as the hospital-owned ambulance operates under community-wide EMS protocols or EMS protocols mandated by state law that direct the ambulance to transport patients to the closest appropriate facility.

9. How does EMTALA define a proper MSE?
An MSE is not an isolated event. It is an ongoing process, conducted by qualified medical personnel, that typically begins with triage, but can involve a wide spectrum of actions ranging from a brief history and physical examination to the performance of diagnostic studies and

procedures and the evaluation by any on-call consultant normally available to the dedicated ED. Triage helps to prioritize the order in which individuals will be seen by qualified medical personnel. The MSE must be appropriate to the individuals presenting signs and symptoms, as well as the capability and capacity of the hospital. The simple answer: the adequate screening of a patient for an emergency medical condition using the resources normally available to the hospital's ED. But the complex reality is that those resources are stated to include any laboratory studies; radiologic examinations such as computed tomography (CT), magnetic resonance imaging (MRI), or angiography; and the services of any on-call consultant normally available to the dedicated ED. Consequently, an adequate MSE can range from a quick history and physical to confirm the presence of an upper respiratory tract infection to a complex work-up involving multiple tests, diagnostic procedures, consultations from specialists, and hospital admission for further evaluation and treatment.

10. Who can perform the MSE?

EMTALA states simply that "qualified medical personnel" (QMP) must perform the MSE. A nurse or mid-level provider may perform the MSE if the hospital's governing board sets forth in the bylaws or hospital rules and regulations that they are qualified to perform screenings for the hospital. The qualified medical personnel must have a job description for this role, qualifications, competencies, and a formal designation to perform MSE in their personnel file.

11. When has the MSE been satisfactorily completed under EMTALA?

An individual is considered stabilized if the treating physician or designated QMP attending to the individual in the ED has determined, with reasonable clinical confidence, that the emergency medical condition has been resolved. Once the emergency medical condition is resolved, the individual may be discharged home with follow-up care instructions, admitted for ongoing care, or transferred to another facility. Psychiatric patients are considered stable when they are protected and prevented from injuring or harming themselves or others. EMTALA ceases to apply once the hospital admits an individual as an inpatient. Importantly, that cessation applies to patients formally admitted to the hospital but who may be boarded in the ED awaiting an inpatient bed.

12. Is it an EMTALA violation if the patient decides to leave against medical advice before the MSE is complete?

It depends on when during the triage and evaluation process that the patient decides to refuse care, and on his or her competency to do so. If, during the course of the MSE, a patient refuses further evaluation and treatment after discussion of the potential risks of such a decision, the patient is considered to have withdrawn the initial request for evaluation, and EMTALA no longer applies. The burden of proof falls on the hospital and the treating physician, however, to demonstrate that no coercion was used to dissuade the patient from consenting to further treatment with suggestions or statements that the continued care could be prohibitively expensive. Proper documentation is essential. The medical record should reflect that screening, examination, or treatment were offered by the hospital prior to the patient's refusal.

A more difficult situation arises when a patient is triaged to the waiting room and then decides to leave before being formally evaluated in the ED. On the surface, this situation can be interpreted as the patient withdrawing the initial request for medical evaluation. EMTALA and the courts have focused considerable attention on the potential for inequity in triage practices, with the uninsured or undesirable patient being subjected to long waiting times in the hopes that he or she will simply leave. In such situations, the hospital must be able to prove that no different standard of triage was used and that a reasonable effort was made to call the patient back to the ED to address the initial complaint.

13. What is meant by *transfer* under EMTALA?

EMTALA does not simply deal with patient transfers and the transferring facilities. EMTALA defines *transfer* as the movement of a patient away from the hospital, not simply as the act of transporting a patient to another hospital. By this definition, even a patient sent home from the ED

is considered to have been transferred under the statute. If such a patient is subsequently found to have been discharged in unstable condition, claim of an EMTALA violation could be made.

14. When does EMTALA say it's OK to transfer a patient?

If a patient is deemed stable (i.e., an emergency medical condition is no longer present, and no significant medical deterioration is likely during or after the transfer), a transfer can proceed without the statute being applicable. EMTALA applies only to the transfer of unstable patients. Unstable patients can be transferred under the following conditions.

- The patient requests the transfer. In that case, an informed request for the transfer must be signed by the patient, and it is important for the hospital and the treating physician to document that a discussion of cost did not enter into the patient's decision to ask for a transfer.
- An unstable patient needs to be moved because the initial facility lacks the capability or the resources to treat the emergent condition adequately. This might occur when a multi-trauma patient presents to a small rural ED and requires transfer to a Level I trauma center to receive proper care. Similarly, a patient with a complicated hand injury who presents to an ED with no hand specialist on call may need to be transferred to a facility capable of providing that service. The expected benefits of the transfer outweigh the risks of the transfer.

15. List the requirements for transferring an unstable patient.

- A physician must certify that the benefits of the transfer outweigh the risks and that when possible this has been discussed with the patient or responsible party.
- Every effort shall be made to minimize the risk involved in the transfer in terms of proper treatment before the patient's departure.
- The receiving facility has accepted the patient and has the capacity and capability to treat the emergency medical condition.
- The receiving facility has been provided with all medical records related to the emergency condition that the patient presented.
- The transfer is conducted with qualified personnel and proper transportation, including the use of necessary and medically appropriate life support measures.

KEY POINTS: REQUIREMENTS WHEN TRANSFERRING AN UNSTABLE PATIENT

1. Physician certifies that benefits of transfer outweigh the risks.
2. Minimize risks prior to transfer.
3. The receiving facility has accepted the patient and has the capacity and capability to treat.
4. Medical records related to the emergency medical condition are copied and sent with the patient (including diagnostic imaging).
5. The transfer is accomplished with qualified personnel and appropriate equipment.

16. Can an on-call consultant refuse to see an unstable patient?

No. If an on-call physician fails or refuses to respond or come to the hospital in a timely fashion (i.e., within a reasonable time under the circumstances or within the time frame established by the hospital's medical staff bylaws), the hospital and the on-call physician may be in violation of EMTALA. If the on-call physician does not respond, the emergency medicine physician treating the patient must decide at what point it is appropriate to transfer the unstable patient to a facility with the capability of treating the emergent medical condition.

In this circumstance, the emergency medicine physician transfers the patient without personally violating EMTALA. Each hospital must have written policies and procedures in place to respond to situations in which a particular specialty is not available or the on-call physician does not respond, and the emergency physician must document the name and address of the consultant who failed to treat the patient on the transfer form.

17. How is the hospital's on-call list determined?

Hospitals have flexibility in determining on-call coverage for their hospitals. However, they must ensure that they are providing sufficient on-call resources to meet the needs of their community. Hospitals must maintain a list of physicians who are on call to stabilize an individual with an emergency medical condition after the initial MSE. A hospital must have written policies and procedures that clearly define the responsibilities of on-call physicians to respond, examine, and treat patients with an emergency medical condition.

18. Can a hospital refuse to accept a transfer under EMTALA?

A receiving hospital cannot refuse an appropriate transfer from a referring hospital within the boundaries of the United States if they have the capacity and capability to treat the patient.

19. If I receive an inappropriate transfer at my hospital, do I have an obligation to report an EMTALA violation?

EMTALA states that any hospital that receives an inappropriate transfer must report the suspected EMTALA violation within 72 hours or face penalties. This is, however, an obligation of the hospital, not of an individual physician.

JOINT COMMISSION (JC)

20. What is JC?

The JC is an independent, not-for-profit organization that evaluates and accredits health care organizations and programs. The JC origins date to 1917, when the American College of Surgeons (ACS) developed the *Minimum Standards for Hospitals* in an effort to establish basic national standards to be met by every hospital operating in the United States. The following year the ACS began on-site inspections to ensure that hospitals met the minimum requirements. Decades later, the ACS, the American College of Physicians (ACP), the American Hospital Association (AHA), the American Medical Association (AMA), and the Canadian Medical Association joined to create the Joint Commission on Accreditation of Hospitals (JCAH) dedicated to further defining a set of standards recommended as essential to the safe and effective delivery of health care by hospitals throughout the nation.

In 1965, Congress passed the Social Security Amendments that included a provision that empowered JCAH by linking each hospital's eligibility to participate in the Medicare program with accreditation by JCAH. This authority allows private, national accreditation organizations to *deem* that an organization is compliant with certain Medicare requirements. Over the years, the standards changed to represent optimal achievable levels of quality, rather than minimum essential levels of quality. As the scope of the organization expanded to include accreditation of clinical laboratories, ambulatory care centers, home health networks, and managed care organizations, the organization changed its name to the Joint Commission on Accreditation of Healthcare Organizations (JCAHO) and, finally, to the JC on January 1, 2007. The JC's mission is to continuously improve health care for the public, in collaboration with other stakeholders, by evaluating health care organizations and inspiring them to excel in providing safe and effective care of the highest quality and value. Currently, the JC evaluates and accredits more than 16,000 health care organizations and programs in the U.S.

21. What are the standards and performance measurements that the JC requires?

The JC collaborates with health care experts, research and quality organizations, providers, performance improvement experts, purchasers and consumers to develop Hospital

Accreditation Standards that focus on an organization's ability to provide safe and high quality care in a safe environment. The *Comprehensive Accreditation Manual* has 17 chapters that include standards and performance in such areas as emergency management, patient care, medication management, infection prevention and control, the record of care, medical staff, and the National Patient Safety Goals (NPSG).

The NPSG became effective in 2003, and were established to help organizations address specific areas of concern in patient safety. The NPSG highlight problematic areas in health care, and focus on system-wide solutions to improve safety and prevent adverse patient outcomes. Annual review and update of the goals is overseen by an expert panel that has hands-on experience addressing patient safety issues in a variety of health care settings. The NPSG often relate to media-grabbing issues such as hospital-acquired infections, patient suicide in a hospital, and wrong-site surgery. Examples of the NPSG include improving the accuracy of patient identification using two patient identifiers to reliably match the patient to the service or treatment provided; standardization of handoff communications (inadequate communication is the leading cause of sentinel events); medication safety; and reducing health-care–associated infections. The NPSG continue to evolve and require greater attention and more resources. They are increasingly important for patient safety and are a critical focus of the accreditation process.

22. How is compliance with the standards evaluated and enforced?

The JC conducts unannounced, on-site surveys that occur 18 to 39 months after the previous unannounced survey. Surveyors are trained and certified in quality-related performance improvement. Their responsibility is to evaluate the hospital's performance and actual care processes using the tracer methodology. The tracer methodology evaluates the patient experience, using the patient's record as a roadmap to move backwards from the patient's current hospital location to their point of access into the hospital. In addition to observing and evaluating the direct care provided to patients, the surveyors scrutinize operational systems that cross all boundaries in the hospital and influence the safety and quality of patient care. Chart review; interviews with staff, patients and families; observation of the processes of care, compliance with the NPSG, and system tracers are central features of the site survey.

To earn and maintain JC accreditation, a hospital must maintain continuous compliance with the JC requirements. In the current, complex health care environment, hospitals are required to meet a variety of accrediting, regulatory, and licensing requirements. The burden is significant and requires organizational commitment. Whenever feasible, hospitals should embed best practice into daily work to ensure compliance; and in an effort to improve operational systems, standard work, computerized provider order entry, hand-held personal digital assistants, bar-coded patient bracelets, *smart* monitors, computerized decision support and electronic medical records are tools that should be considered to promote patient safety and quality of care.

23. What is a sentinel event?

A sentinel event is defined by the JC as "an unexpected occurrence involving death or serious physical or psychological injury or the risk thereof." A sentinel event requires immediate attention, investigation, and response. Not all sentinel events occur as a result of a medical error. An appropriate response to sentinel event is to conduct a timely, credible, and thorough Root Cause Analysis (RCA). An RCA is a process defined by the organization that facilitates the evaluation and identification of the fundamental reason for variation in performance leading to occurrence and sentinel event. The outcome of an RCA should be an action plan designed to implement improvements and reduce risk.

24. How do the JC standards influence the practice of emergency medicine?

As the pressures of increasing patient volume, overcrowding, patient boarding, increasing complexity and limited resources mount, so do the challenges to maintain safe, high-quality patient care in the ED setting. More than 50% of the standards relate directly to patient safety,

making the Joint Commission standards relevant to the ED. Assessment and treatment of pain, emergency preparedness, infection control and prevention, safe medication use, conscious sedation, monitoring restraint use, patient rights, staffing, staff competency, ED security, cultural and linguistic diversity, and standardized hand offs are all areas of focus for the Joint Commission in the ED.

Additionally, there is an expectation that the record of care (patient chart) functions as both a historical record of a patient's episode of care, and a method of communication between care providers. The record needs to tell the story in order to aid in clinical decision making. Since 2005, in keeping with current challenges in health care, there is a Leadership standard that focuses on patient flow and overcrowding in the ED. The JC requires that hospital leadership manage patient flow throughout the hospital to minimize ED overcrowding and minimize delays in care delivery. Leadership must plan, measure, and guide processes to improve patient flow processes and must have plans in place to care for admitted patients in the ED.

HEALTH INSURANCE PORTABILITY AND ACCOUNTABILITY ACT (HIPAA)

25. What is HIPAA?

HIPAA is the *Privacy Rule*. Enacted by Congress in 1996, HIPAA was designed to protect individuals from the unauthorized or inappropriate use of their personal health information. The act's privacy regulations went into full force on April 14, 2004. HIPAA applies to all Covered Entities, public or private, that create, store, or transmit health information pertaining to specific individuals. This includes information in oral, written, or electronic form. HIPAA not only details when and how personal health data may be accessed and shared but also delineates standard transaction formats and data code sets that must be used in transferring such information.

26. Where did the Privacy Rule come from?

The U.S. Department of Health and Human Services (HHS) issued the Privacy Rule to implement the requirement of the Health Insurance Portability and Accountability Act of 1996. Congress had 3 years to enact privacy legislation and when this time passed HHS developed the Privacy Rule that was published in late 2000. The idea of the Privacy Rule is to have patients' medical records protected from anyone who doesn't have consent to review those records. This is coupled with allowing the information to flow between individuals or institutions charged with the patient's care. The Office for Civil Rights (OCR) is responsible for implementing and enforcing the Privacy Rule. In the 23-page paper published by OCR, titled "Summary of the HIPAA Privacy Rule," the OCR clearly states that "This is a summary of key elements of the Privacy Rule and not a complete or comprehensive guide to compliance. Entities regulated by the Rule are obligated to comply with all of its applicable requirements and should not rely on this summary as a source of legal information or advice."

27. What prompted the enactment of such a statute?

Patient privacy and the confidentiality of the physician-patient relationship have been recognized as fundamental ethical and moral obligations in medicine since the time of Hippocrates. With the rise of informatics and the evolution of medical care, individual patient information is now often shared among numerous practitioners, quality assurance auditors, billing coders, and third-party payers. As a result, the potential for unauthorized or inappropriate access to patients' personal information has escalated exponentially. HIPAA is intended to delineate the manner in which personal health data can be accessed, by whom, and for what reason.

28. What is protected health information (PHI)?

PHI is all information pertaining to an individual's medical or psychiatric status, treatment, or payment for health-related services. PHI is linked to specific patients by individually identifiable health information, which HIPAA defines as the person's name; specific contact

information; place of residence by geographic subdivision smaller than the state; Social Security, medical record, or specific account numbers; photographs; biometric identifiers such as finger prints or voice recognition; or any other unique identifier characteristic or code.

29. What is the difference between the use and the disclosure of PHI?
HIPAA defines use of PHI as the sharing, employment, application, utilization, examination, or analysis of PHI within the Covered Entity that maintains the PHI. In general, use of PHI within the Covered Entity for treatment, payment, and normal health care operations without the individual's consent is permissible under HIPAA. The sharing of PHI among physicians, nurses, or other health care providers involved in the direct care of a patient is considered use and is not restricted under HIPAA. Disclosure is the release of PHI to entities outside of the Covered Entity, such as the press, law enforcement, or marketers. PHI disclosure is much more restricted under HIPAA.

30. According to HIPAA, when is it okay to disclose PHI?
PHI may be disclosed under the following circumstances: with the individual's written consent or for 12 national priority purposes. These include required by law; public health activities; victims of abuse neglect or domestic violence; health oversight activities; judicial and administrative proceedings; law enforcement purposes, (which is limited to six specified conditions); and decedents; cadaveric organ, eye, or tissue donation; research; serious threat to health or safety; essential government functions; and lastly workers' compensation.

31. How is the statute enforced, and what are the penalties for a HIPAA violation?
The Office of Civil Rights oversees enforcement of HIPAA privacy standards. Individuals may lodge HIPAA grievances with the Covered Entity or the federal government. Penalties for an established violation include potential monetary fines and jail sentences for the offender(s). Inadvertent violations carry a $100 fine, not to exceed $25,000 per year. If the violation occurred with the knowledge of the offender, punishment can include fines up to $50,000 and up to 1 year in prison. If the violation was committed knowingly and with false pretenses, potential penalties include fines up to $100,000 and a maximum of 5 years in prison. Violation with the intent to sell or profit from PHI disclosure carries a fine of up to $250,000 and up to 10 years in prison.

32. What steps should be taken to prevent disclosure of PHI in the ED?
Maintaining patient privacy is problematic in a busy, crowded ED. Patients and visitors often overhear discussions pertaining to individuals unknown to them in the normal operation of the department. Such inadvertent disclosures are permissible under HIPAA, provided that the department has taken steps in good faith to minimize the likelihood of their occurrence. Examples of such measures include conducting patient interviews and examinations in individual examining rooms when possible; posting signs reminding staff members of the importance of maintaining patient privacy; removing easily identifiable patient information on chalk boards, computer screens, and x-ray view boxes from open view; and documenting staff training with regard to HIPAA issues.

KEY POINTS: BASIC HIPAA COMPLIANCE IN THE ED

1. Perform interviews and examinations in private areas whenever possible.
2. Remove patient identifiers from highly visible areas.
3. Document staff training with regard to HIPAA requirements.

WEBSITES

The Joint Commission Sentinel Event Policy and Procedure:

www.jointcommission.org?SentinelEvents/policyandprocedure

Journey through the History of the Joint Commission:

www.jointcommission.org/AboutUs/joint_commission_history.htm

Privacy Rule: www.hhs.gov/ocr/privacy/

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WEAPONS OF MASS DESTRUCTION

Aaron M. Eberhardt, MD, and Peter T. Pons, MD, FACEP

1. Why is it important for emergency physicians to be familiar with weapons of mass destruction (WMD)?
Emergency physicians (EPs) play an integral role in the planning, preparation, and response to not only unintentional and natural disasters but also terrorist attacks. Casualities from these types of incidents will largely present to an ED, and EPs are increasingly being looked to to take an authoritative position in the management of a WMD event as well as future policy development.

2. We hear about terrorism all the time. Aren't we ready to respond?
Although almost 10 years have passed since the attacks of September 11, 2001, and much has been done to address the issue of terrorism prevention, EPs must continue to be guided by the recognition that the threat of terrorism will never be fully eliminated and that the ability to respond to these events must be maintained. Terrorists continue to refine their techniques and become more efficient at their craft. As such, the emergency medical community must maintain a clear understanding of chemical, biologic, radiologic, nuclear, and explosive (CBRNE) weapons and the ability to effectively respond to an event.

3. Don't we have HAZMAT teams to deal with nuclear, biologic, and chemical (NBC) attacks?
Traditional HAZMAT events usually occur in a relatively well-confined area and usually with a known substance. This allows the HAZMAT team to contain the agent, decontaminate exposed individuals, and control patient flow as needed. NBC attacks will likely occur in population-dense areas to contaminate many people and create significant hysteria. This will make it almost impossible to manage the number of potential patients through a traditional HAZMAT process. A biological weapons attack does not lend itself to a traditional HAZMAT response at all, as a significant amount of time will likely have passed from release of the agent until recognition of the event.

4. What else is unique about a terrorist attack?
Any site of a terrorist attack will automatically be a crime scene. The management of such an event will require the coordination of multiple federal, state, and local agencies. The impact of media coverage of a terrorist event will also require a strategic plan. In addition, the impact of a terrorist attack extends far beyond the people directly affected physically by the NBC attack. Terrorists have the objective to not only kill and destroy, but also to create fear.

5. What makes a good chemical or biologic weapon (in a terrorist's mind)?
The weapon should create the greatest amount of devastation on its intended target. The goal is to create a weapon that is highly lethal or toxic and easy to disperse over large areas. The chemical or biological agent should be relatively stable in the environment so that it doesn't break down too fast. All of this needs to be packaged in such a way that the agent can withstand the energy transfer that occurs during delivery. Lastly, an *ideal* agent and weapon is relatively easy to obtain and inexpensive to manufacture.

6. What should emergency physicians do to prepare and protect themselves?
Emergency physicians must acquire the requisite knowledge about the clinical effects of these various agents, the symptom complex that occurs with each agent to allow for rapid

recognition, and perhaps, most importantly, the steps to take to protect themselves and ED staff from inadvertent exposure to one of these weapons. The type of personal protective equipment (PPE) needed varies depending upon the type of agent used in the attack.

7. Describe the levels of PPE.

- **Level A:** This suit fully encapsulates the body and prevents water and vapor penetration. Respiratory protection is provided by a self-contained breathing apparatus (SCBA) or supplied air. This level of protection is usually worn for the purpose of rescue, assessing, or mitigating a hazardous material event, often when the specific agent is unknown and may be immediately dangerous to life and health.
- **Level B:** Less protective than a level A suit. This is a full-body chemical suit with more limited vapor protection. Can be combined with a SCBA or supplied air to increase protection against vapor. This suit is usually worn by responders who have identified the material or agent and are conducting rescue operations or further incident assessment.
- **Level C:** This is a full-body chemical suit with respiratory protection provided by an ambient air-purifying respirator. Level C protection is appropriate for hospital personnel involved in decontamination.
- **Level D:** Minimal skin protection and no respiratory protection required.

RADIATION

8. What are the basic physics of radiation?

Atoms consist of a nucleus of protons and neutrons (except for hydrogen, which has no neutrons) surrounded by electrons. A given element may exist in the form of different **isotopes**, which have different numbers of neutrons. Some of these isotopes may emit particles or electromagnetic energy and are considered **radioisotopes**. Protection from these particles or energy is afforded by *shielding, distance*, and *decreased time of exposure.*

9. What are the units of radiation?

See Table 101-1.

TABLE 101–1. UNITS OF RADIATION

Radiation Absorbed Dose (Rad) Gray (Gy)	Measure of the energy deposited into matter (the body) by *ionizing radiation* Being replaced by the International System unit the Gray (Gy) 1 Gy = 100 rads 1 Gy = 1 Joule/Kg The Gy dose is the total amount of energy absorbed per gram of tissue
Sievert (Sv) Radiation Equivalent, man (Rem)	The international unit for radiation equivalency Different types of radiation have different effects on the body. These differences are adjusted by multiplying by a Quality Factor (QF) By definition, Gamma radiation has a QF of 1. 1 Gy of pure gamma radiation = 1 Sv 1 Sv = 100 Rem

10. Describe the different types of radiation and their shielding requirements.
See Table 101-2.

11. What are the types of radiation injury?

- **External irradiation**—when all or a portion of the body is exposed to penetrating gamma radiation from an external source. Significant cellular damage can occur. Following exposure, the patient is not radioactive and can be managed like any other patient with no threat to staff, such as a patient undergoing radiation therapy.
- **Contamination**—Radioactive particulate matter is released into the environment and contaminates the person externally, internally (swallowed or inhaled), or both. PPE should be worn when treating or decontaminating these patients.

TABLE 101–2. DIFFERENT TYPES OF RADIATION AND SHIELDING REQUIREMENTS

Radiation	Description	Shielding
α-particles	Consist of two neutrons and two protons that have been ejected from the nucleus of a radioactive atom A doubly charged particle that loses its energy quickly in matter Generally only dangerous if inhaled or swallowed	Stopped by paper
β-particles	High-energy electrons that are emitted from a nucleus along with an antineutrino Much smaller than α-particles and have only one charge Like α-particles, they can cause damage if swallowed or inhaled. May also cause cellular damage to unprotected skin Largely found in fallout radiation	Interact less with target material Require plastic, glass, or thin metal Some levels of PPE
γ	Not particles but rather *uncharged* pulses of very high-energy electromagnetic radiation No mass or charge and only lose its energy when they collide with the electron shell of target atoms Easily pass through the human body Potential to cause significant cellular damage	Concrete, earth, or dense metal such as lead
Neutrons	Uncharged particles emitted during nuclear detonation; not a fallout hazard About the same mass as a proton but no charge Because of this lack of charge, they interact directly with the nucleus of target atom instead of its electrons. Do not react well with material so they can travel large distances Can cause previously stable atoms to become radioactive	Thick concrete or significant amount of earth

PPE, personal protective equipment.

- **Incorporation—**Refers to the uptake of radioactive material in cells, tissues, or organs. Contamination must occur for incorporation to occur. Incorporation allows for continued internal exposure and long-term injury and illness.

12. What are the different types of attacks?

- **Environmental exposure (also known as simple radiologic device)**—Placement of a radioactive source in a public location or within the food or water supply. Although many people would be exposed with this method, very few would likely be significantly contaminated. This type of attack, however, would generate a significant amount of fear and panic.
- **Radiological Dispersal Device (RDD)**—a device designed to spread radioactive material for the purpose of terrorism by using conventional explosives to disperse the radioactive material; this is referred to as a *dirty bomb*. Most of the damage caused by this sort of weapon would be created primarily by the explosion, as the dissemination of radioactive material would be limited in effect. Exposed or contaminated individuals would be those in close proximity to the blast area.
- **Attack/Sabotage of a nuclear reactor**—could lead to significant release of radioactive material into environment.
- **Nuclear bomb**—Obviously the most potentially devastating attack. The least likely method of attack due to strict security measures of existing stockpiles and the money and technology needed to manufacture a new weapon.

13. Describe the three acute radiation syndromes (ARS).

- Bone marrow (hematopoietic syndrome)—This syndrome is caused by damage to stem cells in the bone marrow resulting in a reduction in cell lines. Symptoms include bleeding and infection (low platelets and leukocytes). Usually occurs after exposure to between 0.7 and 10 Gy (70– 1,000 rads).
- Gastrointestinal (GI) syndrome—Irreversible destruction of the GI lining causing nausea, vomiting, and diarrhea. Survival is extremely unlikely as death is caused by overwhelming sepsis and electrolyte disturbances. Usually occurs after exposure to between 6 and 10 Gy (600–1,000 rads).
- Central nervous/cardiovascular syndrome—Symptoms include confusion, seizures, and coma. Death usually occurs within 3 days as a result of circulatory collapse and increased intracranial pressure caused by edema, vasculitis, and meningitis. The full syndrome will usually occur with a dose greater than approximately 50 Gy (5,000 rads) but can occur at lower levels. This is uniformly fatal and, in a mass casualty situation, such patients should be triaged to the *expectant* category.

14. Describe the four stages of ARS.

- **Prodromal (Initial) stage:** Symptoms include loss of appetite, nausea, vomiting, and diarrhea. Symptoms occur minutes to days after the exposure. In general, the more rapid the onset of symptoms, the greater the radiation dose received by the victim and the poorer the outcome.
- **Latent period:** Resolution of symptoms experienced in the initial stage with the patient appearing relatively well. Can last hours to approximately 2 weeks.
- **Manifest illness stage:** Symptoms will vary depending on radiation dose. For doses ranging from 1 to 8 Gy (100 to 800 rad), symptoms are the result of suppression of the hematopoietic system (decreased leukocytes and platelets) and include infection and bleeding. For doses over 8 Gy (800 rads), the primary effects are on the lining of the intestines leading to diarrhea, fever, sepsis, and electrolyte disturbance.
- **Recovery or death:** Survival is highly unlikely with doses exceeding 10 Gy (1,000 rad).

15. All these numbers are great, but what is the bottom line?

- Exposure to 1 Gy is the threshold for nausea and vomiting, but no deaths from acute radiation should occur at this level.
- Exposure to 3.5 Gy will be 50% lethal at 60 days if untreated.
- Exposure to 6.0 Gy is 100% lethal if untreated at 60 days.

TABLE 101–3. ROLE OF THE ABSOLUTE LYMPHOCYTE COUNT

Minimal Lymphocyte Count within 48 Hours of Exposure	Estimated Absorbed Dose (Gy)	Prognosis
1,000–3,000	0–0.5	Likely no injury
1,000–1,500	1–2	Significant but good prognosis
500–1,000	2–4	Severe, may survive
100–500	4–8	Very severe, likely die
<100	>8	Will likely die

From Koenig KL, Goans RE, Hatchett RJ, et al: Medical treatment of radiological casualties: current concepts. *Ann Emerg Med* 45:643–652, 2005.

16. How is the absolute lymphocyte count helpful in evaluating ARS?
See Table 101-3.

17. What treatment options are available for radiation exposure?
A complete primary and secondary survey must be done to ensure that no other, acutely life-threatening injuries exist. After appropriate triage and decontamination, supportive care becomes the foundation of treatment. Local radiation injury should be treated in the same manner as burns with 18% of total body surface area being considered a major burn. Treatment of whole body irradiation becomes more complicated with numerous adjunctive medications that may be useful, including KI, diethylenetriaminepentaacetate (DTPA), Prussian blue, and Neupogen. Such patients should be considered to be immunocompromised and treated as such. There is an excellent overview of weapons of mass destruction in general and these treatments in particular on the Centers for Disease Control and Prevention (CDC) website.

18. What's the deal with potassium iodine (KI) tablets?
Following a radiological or nuclear event, it is possible that radioactive iodine may be released. This radioactive iodine can lead to internal contamination through inhalation or ingestion. The thyroid gland will then absorb this radioactive iodine, which can lead to irreversible destruction of the gland. KI tablets contain stable (non-radioactive) iodine and, if given before exposure to radioactive iodine, can saturate the thyroid gland. This effectively blocks the thyroid from absorbing the radioactive iodine. The CDC website has an informational sheet about KI tablets including indications and appropriate dosing. This information can be found at: http://www.bt.cdc.gov/radiation/ki.asp

CHEMICAL WEAPONS

19. List the characteristics of chemical weapons.

- Volatility describes the tendency of a liquid to evaporate into a gas. Most chemical weapons are liquids at normal atmospheric pressures and temperatures and are dispersed as fine liquid droplets after detonation. The more volatile a chemical is, the more quickly it will evaporate (e.g., phosgene and cyanide). Less volatile agents will remain liquids (e.g., VX and sulfur mustard). Also, all agents except hydrogen cyanide are heavier than air and will concentrate in low-lying places.
- Persistence is inversely related to volatility. Agents are categorized as non-persistent or persistent based on their ability to vaporize in less than or greater than 24 hours respectively. Persistent chemicals will remain on objects and patients longer, creating the potential for ongoing exposure and contamination.

- Toxicity is the ability of an agent to cause harm to a person. The usual measurement is the concentration-time product (Ct). This is the product of the concentration in the air times the amount of time a patient is exposed. One can go further and look at the LCt50, which is the Ct of a vapor or aerosol that will kill 50% of those exposed to the agent.
- Latency describes the time delay between when a patient is exposed to an agent and the clinical manifestation of signs and symptoms. Health care providers must be aware of this principle because victims who do not show any clinical signs or symptoms may still be exposed and need to be decontaminated and treated.

20. What are the different classes of chemical weapons?
See Table 101-4.

21. Describe the pathophysiology and clinical symptoms caused by nerve agents.
Nerve agents are chemicals whose effects are similar to the organophosphate insecticides.

TABLE 101–4. CLASSES OF CHEMICAL WEAPONS

Class	Description	Signs and Symptoms	Examples and Designation
Blister agents/ vesicants	Damage cellular components and create blisters on dermal and mucosal surfaces minutes to hours later	Dyspnea, dermal irritation and pain, vesicles, conjunctivitis, possibly severe respiratory compromise	Lewisite (L) Nitrogen mustard Phosgene oxime Sulfur mustard
Blood agents	Absorbed into the bloodstream and interfere with aerobic metabolism	Dyspnea, chest pain, anxiety, flushed skin	Arsine (SA) Carbon monoxide Cyanogen chloride (CK) Hydrogen cyanide Potassium cyanide (KCN) Sodium cyanide (NaCN) Sodium monofluoroacetate (compound 1080)
Caustics	Directly burn and irritate mucous membranes, skin, and eyes	Burning and severe irritation, pulmonary irritation if inhaled	Hydrofluoric acid
Choking/ pulmonary agents	Irritate the lining of the lungs and throat, causing edema of the mucous membranes	Coughing, dyspnea, dysphagia, chest pain, eye irritation, burning sensation in throat	Ammonia Bromine (Br) Chlorine (Cl) Hydrogen chloride Phosgene (CG) Sulfuryl fluoride
Incapacitating agents	Cause altered mental status and affect victim's ability to think clearly	Altered mental status, anticholinergic syndrome (BZ), opioid toxidrome	3-quinuclidinyl benzilate (BZ) Fentanyl (aerosolized)

TABLE 101–4. CLASSES OF CHEMICAL WEAPONS—cont'd

Class	Description	Signs and Symptoms	Examples and Designation
Nerve agents	Inhibit acetylcholinesterase, thereby interfering with nerve transmission	Cholinergic toxidrome, salivation, lacrimation, paralysis	Sarin (GB) Soman (GD) Tabun (GA) VX
Riot control/ tear gas	Very irritating but nonlethal agents used for crowd control and riot suppression	Mucous membrane irritation. Lacrimation, rhinorrhea, coughing, sneezing	Bromobenzylcyanide (CA) Chloroacetophenone (CN) Chlorobenzylidenemalone nitrile (CS) Chloropicrin (PS) Dibenzoxazepine (CR)
Vomiting agents	Ocular, nasal, and respiratory tract irritation; GI upset and vomiting	Vomiting starting minutes to hours after exposure	Adamsite (DM)

GI, gastrointestinal.
From *Emergency preparedness and response: chemical emergencies*, Centers for Disease Control: www.bt.cdc.gov/chemical

The nerve agents inhibit acetylcholinesterase at the postsynaptic nerve receptors. This leads to excessive acetylcholine accumulation and causes overstimulation of muscarinic and nicotinic receptors in the parasympathetic nervous system (PNS) and central nervous system (CNS), resulting in a clinical cholinergic toxidrome. Stimulation of muscarinic receptors causes activity of exocrine glands (e.g., salivation, bronchorrhea). Stimulation of the nicotinic receptors is responsible for muscle fasciculations, flaccid paralysis, hypertension, and tachycardia.

The clinical toxidrome is complex, involving many different organ systems. One of the best ways to remember the toxidrome is by the SLUDGE mnemonic which stands for: Salivation, Lacrimation, Urination, Defecation, GI symptoms, and Emesis. The patient can have life-threatening bronchorrhea and bronchospasm. The CNS symptoms can include seizure, coma, or apnea.

22. How deadly are nerve agents?

The dose of VX, which will kill half of exposed victims (LD50), is 10 mg (skin exposure) on a 70-kg man. To give you a frame of reference, this means that a drop of VX that is large enough to cover two columns of the Lincoln Memorial on the back of a U.S. penny is enough to kill half of exposed victims who have this amount placed on exposed skin.

23. What is the treatment for nerve agent toxicity?

Treatment is based on a three-pronged approach. Atropine is given to counteract the muscarinic effects, thereby drying up secretions and improving ventilation. Pralidoxime chloride (2-PAM) reverses the nicotinic effects of nerve agents, thereby reversing paralysis. When nerve agents combine with ACHE, a process called "aging" takes place during which a permanent covalent bond is formed and the enzyme is permanently deactivated. To be effective, 2-PAM must be

TABLE 101–5. TREATMENT FOR NERVE AGENT TOXICITY

Patient Age	Mild to Moderate Symptoms	Severe Symptoms
Infant (0–2)	Atropine: 0.05 mg/kg IM or 0.02 mg/kg IV 2 PAM: 15 mg/kg IV slowly	Atropine: 0.1 mg/kg IM or 0.02 mg/kg IV 2 PAM: 15 mg/kg IV slowly
Child (2–10)	Atropine: 1 mg/kg IM 2 PAM: 15 mg/kg IV slowly	Atropine: 2 mg/kg IM 2 PAM: 15 mg/kg IV slowly
Adolescent (10–18)	Atropine: 2 mg IM 2 PAM: 15 mg/kg IV slowly	Atropine: 4 mg IM 2 PAM: 15 mg/kg IV slowly
Adult	Atropine: 2–4 mg IM 2 PAM: 15 mg/kg (1.0 gram) IV slowly	Atropine: 6 mg IM 2 PAM: 15 mg/kg (1.0 gram) IV slowly

Notes:

1. May repeat atropine (2 mg IM or 1 mg IM for infants) every 5–10 minutes until secretions dry and breathing become more comfortable.
2. Phentolamine can be used for 2PAM-induced hypertension (5 mg IV for adults and 1 mg IV for children)
3. Diazepam can be used for seizures.

IM, intramuscularly; IV, intravenously.
From Department of Health and Human Services. Agency for Toxic Substances and Disease Registry. Medical Management Guidelines for Nerve Agents: Tabun (GA); Sarin (GB); Soman (GD); GF; and VX. http://www.atsdr.cdc.gov/MHMI/mmg166.html

administered before this happens. This time ranges from 2 minutes for Soman (GD) to 48 hours for VX. Finally, seizures are treated with diazepam. **See Table 101-5 for specific dosages.**

BIOLOGICAL AGENTS

24. What is bioterrorism?

Bioterrorism is an attack in which there is the deliberate release of viruses, bacteria, or biologic toxins used to cause illness or death in people, animals, or plants.

25. Have terrorists really used biological agents?

Biological weapons have been used in warfare since antiquity. In the 14th and 15th centuries, warring armies would hurl plague-infected corpses over the walls of cities they were attempting to conquer. Accounts exist of biological weapons being used in both World War I and World War II. Many terrorist groups have used biological weapons as well. In 1984, 750 people in Oregon became sick after eating at salad bars in four different restaurants that had been intentionally laced with *Salmonella* by the Bagwan Sri Rajneesh sect. Beginning September 18, 2001, anthrax spores were sent via the U.S. mail system. This attack led to 22 cases of inhalational and cutaneous anthrax, including five fatalities.

26. Doesn't the manufacturing of biological agents require a lot of money and sophisticated equipment?

The manufacture and dispersal of a biological agent is significantly easier to accomplish than a nuclear attack. The previously mentioned incidents with *Salmonella* and anthrax demonstrate how easily this can happen.

27. How are biological attacks different from exposure to radiation or chemical agents?

A biological attack creates two significant challenges not present with a radioactive exposure or chemical attack. First, the development of symptoms can be delayed. Second, symptoms may initially be very non-specific and initially be attributed to a less serious cause. These factors will likely lead to a significant delay in recognition and therefore the ability to appropriately respond to a biological attack. If the agent used is highly communicable, an epidemic could develop very quickly.

28. How does the CDC categorize biological agents?

The CDC prioritizes biological agents into three categories (A, B, and C), based on the characteristics of the agents, including ease of dissemination and transmission and ability to create a significant negative impact on public health infrastructure. The U.S. Public Health System and primary health care providers, including emergency physicians, must be prepared to address various biologic agents, including pathogens that are rarely seen in the United States.

- Category A:
  - Definition: High-priority agents include organisms that pose a risk to national security because they can be easily disseminated or transmitted from person to person, result in high mortality rates and have the potential for major public health impact, might cause public panic and social disruption, and require special action for public health preparedness.
  - Examples: Anthrax (*Bacillus anthracis*), botulism (*Clostridium botulinum* toxin), Plague (*Yersinia pestis*), smallpox (Variola major), tularemia (*Francisella* tularensis), viral hemorrhagic fevers (filoviruses [e.g., Ebola, Marburg] and arenaviruses [e.g., Lassa, Machupo])
- Category B:
  - Definition: Second highest priority agents include those that are moderately easy to disseminate, generally result in moderate morbidity rates and low mortality rates, and require specific enhancements of CDC's diagnostic capacity and enhanced disease surveillance.
  - Examples: Brucellosis (*Brucella* species), epsilon toxin of *Clostridium perfringens*, food safety threats (e.g., *Salmonella* species, *Escherichia coli* O157:H7, *Shigella*), Glanders (*Burkholderia mallei*), melioidosis (*Burkholderia pseudomallei*), psittacosis (*Chlamydia psittaci*), Q fever (*Coxiella burnetii*), ricin toxin from *Ricinus communis* (castor beans), staphylococcal enterotoxin B, typhus fever (*Rickettsia prowazeki*), viral encephalitis (alphaviruses [e.g., Venezuelan equine encephalitis, eastern equine encephalitis, western equine encephalitis]), water safety threats (e.g., *Vibrio cholerae*, *Cryptosporidium parvum*)
- Category C:
  - Definition: Third highest priority agents include emerging pathogens that could be engineered for mass dissemination in the future because of availability, ease of production and dissemination, and potential for high morbidity and mortality rates and major health impact
  - Examples: Emerging infectious diseases such as Nipah virus and hantavirus

29. What are the general descriptive characteristics of biological agents?

See Table 101-6.

30. Give me the basics about anthrax.

Bacillus anthracis is an encapsulated, Gram positive, spore-forming bacterium. The spores are highly resistant to environmental factors, allowing them to survive for decades. Anthrax can manifest in three forms: inhalational, GI, and cutaneous. After a biological attack, the aerosolized anthrax spores are inhaled, taken up by macrophages in the lung, and transported

TABLE 101–6. GENERAL CHARACTERISTICS OF BIOLOGIC AGENTS

Infectivity	The ability of an agent to enter, multiply, and survive in a host ID_{50} is the dose that would infect 50% of an exposed population
Virulence	The relative severity of the disease Different strains of the same agent can cause varying severities of disease
Incubation period	Time between exposure and onset of symptoms
Lethality	The ability of an agent to cause death
Contagiousness	Measured by the number of secondary cases occurring after exposure to the primary case
Mechanisms of transmission	Manner by which the disease is transmitted (i.e., respiratory, blood borne, vector borne, food contamination)

From Public health response to biological and chemical weapons: WHO guidance (2004). www.who.int/csr/delibepidemics/biochemguide/en/

to mediastinal lymph nodes where they transform into a vegetative state. Once the bacteria start replicating, they produce toxins that cause edema (edema factor) and hemorrhage and necrosis (lethal factor).

31. What are the signs and symptoms of anthrax?

Cutaneous anthrax begins with an area of localized redness and swelling and progresses to a painless, necrotic black lesion or ulcer. **GI anthrax** presents with stomach pain, fever, diarrhea, and loss of appetite. **Inhalational anthrax** initially is a very nonspecific, flu-like syndrome with fever, nausea, vomiting, muscle aches, and fatigue. This can quickly progress to difficulty breathing, respiratory failure, shock, and death. Of 10 patients presenting with inhalational anthrax in the 2001 attacks, all had fever, chills, malaise, and fatigue. Most of the patients developed cough, chest discomfort, and dyspnea. Chest radiograph abnormalities were universal and included widened mediastinum, pleural effusions, air bronchograms, necrotizing pneumonic lesions, or consolidations.

32. How should I treat anthrax?

Current CDC recommendations for inhalational anthrax include ciprofloxacin and doxycycline, plus one or two additional antibiotics. For cutaneous anthrax, ciprofloxacin and doxycycline also are first-line therapy. A potential for reactivation of latent infection may exist. Therefore, persons with cutaneous anthrax associated with this attack should be treated for 60 days. A recent addition to the Strategic National Stockpile is raxibacumab (ABthrax^{tm}), which is a monoclonal antibody active against the factor produced by anthrax that allows edema factor and lethal factor to enter a cell. Animal studies have shown great promise for benefit of this new medication.

33. What other sources are available to learn more about biological weapons?

The *Journal of the American Medical Association (JAMA)* has a series of articles titled: Medical and Public Health Management Following the Use of a Biological Weapon: Consensus Statements of the Working Group on Civilian Biodefense. They are available through the *JAMA* website:

- Anthrax: jama.ama-assn.org/cgi/reprint/287/17/2236.pdf
- Botulism: jama.ama-assn.org/cgi/reprint/285/8/1059.pdf
- Hemorrhagic fevers: jama.ama-assn.org/cgi/reprint/287/18/2391.pdf
- Plague: jama.ama-assn.org/cgi/reprint/283/17/2281.pdf
- Smallpox: jama.ama-assn.org/cgi/reprint/281/22/2127.pdf
- Tularemia: jama.ama-assn.org/cgi/reprint/285/21/2763.pdf

The CDC also maintains extensive information on biological weapons including case definitions. You can access this information by going to: http://emergency.cdc.gov/bioterrorism/

34. How should I protect myself when I am caring for patients exposed to biological weapons?

Universal precautions should be observed at all times when dealing with a biological event. The following table lists person-to-person transmissibility and isolation requirements by biological agent, when known. If the agent causing the patient's symptoms is unknown, as it likely will be on initial presentation to the ED, strict isolation precautions should be utilized. See Table 101-7.

35. How will I recognize if a biological attack has occurred?

Recognizing a biological attack can be very difficult for the reasons that have already been stated. In general, disease pattern recognition will be vital to the early identification of a biologic event. In 2000, Dr. Paul Rega published a list of *covert assault clues* that can aid clinicians in this process. These covert assault clues are as follows:

- Severe manifestations of disease in previously healthy people
- Greater than normal numbers of patients with fever, respiratory, or GI complaints
- Multiple patients with similar complaints from a common location
- An endemic disease that occurs during an unusual time of year
- An unusual number of rapidly fatal cases
- A greater number of sick or dead animals
- Rapidly rising and falling epidemic curve
- Larger number of patients with severe pneumonia, sepsis, sepsis with coagulopathy, fever with rash, or diplopia with progressive weakness

TABLE 101–7. TRANSMISSIBILITY AND ISOLATION REQUIREMENTS FOR BIOLOGICAL WEAPONS

Illness	Person to Person?	Isolation Required?
Anthrax	No	No
Botulism	No	No
Hemorrhagic fevers	Yes	Yes
Plague	Yes (pneumonic form)	Yes
Smallpox	Yes	Yes
Tularemia	Likely not	No

36. What should I do if I suspect an attack has occurred?

Every emergency provider should familiarize themselves with their hospital's internal disaster plan and reporting process. Early involvement of local and state public health is important for both diagnostic and epidemiologic followup. The CDC provides a mechanism to report an incident as well as helpful phone numbers that may be needed during the management of an incident. This information can be accessed at the CDC's Emergency Preparedness and Response website located at: http://emergency.cdc.gov/

EXPLOSIVES

37. With all these other highly effective and lethal terrorist weapons, are they really still using explosives?

Absolutely. The widespread use of NBC weapons is significantly limited as they are expensive to acquire and difficult to manufacture in an effective dispersal mechanism. Explosive devices are much easier to acquire and are increasingly being improvised to create maximal body counts. In the year 2008, worldwide, over 8,000 people were killed in terrorist attacks by bombing or combined bombing/arson. There has also been a significant increase in "high-fatality attacks" (those killing more than 10 people), many of which are carried out using explosive devices. From 2004 to 2006, approximately 205 people were injured or killed in the United States as a result of criminal bombing incidents.

38. Describe the five blast injury categories after explosions.

- **Primary:** The direct effect produced by contact from the blast shockwave with the body. This creates shear and stress forces on tissues. Typical injuries include tympanic membrane (TM) rupture, blast lung, ocular injuries, and concussions.
- **Secondary:** Injury produced by impact of primary fragments (pieces of the exploding device) or secondary fragments (fragments from the surrounding environment). Typical injuries include penetrating trauma, amputations, or lacerations.
- **Tertiary:** Injuries created when the blast wave propels victims' bodies into objects or large objects strike the body. Typical injuries include crush injuries and blunt trauma.
- **Quaternary:** Effects include burns, inhalational injury, exposure to toxic substances, and injury from environmental contamination that was created as the result of the explosive device.
- **Quinary:** Injuries resulting from additives such as bacteria or radiation (*dirty bombs*).

39. Is there a quick screening method to triage victims of blast injuries?

Otoscopic examination of the TM is a quick (but not foolproof) way to assess the severity of blast injury. The TM can be ruptured by an increase in atmospheric pressure as low as 5 psi above normal. If there is no TM rupture, then the chance of hollow organ injury is significantly lower. It is not zero, however. It was found that in 17 critically injured patients after the Madrid train bombing in 2004, 13 had ruptured TMs, but 4 did not. Obviously, if other symptoms such as shortness of breath are present, one must suspect other injuries.

40. What is *blast lung*?

Blast lung is the term used to describe the significant pulmonary barotrauma after a high-order explosive detonation. The blast wave's impact with the lung causes tearing, hemorrhage, contusion, and edema with resultant ventilation-perfusion mismatch. Blast lung is a clinical diagnosis characterized by respiratory difficulty and hypoxia.

In general, blast lung is treated similarly to any other pulmonary contusion. Patients exposed to a significant explosion who have normal chest radiographs, normal arterial blood gases, and no complaints that would suggest blast lung injury can be considered for discharge after 4 to 6 hours of observation.

DECONTAMINATION

41. What should I know about decontamination?

Decontamination is a critical aspect of the medical management of NBC attacks. The benefits of decontamination are threefold. It protects the patient from continued injury due to residual agent on the patient's clothes or skin, the health care providers from exposure and injury, and the health care facility itself, allowing it to remain open and to care for more patients.

42. How do I decontaminate victims of chemical exposure?

Decontamination of patients exposed to chemical agents should ideally be performed in the prehospital setting. However, hospitals should be prepared to provide decontamination outside of the ED for the patients that will inevitably self-present for evaluation and care. Hospital staff members involved in the decontamination process should utilize Level C protection. Wet decontamination is the method of choice for liquid chemical exposure. Patients should first have their clothes removed, maintaining as much modesty as possibly. Soap and copious amounts of water should then be applied and are considered adequate for decontamination. While a variety of neutralizing solutions, such as dilute bleach solution, have been suggested as decontamination solutions, most are not used in the hospital setting as they require prolonged contact time (15-20 minutes) and have the potential for causing additional skin injury.

43. How do I decontaminate a patient that has been exposed to radioactive material?

Once the patient is removed from the radiological hazardous environment, standard PPE is adequate for decontamination personnel (scrubs, masks, gloves, eye protection, and shoe coverings). Decontamination personnel should be equipped with dosimeters to monitor radiation exposure. Decontamination can then be broken down into two components:

- **Gross Decontamination:** This involves removing all of the patient's clothes and bagging the clothes appropriately. The patient should then be washed with copious amounts of soap and water or the commercially available 0.5% hypochlorite solution. Care must be taken to avoid washing contaminated water towards mucous membranes. Care must also be taken to not abrade the skin as radionuclides can be absorbed through skin abrasions. This process will successfully remove approximately 95% of the contamination.
- **Secondary Decontamination:** This is a meticulous process to ensure the patient is fully decontaminated. Eyes, ears, mucous membranes, and wounds should be swabbed, and the swabs should be analyzed for radioactivity. Additionally, these same areas should be copiously irrigated. The patient's eyes should be anesthetized and copiously irrigated. The ears should be checked for perforated TMs and irrigated copiously if intact. Dentures should be removed and the mouth rinsed copiously without swallowing the rinse water. Wounds should also be irrigated and covered with waterproof dressings to avoid run-off contamination from irrigating other areas. The hospital's radiation safety officer should be involved with this process.

44. How are victims of biologic agent exposure decontaminated?

In most cases, victims of a biologic attack will present when they become clinically ill. This finding indicates that the exposure occurred days earlier, in which case decontamination is not necessary. Only in those instances of recognized powder exposure is decontamination required. As with chemical exposure, decontamination personnel must use appropriate PPE during the decontamination process. Clothing should be removed and then showering with soap and water accomplished.

KEY POINTS: WMD

1. The ideal terrorist weapon is cheap, easy to manufacture, easy to disseminate, and will produce large numbers of casualties.
2. The most likely WMD to be used by a terrorist is a conventional explosive.
3. A chemical agent attack will generally be recognized by having a large number of casualties present with similar symptoms over a short period of time in a relatively small geographic area.
4. In the event of a biological agent attack, all patients should be considered to be infectious until a definitive diagnosis of a nontransmissible agent is confirmed.
5. The most likely radiologic incident will be a dirty bomb, i.e., radiologic dispersal device that involves using a conventional explosive to disperse some radioactive material.

WEBSITES

Anthrax: http://www3.niaid.nih.gov/topics/anthrax/overview.htm

Biological weapons: http://emergency.cdc.gov/bioterrorism/

Bioterrorism agents: http://emergency.cdc.gov/agent/agentlist-category.asp#catdef

CDC's Emergency Preparedness and Response website located at: http://emergency.cdc.gov/

Journal of the American Medical Association: http://jama.ama-assn.org

KI tablets: http://www.bt.cdc.gov/radiation/ki.asp

U.S. Department of Health & Human Services: http://www.hhs.gov/disasters/emergency/manmadedisasters/bioterorism/index.html

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